DISCLAIMER

Medical science is ever changing and advancing subject. To formulate a guideline is to document the prevailing recommendations of experts in the field at the present time. The publishers and authors take no responsibility for the changes that will be made to these guidelines in current or future point in time. The readers are advised to consult authentic referral guidelines/manuals in case of need.
MESSAGE

I am extremely happy to know that the Standard Treatment Guidelines-2018 have been prepared by the State Drug Management Unit, Directorate of Health Services, Odisha, Bhubaneswar in consultation with specialists of all Medical Colleges and peripheral health institutions of Odisha to rationalise medical practice and reduce costs for the public health system. With the publication of this book, the Government affirms its commitment to the level of diagnostics and the package of services that are available at different health institutions of our State. The Guidelines cover all common diseases. The adoption of Standard Treatment Guidelines should be recognised as a significant milestone for providing quality health care and services to the people of our state.

I am sure the guidelines will be useful to the health professionals of our State in making rational clinical decisions.

(Pratap Jena)
Minister
Health & Family Welfare, Law, I & PR
Odisha
MESSAGE

The Standard Treatment Guidelines (STG)-2018 are a huge step forward in treatment of patients in Government hospitals of our State. For want of standard guidance, it becomes challenging for clinicians in Government Health Facilities to observe common treatment protocols demanded by health policies and programmes implemented from time to time. With this objective in mind, the State Drug Management Unit, Directorate of Health Services, Odisha, along with experts and specialists from Government Medical Colleges and peripheral health institutions has done a commendable job in bringing out standard guidelines for treating a majority of common ailments. The STG-2018 are meant to reach out and guide health professionals to manage their routine clinical decisions. Another important objective of STG-2018 is to rationalise use of drugs and optimise the expenditure on treatment of patients, while preserving the much needed quality of care.

I take this opportunity to extend my sincere gratitude and appreciation for the SDMU team & the experts for their valuable input in bringing out an excellent document on treatment guidelines.
MESSAGE

The Standard Treatment Guidelines - 2018 have been prepared by the State Drug Management Unit, Directorate of Health Services, Odisha, Bhubaneswar in consultation with specialists of all Medical Colleges and peripheral health institutions of Odisha with a purpose to rationalise medical practice across the public health system. The adoption of these Guidelines will ensure quality health care services across the state.

I hope the Guidelines prove useful to healthcare professional and set new standards for health service delivery in the State.

(Shalini Pandit)
Mission Director, NHM
Odisha, Bhubaneswar
MESSAGE

I am extremely happy to note that after much awaited, the Standard Treatment Guideline-2018 for different diseases is getting published. This will focus on scientific treatment of diseases with specific drugs. It will be very much beneficial to all Doctors of Medical Colleges as well as peripheral Health Institutions of the State.

I extend my warm greetings and best wishes for the publication of the STG-2018.

(Dr. Braja Kishore Bramha)
Special Secretary to Govt. (MS)
Health & Family Welfare Department
Odisha, Bhubaneswar
MESSAGE

I express my heartfelt congratulations to the State Drug Management Unit, Directorate of Health Services, Odisha for preparing the Standard Treatment Guideline - 2018 in consultation with specialists of all the Medical Colleges and peripheral health institutions of our State. It contains guiding principles for health professionals in better treatment of common diseases.

It will not only set standard protocols but also ensure rational use of drugs.

(Ms. Archana Patnaik, IAS)
Managing Director
Odisha State Medical Corporation Ltd.
I convey my heartfelt congratulations to the State Drug Management Unit, Directorate of Health Services, Odisha for preparing the Standard Treatment Guideline-2018 in consultation with different Specialists of Medical Colleges and peripheral Health Institutions of our State to rationalize medical practice and make the cost less for the public health system.

It will be highly beneficial for all health professionals of our State delivering better health care to the patients using the available resources.

(Dr. Hara Prasad Patnaik)
Director of Health Services, Odisha
Bhubaneswar
MESSAGE

The State Drug Management Unit, Directorate of Health Services, Odisha has done a praise worthy-work in preparing the Standard Treatment Guideline-2018 in consultation with different specialist of all medical colleges and peripheral health institutions of our State. It contains the guiding principles for treatment of common diseases.

I hope this guideline will help in guiding all the professionals for better treatment of diseases.

(Prof. Dr. Sonamali Bag)
Director of Medical Education & Tr.
Odisha, Bhubaneswar
PREFACE

It’s not practically possible to implement the standard recommended treatment provided in the text books or high standard journals in resource constraint settings. This leads to significant variation in practice of medicine in prevalent health care set up. Standard treatment guidelines or protocols provide consensus on evidence based practice that is practicable and feasible at ground level.

Present version of standard treatment guideline (STG) has been prepared after thorough consultation among different levels of health care of the state. The interactive sessions were initiated by two state level workshops conducted at two different occasions on 15-17 November 2016 and 5.8.2017. Many important changes have been incorporated from the last version of STG Odisha. This includes incorporation of many new chapters in trauma and emergency care, chapters from nephrology and neurology depending upon the high prevalence of the disease or conditions in recent years.

Though the guideline has been prepared by most experienced and sort out clinicians of the state, this needs to be balanced with other available information and in the context of individual patient. Thus the books neither restricts clinical flexibility nor prevent any health care personnel form replacing his clinical judgement. Though the broad objective is to develop consensus based on evidence based practice in the different health care level, expert opinion has been sorted for few clinical situations where there is paucity of evidence or not available.

The unique features of STG Odisha 2018 is its presentation style with different sections comprising of clinical features, Lab investigations, Non drug treatment, drug treatment patient education and referral criteria. Reference chapter has been incorporated, in the present edition to provide authenticity of information.

We are hopeful the book will provide a reference material for the health care personals who are working in the remote areas of the state. We are hopeful.
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****
1. MEDICINE
SHOCK

Introduction:
It is a clinical syndrome which is characterized by reduced tissue perfusion leading to imbalance between delivery of oxygen and substrate causing cellular dysfunction.

Classification of Shock:
1. Hypovolaemic shock
   - Hemorrhagic shock
   - Fluid loss from vomiting and/or diarrhoea, e.g. in cholera and other GI infections.
   - Fluid loss in diabetes mellitus, adrenal insufficiency, excessive sweating, exfoliative dermatitis, diabetes insipidus, re-accumulation of ascites after tapping.
   - Sequestration of fluid, e.g. in intestinal obstruction, pancreatitis.
   - Burns.
   - Traumatic/Crush injuries
2. Cardiogenic
   - Acute myocardial infarction
   - Cardiomyopathy
   - Cardiac arrhythmias
   - Mechanical causes, e.g. valvular diseases, outflow tract obstruction, ruptured ventricular septum
3. Distributive Shock
   - Septic shock
   - Anaphylactic
   - Neurogenic
4. Obstructive Shock
   - Cardiac tamponade
   - Tension pneumothorax

Clinical Features
The presentation of shock largely depends upon the etiology. In general, the symptoms include one or more of the following:
- Restlessness
- Dizziness or faintness
- Confusion
• Bluish lips and finger nails
• Chest pain
• Profuse sweating
• Cold and clammy skin
• Hypotension, Tachycardia and Tachypnea
• Low or nil urine output

Investigations:
CBC, blood sugar, serum electrolytes, LFT, serum lactate, ABG (where possible), imaging studies (X-rays, USG), ECG and other investigations according to etiology.

Treatments (Stepwise management)

NonDrug Treatment:
1. Airway management (suction, endotracheal intubation, as and when required) and oxygen therapy 4-6 L/min with oxygen mask.
2. IV fluids: Peripheral veins or central veins like jugular/femoral can be used and infuse crystalloids like NS/RL with careful observation to avoid fluid overload i.e. chest examination after each 250 ml infusion.
3. Volume of fluids: Boluses of 20 ml/kg to be infused in 30 minutes to restore blood volume quickly. Features of recovery, i.e. warm skin and improved capillary filling time and good volume pulses, blood pressure restoration, improvement in sensorium and urine output. If these do not appear, give a second bolus.

Drug Treatment

Use of vasopressors and inotropes:
Vasopressors like noradrenaline, dopamine or vasopressin can be considered. Inotropes are used to increase myocardial contractility. These are given as continuous intravenous infusions preferably with an infusion pump.

Nor-adrenaline is the preferred vasopressor in septic shock.

<table>
<thead>
<tr>
<th>Name</th>
<th>Dose</th>
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<tr>
<td>Nor-adrenaline</td>
<td>0.01-0.4 µg/kg/min</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>0.04 units/min</td>
</tr>
<tr>
<td>Dopamine</td>
<td>5-20 µg/kg/min</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>5-20 µg/kg/min</td>
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Metabolic corrections

Metabolic acidosis as a consequence of tissue ischaemia is the most important secondary complication. Correction is indicated only when marked acidosis (pH <7.2) is present. Sodium bicarbonate 1-2 mEq/kg can be used initially but subsequent doses should be based on base deficit (mEq = body weight in kg × base deficit × 0.3). Ventilatory support may be required when there are signs of respiratory failure.
Specific treatment according to etiology:-

1. **Hypovolemic shock** :- After airway, breathing and fluid resuscitation, blood or blood products may be infused in case of haemorrhage.

2. **Cardiogenic shock** :- Immediate treatment is same as mentioned above, however, invasive monitoring and advanced life support systems are required, hence patient should be referred to a tertiary level centre after initial resuscitation.

3. **Septic shock** :- where systolic blood pressure < 90 mmHg or mean arterial pressure < 70 mmHg inspite of adequate fluid resuscitation or serum lactate >2mmol/L or requires vasopressors to maintain SBP> 90 mm Hg and MAP > 70 mm Hg.

   After airway, breathing and fluid resuscitation, broad spectrum antibiotics to be given promptly. One or more antibiotics or anti-fungals may be added. Removal or drainage of a focal source of infection. Blood or blood products transfusion as and when required. General support, nutrition, blood glucose to be kept between 120-150 mg/dl, stress ulcer prophylaxis, DVT prophylaxis are also to be taken care. Therapeutic endpoints of resuscitation of septic shock:
   - Normalisation of the heart rate
   - Capillary refill of <2 seconds
   - Good volume pulses
   - Warm extremities
   - Normal range of systolic pressure and pulse pressure
   - Urine output >0.5 ml/kg/h
   - Return to baseline mental status, tone and posture
   - Normal range respiratory rate

4. **Anaphylactic shock** :-
   In anaphylaxis; Adrenaline 1:1000( 1mg/ml)is the most effective drug , 0.3-0.5 ml (mg) to be given deep IM in the thigh every 5 min as needed.

   In anaphylactic shock, Adrenaline 1:100,000 (10 microgram/ml) i.e. 1 ml of 1;1000 solution to 100 ml of saline and infuse at 30-100 ml/hr (5-15 microgram/min).

5. **Obstructive shock** :-
   Cardiac tamponade - pericardiocentesis , better refer to a tertiary care centre.
   Tension pneumothorax- ICT( Inter Costal Tube) drainage, better refer to a tertiary care centre

**Patient/Parent education:**
Immunocompromised patients should be informed about features of early sepsis. Early hospital care gives better outcome.

**References:**

****
FLUID AND ELECTROLYTE IMBALANCE AND REPLACEMENT (IN ADULTS)

Introduction
Disturbances in fluid and electrolyte balance occur in a wide spectrum of diseases, are not confined to any particular field of medicine and are common following burns, trauma and major surgery.

Electrolyte abnormalities can cause cardiac arrhythmias or cardiopulmonary arrest. Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium. In some cases therapy for life-threatening electrolyte disorders should start before laboratory results become available. The electrolyte values for definitions have been chosen as a guide to clinical decision-making. The precise values that trigger treatment decisions depend on the patient’s clinical condition and rate of changes of electrolyte values.

HYponatremia

Definition: Hyponatremia is defined when plasma [Na] <135mEq/L.

Types-
1. Hypovolemic hyponatremia- diarrhoea, vomiting, heat stroke where there is sodium loss as well as excess water loss.
2. Euvolemic- SIADH, hypothyroidism & adrenal insufficiency.
3. Hypervolemic hyponatremia- cirrhosis of liver, congestive cardiac failure & nephrotic syndrome where excessive water retention causes dilutional hyponatremia.

Signs & Symptoms: Lethargy, weakness, headache, altered sensorium, convulsions, coma. It may be acute (within 48 hrs) or chronic (more than 48 hrs).

Treatment: Treatment depends on severity of symptoms. Treatment is required when plasma [Na] <125mEq/L. If symptoms are severe- 100 ml of 3% saline is infused over a period of half-an hour to one hour followed by 0.5 ml/kg/hr infusion. If symptoms are not severe then one can start infusion at the rate of 0.5 ml/kg/hr. But the total increase in sodium should not exceed 12 meq/ 24 hrs to avoid osmotic demyelination. Hence repeated serum sodium estimation is to be done. For patients with SIADH fluid restriction and treatment of underlined cause are important and sodium correction as mentioned above. For hypovolemic hyponatremia, normal saline infusion to correct volume loss as well as hypertonic saline (3% saline) to be infused if the patient is having severe symptom. Treatment of euovolemic hyponatremia is fluid restriction and 3% saline infusion accordingly. For hypervolemic hyponatremia fluid restriction, diuretics (Furesomide/ Torsemide) and tolvaptan 15-30 mg OD may be given. The correction of hyponatremia is up to plasma [Na] 130mEq/L.

If the patient is severely symptomatic, the patient may be referred to the tertiary care centre after initial infusion of 100-300 ml 3% saline.

Patient Education:
Patient must be educated to take adequate fluid & extra salt (10gm/day) and for hypovolemic hyponatremia judicious fluid intake is required.
**HYPERNATRAEMIA**

**Definition:** serum sodium >145 mEq/L

**Causes:**
1. Excessive administration of sodium (accidental salt administration in ORS), inadequate water intake or excessive water losses.
2. Symptoms are non-specific or relate to CNS such as altered sensorium weakness, irritability, focal neurologic deficits and coma or convulsions. The severity of symptoms is determined by the speed and magnitude of the change in serum sodium concentration.

**Treatment**
- Identify and treat the underlying causes.
- Replace water deficit as assessed by degree of dehydration over a period of 48 hours with a solution containing 40 mEq/L of sodium. The quantity of water needed to correct hypernatraemia can be calculated by using the following equation:

\[
\text{Estimated water deficit} = \text{Normal Total Body Water} - \text{Estimated Body Water}.
\]

\[
\text{Normal total Body Water} = 0.6 \times \text{Body Weight}.
\]

\[
\text{Estimated Body Water} = \frac{\text{Normal Plasma Sodium} \times 0.6 \times \text{Body weight}}{\text{Current Plasma Sodium}}
\]

\[
= \frac{140 \times (0.6 \times \text{Body Weight})}{\text{Current Plasma Sodium}}
\]

Rapid correction may lead to cerebral edema. (Caution: Sodium-free solutions are never used except when hypernatraemia is acute, i.e. onset within few hours.) Monitor serum sodium closely to ensure a gradual correction (and prevent rapid fall) in serum sodium.

- Serum sodium >180 mEq/L may require urgent dialysis.

**HYPOKALAEMIA**

It is defined as serum potassium <3.5 mEq/L. Clinical symptoms include muscle weakness, hypotonia, respiratory difficulty, paralytic ileus, leg cramps. ECG changes include ST depression, T-wave flattening/inversion, U waves and arrhythmias. Pulse less electrical activity or asystole may develop.

**Treatment**
- Identify and treat the underlying causes.
- Correct the deficit with potassium supplements. Potassium chloride may be given orally (15 ml = 20 mEq). Intravenous correction with KCl (0.5-1 mEq/kg over 2 hours) is required when patient is unable to take orally, serum potassium is <2.5 mEq/L, has respiratory paralysis, or in the presence of arrhythmia.
- Correct the potassium deficit over a period of 24 hours.
- Potassium-rich foods such as banana or fruit juice may be advised on long-term basis.
HYPERKALEMIA

It is defined as serum potassium >5.5 mEq/L. Symptoms include paraesthesias, weakness, ascending paralysis, respiratory failure. ECG changes are characteristic including tall-peaked T waves, prolonged PR interval, widened QRS complex and arrhythmias in order. It is life-threatening and can cause sudden death, requires immediate therapy.

Treatment

Exclude pseudohyperkalaemia due to lab error, like haemolysis.

1. Mild hyperkalaemia: (Serum K+ 5.5-6 mEq/L.) Stop the potassium intake and offending drugs such as potassium sparing diuretics, ACEI, NSAIDs, recheck potassium levels. Consider correction of acidosis and intravascular volume. Diuretics – Inj. Frusemide 40-80 mg IV and K-bind sachets PO.

2. Moderate hyperkalaemia: (Serum K+ 6-7 mEq/L) Administer a glucose insulin infusion (100 ml of 25% dextrose with 10 U regular insulin, over 1 hour; close monitoring of glucose every 30-60 minutes) and/or a sodium bicarbonate infusion (2 mEq/kg over 5-10 min). Can be repeated 4-6 hourly. Do not give sodium bicarbonate simultaneously with calcium gluconate infusion.

3. Severe hyperkalaemia: (Serum K+ >7 mEq/L or ECG changes, tall T waves.) Urgently administer intravenous 10% calcium gluconate 0.5 ml/kg over 5-10 minutes. This immediately reverses the cardiac effects of hyperkalaemia. This should be followed up with the measures as for moderate hyperkalaemia. Salbutamol nebulization (2.5-5.0 mg) given over 15-20 minutes also acts rapidly to lower serum K+. Haemodialysis has to be done in case the hyperkalaemia refractory to therapy as in renal failure. Consider hydrocortisone 1-2 mg/kg IV to consider if suspicion of adrenal insufficiency.

Once the potassium level is restored to normal, discontinue potassium-lowering therapies and monitor serum potassium level daily. Further work-up should be initiated to determine the cause and to prevent future episodes.

References


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JAUNDICE

Jaundice is defined as yellow discoloration of skin, sclera and tissues caused by increased levels of circulating bilirubin > 2.5 mg/dl.

Prehepatic Jaundice / Hemolytic Jaundice :
Due to haemolysis there is excessive production of unconjugated bilirubin. There is acholuric jaundice, as the urine does not become deep yellow although the unconjugated bilirubin level is high.

Hepato cellular Jaundice:
Occurs due to damage of hepatocytes. Generally the conjugated and unconjugated bilirubin ratio is 1:1 and there is elevation of the liver enzymes.

Post Hepatic Jaundice / Obstructive Jaundice :
Due to cholestasis (both intra and extra hepatic) there is impaired excretion, resulting in conjugated hyperbilirubinaemia (>50% conjugated). About 3/4th of the serum bilirubin is in conjugated form. There is absence of urobilinogen in urine.

Common causes of jaundice in clinical practice include acute viral hepatitis, alcoholic hepatitis, Bile duct obstruction of various etiologies (CBD stone, Carcinoma of GB, Pancreas and biliary system.

Approach to diagnosis of jaundice
Appropriate clinical history, examination and investigations includes Complete blood counts(CBC), liver function tests (LFTs), viral markers, ultrasound examination of liver and biliary tract and if indicated CT scan of abdomen and MRCP.

Acute Viral Hepatitis
Acute viral hepatitis is generally caused by Hepatitis virus A, B and E.

Clinical Features:
- Prodromal symptoms like nausea vomiting, anorexia, fever, myalgia.
- Icterus
- Tender Hepatomegaly

Diagnosis
- LFT (Liver Function tests): Serum bilirubin, SGOT, SGPT, Serum Alkaline Phosphatase. There is equal elevation of both forms of bilirubin and SGOT/SGPT levels are very high (> 5 folds).
- Urine: Bile salt, Bile pigments
- Viral markers :
  - Positive IgM anti HAV : Indicates acute Hepatitis A.
  - Positive IgM anti HEV: Indicates acute Hepatitis E.
  - Positive IgM Anti-HBc: Indicates Acute Hepatitis B.
Detection of HBsAg is not enough for diagnosis of Acute Hepatitis B. The person may merely be a Hepatitis B carrier.

- USG Abdomen & Pelvis:
  This is not necessary for routine diagnosis of Acute Viral hepatitis. However it may be done sometimes during diagnostic confusion.

**Treatment:**

- Adequate fluid and calorie intake. The patient should always be advised to take normal diet without restriction of fat and salt.
- Hospitalization is required if there is severe prodromal symptoms causing dehydration, presence of early signs of hepatic encephalopathy e.g. altered sensorium, disturbed sleep pattern, flapping tremors and decreased liver span.
- If the patient has severe nausea or vomiting anti-emetic drugs: Domperidone 10mg TDS may be prescribed.
- There is no role of Antiviral therapy in routine case of acute hepatitis B.

**Special Tips**

- A short history of fever followed by jaundice (fever subsides when jaundice appears) favors the diagnosis of Acute Viral hepatitis.
- If the patient continues to have fever after the onset of jaundice, then one should rule out other condition like complicated malaria, enteric fever.
- Severe pain abdomen with chill and rigor favours Acute cholangitis resulting from bile duct obstruction.

**Patient Education:**

- Explain the relatives to report and hospitalise the patient if there is alteration in the behaviour or sensorium of the patient.
- There is no need to isolate the patient.
- Patient should be warned to abstain from alcohol and to refrain from consuming indigenous drugs.
- Family members of the patient with acute viral hepatitis B should use precautions to prevent transmission and be vaccinated against hepatitis B.

**Referral Criteria**

Patient should be referred to a tertiary care centre if there is development of ascitis, edema, altered sensorium and bleeding manifestations, indicative of severe disease.

**Reference:**

LYMPHATIC FILARIAISIS

Introduction:

Lymphatic filariasis is caused commonly by nematode worms called Wuchereria bancrofti and Brugia malayi. The reside in the human lymphatic system comprising of nodes and vessels. The adult worm lives in the afferent lymphatic or the sinuses of the lymphnode. They remain viable for over two decades. Humans are the only definitive host.

Clinical profile:

The manifestations can be widespread and variable. The vast majority of infected people are asymptomatic microfilaremic. The lymphatic vessels are affected in microfilaremic patients in the absence of symptoms. The notable manifestations are:

1) Acute adenolymphagitis (ADL): Recurrent acute attacks of lymphadenitis and lymphangitis with or without fever. The lymphnodes commonly affected are epitrochlear, axillary, inguinal and femoral. The lymphangitis is retrograde originating from the affected lymphnode. There may be associated transient local oedema. It is mostly self limiting.

2) Chronic lymphatic disease: Lymphodema of the limbs progressing to elephantiasis.

3) Genital affection: Invariably due to W. bancrofti infection. Funiculitis, epididymitis, hydrocoele, chylocoele, swelling of scrotum and penis are the common presentation.

4) Uncommon: Microscopic haematuria, chyluria, haematochyluria and tropical pulmonary eosinophilia.

Diagnosis:

1) Demonstration of microfilaria in finger prick blood to be collected at night or depending on the periodicity of the microfilariae

2) Detection of circulating filarial antigen in the blood or body fluid for W.bancrofti infection. There are two methods: a) Rapid diagnostic card test b) Assessment by ELISA(Enzyme linked immunosorbent assay). They are 93 to 100% sensitive and 100% specific.

3) PCR based analysis for DNA. Research based study.

4) High frequency ultrasound in conjunction with Doppler technique for detection of motile adult worm within dilated lymphatics in scrotum, lymph node and breast. It has been termed the ‘filaria dance sign’.

Treatment:

1) Active filariasis defined as patients who have microfilaremia, or positive circulating filarial antigen or adult worm detected by ultrasound should receive treatment to eliminate microfilariae. Drugs have limited effect on adult worms.

   a) DEC(Diethycarbamazine) 6mg/kg /day for 12 days

   Or

   b) Albendazole 400mg plus DEC 6mg/kg single dose have been found to have sustained microfilaricidal effect.

2) Acute ADL(Adenolymphangitis):

   a) Analgesics and antipyretics

   b) Antibiotics for secondary bacterial infection

   c) DEC 6mg/kg/day for 12 days
3) Lymphoedema and elephantiasis:
   a) Basic skin hygiene
   b) Prevention of secondary bacterial infection
   c) Antibiotics in the presence of fever and lymphangitis
   d) Physiotherapy of the affected limb
4) Hydrocoele: Surgery

**Preventive measures:**
1) Avoidance of mosquito bite. Use of bed net
2) Anti mosquito measure
3) Mass Drug Administration (MDA): Single dose of Albendazole 400mg and DEC 6mg/kg at community level

**Reference:**
1) Harrison’s Textbook of Internal Medicine, 19th Edition Vol 2, 2015
2) WHO- Lymphatic filariasis, 2017

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**MALARIA**

Malaria is a parasitic infection due to the protozoa of genus Plasmodium transmitted by the female Anopheles mosquito. There are five plasmodia species: P. falciparum, P. vivax, P. malariae, P. ovale and P knowelsi.

Malaria can be classified as:
1) Asymptomatic
2) Uncomplicated
3) Severe

**Salient Clinical features:**

Malaria is an acute illness characterised by paroxysms of fever, chills, sweats, fatigue, anaemia and splenomegaly. In endemic areas with continuous transmission classical symptoms may not manifest.

In a patient with P falciparum asexual parasitemia presence of one or more of these clinical or laboratory features defines the patient as suffering from severe malaria.
1) Impaired consciousness
2) Repeated convulsions,
3) Circulatory shock
4) Respiratory distress
5) Pulmonary odema
6) Bleeding
7) Jaundice  
8) Oliguria/anuria  
9) Anaemia Hb<5gm/dl  
10) Hypoglycaemia Blood sugar < 40mg/dl  
11) Hyperpyrexia (>42°C or 106F),  
12) Hyperparasitemia  

It is to be kept in mind that P vivax malaria which was thought to give rise to uncomplicated malaria has been reported to present with severe manifestations.  

**Diagnosis:**  
Parasite can be detected by:  
1) Demonstration in the peripheral thick and thin blood smear slides. Thick smear for easy detection of parasite and thin smear for identification of species. Note that blood films may be negative even in a severe attack because of sequestration of parasites in the deep capillaries. The limit of detection by thick smear microscopy is 50 parasites/microL.  
2) Rapid diagnostic kits (RDT) monovalent (for P. falciparum) or bivalent (both P. vivax and P. falciparum) can be used for rapid detection. It detects parasite antigens such as HRP2 (Histidine rich protein) and Lactate dehydrogenase (LDH). There can be false negative or false positive.  
3) Detection of parasite DNA based on PCR  

**Treatment:**  
All fever cases suspected to be malaria, after ruling out other common causes, should be investigated by microscopy or RDT. In all confirmed cases, treatment to be started as early as possible.  

1) Patients of uncomplicated malaria can be managed at primary level but patients with severe malaria with complications should be admitted and managed in a hospital where facilities for detailed investigations and blood transfusion exist.  
2) P. vivax cases should be treated with chloroquine, 25mg/kg in divided doses for three days and Primaquine 0.25mg/kg/d for 14 days. Chloroquine: 25 mg/kg body weight divided over 3 days, i.e. 10 mg/kg on day 1, 10 mg/kg on day 2 and 5 mg/kg on day 3.  
Primaquine is used to prevent relapse but is contraindicated in pregnant women, infants and individuals with G6PD deficiency. Note: Patients should be instructed to report back in case of haematuria or high-coloured urine/cyanosis or blue coloration of lips and Primaquine should be stopped in such cases. Care should be taken in patients with anaemia.  
3) Uncomplicated P. falciparum malaria:  
a) Artemisinin based combination therapy (ACT). Cases should be treated with area-specific artemisinin-based combination therapy (ACT) for 3 days. ACT-SP: Artesunate 4 mg/kg body weight daily for 3 days plus Sulfadoxine (25 mg/kg body weight) – Pyrimethamine (1.25 mg/kg body weight) on first day or ACT-AL (Artemether 20 mg + Lumefantrine 120 mg).
This is to be accompanied by single dose primaquine on day 2; 0.75mg/kg single dose for its gametocidal effect.

b) Pregnant women with uncomplicated P. falciparum should be treated as follows:

- **1st trimester:** Quinine
- **2nd and 3rd trimesters:** ACT

Note: Primaquine is contraindicated in pregnant women.

1st trimester: Quinine salt 10 mg/kg 3 times daily for 7 days. (Caution: Quinine may induce hypoglycaemia; pregnant women should not start taking quinine on an empty stomach and should eat regularly, while on quinine treatment.)

2nd and 3rd trimesters: Area-specific ACT as per dosage given above.

3) All cases of mixed infection of *P. vivax* and *P. falciparum* needs to be treated as *P. falciparum* infection.

4) Treatment of *P. ovale* and *P. malariae*:

   In India, these species are very rarely found in few places. Treat *P. ovale* as *P. vivax* and *P. malariae* as *P. falciparum*.

5) Treatment of severe malaria:

   Severe malaria is to be treated as a medical emergency.
   a) Inj. Artesunate 2.4 mg/kg IV given on admission (time = 0 hour); then at 12 hours and 24 hours and then once a day, for seven days or till patient can take orally, ACT can be started. It is the drug of first choice. (Caution: Care should be taken to dilute artesunate powder in 5% sodium bicarbonate provided in the pack only.) If not available,
   b) Inj. Artemether 3.2 mg/kg IM given on admission and then 1.6 mg/kg per day for three days

   If artesunate derivatives are not available

   Inj. Quinine: 20 mg/kg on admission (IV infusion by diluting quinine in 500 ml of 10% Dextrose solution in four hours) followed by maintenance dose of 10 mg/kg 8 hourly. The infusion rate should not exceed 5 mg salt/kg per hour. Do not give loading dose of Quinine, i.e. 20 mg/kg on admission, if the patient has already received quinine or if the clinician feels inappropriate. NEVER GIVE BOLUS INJECTION OF QUININE. If parenteral quinine therapy needs to be continued beyond 48 hours, reduce dose to 7 mg/kg 8 hourly.

   Note: The parenteral treatment in severe and complicated malaria cases should be given for minimum of 48 hours once started (irrespective of the patient’s ability to tolerate oral medication earlier than 48 hours) followed by a full course of oral area-specific ACT for 3 days.

   Those patients who received parenteral Quinine therapy and can take orally should receive: Oral Quinine 10 mg/kg three times a day for 7 days (including the days, when parenteral Quinine was administered) plus Doxycycline 3 mg/kg once a day or Clindamycin, 10 mg/kg 12-hourly for 7 days (Doxycycline is contraindicated in pregnant women and children under 8 years of age; instead, give clindamycin 10 mg/kg 12 hourly for 7 days). Avoid use of mefloquine in cerebral malaria due to neuropsychiatric complications associated with it.

   Do not give corticosteroids, intravenous mannitol, heparin, anticoagulants, adrenaline or overhydrate.
Monitoring

Monitor core temperature (preferably rectal), respiratory rate and depth, pulse, blood pressure and level of consciousness every 4 hours; record urine output, and look for the appearance of brown or black urine (haemoglobinuria) or oliguria; monitor therapeutic response, both clinical and parasitological, by regular observation and blood films; carry out regular laboratory evaluation of haematocrit or haemoglobin, glucose, urea or creatinine and electrolytes; avoid drugs that increase the risk for gastrointestinal bleeding (aspirin, corticosteroids).

Supportive treatment

Treat fever, hypoglycaemia, electrolyte imbalance, hypotension, renal failure, anaemia, convulsions appropriately.

Chemoprophylaxis

Chemoprophylaxis should be administered only in selective groups in high P. falciparum endemic areas.

Short-term chemoprophylaxis (Up to 6 weeks)

1) Tab. Doxycycline 100 mg once daily for adults. The drug should be started 2 days before travel and continued for 4 weeks after leaving the malarious area. Note: It is not recommended for pregnant women and children less than 8 years.

2) Atovaquone–Proguanil daily one tablet.

Chemoprophylaxis for longer stay (More than 6 weeks):

1) Tab. Mefloquine 250 mg weekly for adults and should be administered 2 weeks before, during and 4 weeks after exposure.

Referral Criteria: Patients with manifestations of severe disease as enumerated earlier needs to be referred to a tertiary care hospital

Patient education:

- Avoid mosquito bite. Insecticide impregnated bed net.
- Mosquito control: Insecticide spray

Chemoprophylaxis

References


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TYPHOID OR ENTERIC FEVER

The causative microorganism are *Salmonella typhi* and *paratyphi*. *Salmonella typhi* causes a variety of illnesses including asymptomatic carriage, gastroenteritis, and enteric fever.

**Clinical Features:**
- Onset of fever is typically gradual, continuous (temperature up to 40°C) with constitutional symptoms like headache, malaise, anorexia, lethargy, constipation or diarrhoea.
- Associated abdominal pain and tenderness including a coated tongue.
- Patients with complications (usually in 3rd week) present with G.I bleeding, intestinal perforation, neuropsychiatric symptoms (muttering delirium or coma vigil - staring look with unarousable coma).

**Diagnosis:**
- Examination reveals a toxic look with relative bradycardia and mild soft splenomegaly.
- Complete blood counts in most cases are normal. Leucopenia or neutropenia seen in 15-25% cases. Non specific lab tests include a moderate elevation in liver function tests and muscle enzymes.
- Definite diagnosis requires the isolation of causative organism in blood, bone- marrow, rose spots, stool or intestinal secretions.
- Widal test (Serological diagnosis) for “febrile agglutinins” is not very specific or sensitive test; hence diagnosis is made mostly on clinical basis as blood culture is positive in only 40-80% cases.
- Newer serologic assays using ELISA and dipstick methods have still not been approved for routine diagnostic use in view of the variable sensitivity and specificity.

**Complications**
- GI Bleeding, intestinal perforation, hepatitis, peritonitis, meningitis, pneumonitis, and myocarditis can occur, usually after the first week.

**Treatment**

**Non-pharmacological:**
- Adequate nutrition and hydration should be maintained ensuring adequate intake either orally or with intravenous fluids (in severely ill).
- Cold sponging for increasing temperature.
- Hospitalization is recommended, if patient is very sick, not accepting orally with inadequate urine output, patient has altered sensorium/drowsiness or is having very high pyrexia particularly in the third and fourth week of illness when the risk of complications increases or if the complications have already ensued.

**Pharmacological:**
- Treatment of OPD patients:
  - Tab Cefixime 400mg BD 14 days.
• Tab Azithromycin 1 gm OD PO 7-10 days.
• Tab Ofloxacin 400mg BD PO 10-14 days.

Treatment of Indoor patients:
• Inj. Ceftriaxone 2 gm OD IV 10-14 days or,
• Inj. Cefotaxime 2 gm TID IV 10-14 days.
And shift to Tab Cefixime once fever resolves.
• Second line
  ➢ Tab Ofloxacin 400mg BD PO 10-14 days.
  ➢ Cap Chloramphenicol 25mg/kg TID PO 14 days.
  ➢ Tab TMP + SMX (160/800) BD PO 14 days.

Patient Education:
• Patient should take bed rest. Small frequent feeds should continue. Give plenty of oral fluids and compensate
  for increased fluid loss from the body due to high-grade fever.
• Fever usually lasts 5-7 days even after starting effective treatment in most cases. Frequent change of therapy
  should, therefore, be avoided.
• The treatment should be administered for at least 10 days or for 5 days after the fever subsides due to
  increased the risk of relapse and emergence of resistance.
• The caregivers of the patients should be informed about the complications.

Referral Criteria:
Patients not responding to treatment after 5 days or having complications like perforation, bleeding, shock,
encephalopathy should be referred to a nearest hospital or tertiary care centre.

Vaccination
• Type 21a (Live attenuated vaccine) to be taken as 1 cap P.O on days 1, 3, 5 and 7. Minimum age of vaccination
  6 years and booster doses needed every 5 years.
• Vi-CPS Conjugate vaccine (Killed vaccine): Single dose I.M injection. Minimum age of vaccination 2 years
  and booster required every 2 years.

References:
  Chap 190; 1049-1055.
DENGUE

Introduction:
Dengue is a vector borne disease caused by Dengue virus (Sero types = 1, 2, 3 & 4) and transmitted by Aedes aegypti mosquito (a day biter). It is the fastest growing vector borne disease and has increased by 30 times in last 5 decades.

Diagnostic Criteria:
Dengue Fever = An acute febrile illness of 2-7 days duration +
Two or more of the following manifestations:
- Headache, retro-orbital pain, myalgia, Arthralgia, rash, haemorrhagic manifestations.

Dengue Hemorrhagic Fever= (DHF):
(a) Clinical criteria of dengue fever +
(b) Hemorrhagic tendencies evidenced by one or more of the following
   1) Positive tourniquet test
   2) Petechiae, ecchymosis or purpura
   3) Bleeding from mucosa, gastrointestinal tract, injection sites etc.
   +
(c) Thrombocytopenia (<1 lakh cells per Cumm)
   +
(d) Evidence of plasma leakage due to increased vascular permeability manifested by one or more of the following:
   1) A rise in haematocrit by >20%
   2) > 20% drop in haematocrit following volume replacement treatment compared to baseline.
   3) Signs of plasma leakage (pleural effusion, ascites, hypoproteinaemia)

Dengue shock syndrome (DSS):
Criteria for DHF +
Evidence of circulatory failure
Manifested by rapid and week pulse and pulse pressure (<“20mm Hg) or hypotension for age, cold clammy skin, restlessness, oliguria.

Probable Dengue fever:
A case compatible with clinical description of dengue fever
(A positive test by RDT will be consider as probable due to poor sensitivity and specificity of currently available RDTs)

Confirmed Dengue fever:
A case compatible with clinical description of dengue fever with atleast one of the following.
- Isolation of dengue virus by culture from serum, plasma, leukocytes.
- Demonstration of dengue virus antigen (NS1Ag) in single serum sample if fever duration is <5 days.
- Demonstration of IgM antibody by ELISA (fever more than 5 days)
- Detection of virus by PCR
- IgG seroconversion in paired sera (after 2 weeks 4 fold increase in IgG titre)

**Natural Course of Dengue fever:**
The clinical course passed in 3 phases.

a) Febrile phase last for 2-7 days

b) Critical phase usually comes after 3-4 days of onset of fever. Usually fever subsides at this stage. This is also associated with rising hematocrit and falling platelet count. This phase lasts for 36-48 hours. Secondary attack of dengue leads to more complication than primary.

c) Convalescent phase (Recovery phase)

This lasts for 2-3 days. During this phase the extracellular fluid which was lost due to capillary leakage returns to the circulatory system. Patient may develop pulmonary edema if fluid replacement is not optimized carefully.

**Investigation and Management Plan:**
Guidelines to be followed in the PHC for management of Dengue cases:

**Criteria of Discharge:**
No fever for at least 24 hours (48 hours if he had shock), normal blood pressure, adequate urine output, no respiratory distress, persistent platelet count >50,000/Cumm.

**Specific treatment:**
Not available

**Referral Criteria:** Patients with persistent fever, bleeding tendencies, shock and low platelet count.

**Prevention:**
Aedes breeds in clean water collection in and around houses like unused water tank, cooler, air conditioner, flower pot, broken pots, disposable glass-cup-plates, tyres etc. Increased virus transmission occurs during monsoon and post-monsoon season. These sources should be removed at that time. Other measures like use of mosquito nets, mosquito repellent creams, use of dress to cover body etc. can be useful. One tetravalent vaccine is available in some countries. But efficacy is not unequivocal so not yet allowed in India.

**References:**
CHIKUNGUNYA

Introduction:

The agent is chikungunya virus which has 3 distinct lineages – West Africa Lineage, East-Central South African (ECSA) lineages and Asian lineage. Aedes Aegypti and Albopictus mosquito are the vectors.

Diagnostic criteria:

After an incubation period of 3-7 days, chikungunya has an abrupt onset with high grade fever, single or multiple joint pain, skin rashes, headache and myalgia. Uncommon manifestations include photophobia, retro-orbital pain, hypotension, vomiting, diarrhea, meningeal syndrome and acute encephalopathy. However, these later manifestations are common in children.

Clinical presentation of chikungunya

It has 3 phases which are as follows-

(a) Acute phase: Less than 3 weeks
(b) Sub-acute phase : 3 weeks to 3 months
(c) Chronic phase : More than 3 months

10-15% of chikungunya progress to sub-acute or chronic phase.

Based on clinical presentation chikungunya is classified into 3 categories-

<table>
<thead>
<tr>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low grade fever</td>
<td>Low to high grade persistent fever</td>
<td>Persistent high grade fever</td>
</tr>
<tr>
<td>Mild Arthralgia</td>
<td>Moderate joint pain</td>
<td>Severe joint pain</td>
</tr>
<tr>
<td>Mild focal Myalgia</td>
<td>Generalized Myalgia</td>
<td>Persistent vomiting/Diarrhoea</td>
</tr>
<tr>
<td>General Weakness</td>
<td>Hypotension</td>
<td>Altered sensorium</td>
</tr>
<tr>
<td>Skin rash/itching</td>
<td>Mild Bleeding usually due to use of NSAIDS</td>
<td>Bleeding (GI tract bleeding</td>
</tr>
<tr>
<td></td>
<td>Retro-orbital pain</td>
<td>Shock due to persistent vomiting and diarrhea</td>
</tr>
</tbody>
</table>

High risk group include co-morbid condition like Hypertension, Diabetes, CAD/CVA, Geriatric age, pregnancy, COPD, Hypothyroid state and co-infections like Tuberculosis, Enteric Fever, Pneumonia, HIV, Malaria and Dengue.

Diagnosis:

Clinical criteria:
Acute onset of fever and severe arthralgia/arthritis ± skin rash and residing or visited an epidemic area within 15 days prior to onset of symptoms.

**Laboratory criteria:**

At least one of the following tests done in acute phase of illness.

(A). Direct evidence - Virus isolation or presence of viral RNA by RT-PCR

(B). Indirect evidence-

- Presence of virus specific IgM antibodies in single serum sample collected in acute or convalescent stage.
- Four-fold increase in IgG values in samples collected at least 3 weeks apart.

**Differential diagnosis:**

In acute phase, it has to be differentiated from Dengue fever, Malaria, Leptospirosis, and Enteric fever.

In chronic phase, it has to be differentiated from various types of arthritis.

In particular, it has to be differentiated from Dengue fever because the later may have fatal outcome and NSAIDS should not be used to relieve pain. The following differences are useful to reach at a diagnosis.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Chikungunya (%)</th>
<th>Dengue Haemorrhagic Fever (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal pain</td>
<td>31.6</td>
<td>50.0</td>
</tr>
<tr>
<td>Conjunctival injection</td>
<td>55.6</td>
<td>32.8</td>
</tr>
<tr>
<td>Maculo-papular rash</td>
<td>59.6</td>
<td>12.1</td>
</tr>
<tr>
<td>Myalgia/Arthralgia</td>
<td>40.0</td>
<td>12</td>
</tr>
<tr>
<td>Coma</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Fever onset</td>
<td>Acute</td>
<td>Gradual</td>
</tr>
<tr>
<td>Duration</td>
<td>2-4 days</td>
<td>5-7 days</td>
</tr>
<tr>
<td>Hypovolumic shock</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Leucopenia</td>
<td>Common</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Infrequent</td>
<td>Common</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>Normal</td>
<td>High</td>
</tr>
</tbody>
</table>
Treatment:

No specific antiviral treatment is available. So patient has to be treated symptomatically – particular’s to relieve pain. If Dengue diagnosis is excluded, patient can be given NSAIDS. Otherwise paracetamol and tramadol are to be used for pain relieve.

In chronic cases presenting with arthritis, hydroxychloroquine in the dose of 6mg/kg/day may be useful.

Referral Criteria:

In acute and severe cases of chikungunya and in chronic cases, to rule out other rheumatological disorders.

Prevention:

Aedes breeds in clean water collection in and around houses like unused water tank, cooler, air conditioner, flower pot, broken pots, disposable glass-cup-plates and tyres. Increased virus transmission occurs during monsoon and post-monsoon season. These sources should be removed at that time. Other measures like use of mosquito nets, mosquito repellent creams, use of dress to cover body etc. can be useful.

References:


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SNAKE BITE

Introduction:
Snake bite envenomation is a common medical emergency. In Odisha 45 species of snakes are found of which 11 are poisonous, out of which four, namely common cobra (Naja naja), Russell’s viper (Daboia russelii), saw-scaled viper (Echis carinatus) and common krait (Bungarus caeruleus) are highly venomous and believed to be responsible for most of the poisonous bites.

Diagnostic Features:

Viperidae Snake bite (Vasculotoxic)-Russell’s viper, saw-scaled viper
- Local effects: Within a few minutes of bite local swelling, burning pain at the site along with painful lymphangitis and regional lymphadenitis develops.
- Generalised haemorrhagic manifestations: haematuria, epistaxis, bleeding gums, haematemesis, malena, bleeding from the bite site, ecchymosis, petechial haemorrhages and the dreaded complication like intracerebral haemorrhage.
- Acute renal failure is the most important complication and cause of death.
- Hypotension develops in some cases

Elapidae Snake bite (Neurotoxic)- common Krait, Cobras
- Predominantly neurotoxic effects produced which usually develop early but may be delayed for several hours
- Ptosis is the commonest neurological deficit. Ophalmoplegia, bulbar palsy (dribbling of saliva, difficulty in speech), quadriplegia, respiratory paralysis are other manifestations. Myalgia and muscle tenderness are very common
- Respiratory paralysis is the common cause of death. Hypotension develops sometimes.

Hydrophidae (myotoxic and neurotoxic) – sea snakes
- Muscle pain, tenderness, weakness, rhabdomyolysis, myoglobinuria, renal failure, respiratory paralysis

Laboratory tests:

20 minutes Whole Blood Clotting test (20 WBCT):
Place 2ml of venous blood in a new, clean, dry glass test tube, then leave it undisturbed in erect position for 20 min. Tip the test tube once to see if the blood has clotted. If the blood is still liquid (uncotted), the patient has hypofibrinogenemia (incoagulable blood) due to venom-induced consumption coagulopathy. This is diagnostic of Viper bite (vasculotoxic) and requires ASV treatment.

Other useful tests (if facilities available)
- Hb/platelet count/peripheral smear/prothrombin time (PT)/ activated partial thromboplastin time (APTT)/fibrin degradation products (FDP)/D-Dimer, Urine exam for proteinuria/RBC/hemoglobinuria/ Myoglobinuria, Biochemistry for serum creatinine/Urea/Potassium
Treatment
Non-drug treatment:

Reassure the victim that death is not imminent and medical care is available. Transport the patient rapidly to the nearest equipped medical facility after first aid.

1) First aid – Immobilization of the whole patient, especially the bitten limb by a splint, keeping the bitten part below heart level is preferred to delay venom spread. A pressure-pad or pressure-bandage (crepe bandage) with a pressure enough that one can easily introduce a finger between skin and bandage is helpful.

But tight tourniquet, suction, washing the area, local incision, vaccum pumps, chemical compounds, ice packs or electric shock should not be used. A tight tourniquet if already applied, should only be removed after full dose of snake antivenom is administered.

2) All patients should be kept under observation for at least 24 hours.

3) Ensure airway, breathing and circulation.

Drug Treatment:

Anti-snake venom (ASV) Therapy

ASV should not be given to every case of snake bite, as it is associated with risks of antivenom reactions. It should only be administered when features of envenomation are present:

Systemic Envenoming

Evidence of coagulopathy: Primarily detected by 20WBCT or visible spontaneous systemic bleeding.

Evidence of neurotoxicity: ptosis, external ophthalmoplegia, muscle paralysis, inability to lift the head etc.

Cardiovascular abnormalities: hypotension, shock, cardiac arrhythmia, abnormal ECG.

Persistent and severe vomiting or abdominal pain.

Severe Current Local envenoming

- Severe current, local swelling involving more than half of the bitten limb (in the absence of a tourniquet).

Dose- Initially, 100 ml (10 vials) ASV is diluted in 5 ml of normal saline per kg body weight and administered by intravenous infusion over 60 minutes. In patients who continue to bleed briskly or in those with deteriorating neurotoxic or cardiovascular signs, the dose of ASV should be repeated in 1-2 hours.

Children should receive the same ASV dosage as adults as snakes inject the same amount of venom into adults and children.

Adrenaline should always be in readiness before starting ASV. Close monitoring of patients for at least 1 hour after starting ASV is required for detection of early anaphylactoid reactions. If urticaria or itching or tachycardia occurs stop ASV, immediately treat with adrenaline (1:1000) 0.5mg IM, antihistaminics (chlorpheniramine 10 mg IV) and corticosteroids (hydrocortisone 100 mg IV).

No ASV test dose to be administered, as it is not predictive of reactions and may delay treatment.
20WBCT is to be repeated 6 hrs. after administration of ASV, if abnormal, repeat dose of 5-10 vials of ASV should be given, and continued 6 hourly till coagulation is restored.

*Neurotoxic snake bite:* ASV administered as above. Inj. Neostigmine 0.5 mg IV half hourly preceeded by inj. Atropine 0.6 mg IV (to block its muscarinic side effects), 5 such doses and then 4-6 hrly till full recovery of neuroparalytic symptoms. But if no relief after 3 doses it can be stopped. Assisted ventilation may be required.

Severe pain at the bit site can be treated with paracetamol or tramadol tablets.

Renal failure should be treated.

Respiratory failure – Oxygen, assisted ventilation required

Treatment of the bitten part – The painful, swollen limb should be nursed in the most comfortable position avoiding excessive elevation. Leave blisters, aspirate abscess and culture pus. Necrotic tissue needs early surgical debridement and split-skin grafting. Prompt antibiotic treatment of necrotic tissue with amoxicillin or a cephalosporin or ciprofloxacin or Piperacillin/tazobactum along with metronidazole is recommended.

Tetanus toxoid booster is recommended.

**Patient education:**

The patient is advised to return if there is any worsening of symptoms such as bleeding, pain or swelling at the site of bite, difficulty in breathing, altered sensorium, or any signs of serum sickness like fever, joint pain or swelling.

Care of the bitten part, blisters or abscesses should be taken care.

**Referral criteria:**

1. Respiratory failure requiring mechanical ventilation (frequently check for the ability to perform ‘neck lift’) even after full dose of ASV
2. Continuous bleeding even after ASV full dose
3. Renal failure requiring dialysis
4. Progressive septicaemia

**References:**

2. Guidelines for the Management of Snake Bites. World Health Organization 2016. Regional Office for South-East Asia

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ORGANOPHOSPHORUS POISONING

Introduction:
Organophosphorus (OP) pesticides poisoning can result from occupational, accidental or intentional exposure. The commonly used OP insecticides are chlorpyrifos, dichlorvos, diazinon, dimethoate, fenitrothion, methyl parathion, phenthoate, phorate, quinalphos, etc. These compounds can be absorbed through skin, conjunctiva, oral mucosa, GI tract or respiratory tract. Lipid solubility makes easy access to CNS and fat stores. They are intermittently released from fat stores to circulation or secreted to stomach and have been incriminated for the sudden deterioration in a stable patient.

Diagnostic features:

Clinical features
Onset is usually within 3 hours of exposure

Common symptoms: Dizziness, anxiety, headache, tightness of breath, restlessness, confusion, disorientation, abdominal pain, vomiting, diarrhoea, drowsiness

Important signs: Rhinorrhea, sweating, excessive lacrimation and salivation, miosis/mydriasis, bradycardia/ tachycardia, hypotension/ hypertension, coughing, bronchorrhea, bronchospasm, cyanosis, pulmonary edema, respiratory depression, respiratory failure, muscle twitching, fasciculation, flaccid paralysis, involuntary micturition or defeation, loss of consciousness, convulsions, coma.

A high index of suspicion is needed as the history of OP exposure may be denied.

Laboratory tests:
Measurement of Cholinesterase level in RBC, which is reduced to 20% normal values and serum cholinesterase level which is reduced to < 50% of the normal values confirms the diagnosis.

Treatment:

Non-drug Treatment:
1) Skin decontamination – Remove contaminated clothings. Wash skin with soap and water.
2) Gastric lavage with tap water- if presents within 1 hour of ingestion.
3) Activated charcoal- if presents within 4 hours of ingestion
4) Establish airway, throat suction and Oxygen if required. Establish IV line, monitor BP and do not rush fluids

Drug Treatment:
1) Inj. Atropine is the only life saving antidote and should be started immediately (may eliminate the need for intubation).

   Inj. Atropine IV 0.5-2 mg every 5-15 minutes till atropinisation, then atropine infusion at the rate of 10-20% of total atropinisation dose per hour, titrated further to maintain the heart rate at 100/min and the atropinised state. The signs of atropinisation are: drying of all secretions (most reliable), fever, dilated pupils, heart rate > 80 beats/min, systolic blood pressure > 80 mm of Hg.

   Atropine toxicity: - Delirium, agitation, hyperthermia, ileus, tachycardia. Discontinue the atropine infusion and observe. When these settle down the infusion is to be started at 70- 80 % of the previous rate.
Note –

1) There is no fixed dose of atropine in OP poisoning. The aim is to keep the patient atropinised till poison effect weans off.

2) Inj.Pralidoxime (PAM) in a dose of 30 mg/kg slow IV loading dose over 30 min. followed by a continuous infusion of 8-10 mg/kg/hr until clinical recovery or seven days, whichever is later.

3) Furosemide is recommended if pulmonary oedema persists, even after full atropinisation.

4) Benzodiazepenes (Diazepam/Lorazepam) administered in agitation or seizures.

5) Antibiotics (broad spectrum) are to be instituted considering the risks of infections due to multiple interventions.

Continuous monitoring is required for 72 hours or longer as organophosphates may be intermittently released from fat stores.

Intermediate syndrome: which occurs within 24-96 hours is characterised by weakness of neck muscles, proximal limb muscles and respiratory paralysis. Treatment is supportive, may require assisted ventilation.

Note- Morphine, theophylline, phenothiazines, succinylcholine are contra-indicated.

Patient Education:

The importance of skin decontamination should be taught to the attendants of the patient.

Referral Criteria:

1. Patients with serious clinical manifestations of OP poisoning (after full atropinisation).
2. Patients with respiratory failure needing assisted ventilation.

References:


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2. PAEDIATRICS
NEONATAL RESUSCITATION

Introduction - Out of total delivery only 10% babies who don’t cry after birth requires resuscitation. If done timely and following proper procedure, 9 out of 10 will be normal within one minute in labour room. A newborn care corner with radiant warmer, Bag(250,500) and mask(0,1), foot operated suction, shoulder roll, mucous extractor, cord clamp, IV Canula, NG Tube, 2 sterilised towels and other necessary items should be kept ready in a baby tray for immediate use.

Diagnostic criteria-
- Baby is not crying or breathing normally after birth.
- A sick newborn comes with apnea or gasping (RR<30/min).
- Persistent cyanosis in spite of supplemental oxygen.

Lab investigation - Nil

Non drug Treatment-
- Suction Mouth and then gently. (Pressure maximum 100mm of Hg)
- Dry the baby with sterile towel and remove the wet towel
- Assess for breathing if not breathing well
- Cut the cord
- Transfer the baby to radiant warmer
- Position the baby with shoulder roll
- Suction of mouth and nose
- Gentle stimulation by rubbing the back or flicking the sole

Then Assess breathing - If not breathing well
- Start Bag and mask ventilation at 40-60/min
- Sequence is Squeeze—two—three—squeeze—

Look for chest rise If not
- Adjust the position (slight extension of neck) with shoulder roll
- Correct seal of the mask over face (Covering chin, mouth and nose)
- Suction of mouth and nose if any secretion
- Look for closure of mouth
- Increase pressure by pressing bag little harder

Continue for 30 secs and assess breathing

If not breathing
Count heart rate at base of umbilicus by feeling the pulse for 6 secs and multiply by 10.
- If heart rate >60/min
  - Continue ventilation
  - Add oxygen

Drug Treatment
If heart rate <60/min
Chest Compression with Bag and Mask ventilation (3:1 ratio)
Sequence is one and two and three and squeeze and one (120 events in 1 min)
- Inj Adrenaline (1:10000) 0.1ml-0.3ml/kg IV or umbilical route
- Repeat 2 times if no improvement
• Inj Normal saline 10ml/kg IV over 10-20 min slowly
  No sodi bicarb or Dexamethasone to be given during resuscitation.

Patient Education
• Regular ANC to be done to detect any high risk pregnancy
• All mothers are to be motivated for institutional delivery
• Should have faith on doctors decision

Referral Criteria
• If the heart rate decreases inspite of Bag and mask ventilation
• No spontatnous breathing even after 10 mins of resuscitation
• Major cong anomalies
• Extremly premature babies

***

NEONATAL SEPSIS

Introduction
One of the commonest and preventable cause of neonatal deaths in the community. Most cases of neonatal sepsis can be saved, if diagnosed early and treated with good supportive care and antibiotics. Most cases of neonatal sepsis in the community are caused by Escherichia coli and Staphylococcus aureus. In hospitals, Klebsiella pneumoniae is also a common organism.

Diagnostic criteria
General features with sepsis in newborn are Lethargy, Refusal to suckle, Poor cry, Abdominal distension, Hypothermia, Bleeding etc. Specific features in respiratory tract infections are Cyanosis, Tachypnea, Chest retractions, Grunt, Apnea/ gasping and in meningitis fever, seizures, high pitched cry, blank look, convulsions like features may appear.

Lab Investigations
Direct evidence - Blood culture before antibiotics is the gold standard.
Indirect evidence - Sepsis Screen-
If any two of following is positive
1. TLC<5000/ cmm  2. ANC<1800/ cmm
3. I/T Ratio>0.2  4. Micro ESR>15 in 1st hr
5. CRP>10mg/Ltr

Non drug treatment-
Hand washing and using clean cloth for the newborn
Keep the baby warm by doing KMC
Breast fed the baby if sucking
Urgently attend health care facility if any signs of sepsis appears.
Drug Treatment

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Each Dose</th>
<th>Frequency (&lt;7 days age)</th>
<th>Frequency (&gt;7 days age)</th>
<th>Route</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inj. Ampicillin</td>
<td>50 mg/kg/dose</td>
<td>12 hrly</td>
<td>8 hrly</td>
<td>IV</td>
<td>7-10 days</td>
</tr>
<tr>
<td>or Inj. Cloxacillin</td>
<td>25 mg/kg/dose</td>
<td>12 hrly</td>
<td>8 hrly</td>
<td>IV</td>
<td>7-10 days</td>
</tr>
<tr>
<td>B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inj. Gentamicin</td>
<td>5 mg/kg/dose</td>
<td>24 hrly</td>
<td>24 hrly</td>
<td>IV</td>
<td>7-10 days</td>
</tr>
<tr>
<td>or Inj. Cloxacillin</td>
<td>15 mg/kg/dose</td>
<td>24 hrly</td>
<td>24 hrly</td>
<td>IV</td>
<td>7-10 days</td>
</tr>
</tbody>
</table>

Table 1: Summary of antibiotic treatment for sepsis in a young infant

<table>
<thead>
<tr>
<th>Young infant's weight</th>
<th>Amount of Gentamicin to be given intramuscularly as Injection (contains 80 mg in 2 ml vial)</th>
<th>Amount of Amoxicillin to be given per-orally as Syrup (contains 125mg / 5 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 1.5 Kg</td>
<td>To be referred to higher facility</td>
<td></td>
</tr>
<tr>
<td>Above 1.5 Kg - up to 2.0 Kg</td>
<td>0.2 ml</td>
<td>2 ml</td>
</tr>
<tr>
<td>Above 2.0 Kg - up to 3.0 Kg</td>
<td>0.3 ml</td>
<td>2.5 ml</td>
</tr>
<tr>
<td>Above 3.0 Kg - up to 4.0 Kg</td>
<td>0.4 ml</td>
<td>3 ml</td>
</tr>
<tr>
<td>Above 4.0 Kg - up to 5.0 Kg</td>
<td>0.5 ml</td>
<td>4 ml</td>
</tr>
<tr>
<td>Route of administration</td>
<td>Intramuscular</td>
<td>Oral</td>
</tr>
<tr>
<td>Dosage</td>
<td>5 mg/kg/dose *</td>
<td>25 mg/kg/dose**</td>
</tr>
<tr>
<td></td>
<td>Once a day</td>
<td>Twice a day</td>
</tr>
</tbody>
</table>
*Precaution: If the treatment is to be continued same vial can be reused for the entire course of 7 days, provided it is stored properly and its contents do not change colour or have turbidity. In case of any doubt it is better to use a new vial.

Treatment 2ND table can be followed if unable to put IV line and sepsis is less serious.

Referral Criteria to SNCU:
- No improvement within 48hrs of treatment
- Persistent central Cyanosis
- Distention abdomen
- Sclerema

****
NEONATAL HYPOTHERMIA

Introduction

Normal temperature of a newborn baby is 36.5-37.4 degree celsius. Prolonged Hypothermia in a normal healthy newborn may lead to hypoglycaemia-Hypocalcemia and death. Heat loss occur by Evaporation,conduction,convection and radiation methods So it is very essential to maintain appropriate temperature of the baby during management of sick newborn.

Diagnostic features-

- Presentation are nonspecific.
- Irritability, Lethargy , Refusal to feed ,acro cyanosis etc.
- A hypothermic baby may have underlying sepsis.

<table>
<thead>
<tr>
<th>Cold Stress</th>
<th>Abdomen is warm but feet are cold</th>
<th>36.4-36</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothermia</td>
<td>Both abdomen and feet are cold</td>
<td>&lt; 36</td>
</tr>
</tbody>
</table>

Lab Investigation-

- After initial management if the hypothermia persists
- Blood sugar, sepsis screening and blood calcium can be estimated if hypothermia persists.

Non Drug Treatment-

- If in cold stress keep baby with mother in skin to skin contact .Kangaroo Mother care to be practised in all the LBW babies .If the baby is hypothermic, keep baby in radiant warmer setting temp at 37degree celsus.

Drug Treatment

- If with above procedure the hypothermia persists then treat for sepsis as per guideline .Give humidified oxygen ,IV fluid and broad spectrum antibiotics as required.

Patient Education-

- Baby should be always kept with mother.
- Cover the head, hand and feet of the newborn.
- Don’t give bath to LBW and sick babies
- Mother should feed breast milk to keep baby warm.

Referral Criteria-

- If the baby is very sick he should be refd to SNCU after correcting the hypothermia in the facility.
NEONATAL JAUNDICE

Introduction

60% term and 80% preterm babies develop jaundice. It might signal a serious, potentially treatable condition and may cause neurological damage if bilirubin is sufficiently elevated.

Physiological jaundice

Jaundice that first appears between 24-72 hours of age

Maximum intensity is seen on 4-5th day in term and 7th day in preterm neonates

Does not exceed 15 mg/dl

Clinically undetectable after 14 days

Pathological jaundice

Clinical jaundice in first 24 hrs of life

Total serum bilirubin (TSB) increasing by > 5mg/dL/day or 0.5 mg/dL/hr

TSB >15 mg/dl

Conjugated serum bilirubin > 2 mg/dl

Clinical jaundice persisting for > 2 week in full term and > 3 weeks in preterm neonates

Diagnostic criteria

Clinically jaundice spreads from cephalo caudal direction.

Clinically level of jaundice can be assessed as

1-6mg, 2-9mg, 3-12mg, 4-15mg and 5-18-20mg/dl

Lab investigations:

Serum Bilirubin—Total and direct, Blood grouping, Rh typing, Peripheral smear, G6PD, Sepsis screen etc.

Non Drug Treatment

Physiological jaundice can be observed carefully on its progress. Timely use of phototherapy in case of pathological jaundice can completely cure the baby.

Guidelines for phototherapy and exchange transfusion as per bilirubin level in <35 weeks babies

<table>
<thead>
<tr>
<th>Weight (gm)</th>
<th>Phototherapy</th>
<th>Exchange Transfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>500-750</td>
<td>5-8</td>
<td>12</td>
</tr>
<tr>
<td>750-1000</td>
<td>6-10</td>
<td>15</td>
</tr>
<tr>
<td>1000-1250</td>
<td>8-10</td>
<td>15</td>
</tr>
<tr>
<td>1250-1500</td>
<td>10-12</td>
<td>17</td>
</tr>
<tr>
<td>1500-2500</td>
<td>15-18</td>
<td>20</td>
</tr>
</tbody>
</table>
• Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin.
• Risk factors: isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin < 3.0g/dL (if measured)
• For well infants 35-37 6/7 wk can adjust TSB levels for intervention around the medium risk line. It is an option to intervene at lower TSB levels for infants closer to 35 wks and at higher TSB levels for those closer to 37 6/7 wk.
• It is an option to provide conventional phototherapy in hospital or at home at TSB levels 2-3 mg/dL (35-50μmol/L) below those shown but home phototherapy should not be used in any infant with risk factors.

• The dashed lines for the first 24 hours indicate uncertainty due to a wide range of clinical circumstances and a range of responses to phototherapy.
• Immediate exchange transfusion is recommended if infant shows signs of acute bilirubin encephalopathy (hypertonia, arching, retrocollis, opisthotonos, fever, high pitched cry) or if TSB is ≥5 mg/dL (85μmol/L) above these lines.
• Risk factors: isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis.
• Measure serum albumin and calculate B/A ratio (See legend)
• Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin
• If infant is well and 35-37 6/7 wk (median risk) can individualize TSB levels for exchange based on actual gestational age.
Charts to be used for >35 weeks babies as per bilirubin level for phototherapy or exchange transfusion.

**Patient Education**

Though neonatal jaundice is commonly seen in newborns, Parents should consult paediatricians if yellowness crosses umbilicus.

- No treatment with sunlight or under tube light at home can cure jaundice.
- Frequent breast feeding to be done when the baby is under phototherapy.

**Referral Criteria**

As per the chart if the bilirubin level exceeds normal range and enters phototherapy range baby should be refd to facility having phototherapy. After giving phototherapy always measure sr. bilirubin instead of estimating clinically.

- If during phototherapy bilirubin level enters exchange transfusion level, then it should be refd to institution having exchange transfusion facility.
- All babies having conjugated bilirubin >2mg/dl should be refd tertiary care centres having surgery facility.

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**FLUID AND FEEDING IN SICK NEWBORN**

**Introduction**

All the term babies after birth should be breast fed as soon as possible in the labour room. But some premature or asphyxiated babies may require feeding support to survive. By timely feeding and proper weaning from one method to another method prevents hypoglycaemia and better recovery. Different methods of feeding are-

- Breast feeding, Katori-spoon, Gavage and IV Fluid. But in assisted feeding importance should be given to use expressed breast milk.

**Diagnostic criteria**-

- Usually babies with gestational week >34 and birth weight >1800gm can breast fed.
- If the gestational age <34 weeks or birth weight <1800gm need assisted feeding or fluid.

**Non Drug Treatment**

- If 1200-1800 gm start gavage initially and switch over to Katori spoon and breast feeding subsequently
- In <1200 gm babies start with IV fluid and wean as per schedule if baby tolerates
- Give ONLY expressed breast milk by either spoon or by tube.
- For babies on intra gastric tube feeds, one can try cup or spoon feeds once or twice a day.

**Drug Treatment**

- If the child is <1200 gm or sick IV fluid should be started as per the given table. Wean from fluid to feed at 20-30ml/kg/day if baby improves subsequently.

<table>
<thead>
<tr>
<th>Day of life</th>
<th>Amount of fluids required (ml/kg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Birth weight &gt;1500 g</td>
</tr>
<tr>
<td>1</td>
<td>60</td>
</tr>
<tr>
<td>2</td>
<td>75</td>
</tr>
<tr>
<td>3</td>
<td>90</td>
</tr>
<tr>
<td>4</td>
<td>100</td>
</tr>
<tr>
<td>5</td>
<td>120</td>
</tr>
<tr>
<td>6</td>
<td>135</td>
</tr>
<tr>
<td>Day 7 onwards</td>
<td>150</td>
</tr>
</tbody>
</table>
Patient Education
The mother can also let baby suck on her breast after she expresses milk to stimulate her lactation. Mother should be involved in the care of baby and should be trained and supervised for paladai feeding. Continue to follow-up and weigh them regularly to make sure that they are getting enough breast milk.

Referral criteria
The premature and sick babies should be referred to SNCUs if they need assisted feeding and management of other conditions.
Babies having cleft lip and palate are to be managed with katori spoon or paladin till 3 months before referring for surgery.

BIRTH ASPHYXIA

Introduction: Birth asphyxia is one of the common cause of neonatal mortality and morbidity. This can be reduced by establishing NBCC and imparting training to service providers on neonatal resuscitation. Proper post asphyxia management can also minimise neurological deficits.

Diagnostic criteria- Those babies required some assistance to breathe at the time of birth may develop mild to severe form of symptoms.
- Mild- Excessive cry, refusal to feed, focal convulsions
- Moderate to severely asphyxiated babies may have seizures.
- Severely affected babies may manifest with stupor or coma, periodic breathing or irregular respiration, hypotonia and loss of complex reflexes like Moro’s and sucking.

Lab Investigations-
Blood Sugar, Sr Urea, creatinine, Calcium, Electrolytes, ABG analysis, Sepsis screening are essential for management of these babies.

Non Drug Treatment
- Temperature - Baby should be placed under a radiant warmer. The temperature should be maintained in the normal range of 36.5 – 37.5°C as hypothermia imposes additional stress to the baby by increasing the metabolic needs. This may lead to acidosis, myocardial depression, hypotension, bleeding tendency and pulmonary hemorrhage. Hyperthermia is detrimental and should be avoided.
- Oxygenation should be kept in the normal range by monitoring oxygen saturation by pulse oximetry, if facilities exist. SpO2 should be maintained between 88-92%. Hypoxia should be treated with O2 and if required ventilation. Hyperoxia should always be avoided.

Drugs Treatment
- Blood glucose level should be kept at 75-100 mg/dl to provide adequate substrate for the brain. If the baby is hypoglycaemic, 2ml/kg 10% DS to be given. The baby should be on 60mL/kg/day of 10% Dextrose as maintenance fluid. If still hypoglycaemia persists follow GIR chart for administration of glucose.
- Calcium level should be kept in the normal range and serum calcium should be maintained between 9-11 mg/dl. Hypocalcaemia is a common metabolic alteration and should be monitored within the first 24 hours. If hypocalcemic (<9mg/dL), give IV 10% Calcium gluconate @ 2mL/kg diluted 1:1 with 5% Dextrose under heart rate monitoring. Infuse at a rate of 1ml/min. Withhold infusion if HR <100/min.
• **Vitamin K** 1mg IM must be administered to all those babies who have not received Vit K at birth.

• **Management of Shock**-If CFT is >3 sec bolus NS10ml/kg bolus over 20-30mins can be administered which can be repeated if not improved. Subsequently Dopamine may be considered.

• Management of seizure as per guidelines given in following chapter.

• Patient Education Some cases of asphyxia can be prevented by better antenatal and intranatal management of high risk cases.

• The approach should be regular antenatal check ups to detect high risk cases and adoption of an ‘at risk approach’ to anticipate complications so that timely intervention in terms of emergency LSCS can be instituted.

**Referral criteria**-
- Failure to establish respiration by 5 minutes of life,
- Apgar score of 3 or less at 5 minutes,
- Onset of seizures within 12 hours
- Refractory Seizures
- Severe HIE
- Persistent Oliguria (<1 ml/kg/hr) for the first 36 hrs of life
- Inability to establish oral feeds by 1 week

****

**ORAL THRUSH**

**Introduction**
- Occurs in the babies where mother or family members clean the mouth with ghee or honey from an old container. The baby was not able to take breast feeding which results hypoglycaemia.

**Diagnostic criteria**-
- White patches inside the mouth and tounge which bleeds on attempt to remove the patch

**Lab Investigation**-
- Not required. Only by seeing the patch one can diagnose the disease.

**Non Drug Treatment**-
- Katori spoon feeding during treatment

**Drug Treatment**-
- Clotrimazole mouth paint should be applied 3 times in a day.

**Parent Education**-
- Mouth should not be cleaned for first 6months during exclusive breast feeding.
- Mother should clean her hand before applying the medicine.

**Referral criteria**-
- Can be managed at any facility or community by proper counselling of mother. But if associated with any other disease they should be refd for necessary treatment.

****
**PNEUMONIA IN CHILDREN**

**Definition**

Pneumonia refers to inflammation and infection of the lower respiratory tract, usually confirmed by parenchymal infiltrates on chest radiograph. Bacteria cause a significant proportion of pneumonias and resultant complications in children.

**Risk factors**

**Etiology**

*Streptococcus pneumoniae* (pneumococcus) Haemophilus influenzae are the most common bacterial pathogen in children 3 month to 4 yr of age, whereas *Mycoplasma pneumonia* and *Chlamydophila pneumoniae* are the most frequent bacterial pathogens in children age 5 yr and older. *Staphylococcus aureus*, and *group A streptococci* are less common.

**Diagnostic Criteria** –

Tachypnea is the most consistent clinical manifestation of pneumonia. Increased work of breathing accompanied by intercostal, subcostal, and suprasternal retractions, nasal flaring, and use of accessory muscles is common. Severe infection may be accompanied by cyanosis and lethargy, especially in infants. Abdominal pain is common in lower-lobe pneumonia. Cough is a predominant symptom in *Chlamydia* and *mycoplasma pneumonia*.

**Laboratory Test** –

- higher WBC count, erythrocyte sedimentation rate, procalcitonin, and C-reactive protein level-1

**Imaging** :

An infiltrate on chest radiograph (posteroanterior and lateral views) supports the diagnosis of pneumonia; the film may also indicate a complication such as a pleural effusion or empyema. Viral pneumonia is usually characterized by hyperinflation with bilateral interstitial infiltrates and peribronchial cuffing. Confluent lobar consolidation is typically seen with pneumococcal pneumonia

**Non drug treatment**:–

- Plenty of fluid intake if CCF is ruled out.

**Indications of hospitalization**:-

- Age <6 mo
- Sickle cell anemia with acute chest syndrome
- Multiple lobe involvement
- Toxic appearance
- Moderate to severe respiratory distress
- Requirement for supplemental oxygen
Dehydration

Vomiting or inability to tolerate oral fluids or medications

No response to appropriate oral antibiotic therapy

Social factors (e.g., inability of caregivers to administer medications at home or follow-up appropriately

**Drug Treatment –**

**Outpatient therapy:** • Oral high-dose amoxicillin (80–90 mg/kg/day divided b.i.d.). • A 2nd-generation cephalosporin (high dose) with activity against resistant S. pneumoniae is an alternative (eg, cefuroxime, cefpodoxime, or cefdinir).

• A single dose of ceftriaxone IV/IM may be given to initiate therapy.

**Inpatient therapy:** • IV ampicillin or a cephalosporin (cefuroxime, ceftriaxone cefotaxime).

Antibiotics should generally be continued until the patient has been afebrile for 72 hr, and the total duration should not be less than 10 days

Children >5 yr of age: – Coverage for both atypical pneumonia caused by M. pneumoniae or C. pneumoniae and typical pneumonia caused by S. pneumoniae should be considered. Macrolides(Azithromycin 12.5 mg/kg for 5 days ) effective for atypical pneumonia along with amoxicillin or cefuroxim.

Oral zinc (10 mg/day for <12 mo, 20 mg/day for ≥12 mo) reduces mortality among children with clinically defined severe pneumonia.

**COMPLICATIONS:**

Local complications: Pleural effusion, empyema, lung abscess, pneumatocele, pneumothorax, and pericarditis.

Hematogenous complications: Bacteremia, sepsis, osteomyelitis, septic arthritis, meningitis.

**Patient education**

• Treat upper respiratory and sinus infections at the earliest.

• Do not instil oil drops in cars.

**Referral Criteria –**

Any signs of complication. Immunocompromised state.s

**PEARLS AND PITFALLS:**

School-aged children may have either typical or atypical pneumonia and may require antibiotic coverage for both. Tachypnea is the most sensitive indicator of pneumonia in young children and infants.
BRONCHIAL ASTHMA

Asthma is a chronic inflammatory disorder of airways in which there is Bronchial hyper responsiveness. It is associated with episodic widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment.

Diagnostic Features –

All Asthma is characterized by the coincidence of recurrent symptoms of

• Wheezing
• Dyspnea
• Chest tightness
• And Cough which is usually nocturnal or in the early morning

Lab Investigation –

• Peak expiratory flow rate evaluation by Peak expiratory flow meter where available is a cost effective and valuable investigation for confirmation of reversibility of airway obstruction and monitoring of Asthma.
• X-ray chest P/A view in all patients with acute severe Asthma (especially in patients not responding to treatment) to exclude presence of Pneumothorax a rare but potentially fatal complication.

Non Drug Treatment

• Avoidance of exposure to known allergens and irritants

Drug Treatment –

<table>
<thead>
<tr>
<th>SEVERITY</th>
<th>DRUG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step – 1 / Mild intermittent</strong></td>
<td>• No daily medication needed&lt;br&gt;• Short acting $\beta_2$ agonist inhalation as needed for symptoms.</td>
</tr>
<tr>
<td>• Symptoms ≤ 2 times a week&lt;br&gt;• Asymptomatic and normal PEFR between exacerbations&lt;br&gt;• Night time symptoms ≤ 2 times a month&lt;br&gt;• FEV 1 or PEFR ≥ 80% predicted PEFR variability &lt;20%</td>
<td></td>
</tr>
<tr>
<td><strong>Step – 2 / Mild persistent</strong></td>
<td>• Once daily medication-&lt;br&gt;• Low dose inhaled corticosteroid or cromolyn&lt;br&gt;• Sustained release Theophylline (Orally)&lt;br&gt;• Inhaled short acting $\beta_2$ agonists (Solbutamol) as and when required.</td>
</tr>
<tr>
<td>• Symptoms &gt; 2 times a week but &lt; 1 time a day&lt;br&gt;• Exacerbations may effect activity&lt;br&gt;• Nighttime symptoms &gt; 2 times a month&lt;br&gt;• PEFR or FEV 1 ≤ 80% predicted &amp; PEFR Variability 20-30%</td>
<td></td>
</tr>
<tr>
<td><strong>Step – 3 / Moderate persistent</strong></td>
<td>• Daily medications-&lt;br&gt;• Medium dose inhaled corticosteroid&lt;br&gt;• Long acting inhaled $\beta_2$ agonist</td>
</tr>
<tr>
<td>• Daily symptoms&lt;br&gt;• Daily use of inhaled short acting beta 2 agonist</td>
<td></td>
</tr>
</tbody>
</table>
• Exacerbations ≥ 2 times a week, may last for days
• Night symptoms > 1 time a week
• FEV1 or PEFR > 60% - <80% predicted
• PEFR variability > 30%

**Step – 4 / Severe persistent**
• Continual symptoms
• Limited physical activity
• Frequent exacerbations
• Frequent Nighttime symptoms
• FEV1 or PEFR < 60% predicted & PEFR variability >30%

- Sustained release theophylline or long acting beta 2 agonist tablets
- Daily medications -
  - High dose inhaled corticosteroid
  - Long acting inhaled β2 agonist
  - Sustained – release theophylline or Long acting β2 agonist tablets
  - Corticosteroid tablets or syrup (2 mg / kg / day)

**Emergency Management of Asthma:**
• Nebulisation with short acting beta-2 Agonists and steroids
• I.v Hydrocortisone 5mg/kg to be started.

**Inhaled Corticosteroids –**

<table>
<thead>
<tr>
<th>Corticosteroid</th>
<th>Low Dose</th>
<th>High Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Budesonide</td>
<td>400mg / day</td>
<td>800 – 2000 mg / day</td>
</tr>
<tr>
<td>Beclomethasone dipropionate</td>
<td>800 g / day</td>
<td>800 – 2000 mg / day  Or,</td>
</tr>
<tr>
<td>Fluticasone propionate</td>
<td>400 -800 mg/day</td>
<td>800 – 2000 mg / day</td>
</tr>
</tbody>
</table>

**Inhaled Long acting β-2 agonist –**
• Formoterol 6 μg/12 hourly Or, Salmeterol 50μg/12hourly
• Usually step up approach (Step – 1 to 4) is adopted for management of Bronchial Asthma.
• When patient presents with exacerbations start high (Step – 4) and then step down if good symptom control for 3 months or more is obtained. If patient is well for 6 months anti-inflammatory treatment (Steroid) may be withdrawn.

**Aims of Management**
• Recognize asthma, Abolish symptoms, Restore normal or the best possible long, term airway function, Reduce the risk of severe attacks, Enable normal growth to occur in children, Minimize absence from school or work.

**Patient Education –**
• Teach basic facts about Asthma
• Inhaler / spacer technique
• Develop self management plan
• Develop action plan for when and how to take rescue actions specially for patients with history of severe attacks

**Referral Criteria –**
• Patient not controlled with above treatment protocol
• Any associated complications

**PEARLS AND PITFALLS**
Albuterol and levalbuterol, at equipotent doses, have similar efficacy and safety profiles.  
In severe asthma with very poor aeration, wheezing may not be heard.  
Ketamine is the sedative of choice for endotracheal intubation due to bronchodilatory properties.

****
**BROCHIOLITIS**

**Definition** –
Defined as inflammatory obstruction of small airways occurring commonly among infants aged 1 -3 months but may be encountered up to 2 years of age. **Acute bronchiolitis** is predominantly a viral disease. RSV is responsible for more than 50% of cases. Other agents include parainfluenza, adenovirus, rhinovirus, and *Mycoplasma*

**Diagnostic Criteria** –
- Previously healthy infants presenting with a first time wheezing episode during a community outbreak.
- Following mild upper respiratory tract infection patients present with fever (101 – 102°F), Gradually, respiratory distress ensues, with paroxysmal wheezy cough, dyspnea, and irritability. The infant is often tachypneic, which can interfere with feeding.
- Apnoea may be more marked during early part of the disease.
- Physically examination reveals wheezes with prolonged expiratory phase of breathing.
- Respiratory distress is out of proportion to the extent of the physical signs.

**Laboratory Test** –
Chest radiography can reveal hyperinflated lungs with patchy atelectasis but is not indicated in all patients with bronchiolitis.

**Non Drug Treatment** –
Positioning should be maintained to make the patient more comfortable in sitting posture with head and chest elevated 300 angle with neck extended. Frequent suctioning of nasal and oral secretions often provides relief of distress or cyanosis. Suctioning of secretions is an essential part of the treatment of bronchiolitis.

**Drug Treatment** –
Infants with **acute bronchiolitis** who are experiencing respiratory distress (hypoxia, inability to take oral feedings, apnea, extreme tachypnea) should be hospitalized; risk factors for severe disease include age <12 wk, preterm birth.
- Acetaminophen 15 mg/kg/dose PO/PR q4h PRN
- Cool humidified O₂ – 40 – 70%
- Maintenance with parenteral fluids
- Epinephrine – Nebulised solution – 0.25 = 0.5 ml of 2.25% recemic epinephrine diluted in 3 ml normal saline.
  Nebulized hypertonic saline 3% has been reported to have benefit.

**Patient Education** –
1. Exclusive breast feeding up to 6 months.
3. Sedatives are to be avoided because they may depress respiratory drive.
4. Antibiotics have no role unless there is secondary bacterial infection.
5. Corticosteroid has no role and is not indicated in previously healthy infants with RSV.

**Referral Criteria** –
Practically all cases should be admitted to hospital where there is O₂ facility & IV access.

**PEARLS AND PITFALLS**
Pay close attention to vital signs.
Very young infants and patients with comorbidities or highly abnormal vital signs have the greatest risk of decompensation.
The majority of infants with bronchiolitis require no investigations and no pharmacotherapy.
ACUTE OTITIS MEDIA IN CHILDREN

Definition
Otitis media refers to the presence of inflammation or infection in the middle ear space. A middle ear effusion (MEE) without infection is called otitis media with effusion (OME) or serous otitis. Infection of fluid in the middle ear is called acute otitis media (AOM).

Risk factors
- Day care attendance
- Smoke exposure
- Pacifier use
- Craniofacial anomaly (cleft palate)
- Genetic predisposition
- Previous AOM
- Persistent MEE/OME

Etiology AOM
is bacterial in 70–80% of cases
- S. pneumoniae
- Haemophilous influenzae
- Nontypable Haemophilous influenzae
- Less commonly Moraxella catarrhalis

Diagnostic Criteria –
The course of illness is important. With AOM, there is often a preceding URI, followed by acute onset fever and otalgia. Otalgia has a high specificity but low sensitivity. Restless sleeping or crying is neither sensitive nor specific.

In cases of perforated AOM, visualization of the TM may be impossible, and diagnosis is made by visualizing purulent fluid in the external auditory canal.

Laboratory Test –
- Complete blood count shows leucocytosis.
- Gram stain and culture of middle ear fluid obtained by tympanocentesis may be helpful in directing antibiotic therapy in complicated or resistant infections but is not routinely performed

Non-pharmacological
Stem inhalation and to keep the ear clean in case of pus discharge

Drug Treatment –
- Amoxicillin (high dose) 80–90 mg/kg/day PO divided b.i.d.: – High-dose amoxicillin is needed to overcome S. pneumoniae resistance. Duration of antibiotic therapy: 10 days in children
  In less than 2 years 5-7 days in more than 2 years. In case of recurrent ASOM 10 days course is required and immune suppression should be suspected.
- Second line drug are cefuroxime, cefdinir, macrolides.
- Nasal drops (saline) 4-5 times a day should be frequently instilled in both the nostrils.
COMPLICATIONS: Common complications of AOM are TM perforation and persistent effusion. Other complications include tympanosclerosis, cholesteatoma, chronic otitis, hearing loss, tinnitus, balance problems, facial nerve injury, mastoiditis, meningitis, intracranial abscess, and venous sinus thrombosis.

Patient education
• Treat upper respiratory and sinus infections at the earliest.
• Do not instil oil drops in ears.

Referral Criteria –
Any signs of complication.

PEARLS AND PITFALLS
Otitis-conjunctivitis syndrome is more likely to be caused by H. influenzae, and amoxicillin/clavulanic acid should be given.
Bullous myringitis (blisters on the TM) may be caused by the typical bacterial pathogens or M. pneumoniae.

****

ACUTE DIARRHOEA IN CHILDREN

Definition: Acute Diarrhoea is defined as passage of liquid/watery stools, which starts suddenly & may continue for number of days (usually 3-7 days), but not more than 14 days. It is the recent change in consistency and character of the stools which is more important than frequency of stools.

Note: Passage of frequent formed stools, pasty stools in breastfed infants, transitional stools on the 3rd to 7th day of life, passage of stools during or immediately after feeds due to initiation of gastrocolic reflex are not considered as diarrhoea.

Diagnostic Criteria:
1. Loose stools, which may be voluminous (Stool output is more than 10gm/kg/24hour or more than adult limit of 200gm/24 hour) or of less amount (osmotic diarrhoea).
2. Dehydration - This may be recognized as follows as per WHO Criteria.
   A young infant with diarrhoea is assessed for:
   - how long the young infant has had diarrhoea
   - blood in the stool to determine if the young infant has dysentery, and for
   - signs of dehydration.

<table>
<thead>
<tr>
<th>SI no.</th>
<th>No dehydration</th>
<th>Some dehydration</th>
<th>Severe dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Look at condition(a)</td>
<td>Well, alert</td>
<td>Restless, Irritable*</td>
</tr>
<tr>
<td>2.</td>
<td>Thirst</td>
<td>Drinks normally not thirsty</td>
<td>Drinks eagerly, thirsty*</td>
</tr>
<tr>
<td>3.</td>
<td>Skin pinch(b)</td>
<td>Goes back quickly</td>
<td>Goes back slowly* (pinch stays for a brief period)</td>
</tr>
<tr>
<td>4.</td>
<td>Mouth &amp; Tongue (c)</td>
<td>Normal</td>
<td>Dry</td>
</tr>
<tr>
<td>5.</td>
<td>Eyes(d)</td>
<td>Moist</td>
<td>Sunken</td>
</tr>
<tr>
<td>6.</td>
<td>Treatment</td>
<td>PlanA</td>
<td>PlanB</td>
</tr>
</tbody>
</table>
* Highlighted signs
- Presence of two or more signs including at least one highlighted sign gives the diagnosis of dehydration
  a) A lethargic child is not simply asleep; the child’s mental state is dull; the child may appear to be landing into unconsciousness.
  b) The skin pinch is less useful in infants or children with marasmus or Kwashiorkor or in obese children.
  c) The mouth may be dry in child who habitually breathes through the mouth. The mouth may be wet in a dehydrated child owing to recent vomiting or drinking.
  d) In some infants and children the eyes normally appear somewhat sunken. It is helpful to ask the mother if the child’s eye are normal or more sunken than usual.
3) Fever may also be associated.

**Laboratory Tests**
1. Routine & microscopic examination of stool
2. Stool culture
   - This is indicated in the following conditions-
     a) Outbreaks of diarrhoea
     b) Stools containing fecal leucocytes
     c) Patients with bloody diarrhoea
     d) Suspected Haemolytic Uremic Syndrome
     e) Diarrhoea in immune-compromised individual
   In microscopic examination if pus cells are more than 10 per high power field, antimicrobials are indicated.

**Non Drug Treatment**
1. Breast feeding is to be continued in infants. Older children should be re-fed as soon as they can tolerate feeding (after ORT)
   - Food with complex Carbohydrate (e.g. rice, wheat, potatoes, bread & cereals), meats, yogurt, fruits & vegetables are better to tolerate.
   - Fatty foods or foods high in simple sugars (including juices and carbohydrates, sodas, glucose drinks) should be avoided as they may cause osmotic diarrhoea.
2. Home available fluids-
   - Fluids like rice water, buttermilk, lassi with salt, lemon water with salt, soup, coconut
   Water and plain water.

**Plan A**
- Mother should be allowed to give increased amount of home available fluids
- WHO ORS to be given by health care provider. One pack to be dissolved in 1 litre of water
- Mother should be asked to bring the child to the health worker if diarrhoea does not improve by 3 days or develop danger signs: many watery stools, repeated vomiting, marked thirst, eating or drinking poorly, fever or blood in stool
Quantity of ORS

<table>
<thead>
<tr>
<th>Age</th>
<th>Amount of ORS after each loose stool</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 24mo</td>
<td>50-100ml</td>
</tr>
<tr>
<td>2mo to 10yrs</td>
<td>100-200ml</td>
</tr>
<tr>
<td>10yrs or more</td>
<td>as much as wanted</td>
</tr>
</tbody>
</table>

- Describe and show the amount to be given after each loose stool and mixing of ORS
- Give a teaspoonful every 1-2 minute for a child below 2yrs
- Give frequent sips from a cup for an older child
- If child vomits, wait for 10 minute. then give slowly-1 spoon every 2-3 min
- If diarrhoea continues tell mother to give other fluids or ORS

**Plan B-for some dehydration**

ORS should be started promptly
a) Correction of existing water and electrolyte deficit-give 75ml/kg of ORS in 4hrs
b) Maintenance fluid therapy-after dehydration correction in 4 hrs. ORS should be administered according to loss of fluid in stool-10-20ml/kg body weight for each liquid stool till diarrhoea stops. Offer plain water inbetween.
c) Provision of normal fluid requirement-Breast feeding to continue during rehydration also. Offer semisolid food soon after deficit replacement.

If the child has some dehydration after 4hrs, repeat another 4hr treatment with ORS

**Plan C-for severe dehydration**

Start IV fluid immediately. While the drip is being set up, give ORS solution if child can drink
Best IV solution is Ringer’s lactate. Normal saline can be given if Ringer’s lactate is not available. IV is given 100ml/kg

<table>
<thead>
<tr>
<th>Age</th>
<th>First give</th>
<th>Then give</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;12mo</td>
<td>30ml/kg in1hr*</td>
<td>70ml/kg in 5hrs</td>
</tr>
<tr>
<td>&gt;12mo</td>
<td>30ml/kg in 1/2hr*</td>
<td>70ml/kg in 2 1/2 hr</td>
</tr>
</tbody>
</table>

* Repeat again if the radial pulse is weak

All children should be started with ORS about 5ml/kg/hr when they are able to drink
If one is unable to give IV fluid start rehydration with ORS 20ml/kg/hr (total of 120ml/kg)

Reasses every 1-2hr

*In case of vomiting or abdominal distension give more slowly
*Antimotility drug, antisecretory drug have no role in diarrhoea

**Antibiotics**

- Large majority of cases of diarrhoea are due to viral origin
- Shigella, vibrio cholera, Entamoeba histolytica and giardia needs treatment
- Cholera-Tetracycline-12.5mg/kg/dose 4times/day for 3days in older children, cotrimoxazole-TMP 5mg/kg/dose twice a day for 3 days

**Zinc**: <6mo child -10mg/day for 14days and 20 mg/kg for >6mo child for 14days
Probiotics
Can enhance host protective immunity
lactobacillusBifidobacterium have good safety
Saccharomyces boulardii is effective in antibiotic associated and C difficilediarrhea

Antiemic
Ondansetron is an effective and less toxic antiemetic drug-4mg 4-11yr and 8mg fot children older than 11yrs(.2mg/kg)

Rotavirus immunization
Vaccine causes 41% reduction in diarrhoea related mortality in infants.protective efficacy against gastroenteritis is from 49-61%

Cholera vaccine
Vaccine reduces the risk of cholera by 52%

Referral criteria
Young infants less than 2mo of age
Anuria aftercorrec ting fluid challenge
Severe dehydration not corrected after giving IV fluid
Associated systemic infection like septicaemia,meningitisetc

DYSENTRY IN CHILDREN

Dysentry is defined as presence of blood and mucus in stool,usually caused by Shigella,entero invasive E.Coli and entamoebahistolytica.It is often associated with systemic complications and high mortality rate,The two main causes of dysentery are Bacillary (Bacterial)and Amoebic(Protozoal).

Diagnostic Criteria
Fever,Tenesmus,Anorexia,AbdominalPain,frequent passage of small quantity of stool mixed with blood and mucus.

Laboratory test
Routine and microscopic examination of stool

Non Drug Treatment
1. Breast feeding is to be continued in infants.Older children should be re-fed as soon as they can tolerate feeding(after ORT)
   - Food with complex Carbohydrate(e.g-rice, wheat, potatoes, bread & cereals) meats, yogurt, fruits & vegetables are better to tolerate.
   - Fatty foods or foods high in simple sugars(including juices and carbohydrates,sodas,glucose drinks)should be avoided as they may cause osmotic diarrhoea.
2. Home available fluids-
   Fluids like rice water,buttermilk,lassi with salt ,lemon water with salt,soup ,coconut Water and plain water.
Drug Treatment

Bacillary Dysentry
1) Cotrimoxazole-Syrup 5ml(40mg T +200mg S) or Tab (80mg T +400mg S) or DS Tab (160mgT +800mg S) given 10mg/kg /day in two divided doses for 5 days
2) Nalidixic Acid-Syrup 5ml-125mg or Tab 250 mg,500mg given 55mg/kg/day -4divided doses for 5 days
3) Norfloxacin-Indications-seriously ill patients or suspected or known to be resistant to other antimicrobials-syrup 5ml=100mg Or Tab 100mg,200mg,400mg .Give 10mg/kg/day in 2 divided doses for 5 days
   Amoebic dysentery
   Metronidazole-syrup 5ml=100mg & 200mg or Tab 200mg,400mg
   For Amoebiasis-30-50mg/kg/day(Max 500-750mg/dose ) in 3 devided doses for 5-10 days
   For Giardiasis-15mg/kg/day-in 3 divided doses(Max 750mg/d)-5days

Patient/Parent Education
1) The mother should be educated to practice exclusive breastfeeding upto 6months of age with improved weaning practices
2) Measles immunization at 9-12 months of age
3) Maintenance of personal hygine of child particularly in relation to food ,use of safe drinking water ,use of latrines and proper disposal of stools

Referral Criteria
1) Young infant of less than 2month of age with diarrhoea
2) Anuria after correction of dehydration & further fluid challenge
3) Severe dehydration not corrected after IV rehydration therapy
4) Associated systemic infections like septicaemia,meningitis or pneumonia
5) Assess and classify diarrhoea:p 144-153,part 1 integrated management of neonatal and childhood illness:WHO

MALARIA

Definition : Malaria is among the most common vector-borne illnesses worldwide. Its a febrile illness due to the Plasmodium species of Protozoan parasites. Plasmodium falciparum and Plasmodium vivax most commonly infect humans, though Plasmodium malariae and Plasmodium ovale also produce infection. The Anopheles mosquito serves as the vector for Plasmodium transmission.

Diagnostic Criteria :

History:
• Recent travel to malaria-endemic region and prescription / compliance with malaria prophylaxis.
• Symptoms typically develop within the 1st month after return from an endemic zone, though presentation up to 12 months later may occur.
• Malaise, High fever, Headache, Chills, Sweating, Rigors, and Myalgia / Arthralgias are common presenting complaints.
• Classic malarial paroxysm is 15-60 mins of chills followed by 2-6 hrs of non-diaphoretic fever:
Profuse sweating occurs with defervescence.
Pattern of fever can be helpful; with *P. falciparum* typically recurs every 72 hrs, but with other *Plasmodium* infections, fever occurs at 48 hrs interval.
- Cough, Irritability, Anorexia, Vomiting, abdominal pain, back pain and Arthralgias may also be reported.
- Dark Urine.

**Physical Examination:**
- Ill-appearance during fever, with relatively well appearance in between (inter-febrile phase).
- Jaundice, Pallor.
- Hepato-splenomegaly may be present.
- Rash is not a typical feature of Malaria; the presence of rash should elicit consideration of alternative diagnosis.
- GI symptoms of vomiting, diarrhoea and fever may occur.
- Pulmonary findings including dyspnoea, rales and pulmonary oedema may occur.
- Cerebral Malaria, the most severe complication of malaria, may manifest with signs of increased intracranial pressure, encephalopathy, focal neurologic findings and seizures.

**Rational Investigation:**
- **CBC**
  - **Haemoglobin:** Haemolytic anaemias present initially as mild but may be more severe depending on the *Plasmodium* species.
  - **Leucocyte counts:** Are usually normal or low, **eosinophilia is not present.**
  - **Thrombocytopenia:** May occur in severe cases due to liver and splenic sequestration.
- **Peripheral Blood Smear**
  - **Microscopic identification** is essential for the diagnosis of Malaria.
  - **Thick and Thin peripheral blood smears** are both required (*Thick* smears enable better sensitivity level if parasitaemia is low; *Thin* smears allow for species identification).
  - **If initial smears are negative**, repeat specimens should be obtained q8-12h during a 72-hr period to confirm a truly negative result.
- **The percent of infected red cells (Parasitaemia)** is an important risk factor for illness severity:-
  - **Parasitaemia >5%**, CNS involvement (e.g. Mental status changes), or **other organ involvement** are indications of more aggressive, intensive therapy.
- **Quantitative Buffy Coat (QBC)** analysis is available for rapid screening, but confirmation by blood smear is necessary.
- **Serologic tests and indirect immunofluorescent assays** have low sensitivity in early phases of acute infection.
- **Other tests including Rapid Detection Kits (Rapid Diagnostic tests—RDT-ICT) and Polymerase Chain Reaction (PCR)** techniques have demonstrated variable sensitivity and specificity, but may be of use.
- **RDTs** The NVBDCP has recently rolled out bivalent RDTs (For detecting *P. falciparum* and *P. vivax*) for use in the public health sector. It should be noted that Pf HRP-2 based kits may show positive result up to three weeks after successful treatment and parasite clearance. In these cases, results should be correlated with microscopic diagnosis.
- **Relevant investigations to R/O** (a) Other common endemic diseases in high-malaria zones, (b) Common aetiologies of Fever, (c) Other causes of altered mental status and (d) Other causes of Diarrhoeal diseases.
Initial Stabilization Therapy:

- **Airway** protection and **Circulatory** stabilization are frequently indicated.
- **Hypoglycaemia** is a common complication of Severe Malaria; Early detection and treatment is necessary.
- Using blood gas measurement, treat **tissue hypoxia** using IV fluid and supplemental Oxygen.
- **Seizures** may result from cerebral malaria and should be treated promptly with Benzodiazepines.
- **Aggressive antimalarial** treatment should be promptly administered.

**Drug Treatment:**

- **Treatment of Uncomplicated Malaria:**
  - All fever cases diagnosed as malaria by RDT or Microscopy, should be given effective treatment.
    - **Treatment of P. vivax Malaria:**
      - Confirmed Pv cases should be treated with Chloroquine in full therapeutic dose of 25 mg/kg (1st dose 10 mg/kg, 2nd dose 10 mg/kg after 24 hr and 3rd dose 5 mg/kg after 48 hr of 1st dose) PO.
      - Primaquine should be given at a dose of 0.25 mg/kg body weight daily PO for 14 days under supervision (P. vivax or P. ovale). Primaquine is contra-indicated in Pregnant women, Lactating mother up to 6 M, Infants up to 6 M and KCO G-6-PD deficient patients.
    - **Chloroquine resistant Pv should be treated as Pf. Cases.**
  - **Treatment of P. falciparum Malaria:**
    - Artemisinin Combination Therapy (ACT) should be given to all the confirmed Pf case found positive by RDT or Microscopy.
    - This is to be accompanied by single dose of Primaquine (0.75 mg/kg BW) on Day 2.
    - Dosage Schedule;
    - AS+SP and PQ (Day-1 AS+SP, Day-2 AS+PQ and Day-3 AS only) as per National Malaria treatment Guideline.
    - Fixed Drug Combination (FDC) of Artemether-Lumefantrine (AL) as per National Malaria treatment Guideline(Twice a daily dose for 3 days, preferably after fatty meal).
    - **MONOTHERAPY OF ORAL ARTEMISININ DERIVATIVES IS BANNED IN INDIA.**
  - **Treatment of Mixed Infections:**
    - Mixed infections with Pf should be treated as falciparum malaria. Since AS+SP is not effective in Vivax malaria, AL should be used.
    - Anti-relapse treatment with PQ can be given for 14 days, if indicated.
  - **Treatment based on Clinical Criteria without Laboratory Confirmation;**
    - All the efforts should be made to diagnose malaria either by Microscopy or RDT. However, special circumstances should be addressed as below:
      - If RDT for only Pf is used, negative cases showing signs and symptoms of malaria without any other obvious cause for fever, should be considered as CLINICAL MALARIA, and treated with Chloroquine in full therapeutic dose. Treatment to be completed according to species as per the slide results obtained later.
      - Suspected malaria cases not confirmed by RDT or Microscopy, should be treated with Chloroquine in full therapeutic dose.
  - **General Recommendations for management of Uncomplicated Malaria:**
    - Avoid starting treatment on an empty stomach. The first dose should be given under observation.
    - Dose should be repeated if vomiting occurs within 30 minutes of antimalarial intake.
    - The patient should be asked to report back, if there is no improvement after 48 hrs or if the situation deteriorates.
    - The patient should also be examined and investigated for concomitant illnesses.
Treatment of Severe Malaria;
- Severe manifestations can develop in Pf infection over a span of time as short as 12-24 hours and may lead to death if not treated promptly and adequately. Severe Malaria is characterized by one or more of the following features:
  - Impaired consciousness / coma.
  - Repeated generalized convulsions.
  - Renal failure (Serum Creatinine > 3 mg / dl).
  - Jaundice (Serum Bilirubin > 3 mg / dl).
  - Severe anaemia (Hb < 5 g / dl).
  - Pulmonary oedema / ARDS.
  - Hypoglycaemia (Plasma Glucose < 40 mg / dl).
  - Metabolic acidosis.
  - Circulatory collapse / Shock.
  - Abnormal bleeding and DIC.
  - Haemoglobinuria.
  - Hyperpyrexia (Temperature > 106 d F or 42 d C).
  - Hyperparasitaemia (> 5 % parasitized RBCs).
- Specific antimalarial treatment of severe malaria;
  - Artesunate: 2.4 mg / kg BW IV or IM given on admission (time=0), then at 12 and 24 hours, then once a day (Care should be taken to dilute artesunate powder in 5 % Sodium bi-carbonate provided in the pack) for 7-10 days. OR
  - Quinine: 20 mg Quinine salt / kg BW on admission (IV infusion in 5 % Dextrose / Dextrose Saline over a period of 4 hrs) followed by maintenance dose of 10 mg / kg BW 8 hourly; infusion rate should not exceed 5 mg / kg / hour. Loading dose of 20 mg/kg should not be given if already received Quinine. (NEVER GIVE BOLUS INJECTION OF QUININE). If parenteral quinine therapy needs to be continued beyond 48 hrs, dose should be reduced to 7 mg/kg BW, 8 hrly.

Patient /Parent Education: -
- Anti-Mosquito measures: Use of personal protection measures like insecticide-treated bed nets should be encouraged for pregnant women and other vulnerable populations.
- Chemoprophylaxis: Recommended for travellers, migrant labourers and military personnel exposed to malaria in highly endemic areas.

Referral / Admission Criteria: 
- Most patients should be admitted and managed as inpatients.
- Even if initial peripheral blood smears are normal but there is still a high clinical suspicion for malaria, the patient should be admitted.
- Critical care admission criteria: 
  - Severe or complicated malaria (eg Cerebritis, Severe anaemia, Shock, Acidosis, DIC) should be managed in intensive care setting.

Pearls and Pitfalls: 
- Delay in diagnosis increases morbidity and mortality up to 20-fold compared to diagnosis within 24 hr of presentation,
- Even with optimal therapy, severe malaria has high morbidity and mortality.
- Periodicity of fever is less commonly seen in young children and travellers.
- Malaria must be ruled out in any febrile traveller returning from an endemic zone.
- If treated promptly, even P. falciparum malaria responds well to current treatment options if severe parasitaemia is not present.
MEASLES

Definition:
- Measles is an exanthematous, highly contagious paramyxovirus infection.
- Clinical syndrome includes:
  - Classical measles in immunocompetent patients marked by high fever, rash, conjunctivitis and cough.
  - Modified measles in patients with partial immunity:
    - Longer incubation period (17-21 days).
    - Milder symptoms than classic disease.
  - Severe, progressive measles in patients with defects in cell-mediated immunity (AIDS, Lymphoma, Malignancies).

Diagnostic Criteria:
- History:
  - Prodromal phase (lasts 2-4 days);
    - Characterised by malaise, fever, coryza, conjunctivitis and cough.
    - Fever is an early sign, often before the start of respiratory symptoms.
    - Early respiratory symptoms mimic the common cold.
    - Photophobia, increased lacrimation from conjunctivitis.
  - Exanthema phase (appears on the 14th day after exposure—4th day of fever);
    - Spread of rash is centrifugal—from head to feet, starts behind the ears and on the forehead at the hairline.
    - Fever peaks on the 2nd day of rash, (Fever persistent beyond the 4th day of rash is often a sign of complication).
    - Vomiting, diarrhoea and abdominal pain may be seen in young children.

- Physical Examination:
  - Prodromal phase;
    - May have transitory macular rash early in the prodrome phase.
    - Slit lamp reveals corneal and conjunctival lesions.
    - Cough is described as having “BRASY” quality and becomes worse through the prodromal phase.
    - On day 10 after exposure, Koplik spots will be seen (Pathognomonic of Measles).
    - 4-Cs (Cough, Coryza, Conjunctivitis and Koplik spots)
  - Exanthem phase (lasts 6-7 days);
    - Appears after Koplik spots have peaked.
    - Starts as erythematous and maculopapular, spreading from head to feet.
    - Rash then confluences in the same centrifugal pattern, always worse on the face.
    - After rash fades (by 4th day of rash), skin may look coppery and then desquamate.
    - Cough may still be persistent in this phase for 10 more days.
    - Pharyngitis, lymphadenopathy and splenomegaly also are seen in this phase.

Rational Investigation:
- CBC; May have leucopenia during prodrome and exanthem phases.
- Diagnosis mostly clinical and suspected cases should be reported to local health authorities.
• Imaging: CXR→ Either primary Viral or subsequent Secondary Bacterial Pneumonia.

Non-Drug Treatment:
• Assess and stabilize Airway, Breathing and Circulation.
• Isolation from other individuals particularly unimmunized children for at least 4 days after the appearance of the rash.
• Primary therapy → Supportive care in normal hosts and prevention of subsequent complications.
• Bed rest is usually required and tepid sponging may be required for febrile patients.
• Small frequent feeds and plenty of oral fluids should be continued.
• Antitussives when indicated for persistent cough.

Drug Treatment:
• No specific antiviral treatment is available.
• Antipyretic;
  o Acetaminophen 15 mg/kg BW PO/PR q4h PRN.
  o Ibuprofen 10 mg/kg BW PO/IV q6h PRN.
• Antitussive;
  o Dextromethorphan:-
    ▪ 2-6 yrs of age; 2.5 – 7.5 mg PO q6h PRN.
    ▪ 7-12 yrs; 5 — 10 mg PO q4h PRN.
    ▪ > 12 yrs; 10 – 30 mg q4-8h PRN.
  o Codeine:-
    ▪ > 2 yrs; 1.5 mg/kg/day divided q4-6h PR.
• Vitamin A;
  o 6-12 M of age; 100,000 IU PO / day X 2 days.
  o 1-2 yrs; 200,000 IU PO / day X 2 days.
  o Although the mechanism of action of Vitamin-A is not known, it is thought that it may help treat a viral-induced state of Hyporetinaemia.
  o The WHO recommends Vitamin-A to the children of all areas where Vitamin-A deficiency is prevalent or mortality for measles is > 1 %.

Patient/Parent Education:-
• Measles leads to marked anorexia and also often precipitates PEM and other deficiencies. Regular frequent feeds must continue.
• The disease usually lasts for 10-12 days and the maximum risk of infectivity is 5 days prior to and 4 days after the appearance of rash. The rash usually heals by desquamation and often leaves some hyperpigmented stains on the body which disappears over weeks subsequently.
• The parents must report to the hospital in case the child develops any of the following warning signs;
  o Stiff neck, facial twitching or convulsions, extreme drowsiness, loss of consciousness or altered behaviour.
  o Rapid and laboured breathing, difficulty in feeding or cyanosis.
  o Significant dehydration or passing blood in stool.
• Vaccination against measles is recommended at 9 months of life and subsequent doses in the form of MMR is mandated at 12 through 18 months of age. Catch up vaccination beyond 12 months should be MMR. There is no role of giving measles vaccination to a child who has already suffered from the disease. During outbreaks of measles, vaccination can be administered to unimmunized infants at 6 months of age.
Referral Admission Criteria:
- Severely ill patients with dehydration, altered mental status, seizures, immune-compromise or respiratory distress should be hospitalized.
- Critical Care admission criteria;
  - Severe Respiratory compromise.

Pearls and Pitfalls:
- The characteristic spread of the measles rash from head to feet helps to distinguish it from other viral exanthems.
- In the emergency department waiting and exam areas, children suspected to have measles must be separated from others.
- Children with risk factors for lower vitamin-A levels due to virus (and thus at increased risk for morbidity from the illness) should receive vitamin-A therapy.

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MUMPS IN CHILDREN

Mumps virus is in the family Paramyxoviridae and the genus Rubulavirus. Mumps is an acute self-limited infection. It is characterized by fever, bilateral or unilateral parotid swelling and tenderness, and the frequent occurrence of meningoencephalitis and orchitis. Only cause of epidemic parotitis.

Commonly associated conditions: Parotitis, Orchitis, Aseptic meningitis, Pancreatitis

Diagnostic Features:
- Often presents with nonspecific prodromal symptoms including fever, malaise, myalgias, anorexia, and headache
- Parotitis usually ensues within <48 hr of symptom onset:
  - Can be unilateral, bilateral, or subclinical
  - Often begins as otalgia and pain over the angle of the jaw
  - Progresses to pain and swelling over the parotid gland, both pre- and postauricular 1/3 of patients present with respiratory symptoms, with no significant parotitis.
  - Complaints of localized pain may point to specific complications such as meningitis, orchitis, or pancreatitis.
  - Swelling and erythema of Stensen’s duct, where the parotid gland inserts in the mouth (at level of upper 2nd molar)
  - Parotid pain worsens with salivation.
  - Morbilliform rash may rarely be present.
  - Mumps orchitis presents with testicular pain and swelling with associated systemic symptoms. Aseptic meningitis presents with fever and neck stiffness.
  - Epigastric tenderness may result from pancreatitis

Laboratory Test:
- Leukopenia with lymphocytosis and elevated serum amylase support the diagnosis. Serum lipase may be used to detect pancreatic involvement.
Treatment

Non-pharmacological

- Child should be encouraged to drink plenty of fluids, water, decaffeinated soft drinks and tea are better tolerated than acidic fruit juices (like orange juice, grape fruit juice or lemonade) that make parotid pain worse.
- Either warm or cold packs, whichever feels better, may be used to soothe the swollen parotid glands.
- Patients with abdominal pain, testicular swellings or signed intracranial tension need to be admitted in the hospital.

Pharmacological

1. Most cases are treated symptomatically on OPD basis.
2. Fever when troublesome may be brought down using non-aspirin fever medications such as paracetamol (10-15 mg/kg/day SOS or every 4-6 hours). These medicines will also help relieve pain in the swollen parotid glands.
   (Caution: Aspirin is contraindicated in children with viral illnesses due to risk of Reye’s syndrome)
3. Being a viral illness, antibiotics have no role. There is no specific therapy available.

Patient / Parent education

- Parents should be explained the warning signs, e.g.
  - boys, parents are told to watch for high fever, with pain and swelling of the testicles.
  - Watch for abdominal pain that can mean involvement of the pancreas in either sex or involvement of the ovaries in girls.
  - Severe headache, stiff neck, convulsions seizures), extreme drowsiness, etc., suggest CNS involvement and need for admission to a tertiary level centre.
  - Recurrence of high grade fever (above 101°F / 38.3°C) often herald onset on the above complication and can be used as an early referral sign.
- Children usually recover from mumps in about 10-12 day. First attack of mumps almost always gives lifelong protection against another; therefore, such children not benefit from any immunisation later.

Prevention:- By vaccination(MMR or MR)

Referral Criteria :

Sometimes necessary in cases of mumps orchitis or pancreatitis Meningitis with need for IV antibiotics pending CSF cultures

PEARLS AND PITFALLS:

A failure to recognize mumps infection can lead to local outbreaks. The MMR vaccine should not be deferred for children with mild febrile illnesses. Close contacts of immunocompromised children should be vaccinated with the MMR vaccine; they will not transmit the mumps virus. Expression of pus through Stensen’s duct can help differentiate bacterial parotitis from viral parotitis.

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CHICKEN POX OR VARICELLA IN CHILDREN

Varicella zoster virus (VZV), a herpes virus, results in two common clinical entities:
Varicella (Chickenpox), is a result of primary infection, and is characterized by fever and disseminated rash.
Herpes Zoster (Shingles), is reactivation disease of latent virus residing in the dorsal root ganglion from previous infection (or immunization). It is characterized by a painful, vesicular rash of dermatomal distribution.

Diagnostic Features:

Varicella:
– Prodrome of fever and malaise 1–3 days prior to rash
– Resolution of fever within 24 hr of rash
– Universally pruritic centripetal rash starting on trunk/neck/face and progressing outward
– Rash: Erythematous papules (2–4 mm) evolving to vesicles, then pustules over 6–8 hr, then crusting within days
– Successive crops appear over 2–4 days, so lesions (250–500) are in various stages of evolution. – Mucosal exanthem (oropharynx; less commonly, the vagina)
– Secondary family cases have more lesions than the index case.

Treatment:

Non-pharmacological
• Itching is bothersome and scratching effect may be minimised by making the patient wear mittens, daily change of clothes and good personal hygiene may decrease the risk of secondary infection.

Pharmacological:

Local antipruritic agents like calamine lotion may alleviate itching. If itching is not relieved with above, Tab. Pheniramine 25 mg 2 times a day.
In children : Syr. 0.5 mg/kg/day every hours
Or
Tab. Cetirizine 10 mg once a day
In children (2-6 years) : 5 mg; (>6 years) 10 mg once a day.

Analgesic may be needed, particularly for varicella zoster:
– Acetaminophen 15 mg/kg/dose PO/PR q4h PRN ,Ibuprofen 10 mg/kg/dose PO/IV q6h PRN
– Consider acyclovir if increased risk for complications (chronic lung diseases, patients receiving long-term salicylate or steroid [oral/inhaled] therapy, secondary household cases, pregnant women):
  • Oral acyclovir, if given within 24 hr of onset of rash, will reduce the duration of fever and the number/duration of lesions (80 mg/kg/day divided q6h × 5 days).
  • Acyclovir for any hospitalized child with ongoing vesicle formation (1,500 mg/m2/day IV divided q8h × 7–10 days)
  • Immunocompromised hosts require IV acyclovir (prevents visceral dissemination)

Patient/Parent education
• The disease commonly is self-limiting in healthy children. Child should be excluded from day care or school till after 6th day of the rash or till scabs are formed.
• Do not use over-the-counter fever medicines as they may contain aspirin or other salicylates.

Prevention: By vaccination

Referral Criteria:
• Immunocompromised patient, Newborn,

PEARLS AND PITFALLS:
Postvaccine varicella occurs in 3–5% of children at 5–26 days after immunization (typically 2–5 lesions, rash may be maculopapular). Immunosuppressed individuals, pregnant women, and infants 10–20 yr. VariZIG is not indicated for healthy term infants with postnatal varicella exposure (including those whose mother’s rash developed >48 hr post delivery). Avoid salicylates due to increased risk of Reye syndrome.

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ACUTE BACTERIAL MENINGITIS

Clinical Features:
Fever, headache, neck stiffness, photophobia, nausea, vomiting and altered mental status → (lethargy to coma).

Infants, elderly, and immunocompromised patients - only mild behavioural changes, low-grade fever and little clinical evidence of meningeal inflammation.
Focal cerebral findings,
Seizures→

Diagnosis
Urgent lumbar puncture (LP)-CSF examination, CSF culture & gram staining. CECT before LP - focal findings or clinical evidence of raised intracranial pressure → CSF examination - elevated pressure (200-500 mm H2O) and protein (100-500 mg/dl, normal 15-45 mg/dl), decreased glucose (<40% of serum glucose), and marked pleocytosis (100-10,000 white blood cells / ul, normal <5) with 60% or greater polymorphonuclear leucocytes. Early in disease, 10-20% of patients have CSF cell counts less than 1,000 cells/ul. Otherwise, cell counts below 1,000 cells/ul in a patient with a compatible clinical syndrome indicate partially treated meningitis, concurrent immunosuppression, or a nonbacterial cause.

The likelihood of finding Gram’s stain or culture-positive CSF may decrease, if antibiotics are administered before doing LP. Petechial or purpuric rash suggests N. meningitidis, or, less often, Staphylococcus aureus, Pneumococcus, or the Rickettsiae.

Treatment Nonpharmacological: Hospitalize in a quiet place with no bright lights preferably in ICU. Maintain vitals, endotracheal intubation in patient with poor respiratory effort. Elevation of head to 30° and hyperventilation, if evidence of raised intracranial pressure. Pharmacological: Empiric antimicrobial to be initiated based on the patients age and underlying disease status; once a bacterial pathogen is isolated, antimicrobial therapy can be modified for optimal treatment. 1. Inj. Ceftriaxone 4 g/day in 2 divided doses administered every 12 hours. It can also be administered as a single dose. Or Inj. Cefotaxime 8-12 g/day, in divided doses administered every 4 hours. If age is more than 50 years to cover Listeria monocytogenes and Pseudomonas or in immunocompromised patients. Inj. Ceftazidime 8 g/day in divided doses administered every 6 hours plus Inj. Ampicillin 10-12 g/day in divided doses...
every 4 hours. Or In case of resistant Gram-negative infection and are sensitive to Inj. Meropenem 1-2 g 8 hourly. Continue treatment for 10-14 days with antibiotics. Clinical response is observed within 24-48 hours. Repeat lumbar puncture.

In case of head trauma, CSF rhinorrhoea, intracranial shunt or history of neurosurgical intervention, when penicillin or cephalosporin resistant strains of Streptococcus pneumoniae are suspected, add Inj. Vancomycin 500 mg IV 6 hourly. 3. In H. influenzae type-B meningitis, pneumococcal meningitis in children and in children over 2 months of age with neurological sequelae Inj. Dexamethasone 0.15 mg/kg every 6 hour IV for 2 days. It should be started at the same time or shortly before, the first dose of antibiotic (not effective, if given after antibiotics). 4. In patient with papilloedema, altered sensorium, 6th nerve palsy, convulsions or decerbrate posturing Inj. Mannitol 20% 1 g/kg stat followed by 0.5 g/kg every 4-6 hours. 5. Chemoprophylaxis for close contacts of patients with meningococcal meningitis Cap Rifampicin 600 mg twice a day 5 days Or Cap Ciprofloxacin 500 mg single dose Or Tab. Ofloxacine 400 mg single dose Or Inj. Ceftriaxone 250 mg IM single dose. Note: Diagnosis and management of underlying cause, e.g. CSOM is important.


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BACTERIAL MENINGITIS

Introduction

Meningitis is a acute bacterial infection of brain which may affect the brain and lead to irreversible neurological damage and auditory impairment. Bacterial meningitis is a medical emergency ,the treatment is based upon early parenteral antibiotics that penetrates well into cerebrospinal fluid.

Empiric antibiotics is administerd if the pathogen cannot be identified or while waiting for the laboratory reports. Infants, elderly, and immunocompromised patients may show only mild behavioural changes with low-grade fever and little clinical evidence of meningeal inflammation. Meningitis in newborns usually results from an infection of the bloodstream (sepsis). The infection is typically caused by bacteria acquired from the birth canal, most commonly group B streptococci, Escherichia coli, and Listeria monocytogenes.

Older infants and children usually develop infection through contact with respiratory secretions (such as saliva or mucus from the nose) containing causative bacteria. Bacteria that infect older infants and children include Streptococcus pneumoniae and Neisseria meningitidis. Haemophilus influenzae type b was the most common cause of meningitis

Clinical feature:- common-Fever, vomiting, lethargy, irritable, high-pitched cry, refusal to feed, headache, muscle pain, cough and respiratory distress, otitis media.

Less common/non specific - chill & rigor, pain abdomen,sore throat, coryza

More specific sign-Bulging fontanelle, stiff neck, altered mental status, non blanching rash, signs of shock, like cold hand and feet, hypotension, unusual skin colour, capillary refill time more than 2 second , Kernig’s sign,
Brudzinski’s sign, Focal neurological deficit including cranial nerve involvement and abnormal pupil, Seizures. Neck stiffness, bulging fontanelle, high-pitched cry are often absent in infants with bacterial meningitis. High possibility of meningococcal meningitis in patients with signs of shock with petechial rash.

**Signs of shock**
- Capillary refill time more than 2 seconds
- Unusual skin colour
- Tachycardia and/or hypotension
- Respiratory symptoms or breathing difficulty
- Cold hands/feet
- Toxic/moribund state
- Altered mental state/decreased conscious level
- Poor urine output

**Diagnostic criteria**

<table>
<thead>
<tr>
<th>Components</th>
<th>Normal Newborn</th>
<th>Normal Children</th>
<th>Bacterial Meningitis</th>
<th>Viral Meningitis</th>
</tr>
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<tbody>
<tr>
<td>Glucose (mg/dL)</td>
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<td>Protein (mg/dL)</td>
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<td>50-100</td>
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<td>Leukocytes/mL</td>
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<td>&gt;1,000</td>
<td>100-500</td>
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<td>Neutrophils (%)</td>
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<td>&gt;50</td>
<td>&lt;40</td>
</tr>
<tr>
<td>Erythrocytes/mL</td>
<td>0-2</td>
<td>0-2</td>
<td>0-10</td>
<td>0-2</td>
</tr>
</tbody>
</table>

Rational investigation—full blood count, C-reactive protein (CRP), CSF examination.

CSF examination should include white blood cell count and examination, total protein and glucose concentrations, Gram stain and microbiological culture. A corresponding laboratory-determined blood glucose concentration should be measured. In children and young people with suspected meningitis or suspected meningococcal disease, perform a lumbar puncture unless any of the **following contraindications are present:**

- signs suggesting raised intracranial pressure
  - reduced or fluctuating level of consciousness (Glasgow Coma Scale score less than 9 or a drop of 3 or more)
  - relative bradycardia and hypertension
  - focal neurological signs
  - abnormal posture or posturing
  - unequal, dilated or poorly responsive pupils
  - papilloedema
  - abnormal ‘doll’s eye’ movements
- shock
- extensive or spreading purpura
- after convulsions until stabilised
- coagulation abnormalities
  - coagulation results (if obtained) outside the normal range
Platelet count below 100 x 10^9/litre
- receiving anticoagulant therapy
- local superficial infection at the lumbar puncture site
- respiratory insufficiency (lumbar puncture is considered to have a high risk of precipitating respiratory failure in the presence of respiratory insufficiency).

**Perform a repeat lumbar puncture in neonates with:**
- persistent or re-emergent fever
- deterioration in clinical condition
- new clinical findings (especially neurological findings) or
- persistently abnormal inflammatory markers

**Do not perform a repeat lumbar puncture in neonates:**
- who are receiving the antibiotic treatment appropriate to the causative organism and are making a good clinical recovery
- before stopping antibiotic therapy if they are clinically well.

In children and young people with a reduced or fluctuating level of consciousness (Glasgow Coma Scale score less than 9 or a drop of 3 or more) or with focal neurological signs, perform a CT scan to detect alternative intracranial pathology. Do not delay treatment to undertake a CT scan. Clinically stabilise children and young people before CT scanning. If a CT scan has been performed, do not perform a lumbar puncture if the CT scan shows radiological evidence of raised intracranial pressure.

**Nondrug treatment**—Hospitalize in a quiet place with no bright lights preferably in ICU. Maintain vitals, endotracheal intubation in patient with poor respiratory effort. Elevation of head to 30° and hyperventilation, if evidence of raised intracranial pressure.

In children and young people with suspected bacterial meningitis or meningococcal septicaemia, undertake and record physiological observations of heart rate, respiratory rate, oxygen saturations, blood pressure, temperature, perfusion (capillary refill) and neurological assessment (for example the Alert, Voice, Pain, Unresponsive [AVPU] scale) at least hourly.

**Drug treatment**— Patients must be treated in a facility where emergencies can be managed and nursing and medical staff are experienced in caring for critically ill patients. Accordingly, they may require a transfer to a pediatric hospital or large general hospital. Depending on the child’s condition, admission to a pediatric intensive care unit (ICU) may be warranted. If the child is critically ill or experiencing a seizure, immediate stabilization and support are necessary. If the child is hemodynamically stable, intravenous (IV) fluids should be administered at maintenance, Patients who present with dehydration should be rehydrated and should not undergo fluid restriction. Seizures should be treated promptly and should be expected at any time during the initial management.

*Age*

**Younger than 1 month:**
Commonly observed pathogens include group B Streptococcus (GBS) (cases lack characteristic stiff neck of more typical bacterial meningitis), Escherichia coli, Listeria monocytogenes, and Klebsiella species.

- Ampicillin 100 mg/kg plus cefotaxime 50 mg/kg q6h or
- Ampicillin 100 mg/kg plus an aminoglycoside (gentamicin 2.5 mg/kg or tobramycin 2.5 mg/kg) q8h

**One to 23 months:**

Commonly observed pathogens include Streptococcus pneumoniae, Neisseria meningitidis (rapidly evolving skin rash indicative of infection with Meningococcus species; immediately begin a regimen of benzyl penicillin, ceftriaxone, or cefotaxime), GBS, Haemophilus influenzae type b, and E coli.

- Vancomycin 15 mg/kg q6h plus a third-generation cephalosporin (ceftriaxone 75-100 mg/kg q12-24h or cefotaxime 75-100 mg/kg q6-8h

**More than 2 years**

Vancomycin 15 mg/kg q6h plus ceftriaxone 75-100 mg/kg q12-24h or cefotaxime 75-100 mg/kg q6-8h

**Patient education:** Before discharging children from hospital, consider their requirements for follow-up, taking into account potential sensory, neurological, psychosocial, orthopaedic, cutaneous and renal morbidities, discuss potential long-term effects of their condition and likely patterns of recovery with the child and their parents and provide them with opportunities to discuss issues and ask questions.

**Referral criteria:** signs of shock (stabilize before referral), respiratory embarrassment, any complication

**Complication:**

1. Subdural effusion :-20-30% of infants with meningitis, Commonly with H. influenza type b & pneumococcal meningitis, Drain only with neurological symptoms from mass effect
2. Subdural empyema – drainage & prolonged antibiotics
3. Hearing loss
4. SIADH – very cautious with fluid restriction because cerebral vascular autoregulation is compromised in meningitis
4. Loss of cognitive functions

**PREVENTION:**

Hib vaccine for H influenzae, pneumococcal conjugate vaccine for pneumococcus and meningococcal conjugate vaccine for meningococcus

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ACUTE ENCEPHALITIC SYNDROME IN CHILDREN

Definition:
Clinically, a case of AES is defined as a person of any age, at any time of year with the acute onset of fever and a change in mental status (including symptoms such as confusion, disorientation, coma, or inability to talk) AND/OR new onset of seizures (excluding simple febrile seizures).

Lab Diagnosis –
A. Investigations
i. Complete blood counts
ii. Peripheral blood smear-Malarial parasite
iii. Blood glucose, Electrolytes
iv. CSF and Blood for serology by IgM ELISA/ virus isolation, CSF is preferred since by the time patient presents with CNS manifestations the level of viremia in blood has decreased and there is cross reaction with other flaviviruses.

B. Specimen Collection
Blood(serum) and CSF specimen are to be collected.

Imaging
Unnecessary in emergency department.

MRI can be done to see specific abnormalities in specific diseases.

Treatment mainly supportive and empirical antiviral ,anti malarial and anti meningitic treatment should be started if any specific etiology is found during course , treatment should be modified accordingly.

The treatment of the patients may require, as follow :
1. Management of Airways and Breathing.
3. Control of Convulsion and Intracranial pressure
4. Control of Temperature
5. Fluid and Electrolytes and Calories/ Nutrition
6. General management
7. Specific treatment of any for treatable cause
8. Investigations, Samples Collection & Transportation
9. Reporting of case
10. Rehabilitation.

MANAGEMENT OF AIRWAY AND BREATHING

- Assessment of Airway and Breathing
- Clear Airways: No oral feed, Nurse in semi-prone and prone position
- Obstructed breathing / Severe respiratory distress
- Give Oxygen if needed
  - Clear secretions from mouth: Wiping oral cavity, Suction of mouth, turning head on one side, Give Oxygen
  - Ventilate with Bag and Mask / Endo Tracheal Tube if breathing is laboured.
  - Refer the case to tertiary care centre for Ventilatory support if need

MANAGEMENT OF CIRCULATION

- Establish IV line. Look for sign and symptoms of shock:
  - Capillary refill > 3 secs (pediatric patient)
  - Cold extremities
  - Weak and rapid pulse
- Assess pediatric patient for dehydration
- No dehydration
  - Symptomatic management
  - Look for signs of referral
  - 2/3rd of maintenance fluid by Intravenous route
- Grade dehydration as some/ severe dehydration
- Severe dehydration:
  - IV fluid Ringer lactate/ Normal Saline as per WHO guidelines
  - Some dehydration
  - IV fluid – Ringer Lactate/ Normal saline
- Shock present: IV fluid Ringer Lactate 20 ml/kg/hr
  - (Repeat if shock Persists)
- Ringer Lactate – 20 ml/kg, if shock improves, child is euvoletic, give maintenance fluid
- Shock Persists – Inotrope Dopamine drip in maintenance fluid 5 mcg/kg/minute, then again increase Dopamine upto 20mcg/kg/minute and similarly Dobutamine start with 5mcg/kg/minute & increase upto 20 mcg/kg/minute (Till BP stabilizes)
- Reassess
- Improvement: Continue maintenance IV fluid
  - No improvement: Refer to higher centre
MANAGEMENT OF INCREASED INTRACRANIAL PRESSURE (Tension) (only after correction of Dehydration)
1. Mannitol 20% I/V 5 ml/kg in 1/2 hrs as 1st dose than 2.5 ml/kg at 6 hrs. intervals up to 48 hours (8 doses).
2. Injection Lasix I/V - 1 mg/kg upto 40 mg can be given.
3. Glycerol solution : Oral 0.5 ml/kg mix with fruit juice can be given by nasogastric tube - 3 times a day
4. Steroids - are not indicated in viral encephalitis including JE.
5. 3% Hypertonic saline can be used.

Empirical treatment of AES:
Should cover anti viral,anti malarial and anti bacterial meningitis.

Herpes Acyclovir 10 mg/kg/dose, slowly over a period of one hour 8 hourly X 21 days.

Malaria – Artesunate 2.4 mg/kg( 3mg/kg< 20 kg ) stat then after 12 hours then once daily for next 6 days . I/V Quinine . 20 mg/kg in 5% Dextrose slowly over a period of 1hr then 10mg/kg 8 hourly for 7 days . Monitor Blood Sugar and Blood Pressure.

Meningitis (Pyogenic) -
Start with inj. Ampicillin 400 mg kg 6 hourly upto 12gm/day 
+ 
Inj. Ceftrioxone 100-150mg/kg as stat dose than in two dived doses 12 hourly

Prognosis:-
Depends upon supportive management and etiology.

Patient Education –
Early hospitalization if any sign and symptom of altered sensorium along with fever.
Use mosquito net.
FEBRILE SEIZURES IN CHILDREN

It is a brief tonic-clonic generalized self-limited seizure of childhood seen in an otherwise normal child with high fever. Febrile seizures are seizures that occur between the age of 6 and 60 mo with a temperature of 38°C (100.4°F) or higher, that are not the result of central nervous system infection or any metabolic imbalance, and that occur in the absence of a history of prior afebrile seizures.

Diagnostic Features:
A simple febrile seizure is a primary generalized, usually tonic–clonic, attack associated with fever, lasting for a maximum of 15 min, and not recurrent within a 24-hr period.
A complex febrile seizure is more prolonged (>15 min), is focal, and/or reoccurs within 24 hr.

Laboratory Test:
Tests are directed to elucidate the cause of fever for proper diagnosis and therapy. If doubt exists for possibility of meningitis a Lumbar Puncture and study of C.S.F. is indicated.

Non Drug Treatment:
- Patent and clean air way to be maintained.
- Secretion from the mouth should be wiped out.
- Semi-prone lateral position should be advised to prevent secretions entering to trachea. Sponging with tap water should be continued in open space or under fan.
- Child should not be covered with tight clothing.

Drug Treatment:
If the seizure lasts for longer than 5 min, acute treatment with diazepam, lorazepam, or midazolam is needed. Antipyretics can decrease the discomfort of the child but do not reduce the risk of having a recurrent febrile seizure.
Most convulsions stop before child is brought under health facility. If a child is received with convulsion - Diazepam 0.2 mg / Kg body weight by slow IV route not exceeding 2 mg / min is given. If facilities for IV administration is not available 0.5 mg / Kg of Diazepam may be given per rectally.
- Oxygen Inhalation by mask to avoid Hypoxia.
- Because of the benign nature of the disease long term anticonvulsant therapy or short term prophylaxis is not recommended. Oral diazepam during acute febrile illness for 2 to 3 days in a dose of 0.3 mg / Kg 8 hourly will be sufficient to reduce the risk of convulsion.
Iron deficiency is associated with an increased risk of febrile seizures, give iron therapy 2-3 mg/kg in iron def anemia.

Patient Education:
- The benign nature of the disease should be explained to the parents and they must learn the administration of intra-rectal Diazepam at home.
- Immunization should not be withheld. After DPT injection Paracetamol 15 mg / Kg per dose should be given 6 hourly. After meals immunization Paracetamol with above doses can be started 3 to 4 days after to reduce pyrexia.
Referral Criteria:

- Patients not responding to treatment or repeated occurrence of seizure may be referred to referral centers.

PEARLS AND PITFALLS

Pearls:
- Determination if the child has serious bacterial infection including meningitis is critical.
- Consider further lab/imaging evaluation with any child who presents with an atypical history or is less than 1 year.
- Seizure without focal or other abnormal neurologic findings is not consistent with or suggestive of meningitis.
- Caregiver education and reassurance is important to ensure proper care of the child and to assuage fears about the repercussions of febrile seizures.

Pitfalls:
- Failure to diagnose cause of fever that would warrant intervention, such as urinary tract infection or otitis media.
- Not recognizing febrile status epilepticus and treating for status epilepticus.
- Failure to allow 5–10 min of seizure activity before giving a benzodiazepine: æ% Most febrile seizures cease spontaneously. æ% Benzodiazepines prolong sedation after postictal period, making neurologic evaluation difficult.

U.T.I. IN CHILDREN

Urinary tract infections (UTIs) occur in 1% of boys and 1-3% of girls. The prevalence of UTIs varies with age. During the 1st yr of life, the male : female ratio is 2.8-5.4 : 1. Beyond 1-2 yr, there is a female preponderance, with a male : female ratio of 1:10. In boys, most UTIs occur during the 1st yr of life; UTIs are much more common in uncircumcised boys, especially in the 1st yr of life. In girls, the first UTI usually occurs by the age of 5 yr, with peaks during infancy and toilet training.

Infection of Urinary tract by microorganism. UTIs are caused primarily by colonic bacteria. In girls, 75-90% of all infections are caused by *Escherichia coli* (see Chapter 200), followed by *Klebsiella* spp. and *Proteus* spp.

UTI may be –
- a) Lower tract infection – Cystitis(Usually afbrile UTI)
- b) Upper tract infection – Acuta Pyelonephritis.(febrile UTI)

Types –

1. *Cystitis* –

Usually report with dysuria, Frequency, Urgency, Supra pabic pai

2. *A.C. Pyelonephrites* –

- *Fever*, shaking chill, nausca, vomiting.
- Symptom of cystitis may or may not be there.
Clinical features:

**Infants:**
- Nonspecific symptoms, most often have fever alone
- Can have vomiting, irritability, lethargy, poor feeding
- Rarely have failure to thrive or jaundice
- Often no physical findings or fever alone
- Less commonly: Dehydration, abdominal pain or distention, poor weight gain, malodorous urine, jaundice

**Older children:**
- Lower tract infection symptoms typically include urgency, frequency, dysuria, hematuria, hesitancy, suprapubic pain, and malodorous urine.
- Upper tract infection symptoms typically include nausea, vomiting, fever, chills, and flank pain.
- May have a history of constipation – May also present with incontinence or secondary enuresis
- Lower tract: Suprapubic tenderness
- Upper tract: Fever, costovertebral angle tenderness

**Initial Lab Tests:**
- With acute renal infection, leukocytosis, neutrophilia, and elevated serum erythrocyte sedimentation rate, procalcitonin, and C-reactive protein are common.
- Include urinalysis (UA)
- Urine culture (Prompt plating of the urine sample for culture is important within 60 mins. Refrigeration is a reliable method of storing the urine until it can be cultured.
- Bladder catheterization or suprapubic aspirate in young children
- Midstream clean catch if possible
- A specimen should not be obtained by applying a bag to the perineum, as this technique is associated with an unacceptably high rate of contamination. Cultures take 48-72 hr to grow.

The following are suggestive of UTI:
- Conventional UA: >5 WBC/high-power field centrifuged urine
- Enhanced UA (combines microscopy on uncentrifuged urine with Gram stain): >10 WBC/mm3 and/or positive Gram stain

The following are diagnostic of UTI
- If the culture shows >50,000 colonies of a single pathogen (suprapubic or catheter sample), or if there are 10,000 colonies and the child is symptomatic, the child is considered to have a UTI.
- In a bag sample, if the urinalysis result is positive, the patient is symptomatic, and there is a single organism cultured with a colony count >100,000, there is a presumed UTI.

**Imaging**
- Ultrasonography if there is concern for obstructive uropathy or congenital or iatrogenic urinary tract anomalies. Other imaging required are VCUG, DMSA scan in high-risk patient.

**Serile Pyuria** – May indicate infection with Chlamydia, U. Urealyticum, M. Tussenurtoer & fungi, calculi, anatomic abnormal.
**Treatment**

**Admission Criteria:**
- Neonates require hospital admission.
- Ill appearing, dehydrated, unable to tolerate PO (10), or there is concern for compliance and follow-up
- Critical care admission criteria: – Urosepsis or unstable vital signs

**Empiric inpatient therapy for febrile UTI**—IV therapy with a 3rd-generation cephalosporin: Cefotaxime (40 mg/kg/ dose IV q8h); ceftriaxone (50–75 mg/kg IV/IM per day for 10-14 days

**Empiric outpatient therapy:** Cefixime (8 mg/kg/day divided b.i.d.); cefdinir (14 mg/kg PO per day); amoxicillin/clavulanate or amoxicillin (45 mg/kg/day divided b.i.d.); cephalaxin (50 mg/kg/day divided b.i.d.–t.i.d.); co-trimoxazole (5 mg/kg/day divided b.i.d.): for 7-10 days.

**Terminology**
- Recurrent Infection – is classified as same strain or different strain
  - (< 2 week) & early or late (> 2 week)
- Recurrence > 2 weeks after cessation of The infection – reinfection – new strain.
- Recurrence > 2 weeks with same strain – Relapse

**Cause** – most common cause recurrent UTI in children is congenital urinary tract abnormalities like posterior urethral valve, ureterocaly, vescico ureteral reflux.

**Treatment:**- Prophylactic antibiotic therapy and surgery.

**Patient Education** –
- To drink plenty of oral fluid
- Not to control the urge to pass urine. To empty the bladder completely & very often.

**PEARLS AND PITFALLS**:-
- Screening tests are imperfect. Always send a culture if UTI is suspected. Contaminated cultures are difficult to interpret. Obtain urine by catheterization or suprapubic tap. Consider obtaining a urine specimen in febrile children with other sources of fever (bronchiolitis, upper respiratory infection, otitis media, gastroenteritis) since they may have concurrent UTIs.

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**POSTSTREPTOCOCCAL ACUTE GLOMERULAR NEPHRITIS**

**Definition:**
- Pathologically: it is an inflammation of glomeruli
- Clinically: nephritis is a triad of hematuria, hypertension and azotemia
  - Usually, these patient present with smoggy urine, dark urine,
    decrease amount of urine, headache as symptom of HTN, edema as symptom of azotemia in GN.
  - Hematuria (may be gross or microscopic)

**Most common causes**: Postinfectious glomerulonephritis (post streptococcal)

**Presentation**: Hematuria:
- May be sole symptom
- “Cola-colored” urine: 30–50% of AGN
- Gross hematuria: 50% of IgA nephropathy. Pink or red urine and presence of blood clots indicate extraglomerular source of bleeding.
Edema Reduced urine output

Presentation patterns:
- PSAGN: History of pharyngitis 1–3 wk or history of pharyngitis, impetigo, or cellulitis up to 6 wk prior to onset of hematuria

Abnormal vital signs:
- HTN
- Tachycardia
- Tachypnea

Suggestive physical exam findings:
- Change in mental status
- Papilledema
- Gallop, rales, hepatomegaly
- Edema: Pitting, generalized or localized

**Lab Diagnosis**

Urinalysis:
- Presence of RBCs
- Absence of blood clots
- Presence of RBC cast is pathognomonic for glomerular disease.

- Elevated BUN and Cr in renal insufficiency
- Low Serum C3 (low C4 is seen only in SLE GN)
- ASO titers peak 10–14 days post-infection

**Imaging**

Unnecessary in emergency department

Consider chest radiograph in following clinical scenarios:
- HTN: Cardiomegaly
- CHF: Cardiomegaly, pulmonary edema

**Treatment**

**Non Drug Treatment**

Hospitalise every pt of AGN with hypertension
Emergently stabilize patients presenting with CHF and/or malignant HTN.
Restrict sodium intake.
Restrict fluid intake in ill patients only

**Drug treatment**: Management of HTN:

1st line of treatment for BOTH fluid overload & HTN is **DIURETIC** (lasix). If BP gets better, fair enough. If doesn’t, 2nd line of treatment is usually Ca-channel blocker.

In acute phase: - Furesamide (Lasix) 1-2 mg / kg 8-6 hrly if not controlled or rising BP add calcium channel blocker(nifedipine) 0.25 -0.5 mg /kg once or twice daily.

**Prognosis**: Complete recovery occurs in >95% of children with APSGN. Recurrences are extremely rare. Mortality in the acute stage can be avoided by appropriate management of acute renal failure, cardiac failure, and hypertension.

**Patient Education**

Avoid over crowding.
Keep yourself and your baby hyegenic.
Consult doctor in any case decrease urination.
RICKETS IN CHILDREN

Normal bone growth and mineralization require adequate calcium and phosphate, the two major constituents of the crystalline component of bone. Rickets is due deficient mineralisation of growth plate. Mineralization defects are classified according to the predominant mineral deficiency. **Calcipenic rickets** is caused by calcium deficiency, which usually is due to insufficient intake or metabolism of vitamin D, and in some cases insufficient intake or absorption of calcium in the setting of normal vitamin D levels. **Phosphopenic rickets** usually is caused by renal phosphate wasting.

**Diagnostic criteria:**

**Clinical manifestations:**
- Delayed closure of the fontanelles.
- Parietal and frontal bossing.
- Craniotabes (soft skull bones).
- Enlargement of the costochondral junction visible as beading (rachitic rosary) along the anterolateral aspects of the chest.
- Formation of Harrison sulcus (or groove) at the lower margin of the thorax caused by the muscular pull of the diaphragmatic attachments to the lower ribs.
- Widening of the wrist and bowing of the distal radius and ulna.
- Delayed achievement of motor milestones.

**Radiographic findings:**
- Widening of the epiphyseal plate.
- Disorganization of the growth plate becomes more apparent as cupping, splaying, formation of cortical spurs, and stippling with progression of disease.

**Laboratory findings:**
- Increased serum alkaline phosphatase.
- Calcipenic rickets is often but not always associated with low serum calcium levels and high PTH, while phosphopenic rickets is characterized by low serum levels of phosphorus and normal PTH concentration.

**Treatment:**
- Preterm neonate: Vitamin D 1000 IU oral daily and elemental Calcium 70-80 mg/Kg/day for minimum 3 months.
- Infant upto 1 year: Vitamin D 2000 IU oral daily and elemental Calcium 500-800 mg/day for minimum 3 months.
- 1-18 years: Vitamin D 3000-6000 IU oral daily and elemental Calcium 500-800 mg/day for minimum 3 months.
- Or >3months -18 years: Vitamin D 60,000 IU weekly for 6 weeks and and elemental Calcium 500-800 mg/day.

**Patient education:**
- No special oil is required for massage.
- Most of the deformities improve in the due course of time.

**Referral criteria:**
- No response to therapy after 4 weeks, refer to a higher centre for evaluation of non-nutritional rickets.

**References:**
NEPHROTIC SYNDROME (NS) IN CHILDREN

Nephrotic syndrome is an important chronic renal disease in children. Children with NS are classified based upon whether or not there are signs of systemic disease as:

- **Primary/Idiopathic nephrotic syndrome**: absence of an identifiable systemic disease.
- **Secondary nephrotic syndrome**: presence of an identifiable systemic disease.
- **Congenital and infantile nephrotic syndrome**: which occur in children less than one year of age.

Idiopathic nephrotic syndrome is the most common form of childhood nephrotic syndrome, representing more than 90 percent of cases between 1 and 10 years of age and majority of patients respond to steroid therapy.

**Diagnostic criteria:**

The diagnosis of childhood nephrotic syndrome is generally based upon the characteristic clinical and laboratory findings of four criteria, but the first two are used diagnostically because the last two may not be seen in all patients:

- Nephrotic range proteinuria: Urinary protein excretion greater than 50 mg/kg per day or spot urine protein/creatinine ratio >2.
- Hypoalbuminemia: Serum albumin concentration less than 2.5 g/dL.
- Hyperlipidemia: Serum cholesterol >200 mg/dL.
- Edema

**Investigation:**

A detailed evaluation is necessary before starting treatment with corticosteroids. Investigations recommended at the initial episode include:

- Urinalysis
- Complete blood count
- Serum albumin, cholesterol, urea and creatinine.
- Estimation of blood levels of antistreptolysin O and C3 is required in patients with gross or persistent microscopic hematuria.
- Appropriate tests are performed, if necessary, for associated conditions (e.g., chest X-ray and tuberculin test, hepatitis B surface antigen, antinuclear antibodies).
- Urine culture is not necessary unless the patient has clinical features suggestive of a urinary tract infection.

**Non-drug treatment:**

- Adequate protein (1.5-2 g/Kg) and calories is recommended.
- Reduced salt (1-2 g/day intake, if persistent oedema.

**Drug treatment:**

*Treatment of first episode:*

Prednisolone 2 mg/kg daily for 6 weeks, followed by 1.5 mg/kg on alternate days for 6 weeks.
Treatment of subsequent relapse:
Prednisolone 2 mg/kg daily until remission, then in a single morning dose of 1.5 mg/kg on alternate days for 4 weeks, and then discontinued.

Supportive treatment:
- Treatment of oedema: Oral frusemide 1-3 mg/kg/day. May add spironolactone 2-4 mg/kg/day
- Antihypertensive drug: if BP > 95th cile.
- The use of antacids or histaminereceptor antagonists (e.g., ranitidine) is not necessary, unless there are symptoms of upper gastrointestinal discomfort.
- Long-term calcium supplementation (calcium carbonate, 250-500 mg) is unnecessary if the patient receives more than 3 monthstreatment with prednisolone

Patient education:
- Maintain a diary showing results of urine protein examination, medications received and intercurrent infections.
- Encourage normal activity and school attendance.
- Since infections are an important cause of morbidity, patients should receive appropriate immunization.

Indications for referral to a Paediatric Nephrologist:
- Onset < 1 year of age; family history of nephrotic syndrome.
- Nephrotic syndrome with hypertension, gross/persistent microscopic hematuria, impaired renal function, or extrarenal features (e.g., arthritis, serositis, rash).
- Complications: refractory edema, thrombosis, severe infections, steroid toxicity.
- Steroid resistance NS (SRNS): absence of remission despite therapy with daily prednisolone at a dose of 2 mg/kg per day for 4 weeks.
- Frequently relapsing NS (FRNS): ≥ 2 relapses in initial six months or > 3 relapses in any twelve months
- Steroid dependent NS (SDNS): Two consecutive relapses when on alternate day steroids or within 14 days of its discontinuation.

References:

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PICA

Definition
Pica involves the persistent eating of nonnutritive, nonfood substances (e.g., paper, soap, plaster, charcoal, clay, wool, ashes, paint, earth) over a period of at least 1 mo. The eating behavior is inappropriate to the developmental level (e.g., the normal mouthing and tasting of objects in infants and toddlers) and, therefore, a minimum age of 2 yr is suggested.

Treatment
- Pica below 2 years does not need any intervention.
- Children with pica are at an increased risk of lead poisoning, iron deficiency, bezoars and parasitic infections. They should be investigated for these problems and if present, treated suitably.
- Education, guidance and counselling of the family.
- The child has to be kept occupied in other tasks and provided with environmental stimulation.

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BREATH HOLDING SPELL

Definition
Paroxysmal self limiting events occurring in up to 5% of healthy children and are rare prior to 6 months of age, peak at about 2 years of age and abate by 5 years of age.
- Child starts crying (precipitated by an upsetting event such as anger, fear or injury) and then holds breath in expiration followed by a colour change (blue or pale)
- Spell may resolve spontaneously or the child may lose consciousness and may have convulsions. Normal breathing and alterness are resumed within a minute.

Subtypes of breath holding spells include cyanotic, pallid, or mixed episodes. Cyanotic are the dominant type and may include a brief loss of consciousness and a very brief tonic-clonic seizure. Pallid spells may be similar to vasovagal related syncopal events in older children and initiated from similar stimuli

Treatment
Reassurance. Explain that attacks are harmless and always abort by itself.

Immediate measures
1. Prevent injury during the episode. Help the child to floor and have him lie flat.
2. If loss of consciousness occurs, place on the side to protect against aspiration.
3. Maintain patent oral airway but do not start CPR.
4. Do not shake the baby, splash water or put anything in the mouth.
   Iron deficiency with or without anemia may be present and some children with breath-holding spells respond to iron therapy

Referral:
When breath holding does not respond to the parent’s coaching or is accompanied by head banging or high levels of aggression, referral for a mental health evaluation is indicated.

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STANDARD TREATMENT GUIDELINES

SCRUB TYPHUS

Introduction

Scrub typhus is an acute febrile illness caused by Orientia tsutsugamushi, an obligate intracellular gram negative bacterium transmitted by Trombiculid mites and humans are accidentally infected when exposed during occupational and recreational activities in the habitat of mites such as grasses and dirty home environments. Early reports of scrub typhus in Indian subcontinent emerged from Assam region during world wars. Since then, several papers have reported cases in adults and children from various parts of India indicating that the disease is ubiquitous. It has a seasonal pattern; most cases are reported during rain and post monsoon months typically between July and November. The cases are seen from rural and urban backward areas which are the natural habitat of the vector.

Diagnostic Criteria

The incubation period range from 6-21 days. The illness severity can vary from an undifferentiated febrile illness to severe systemic disease with multi-organ failure. The initial manifestations include fever, cough, vomiting, headache and myalgia. Examination findings include edema, hepatosplenomegaly, lymphadenopathy and typical eschar. Eschar is seen in 4-68% and if found is a clue to diagnosis. It is 5–20 mm in diameter resembling a cigarette burn, formed at the bite site (mostly inguinal, genitalia, buttock and axillary areas), progressing initially from a papule to vesicle and finally an ulcer with a black necrosis.

Diagnosis in the first week may be missed due to the presentation as undifferentiated fever, however systemic involvement with multi-organ involvement is seen from the second week. Complications seen are respiratory distressand ARDS (30-75%), septic shock and myocarditis(10-34%), meningitis and encephalitis (11-41%), acute kidney injury (8-40%) , hepatitis( increased transaminases in 64-100% and hypoalbuminemia in 54-100%) and thrombocytopenia(60-100%).

Rational Investigations

CBC: Thrombocytopenia with platelet count 50x10^3 /L seen in 50% cases. Mild anemia and leucocytosis.

Biochemical: Hyponatremia, elevated transaminases, hypoalbuminemia

Laboratory confirmation: Weil-Felix is the nonspecific test, done widely in resource limited settings has a poor sensitivity(50%). A high OXK titre(>1:320) is reported to predict scrub typhus. Indirect immunofluorescent antibody (IFA) test is considered as gold standard. IgM Elisa is the most practical useful test for confirmation (97% sensitive and 99% specific).

Non-Drug Treatment:

Initial management focuses on stabilisation of airway, breathing and circulation. Monitoring of complication is important for early recognition and further management and timely referral.

Drug Treatment

If a definitive diagnosis is not possible at the outset, it is recommended to treat children empirically for scrub typhus till serological confirmation is available.

Early treatment with doxycycline (5mg/kg/day in 2 divided doses, i.v. or oral) shortens the disease course and reduces mortality. Duration of therapy is variable; 5 days or 3 days after defervescence in uncomplicated cases. In severe scrub typhus with organ failure it may be given for 7-14 days. Azithromycin (10mg/kg/day for 5 days) is an effective alternative.
Patient Education:

Children are exposed to the mites during occupational and recreational activities in the habitat of mites such as grasses and dirty home environments. Children present often in post-monsoon season with undifferentiated fever and various degrees of organ involvement that progress to fatal multi organ failure if untreated. Early treatment shortens the disease course and reduces mortality.

Referral Criteria:
1) Children with respiratory distress with hypoxia and features of ARDS
2) Severe encephalopathy with GCS< 8 or uncontrolled seizure
3) Septic shock and myocardial dysfunction.

DENGUE

Introduction
Dengue fever manifests as a wide spectrum of clinical illness ranging from mild undifferentiated fever to severe hemorrhagic illness requiring intensive care. Capillary leakage leading to third space and clinically apparent and occult bleeding contribute to hypoperfusion and shock. Dengue is caused by dengue virus (DENV), which is an arthropod borne single stranded RNA virus from genus Flavivirus, family Flaviviridae. It has four distinct antigenically different serotypes, DENV 1, 2, 3 and 4. DENV is transmitted principally by Aedesaegypti, which is a day biting mosquito.

Diagnostic Criteria
Intrinsic incubation period is usually 4-10 days (human bite by infected mosquito to appearance of symptoms). New WHO 2009 classification is summarised below.

Classical dengue is a dynamic and unpredictable disease. It has three phases of illness: febrile phase, critical phase and recovery phase.

Figure 1.4 Suggested dengue case classification and levels of severity

Febrile Phase: It lasts approximately 3-7 days and characterised by high grade fever. Other common symptoms are conjunctival injection, coryza, myalgia, headache, abdominal pain and retro-orbital pain. Body aches and joint pain are common and severe, hence termed breakbone fever. Rash is seen in approximately one fourth of patients which is flat generalised blanchable erythema appearing on day 2-4 of illness. Mild hemorrhagic manifestations can be seen during this period including petechiae and epistaxis. Tourniquet test (done by inflating blood pressure cuff between systolic and diastolic pressure around the arm for 5 minutes, e’10 petechiae/square inch, test is considered positive) is usually positive during this period.

Critical Phase: It lasts approximately 48 hours. Capillary leakage heralds onset of critical phase which is usually associated with improvement in fever. Capillary leak manifests as pleural effusion and ascites. Severe pleural effusion may lead to respiratory distress. Severe intravascular depletion manifests as cool extremities, tachycardia, poor pulses which can progress to hypotensive shock if appropriate volume resuscitation is not provided. Severe bleeding manifestation (due to abnormal hemostasis and thrombocytopenia) can include melena, hematemesis, hematuria and occult bleeds like capsular hematoma of liver.

Recovery phase: It is associated with absorption of leaked fluid which manifest as polyuria, hypertension and sinus bradycardia. Patient may develop fluid overload leading to congestive cardiac failure and pulmonary edema. An erythematous maculo-papular rash with small islands of hypo-pigmentation may appear during this phase distributed over lower extremities and trunk, commonly called “islands of white in sea of red”. Pruritis is common.

Rational Investigations

Initial investigations: Hemogram with HCT, coagulation studies, blood sugar, blood gas, serum electrolytes, blood grouping and cross-matching. Chest X ray should be done in children with respiratory distress.

Monitoring:

i) TLC: normal - early febrile stage
decrease rapidly - disease progression (EARLY SUSPICION)

ii) HCT: Rising HCT – a marker of Plasma leakage (differentiate DF & DHF)
Falling HCT after IV fluid – retrospectively indicate plasma leak
Low HCT- Bleeding (Occult/overt)

iii) TPC: normal - early febrile stage
decrease rapidly - disease progression (correlate with severity)

iv) LFT: AST>ALT elevation more in DHF compared to DF.

Definitive Diagnosis:

NS1 ELISA provides results in 6 hours with good sensitivity (67-98%) and specificity (100%). Point of care NS1 test strips are also being utilized with comparable sensitivity and specificity. IgM appears by day 3-5 in primary infection and results are positive in 93-99% sixth day onwards. IgG becomes positive on day 9 of illness which stays positive for months. During secondary infection, IgM level are low but IgG response is very rapid and strong; titres of IgG> 1:1280. Point of care IgM enzyme immunoassay kits are also available.
Non-Drug Treatment

Dengue illness should be suspected in children presenting with acute febrile illness in endemic areas especially during rainy season. Dengue is a self-limiting disease and needs only supportive and symptomatic management. Being a dynamic disease it needs frequent clinical & laboratory monitoring to recognize disease progression i.e. plasma leakage, shock, MODS. During the febrile phase bed rest, oral adequate fluid intake, reduction of fever (tepid sponging and paracetamol), antiemetic is needed. If not admitted parents must be made aware of the warning signs especially from day 3 until afebrile for at-least 48 hr.

Indication for hospitalization (Warning signs) : Abdominal pain or tenderness, persistent vomiting, clinical fluid accumulation (pleural effusion, ascites), mucosal bleeding, restlessness or lethargy, enlarged and tender liver, increase in Hct with rapid decrease in platelet count.

Hemodynamically stable with danger signs: Children with danger signs are started with intravenous crystalloid like normal saline (NS) or Ringer’s lactate (RL) at 5-7 ml/kg/hr for 1-2 hours. Further fluid therapy will depend on clinical improvement and HCT change. If improving clinically and HCT decreases, fluid rate should be decreased to 3-5 ml/hr for 2-4 hours and then 2-3 ml/hr. If worsening clinically and hematocrit increases, fluid rate should be increased to 5-10 ml/hr and child should be rigorously monitored for hemodynamic instability. If clinical worsening and falling hematocrit, suspect bleeding, this can be occult gastrointestinal hemorrhage. Urgent blood product transfusion should be sought. Blood transfusion should only be given in children with suspected/severe bleeding. Platelet transfusions may be considered for children with severe bleeding. FFP may be considered in children with significant coagulopathy.

Children with shock, need of respiratory support, significant bleeding or encephalopathy should be referred for intensive care management.

Drug Treatment:

There is no specific therapy for dengue fever. The mainstay of therapy is supportive and symptomatic.

Patient Education

Predominant dwelling sites for mosquitoes are household water collections and construction sites and need to be taken care of. Mosquitoes control and individual protection from mosquitobites are important. Dengue is rarely fatal and all patients don’t need hospitalization, stable for >48 hr after defervescence means danger period is over. However fatality can be reduced by awareness regarding warning signs and appropriate early management.

Referral Criteria:

1) Shock despite initial fluid management.
2) Suspected myocarditis or cardiac dysfunction requiring inotropic support
3) Fluid overload/ renal failure requiring peritoneal dialysis
4) Seizure and encephalopathy
5) Respiratory failure due to pleural effusion.

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3. PSYCHIATRY
SCHIZOPHRENIA AND ACUTE PSYCHOTIC DISORDER

Schizophrenia is a psychotic disorder, characterised by disturbances in thinking, emotions and perception and disorganised behaviour. The illness tends to be chronic.

Patients may present to a physician when they develop a physical or heluviound problem.

DIAGNOSTIC FEATURES:
- Socially disorganised behaviour (abusive, aggressive, violent, destructive, roaming aimlessly).
- Talking irrelevantly, suspiciousness, fearfulness, thoughts of being harmed or cannoned by some external agencies.
- Laughing, smiling or crying without any obvious reason.
- Hearing voices, muttering or talking to self with imaginary figures.
- Standing or sitting in odd postures for long. Remaining quiet and withdrawn, neglecting personal care and disturbed sleep.
- Symptoms of schizophrenia are often present for a long time varying from a few months to many years. In acute psychotic disorder, duration varies from a few days to weeks.

Treatment

In patients already on treatment from a psychiatrist, the same may he continued. In others the treatment as given below may be started but the patient should preferably be referred to a psychiatrist.

Non-pharmacological
- Psychological support by family and therapist
- Psycho education: Educating the family about nature of illness need of treatment etc.

Pharmacological

Goals of treatment in acutely violent patients
1. Prevent harm
2. Control of disturbing behavior
3. Reduce the severity of psychosis and associated symptoms, viz., agitation, aggression, negative symptoms, and affective symptoms.
4. Determine and address the factors that led to the occurrence of the acute episode.
5. Facilitate a rapid return to the best level of functioning.
6. Develop alliance with the patient and family.
7. Formulate short- and long-term treatment plan.
8. Connect the patients with appropriate aftercare in the community.

In a newly diagnosed case, select medication depending on the following factors: Prior response to treatment; past experience of side effects; side-effect profile of the prospective medication; patients’ preference for a particular medication including route of administration; availability of the medicine locally.

Treatment can be started as below:

Tab. Risperidone 1 mg/day, gradually increased to 2-4 mg/day in 2 divided doses over 2-4 days, which can be further increased depending on tolerability and clinical response (usual therapeutic dose 4-8 mg/day, though most patients are likely to respond at 4 mg/day).
Or

Tab Olanzapine 5 mg/day as a single night-time dose; can be gradually increased up to 20 mg/day over 2-3 weeks depending on response and tolerability. Usual therapeutic dose is 10-20 mg/day
(Caution: Olanzapine has a potential to cause hyperlipidaemia and precipitate diabetes mellitus. Hence, patients require weight monitoring every month and lipid and blood sugar monitoring every 6 months.)

Or

Tab, Quetiapine 25 mg twice a day on the first day, 75-100 mg in two divided doses on days 2-3, 150-200 mg in two divided doses on days 4-5 and 200-400 mg on days 6-7, can increase up to max dose of 900 mg/day over 2-3 weeks depending on the response and tolerability, Slow release (SR) preparations available in strengths of 50, 100, 200, 300 mg; patient can be shifted to SR preparations after 2-3 weeks.

Or

Tab. Haloperidol 5 mg/day, which can be increased up to 10 mg/day (in 2 divided doses) over 1-2 weeks.

Or

Tab. Trifluoperazine 10 mg/day, which can be increased to 15-20 mg/day (in 2-3 divided doses) over 1-2 weeks.

Risperidone and Olanzapine have been associated with weight gain when used for a long period. Patients should be encouraged for lifestyle modification like regular physical exercise and diet control.

In case of acute excitement or violent behaviour, the patient may be given Inj. Haloperidol 5-10 mg IM + Inj. Promethazine chloride 25-50 mg IM Stat.

Or

Inj. Olanzapine 10 mg IM
And / Or
Inj. Lorazepam 2 mg IM.

In case of non-response to medication patient should be considered for Electro Convulsive Therapy (ECT).

In case of chronic schizophrenia the treatment should be lifelong. The injection can be repeated after 8 hours. Antipsychotics may cause mild-to-moderate side effects like sedation, slowness of movements, changes in facial expression and gait, rigidity, excessive salivation, dryness of mouth and constipation. Patient usually develops tolerance to these over a few weeks. If the patient develops extrapyramidal symptoms like tremors, rigidity, slowness of movements, parkinsonian face, sialorrhoea, akathisia add

Tab. Trihexyphenidyl - 2 mg once in morning and once in afternoon (attempts may be made to taper it off after 3 months)

For sleep disturbance, give

Tab. Lorazepam 1-2 mg or clonazepam 0.25-1 mg at bedtime may be given in the initial period (usually for 10-15 days, to be tapered off thereafter).

Follow-up is required weekly initially. Once the symptoms stabilise, frequency of follow-ups may be gradually reduced to once in fortnight to once in 1-3 months. Improvement starts within one week. However, it may take few weeks to months for full response to come. The illness needs long-term treatment, which may go on from one to many years in schizophrenia. Treatment for acute psychotic disorder is usually required for 6-9 months. For patients not compliant to medication and with a history of relapse, Inj. Fluphenazine decanoate 25-50 mg deep IM technique

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in the gluteal region every 15 days or Inj Haloperidol decanoate 50-100 mg deep IM in the gluteal region and repeat every month.

Typical antipsychotic depot injections (Risperidone and Paliperidone) are available in the market but are very expensive and consider for a select group of patients. Refer treatment-resistant cases to a tertiary care centre for clozapine therapy to be prescribed and monitored by a psychiatrist since it requires vital parameter monitoring along with blood count monitoring on a regular basis.

**Patient/Family education**

- Both the patient and the caregivers to be educated that schizophrenia is a psychiatric illness, which can be effectively treated by medicines. Family should be advised to be supportive and not to criticise the patient. Try to maintain low expressed emotion family environment.
- Sensitize the patient to the common side effects. If the patient develops spasm of a part of body like neck or extremities or high-grade fever or alterations in sensorium, immediately contact the treating doctor or the psychiatrist.
- Antipsychotics are often associated with weight gain. Encourage the patient for lifestyle modification like avoidance of fats. Encourage high-fibre diet and regular physical activity.
- Treatment should not be stopped abruptly without the advice of the treating psychiatrist.

**References**


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**BIPOLAR AFFECTIVE DISORDER**

The illness is characterized by episodes of mania and depression or mania alone with intervening periods of normalcy. Patients may present to a physician in a new episode or when they develop a physical or behavioural problem, while on treatment with a psychiatrist.

**SALIENT FEATURES**

Episodes of mania are characterized by:

- Elevated, expansive or irritable mood, inflated self-esteem, or grandiosity, decreased need for sleep, over talkativeness, over activity, increased sociability, interfering behaviour, and excessive involvement in pleasurable activities that have a potential of harmful consequences (buying sprees; sexual indiscretions).
Symptoms should be present for a minimum duration of one week for a diagnosis of mania to be made (for details about depressive episodes, see section on Depression).

Treatment

Treatment is for the current episode and for prophylaxis since the episodes tend to recur. Prophylaxis is usually indicated if there are more than 2-3 episodes in the previous 4-5 years. In patients of bipolar affective disorder already on treatment, the same may be continued. The patient should preferably be referred to a psychiatrist.

In a newly diagnosed case, treatment can be started as below:

Tab. Risperidone 1 mg/day gradually increased to 2-4 mg/day in 2 divided doses over 2-4 days, which can be further increased depending on tolerability and clinical response (usual therapeutic dose 4-8 mg/day though most patients are likely to respond at 4 mg/day),

Or

Tab Olanzapine 5 mg/day as a single night-time dose; can be gradually increased up to 20 mg/day over 2-3 weeks depending on response and tolerability. Usual therapeutic dose is 10-20 mg/day

(Caution: Olanzapine has a potential to cause hyperlipidaemia and precipitate diabetes mellitus. Patients on olanzapine may require lipid and blood sugar monitoring every 6 months.)

Or

Tab Quetiapine, as in schizophrenia Or Tab. Haloperidol 5 mg/day, which can be increased up to 10 mg/day (in 2 divided doses) over 1-2 weeks

Or

Tab. Divalproex (combination of sodium valporate and valproic acid) and lithium carbonate are other medications (mood stabilizers), also used for treatment of mania, but should be used only under strict psychiatric supervision. Risperidone and olanzapine have been associated with weight gain when used for a long period. Patients should be encouraged for lifestyle modification like regular physical exercise and diet control.

If the patient develops extra pyramidal symptoms like tremors, rigidity, slowness of movements, parkinsonian face, sialorrhoea while on antipsychotics, add Tab. Trihexyphenidyl - 2 mg once in morning and once in afternoon (attempts may be made to taper it off after 3 months)

For sleep disturbance

Tab. Lorazepam 1-2 mg or Clonazepam 0.25-1 mg at bedtime may be given in the initial period (usually for 10-15 days, to be tapered off thereafter). In case of acute excitement or violent behaviour, the patient may be given

Inj. Haloperidol 5-10 mg IM Stat + Inj. Promethazine chloride 25-50 mg IM

Or

Inj. Olanzapine 10 mg IM

Or

Inj. Lorazepam 2 mg IM

The injection can be repeated after 8 hours.

Improvement starts within 1 week. The treatment may need to be given for a period of 3-6 months, usually at least for 3-4 months after the patient becomes asymptomatic. If there is no improvement in a week, the patient should be referred to a psychiatrist.
Current episode of depression

Line of treatment is similar to that as described under depression section. However, the patients of bipolar depression should always be prescribed a mood stabilizer along with the antidepressant. Most antidepressants if given alone in a patient with bipolar disorder without cover of mood stabilizer have a strong potential to cause switch to mania.

Prophylactic treatment
Tab. Lithium carbonate 900-1500 mg/day in 2-3 divided doses or
Tab. Carbamazepine 600-1200 mg/day in 3 divided doses.

Or
In rapid cyclers (>5 episodes/year), Tab. Sodium valproate or divalproex 500-1500 mg/day in 2-3 divided doses.

Note: Prophylactic treatment should only be given under psychiatric supervision. Prophylaxis is required generally after 2-3 episodes. Prophylactic treatment may continue for a duration varying from 3 years to lifelong. Patients on lithium require regular blood level monitoring. Liver function test and blood cell counts should be performed at baseline and once in 6 months in patients on carbamazepine and sodium valproate.

Patient education
• General guidelines about the illness and medications similar to that for schizophrenia.
• Emphasise on recurrent course of illness and not to get too much worried on recurrences.
• Relapses can be treated as successfully as the first episode.
• When on lithium, advised to take plenty of fluids, especially during summer; not to restrict salt.
• the patient develops fever, vomiting or diarrhoea while on lithium, reduce the dose of lithium to half and contact the physician or the psychiatrist immediately.

In case of non-response to medication patient should be considered for Electro Convulsive Therapy (ECT).

References

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DEPRESSION

Depression is one of the commonest psychiatric disorders. Patients often present to the general practitioners and the physicians with vague somatic symptoms or aches and pains for which no physical cause is found on assessment. A careful screening for depressive symptoms (as outlined under salient features) usually elicits the diagnosis.

SALIENT FEATURES

- Sadness of mood, loss of pleasure in activities, which one enjoyed earlier, generalised lack of interest; anxiety is often associated.
- Lack of energy, slowness of thought, decreased concentration and efficiency.
- Lack of sleep, appetite and libido.
- Ideas of insufficiency, inadequacy and worthlessness, unexplained ideas of guilt, death wishes, suicidal ideas, history of suicidal attempt.
- Disruption of social and occupational functioning.
- Symptoms should be present for a minimum period of 2 weeks for a diagnosis of depression to be made. Hypothyroidism may co-exist.

Treatment

Non pharmacological

- Counselling, reassurance, psychological support, encouragement.
- Cognitive behaviour therapy (to be given by a psychiatrist/clinical psychologist).

Pharmacological

Cap. Fluoxetine 20-60 mg/day as a single dose in morning with food; starting with 20 mg/day which can be increased up to 60 mg/day in increments of 20 mg after 5-6 weeks in case of non-response.
Or
Tab. Sertraline 50-200 mg/day as a single dose in morning with food; starting with 50 mg/day which can be increased up to 200 mg/day after 5-6 weeks in increments of 50 mg in case of non-response.
Or
Tab. Escitalopram 10-20 mg/day as a single dose in morning with food; starting with 5 mg/day which can be increased to 10 mg/day over a week, and further up to 20 mg/day after 5-6 weeks in case of non-response. Most patients would respond at 10 mg/day.
Or
Tab. Venlafaxine 75 mg/day, can be increased to 150 mg/day over 1-2 weeks; can be further increased to 225 mg/day after 5-6 weeks if there is inadequate response; needs to be given in two divided doses; extended release preparations can be given as a single dose.
Or
Tab. Mirtazapine 15-45 mg/day as a single night-time dose starting with 15 mg/day which can be increased up to 45 mg/day in increments of 15 mg after 5-6 weeks in case of non-response.
Or
Tricyclic antidepressants (TCAs) like Tab. Imipramine or Tab. Amitriptyline 75-150 mg/day in 2-3 divided doses; to be started at 25 mg twice a day, and increased by 25 mg every third day till 150 mg/day.
(Caution: TCAs to be avoided in patients with epilepsy, heart disease, glaucoma, and benign prostatic enlargement.)

Antidepressant medication begins to improve sleep, appetite and anxiety feelings within about one week. Feelings of depression may take from 2 to 4 weeks to improve. By about 12 weeks, most of the patients substantially improve. Observe closely patients with suicidal tendencies during this period. If there is no response to treatment in 5-6 weeks, check compliance and reconfirm diagnosis. If compliant with medication, increase the dose or change antidepressant or augmentation with another SSRI or antidepressant from another class may be warranted or refer to a psychiatrist at this stage.

For the first episode of depression, treatment needs to be continued for 6-9 months. Dose may be tapered off over a period of 6-8 weeks. However if symptoms recur during this period, treatment needs to be continued for another 3-4 months. In case of multiple episodes of depression, treatment may need to be continued indefinitely.

In cases of bipolar depression, patients while on antidepressants may have a sudden switch to mania. In such cases, stop antidepressants immediately, and refer the patient to a psychiatrist.

Depression with suicidal ideas or suicidal intent pharmacological treatment should be augmented with Electro Convulsive Therapy (ECT).

**Patient education**

- Explain the nature of illness, consequences of untreated depression, suicidal risk, need of adequate doses for adequate duration and other supportive measures.
- The therapeutic response takes time to appear but side effects may appear earlier. Common side effects of tricyclic antidepressants are dry mouth, constipation, postural hypotension (giddiness), blurred vision, sweating, palpitation, tremors, delayed micturition, sedation, erectile dysfunction, etc.
- Common side effects of SSRIs are agitation, headache, nausea or heartburn, tremors, delayed ejaculation, erectile dysfunction and loss of appetite.
- Mirtazapine causes sedation, giddiness and increased appetite and weight gain.
- The drug may impair mental or physical abilities initially, avoid driving or operating machinery if patient is drowsy.
- One should avoid alcohol during treatment, as it may cause oversedation and dizziness.
- Patient should be cautioned against increasing or decreasing the dose without medical advice.
- Patient should be advised not to stop drug suddenly as it may result in withdrawal symptoms.

**References**


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MENTAL AND BEHAVIOURAL PROBLEMS IN CHILDREN

Psychological and behavioral problems are quite common in children. Behavioural problems that are not developmental stage specific point towards an underlying psychological/psychiatric disorder if these occur commonly, are intense, pervasive and interfere with the child’s functioning. Some of these problems have been enlisted below and children exhibiting such problems require would need a detailed assessment. An early intervention would be helpful to prevent functional decline in a child. The child may be referred to a psychiatrist, paediatrician or a clinical psychologist for further assessment and treatment.

- Frequently experiencing mood swings
- Avoiding friends and family
- Anger and rage attacks
- Abusing drugs
- Spending excessive time in bathroom or room alone
- Talking to self/muttering
- Not doing the things he or she used to enjoy
- Worrying constantly
- Lacking energy or motivation
- Poor appetite
- Having sleep difficulties
- Academic decline
- Rebelling against authority
- Hitting or bullying other children
- Damaging other people’s property
- Not concerned with his or her appearance
- Obsessed with his or her weight
- Attempting to injure him or herself or others

A child should be referred to a higher centre in the following situations:

- If the child is suicidal
- If the child exhibits verbal, physical or sexual aggression
- If the child is suspected of using drugs
- If the child has made attempts in the past to self-harm
- If the child exhibits bizarre behaviour
- If the child has a medical problem such as seizures

Management of some common psychiatric conditions encountered in children is given in the following sections.

Parent/Patient education

- Parental counseling; learning to anticipate the situations that allow behavioural problems to appear and plan ahead so as to minimize disruption.
- Encouraging parents to screen the peer relationships of the child so as to protect the vulnerable child.
- Recognising the attention difficulties of the child and tailoring the work expectations by reducing the length and complexity of assignments.
A coordinated effort both at home and school is required. Refer to a psychiatrist, or a higher centre for treatment and management.

References

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DEPRESSION IN CHILDREN

Depression in children is not uncommon, though the presentation may vary. The predominant mood is often irritable. Diagnosis is made on the same criteria as in adult depression. Depression should be suspected in a child presenting with decline in school performance, withdrawal from peers, and increased conflict with peers, siblings, parents and other adults, and irritability. Children may be anxious and tearful and may also present with somatic symptoms.

Treatment should mainly be supportive. Pharmacological treatment is generally not encouraged. TCAs and SSRIs can be used, if depression is severe. Imipramine and Fluoxetine, can be given. Other SSRIs may not be as safe as fluoxetine. Duration of treatment is as described in adult depression.

References

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GENERALISED ANXIETY DISORDER

It is one of the commonest psychiatric disorders in general clinical practices, more common in women than in men. Patients often present in primary care with symptoms of sympathetic over activity or vague aches or pains, sleep disturbance, forgetfulness or worrying too much.

SALIENT FEATURES
• Persistent anxiety, present all the time.
• Tremulousness, shakiness, generalised aches, restlessness
• Apprehension, worries of future, irritability, sleeplessness.
• Palpitations, sweating, dry mouth, increased frequency of micturition, abdominal distress.
• Intensity, duration and frequency of the anxiety and worry are far out of proportion to the actual likelihood or the impact of the feared event and it interferes with the task in hand.

Treatment

**Non pharmacological**
• Reassurance, psychological support, encouragement.
• Anxiety management — relaxation exercises, breathing exercises, meditation and yoga.

**Pharmacological**

Tab. Diazepam 5-20 mg/day
Or
Tab. Lorazepam 1-4 mg/day
Or
Tab. Alprazolam 0.75-1.5 mg/day in 2-3 divided doses.
Or
Tab. Clonazepam 0.5-1.0 mg/day in 2-3 divided doses.

Treatment should be started at the lowest dose, which can be increased up to the maximum dose to achieve a therapeutic response, but attempt should be to keep it at the minimal possible level.

Treatment with above should not be given for more than 2-4 weeks because of the abuse potential.

Or
Cap. Fluoxetine 20-60 mg/day as a single dose in morning with food; starting with 10 mg/day which can be increased to 20-60 mg/day in increments of 20 mg after 5-6 weeks in case of non-response. Most patients may not require more than 20 mg/day.
Or
Tab. Sertraline 50-200 mg/day as a single dose in morning with food; starting with 25 mg/day which can be increased up to 200 mg/day after 5-6 weeks in increments of 50 mg in case of non-response. Most patients may not require more than 50 mg/day.
Or
Tab. Escitalopram 10-20 mg/day as a single dose in morning with food; starting with 5 mg/day which can be increased up to 20 mg/day after 5-6 weeks in case of non-response. Most patients may not require more than 10 mg/day.

(Caution: SSRI's may exacerbate anxiety symptoms in the first few days; usually a low dose is required for anxiety disorders.)

Or
Tab. Buspirone 15-60 mg/day in 2-3 divided doses. It is effective in 60-80% of patients especially in reducing the cognitive symptoms. It takes 2-3 weeks to show its effect.
Or
Tab. Paroxetine 12.5 mg/day as a single daily dose with or without food usually in the morning, it can be increased up to 37.5 mg/day at the interval of 1 week.
Or
Tab. Propranolol 40-80 mg/day in 2 divided doses, given especially if the predominant symptoms are those of sympathetic overactivity.

(Caution: To be avoided in patients with history of chronic obstructive airway disease and bronchial asthma.)
SSRIs to be used if the patient needs treatment for longer period. Both SSRIs and benzodiazepines can be started together. Benzodiazepines can be withdrawn over 2-4 weeks as the SSRIs take over the effect. In another approach, buspirone may be combined with benzodiazepines initially as it shows its effect after 2-3 weeks after which benzodiazepines may be gradually withdrawn.

**Patient education**

- Patient should be encouraged to bring changes in lifestyle like mild exercise such as morning walk, keeping some time for leisure or entertainment.
- Patients should be informed about the abuse potential of the drug.

**References**


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**OBSESSIVE COMPULSIVE DISORDER**

Obsessive compulsive disorder is characterized by obsessions and compulsions and often tends to be chronic. Illness usually begins in adolescent or early adult life and majority of patients have a chronic waxing and waning course.

**SALIENT FEATURES**

- Recurrent obsessional thoughts may present in form of repetitive ideas, images or impulses (e.g. constantly thinking that the door has been left unlocked).
- Perceived as senseless by the sufferer, who feels distressed and tries to resist them unsuccessfully.
- Compulsive acts are repetitive behaviour which are not enjoyable and do not result in the completion of inherently useful tasks (e.g. constantly going back to check the door lock) and cause marked anxiety and distress in the individual.
- Significantly interfere with the person’s normal routine, occupational (or academic) functioning, or usual social activities or relationship

**Treatment**

**Non pharmacological**

- Counselling, reassurance, support
- Cognitive behaviour therapy (to be given by a psychiatrist/clinical psychologist)
- Exposure and response prevention (to be given by a psychiatrist/clinical psychologist)
Pharmacological

Cap. Fluoxetine 20-60 mg/day as a single dose in morning with food; starting with 10 mg/day which can be increased to 20-60 mg/day in increments of 20 mg after 5-6 weeks in case of non-response.

Or

Tab. Sertraline 50-200 mg/day as a single dose in morning with food; starting with 50 mg/day which can be increased up to 200 mg/day after 5-6 weeks in increments of 50 mg in case of non-response.

Or

Tab. Escitalopram 10-20 mg/day as a single dose in morning with food; starting with 5 mg/day which can be increased up to 20 mg/day after 5-6 weeks in case of non-response.

Or

Tab. Fluvoxamine 50 mg twice a day to be increased to 100-200 mg twice a day in 1-2 weeks. (Caution: SSRIs may exacerbate anxiety symptoms in the first few days; usually a low dose is required for anxiety disorders.)

Or

Tab. Clomipramine 75-150 mg/day in single or divided doses; to be started with 25 mg twice a day, increased by 25 mg/day every third day till 150 mg/day (to be avoided in patients with epilepsy, heart diseases, glaucoma and benign prostate hypertrophy). In the initial 2-4 weeks of treatment, one may need to add benzodiazepines like diazepam, lorazepam or alprazolam if anxiety symptoms are troublesome. These can be given in doses as described under generalized anxiety disorder.

Anxiety and distress are the first symptoms to respond. Obsessive and compulsive symptoms respond later. Doses of SSRIs required are often higher than in depression and response is slower than in depression. If no response is seen within 6-8 weeks, the patient should be referred to a psychiatrist.

Patient education

• Reassure the patient that although disease is distressing and disabling, it is treatable.
• The drug takes about 2 weeks for its therapeutic response to manifest.
• Side effects may appear before the onset of therapeutic response. Common side effects of Clomipramine are dry mouth, constipation, postural hypotension (giddiness), blurred vision and sedation. Side effects of fluoxetine and other SSRIs include gastrointestinal distress, nausea, headache, nervousness, anorexia, restlessness and sexual side effects. Patient usually adapts to these side effects with time.
• Advise the patient that treatment may continue for a long time. He/she should not leave drugs without medical advice.
• in case of any untoward effects of drugs, he/she must immediately get in touch with his/her clinician.
• Patient must continue all his regular activities as far as possible.
• Yoga, meditation, physical exercises are useful measures along with drug therapy.

References

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INSOMNIA

Insomnia is one of the commonest complaints in psychiatric, medical and general clinical practice. Common causes include a recent stress, psychiatric illnesses like depression and anxiety disorders, pain in any body part or substance abuse.

**SALIENT FEATURES**

- Difficulty in initiating sleep, frequent awakenings from sleep, early morning insomnia or non-restorative sleep. In the elderly, the physiological reduction in number of hours of sleep does not amount to insomnia. If the patient is distressed by decreased sleep, treatment may be given to increase the duration of sleep.
- Stressful situation leading to insomnia or the symptoms of the causative illness can be elicited on careful enquiry. Duration of symptoms may vary from few days to many months or years depending on the cause.

**Treatment**

Treat the underlying cause. In both primary insomnia (where no cause is identifiable) and insomnia due to other causes, management includes introducing good sleep hygiene and medications for short period, if required.

**Sleep hygiene**

- Set a schedule: Go to bed at a set time each night and get up at the same time each morning. Avoid daytime naps. Limit daily in-bed time to the usual amount present before the sleep disturbance.
- Avoid large meals near bedtime; eat at regular times daily. No stimulant medication or food beverages (caffeine, nicotine, alcohol, etc.) especially in the evenings.
- Mild-to-moderate physical exercise in the morning.
- Relax before going to bed: a warm bath, reading, or another relaxing routine can make it easier to fall sleep. Avoid evening stimulation: substitute television by radio.
- Do not lie in bed awake: If you cannot get to sleep, do not just lie in bed. Do something else, like reading, watching television, or listening to music, until you feel tired.
- Practice evening relaxation routines, such as progressive muscular relaxation or meditation.
- Maintain comfortable sleeping conditions: avoid extreme temperatures.

**Pharmacological**

Tab. Zolpidem 5-10 mg at bedtime.
Or Tab. Lorazepam 1-2 mg or Clonazepam 0.25-0.5 mg at bedtime.

**Precautions**

- Medication to be given 1/2 to 1 hour before the usual time of going to bed.
- Medications should be prescribed at the lower dose for a period of 5-7 days.
- Benzodiazepines have risk of abuse potential if taken for more than 3 weeks. Diazepam and nitrazepam carry risk of dependence and should be avoided.
- Zolpidem also has dependence potential and therefore long-term use should be discouraged.

**Patient education**

- Stress on basic principles of sleep hygiene as above.
- Patient to avoid exceeding the prescribed dose and should not take medicines beyond the prescribed period.
- Sometimes these drugs can lead to sedation during daytime. In such case, reduce the dose to half and contact the doctor.
- Diazepam and nitrazepam carry risk of dependence.

**References**

ALCOHOL DEPENDENCE SYNDROME

Persisting with drinking despite clear evidence of overtly harmful consequences and withdrawal state.

SALIENT FEATURES

- Craving, compulsion to drink, difficulties in controlling alcohol consumption
- Tolerance (increasing amount required to achieve the same effect)
- Progressive neglect of alternative pleasures or interests
- Withdrawal symptoms are tremor, tachycardia, anxiety, sleep disturbance, nausea, vomiting, hallucination, generalised seizure and delirium in severe cases

Treatment

I Detoxification (Treatment of the withdrawal state and associated problems)

Detoxification can be done in an outpatient or inpatient settings. Outpatient treatment is preferred when the withdrawal state is uncomplicated. Inj. Thiamine 100 mg IM.
Or Tab. Thiamine orally along with oral multivitamins and Tab. Folate 1 mg. Tab. Chlordiazepoxide 10-40 mg 4 times a day, depending on severity of dependence. Or Tab. Diazepam 5-20 mg 4 times a day.

Once the patient is well sedated and stable, the dosage should be decreased 20% per day over a maximum period of 2 weeks. The patient should be monitored over this period for the appearance of the signs of delirium. For elderly patients or in presence of significant liver disease, Tab. Oxazepam 15 mg or Tab. Lorazepam 2-4 mg every 6 hour should be started.

Inpatient treatment is advised when withdrawal state is associated with seizures, delirium or emesis, fluid and electrolyte disturbance, medical conditions like pneumonia or surgical problem (e.g. head trauma), hallucinatory behaviour, suicidal risk and previous history of delirium tremens.

The Vital signs and withdrawal symptoms should be monitored 2-4 hourly. Once the patient is stable, the dose should be gradually tapered off (20% per day) over a period of 7-10 days.

Treatment of dependence with complications

Basic treatment will be as described above, but the patient needs to be hospitalised.

Guidelines are as below:

1. Fluid and electrolyte disturbance should be corrected, especially if there is vomiting or fever.
2. Seizures - Rum fits (appearing within 24 hours of abstinence) can be treated with Inj. Diazepam 1 0.mg or Inj. Lorazepam 2 mg IV gat, especially when seizures are repeated. Prophylactic treatment is not recommended for true alcohol withdrawal fits.
3. Delirium tremens - The patient should be preferably treated in an intensive care unit.

   a) An intravenous line should be started immediately and Inj. Thiamine 100 mg administered IV, or IM. Thiamine along with multivitamin should be continued parenterally till normal diet is resumed. Later oral thiamine should be continued for at least 3-4 months.

   b) Dextrose and saline IV should be given at a rate adequate to replace fluid losses and maintain blood pressure.

   c) Hyperthermia should be managed with cold sponge. Tab. Paracetamol 500 nig PO 4 times a day may be used in absence of any hepatic dysfunction.
d) Inj. Diazepam 10 mg should be given, slowly IV and should be repeated every 15-20 minutes till sedation is achieved.

e) Physical restraint may be necessary if the patient is combative.

f) Associated medical and surgical problems should be simultaneously investigated and treated appropriately.

**Long-term treatment (To be treated by a psychiatrist)**

The goal of this treatment is to help the patient maintain long-term abstinence.

**Non pharmacological**

Individual counselling and family support should be planned along with pharmacotherapy. After remission, the patient should be encouraged to join self-help groups like Alcoholic Anonymous (AA).

**Pharmacological**

Deterrents like disulfiram or anticraving agents like naltrexone or acamprosate are used for long-term treatment of alcohol dependence.

Tab. Disulfiram 250 mg a day may be used if the patient desires enforced sobriety and who have remained alcohol-free for at least 7-10 days.

Patients taking disulfiram develop an extremely unpleasant reaction on intake of even small amounts (e.g. 7 ml) of alcohol. The reaction occurs due to accumulation of acetaldehyde and includes flushing, headache, throbbing in head, dyspnœa, hyperventilation, tachycardia, hypotension, sweating and confusion.

In the event of disulfiram-ethanol reaction (DER), fall in BP should be controlled on priority basis. If DER is mild, assurance and oral fluids suffice. In case of moderate or severe DER, IV fluids are required and some patients may even need dopamine infusion.

Generally, DER does not occur in the first week of disulfiram use and if alcohol is consumed after 5-7 days of stopping disulfiram, but can occur up to 2 weeks after stopping disulfiram.

Disulfiram should be continued for several months to establish a long-term pattern of sobriety or Tab. Naltrexone 50 mg orally once daily.

*(Caution: Baseline hepatic functions should be assessed and monitored once a month while on naltrexone treatment. The drug is usually continued for a period of 6 months. However, it may have to be withdrawn in presence of significant liver disease [i.e. several-fold increase in the serum levels of transaminases]).*

Or

Tab. Acamprosate (333 mg) 1-2 g/day in 3 divided doses. There is no optimum duration of therapy but, benefit beyond 12 months has not been demonstrated. *(Caution: Contraindicated in severe renal and hepatic failure; reduce dosage in moderate renal impairment; monitor renal and liver function regularly.)*

**Patient education**

Patient should be told about the nature of illness, course and treatment modalities available through individual sessions.

**References**


4. DENTAL CONDITIONS
OROFACIAL PAIN

Pain in teeth, Jaw bones and oral cavity may be of dental and non-dental Origin

DENTAL CAUSES:

Caries with Pulpitis, Periodontitis, Pericoronitis, Aphthous ulcers, Herpetic, Vesicular, Tubercular, Fracture, Osteomyelitis, Cyst and tumor, Dry socket.

NON-DENTAL CAUSES:

Maxillary sinus diseases, Trigeminal Neuralgia (primary or idiopathic and secondary to dental and non-dental causes), T.M.J disorder (MPDS, T.M.J arthritis), Glossodynia (painful tongue).

Referred Causes: Myocardial infarction pain at times felt as left side jaw pain

Diagnosis: For dental causes there is always a dental focus like caries, periodontal pocket impacted tooth, intra oral sinus which are to be examined with probe, mouth mirror and a good light source. Patient with trigeminal neuralgia has to be excluded from dental, maxillary sinus causes as secondary neuralgia. May have a trigger zone

Investigation: Radiological, Routine blood test.

Treatment:

1. Dental conditions to be treated with local Eugenol dressing, restoration, R.C.T, Extractions.
2. T.M joint pain treated with Etoricoxib 90mg 1 tablet after food bed time, muscle relaxants, anxiolytic/antidepressant, occlusal splints.

Referral Criteria:

1. Neurological pain not responding to conservative treatment T.M.J pain needing for the investigations.
2. Non dental origin facial pain.

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TEMPORO MANDIBULAR JOINT DISORDER (TMD)

Pain in front of the ear without ENT causes are mostly of TMJ origin. These can be MPDS. TMJ Arthritis, Trismus, Ankylosis,

Diagnosis:

MPDS Hypomobility of TMJ resulting in reduced oral opening, Click sound on opening, Pain in TMJ, Anxiety back ground, Faulty Dental Restoration.

Arthritis: Rheumatoid Arthritis or Osteoarthritis.

Lab Tests: ESR, C-reactive protein, ASO, Rh factor. Radiological investigations.

Treatment: Rest to the joint, Correction of Dental Occlusion, Occlusal Splints. Long acting intra capsular Depomedrol injection .05 ml to with Lignocaine 2% in to the joint weekly with aseptic measures, 3 to 4 such.

Referral: Resistant cases for further evaluation of surgical correction.

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TEMPORO-MANDIBULAR JOINT DISLOCATION

Acute TMJ dislocation is characterized by wide open mouth unable to close. Condyle moves anterior to the articular eminence making a hollow in front of the ear. Painful spasm of the muscles of mastication.

**Treatment:** With a Mandibular Nerve block with 2% Lignocaine, Stand in front of the patient with lower occlusal plane at a level below the elbow. With padded fingers the Mandible is pushed down ward, backward and upward to the Glenoid fossa. Failure to achieve reduction needs the same manoeuvre under GA. Following reduction, the jaw is temporarily immobilized with Barrel Bandage.

Chronic or long standing dislocation or Subluxation is referred for surgical Management.

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GUM (PERIODONTAL) DISEASES

**Diagnosis:** Characterized by painful swollen, bleeding gums with bad breath (Halitosis), loose teeth without any systemic diseases background.

**Gingivitis:** It is an inflammatory response of the gingivae without destruction of supporting tissue, without teeth mobility.

**Periodontitis:** Describes a group of inflammatory diseases affecting all the periodontal structures resulting in destruction of all the supporting tissue leading to the development of periodontal pocket or tooth mobility.

**Gingival Hyperplasia (Epulis):** May be localized for local causes or in generalized form as in Idiopathic familial gingival hyperplasia. Phenytoin induced hyperplasia, anti-hypertensive drug induced (Calcium Channel blocker) hyperplasia, pubertal hyperplasia of gum. Pregnancy tumor is a localized growth on gingiva that regresses after pregnancy. Surgical recontouring might be needed in resistant cases.

Intra Oral X rays and OPG shows infra or supra bony defects.

**Treatment:** Rule out systemic causes by appropriate investigations.

Anaerobic infection treated with Tetracyclin 500 mg thrice daily, or Doxycycline 100 mg twice daily. Metronidazole 400 mg thrice daily for 5 days, Chlorhexidene or Povidone Iodine mouthwash. Oral Prophylaxis (Scaling), Surgical correction of the bony alveolar defects.

**Referral:** For surgical correction.

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DENTAL CARIES (CAVITY)

It is a common disease affecting individuals of all age groups but the younger and older individuals are more commonly affected.

**Signs & Symptoms:** Dental caries appears as

- Chalky white, Brown or black discoloration
Cavity in the tooth
- Usually painless in the beginning and on progression it presents as mild sensitivity to hot and cold.
- Pain in advanced stages when cavity becomes very deep

**Treatment:** Restoration – When cavity is small and painless.

- Root Canal Treatment: Painful tooth with or without periapical abscess
- Extraction: Tooth that can’t be restored.

**Analgesics:** Ibuprofen - 400mg SOS, Diclofenac/Aceclofenac – 50mg /100mg SOS.

**Antibiotics:**
- Amoxicillin – 500mg TDS 3-5 Days

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**HYPERSENSITIVITY**

This occurs when the enamel that protects our teeth from sensitivity gets thinner or lost or when gum recession occurs, exposing the underlying dentin.

This is due to chemo-mechanical loss due to faulty brushing technique, excessive consumption of carbonated drinks, psychogenic grinding of teeth (bruxism).

**Signs & Symptoms:** If hot, cold, sweet, sour foods and drinks or cold air, makes a tooth/teeth sensitive or painful then it is known as hypersensitive teeth.

**Treatment:**
- Desensitizing tooth paste containing potassium nitrate, strontium chloride, etc.
- Use of soft bristle brush
- Use of Fluoride varnish/Gel
- Root canal treatment and crown in cases not controlled by the above.

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**CARE OF CHILDREN TEETH**

- Pure deciduous dentition erupts at 6 months and stays up to 6 yrs
- The shedding of teeth starts at the age of 6 yrs & permanent teeth start appearing till 12 yrs making this period as mixed-dentition.
- Deciduous teeth are vulnerable for caries, rampant caries, may be due to lack of oral care due to nursing bottle, night breast feeding.

**Treatment:** Enameloplasty, pit & fissure sealant, topical fluoride application, diet counselling with the parents with restriction of sugar intake particularly in between the meals, pulpotomy, pulpectomy or extraction.

Patients with crowding in the mixed-dentition period should be planned for serial extraction (C),(D),(4).

In selected cases, space maintainer should be planned to prevent crowding or malocclusion in the permanent dentition.
PREMALIGNANT ORAL LESIONS

Oral Leukoplakia: Is a clinical condition of white patches which are not easily scrubbed off and cannot be diagnosed as any other definite oral lesions, mostly with tobacco chewing or smoking habits. Has a malignant potentiality from 3 to 11% cases. Treated with conservative means until dysplasia appears.

Erythroplakia: Appears same as Leukoplakia with speckled areas of redness and/or ulcers with more malignant potential

Oral Sub Mucous Fibrosis: Classified as a pre-malignant condition with gradually noticed closure of oral opening with feeling of tightness, appearance of white bands in buccal mucosa, lip, Faucial pillars, Lip and floor of the mouth burning sensation of mouth more so with spicy food. Mostly associated with Gutka (Processed Arecholine, tobacco) habits

Treatment: Abstinence from habits, Jaw exercises for limited forceful opening of mouth to bring to 35 mm

AntiOxidants, Intralesional injection of Triamcinolone with Placental Extracts and Hyaluronidase sometimes help

Referral Surgical Correction

Oral Lichen Planus: Appears as Localized or diffuse thread like interlacing bluish white ulcers Mostly on Buccal Mucosa, Tongue, Alveolar Mucosa with burning mouth. Often associated with Hypertension, Diabetes Mellitus and Anxiety background

Treatment: Anxiety reduction protocol, Local application/Intralesional use of Triamcinolone, Clobetasol ointment

Patient with Anticoagulants: Patients on anticoagulant therapy like Ecosprin and clopidogrel should be advised to withdraw the same drugs for 3-5 days in consultation with cardiologist. Patients with warfarin can go for simple extraction with INR value under 2.6 or else referred for cardiologist opinion to modify the regime.

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OROFACIAL INFECTION

DENTO-ALVEOLAR ABSCESS:

Symptoms: Pain, Fluctuant Swelling in the vestibule, palate with a definite focus of infection as caries or periodontal pathology.

Treatment: Drainage of abscess, Extraction of non restorable tooth.

Referral: Root Canal/Endodontic treatment

Ludwig’s Angina: Bilateral firm swelling in the floor of the mouth and submandibular region with fever, lymphadenopathy, leukocytosis. Elevated tongue, shrilled voice warrants airway consideration for referral tracheostomy.

Referral: General management of the patient, Injectable Antibiotics – Ceftriaxone 1Gm BD, Metronidazole 100mg –TID.

Space Infections: Buccal Space infection, lateral Pharyngeal Space are marked with swelling trismus with systemic signs. Spread of infection to mediastinum may be borne in mind and suitably referred.

Treatment: Drainage and Injectable Antibiotics – Ceftriaxone 1 gm BD, Metronidazole 100mg –TID.

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ORTAL ULCERS

SINGLE ULCER:

Traumatic Ulcer, Bullous or vesicular ulcers (pemphigus)
(Sharp tooth, denture trauma)

MULTIPLE RECURRENT ULCERS:

Aphthous Ulcer: Small, oval, greyish white ulcer on mobile mucosa with erythematous border lasting for 1 wk with psychogenic background.

Herpetic Ulcer: Multiple, small, involves attached mucosa with systemic viral involvement.

Herpangina: Small, multiple, extremely painful, involving soft palate, faucial palate,
Drug induced (chemotherapy, drug & contact allergy)

Treatment: Most of the ulcers would need palliative treatment with:

Topical Anesthetic Agent – 2% Lignocaine, Bezocaine, Amlexanox as local application.
Local Cauterization with silver nitrate 1-2% (Isolating the Area), Diathermy if available

Topical Steroids: Triamcinolone 0.1%, Clobitasol, Retinoids.

Systemic therapy like pentoxyphyllin 300mg, prednisolone 30mg/day, deflazacort 24mg/day and immunosuppressant depending on the type in consultation with physician.

Referral: Ulcers with systemic background, persistent ulcers of more than 3 wks. Persistent ulcer on the lateral border of tongue and floor of the mouth should have high suspicion index for carcinoma.
5. HAEMATOLOGY
ANEMIA

INTRODUCTION:

Anemia is a very common clinical problem. It commonly refers to a low hemoglobin level and arise out of myriad of causes ranging from nutritional factors to malignancy. Accordingly, its evaluation needs a rational approach and treatment will vary according to the cause.

Common causes of Anemia:

1. Iron deficiency anemia
2. Hemolytic anemia (e.g. Thalassemia and Sickle Cell Anemia)
3. Megaloblastic anemia
4. Leukemia and lymphoma
5. Hypoplastic anemia
6. Anemia of chronic diseases like CKD, connective tissue diseases, chronic infection like tuberculosis, malignancy etc.

DIAGNOSTIC CRITERIA:

Criteria to define anemia varies; however, a hemoglobin level <13 gm/dl in adult males and <12 gm/dl in adult females is adequate for defining anemia.

RATIONAL INVESTIGATIONS:

A rational investigation must include a careful history, physical examination and laboratory investigation. A history of menorrhagia may point to iron deficiency anemia; similarly, repeated pain in extremities may be a pointer for Sickle Cell Anemia (SCA) and repeated blood transfusion since early childhood may be due to Thalassemia major.

Physical examination like koilonychia, glossitis, angular stomatitis suggests iron deficiency anemia; icterus and Splenomegaly may point to hemolytic anemia. Similarly, presence of lymphadenopathy may point to lymphoma, leukemia or chronic infection like tuberculosis.

History and physical examination findings are important in choosing from the various investigations mentioned below.

Laboratory Investigation:

A. HEMATOLOGICAL

i. Hb, DC, TLC, Total Platelet count, comment on peripheral smear (hyper-segmented neutrophil points to Megaloblastic anemia, blasts in leukemia, hypochromic target cells in thalassemia etc.)
ii. CBC (helpful in classifying anemia into microcytic hypochromic, normocytic normochromic and macrocytic anemia)

iii. Reticulocyte count (helpful in classifying between causes of anemia leading to decreased production and increased destruction)

iv. Sickling Test and Hb Electrophoresis

B. Stool examination for hook worm, occult blood.

C. Upper GI endoscopy and colonoscopy when a GI source of blood loss is suspected

D. Lymphnode FNAC/Excisional biopsy

E. Iron studies- Serum Iron, Ferritin, TIBC, Transferrin saturation.

F. Serum B₁₂ and Folate level estimation

G. Others- Chest X-ray, USG of abdomen and pelvis, CT scan as and when required.

NON-DRUG TREATMENT:
1. Nutrition and balanced diet
2. Inclusion of both vegetables and non-vegetarian diets (Fish, Meat, Egg).

DRUG TREATMENT:

A. Iron Deficiency Anemia: The cause of iron deficiency is to be determined and treatment directed to cause be provided. However, the iron deficiency itself needs treatment. Various approaches include, a) Oral Iron Therapy, b) Parenteral Iron Therapy c) Packed red cell transfusion.

a. Oral Iron Therapy: Various types of oral irons used for treatment include ferrous sulfate, ferrous fumarate, ferrous gluconate, ferrous ascorbate etc.. The iron salts contain different amount of elemental iron, e.g. Ferrous sulfate 325 mg contains 65mg of elemental iron, Ferrous fumarate 325 mg contains 107mg of elemental iron, Ferous gluconate 325 mg contain 39mg of elemental iron and ferrous ascorbate 100mg contains 100mg of elemental iron.

Dose of oral iron:- 200mg elemental iron per day.

Duration:- Iron therapy continued till correction of anemia and then for another 6-12 month to restore body iron.

Adverse effects:- Abdominal pain, nausea, vomiting, loss of appetite, constipation.

b. Parenteral Iron Therapy:-

Parenteral iron therapy is needed in case of

i. Oral Iron Intolerance.

ii. Malabsorption

iii. Noncompliance

iv. When rapid iron restoration is needed as in late pregnancy or rapid blood loss.
The presently available parenteral iron preparations include Iron sucrose and Ferric carboxy maltose, which are relatively safe and have a lower chance of anaphylaxis and other reactions, as compared to Iron-dextran. Iron sucrose: 200mg diluted in 100ml normal saline can be given daily. Ferric carboxy maltose: up to 750mg to 1000mg diluted in 100ml normal saline can be given in a day.

Calculation of dose of parenteral iron: Body weight (Kg) × 2.3 × (15 - patients hemoglobin in gm/dl) + 500 or 1000mg (for stores).

Adverse reactions: Anaphylaxis, rash, arthralgia

c. Packed Red Cell Transfusion:
   Only needed in cases of severe symptomatic anemia for hemodynamic stability restoration or in situations like necessity of surgery, delivery etc.

Other Measures:
   i. Albendazole 400mg single dose may be considered in view of parasitic infection.

(B) Nutritional Anemia related to folate and $B_{12}$ deficiency
   a. Folate Deficiency: 5-10mg/day for 4 months may require life long treatment in cases of malabsorption syndrome.
   b. $B_{12}$ deficiency: Initially Hydroxocarbolamin 1000µg IM every 3 to 7 days for 2 months or resolution of neuropathic symptoms followed by 1000µg IM every 3 month.

(C) Other cases of anemia like aplastic anemia, leukemia, myelodysplasia needs evaluation in tertiary care centers.

PATIENT EDUCATION:
   1. To avoid open field defecation and walking bare foot.
   2. To avoid washing of self after defecation in ponds used by the public.
   3. Educate patients regarding sources rich in iron, $B_{12}$, folate.
   4. To advice patients to take both vegetarian and nonvegetarian food items.
   5. To report to doctors if having symptoms like fatigue, breathless, black stool, jaundice etc.
   6. To educate regarding common side effects of iron therapy to maintain compliance.

REFERRAL CRITERIA:
   1. Patients having pancytopenia.
   2. Patients having refractory anemia/ persistent anemia not responding to therapy.
   3. Patients having generalized lymphadenopathy/ purpura/Jaundice/Splenomegaly/ Hepatomegaly
4. When initial evaluation points to hematological and nonhematological malignancies.

REFERENCES:

2. Wintrobe’s Hematology

SICKLE CELL DISEASE

INTRODUCTION

The two most common hemolytic anemia of public health importance in Odisha are Sickle Cell Anemia and Thalassemia.

Sickle Cell hemoglobinopathy is an inherited disorder characterized by sickle shaped RBC in blood. This includes Sickle Cell Trait (AS) and Sickle Cell Disease (SS and compounds heterozygous states). Sickle Cell Traits (AS) remain asymptomatic and act as carriers. Sickle Cell Anemia (SCA), conventionally, includes homozygous Sickle Cell Anemia (SS) and HbSαthalassemia. SCD in steady state manifests with anemia, hemolytic jaundice, hepatomegaly with or without splenomegaly.

Various acute manifestations are

- Increased rate of infection with pneumococcus and other capsulated organisms
- Hand foot syndrome (dactilytis)
- Painful crisis or Vaso-occlusive crisis (VOC)
- Acute chest syndrome
- Splenic sequestration crisis
- Aplastic crisis (Parvo virus B19 infection)
- Hemolytic crisis
- Sickle cell Hepatopathy (Acute hepatic sequestration and acute intrahepatic cholestasis)
- Avascular necrosis of femoral and humeral head
- Osteomyelitis
- Priapism
- Stroke
- Splenic infarction.

In addition, different chronic organ changes include growth retardation, gall stones, pulmonary hypertension, retinopathy, leg ulcer, chronic renal failure, iron overload following repeated transfusion etc.

Sudden death may occur due to splenic sequestration crisis, acute chest syndrome and stroke. Other causes of death are acute painful crisis, sepsis, aplastic crisis, falciparum malaria, hepatopathy, pulmonary hypertension
and progressive renal damage culminating in end stage renal disease (ESRD). Death may also occur during pregnancy
and delivery.

**DIAGNOSIS:**

Sickle Cell Disease should be suspected when patient are having suggestive clinical feature like repeated
episode of pain in extremities, back and ribcage, chronic anemia, splenomegaly, avascular necrosis of femoral head
etc. as well as family history of Sickle Cell Disease. It is more commonly found in certain communities and castes like
agharia, chasa, kulita, scheduled caste and schedules tribes. In such circumstances, the individual should be subjected
to the following tests.

i. **Sickle slide test (24 hours hypoxia):** A simple test which establishes the presence of sickle cell
hemoglobinopathy but can not tell whether trait or disease in absence of other suggestive symptoms or tests.

ii. **Hb Electrophoresis:** Establishes whether the case is a trait or disease.

iii. **High-Performance Liquid Chromatography (HPLC):** Whether the particular case of Sickle Cell
hemoglobinopathy is homozygous or compound heterozygous can only be confirmed by HPLC. But in
certain cases of doubt diagnosis is established by DNA-PCR, and genetic sequencing.

**Laboratory Investigations**

A. **Hematological-** CBC with facility of absolute neutrophil count (before and after starting hydroxyurea)
B. **Biochemical** - Blood urea, Serum creatinine, LFT
C. **Pregnancy test** - In female of reproductive age
D. **Seminal fluid analysis**
E. **Imaging-** Chest x-ray, X-ray hip, Ultrasonogram of abdomen

**NON-DRUG TREATMENT**

1. Avoidance exercise exertion, exposure to extreme heat and extreme cold, avoidance of sporting activities.
2. Plenty of Fluids.
3. Provision of adequate rest and sleep.
4. Blood transfusion: A) Acute transfusion, B) Chronic (Prophylactic) transfusion
5. Bone marrow transplantation.

**Blood transfusion** in sickle cell disease is recommended in splenic sequestration crisis, hyper haemolytic
crisis, aplastic crisis, acute chest syndrome and in pregnancy (hemoglobin level less than 7.0 gm/dL), before surgery
to raise hemoglobin level up to 10.0 gm/dL. The hemoglobin should never be raised beyond 10gm/dL. Simple or
exchange transfusion is recommended for acute chest syndrome, stroke, priapism and resistant VOC not responding
to standard therapy.
Prophylactic blood transfusion every 3 to 4 weeks is recommended for secondary prevention of stroke. This should be combined with iron chelation therapy (Deferasirox 20-40mg/kg/day) wherever there is evidence of iron overload (serum ferritin > 1000ng/ml).

**DRUG TREATMENT**

1. **Folic Acid**: 1 mg/day (However 1mg tablets are not available and 5mg tablets may be given)

2. **Hydroxyurea**: One of the most important diseases modifying measures. Informed consent should be taken from the patient/parents in case of minors. Treatment is usually life long unless stopped for some other reason. Regular follow up and monitoring is necessary.

**Indication of Hydroxyurea**: -
- Repeated painful crisis (3 times or more in last 12 months),
- Repeated blood transfusion for symptomatic anemia (2 times or more in last 12 months)
- History of acute chest syndrome.
- For infants more than 9 months old and children irrespective of their clinical condition,
- As an alternative to repeated blood transfusion in prevention of stroke.
- Sickle Cell Disease patients with Chronic Kidney disease who are on erythropoietin.

**Contraindication of Hydroxyurea**: -
- Absolute neutrophil count < 2000/cmm
- Total platelet count < 80,000/cmm
- Hemoglobin < 5gm/dl
- Pregnancy* and lactation
- Hepatic dysfunction
- Hypersensitivity to hydroxyurea
- Decreased sperm count

* found to be teratogenic in animal studies; child may be born normally.

**Dose**: 10-30mg/kg/day

(In Odisha low dose hydroxyurea i.e- 10mg/kg/day has been found effective)

**Toxicities**: Transient myelosuppression-Anemia, neutropenia, thrombocytopenia

Rarely decrease in sperm count

**Monitoring**: After 2-4 weeks of starting hydroxyurea, CBC, LFT, renal function should be assessed. Hydroxyurea should be stopped when absolute neutrophil count < 2000/cmm, platelet count < 80,000/cmm, Hb < 5gm/dl. It can
be re-started after hematological recovery at a dose of 5mg/kg/day and gradually escalated in every 8 weeks with monitoring of CBC. (Refer to concerned specialist).

Patients at a steady hematological state should be followed up at every 3 months with monitoring of the above parameters.

3. **Oral Penicillin prophylaxis**: 125mg twice daily orally up to 3 yrs and then 250mg twice daily from 3-5 yrs

4. **Vaccination**: against pneumococcus, H. influenza, Hepatitis B and influenza are recommended.

**Treatment of specific complication in Sickle Cell Disease**

1. **VOC/Dactylitis**
   - Hospitalization in moderate to severe cases.
   - Oral or intravenous fluid: 2L/m²q/day.
   - Prompt analgesic use within 30 minutes of arrival (Opioid and/or NSAIDs e.g. inj. **Tramadol** IM, 50-100mg, 2-3 time daily and inj. **Ketorolac** 30-60mg IM stat. then 15-30mg IM, 6-8 hourly; morphine is ideal but not available) for 5-7 days.
   - Alternative analgesics -diclofenac, paracetamol+tramadol.
   - Antibiotic for infection.
   - Oxygen inhalation in case of O₂ saturation < 95%

2. **Acute Chest Syndrome** is characterized by fever, tachypnea, dyspnea, pulmonary infiltrate in X ray, leukocytosis and fall of Hb and O₂ saturation. This condition is treated by:
   - Hospitalization.
   - Analgesics to control pain.
   - Antibiotic covering atypical organisms and pneumococci (Azithromycin 500mg orally once daily, ceftriaxone injection for adults).
   - Oxygen inhalation.
   - Simple or exchange transfusion.
   - Judicious use of IV fluid to avoid fluid overload.
   - Incentive spirometry for prevention.

3. **Stroke**: simple or exchange transfusion and other supportive measures.

4. **Avascular Necrosis of femoral head**: Managed by analgesic and avoidance of weight bearing. Hip replacement later.

5. **Priapism**: Treated with analgesics, IV fluid, exchange transfusion, corporal aspiration and epinephrine irrigation (refer to urologist).
6. **Hepatopathy**: Treated by hydration, rest, close observation, referral for simple or exchange transfusion.

7. **Infection**: Antibiotic effective against capsulated organisms like inj. ceftriaxone 1g IV 12 hourly. Care should be taken in case of children as cetriaxone can produce fatal hemolysis.

8. **Leg Ulcer**: Treated with leg elevation, occlusive zinc oxide dressing, skin grafting, topical GM-CSF.

9. **Chronic Kidney Disease and proteinuria**: Erythropoietin for anemia, ACE inhibitor/ARB for proteinuria (serum potassium should be measured to exclude hyperkalemia before giving ACEI/ARB), (refer to specialist).

10. **Pregnancy and Sickle Cell Disease**: In view of increased complications of SCD, increased fetal and maternal morbidity and mortality pregnancy must be monitored and institutional delivery be conducted. Following precaution should be taken.  
    • Iron supplementation should be given like that of normal counterpart unless there is evidence of iron overload (Serum ferritin>1000 ng/ml)  
    • Haemoglobin and urine examination monthly.  
    • Folic acid (5mg/day).  
    • NSAIDs use only between 12th -28th week for pain crisis, rest same.  
    • Avoidance of general anaesthesia during delivery.  
    • Use of anticoagulants during delivery and postpartum.  
    • Patient should be under care of obstetrician experienced in managing SCD and pregnancy. Sickle cell experts can be consulted.

**PATIENT EDUCATION**

- Patient must be educated to avoid precipitating factors of painful crisis like excess physical and mental exertion, exposure to excess cold and heat, avoidance of exposure to infection and intake of plenty of fluid.
- Education for testing before marriage, partner testing and prenatal diagnosis.
- Avoidance of consanguineous marriage.
- Avoidance of marriage between carriers of sicklers and thalassaemia.
- Early consultation with doctors in case of any complications.

**REFERRAL CRITERIA**

- In case of severe VOC and acute chest syndrome.
- Stroke
- Priapism not responding to analgesics and hydration
• Avascual hip necrosis
• Sickle cell hepatopathy
• Sickle cell kidney disease – CKD, proteinuria.
• Ophthalmic complications
• For initiation of hydroxyurea
• For prenatal diagnosis.
• Pregnancy with SCD.

REFERENCES:
3. Patel DK et al. Low dose hydroxyurea is effective in reducing the incidence of painful crises and frequency of blood transfusion in sickle cell Anaemia patients from Eastern India. Hemoglobin 2012 Aug. 13 (E.pub ahead of print)

THALASSAEMIA

A group of Heterogenetically inherited disorders that result from reduced rate of synthesis of α 0r β- chain’s leading to thalassemia major , minor or Intermedia. Beta- Thalassemia is common and the most important type encountered in our state. Beta- Thalassemia trait can be either asymptomatic or can present with mild anaemia who should be managed with regular Folic acid and rarely needs blood transfusion. The clinical severity of Beta-Thalassemia Intermedia lies between trait and homozygous and the principle of management is similar to Beta -Thalassemia major.

β. Thalassemia Major

Salient Features:
1. Hemolytic Anaemia, Jaundice, Hepato Spleenomegalysometimes with leg ulcers and pigment stones in gallbladder.

2. Severe Anaemia becomes apparent at 3-6 months after birth.

3. Expansion of bone leads to Thalassemia facies: Frontal bossing, malar prominence (maxilla is enlarged) malocclusion of teeth.

4. Patients can be sustained by timely blood transfusions but iron over load caused by repeated transfusion is inevitable. Therefore iron chelation therapy should be introduced when Sr. Ferritin level is >1000ng/dl.

5. Vaccination should be adequately done and specially against, Pneumococcus, H Influenzae, Hepatitis A and B, Meingococcus.

**Diagnosis:**

1. Anaemia with low MCV and MCH : Detected by CBC Machine
2. Confirmation by H.P.L.C/Capillary Zone Electrophoresis
3. Patient presenting with anaemia and hepatospleenomegaly at 3-6 months of life may have Hb Electrophoretic pattern:
   - Thalassemia (Major)
     - HbA₂ - 2%
     - HbF - 98%
   - Thalassemia (Inter media)
     - HbA₂ - variable
     - HbF10 - 80%
     - HbA - 20 -90%
   - Thalassemia (Trait)
     - HbA - >90%
     - HbA₂ - >3.5%
     - HbF - 0-5%

(MCV"< 70 fl/ MCH"< 20pg ) microcytic, hypochromic red blood cells

4. Peripheral blood smear may show target cells with anisocytosis, poikilocytosis, nucleated red cell, reticulocytes and fragmented RBCs.

**Principles of treatment:**

1. **Packed red cell transfusion** :- Patients with β. Thalassemia major should be transfused with Packed RBC (double saline washed / leucoreduced) to maintain Hb around 10gm%.

2. **S-ferritin**:- Serum Ferritin is to be maintained below 1000ng/ml by regular iron Chelation. Iron chelation is to be initiated when Sr. Ferritin is >1000ng/ml or has already received more than 15 units of Red Cell Transfusion.
3. Folic acid supplementation 1-5mg/day
4. Bone Marrow Transplantation is a curative option.

**Indication of Packed Red Cell Transfusion:** Both the criteria should be fulfilled.
1. Confirmed diagnosis of thalassemia
2. Laboratory Criteria:
   a) Hb < 7gm/dl on 2 occasions, >2 weeks apart
   or
   b) Clinical Criteria (Hb>7gm/dl with any of the following)
      Facial changes / Poor growth/ Fractures/ Significant Hepatosplenomegaly

**Iron overload: Treatment by Iron Chelation.**

**Indications of Iron Chelation with Deferasirox:**

Age > 2 yrs
Sr. Ferritin>1000 ng/ml or >10-15 Units of Blood Transfusion

Deferasirox is given orally to Thalassemia major patients at a dose of 20-40 mg/kg/day in single dose in empty stomach with a glass of water or fruit juice to be continued to maintained the Sr. Ferritin around the target level.

**Prevention/ Patient Education:**
1. Carriage State diagnosis and marriage counselling: Young boys and girls should be educated to undergo blood testing to detect any abnormal haemoglobin gene (Thalassemia, Sickle Cell Anemia) by HPLC / Capillary Zone Electrophoresis. Carriers of any abnormal haemoglobin gene should be counselled to avoid marriage among themselves. Awareness programme: “Marriage after Hemoglobin matching instead of horoscope matching” should be taken up in the society.
2. Genetic counselling to parents. If both the husband and wife carry the abnormal haemoglobin gene, Beta globin gene mutation analysis of the foetus can be done from chorionic villous biopsy at 12-14 weeks of pregnancy at various centres like Kolkata, CMC Vellore, AIIMS, Delhi, Mumbai etc. If foetus is found to be homozygous state of thalassemia, the pregnancy should be terminated as per the rule: If found to be healthy or carrier state then it can be allowed to continue.

**Reference:**
BLOOD DISEASES

BLEEDING DISORDERS

INTRODUCTION:

Bleeding disorders is a challenging clinical situation encountered in many discipline. The common etiology may be due to abnormalities vascular and platelet disorders or coagulation disorders. Bleeding may ne life threatening and cause loss of life. A methodical approach integrating both clinical and laboratory parameters are essential for proper diagnosis and thus optimal management.

Causes of Bleeding disorders (Medical)

1. Low Platelet count (Thrombocytopenia)
2. Platelet function Disorders
3. Coagulation Disorders
4. DIC (Disseminated Intravascular Coagulation)
5. Systemic diseases like Liver / Kidney dysfunction etc.

Diagnostic Approach

Correlation of detail history, physical examination and laboratory parameters are essential to determine the cause of the bleeding. Bleeding since birth / similar history among siblings and bleeding into deep structure like muscle and joints indicate congenital coagulation disorders (Hemophilia) while bleeding in mucocutaneous region without other former parameters indicate platelet disorders and acquired etiology. General and various system evaluation will suggest the possibility of systemic diseases like Chronic liver disease and chronic renal failure.

Laboratory Investigation:

A) Hematological:

- Complete Blood Count, Peripheral Blood smear evaluation, PT, aPTT and their correction test, Serum Fibrinogen, Serum FDP (Fibrinogen Degradation products), D-Dimmer, Factor assays (VIII, IX, VII) etc., Plate function test.

- Bone Marrow Examination.

B) Others:

- Liver Function test, Blood Urea, Serum Creatine, test for collagen vascular disorders, etc.
Treatment:

*General measures*

No intramuscular injections; no aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs). Apply local pressure to stop bleeding.

**PLATELET DISORDERS (THROMBOCYTOPENIA)**

Low platelet counts suggest either reduced production or increased destruction of platelets or sequestration. In reduced platelet production, bone marrow examination shows reduced megakaryocytes and common causes are: megaloblastic anemia, aplastic anemia, marrow infiltration by malignancy. If bone marrow shows increased or normal megakaryocytic, it implies increased destruction as in idiopathic thrombocytopenic purpura (ITP), disseminated intravascular coagulation (DIC), hemolytic condition etc. Increased sequestration occurs in hyperplasia and infective conditions.

**Treatment**

Treat the primary cause.

Platelet transfusion is indicated mainly when platelets are not adequately produced and aim is to keep platelet count above 20,000/mm$^3$. If there is no fever and no clinical bleeding, even counts of 10,000/mm$^3$ are acceptable. Transfusing platelets in ITP is not useful as these are rapidly destroyed, but platelet concentrates may be transfused in life-threatening bleeding before specific therapy takes effect.

Platelets are never kept in refrigerator. They can be stored at room temperature (22°C) on an agitator for up to 3-5 days from the day of collection. Once issued they should be transfused rapidly.

Dose: one single donor platelet collected by aphaeresis raises platelet counts by 30,000 to 50,000 in an adult. One random donor platelet collected from a single unit of donated blood raises platelet counts by 5000-10000/mm$^3$ in an adult. The rise is less in DIC, ITP, febrile patients and during active bleeding.

**Immune Thrombocytopenic Purpura (ITP)**

**Treatment**

1. Tab. Prednisolone 1mg/kg/day for 4 weeks and taper off slowly by next 4 weeks.
   Or,
2. IV anti-D : 50-75 µgm / Kg slow IV infusion once
   Or,
3. Inj. Immunoglobulin: 1gm/kg/day for 1 day.

Failure with one therapy may still be followed by response to other therapy. If there is poor response to the above, case must be referred to specialist centre for consideration of 2nd line or reserved drugs or splenectomy.
PLATELET FUNCTION EFFECTS

If clinical presentation suggests platelet defect but platelet counts are normal, suspect platelet dysfunction. Bleeding time is prolonged in most cases. Platelet function tests will confirm the defect but facilities are not routinely available. It could be due to congenital or acquired reasons including drugs like aspirin and NSAID use.

Treatment

To stop NSAID use
If active bleeding occurs, transfuse platelets.

Hemophilia-A

Treatment

1. Factor VIII concentrate: 20 units/Kg, Slow IV push 12 hrly for 3-5 days followed by prophylaxis @ 10 units/Kg slow IV push twice a week (At the interval of 3-4 days) for prolonged period.
2. Tranexamic Acid: 250-500 mg 6 hrly for 1-2 weeks
3. Rest and ice fomentation to the local part

Hemophilia-B

Treatment

1. Factor VIII concentrate: 40 units/Kg, Slow IV push once daily for 3-5 days followed by prophylaxis @ 10 units/Kg slow IV push twice a week (At the interval of 3-4 days) for prolonged period.
2. Tranexamic Acid: 250-500 mg 6 hrly for 1-2 weeks
3. Rest and ice fomentation to the local part

DIC (Disseminated Intravascular Coagulation)

Diagnosis: Presence of primary etiology like trauma, infection, still birth, embolism etc., bleeding manifestations with deranged hematological parameters like low platelet count, Serum Fibrinogen, Prolong PT/aPTT and positive FDP/D-Dimer

Treatment:

1. Treat the underlying cause.
2. If cause is not clear, give Vitamin K and fresh frozen plasma (FFP) depending on investigation. FFP: 20 ml/Kg by continuous IV Infusion. Evaluation should be done at the end of 6-8 hrs followed by administration of FFP as per the coagulation report.
3. For overdose of oral anticoagulants or Vitamin K deficiency.
   - Inj. Vitamin K intravenous (use IV preparation) 10mg IV once daily for 3 days or till response.
   - Cryoprecipitate contains factor VIII and Fibrinogen and may be used in Haemophilia and in DIC and Hemophilia-A as replacement therapy (when Factor VIII concentrates are not available).
6. OTORHINOLARYNGOLOGY
ACUTE SUPPURATIVE OTITIS MEDIA

- ASOM is caused by inflammation of the mucous membrane lining the middle ear cleft, produced by pus forming organisms.

Diagnostic Criteria
- Severe throbbing pain in the ear, difficulty in hearing and rarely giddiness & excessive crying in children
- Often bilateral in children, preceded by URTI

STAGES
- **STAGE I** - stage of tubal occlusion
  Eustachian tube blockage, sensation of fullness in ear, otalgia and congestion
- **STAGE II** - Stage of exudation
  Marked by collection of exudate in tympanic cavity
  Pain in ear, deafness, fever, gross congestion of TM
- **STAGE III** - Stage of suppuration
  Perforation of TM, severe otalgia

Laboratory investigations
- Culture of the aural swab

TREATMENT

Non Pharmacological
- Steam inhalation and to keep the ear clean in case of pus discharge

Pharmacological
- Cap Amoxycillin 40mg/kg/day TDS for 7-10 days or
- Cap. amoxycillin 250-500mg + clavulanic acid 125mg TDS for 7 days
- Or Cefpodoxime or cefuroxime for 7-10 days
- Switch to tab. ciprofloxacin 250-500mg BD for 7 days, if no clinical improvement by 3rd day
- Xylometazoline HCl 0.1% 1-2 nasal drops in each nostril 1-2 times daily - Children 0.05%
- Oxymetazoline 0.05% 1-2 drops twice daily

Children 0.1% oxymetazoline drops
- T.Paracetamol 500mg SOS
- In case of ear discharge add ear drops Ciprofloxacin 2 drops thrice daily
- If symptoms of URTI, add T.Cetrizine 10mg daily
- OR Levocetrizine -5-10 mg od dose

Patient education
- Treat upper respiratory and sinus infection at the earliest
- Drops should be instilled after cleaning the ear meticulously
- Long term use of local antibiotic drops may cause sensitisation and otomycosis
- Burning sensation may occur with chloramphenicol drops
- Avoid entry of water into the ear

Referral criteria
- Patient not responding to treatment should be referred to the tertiary care hospital for surgical myringotomy.
CHRONIC SUPPURATIVE OTITIS MEDIA

- CSOM is characterized by the presence of perforation resulting from acute otitis media. It may present as an active disease when infection may occur through the nasopharynx or through the perforation, thus causing ear discharge. In the inactive disease, the only presenting feature is deafness.

**Diagnostic features**
- Copious mucous or mucopurulent discharge, more during attack of cold
- Foul smelling scanty discharge (unsafe type)
- Central perforation (in safe type)
- Marginal or attic perforation (in unsafe type)
- Deafness

**Laboratory tests**
- Aural swab for culture and sensitivity
- X-ray of mastoids
- Pure tone audiometry

**Nondrug**
- Aural toilet – suction cleaning and dry mopping

**Drug treatment**
- Ciprofloxacin ear drop 0.3% 2-3 drops thrice for 1 week +/- local steroids OR ofloxacin ear drops

Avoid ear drops containing
- Gentamycin
- Neomycin

In presence of URTI and external otitis:
- Cap Amoxycillin 500mg TDS for 7 days Child: 20-40mg/kg in 3 divided doses OR
- In case of severe infection T. Ciprofloxacin 250-500mg BD for 7 days

OR
- Cap Amoxycillin 500mg + clavulanic acid 125 mg TDS for 7 days
- Local nasal decongestants xylometazoline or oxymetazoline nasal drops
- Antihistaminic decongestants-Chlorpheneramine maleate/Cetirizine/Pseudoephedrine orally

**Patient Education**
- Not to allow water to enter ear
- Immediate treatment in case of upper respiratory infection
- Do not instill oil if there is discharge from the ear or if the patient is known to have a perforation of ear drum

**Referral Criteria**
- When ear is dry with central perforation – for tympanoplasty
- If ear is not dry inspite of treatment for further investigation
- In case of unsafe ear for treatment of cholesteatoma or mastoidectomy.

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OTITIS MEDIA WITH EFFUSION

It is characterised by presence of non purulent fluid in the middle ear cleft clinical features conductive type deafness otoscopy-retraction of drum,

- Air fluid level,
- air bubble

Treatment

- **Medical:**
  - Steam inhalation
  - In adults xylometazoline- HCL-0.1%-1-2 drops to be instilled 1-2 times a day.In children -0.05%
  - Ambroxol –in above five years of age-30 mg thrice daily.
  - Valsalva maneuver- in absence of throat infection
- **Surgical management**
  - In case of failure of medical treatment the patient may be referred to tertiary health centre for myringotomy, gromet insertion or adenoidectomy
- **Patient Education**
  - Avoid cold environment ,cold foods,

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WAX

- Wax is secretion of the ceruminous and pilosebaceous glands located in the cartilaginous portion of the external auditory canal.
- features pain, deafness, tinnitus, vertigo & reflex cough
- **Treatment**
  Pharmacological Pain- Ibuprofen 400mg sos, in children 20mg/kg/day
  Wax softener- 3-4 drops, 3-4 times daily for a week then removal of wax by syringing with sterile saline solution at body temperature pushed along the meatus to pull out the wax. Dry mopping.
  - Caution- if previous history of ear discharge or perforated drum, remove wax by instrument manipulation using probe, forceps & suction cleaning if available
- **Patient education**
  Cleaning the ear with buds to be avoided as it can push the wax to the deeper canal
- **Reference**

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OTOMYCOSIS

- It is the fungal infection of the external auditory canal seen more commonly in tropical and subtropical climates. Common fungus are Aspergillus niger and candida albicans

- **Diagnostic criteria**
  - itching with or without pain, greyish white fungal debris with or without black specks ear blockage

- **Treatment**
  - Non-pharmacological-
    - regular ear toilet by suction, dry mopping or instrumentation. No syringing with saline
    - Clotrimazole ear drops 3-4 drops three to four times daily for 7-14 days
  - Pharmacological-
    - topical clotrimazole 1%powder or liquid 3-4 times/day for at least a week after clinical resolution
    - Ibuprofen with or without paracetamol sos
    - Patient education
    - Ear to be kept dry; prevent entry of water into the ear
    - Do not plug the ear with cotton after instillation of eardrops or bathing

**Reference**

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COMMON COLD (CORYZA)

- This is one of the most common acute viral infection affecting upper respiratory tract

- **Diagnostic Criteria**
  - Rhinorrhoea, nasal obstruction, malaise, fever, sneezing

- **Treatment**
  - Non-pharmacological-
    1. steam inhalation via nose 2-3 times a day for 2-3 days
    2. Bed rest, home remedies (ginger,tulsi,honey)

  - Pharmacological
    1. Cetrizine dihydrochloride 10mg once daily/ 7 days, in children 5mg once daily
    2. OR Levocetirizine 5mg once daily
    3. In case of malaise and fever Paracetamol 650mg 3-4 times daily for 2-3 days or sos
    4. In children; 40-60mg/kg/day divided in 4 doses or 10mg/kg/dose sos
    5. If nasal obstruction or rhinorrhoea- saline nasal drops, 1-2 drops in each nostril 2-3 times daily
    6. If nasal obstruction is severe and in older children as short-term nasal decongestant; oxymetazoline HCl 0.05% nasal drops, 1-2 drops in each nostril 2 times a day for 2-3 days

  In children 0.025%, 1-2 drops, 2 times daily for 2-3 days

  **Patient education**
ALLERGIC RHINITIS

This is an IgE-mediated hypersensitivity of the mucous membrane of the nasal passage.

Salient features
1. Sneezing, itching, watery nasal discharge and a feeling of nasal obstruction
2. It may be associated with allergic conjunctivitis and bronchial asthma
3. It can be divided into the following types:
   - Seasonal allergic rhinitis (SAR/hay fever) sneezing, itching, watery, rhinorrhea and conjunctivitis are prominent symptoms
   - Perennial allergic rhinitis (PAR) this differs from SAR as this is due to long-standing nasal mucosal congestion. In this condition, nasal discharge is more visous or purulent. Usual symptoms are nasal blockage, postnasal discharge and hyposmia. Diagnosis can be made if patient is having 2 or more symptoms occurring for more than 1 hour on most days.

Treatment
- non-pharmacological- avoid allergens
- Pharmacological
  - Tab. Cetrizine 10-20mg in a single daily dose for 7 days, in children; 5mg in a single daily dose
  - Or tab levocetrizine 5mg once daily or tab fexofenadine 120-180mg daily for 7 days or tab chlorpheniramine maleate 4mg 6-8hourly for 7 days or montelukast 5-10mg once a day.
  - 2. If nasal obstruction or rhinorrhea- saline nasal drops, 1-2 drops in each nostril 2-3 times daily
  - 3. If nasal obstruction is severe and in older children as short-term nasal decongestant; oxymetazoline HCl 0.05% nasal drops, 1-2 drops in each nostril 2 times a day for 2-3 days
  - In children 0.025%, 1-2 drops, 2 times daily for 2-3 days
  - In case the signs symptoms are persistent:
    - Betamethasone nasal drops 2-3 drops 2-3 times a day or
    - Hydrocortisone nasal drops 2-3drops 2-3 times a day or

Reference

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✓ Beclomethasone inhaler (50mcg/puff) 2 puffs 12hourly or
✓ Budesonide(50-100mcg/puff) intranasal spray 1-2puffs a day
✓ Topical azelastine intranasal spray 2-3 times a day
✓ Fluticasone /mometsone nasal spray-twice for 14 days then once daily for thee months or more.

In case of no response to the treatment outlined above,
✓ Tab. Prednisolone 5-60mg/day in 3-4 divided doses for5-7 days Or
✓ Tab. Dexamethasone 0.5-5.0mg/day in 3-4 divided doses for 5-7 days
Or
✓ Tab. Betamethasone 0.5-5.0 mg/day in 3-4 divided doses for 5-7 days

Patient education
✓ It is due to hypersensitivity and there is no cure but symptoms can be controlled effectively by the judicious use of drugs and the patient can lead a normal life
✓ Medicated nasal drops should not be used for more than 7 days. Medicated nasal drops used for a longer period can cause rebound congestion and rhinitis medicamentosa
✓ Chlorpheniramine, pheniramine, etc can cause sedation , cognitive impairment. The patient should avoid tasks requiring alertness and skill.
✓ Systemic steroids should not be stopped abruptly. Dose to be tapered off before cessation of therapy.

References

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EPISTAXIS

• Epistaxis is bleeding from the nose. It is a symptom and not a disease. So the underlying cause is to be evaluated.
• Epistaxis can be classified into anterior or posterior according to location of bleeding.
• Anterior bleeding accounts for 90% of cases.
• Anterior bleeds are characterised by slow, persistent oozing whereas posterior tend to be more profuse carrying higher risk of airway compromise, aspiration of blood and life-threatening hemorrhage
• Causes of epistaxis include trauma, hypertension, bleeding disorders, nasal mass and acute inflammation

Diagnostic features
• Bleeding from nose
• Bleeding from other sites may be searched to exclude bleeding disorders
• Presence of pallor, tachycardia and hypotension indicate excessive blood loss

LABORATORY INVESTIGATIONS
• Complete hematological investigation including Hb%, DC, TLC, Platelet count, BT, CT, Prothrombin time
• Radiological examination of nose, nasopharynx and paranasal sinuses.

Treatment
• In cases of active epistaxis, check vitals and pinch the nose for 15 minutes nonstop and position the patient sitting upright and leaning forward, keep the head elevated but not hyper extended to avoid aspiration
• Position the patient in supine (or other) lateral position, if signs and symptoms of shock are present
• In epistaxis due to suspected head injury do not pinch the nose
• Application of ice on the bridge of nose cause reflex vasoconstriction and is helpful in traumatic bleeding
• Nasal packing with ribbon gauze soaked in liquid paraffin or any antibiotic (neosporin/soframycin) if persistent bleeding is present
• Cauterisation of bleeding point in nasal cavity by local touching with silver nitrate or trichloroacetic acid
• If bleeding is not controlled, arterial ligation, angiography and embolisation may be required.

Drug treatment
• Sedation – inj. Diazepam 5mg to alley patient’s anxiety
• Haemostatic – Inj. Ethamsylate 250mg IV/orally TDS till active bleeding subsides/trenaxamic acid-tab/injection
• Cap. Amoxycillin 500mg TDS to control infection due to nasal packing
• Replacement of blood loss by blood transfusion when indicated

Patient education
• Avoid nose picking
• Regular BP check up

Referral Criteria
• Patient with excessive blood loss
• Uncontrolled persistent epistaxis
• Bleeding disorders
• Treatment of underlying cause
• Sinusitis, hypertension, trauma, tumour treated with appropriate drugs

RECURRENT EPISTAXIS
• Instructions to minimise the recurrence of bleeding, including limiting local trauma, (no nose blowing or picking)
• Applying nasal hydration with saline mist or ointments, increasing ambient humidity with a cool mist vapouriser to reduce mucosal irritation

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ACUTE RHINO-SINUSITIS

DIAGNOSTIC FEATURES

- Inflammation of the paranasal sinuses
- Symptoms of common cold that persists for more than 1 week like purulent nasal discharge, headache, postnasal drip & facial pain (in acute cases)
- Tenderness over concerned paranasal sinuses
- Pus in middle meatus suggest maxillary sinusitis
- Trans illumination test-shows reduced light transmission through frontal & maxillary sinuses
- Recurrent acute sinusitis-recurrent episodes each lasting <30 days, separated by asymptomatic interval of at least 10 days
- Sub acute sinusitis-signs and symptoms lasting between 30-90 days
- Chronic sinusitis-low grade persistence of signs and/or symptoms longer than 90 days without improvement

INVESTIGATIONS

- X-ray PNS – haziness compared to contralateral side or orbital shadow
- treatment

Non Pharmacological

- Steam inhalation

Pharmacological

- Cap. Amoxycillin 500mg TDS for 10 days (children-20-40mg/day in 3 divided doses)
- Levofoxacin(500) OD for 10 days days (not in children)
- Ciprofloxacin 250-500mg BD for 5-7 days (not in children)
- If no response in 3-5 days/worsens 48-72 hrs after starting treatment – cap Amoxycillin 500mg + clavulanic acid 125mg TDS for >7 days

IN SINUSITIS OF DENTAL ORIGIN

- Cap. Amoxycillin 500mg TDS for 10 days
- Amoxycillin 500mg TDS + Metronidazole 7.5mg/kg TDS for 10 days.
- OR Ofloxacin200mg+Ornidazole 500mg BD for 10 days

FOR SYMPTOMATIC RELIEF

- Paracetamol 500mg TDS(10mg/kg in children)
- OR Ibuprofen 400-600mg TDS
- Bromhexine 8mg/Ambroxol 30mg TDS for 7 days

ADJUNCTIVE THERAPY FOR DRAINAGE

- Topical decongestants i.e, Oxymetazoline HCl 0.05% (0.025% in children)
- OR Xylometazoline -0.1% (in child 0.05%) nasal drops 1-2 drops in each nostril 2-3 times a day
- Antihistamine-Chlorpheniramine maleate 4 mg OD
• OR Cetirizine 10mg /Desloratidine/Fexofenadine 120 mg od dose.
• Patient education
• To complete the course of antibiotics for eradication of the bacteria from the sinuses, to avoid development of resistance
• Topical decongestant use for not more than 1 week to be avoided to prevent development of atrophic rhinitis and rhinitis medicamentosa.

**Referral Criteria**
- Not relieved symptomatically after 48-72 hrs. of antimicrobial therapy should be sent to Otorhinolaryngologist
- CNS complications of sinusitis

**References:**

**ACUTE TONSILITIS**
Inflammation of the palatine tonsil & pharyngeal mucosa, mostly bacterial in origin, streptococci being the most common organism

**Diagnostic features**
- Sore throat associated with fever & malaise
- Pain on throat aggravated by swallowing
- Hawking-tendency to clear throat
- Diffuse congestion of the posterior wall & tonsils (anterior pillar)
- Tonsils may or may not be enlarged
- Tender jugulo-digastric nodes

**Laboratory tests**
- Blood count
- Throat swab for culture & sensitivity test
- ASO titre

**Treatment**

**Non – Pharmacological**
- Warm saline gargle
- Proper oral hygiene
- Sufficient fluid intake
- rest

**Pharmacological**
- **Cap. Amoxycillin** 500mg TDS for 7 days (In children 50mg/kg/day in 3 divided doses)
- Cap. Amoxycillin 250/500mg + clavulanic acid 125mg TDS in severe infection for 5-7 days. **T. Roxythromycin/Azithromycin** 150 mg BD for 5-7 days, azithromycin 12mg/kg once a day (max. 500mg), (in children azithromycin 50mg/kg/day in 3 divided doses)
- **T. paracetamol** 8-10mg /kg 6-8th hourly and then SOS
• **Penicillin**—oral/parenteral given for 6-12 months if ASO titre is positive

**Patient education**

• Avoid exposure to cold
• Avoid cold drinks
• referral Criteria
• If there is peritonsillar abscess(trismus & dysphagia) or peritonsilitis, the patient should be referred for drainage under supervision of ENT surgeon.
• If symptoms persist or recurs with 3-4 attacks/year inspite of antibiotic treatment the patient may be referred for tonsillectomy/adenoidectomy
• (in children)

**References:**


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7. GENERAL SURGERY
WOUNDS

A breach in continuity of lining epithelium or skin.

CLASSIFICATION
1) Clean wound
2) Clean contaminated wound
3) Contaminated wound
4) Dirty wound

1. CLEAN WOUND

DIAGNOSTIC CRITERIA:
Wound looks healthy without contaminants like mud, grease etc. / incised wounds
Pain at the site of injury, oedema

LABORATORY TEST:
Hb, TLC, DC, FBS/RBS, blood Urea, serum Creatinine, BT, CT

IMMEDIATE CARE OF THE WOUND:
Wound should be washed with normal saline
Antiseptic lotion should be applied (Povidone iodine 10%)
Primary closure of the wound with non-absorbable monofilament suture like Polyamide/nylon in superficial wounds with or without drain.
In deep wounds layered closure to be done. Suture (muscle/fascia) with chromic catgut/ polyglactin 910 (vicryl) and skin with monofilament non absorbable sutures like polyamide/nylon/stapler.

DRUG THERAPY
Tetanus toxoid injection 1 ampule intramuscular and human tetanus immunoglobulin 500 IU intramuscular/250 IU injection in children Human upto 14 year one dose

ANTIBIOTICS
Tab. Ciprofloxacin 500 mg BDPC or Tab. Cefadroxyl 500 mg BD PC or
Tab. Cefexime 200mg BD PC for 5-7 days

ANALGESICS
Tab Acelofenac 100 mg BD PC or Tramadol 50/100mg BD after food for 2-3 days

Wound inspection to be done every alternate days.
Sutures can be removed after 7-10 days depending on the location of wound in relation to body part.

REFERRAL CRITERIA
1. Inability to achieve haemostasis/ Bleeding disorders
2. Suspected major vessel/nerve injuries in deep wounds
3. If the wounds fail to heal by above treatment within 7-14 days.
4. Suspected internal injuries.

2. CONTAMINATED WOUND:

**DIAGNOSTIC CRITERIA:**
Visible external injury contaminated with mud, dust, oil, grease and other organic or environmental contaminants/ Foreign body.

**LABORATORY TEST**
Hb TLC, DC, FBS/RBS, BT, CT

**IMMEDIATE PRIMARY CARE**
- Wash the wound with plain water or normal saline to flush out all foreign bodies.
- Apply antiseptic lotion (Povidone iodine 10%)

Grossly lacerated tissue and devitalized tissue to be excised out.
Wound should be kept open.
- Grossly contaminated wounds may be closed by delayed primary suture after 7-10 days basing on the condition of the wound.

**DRUG THERAPY**
Tab. Ciprofloxacin 500 mg BD PC or Tab. Cefixime 200 mg BD PC with Tab. Linezolid 600 mg BD PC for 5-7 days.
Tab. Metronidazole 400 mg TDS after food for 5 days.
- In severely contaminated wound: Inj. Cefotaxime 1 gm IM/IV BD 5 days, in children: Inj. Cefotaxime 50-100 mg/kg/day may be given.
Tab. Aceclofenac 100 mg BD PC/ Paracetamol 650 mg BD PC/ tramadol (50/100 mg) BD PC for 3-5 days.

**REFERRAL CRITERIA**
Large tissue defect requiring secondary surgical closure or skin grafting should be referred to Tertiary centres.

**CELLULITIS**
A spreading inflammation of skin and subcutaneous tissue.

**DIAGNOSTIC FEATURES:**
- A brawny red or reddish brown shining area of edematous skin, Pain in the affected part with or without constitutional symptoms.

**LABORATORY TEST:**
- Hb, TLC, DC, Urine analysis, FBS/RBS, BT, CT
**SUPPORTIVE TREATMENT:**
Rest to the affected part of the body. Elevation of the affected part.

**DRUG THERAPY:**
- Inj Cefotaxime 1 gm iv BD/INJ Piperacillin and Tazobactum 4.5 gm iv 8 hourly in severe cases
- Tab. Cefadroxyl 500 mg or Cefixime 200 mg or linezolid 600 mg BD after food for 5-7 days.

If no response within 24 hours, a hidden abscess should be searched for and drained.
- Tab. Aceclofenac 100 mg BD PC/tab Paracetamol 650 mg after food for 2-3 days for relief of pain & inflammation and indomethacin 75 mg im/tramadol 100 mg/50 mg im in severe pain with PPI injection once a day.

**PATIENT EDUCATION**
Restrict mobility of affected part and limb affected can be kept elevated.

**REFERRAL CRITERIA**
- Presence of rapidly progressive cellulitis with patches of skin necrosis in any part
- Cellulitis in a Diabetic patient.
- Suspected Gangrene and progressing to septicaemia.

**ABSCESS**
- Localised collection of pus in a cavity lined by granulation tissue, covered by pyogenic membrane with inflammatory features over it.

**DIAGNOSTIC CRITERIA:**
- Painful swelling over that part of body, Redness.
- Increased temperature over swelling.
- Fluctuation test positive.
- Impairment of function of the particular part of the body.
- Rule out cold abscess by absence of local inflammatory features.
- Diagnostic confirmation by aspiration by wide bore (18 G) needle at the most fluctuant site.

**LABORATORY TEST:**
- Hb, TLC, DC, FBS/RBS, BT, CT
- Culture and sensitivity of pus

**SURGICAL TREATMENT**
**INCISION AND DRAINAGE**
- Cleaning the abscess site with povidone iodine (10%) and shaving off hairs (if needed) Infiltration of local anaesthesia after skin test. (0.5% plain xylocaine)
- Liberal incision is given on the most dependent part or fluctuant part of abscess.
All loculi and septa are broken by finger gently or by sinus forceps to drain all pus. Abscess cavity to be packed with gauze soaked with povidone iodine 10% lotion and dressing done. Removal of pack after 48 hours.

**DRUG THERAPY**
Features of persistent inflammation may need antibiotics at least for 5-7 days.

**ANTIBIOTICS**
Tab. Cefadroxil 500 mg BD or ciprofloxacin 500mg BD after food/ AmoxyClav 625 mg.

**ANALGESICS**
Tab Aceclofenac (100mg) BD PC/ Tab. Paracetamol 650 mg BD PC for 3 days.

**PATIENT EDUCATION**
Daily dressing of abscess cavity with normal saline and surrounding skin povidone iodine 10% lotion.
Maintain cleanliness of the part

**REFERRAL CRITERIA**
Deep abscess involving vital structures of body like neck, axilla, Cubital fossa, Popliteal fossa, Groin.
Abscess which requires drainage under anesthesia like ischiorectal abscess, breast abscess.

**HEMORRHOIDS**
Piles are dilated veins occurring in relation to the anus cushions.

**TYPES**
1. Internal haemorrhoids (Painless)
2. External haemorrhoids (Painful)
3. Intro-external haemorrhoids (Painless)

**DIAGNOSTIC FEATURES:**
Bright red painless bleeding (splash in the pan)
Mucus discharge, pruritus
Pain during prolapse of piles/ infection/ spasm.
Per rectal examination - Thrombosed Piles are only felt, normal piles not felt.
On Proctoscopy - Just below the anorectal ring internal haemorrhoids bulge into lumen at 3, 7, 11 o clock position (primary piles) and secondary in between.

**NON-DRUG TREATMENT:**
Sitz bath

**DRUG TREATMENT:**
Laxatives /bulk formers
Management of associate anaemia if present orally.
Antibiotics in cases of thrombosed haemorrhoids
Tab tranexamic acid 500mg TID If there is active bleeding
Calcium dobesilate 500 mg BD for 2 weeks
Calcium dobesilate cream to apply intra-anally thrice daily.

**PATIENT EDUCATION**
- Regular bowel habit, sitz bath, Plenty of water, fibres like fruits and vegetables.
- Exercise, Avoid straining at defecation.

**REFERRAL CRITERIA**
- Patients with symptomatic active bleeding with not responding to conservative management/prolapsed piles suspected colo-rectal malignancy.

**ANAL FISSURES**
(FISSURE-IN-ANO)
A longitudinal ulcer in the long axis of lower anal canal, not beyond dentate line.

**DIAGNOSTIC CRITERIA:**
- Constipation with pain during and after defecation.
- Mucus discharge with itching.
- Bright red blood stricking stool.
- SIGNS- a sentinel tag along ith sphincter spasm may be present in long standing cases.
- Tightly closed puckered anus is pathognomonic feature.
- Per rectal examination- is not done usually due to intense pain.

**NON-DRUG TREATMENT:**
- Sitz Bath

**DRUG TREATMENT:**
- Laxatives and stool softeners at bed time.
- Xylocaine 5% ointment for intra anal application before and after defecation / 0.2% glyceryltrinitrate 4 times daily applied into the anus.(may cause headache)

**PATIENT EDUCATION**
- intake of bulk forming diet with plenty of water.
- Avoid spicy/ non vegetarian food.

**REFERRAL CRITERIA:**
- Refer the patients to higher centre for surgery (only in chronic cases).
FISTULA-IN-ANO

An abnormal tract connecting between two epithelial surfaces and is lined by granulation tissue or epithelium. Low level fistula opens into the anal canal below the anorectal ring. High level fistula opens into the anal canal at or above the level of anorectal ring. At times it can be multiple and complex.

DIAGNOSTIC FEATURES:
- Persistent seropurulent discharge may be blood stained, staining the under garment.
- Pain or sometimes history of an abscess that has been drained improperly.
- May be associated with TB, Crohn’s disease or carcinoma, actinomycosis.
- Per rectal examination- Internal opening can be palpated inside anus, if of low variety.

NON-DRUG TREATMENT
- Sitz bath
- Diet modification to avoid constipation
- Local hygiene

DRUG TREATMENT
- Laxatives and bulk forming laxatives
- Tab Ciprofloxacin 500mg or Tab ofloxacin 400mg twice daily for 5-7 days if infected.

PATIENT EDUCATION
- Intake of bulk forming diet
- Avoid spicy/non veg food.
- Don’t take treatment for anal disorders like abscess and fistula from unqualified persons.

REFERRAL CRITERIA
- Refer patients with high or multiple fistulae to higher centre for surgery.

ACUTE ABDOMEN

INTRODUCTION
Acute abdominal pain can occur due to variety of medical and surgical causes. It is important to elicit a detailed clinical history and perform abdominal examination to determine the cause of pain and exclude non surgical causes of referred pain to abdomen. In very severe cases, it may be necessary to give treatment before proper history can be obtained or examination is allowed by the patient.

CAUSES OF ACUTE ABDOMEN

ABDOMINAL CAUSES
1. Inflammation of peritoneum due to bacterial or chemical contamination. Perforation of appendix or bowel, ulcer, pancreatitis or pelvic inflammatory disease.
2. Mechanical obstruction of hollow viscera-intestinal obstruction, ureteric obstruction due to stone or other causes, and obstruction of the biliary tree with ascending cholangitis.


4. Abdominal wall injury or infection in the muscles. Referred pain from pneumonia, angina, spine radiculitis and torsion of testis should be excluded.

**METABOLIC CAUSES**

- lead poisoning, uraemia, diabeticketoacidosis, porphyria and other food allergy causes.

**APPROACH TO THE PATIENT WITH ACUTE ABDOMEN**

1. In view of the variety of causes, it is absolutely essential to elicit an orderly detailed history about the nature of pain and perform a sequential examination. Narcotics or analgesics should be avoided till the diagnosis is made.

2. A proper history can provide more valuable information than the investigations. The features of the history that must be elicited are site, time and nature of onset, severity, nature of pain, progression, ration, relieving factors, exacerbating factors and radiation or referral of pain, history of complaints like vomiting, constipation, haematemesis and melena and urinary complaints Should be obtained.

3. Examination of the patient with respect to facies, posture in the bed, respiration can help in localizing the cause and nature of pain. The abdominal examination should be thorough but extremely gentle starting from the nontender area. Points to be noted are area of maximum tenderness, guarding, rebound tenderness, bowel sounds, obliteration of liver dullness and presence of any associated mass or free fluid in the abdomen.

4. It is important not to miss examination of left Supraclavicular lymphnodes, hernial orifices, peripheral pulsations, genitalia, bowel sounds and rectal and/or vaginal examination.

5. Laboratory investigations depend upon the suspected cause in relation to the quadrant of maximum tenderness and include haemogram, kidney function tests, liver Function tests, serum amylase and lipase, blood sugar and urinalysis. Special analysis like serum lead estimations function tests. serum may be needed, if deemed necessary.

6. Radiological investigations include plain radiograph of abdomen, supine and erect and chest. In a sick patient left lateral decubitus X-ray may provide the same information.

**CONSERVATIVE TREATMENT:**

(only for 24 hours of initial complaint except in case of strangulated hernia)

1. IV fluids preferably RL, DNS etc.

2. Ryle’s tube aspiration 8th hourly.

3. 3rd generation antibiotics like inj. Ceftriaxone 1gm IV BD with Inj. Metronidazole 500mg IV TID

4. Suitable analgesics like NON-OPIATE analgesics, Inj. Aceclofenac 100mg IV BD, Inj. Diclofenac 75mg IV SOS
5. Inj Pantoprazole 40 mg IV OD.

**LABORATORY TEST:** (to be done after resuscitative method)

- Hb, TLC, DC, FBS/RBS, Blood urea, serum creatinine, serum calcium, LFT, serum amylase and serum lipase, blood grouping and cross matching, urine routine and microscopy.
- X-ray chest PA view and abdomen in erect posture (gas under right diaphragm or multiple air fluid levels)

**PATIENT EDUCATION:**
1. Do not administer analgesics for acute abdomen on your own. This may mask surgical signs.
2. Enema should be administered before diagnosis.
4. Workout to decide which case requires urgent surgical intervention.

**REFERRAL CRITERIA:**
1. If patient’s pain doesn’t subside within 24 hours of onset of abdominal pain with conservative treatment or worsens.
2. Non availability of anaesthesia facility.
3. Non availability of blood transfusion facility.
4. Doubt regarding the pathology.
5. Known case of Sickle cell disease with abdominal crisis.
6. All case of peptic perforation, Sigmoid volvulus and strangulated hernia.

**POLY-TRAUMA**

- Life threatening and serious injuries that require specialized surgical care for survival of the patient and prevention of disability. It may occur due to road traffic accidents, fall from height, penetrating injuries, blast injuries.

**DIAGNOSTIC FEATURES:**

Presence of obvious visible injuries fractures/bleeding.
- Loss of consciousness.
- Vitals: Glasgow coma score: Less than 13
- Systolic BP less than: Less than 90mm Hg
- Respiratory rate less than 10 per minute or more than 20/min
- Injury to spine may be present.

**LABORATORY TEST –**

- Hb, TLC, DC, Blood Grouping and typing
- X-ray-Lateral cervical spine, AP chest, Straight X-ray Abdomen, Pelvis AP & Lateral

**Protocol of preferential systemic exam in hospital:**

1. Respiratory system
Standard Treatment Guidelines of Odisha 2018

2. CNS
3. ABDOMEN and CVS
4. Musculo-skeletal system

A) If the victim is having difficulty in breathing - Airway management Is required. Clear the airway, Oxygen inhalation for all patients.
B) If pulse or heart beat not palpable - begin closed chest compression.
C) If there is gross external bleeding - elevate the part and apply external pressure bandage.
D) If injury to neck is suspected - protect the neck and spine by splintage before transporting the patient.
E) IV fluids with 2 wide bore canulae (16G or 18G) F) Look for level of consciousness with AVPU method. (A-alert, V- Voice Response, P- Pain Response, U- Unresponsive)
G) Exposure of the patient done by removing all the clothing.
H) Put Ryle’s tube and Foley’s catheter.
I) Splintage of fractured bone or joints.

DRUG TREATMENT

Diclofenac sodium Tab 50mg thrice daily/Inj.diclofenac 75mg BD or thrice daily (preferable)
Inj Cefotaxime 500mg/1 gm twice a day for 5-7 days
Inj Gentamicin 80 mg 12th hourly for 5-7 days
Tab Metronidazole 400mg 8th hourly/ Inj 100 m1 8th hourly for 5-7 days

REFERRAL CRITERIA

All patients with Multiple trauma
Before referral to tertiary centre external bleeding must be controlled either by local pressure or suture.
History of anticoagulation therapy or bleeding diathesis.

ACUTE APPENDICITIS

Appendicitis is one of the commonest cause of acute abdomen and may appears catarrhal appendicitis or as obstructive appendicitis and sometimes it may present as an Appendicular lump or Appendicular abscess or as burst appendix with peritonitis.

DIAGNOSTIC CRITERIA

Acute peri-umbilical pain with nausea and or vomiting and mild fever
Shifting of pain to the right lower abdomen after a 6-12 hours in obstructive type.
Maximum tenderness at the McBurney’s point
Guarding in right iliac fossa and Rebound tenderness present.
An inflammatory tender lump in right iliac fossa or signs of peritonitis in an abscess.
LABORATORY TEST
HB, DC, TLC (Poly morphonuclear Leukocytosis), BT, CT
Ultrasound of the abdomen
Straight x ray of abdomen in erect posture.
Urine Routine & Microscopy

CONSERVATIVE TREATMENT
Nil orally
IV Fluids and catheterization
inj Ceftriaxone Ig IV 12th hourly
inj Gentamicin 80mg or Inj. Amikacin 500mg BD and Inj. Metronidazole 100ml IV 8 hourly
Inj. Diclofenac I amoule IM intragluteal SOS

DEFINITIVE TREATMENT
Emergency Appendectomy, provided there is no appendicular lump formation or patient comes within 72 hrs of pain onset.

PATIENT EDUCATION
Normal routine activity after 15 days of emergency appendectomy and 7 days after laparoscopic appendectomy.

REFERRAL CRITERIA:
1. Suspected acute appendicitis with other associated co-morbid conditions.
2. Non availability of anesthesia facility.
3. Any appendicular lump, not resolving with antibiotics after 2 weeks. (no decrease in size of the mass).

FURTHER READINGS:
8. ORTHOPEDICS
LOW BACK PAIN

INTRODUCTION:

Pain over low back region interfering with normal day to day activities associated with or without radicular pain.

DIAGNOSTIC CRITERIA

- Usually there is Pain over low back associated with stiffness
- Tenderness over affected area
- Movement is painful and restricted with muscle spasm
- Straight leg raising test (SLR) may be restricted
- Neurological evaluation of nerve root affection is essential

LABORATORY INVESTIGATIONS:

- X-Ray of affected spine (Lumbosacral/dorsolumbar spine).
- DC, TLC, HB, ESR, FBS, Serum uric acid, S Creatinine
- Urine routine and microscopy.

NON-PHARMACOLOGICAL TREATMENTS:

- Bed rest
- Hot fomentation
- L.S Belt or corset
- Proper postural training
- Physiotherapy

PHARMACOLOGICAL TREATMENTS:

- Tab. Pantoprazole 40mg. Tab. Rabeprazole 20mg.
- Analgesics
  - Tab. Diclofenac 50mg. 1 Tab. thrice daily after food.
  - Tab. Aclophenac and Paracetamol 1 Tab. twice daily after food.
  - Tab. Tramadol 50mg. 1 Tab. thrice daily after food.
  - Tab. Thiocolchicosid 4mg. 1 Tab. twice daily after food.

PATIENT EDUCATION:

- Adoption of proper posture
- Sleep on firm bed
- Regular Back Exercise
REFERRAL CRITERIA:

- Back pain not responding to conservative treatment
- Appearance of neurological symptoms like weakness of great toe extension and flexion, foot drop, exaggerated jerks or absence of jerks
- Associated Fever, weight loss & Trauma

OSTEOARTHRITIS

INTRODUCTION:

Chronic degenerative joint disorder in elderly individuals in which there is progressive softening and degeneration of articular cartilage with associated bony changes

DIAGNOSTIC CRITERIA:

- Pain and swelling over joint
- Stiffness or crepitis of the joint
- Difficulty in climbing stairs, sitting crossed legged, squatting, walking.
- Deformity – varus or valgus

LABORATORY INVESTIGATIONS:

X-Ray of affected part – Hip, Knee (AP/Lat)

- Joint space narrowing
- Peripheral osteophytes, loose bodies.
- Cysts in apposing articular surfaces.
- Deformity

Blood test

- FBS, ESR, Serum Uric Acid, RA Factor.

NON-PHARMACOLOGICAL TREATMENT:

- Weight reduction
- Hinged knee cap
- Quadriceps strengthening exercises
- Use of Western Toilets, Assistance device like crutches or walkers.
- Hot fomentation

PHARMACOLOGICAL TREATMENT:

1. Analgesics

   - Tab. Paracetamol 650 mg./1000mg. 1 Tab. Thrice daily after food for 10 to 15 days.
➢ Tab. Diclofenac 50mg. 1 Tab. Thrice daily after food for 10 to 15 days.
➢ Tab. Tramadol 50mg. 1 Tab. 2 to 3 times daily after food for 10 to 15 days.
2. Local application of analgesic gel with hot formation.
3. Intra articular steroid injection of methylprednisolone 40mg. or Triamcinolone 40mg. under strict aseptic measures if not responding to NSAIDs. (Maximum of 3 injection in a year)

PATIENT EDUCATION:
➢ No curative pharmacological agent as the disease is progressive in nature. The disease is due to ageing & aggravated by obesity & life style.
➢ To avoid and reduce activities which exaggerated pain.
➢ Take minimal medication which provides symptomatic relief.
➢ Physiotherapy and Muscle strengthening exercise.

REFERRAL CRITERIA:
➢ Grossly deformed and unstable joint.
➢ Severe pain hampering with day to day activities.
➢ Gross swelling and recurrent effusion of affected joint.
➢ Not responding to pharmacological and non pharmacological measures.

OSTEOMYLITIS

INTRODUCTION:
Acute Pyogenic Osteomyelitis is an acute infection or inflammation of bone and bone marrow commonly seen in children’s.

DIAGNOSTIC CRITERIA:
➢ Pain and swelling over affected bone.
➢ Fever
➢ Loss of function of affected limb
➢ Local rise of temperature.

LABORATORY INVESTIGATIONS.
➢ Blood- DC, TLC, ESR, CRP, (Acute Phase reactant rises) Blood Culture & Sensitivity if facility available
➢ Urine routine and microscopy.
➢ X-Ray of affected part (X-Ray changes usually appeared 7 to 10 days after onset of diseases).
  • Soft tissue swelling.
  • Periosteal reaction.
  • Lytic aea in metaphysis of bone.
NON-PHARMACOLOGICAL TREATMENT:
- Splintage of affected area by Plaster/Slab/Traction
- Elevation of limb.

PHARMACOLOGICAL TREATMENT:
- IV Fluids to correct dehydration.
- Tab. Paracetamol 15mg. /kg. 4 times per day.
- Broad spectrum antibiotics like Inj. Ceftriaxone 100mg. /kg. Per day in 2 divided dose, Inj. Cefotaxim 100-200 mg. /kg. / Per day in 2 to 4 divided dose.

PATIENT EDUCATION:
- Hot fomentation and massages to be avoided.
- Not to make the child walk on the affected limb.
- As it is an acute emergency seeking early medical intervention is of prime importance.

REFERRAL CRITERIA:
- Acute symptoms like Fever, Pain and Joint swelling not subsiding in 48 hours of treatment.
- Pus pointing or appearance of discharging sinus.

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SEPTIC ARTHRITIS

INTRODUCTION:
Septic arthritis is acute inflammation of joint usually seen in children less than 10 years. It is an acute emergency.

DIAGNOSTIC CRITERIA:
- Throbbing pain , Swelling and redness of affected joint
- High grade fever and malaise
- Gross restriction of movement of affected limb around the joint

LABORATORY INVESTIGATIONS:
- Blood – DC, TLC, ESR, CRP
- X-Ray of the affected joint (showing increased soft tissue shadow).
- Aspiration of affected joint under strict aseptic measures. Gram staining and culture sensitivity of the aspirate to be done.

NON-PHARMACOLOGICAL TREATMENT:
- Splintage with plaster slab or splint.

PHARMACOLOGICAL TREATMENT:
- IV fluids to correct dehydration.
Broad spectrum antibiotics like Inj. Ceftriaxone 100mg./kg. Per day in 2 divided dose, Inj. Cefotaxim 100-200 mg./kg./ per day in 2 to 4 divided dose.

Analgesics
- Tab. Paracetamol 625mg./1000mg. thrice daily after food
- Tab. Diclofenac 50 mg. thrice daily after food.
- Tab. Acclophenac and Paracetamol 1 Tab. twice daily after food.

PATIENT EDUCATION:
- As it an acute emergency seeking early medical intervention is of prime importance.
- Avoidance of Hot fomentation and massaging of the joint.
- Prolonged drug intervention is to be avoided and seeking early surgical intervention is preferable.

REFERRAL CRITERIA:
- Purulent joint aspirate.
- Acute symptoms like Fever, Pain and Joint swelling not subsiding in 48 hours of treatment.
- Very sick child.

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9. PULMONARY MEDICINE
**BRONCHIECTASIS**

Bronchiectasis is defined as permanent abnormal dilatation of bronchi due to destruction of ciliated epithelium, elastic and muscular tissue. The destruction process may be initiated by primary microbial infection (tuberculosis, necrotizing pneumonia, aspergillosis etc) or obstruction (foreign body, tumour, lymph node, etc.) or congenital defect of large airways or due to underline immune deficiency state or underline connective tissue disorder (rheumatoid arthritis) resulting inadequate clearance and pooling of secretion and secondary infection. It is synonymous with non-CF bronchiectasis.

**Clinical features**

Majority of patient presented with chronic persistent productive cough, increasing volume, change of colour of sputum & purulence due to recurrent infection. Next common symptoms are haemoptysis, breathlessness & pleuritic chest pain. Patient may presented with symptoms due complication (cor-pulmonale or respiratory failure) like swelling of feet, bluish coloration of skin, breathlessness. Signs like clubbing of fingers, presence of course, fixed, leathery crepitation are the hallmark of the disease.

**Investigation**

X-ray chest may be normal. Finding like tram track, hyperinflation/air-trapping, multiple ring like shadow may be seen in x-ray. Increased TLC count & presence of fever are the evidence of active infection.

**Management**

Objective of management to relief symptoms, prevent complication, prevent exacerbation & reduction of mortality.

**Non-Pharmacological**

Airway clearance by postural drainage, chest physiotherapy which enhance gravity aided clearance of secretion.

**Pharmacological**

a) **Antibiotics:** These are to be used for the treatment of exacerbation or for any evidence of infection (pyrexia, purulent sputum, raised TLC & raised neutrophils).

- Amoxicillin 500 mg with/without clavulanic acid 125mg three times a day for 7-10days.
- Or Cotrimaxazole (SMZ 800mg + TMP 160 mg) two times a day for 7-10 days
- Or Tetracycline 25-50 mg/kg/day in 3 divided doses for 7-10 days
- In presence of foul smelling sputum, Metronidazole (400mg) three times a day for 7 days.
- If there is any suspicious of pseudomonas infection (frequent exacerbation, copious purulent sputum, yellow /green color sputum) Ciprofloxacin 500mg BD/levofloxacin 750mg OD for 14 days. IV Drugs like Inj. Ceftazidime (1-2gm 8hrly) and in. Gentamycin (3-5mg/kg/day) / Inj. Amikacin (750mg OD)/ inj.Tobramycin (3-5mg/kg/day) OD should be used in sick patients.
NB. Injectable Beta Lactam group of antibiotics should be given in infusion form instead of bolus whereas Aminoglycosides & Fluoroquinolone in bolus & once a day instead of divided dose.

**b) Bronchodilator:** These drugs are used for stimulation of mucociliary clearance. Salbutamol inhaler 200 mcg four times a day if patient is more breathlessness. Tab Etophylline + Theophylline 100 mg three times a day.

Hospitalization is required for severe breathlessness, significant haemoptysis or very sick patient.

**Patient Education**

Emphasis on quit smoking, prompt treatment of exacerbation or haemoptysis, regular physiotherapy and constant follow up to be given to all patients.

**When to refer**

Condition like uncontrolled or life threatening haemoptysis, frequent exacerbation, localized disease (for surgery), presence of complication like respiratory failure or cor pulmanale; where patient has to be referred to higher center for further better management.

**SWINE FLU**

Swine flu is a highly contagious respiratory disease in pigs caused by one of several swine influenza A viruses. Transmission of swine influenza viruses to humans is uncommon. However, the swine influenza virus can be transmitted to humans via contact with infected pigs or environments contaminated with swine influenza viruses.

**Clinical Feature**

Manifestations of H1N1 influenza are similar to those of seasonal influenza. Patients present with symptoms of acute respiratory illness, including Fever, Cough, Sore throat, Body aches, Headache, Chills and fatigue, Diarrhea and vomiting (possible). In children, signs of severe disease include apnea, tachypnea, dyspnea, cyanosis, dehydration, altered mental status, and extreme irritability. One can suspect if any one of the criteria fulfil,

- Onset of acute febrile respiratory illness within 7 days of close contact with a person who has a confirmed case of H1N1 influenza A virus infection, or
- Onset of acute febrile respiratory illness within 7 days of travel to a community where one or more H1N1 influenza A cases have been confirmed, or
- Acute febrile respiratory illness in a person who resides in a community where at least one H1N1 influenza case has been confirmed.

**Diagnosis**

For confirmation of clinical suspicion of H1N1, patient’s nasopharyngeal swab or throat swab to be collected with proper Personal Protective Equipment (PPE) and to be transported through Viral Transport Media (VTM) by maintain proper cold chain to the nearest laboratory for real time PCR.

**Treatment**

Principles of treatment are
• Early implementation of infection control precautions to minimize the spread of disease. Voluntary quarantine for close contacts of cases for at least 7 days of last contact.
• Prompt treatment to prevent severe illness and death.
• Early identification of cases and notification to proper authority

**Category A:** Patients with mild fever plus cough / sore throat with or without bodyache, headache, diarrhea and vomiting. **No testing for H1N1 required** for these group of patient. **They do not require anti-viral** and should be treated for the symptomatically. The patients should be monitored for their progress and reassessed at 24 to 48 hours. Patients should confine themselves at home and avoid mixing up with public and high risk members in the family.

**Category B:** In addition to all the signs and symptoms mentioned under Category-A, if the patient has high grade fever and severe sore throat, with/ without presence of risk factor like; Children with mild illness but with predisposing risk factors, Pregnant women, Persons aged 65 years or older, Patients with lung diseases, heart disease, liver disease, kidney disease, blood disorders, diabetes, neurological disorders, cancer and HIV/AIDS, Patients on long term cortisone therapy. **No tests for H1N1 is required** for this Category-B but **required anti-viral treatment.** Patients should confine themselves at home and avoid mixing up with public and high risk members in the family.

**Category C:** In addition to the above signs and symptoms of Category-A and B, if the patient has one or more symptoms like Breathlessness, chest pain, drowsiness, fall in blood pressure, sputum mixed with blood, bluish discoulouration of nails & worsening of underlying chronic conditions. Children with influenza like illness who had a severe disease as manifested by the **red flag signs** (Somnolence, high and persistent fever, inability to feed well, convulsions, shortness of breath, difficulty in breathing, etc).These group of patients **require testing**, immediate hospitalization and treatment.

**Anti-viral Therapy**
*Oseltamivir* in 12hrly for 5 days recommended as anti-viral for H1N1 for category B & C group of patients. Dose for treatment is as per weight of patient. > 40kg -75 mg B.D, 24 to 40Kg -60mg B.D., 15 to 23kg -45mg B.D. & <15 Kg -30mg B.D.. For infants: 0 < 3 months- 12 mg B.D., 3-5 months- 20 mg B.D., 6-11 months- 25 mg B.D.. Dose & duration can be modified as per clinical condition.

**Supportive therapy:** IV Fluids, Parenteral nutrition, Oxygen therapy should be used based on requirement. Antibiotics can be used for secondary infection. Vasopressors are indicated if there is any evidence of shock. Paracetamol or ibuprofen is prescribed for fever, myalgia and headache. Patient is advised to drink plenty of fluids. For sore throat, short course of topical decongestants, saline nasal drops, throat lozenges and steam inhalation may be beneficial. Salicylate / aspirin is strictly contra-indicated in any influenza patient due to its potential to cause Reye’s syndrome.

**Chemoprophylaxis:** Chemoprophylaxis should be administered to all close contact of suspected, probable & confirmed cases including healthcare personnel. Close contact include household, social contact, family members, workplace/school contact, fellow travelers. Oseltamivir same strength bases on weight once a day till 10 days after last exposure but maximum period of 6 week.

**Patient Education**
Mild illness does not need medical care or antiviral drugs and recovers in less than 2 weeks. Some people however are more likely to get flu complication that result in being hospitalized and occasionally results in death. The flu also can make chronic health problems worse. The family of the patient should be educated on personal hygiene and infection control measure at home. The risk of infection and of influenza viruses between people can be reduced by taking a combination of preventive actions including: Covering nose and mouth with a tissue while coughing or
sneezing and disposal of tissue in the trash after use; hand washing often with soap and water, especially after coughing or sneezing, avoid touching eyes, nose or mouth; avoiding close contact with sick people; staying home from work or school until illness is over.

**When to Refer**

Any clinical sign or symptoms of deterioration like features of hypoxia (increase respiratory rate, decrease oxygen saturation, decrease level of consciousness), features of circulatory failure (cold skin, low blood pressure, decrease urine output) to be referred to higher center for requirement of intensive therapy or Ventilatory support.

**BRONCHIAL ASTHMA**

Asthma is a chronic inflammatory disorder of the airways in susceptible individuals associated with widespread but variable airflow obstruction and an increase in airway response to a variety of stimuli. Obstruction is reversible spontaneously or with treatment.

**Diagnosis**

A clinical diagnosis of asthma should be suspected in the presence of recurrent/episodic wheezing, breathlessness, cough, and/or chest tightness which is variable even worse at night and provoked by triggers. Triggers means things those aggravate the asthma symptoms, those are like; outdoor & indoor allergens (house dust mites, grass pollens, animal proteins, moulds), viral & bacterial respiratory infection, drugs, exercise, certain occupation, GERD, pollution, psychological factors etc. None of the symptoms and signs is specific for asthma. Absence of signs and symptoms at the time of presentation does not rule out the presence of asthma.

**Investigation**

Wherever available, spirometry is recommended for all patients suspected to have asthma for confirming diagnosis, assessing severity of airway limitation and monitoring asthma control but normal spirometry does not rule out asthma. If spirometry is not available, airways obstruction can be measured by Peak Expiratory Flow (PEF) meters. Chest radiograph is not routinely recommended for patients suspected to have asthma but may be considered when alternate diagnosis or complication of asthma is suspected. DC & TLC are not indicated routinely but may require whenever any suspicion of superadded bacterial infection.

**Treatment**

**Pharmacological**

The goals of asthma management include relief of patient’s current symptoms and prevention of further disease progression. For the purpose of management asthma can be classified based on the requirement of type of medication such as Step1, step 2, step 3, step 4 & step 5. Broadly there are two types of medication, one is **“Reliever”** means which required only to relieve of symptoms (Which should be used when ever patient is symptomatic), other is **“Controller”** means required for control of asthma (Which should be used irrespective of symptoms to control the asthma & to prevent future exacerbation). Drugs used as “Reliever” are Short Acting Beta2 Agonist (SABA), injection Theophylline & Short Acting Muscarinic Antagonist (SAMA) and as “Controller” are Long Acting Beta2 Agonist (LABA), Inhaled Corticosteroid (ICS), Leukotriene Receptor Antagonists LTRAs), oral methylxanthines (Theophylline, Doxophylline, Acebrophylline), Long Acting Muscarinic Antagonist(LAMA) & oral corticosteroid. Among SABA, there are Salbutamol, Terbutaline and among LABA, there are Formoterol, Salmeterol and among ICS there are Budesonide, Fluticasone, Beclomethasone, Ciclesonide and among SAMA, Ipratropium bromide and among LAMA, Tiotropium Bromide. Among the ICS, based on the strength there are divided into Low dose, Medium dose or High dose.
Commonly available inhaled corticosteroids and their equipotent doses (in \text{\textmu}g)

<table>
<thead>
<tr>
<th>Drug name</th>
<th>Low dose (mcg)</th>
<th>Medium dose (mcg)</th>
<th>High dose (mcg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beclomethasone</td>
<td>100-250</td>
<td>&gt;250-500</td>
<td>&gt;500-1000</td>
</tr>
<tr>
<td>Fluticasone</td>
<td>100-250</td>
<td>&gt;250-500</td>
<td>&gt;500-1000</td>
</tr>
<tr>
<td>Budesonide</td>
<td>200-400</td>
<td>&gt;400-800</td>
<td>&gt;800-1600</td>
</tr>
<tr>
<td>Ciclesonide</td>
<td>80-160</td>
<td>&gt;160-320</td>
<td>&gt;320-1280</td>
</tr>
</tbody>
</table>

Practical considerations while initiating therapy in asthma patients

<table>
<thead>
<tr>
<th>Steps</th>
<th>Choice of Patients</th>
<th>Treatment Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. (As needed reliever medication)</td>
<td>Patients who have only occasional symptoms of asthma (less than twice a month) and remain completely asymptomatic between these episodes.</td>
<td>Patient should use a reliever medication (SABA), as and when required; no controller medication is used.</td>
</tr>
<tr>
<td>2. (Single controller plus as-needed reliever medication)</td>
<td>Patients with symptoms between twice a month and twice a week and no (rare) nocturnal symptoms</td>
<td>Patient should receive a controller medication in addition to the as-needed reliever medication. Preferred choice: Low-dose ICS. Alternate Controller: LTRA. Monotherapy with LABA and/or oral methylxanthines should be avoided.</td>
</tr>
<tr>
<td>3. (Two controllers plus as needed reliever medication)</td>
<td>Symptoms more than twice a week, those with nocturnal symptoms or in patients who remain uncontrolled despite using low-dose ICS, patients with acute exacerbation (besides manage-ment of the acute episode)</td>
<td>Preferred Choice: LABA with low-dose ICS. Alternate choice: (a) doubling the dose of ICS (to a medium-dose ICS range), (b) adding a LTRA to ICS, or (c) adding theophylline to ICS.</td>
</tr>
<tr>
<td>4. (Two or more controllers plus as-needed reliever medication)</td>
<td>Patients uncontrolled despite step 3 therapy</td>
<td>Preferred Choice: Continue on ICS/LABA combination and increase ICS dose to medium and high doses sequentially. High-dose ICS should preferably be given on a trial basis for 3 months to assess patient response, and de-escalated if no clinical benefit is observed. For patients who were not receiving LABA at step 3, addition of LABA should be done before any further change in treatment. We suggest adding tiotropium, LTRA, and/or methylxanthine in those who remain uncontrolled despite the moderate to high-dose ICS/LABA combination At this stage, further treatment needs to be individualized.</td>
</tr>
<tr>
<td>5. (Difficult asthma) to treat asthma</td>
<td>Patients uncontrolled despite step 4 therapy</td>
<td>In patients who fail to respond to all therapeutic interventions detailed in step 4, we recommend a trial of oral corticosteroids or other targeted therapies (such as anti-IgE etc.)</td>
</tr>
</tbody>
</table>
Asthma can be categorized as either adequately controlled or inadequately controlled by assessing four parameters—day time symptoms (or reliever medication use), night time symptoms/awakening, limitation of activities, and lung function i.e. Peak Expiratory Flow (PEF).

**Level of current asthma control (over the preceding 4 weeks)**

<table>
<thead>
<tr>
<th>Components</th>
<th>Adequately controlled (all four need to be present)</th>
<th>Inadequately controlled (any one)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day time symptoms or rescue medication use</td>
<td>Twice or less in a week</td>
<td>More than twice a week</td>
</tr>
<tr>
<td>Night time symptoms/ awakening</td>
<td>None</td>
<td>Any</td>
</tr>
<tr>
<td>Limitation of Activity</td>
<td>None</td>
<td>Any</td>
</tr>
<tr>
<td>Pulmonary Function, Peak Expiratory Flow (PEF)</td>
<td>&lt; 80% of predicted or person’s best</td>
<td>&gt; 80% of predicted or person’s best</td>
</tr>
</tbody>
</table>

**Stepping Up Therapy:** Patients whose asthma remains inadequately controlled on the existing treatment need to augment therapy with an aim of achieving adequate asthma control by stepping up of therapy at intervals of 2-3 month.

**Stepping Down Therapy:** Patients who remain well controlled on treatment require step down of their treatment to maintain control at the lowest possible step by stepping down every 2-3 months. For patients with seasonal exacerbations, not to step down treatment at the time when asthma control is likely to be poor. Patients who are being stepped down should be closely monitored for any loss of asthma control.

**Non-Pharmacological:** Avoidance of trigger factor, environmental control measure (Occupational Asthma) are to be followed for prevention of acute attack as well to maintain the control status of asthma.

**Patient Education:** During the time of consultation to the patient, some asthma education to be given to the patient as well as the family member of patients like proper use of inhalers, identification of trigger factor, early identification of severity, regular physician review etc.

**When to Refer:** In spite of proper use of inhalers, still asthma in step 5 (difficult to treat asthma), patient has to refer for specialist care.

****

**MANAGEMENT OF ACUTE EXACERBATION OF ASTHMA**

An exacerbation of asthma is characterized by worsening of one or more of the asthma symptoms (cough, wheezing, chest tightness, dyspnea), leading either to increased need for reliever medications or hospitalization. The risk factor for exacerbation include, previous history of need for mechanical ventilation or hospitalization for asthma care in previous 1 year, currently not using inhaled corticosteroids, currently using or have recently stopped oral glucocorticosteroids, use of >1 canister/month on inhaled short-acting beta-2 agonist (or equivalent dry powder inhaler doses or nebulizer doses, need of three or more classes of asthma medication, poor adherence to treatment, history of psychiatric illness or drug abuse, lack of social support, use of NSAIDs, presence of co-morbidities etc. The severity of an asthma exacerbation is defined on a combination of signs and symptoms and the extent of accompanying cardiorespiratory dysfunction into non severe, severe and life threatening.
Assessment of the severity of acute asthma exacerbation

<table>
<thead>
<tr>
<th>Severity</th>
<th>Symptoms</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non Severe</td>
<td>Not fulfilling the criteria for severe or life threatening asthma</td>
<td></td>
</tr>
<tr>
<td>Severe (Presence of two or more)</td>
<td>Inability to complete sentences, Agitation</td>
<td>Use of accessory muscles, Respiratory rate &gt;30/min, Heart rate &gt;110/min, Pulsus paradox &gt;25 mmHg, Silent chest, PEF &lt;60% of predicted or personal best, SpO2 &lt;92% (if possible)</td>
</tr>
<tr>
<td>Life threatening (Presence of any)</td>
<td>Alteration in mental status, Orthopnea</td>
<td>Cyanosis, Paradoxical breathing, Heart rate &lt;60/min (excluding drug related bradycardia)</td>
</tr>
</tbody>
</table>

Patients classified as having a severe asthma attack are best managed in a hospital setting, while those classified as having non-severe asthma exacerbations can be safely managed on an outpatient basis.
Non-severe exacerbations: Patients should be initially managed with an increase in dose of inhaled salbutamol (4-6 puffs of 100 ìg salbutamol every 30 min) If there is no response in 1 h, oral prednisone 30-40 mg once a day for 5-7 days should be started.

Severe exacerbations: Oxygen should be titrated to maintain a SpO2 between 93% and 95% (>95% in pregnancy). Lack of pulse oximetry/arterial blood analysis should not preclude administration of oxygen Inhaled salbutamol (4-6 puffs of 100 ìg every 15 min; nebulizer: 2.5 mg salbutamol every 15 min) plus ipratropium (2 puffs of 20 ìg every 4 h; nebulizer: 500 ìg once then 250 ìg q4-6 h), the duration needs to be individualized depending on clinical response 30-40 mg of prednisolone or equivalent (0.75 mg dexamethasone ~4 mg methylprednisolone ~5 mg prednisolone ~20 mg hydrocortisone) for 5-7 days. If the patient fails to respond within 1 h or worsens, patient to be referred to higher centre.

Patient Education: During the time of discharge to the patient, asthma education like proper use of inhalers, identification of trigger factor, identification of warning sign (Cyanosis, paradoxical respiration, clouding of consciousness) to be delivered to the patient as well as family members of patient.

When to Refer: Presence of following signs like cyanosis, paradoxical breathing, bradycardia, alter mental status and unresponsive to severe asthma (at least 1 hr after the initiation of treatment) should be refer to higher centre for ICU management.

COPD

(Chronic Obstructive Pulmonary Disease)

Definition:
A common preventable and treatable disease characterized by persistent airflow limitation, usually progressive, and associated with enhanced chronic inflammatory response in the airways. Exacerbations and co-morbidity contribute to severity.

Risk factors:
Major risk factors are cigarette smoking, biomass fuel (home cooking and heating fuels), occupational exposure to dust and chemicals, air pollution, and asthma and chronic bronchitis.

Diagnostic features:
The symptoms are progressive exertional shortness of breath, persistent chronic cough and chronic sputum production. The general findings include barrel shaped chest, purse lip breathing and use of accessory muscles. The chest findings are vesicular breath sounds of diminished intensity, prolonged expiration and expiratory wheeze.

Laboratory investigations:
Routine blood count (DC, TLC) detects presence of infection during exacerbations. X-ray chest is done to exclude pneumonia and pneumothorax. Spirometry (if available) shows post bronchodilator FEV1/FVC < 0.7 (70%), without FEV1 reversibility.

Non pharmacologic treatment:
The modalities include smoking cessation, avoidance of exposure to risk factors, physical activities for rehabilitation, and vaccination.
Pharmacologic treatment:

1. **Bronchodilators:**
   - Inhaled short acting and long acting bronchodilators are used. Long acting formulations of beta2 agonists and anti cholinergics are preferred over short acting drugs in case of stable COPD. Oral theophyllines are added when patient is symptomatic in spite of inhaled bronchodilators. The inhalational drugs are:
     - Salbutamol 200 mcg 4 times daily
     - Tiotropium 9 mcg twice daily
     - Formoterol 6 mcg twice daily
   - PMDI, DPI, or nebulizers are used.

2. **Corticosteroids:** (severe and very severe COPD)
   - Inhaled are preferred over oral corticosteroids. Monotherapy corticosteroids are not recommended. Inhaled fluticasone 100-500 mcg twice daily, followed by washing of mouth. (Dose as per need and severity)

3. **Phospho diesterase 4 inhibitor**
   - Roflumilast is a PDE4 inhibitor, used in case of frequent exacerbators of chronic bronchitis, severe and very severe COPD. Dose = 500 mcg OD.

4. **Oxygen:**
   - Long term oxygen therapy (LTOT) > 15 hours/day, with nocturnal coverage, if SaO2<88%, with or without increase in PaCO2.

5. **Ventilator support**

Exacerbations:
- An acute event characterized by worsening of respiratory symptoms (cough, sputum, purulence, SOB) beyond normal day to day variations.

**Management (A, B, C, Adjunct)**

A=Antibiotics  B=Bronchodilators  C=Corticosteroids

A=Antibiotics
- Amoxicillin clavulanate (500mg+125mg) tds 7-10 days or Azithromycin 500 mg od * 5 days

B=Bronchodilators
- Short acting B2 agonist with or without short acting anticholinergics via nebulizer or PMDI with spacer. Salbutamol (1.25mcg) + ipratropium (0.5mg) every 4-6 hours.

C=Corticosteroids
- Prednisone 40mg od * 5-10 days, Nebulized budesonide.

  - If patient’s oxygen saturation by pulse oxymetery<88%, oxygen inhalation can be given but in a controlled manner i.e. low flow by nasal prong & target of SPO2 will be 88-93%.
Adjunct:
Adjunctive t/t includes fluid balance, nutrition, thromboembolic prophylactic measures, diuretics, anti coagulants and management of comorbidities.

Patient education:
Patients need to be educated about vaccination (pneumococcal and influenza vaccine in patients with age > 65 years, DM-2, COPD), smoking cessation, proper use of inhaler devices, avoidance of noxious gases and fumes, exercise, maintenance therapy at home, and follow up at 4-6 weeks.

Referral criteria:
- Confusion, lethargy, coma, fatigue
- Severe dyspnea with inadequate response to t/t
- Paradoxical respiration, accessory muscle use, cyanosis
- Hemodynamic instability

COMMUNITY ACQUIRED PNEUMONIA

Definition:
Inflammation of alveolar tissue often by a microbial agent. The typical organisms are pneumococcus, H.influenzae, staphylococcus. The atypical organisms are mycoplasma, legionella, chlamydia.

Diagnostic features:
The symptoms are fever with or without chills and rigor, cough with or without expectoration (purulent or mucopurulent), fever and shortness of breath.
The signs are tachycardia, tachypnea and increase in temperature.

Chest examination may reveal bronchial breath sounds and crepitations, with or without evidence of pleural effusion.

Chest X-ray shows infiltrates and consolidation.
Lab test findings are elevated TLC, neutrophilia and toxic granules.

Severity assessment: (CURB-65)
- C=confusion
- U=urea > 20 mg/dl
- R=respiratory rate >30/ min
- B=blood pressure < 90/60 mm Hg
- Age > 65 years

Score 0-1 à OPD t/t, Score 2 à inpatient t/t, Score 3-5 à ICU t/t.

Management:
- Supportive management includes hydration, oxygenation, analgesics and antipyretics.
- Antibiotics are selected according to plans elicited below.

✦ PLAN-A : OPD t/t of previously healthy
Tab Azithromycin(500) od for 10 days or
Cap Clarithromycin(500) bd for 10 days or
Tab Erythromycin(500) qid 10 days or
Cap Doxycycline(200) Loading Dose followed by 100mg od for 10 days

PLAN-B: CAP with comorbidities
- Cap Amoxicillin (500) tid for 10 days or Tab Cefpodoxime (200mg) bd for 10 days and
- Tab Azithromycin(500) od 10 days or Cap Clarithromycin(500) bd for 10 days or Cap Doxycycline(100) bd * 10 days

PLAN-C: inpatient
- Inj Cefotaxime 1-2g iv tds or Inj Ceftriaxone 2g iv/im od with Inj. Azithromycin 500 iv od

PLAN-D: ICU t/t
- Referral to higher centre

NB: Injectable Beta Lactam group of antibiotics should be given in infusion form instead of bolus whereas Aminogycosides & Flouroquinolone in bolus & once a day instead of divided dose.

Patient education:
Patients must be educated about hand hygiene, referral criteria and vaccination.

Referral criteria:
Referral to higher centre is needed in case of severe CAP, shock (hemodynamic instability), cyanosis, Empyema and complications.

PULMONARY TUBERCULOSIS

Classification of tuberculosis
1. PTB (Pulmonary tuberculosis): Sputum smear positive, sputum smear negative.
2. EPTB (Extra-pulmonary tuberculosis).

Classification of Treatment categories
Cat I (New cases), Cat II(Previously treated cases- Recurrent, Lost to Follow-up, Treatment failure), Cast IV( MDR-TB cases) , Cat V (XDR-TB cases).

Diagnosis of Tuberculosis
It is based on history, physical symptoms and signs and sputum smear examination for AFB.

History:
Contact history is very important for suspecting a case of tuberculosis. History reveals previous treatment for tuberculosis which helps in case definitions, risks for resistance and appropriate treatment.

Definition of presumptive TB:
1. **Presumptive PTB**: Cough >2wks, Fever >2wks, Significant weight loss, Hemoptysis, Any abnormality in CXR. Contact history with Smear Positive cases, Cough of any duration in suspected or confirmed EPTB and known HIV cases. 2-3% of new adult out-patients in PHC level and 5-15% of them are found to be smear positive.

2. **Presumptive EPTB**: Presence of organ specific symptoms and signs like swelling of lymph nodes, pain and swelling in joints, neck stiffness, disorientation etc. and/or constitutional symptoms like significant weight loss (loss of > 5% body weight as compared to highest weight recorded in last 3 months), persistence of fever >2wks, night sweats.

3. **Presumptive Pediatric TB**: Persistent fever and/or cough >2wks, loss of weight/no weight gain and/or history of contact with infectious TB cases.

4. **Presumptive DR-TB**: TB patients who have failed treatment with first line drugs (FLDs), pediatric TB non-responders, TB patients who are contacts of DR-TB or rifampicin resistant (RR), TB patients who are found positive on any follow up sputum smear examination during treatment with first line drugs, previously treated TB cases, TB patients with HIV co-infection.

**Definition of MDR-TB:**

MDR-TB is defined as a DR-TB displaying resistance to at least INH and Rifampicin with/without other first line anti tubercular drug.

**Definition of XDR-TB:**

MDR-TB plus resistance to one of Flouroquinolones (Lfx, Mfx) and one injectable agent (Km, Am, Cm).

**CLINICAL FEATURES:**

There is no characteristic distinguishing clinical presentations between susceptible and drug resistant cases.

1. **Symptomatology:**

   Constitutional symptoms are low grade fever specifically in evening hours, night sweats, lassitude, loss of appetite, weight loss, failure to thrive (in children). Cough with scanty mucoid expectoration (Dry cough in children) is most frequent. Hemoptysis is a classical but nonspecific symptom.

2. **Signs:**

   Emaciated body, flattening of chest, Inspiratory crackles or cavernous breath sounds over apical/posterior segments of upper lobe or apical segment of lower lobe are classical findings.

**LABORATORY METHODS:**

Chest X-ray revealing upper zone patchy or nodular opacities or cavitations present unilaterally/bilaterally are highly sensitive but not specific of tuberculosis. Sputum smear for AFB positivity is less sensitive but more specific. At least 2 morning samples/one morning with one spot sample are economical and more informative. Culture is confirmatory. Recent molecular methods like (a) CBNAAT (Cartridge Based Nucleic Acid Amplification test to diagnose both Tuberculosis and RR-TB (rifampicin resistant TB) cases, used for screening and (b) LPA (Line Probe Assay) to diagnose MDR-TB cases, are recently adapted diagnostic procedures for rapid diagnosis and early institution of chemotherapy.
CHEMOTHERAPY:

Multi-drug Regimen is scientifically more rational, should be the basis of treatment to reduce infectiousness and initial INH resistance in IP and subsequent relapse or resistance in CP. Single anti-tubercular drug is irrational and should not be added to a failing regimen. Short course chemotherapy with supervision (DOTS) is more compliant and most effective. “Daily regimen” is now preferred to be more effective with high cure rates than “Intermittent regimen”. Fixed dose combination (FDC) in spite of its disadvantage in few aspects, causes better adherance and compliance to the patients.

Table-1 Essential Anti-tubercular Drugs WHO (1997, 2003) for CAT I and CAT II DOTs

<table>
<thead>
<tr>
<th>i-TB Drugs</th>
<th>Intermittent (thrice weekly) mg/Kg</th>
<th>Daily mg/kg</th>
<th>Adverse Reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>(INH)</td>
<td>10</td>
<td>5</td>
<td>HR, PNP, Psychosis, Hepatitis, SLE, Skin rashes, Gynecomastia</td>
</tr>
<tr>
<td>(Rifampicin)</td>
<td>10</td>
<td>10</td>
<td>HR, Gastric upset, Skin rashes, Hepatitis, Flu syndrome, Resp Failure, ARF, Hemolysis, Thrombocytopenia</td>
</tr>
<tr>
<td>Pyrazinamide)</td>
<td>35</td>
<td>25</td>
<td>HR, Skin rashes, Arthritis, Hepatitis, Gastric upset, Photosensitivity</td>
</tr>
<tr>
<td>Ethambutol)</td>
<td>30</td>
<td>15</td>
<td>Optic neuritis, Color vision</td>
</tr>
<tr>
<td>Streptomycin)</td>
<td>15</td>
<td>15</td>
<td>HR, Circum oral parasthesia, Vertigo, Acute tubular necrosis</td>
</tr>
</tbody>
</table>

NB: Twice weekly therapy is not recommended by WHO & IUATLD because of missing a dose resulting in insufficient treatment and higher risk of toxicity. HR- Hypersensitivity reaction, PNP- Peripheral neuropathy.

Table-2 WHO (2016): DR-TB Guidelines. New Classification of Anti-TB Drugs:

<table>
<thead>
<tr>
<th>Class</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.</td>
<td>Fluoroquinolones</td>
</tr>
<tr>
<td></td>
<td>Lfx (levofloxacin), Mfx (Moxifloxacin), Gfx (Gatifloxacin)</td>
</tr>
<tr>
<td>B.</td>
<td>Second line injectable agents</td>
</tr>
<tr>
<td></td>
<td>Kn (Kanamycin), Am (Amikacin), Cm (Capreomycin)</td>
</tr>
<tr>
<td>C.</td>
<td>Other core Second Line Drugs</td>
</tr>
<tr>
<td></td>
<td>Eto (Ethionamide), Pto (Prothionamide), Cs (Cycloserine), Trd (Pirazamide), Czf (Clofazimine), Lzd (LINZOLID)</td>
</tr>
<tr>
<td>D.</td>
<td>Add-on agents</td>
</tr>
<tr>
<td></td>
<td>D1 Z (Pyrazinamide), E (Ethambutol)</td>
</tr>
<tr>
<td></td>
<td>D2 Brd (Bedaquine), Dlm (Delamanic)</td>
</tr>
<tr>
<td></td>
<td>D3 PAS (Para-aminosalicylic acid), Ipm (Imipenem/clavulanic acid), Mpm (Meropenem), Amx-clv (Amoxicillin-clavulanic acid), T (Thiacetazone)</td>
</tr>
</tbody>
</table>

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Table 3: Standardized Anti-tubercular Regimens:

<table>
<thead>
<tr>
<th>Current Regimens</th>
<th>Proposed Regimens</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAT I 2HRZE/4HR (Ideal Initial FLD Regimen), 2(HREZ),4(HR),</td>
<td>2(HREZ),4(HRE)</td>
</tr>
<tr>
<td>CAT II 2SHRZE/1HRZE/SHRE, 2(SHERZ),1(HREZ),5(HRE),</td>
<td>2(SHERZ),1(HREZ),5(HRE),</td>
</tr>
<tr>
<td>CAT IV (MDR-TB) 6-9 (Km, Lfx, Et, Cs, E, Z) /18 (Lfx, Et, Cs, E)</td>
<td>4-6 Km Mfx Plt Ctz Z H (high dose) / 5 Mfx Ctz E (Shorter MDR-TB regimen)</td>
</tr>
<tr>
<td>CAT V (XDR-TB) 9-12 (Cm, PAS, Mfx, Lzd, Amx, Clv, Ctz, High dose H) / 18 (PAS, Mfx, Lzd, Amx, Clv, Ctz, High dose H)</td>
<td>6 Am Lfx Cs Lzd Bdq PAS / 12 Lfx Cs Lzd PAS Cm Lfx Lzd Imp-Civ Bdq PAS</td>
</tr>
</tbody>
</table>

Table 4: Standardized Daily Regimens:

Drugs Dosage for Adult TB

<table>
<thead>
<tr>
<th>Weight category</th>
<th>Number of tablets (FDCs)</th>
<th>Inj. Streptomycin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intensive phase</td>
<td>Continuation phase</td>
</tr>
<tr>
<td>25-39 kg</td>
<td>75/150/400/275</td>
<td>75/150/275</td>
</tr>
<tr>
<td>40-54 kg</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>55-69 kg</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>&gt;=70</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

*Inj. Streptomycin to be added in IP phase for 2 months in the previously treated regimen of drug sensitive patients. In patients above 50 years of age, maximum dose of streptomycin should be 0.75 gm.
Adults weighing less than 25 kg will be given loose drugs as per body weight. Dosages of loose drugs are given in appendix.

Drug Dosage for Pediatric TB

<table>
<thead>
<tr>
<th>Weight category</th>
<th>Number of tablets (dispersible FDCs)</th>
<th>Inj. Streptomycin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intensive phase</td>
<td>Continuation phase</td>
</tr>
<tr>
<td>4-7 kg</td>
<td>50/75/150</td>
<td>100</td>
</tr>
<tr>
<td>8-11 kg</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>12-15 kg</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>16-24 kg</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>25-29 kg</td>
<td>3 + 1A</td>
<td>3 + 1A</td>
</tr>
<tr>
<td>30-39 kg</td>
<td>2 + 2A</td>
<td>2 + 2A</td>
</tr>
</tbody>
</table>

A = Adult FDC (HRZE = 75/150/400/275; HRE = 75/150/275)
Patient Education:

Tuberculosis is an infectious communicable disease and spreads on coughing (droplet infection) and contact. Health screening at health facility is necessary for all symptomatic adult contacts and all children <6yrs age. Prompt attendance at nearest health care facility is more valuable if one has cough of ≥2wks or any duration (in cases of EPTB, HIV). Hence use fomites /facemasks during coughing is most effective control of infection when in family or crowds. The patient should not spit and speak in loud voice and should avoid smoking and alcohol. Coughing material must be discarded after treating with disinfectant (phenol 5% for 20 minutes) to avoid biohazards. Two sputum specimens (either 2 mornings or 1 morning with 1 spot) are required for sputum AFB smear result in DMC. There should be well ventilated and well lighted with sunrays to avoid spread of infection. Prompt medical attendance at nearest medical center is essential, once duration of cough is ≥2wks or more or persistent fever >1 month. The slogan of awareness should be “TB is completely curable”, if treated earlier with complete treatment. But if untreated or partially treated turns to complications, DR-TB and death. Treatment is available in free of cost at each DOT center. Medication with proper dosage, proper duration and by proper healthcare worker is highly successful. Early disappearance of symptoms during treatment is not a sign of cure. Adverse drug reactions (gastric upset, swelling of joints, yellow conjunctiva etc.) during treatment are usual and the patient should approach a medical officer before stopping medication. Adherence to treatment and periodic follow up for sputum conversion and for drug side effects are fore-runner of successful treatment. “MDR-TB is man-made”.

Referrals:

Presumptive PTB cases to nearest DMC (Designated Microscopic centers) Presumptive Drug Resistant (MDR-TB) cases to Tertiary care centers (DR-TB centers) for Culture/DST at Intermediary Referral Laboratory (through DTO for recording and reporting). For Adverse Drug Reactions (ADR), to Chest Physicians. For complications (Pneumothorax/BPF), to Tertiary centers.

LUNG ABSCESS

Introduction:

Lung abscess is defined as a localized area of destruction of lung parenchyma characterized by suppuration and necrosis, mostly due to bacterial infection. Lung abscess are classified as “acute” or “chronic” based on symptoms duration(< or >/= 4-6weeks).

Lung abscess are characterized as “primary”; when they appear after lung infection in previously healthy person or in patients prone to aspiration of nasopharyngeal or oropharyngeal material, e.g. alcoholics, drug addicts, patients with reduced level of consciousness, comatose patients or after epileptic seizures.

Abscess formation may occur secondarily in cases of mechanical bronchial obstruction caused by endobronchial mass, a foreign body or extraluminal compression, septic pulmonary emboli due to infective endocarditis, contamination of bullae or bronchiectasis.

CLINICAL MANIFESTATIONS:

Clinical findings of lung abscesses depend on the nature of the infections. Most cases are due to mixed infections; whereas dominant pathogens isolated are anaerobic bacteria found in normal oral and upper intestinal microbial flora.
Lung abscesses caused by anaerobic or mixed bacteria present with non-specific symptoms and signs e.g. fever, night sweats, dull chest pain, fatigue, anorexia, weight loss, productive cough with foetid sputum or sometimes bloody.

Physical examination includes fever, poor dentition & gingival disease, anaemia, clubbing, tachycardia, tachypnea, and abnormal findings consistent with lung parenchymal infection, pleural fluid, amphoric or cavernous breath sound on auscultation.

INVESTIGATIONS:

On routine blood examination; anaemia of chronic disease and leukocytosis are usually present.

The diagnosis of lung abscess is usually made by chest radiography; which shows a lung cavity with an air-fluid level. Typically the cavity wall is thick and irregular, and a surrounding pulmonary infiltrate is often present.

Computed tomography (CT) is more sensitive than chest radiography, as it is useful to detect small cavities, provide evidence for obstructing endobronchial lesions and again distinguish lung abscess from hydropneumothorax.

For microbiological diagnosis; culture of sputum is not suitable for the detection of anaerobic pathogens due to their frequent contamination from normal flora of oral cavity. But sputum cultures have been proved to be useful in cases of lung abscesses attributed to aerobic pathogens, e.g. *Klebsiella, Staphylococcus* and *Pseudomonas*. Blood cultures and pleural fluid cultures are usually negative. Fiberoptic bronchoscopy with quantitative cultures of samples obtained by protected specimen brush or broncho-alveolar lavage (BAL) or Trans-thoracic needle aspiration are usually required for targeted antimicrobial treatment.

MANAGEMENT:

A: GENERAL PRINCIPLES:

Reversibility of the underlying conditions predisposing to aspiration must be considered. Procedures such as nasogastric feeding tube, sedation, lying flat on the bed while sleeping, reflux and frequent choking are associated with aspiration and hence need strategies for remediation. Measures like semi-recumbent or upright position, reduction of stomach volume with suction or metoclopramide, neutralization of gastric acid with H2 blockers & antacids should be taken. Chest physiotherapy for drainage of pus should be advocated.

B: ANTIMICROBIAL THERAPY:

The initial management of lung abscess is based on empirical therapy with broad spectrum antibiotics and is followed by antibiotics streamlining driven by microbiological documentation by cultures.

Previously, the standard drug for lung abscess involving anaerobic bacteria was intravenous or high dose oral Penicillin. But presently Clindamycin (600mg intravenous every 8 hourly followed by 150-300mg 6 hourly PO) is considered the first choice of antibiotics, which is superior to Penicillin in terms of response rates and time to defervescence.

Alternative regimens that can be used successfully include â lactam/â lactamase inhibitor combinations (Amoxicillin-Clavulanate, Ampicillin-Sulbactam, Piperacillin-Tazobactam) combined with Metronidazole. But monotherapy with metronidazole should be avoided due to inadequate coverage for aerobic and microaerophilic streptococci.
Linezolid (initially 600mg intravenous twice daily followed by oral administration after clinical improvement) is preferred in cases of lung abscess caused by MRSA. An alternative choice is Vancomycin (15mg/kg intravenous twice daily).

The appropriate duration of therapy is dependent on the clinical and radiographic response of the patient. Patients should be treated at least until fever, putrid sputum and abscess fluid have resolved and hence a minimum of 2-3 weeks of antibiotics is recommended. Treatment interval may be prolonged to several months (>2 months), especially when the initial lesion is of large size (maximum diameter >6 cms).

NB: Injectable Beta Lactam group of antibiotics should be given in infusion form instead of bolus whereas Aminoglycosides & Fluoroquinolone in bolus & once a day instead of divided dose.

PATIENT EDUCATION:

The symptoms of a lung abscess are similar to the symptoms of other lung conditions including pneumonia and tuberculosis. The most noticeable symptoms of a lung abscess is productive cough, which may be bloody or pus-like with a foul odour.

Most lung abscess can be treated with a course of antibiotics. Chronic abscess or an abscess left untreated could lead to fatal complications (e.g. Empyema, Pneumatocele, Broncho-Pleural Fistula).

Alcoholism is the most common conditions that predisposes people to lung abscess. Other people with weakened immune systems, such as Cancer, HIV, Organ transplant and Autoimmune disease are also at risk.

REFERRAL:

Persistence of bacteremia or high grade fever after 72hr, hemoptysis (moderate/massive), absence of change in sputum production/character in the radiographic images over 7-10 days suggests unappreciated anatomic or microbiologic problems and require referral to tertiary care center for bronchoscopy, CT-guided percutaneous drainage or cardiothoracic surgical intervention.

Surgical resection of necrotic segments of lung is helpful, if the response to antibiotic is poor, for large abscesses or ventilation-perfusion scans suggesting little residual lung function, airway obstruction limiting drainage.

Where there is slow resolution, the possibility of malignancy or unusual micro-organisms must be considered. A thick walled lung abscess should be further investigated to rule out malignancy.

REFERENCES:

PNEUMOTHORAX

INTRODUCTION

Pneumothorax is the accumulation of air in pleural space. Pneumothorax divided into two type:- spontaneous and traumatic. Spontaneous divided into primary and secondary. spontaneous Pneumothorax occur without antecedent trauma or other obvious causes. Primary spontaneous Pneumothorax occur in otherwise healthy individuals. Causes of secondary pneumothorax: When it occurs as complication of underlying lung disease. COPD most common cause world wide & most common cause in India is pulmonary tuberculosis. Other less common causes are tumours, sarcoidosis. Traumatic Pneumothorax may be due to Iatrogenic (Thoracocentesis, Bronchoscopy) or Non-iatrogenic.

Diagnostic feature-

Symptoms

Patient will present to us with complain of acute onset of breathlessness and chest pain, pain limited to side of pneumothorax. Possibility of pneumothorax should be consider if in COPD patient who has increase in breathlessness particularly associated with chest pain

Past history of COPD, TUBERCULOSIS, HIV INFECTION, BRONCHIECTASIS should be taken. history of smoking and occupational history should be taken. Patient will be very much dyspnoic and use of accessory muscle of respiration, tension pneumothorax, may be associated with tachycardia, tachypnea, hypotension, and cyanosis.

On chest examination–side of pneumothorax is larger than contralateral side, moves less during respiration with trachea shifted towards opposite side, percussion note will be hyperresonant. Decrease or absence of breath sound over the affected side.

INVESTIGATIONS

In this above scenario urgent CHEST X RAY should be done. in chest x-ray there will be presence of hyperlucency with visible visceral pleural line and absence of vascular markings.

TREATMENT-

GENERAL PRINCIPLE-

Goal of treatment is get rid of pleural space of air and decrease of likely hood of recurrence.

High flow Oxygen inhalation, Monitoring of vitals, Analgesic for pain, Cough suppressant are the main stay of treatment.

Secondary spontaneous Pneumothorax should managed with Intercostal drainage tube insertion under water seal drainage.

Primary spontaneous Pneumothorax having less than 15% of hemithorax kept under observation, if more than 15% than closed aspiration should done if failure Intercostal tube drainage given.

Appropriate treatment of underlying disease like bronchodilator and inhalation corticosteroid for COPD. ATT for tuberculosis.
**Referral**—when patient develops signs of tension Pneumothorax (tachycardia pulse rate more than 140/min. hypotension, cyanosis), chest x-ray reveals contralateral shift of mediastinum and inversion of diaphragm. Tension Pneumothorax is a medical emergency. No delay in treatment or referral should be done. Recurrence of primary spontaneous Pneumothorax for procedure for prevention of Pneumothorax.

**PATIENT EDUCATION**

**Smoking cessation.**

As there is high chance of recurrence in both primary and secondary Pneumothorax patient should taught that if any patient developed sudden onset breathlessness or/and pleuritic chest pain than urgently come to healthcare facilities.

**When to Refer—**

Any case of Pneumothorax with heamodynamically unstable refer to higher centre and immediate measure taken to get rid of pleural air at PHC/CHC level a 16 gauge iv canula with water seal drain to second intercostals space in mid clavicular line.

### PLEURAL EFFUSION

**INTRODUCTION:**

Pleural effusion is an abnormal accumulation of fluid in the pleural space due to disruption in the homeostatic forces that control the flow of fluid into & out of the pleural space.

Pleural effusion can be of two types: “exudative”; when local factors that influence the formation and absorption of pleural fluid are altered, & “transudative”; results from the alteration of systemic factors.

The common causes of exudative pleural effusions are infections (tuberculosis, bacterial, viral); malignancy (mesothelioma, secondary malignancy); collagen vascular disorders (RA, SLE); pulmonary infarction, gastro-intestinal diseases, drug-induced etc. The causes of transudative pleural effusions are congestive heart failure, cirrhosis of liver, nephrotic syndrome, myxedema etc.

**CLINICAL MANIFESTATIONS:**

The common symptoms of pleural effusion are pleuritic chest pain, cough, dyspnea, fever. A constant dull aching chest pain may suggest pleural malignancy. Cough is often dry and non-productive. The symptoms are more manifest where the quantity of fluid is large.

On physical examination; there is enlargement and decreased movement of the hemithorax, shifting of the mediastinum to contralateral side, decreased vocal fremitus, stony dull note over the area of pleural effusion and decreased or absent breath sounds on auscultation. Localized intercostal tenderness in a febrile patients with pleural effusion must raise the strong suspicion of “empyema thoracis”.

**INVESTIGATIONS:**
Plain chest X-ray PA view shows the classical feature of homogenous opacity of lower hemithorax with a concave upper border and obliteration of costo-phrenic & cardio-phrenic angles. Loculated effusion may produce an opacity of “D” shape.

Ultrasonography of thorax can confirm, quantify and localize pleural fluid collection and also help in aspiration. Diagnostic thoracentesis is a must to arrive at the etiology. The physical, biochemical, cytological and microbiological characterization of pleural fluid gives a diagnosis in majority of cases. The gross appearance of pleural fluid may be clear, serous, serosanguinous, turbid, or frankly purulent. Hemorrhagic pleural fluid suggests underlying malignancy, trauma or pulmonary infarction. Some cases of tubercular pleural effusion and pneumonia may show blood-tinged fluid. Measurements of pleural fluid protein content and LDH level help in differentiating effusion into exudative and transudative as per Light’s criteria. Pleural effusions are classified as exudates if they meet any one of the criteria: pleural fluid protein to serum protein ratio > 0.5; pleural fluid LDH to serum LDH ratio > 0.6; and pleural fluid LDH is more than 2/3rd the upper limits of serum LDH. Reduced values (< 50mg/dl) of pleural fluid glucose suggest tubercular, bacterial or rheumatoid effusions. Pleural fluid ADA > 40 IU is suggestive of tubercular effusion. Differential leukocyte count helps in supporting the diagnosis. Tubercular effusion has predominant lymphocytes, while predominance of polymorphonuclear cells points to a bacterial infection. Mesothelial cells and malignant cells to be searched to rule out mesothelioma and malignant pleural effusion.

**MANAGEMENT:**

a. **Tubercular pleural effusion:** This is the commonest type of pleural effusion, should be put on standard chemotherapy as per RNTCP guidelines. Therapeutic Thoracocentesis is necessary in moderate to massive effusion with respiratory distress and in hydropneumothorax/pyopneumothorax.

b. **Bacterial infections leading to pleural effusion (parapneumonic effusion):** They must be managed by appropriate antibiotic therapy. Repeated therapeutic aspirations are necessary to drain the pleural space completely. Patients with Empyema should be managed with Intercostal chest tube insertion and appropriate antibiotic therapy for 6-8 weeks.

c. **Malignant effusions:** Carcinoma lung, carcinoma breast and lymphoma are the common causes of malignant effusions. Presence of effusion in a case of malignancy indicates advanced disease, not curable with surgery or chemotherapy. Management is mainly symptomatic.

**REFERRAL:**

Some cases of pleural effusion needs referral to tertiary care centre for tube Thoracoscopy; where frank pus is aspirated, pyopneumothorax/ hydropneumothorax, disproportionate dyspnea, hemorrhagic pleural effusion, USG showing multiple loculations of fluid with debris. If a pleural fluid persists after 3 months of starting anti-tubercular therapy in cases of tubercular effusion, patients should be referred for repeat evaluation and reconsideration of etiological diagnosis.

Thoracoscopic breakdown of adhesions, thoracotomy with decortications may be required in cases of multiple loculations. Pleurodesis may help some patients with malignant effusions and should be referred to higher center for palliative treatment.
10. GASTROENTEROLOGY
DYSPEPSIA

Dyspepsia is defined as having one or more symptoms of epigastric pain, burning, postprandial fullness, or early satiety. This may be organic due to acid-peptic disorders, upper GI malignancy or functional which may again be classified as ‘ulcer-like dyspepsia’ (upper abdominal pain related to food intake), ‘dysmotility type’ (nausea, vomiting, belching, early satiety, bloating) or ‘non-specific dyspepsia’. Organic dyspepsia can be excluded by history and upper GI endoscopic examination.

Treatment Nonpharmacological
- Reassurance and patient education
- Small and frequent meals
- Avoid meals with high fat content
- Discourage smoking and alcohol.
- Avoid excess tea, coffee, and spicy foods if they aggravate symptoms.
- Avoid NSAID and aspirin
- Treat anxiety and depression if present.

Pharmacological Treatment:

Ulcer like dyspepsia
- Tab. Ranitidine 150 mg 2 times a day for 4-6 weeks. Or
- Proton pump inhibitors (Pantoprazole 40 mg or Rabeprazole 20mg or Esomeprazole 40mg tablets once daily in morning) for 2-4 weeks.

Dysmotility type dyspepsia (postprandial fullness and early satiety)
- Tab. Domperidone 10mg 3 times a day for 4-6 weeks (to be taken 30 minutes before meals). or
- Tab. Levosulpride 25 mg 3 times a day for 4-6 weeks (to be taken 30 minutes before meals).

Nonspecific dyspepsia
- Any of the above drug given alone or in combination and assess for clinical response after 4 weeks of the treatment.

Non responders:
- Trial of low dose tricyclic antidepressant in patients who failed to improve with above medications.
- If they have features of anxiety and depression, then adequate dose of tricyclic antidepressant can be tried.
- Short courses (4-6 weeks) of the drug may be repeated or alternative drug tried if patient comes back with relapse after stopping the drug. Long term treatment may be continued for up to a year and to rule out organic cause.

Alarm features [if present urgent upper GI endoscopy must be performed]:
- New onset of symptoms in someone over 50
- Family history of upper-GI malignancy
- Unintended weight loss
- GI bleeding or iron deficiency anemia
- Progressive trouble swallowing and pain with swallowing
• Persistent vomiting
• Palpable mass or lymphadenopathy, and jaundice.

Summary:
- Patients with dyspepsia who are older than 50 years of age and/or those with alarm features should undergo endoscopic evaluation.
- Patients with dyspepsia who are younger than 50 years of age and without alarm features may undergo an initial test-and-treat approach for H. pylori.
- Patients who are younger than 50 years of age and are H. pylori negative can be offered an initial endoscopy or a short trial of PPI acid suppression.
- Patients with dyspepsia who do not respond to empiric PPI therapy or have recurrent symptoms after an adequate trial should undergo endoscopy.

Patient education
- Reassure patient that this is a very common symptom and not a very serious problem.
- Avoid precipitating factors [Only Dietary substances which induce or aggravate symptoms].
- Patient should be advised to avoid mental stress and learn to relax either through exercise, yoga or music.

References:

GASTRO ESOPHAGEAL REFLUX DISEASE [GERD]
A common disorder caused by retrograde flow of gastric contents through an incompetent gastro esophageal junction. Retrosternal pain, heart burn and regurgitation mostly occurring after meals. Rarely may present with chronic cough, laryngitis recurrent pulmonary infections especially in children and bronchospasm.

- Disease is classified as mild if endoscopy reveals no or minimal oesophageal mucosal inflammation and moderate-to-severe if there are ulcers with or without stricture formation in distal oesophagus.
- Diagnosis is by history and upper GI endoscopy; 24-h pH monitoring required in only very rare difficult situations
  - Endoscopy is mandatory in a patient with GERD if any of the following symptoms are present:
    • Painful swallowing
    • Difficulty in Swallowing
    • Weight loss
    • Hematemesis or Melena
Treatment of GERD:
The treatment is based on

(1) Lifestyle modification
(2) Control of gastric acid secretion through
   • medical therapy with antacids or PPIs
   • surgical treatment with corrective antireflux surgery [rarely]

Nonpharmacological Treatment of GERD:
Life-style modification

• Losing weight (if overweight)
• Avoiding alcohol, chocolate, citrus juice, and tomato-based products.
• Stop smoking.
• Avoiding peppermint, coffee, and possibly the onion family
• Avoiding large meals; Avoiding uses water immediately after meals.
• Waiting 3 hours after a meal before lying down
• Elevating the head of the bed by 8 inches in severe cases.

Pregnant women with GERD

• Lifestyle modifications are the first line of management
• Advise patients to elevate the head of the bed
• Avoid bending or stooping positions
• Eat small, frequent meals; and refrain from ingesting food (except liquids) within 3 hours of bedtime.

Pharmacological Treatment of GERD:
A step wise approach as indicated below:

Mild gastro-oesophageal reflux
1. Antacid Gel 10-15ml or 2-3 tablets (chewed) taken 4-6 times a day ½ to 1 hour after meals may be given for a long time depending upon patients symptoms. If no relief add (2) and/or (3).
2. Tab. Domperidone 10 mg 3 times a day 30 minutes before meals for 4-6 weeks or even for longer if needed.
   Or Tab. Levosulpride 25 mg 3 times a day 30 minutes before meals for 4-6 weeks or longer if needed.
3. Proton pump inhibitors (Pantoprazole 40 mg or Rabeprazole 20mg or Esomeprazole 40mg tablets once daily in morning) for 4-8 weeks.

Follow up:
Ranitidine or Proton pump inhibitor courses may be repeated or continued for several months if patient relapses while on antacids or Domeperidone/Levosulpride. If necessary [symptoms persist off medications], then patient can take these medicines indefinitely.

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PEPTIC ULCER

Peptic ulcer is an acid-pepsin related ulceration of mucosa of stomach and duodenum.

**Salient features**

- Sharp or gnawing epigastric pain, may be worsened (gastric ulcer) or relieved by intake of food (in duodenal ulcer). Nocturnal pain may awakens the patient at midnight but early morning pain is very rare. Symptoms are recurrent and periodic.
- Complications include upper GI bleed, perforation and gastric outlet obstruction. 95% of duodenal ulcers and 60% of gastric ulcers are related to H. pylori infection and most of the remaining are related to NSAID intake.
- Diagnosis of peptic ulcer is confirmed by upper GI endoscopy. H. pylori infection may be diagnosed by serology, rapid urease test, histopathology of antral mucosa or C (13) breath test.

**Treatment:**

**Nonpharmacological measures:**

Stop smoking and avoid/minimize intake of NSAIDs.

**Pharmacological Treatment:**

It is recommended that the presence of H. pylori is confirmed before starting eradication treatment. Endoscopy is required to document healing of gastric ulcer and to rule out gastric cancer. This usually is performed 6-8 weeks after the initial diagnosis of PUD.

**H Pylori therapy**

**Preferred 10-14 days triple therapy for eradication of H. pylori regimen :**

- PPI †, 2 times a day.
  - Clarithromycin 500 mg 2 times a day.
  - Amoxycillin 1 g 2 times a day
  
  or

- Tab. PPI†, 2 times a day. Tab.
  - Clarithromycin 500 mg 2 times a day.
  - Tab Metronidazole 400 mg 2 times a day

**Sequential regimen :**

- PPI†, amoxicillin 1000 mg (each twice daily) for initial 5 days followed by PPI†, clarithromycin 500 mg, tinidazole 500 mg (each twice daily) for next 5 days

**Quadruple therapy:**

- Bismuth subsalicylate 525 mg, metronidazole 500 mg, tetracycline 500 mg (each four times daily) plus PPI† (twice daily) for 14 days

**Rescue therapy:**

- PPI†, amoxicillin 1000 mg, levofloxacin 250 mg (each twice daily) for 4 days
Standard Treatment Guidelines of Odisha 2018

Or

PPI† amoxicillin 1000 mg, rifabutin 150 mg (each twice daily) for 14 days.
†Lansoprazole 30 mg, pantoprazole 40 mg, rabeprazole 20 mg, omeprazole 20 mg, or esomeprazole 40 mg (esomeprazole can be taken once daily). All medicines to be taken 15-30 minutes before meals.

Symptomatic treatment:

PPI [Lansoprazole 30 mg, pantoprazole 40 mg, rabeprazole 20 mg, omeprazole 20 mg, or esomeprazole 40 mg] Tablet to be taken once daily 30 minutes before breakfast for 6 to 8 weeks.

Follow up:

To report to the physician urgently if vomiting of blood or passage of black tarry stools. Refer to a specialist if pain becomes continuous, frequent vomiting or symptoms of GI bleed.

Indications of surgery:

- Spurting hemorrhage which can not be stopped by endoscopic means.
- Bleeding point which can not be seen because of heavy active bleeding.
- Recurrent bleeding which appears after initial endoscopic control.
- Elderly patients with active bleeding and associated co-morbid illness.
- Peptic perforation.
- Gastric outlet obstruction.

Patient education

- Patient should avoid frequent use of unsupervised pain killers. If these are required on long term basis, a safer drug should be taken in consultation with a doctor.
- Smoking should be stopped.
- There is no use of bland diet or drinking cold milk in the treatment of peptic ulcer.

References:


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GASTROINTESTINAL (GI) BLEEDING

Gastrointestinal (GI) bleed may present as occult or overt bleed. Upper GI bleed is defined as bleeding from any site from pharynx to duodenojejunal (DJ) flexure or more specifically upto ligament of Trietz and usually presents as haematemesis or melena (black, tarry, sticky, foul smelling stools). Bleeding from GIT distal to DJ
flexure is called lower GI bleed. Presence of identifiable fresh/altered blood in stool is called haematochezia. Minimum of 60 ml of blood is required in the stomach to produce melena. Black stools may be seen in patients taking iron, charcoal or bismuth salts.

**Salient features**
- Occult or overt bleed or fresh blood.
- Diagnosis of GI bleed requires clinical history, examination and radiological/endoscopic examination.
- Active bleed is indicated by presence of fresh blood in vomitus, nasogastric tube aspirate, melena, passage of fresh blood in stool.

**Treatment**
Acute GI bleed is an emergency and needs active management. An assessment of activity and severity of bleed should be done immediately.

**Nonpharmacological Measures:**
- Maintain vital signs (blood pressure, airways, respiration, temperature).
- Insert a large bore IV cannula and sent the blood samples for Hb, TLC, platelets, coagulation profile, renal and liver function tests, blood grouping and cross-matching.
- Start IV fluids like normal saline/Ringer’s lactate/haemacoel.
- Severity of GI bleed is assessed as mild (patient has tachycardia but blood pressure is maintained), moderate (tachycardia with postural hypotension, tachypnoea, sweating, cold skin) and severe (hypotension and shock).
- Replace the blood as soon as available if moderate to severe bleed or there is active ongoing bleed.

**Pharmacological Treatment:**
1. Treatment should be initiated with Intravenous PPI (Rabeprazole 20mg 6 hourly) or Pantoprazole, 80 mg bolus and 8 mg/hour for 72 hours for 72 hours. At the same time oral PPI [Rabeprazole 20 mg daily or Pantoprazole 40 mg] should be started to maintain therapy beyond 72 hours.
2. If variceal bleed is suspected (chronic alcoholic, jaundice, splenomegaly, dilated abdominal veins, ascites, encephalopathy).
   - Inj. Octreotide 50 mcg IV immediately followed by 50 mcg/continus infusion hour infusion upto 5 days
   - Or
   - Inj. Terlipressin 1-2 mg IV given 4-6 hourly upto 5 days.

**Bleeding peptic ulcer:**
- Blood transfusions should target hemoglobin e” 8 gm / dl, with higher hemoglobin targeted in patients with clinical evidence of intravascular volume depletion or comorbidities, such as coronary artery disease.
- Discharge from the emergency department without inpatient endoscopy may be considered in patients with urea nitrogen < 18.2 mg/dl; hemoglobin greater than e” 13.0 g/dl for men (12.0 g/dl for women), systolic blood pressure >110 mm Hg; pulse < 100 beats / min; and absence of melena, syncope, cardiac failure, and liver disease, as they have < 1% chance of requiring intervention.
- Nasogastric or orogastric lavage is not required in patients with UGIB for diagnosis, prognosis, visualization, or therapeutic effect.
Patients with UGIB should generally undergo endoscopy profound within 24 hours of admission, following resuscitative efforts.

In patients who are hemodynamically stable and without serious comorbidities endoscopy should be performed as soon as possible in a non-emergent setting to identify the substantial proportion of patients with low-risk endoscopic findings who can be safely discharged.

In patients with higher risk clinical features (e.g., tachycardia, hypotension, bloody emesis or nasogastric aspirate in hospital) endoscopy within 12 h may be considered to potentially improve clinical outcomes.

**Lower gastrointestinal bleeding (LGIB)**

Lower gastrointestinal bleeding (LGIB) is a common GI problem and is defined as bleeding from a colonic source.

- It can present as
  - Acute and life threatening event.
  - Chronic bleeding, which might manifest as iron deficiency anemia, fecal occult blood or intermittent scant hematochezia.
- Anorectal disorders, neoplasms, colitis, ischemia, diverticula, angiodysplasias, and postpolypectomy bleeding are the most common causes of LGIB.
- Volume resuscitation should take place concurrently during initial patient assessment.
- Colonoscopy is the diagnostic and therapeutic procedure of choice, for acute and chronic bleeding.
- Angiography is used if colonoscopy fails or cannot be performed. The use of radioisotope scans is reserved for cases of unexplained intermittent bleeding, when other methods have failed to detect the source.
- Embolization or modern endoscopic techniques, such as injection therapy, thermocoagulation and mechanical devices, effectively promote hemostasis.
- Surgery is the final approach for severe uncontrolled bleeding.

**Bleeding piles:**

The treatment of bleeding piles is normally quite easy, though it may take some time.

1. **Toileting habits and constipation:**
   - Bowel motions / stools should be softened
   - Patient should not sit too long nor try forcing bowel movement. If nothing has come out in five minutes, patient should get up and move around. Sitting and straining puts a heck of pressure on piles, and if they are prone to bleeding, the pressure can precipitate the bleed.

2. **Sitz baths:**
   - Patient should sit in a few inches of hottish water - NOT burning hot - for 15 to 20 minutes, about four times a day. You can add some epsom salts if symptoms of aching and pain are present, and or oatmeal for just general soothing.
   - Patient should wrap ice into a towel and sit on it for 10 minutes. It works by reducing the inflammation of the piles, helps the pile not to be so prone to bleeding.

**Follow up:**

- Monitor pulse, blood pressure and urine output and activity of bleed. Presence of identifiable blood/clots per rectum in patient with upper GI bleed indicates a severe ongoing bleed and need for very active management.
Transfer patient urgently (after starting the above treatment) to a higher center where endoscopic intervention, competent surgeons are available.

References:


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CONSTIPATION

Constipation refers to persistent, difficult, infrequent or incomplete defecation associated with excessive straining and passage of hard stool.

Pathophysiological subgroups:

- Functional constipation (slow-transit constipation)
- Irritable bowel syndrome (IBS)
- Outlet obstruction.

Functional Constipation:

Rome III Criteria for Functional Constipation

Two or more of the following six must be present:*  
- Straining during at least 25% of defecations  
- Lumpy or hard stools in at least 25% of defecations  
- Sensation of incomplete evacuation for at least 25% of defecations  
- Sensation of anorectal obstruction/blockage for at least 25% of defecations  
- Manual maneuvers to facilitate at least 25% of defecations (e.g. digital evacuation, support of the pelvic floor)  
- Fewer than three defecations/week.

*Criteria fulfilled for the previous three months with symptom onset at least six months prior to diagnosis. In addition, loose stools should rarely be present without the use of laxatives, abdominal pain is not required, and there should be insufficient criteria for irritable bowel syndrome. These criteria may not apply when the patient is taking laxatives.

Risk situations, groups and factors:

- Infants and children, age >55 years  
- Recent abdominal or perianal/pelvic surgery, limited mobility  
- Late pregnancy
• Inadequate diet (fluid or fiber), medication (polypharmacy)
• Known comorbidities (neurological and structural abnormalities)
• Terminal care patients, travel, history of chronic constipation.

**Major alarm symptoms**, especially in patients over the age of 50 are new-onset constipation, anemia, weight loss, rectal bleeding, positive occult blood test, vomiting and sudden change in bowel movements. Urgent colonoscopy is indicated in these patients.

**Management:**

**Non-pharmacological:**

1. **Bowel training:** The optimal time to have a bowel movement typically is soon after walking and 30 minutes after meals when colonic activity is greatest. Regular physical exercise in the morning is beneficial.
2. **Fluid intake:** Adequate hydration is considered important to maintain bowel motility
3. **Dietary fiber intake:** The daily recommended fiber intake is 20-30 gm/day both foods included in the diet and as supplements (bran, fruits, vegetables and nuts), and/or an inexpensive osmotic agent, such as milk of magnesia or polyethylene glycol.

**Pharmacological**

1. Depending on stool consistency, the next step may be to supplement the osmotic agent with a stimulant laxative (e.g., bisacodyl or glycerol suppositories), which is preferably administered 30 minutes after a meal to synergize the pharmacologic agent with the gastrocolonic response.
2. Drugs such as lubiprostone, linaclotide and prucalopride, can be tried when symptoms do not respond to laxatives.

**Special Situations:**

**The elderly:**

• Recent unexplained constipation in an elderly whose bowel habits were always regular should be investigated to rule out malignancy.
• The main problems here are lack of mobility and polypharmacy.
• Emphasis should be placed on change of lifestyle and diet.
• For immobility, it is better to use stimulant laxatives instead of bulking agents.
• Senna-fiber combinations are more effective than lactulose.
• It is important to try and stop potentially constipating drugs.

**Pregnancy:**

• Use dietary fiber, increased fluid intake, and exercise as the main treatment options.
• Laxatives can be used if this fails.
• Use drugs only for short periods. Drug safety is the main concern in pregnancy. Bulking agents are thought to be safer than stimulants.
• Senna is considered safe at normal doses, but caution is necessary when it is used near term or if the pregnancy is unstable.
• Bulking agents and lactulose will not enter breast milk. Senna, in large doses, will enter breast milk and may cause diarrhea and colic in infants.
Children:
- Use a high-fiber diet and plenty of fluids first.
- Avoid excessive milk consumption.
- Laxatives can be given (oral therapy is best) if increased fiber and fluids fail.
- There is no robust evidence regarding which class of laxatives is superior.
- Early commencement of treatment is important, as chronic constipation can lead to mega-rectum, impaction, and overflow soiling.
- Long-term use of a stool softener is often prescribed to prevent the recurrence of fecal impaction.
- Regular use is important, as intermittent use can cause relapses.

Diabetic patients:
- Bulk-forming laxatives are safe and useful for those unable or unwilling to increase dietary fiber.
- Diabetics should avoid stimulant laxatives such as lactulose and sorbitol, since their metabolites may influence blood glucose levels, especially in patients with brittle type 1 diabetes.

Terminal care:
- Prevention of constipation is of great importance in the terminally ill.
- Dehydration and use of prophylactic laxatives is important.
- If feces are hard and the rectum is full, glycerin suppositories are recommended.
- If feces are soft, stimulant laxatives such as senna or bisacodyl can be used.

Follow up:
- If patient continues to have severe constipation or symptom worsen, refer the patient to a specialist gastroenterology centre for investigations to rule out secondary cause.

References:
11. ENDOCRINOLOGY
DIABETES MELLITUS

Diabetes mellitus refers to a group of metabolic disorders characterized by chronic hyperglycemia with disturbance of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both.

Types of diabetes:

1. **Type 1 diabetes mellitus** (due to autoimmune beta cell destruction, usually leading to absolute insulin deficiency)

2. **Type 2 diabetes mellitus** (due to a progressive loss of beta cell insulin secretion frequently on the background of insulin resistance)

3. **Gestational diabetes mellitus** (GDM) (diabetes diagnosed in the second or third trimester of pregnancy that was not clearly overt diabetes prior to gestation)

4. **Specific types of diabetes** due to other causes, e.g., monogenic diabetes syndromes (such as neonatal diabetes and maturity-onset diabetes of the young [MODY]), diseases of the exocrine pancreas (such as cystic fibrosis), and drug- or chemical-induced diabetes (such as with glucocorticoid use, in the treatment of HIV/AIDS, or after organ transplantation)

**TYPE 2 DIABETES MELLITUS**

**Introduction:** Type 2 Diabetes mellitus is characterized by insulin resistance and beta cell dysfunction leading to insulin deficiency.

**Diagnostic Criteria:**

1. Symptoms of diabetes (polyuria, polydipsia, weight loss) plus random plasma glucose concentration greater than or equal to 200 mg/dL

2. Fasting plasma glucose greater than or equal to 126 mg/dL

3. HbA1C greater than or equal to 6.5% or

4. 2-hr plasma glucose greater than or equal to 200 mg/dL during an oral glucose tolerance test

In the absence of unequivocal hyperglycemia and acute metabolic decompensation, these criteria (2, 3, 4) should be confirmed by repeat testing on a different day.

**Laboratory Investigation:**

FPG, 2hr PPG, HbA1C are the principal biochemical tests in Type2 DM. For screening of chronic diabetic complications, Urine routine microscopy, spot urine albumin/creatinine, serum urea and creatinine, serum lipid profile, Fundoscopy, NCS (Nerve conduction study) and ECG should be done.
Treatment:

A. Non-pharmacological

1. Diet- Macronutrient distribution should be individualized while keeping total calorie and metabolic goals in mind.
   - Carbohydrate: intake from whole grains, vegetables, fruits, legumes, and dairy products, with an emphasis on foods higher in fibre and lower in glycemic load
   - Protein: (1–1.5 g/kg body weight/day or 15–20% total calories) without diabetic kidney disease
   - Dietary fat: (20–35% total calories). People with diabetes should be advised to follow the guidelines for the general population for the recommended intakes of saturated fat (<7%), dietary cholesterol and transfat. Ideally transfats should be avoided.
   - Alcohol: No intake or moderate intake (no more than one drink per day for adult women and no more than two drinks per day for adult men)
   - Discourage smoking

2. Exercise- 150 min or more of moderate-to-vigorous intensity physical activity per week, spread over at least 3 days/week, with no more than 2 consecutive days without activity. Shorter durations (minimum 75 min/week) of vigorous-intensity or interval training may be sufficient for younger and more physically fit individuals.

B. Pharmacological

1. Biguanides- Metformin (500 - 2000mg) after food. Dose to be modified if eGFR<45. Should not be used if eGFR<30
2. Sulphonylurea- Glipizide(2.5-20mg/day), Glyclazide(40-320mg/day), Glimepiride(1-6mg/day), Glibenclamide (1.25-15mg/day)
3. Meglititinides- Repaglinide(0.5-16mg/day), Nateglinide (180-360mg/day)
4. Thiazolidinediones- Pioglitazone(15-45 mg/day)
5. á-Glucosidase inhibitors- Voglibose(0.2-0.3 mg/meal), Acarbose(25-100mg/meal)
6. GLP-1 receptor agonists- Exenatide(0.01-0.02mg/day), Liraglutide (0.6-1.8mg/day)
7. DPP4 inhibitors- Vildagliptin (50-100mg/day), Sitagliptin (100mg/day), Saxagliptin (5mg/day), Linagliptin (5mg/day)
8. SGLT2 inhibitors- Dapagliflozin (5-10mg/day), Canagliflozin (100-300mg/day), Emapgliflozin (10-25mg/day)
9. Insulin- Initial therapy in very high blood glucose (FPG>250 mg/dl), secondary failure to OADs, renal/hepatic disease, acutely ill or hospitalized patients and pregnancy (For insulin therapy regimen and protocols please refer to section of insulin therapy in type 1 Diabetes section.)
**Glycemic and metabolic goals:** Therapy should be individualized according to patient needs and clinical condition.

<table>
<thead>
<tr>
<th>Glycemic recommendations for nonpregnant adults with diabetes</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preprandial capillary plasma glucose*</td>
<td>80-130 mg/dL</td>
</tr>
<tr>
<td>Peak postprandial capillary plasma glucose†</td>
<td>180 mg/dL</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>&lt;130/80 mm Hg</td>
</tr>
<tr>
<td>LDL</td>
<td>&lt;100 mg/dl, ideally &lt; 70 mg/dl</td>
</tr>
<tr>
<td>TG</td>
<td>&lt;150 mg/dl</td>
</tr>
<tr>
<td>HDL</td>
<td>HDL&gt;40mg/dl in men and &gt;50mg/dl in women</td>
</tr>
</tbody>
</table>

*More or less stringent glycemic goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations.

†Postprandial glucose may be targeted if A1C goals are not met despite reaching preprandial glucose goals. Postprandial glucose measurements should be made 1–2 h after the beginning of the meal, generally peak levels in patients with diabetes.

**Patient Education:**
1. Avoid smoking, limit alcohol intake (one drink/day in women, two drinks/day in men)
2. Regular exercise and diabetic diet
3. Explain about the risk and management of hypoglycemia
4. Daily foot inspection, foot care and avoid walking barefoot.
5. Regarding micro and macrovascular complications of Diabetes and their prevention.
6. Regular follow up to monitor FPG, 2 hr PPG at each visit, BP measurement (quarterly), HbA1c testing(2-4 times/year), lipid profile and serum creatinine (annual), Eye exam(annual), foot exam(1-2times/year by physician, daily by patient).

**Referral:** Inability to achieve optimal metabolic control.

Complications that cannot be managed on site, especially ophthalmic, e.g. cataracts and proliferative retinopathy, advanced nephropathy, severe neuropathy, cardiovascular, cerebrovascular and peripheral vascular diseases.
TYPE 1 DIABETES MELLITUS

Introduction:

It is characterized by absolute insulin deficiency due to autoimmune destruction of pancreatic beta cells. Onset is mostly in childhood or in young adults.

Symptoms:

Abrupt onset of severe hyperglycemic symptoms like polyuria, polydipsia, polyphagia and weight loss. In severe cases they may present as diabetic ketoacidosis characterized by pain abdomen, vomiting, tachypnoea and/or altered sensorium.

Diagnostic Features:

1. Symptoms of diabetes (polyuria, polydipsia, weight loss) plus random blood glucose concentration greater than or equal to 200 mg/dL

or

Fasting plasma glucose greater than or equal to 126 mg/dL or

Hemoglobin A1C greater than or equal to 6.5%

or

2-hr plasma glucose greater than or equal to 200 mg/dL during an oral glucose tolerance test

In the absence of unequivocal hyperglycemia and acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day.

2. Apart from documentation of hyperglycemia using ADA criteria, measurement of beta cell autoantibody like GAD65, IA2 and IAA are crucial in establishing diagnosis.

3. Measurement of ketones and C-peptide are also helpful.

Laboratory Investigation:-

FPG, 2hr PPG, HbA1C and urine ketone are the principal biochemical tests in Type1 DM.

Treatment:

A. Non pharmacological

1. The children should be encouraged to take a well balanced diet with adequate macro and micro nutrients.

2. Simple sugars should be avoided.

3. In all children energy and nutrient intakes should be adequate to ensure normal growth and development.

4. Children with diabetes should be encouraged to engage moderate physical activity each day.
B. Pharmacological

<table>
<thead>
<tr>
<th>Insulin types</th>
<th>Onset of action</th>
<th>Peak action</th>
<th>Duration of action</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultra short acting</td>
<td>15 min</td>
<td>1 – 4 hours</td>
<td>Lispro, Aspart, Glulisine.</td>
<td></td>
</tr>
<tr>
<td>Short acting</td>
<td>30 min</td>
<td>2 – 4 hours</td>
<td>Regular human insulin</td>
<td></td>
</tr>
<tr>
<td>Intermediate acting</td>
<td>1 – 3 hrs</td>
<td>6 – 8 hrs</td>
<td>Neutral Protamine Hagedorn (NPH) insulin</td>
<td></td>
</tr>
<tr>
<td>Biphasic</td>
<td>30 min</td>
<td>1 – 2 hrs</td>
<td>Insulin 30/70, Insulin 50/50</td>
<td></td>
</tr>
<tr>
<td>Long acting</td>
<td>60 min</td>
<td>No peak</td>
<td>Upto 24 hrs</td>
<td>Glargine, Detemir</td>
</tr>
</tbody>
</table>

**Insulin protocols**

1. **Basal bolus regimen**

   All type 1 diabetics should preferentially be managed with combined intermediate/long acting (basal) and short-acting insulin (bolus) or so-called basal bolus regimen. This consists of pre-meal short-acting insulin and bedtime intermediate/long acting insulin not later than 22:00 hours.

   The initial total daily insulin dose: 0.6 units/kg body weight. The dose is divided into:

   - 40-50% basal insulin, the rest as bolus insulin split equally before each meal. Adjust dose on an individual basis.

2. **Pre-mixed insulin**

   Twice daily pre-mixed insulin, i.e. a mixture of intermediate- or short-acting insulin provides adequate control, when used with at least daily blood glucose monitoring. It is a practical option for patients who cannot monitor blood glucose frequently.

   In visually impaired patients, prefilled syringes may be used.

**Home glucose monitoring**

T1 DM patients should measure glucose at least twice daily. All patients should be taught about importance of glycemic control, blood glucose monitoring and hypoglycemia management.

Blood glucose and A1C goals for type 1 diabetes across all pediatric age-groups
Blood glucose goal range

1. Before meals 90–130 mg/dL
2. Bedtime/overnight 90–150 mg/dL
3. A1C* < 7.5%

*A lower goal (7.0%) is reasonable if it can be achieved without excessive hypoglycemia

Key concepts in setting glycemic goals:

1. Goals should be individualized, and lower goals may be reasonable based on a benefit–risk assessment.
2. Blood glucose goals should be modified in children with frequent hypoglycemia or hypoglycemia unawareness.
3. Postprandial blood glucose values should be measured when there is a discrepancy between preprandial blood glucose values and A1C levels and to assess preprandial insulin doses in those on basal–bolus regimens.

Referral:

Inability to achieve optimal metabolic control or patient is in DKA.

Onset of diabetic complications that cannot be managed on site.

Reference:


GESTATIONAL DIABETES MELLITUS (GDM)

GDM is diabetes that is first diagnosed in the second or third trimester of pregnancy that is not clearly either preexisting type 1 or type 2 diabetes. Women diagnosed with diabetes in the first trimester should be classified as having preexisting pre-gestational diabetes (type 2 diabetes or, very rarely, type 1 diabetes).

Screening:

Any woman at high risk of diabetes (marked obesity, previous history of GDM, impaired glucose metabolism or glycosuria, or a strong family history of diabetes) should be tested for diabetes using standard diagnostic criteria as soon as possible after confirmation of pregnancy.

All women who have not already been diagnosed with pre-existing diabetes or gestational diabetes should undergo a 75-g, 2-h oral glucose tolerance test at 24–28 weeks gestation.

Laboratory Investigation:

Diagnostic criteria- Perform a 75-g OGTT, with plasma glucose measurement when patient is fasting and at 1 hour and 2 hour, at 24–28 weeks of gestation in women not previously diagnosed with overt diabetes. The OGTT should be performed in the morning after an overnight fast of at least 8 h.

The diagnosis of GDM is made when any of the following plasma glucose values are met or exceeded:
Fasting: 92 mg/dL
1 hour PG: 180 mg/dL
2 hour PG: 153 mg/dL

Management:

The mainstay of therapy is the dietary prescription. The goal of medical nutrition therapy (MNT) is to keep the peak postprandial glucose response in the normal range.

Criteria for beginning insulin are based on the fasting and postprandial responses to the prescribed meal plan. There is gradual increase in insulin requirement throughout pregnancy.

Both short-acting insulin (regular insulin) and rapid-acting insulin analogues can be used.

The rapid-acting insulin lispro and aspart have greater postprandial control, reduced hypoglycaemia and greater convenience of administration than regular insulin.

Glucose monitoring is paramount in the woman with gestational diabetes. All women diagnosed with GDM need to be taught to self-monitor their blood glucose concentration, and perform daily tests fasting and 1-hour after meals.

Aim to maintain the glucose level- FPG < 95mg/dl, 1hr PPG < 140mg/dl, 2hr PPG < 120mg/dl.

Though metformin and glibenclamide are used in GDM management, its routine use is not advocated by the ADA.

Timing of delivery:

Timing and mode of delivery are determined not only by the obstetric indications but also by the glycemic control of the mother.

Immediately following birth, insulin requirement falls precipitously to below the pre-pregnancy levels. Insulin dose should be reduced at this time and to monitor blood glucose frequently.

A glucose tolerance test 6–8 weeks postpartum should be performed to ensure that the woman is not left with pre-diabetes or type 2 diabetes mellitus (T2DM).

Patient Education:

Exercise programs are considered safe as an adjunct therapy for GDM.

A smaller, more frequent meals leads to better satiety and compliance, with a reduction in postprandial glucose peak.

A prevention program should be started immediately postpartum to keep the woman lean and fit, which markedly decreases the chances that she will develop T2DM as she ages.

Breast feeding provides health advantages for both mother and child.

Referral Criteria:

Such patients should ideally be referred to tertiary care center for obstetric and glycemic management.

Reference:


COMPLICATIONS OF DIABETES MELLITUS

A. Acute
1. HHS (Hyperglycemic Hyperosmolar State)
2. DKA (Diabetic Ketoacidosis)
3. Hypoglycemia

B. Chronic

HYPERGLYCEMIC HYPEROSMOLAR STATE (HHS)

HHS is a medical emergency that usually occurs in an elderly individual with type 2 DM with several-week history of polyuria, weight loss and diminished oral intake that culminates in severe dehydration, mental confusion, lethargy or coma. Often precipitated by a serious, concurrent illness such as myocardial infarction or stroke or sepsis.

Diagnostic Features:

On physical examination, there is profound dehydration with hypotension, tachycardia and altered mental status. Nausea, vomiting, abdominal pain and Kussmaul’s respiration that are characteristic of DKA are absent.

Laboratory investigations:

Marked hyperglycemia (plasma glucose 600-1200 mg/dL), hyperosmolality (>350mosmol/L) and prerenal azotemia. The measured serum sodium may be normal or slightly low despite the marked hyperglycemia. The corrected serum sodium is usually increased (add 1.6 meq to measured sodium for each 100-mg/dL rise in the serum glucose). In contrast to DKA acidosis and ketonemia are absent or mild.

Treatment:

1. Fluid replacement

Fluid loss and dehydration in HHS is more pronounced than DKA. Initially stabilize the hemodynamic status of the patient (1-3 L of 0.9% normal saline over the first 2-3 h). Too rapid a reversal may worsen neurologic function. If the serum sodium is >150 meq/L, 0.45% saline should be used. Once hemodynamic stability is achieved, thereafter the IV fluid administration is directed at reversing the free water deficit. The calculated free water deficit (which averages 9-10 L) should be reversed over the next 1-2 days (infusion rates of 200-300 mL/h of hypotonic solution).

2. Insulin therapy:

IV regular insulin bolus of 0.1 unit/kg followed by IV insulin at a constant infusion rate of 0.1 unit/kg per hour. If the serum glucose does not fall, increase the insulin infusion rate by twofold. Glucose should be added to IV fluid when the plasma glucose falls to 250 mg/dL and the insulin infusion rate should be decreased to 0.05-0.1 unit/kg per hour. The insulin infusion should be continued until the patient has resumed eating and can be transferred to a SC insulin regimen.

3. Replace Potassium:

KCL 10 meq/h when plasma K+ < 5.0-5.2 meq/L; 40-80 meq/h when plasma K+ < 3.5 meq/L. If initial serum potassium is > 5.2 meq/L, do not supplement K+.
4. Treatment of underlying precipitating event

5. Monitoring- Monitor blood pressure, pulserespiratory rate, mental status, fluid intake and output every 1 -4 hour.

Mortality rate in HHS is higher than DKA.

DIABETIC KETOACIDOSIS (DKA)

Ketoacidosis is an acute complication of diabetes, usually occurring in type 1 but can also occur in type 2.

Clinical features:
DKA often occurs in younger patients and develops over hours to days. There may be nausea, vomiting, abdominal pain, dehydration and deep rapid breathing (Kussmaul’s sign), altered sensorium and ultimately coma and death if left untreated.

Diagnostic criteria:
- Blood glucose usually > 250mg/dl but < 600 mg/dl
- Urine ketones positive (Blood ketones are positive)
- Serum osmolality < 320 mOsm/L.
- Acidosis with arterial pH usually <7.3
- Low bicarbonate < 15mmol/l.

Treatment:

GENERAL MEASURES
Set up an intravenous line.
Protect airway and insert a nasogastric tube, if unconscious.
Access fluid loss, degree of acidosis, Monitor urine output.
Monitor plasma glucose, urine ketone, CBC, renal function, electrolytes and arterial blood gas.
Look for precipitating causes, e.g. infection or missed insulin dose

MEDICAL TREATMENT

Fluids
Average deficit is around 6 litres, but may be as much as 12 litres. If renal or cardiac disease is present, monitor with central venous pressure.
In the absence of renal or cardiac compromise: In adults - Sodium chloride 0.9%, IV, 15–20 ml / kg in the first hour. For patients < 20 years of age, initial volume: 10–20 ml/kg in the first hour. Subsequent infusion rate varies from 5–15 ml/kg/hour depending on the clinical condition. Correction of estimated deficits should take place over 24 hours. The volume infused in the first 4 hours should not exceed 50 mL/kg. Reduction in serum osmolality should not exceed 3mOsm/kg/hour

Caution
Cerebral edema may occur with over-aggressive fluid replacement or rapid sodium change.

Potassium
Potassium will fall on insulin treatment and patients with DKA have potassium depletion even if initial potassium is normal or high. It is therefore essential to monitor and replace potassium. An useful guide for potassium
supplementation is 40 mmol (when K+ < 3.5 mmol/L), 20 mmol (when K+ = 3.5–5.5 mmol/L) and no supplementation when K+ > 5.5 mmol/L.

**Bicarbonate**
Use is restricted only when severe acidosis.

**Insulin therapy**
Patients should be preferentially managed with insulin in a high care ward, with appropriate monitoring. Ketonaemia takes longer to clear than hyperglycaemia and combined insulin and glucose (and potassium) are needed to ensure clearance of ketonaemia.

**Insulin Protocol: Continuous intravenous infusion**
Insulin, short-acting, IV infusion, 50 units in 200 ml sodium chloride 0.9%. 4 mL solution = 1 unit insulin. Initial infusion: 0.1 unit /kg /hour. Usually 5–7 units/hour (20–28 ml/hour).

If plasma glucose does not fall by 54mg/dl (3 mmol/l) in the first hour, double the insulin infusion (hourly) until a steady reduction of plasma glucose is achieved, i.e. at least 54-72mg/dl (3–4 mmol/l) per hour.

If plasma glucose < 250mg/dl, reduce the insulin infusion rate to 0.05–0.1 units / hour and add D5/2 NS solution.

Continue treatment until the acidosis has resolved and the patient is able to eat. Subcutaneous insulin therapy is instituted either at previous doses or, for newly diagnosed diabetes at 0.5–1 unit/kg total daily dose divided into at least 2 doses with mixed short and long acting insulin (biphasic insulin 2/3rd in the morning and 1/3rd at night).

**HYPOGLYCEMIA**
Hypoglycemia is, by definition, a plasma glucose concentration low enough (usually < 70 mg/dl) to cause symptoms or signs, including impairment of brain function.

It is commonly seen in diabetics with sulphonylurea or insulin overdose, alcohol abuse, renal failure or poor food intake.

**Diagnostic Features:**

**Clinical**

Symptoms:
- Anxiety, sweating, palpitations, hunger, headaches and behavioral changes

Signs:
- Sweating, confusion, tremor, tachycardia, seizures and ultimately coma if left untreated.

**Laboratory Investigation:**
- Blood Glucose < 70 mg/dl on finger prick blood glucose. Confirm with laboratory measurements if uncertain.

**Treatment:**

**At home**
Start immediately.
- Oral sugary drinks or glucose solution if patient is able to swallow (20-50 mg in 200 ml water).
Drug Treatment

In hospital:
1. Dextrose 50%, rapid IV injection, 50 mL infusion should be started. Assess clinical status and finger prick glucose level over the next 5–10 minutes. Further infusion of 5-10% Glucose IV 500 ml 4 hourly until the blood glucose sufficiently rises and patient is able to eat normally a meal.
2. Alternatively Inj. Glucagon 1 mg IM may be tried.

Patient Education:
- Educating about the symptoms of hypoglycemia and managing with oral glucose drinks.
- Insulin dosing adjustment in case of decreased appetite or vomiting.

Referral Criteria:
- In case of recurrent hypoglycemia should be referred to find underlying cause.
- If the patient has not regained consciousness after 30 minutes with a normal or elevated blood glucose should be referred to look for other causes of coma.

HYPOTHYROIDISM

Introduction:
Clinical condition which occurs due to decreased availability of thyroid hormones (T4 & T3). It may be due to primary (autoimmune / iatrogenic) or secondary causes (destruction of pituitary/hypothalamus).

Diagnostic features:
- Mild to moderate cases- Dry skin, hoarse voice, facial puffiness, weight gain, cardiac enlargement/effusion, prolonged relaxation phase of deep tendon reflexes, goitre (in cases of primary hypothyroidism)
- Severe and prolonged - Myxoedema coma characterized by hypothermia, hypoventilation, hyponatremia, hypoxia, hypertension or hypotension

Investigation:
1) An elevated serum TSH is sufficient to diagnose primary hypothyroidism, however serum free T3 & free T4 are needed for secondary hypothyroidism diagnosis because TSH may be low normal.
2) Free T3, free T4 levels- Low, Serum TSH level-High in primary but low in secondary hypothyroidism.

Treatment:
1) Non pharmacological treatment-avoidance of exposure to cold in elderly and consumption of iodized salt intake.
2) Pharmacological treatment-Initiate Tabletevo-thyroxine 25mcg/day. The dose may be enhanced by 25mcg/day at weekly intervals till the patient improves clinically (mean replacement dose is 1.5-2mcg/kg/day).

Precautions
- Higher dose may be started in newborn and younger patients. In elderly and patients with cardiac disease thyroxine needs to be given with caution. Levothyroxine dose has to be enhanced on gradual escalation in this group of patients. In pregnancy thyroxine has to be continued throughout with a dose requirement of 20-25% more than the pre-pregnancy dose. Response to therapy is to be assessed clinically and by serum TSH measurement at 8 weekly intervals. Once euthyroid state is achieved, follow up at 6-12 months intervals is usually sufficient.
Pregnancy and hypothyroidism

1) Women with hypothyroidism should be euthyroid before planning to conceive.
2) Levothyroxine dose requirement increases by 20% during pregnancy in hypothyroid women on levothyroxine supplementation.
3) Levothyroxine dose titration in pregnancy is to be done by monitoring the trimester specific ranges of TSH (first trimester-0.1-2.5miu/ml; second trimester-0.2-3.0 miu/ml; third trimester 0.3-3.0miu/ml)

Myxedema coma

Myxedema coma is the ultimate stage of severe long standing hypothyroidism, especially in elderly patients during winter months associated with high mortality.

Treatment of Myxedema coma:

These patients are to be managed carefully as they take longer time for recovery. Underlying precipitating causes (stroke, infection, MI) should be promptly identified and treated accordingly.

1) Warm blankets, mechanical ventilation for respiratory failure, correction of electrolyte disturbances, are to be done as immediate measure.
2) Levothyroxine 500-800mcg as a loading dose replete the peripheral hormone pool and may cause improvement within hours. Daily dose of i.v levothyroxine 100mcg are given thereafter. If IV preparations are not available then same dose can be given through Ryle’s tube. Once acute phase is over, maintenance dose with oral thyroxine (1.5-2micgm/kg/day) needs to be continued.
3) In acute phase Inj hydrocortisone 100mg iv to be started and then 25-50mg tid to be given, subsequently steroid to be withdrawn as patients recovers.
4) Broad spectrum antibiotics may be administered

Patient education:

Levothyroxine has to be taken early morning in empty stomach at least 60 min before food; lifelong replacement therapy may be needed on individualized basis with regular monitoring; over treatment leads to reduced bone mineral density and adverse cardiac effects.

Referral criteria:

Patients with secondary hypothyroidism require detailed investigations; patients of myxoedema coma after resuscitation should be referred to tertiary care hospital.

References:

1. Braverman.L, Cooper.D-Werner &Ingbars;The thyroid a fundamental & clinical text, 10th edition-pg775-803.
HYPERTHYROIDISM

Introduction:
The clinical state which occurs due to excess production of thyroid hormone from the thyroid gland as in Graves’ disease, toxic multinodular goitre & toxic adenomas. Whereas thyrotoxicosis is defined as a clinical state of thyroid hormone excess due to any aetiology including use of exogenous drugs.

Diagnostic features:
- Sweating, tremors, unexplained weight loss despite good appetite, heat intolerance, wide pulse pressure, sinus tachycardia and atrial arrhythmias, worsening of angina or cardiac failure may predominate in older patients. Elderly may also manifest with features of apathetic thyrotoxicosis. Graves’ disease is characterized with cardinal features of goitre, ophthalmopathy & dermopathy.

Investigations:
- Low to undetectable serum TSH and increased serum total/free thyroid hormones

Treatment:
1) Adjunctive treatment- For adrenergic symptoms like sweating, tremor & tachycardia- Tab propranolol 40mg gradually titrated up to 120mg.
2) Antithyroid drugs (ATD): Tab Carbimazole- 10mg-20mg every 8-12hrs; after euthyroid state achieved in 6-8 weeks once daily dose may be tried; Tab Methimazole 10-40mg once daily; Tab Propylthiouracil 100-150 mg every 8 hours, preferred in first trimester pregnancy
3) Definitive treatment is surgery or radioactive thyroid ablation (surgery-young patients with large goitres, radioactive iodine-elderly, young patients with recurrent thyrotoxicosis, small sized goitres or when surgery is contraindicated.

Precautions
- Hyperthyroidism in specific situation needs to be carefully monitored due to increased risk of adverse events. These include:
  1) If patients on ATD develop sore throat with high grade fever, agranulocytopenia needs to be ruled out.
  2) Monitoring of pregnant women with hyperthyroidism on ATD needs to be done due to increased risk of both fetal hypo and hyperthyroidism.
  3) Risk of worsening of eye signs needs to be explained.

Patient education:
1) ATD carry risk of adverse effects such as, allergic rash, drug sensitivity, hepatitis, drug fever and arthralgias.
2) Smoking and tobacco abuse to be stopped in cases with pre existing ophthalmopathy.

Thyrotoxic crisis/thyroid storm
- Life threatening condition due to exacerbation of hyperthyroidism characterized with features of fever, vomiting, diarrhoea, jaundice, delirium and coma. Usually precipitated by acute illness like stroke, infection, trauma, patients undergoing surgery, DKA and radioiodine treatment in a poorly prepared patient. Treatment includes - Tab PTU 600mg loading, then 150mg-200mg every 6 hourly orally or through Ryle’s tube (Tab Carbimazole 15-25mg 6 hourly may be used as an alternative); SSKI 3-5 drops qid or Lugol’s iodine 5-10 drops tid; beta-blockers (Tab Propranolol) in doses of 80-120 mg/day in divided doses, Inj Dexamethasone 2mg iv qid.
Patient referral:

Ideally all the cases must be referred to tertiary centre especially elderly, pregnant women and children with features of thyrotoxicosis.

References:
1. Braverman.L, Cooper.D-Werner &Ingbars; The thyroid a fundamental & clinical text, 10th edition-pg775-803.

ADRENAL INSUFFICIENCY

Introduction:

It is caused due to deficient production of glucocorticoids and/or mineralocorticoids and their resultant effect on our body. They can be due to defect in adrenals (primary adrenal insufficiency) or pituitary defect (secondary adrenal insufficiency).

Diagnostic features:

Acute crisis

Fever, vomiting, dehydration, weakness, pain abdomen, altered sensorium, hypoglycemia, hyponatremia, hyperkalemia, acidosis and hypotensive shock

Chronic

Hyperpigmentation, GIT disturbances, weakness and fatigue, hypotension, loss of weight, hypoglycemia, postural dizziness, hyponatremia

Lab Investigations:

A serum 8 AM cortisol level (or at time of presentation in acute crisis): > 20 µg/dl: virtually excludes the diagnosis. A value of <3 µg/dl is highly suggestive of hypoadrenalism.

Values from 3-18 µg/dl are indeterminate and require an adrenocorticotropic hormone (ACTH) stimulation test for confirmation.

ACTH 250 µg iv with blood sampling at 60 minutes, if serum cortisol level: > 20 µg/dl –Normal response

Treatment:

General Measures

All patients should wear a notification bracelet.
Medical Treatment

Acute crisis

Exclude sepsis.

To maintain adequate intravascular volume guided by blood pressure: Sodium chloride 0.9%, IV infusion. Hydrocortisone, IV, 100 mg 6 hourly. Change to oral maintenance therapy once stable.

Monitor glucose levels closely and treat hypoglycemia if present.

Chronic

As maintenance therapy:

Oral Hydrocortisone. Start with 10 mg in the morning and 5 mg at night. Increase the dose according to clinical response up to 20 mg in the morning and 10 mg at night.

Alternatively oral Prednisone can be given. Start with 5 mg daily. Increase to maximum of 7.5 mg daily, if necessary.

For patients who have primary adrenal insufficiency and remain symptomatically hypotensive:

Oral Fludrocortisone 50–100 µg daily. Monitor response to therapy with symptoms, blood pressure and electrolytes.

Patient education:

During times of severe “stress” i.e. acute illness, surgery, trauma, etc: IV Hydrocortisone 100 mg start and referral to tertiary care center.

With minor stress maintenance therapy dose should be doubled for the duration of illness and gradually tapered to usual dose.

Referral:

All suspected cases for full evaluation.

Addisonian Crisis.

Reference:


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12. OPHTHALMOLOGY
ACUTE PAINLESS RED EYE

CAUSES
conjunctivitis, episcleritis, subconjunctival haemorrhage, inflamed pinguecula, pterygium.
Lid abnormalities, e.g. trichiasis, entropion, blepharitis, meibomianitis, ectropion, lagophthalmos

CONJUNCTIVITIS
Infective or Inflammatory conjunctivitis by bacteria / Chlamydia / virus or Allergic conjunctivitis.

BACTERIAL CONJUNCTIVITIS
Manifests as acute mucopurulent, purulent, angular or membranous conjunctivitis.

ETIOLOGY
Staphylococcus aureus, Haemophilus aegyptius, Streptococcus pneumoniae,

DIAGNOSTIC CRITERIA
Unilateral or bilateral red eye, conjunctiva congestion, mucopurulent / purulent discharge, sticky lids, matting of cilia, no photophobia in uncomplicated cases.

NON-PHARMACOLOGICAL TREATMENT (PATIENT EDUCATION)
Do not patch or bandage eye, use dark glasses, maintain good ocular hygiene-wipe eye with clean water 3-4 times a day. Patient’s towel, handkerchief should not be shared. The examiner must wash hands after examining patient, tonometer should be disinfected each time.

PHARMACOLOGICAL
Eyedrops Ciprofloxacin 0.3% or Chloramphenicol 0.5% or Gatifloxacin 0.3% e/d 1 drop 4-6 hourly during day time and eye ointment at bedtime for 5-7 days. Tab Ibuprofen 300mg 3 times a day for 2-3 days.

LAB TEST
If there is no response to above therapy after 7 days, take conjunctiva swab for Gram’s stain, culture and sensitivity.

OPHTHALMIA NEONATORUM
Acute purulent conjunctivitis of the newborn, during first 28 days of life, may be gonococcal or non-gonococcal (milder disease occurring within 5-14 days after birth by Herpes simplex II and Chlamydia in 80% cases) and is acquired from the maternal birth canal. Complications like corneal blindness, cataract, nystagmus, endophthalmitis or panophthalmitis if untreated.

TREATMENT
NON-PHARMACOLOGICAL
Irrigate conjunctiva sac hourly with warm normal saline before antibiotic instillation until the discharge is eliminated, wipe away the discharge with moistened cotton wool.

PHARMACOLOGICAL (GONOCOCCAL OPHTHALMIA NEONATORUM)
Treat gonococcal infection as recommended. Topical Tobramycin / Gentamycin 0.3% e/d hourly or Ciprofloxacin 03% eyedrops every 2 hourly and ointment at night for 10 days. If corneal involvement -see section on Corneal ulcer. Also treat mother with systemic therapy, treat Chlamydia infection simultaneously. Suspect ophthalmia neonatorum if there is any mucopurulent discharge from eyes during first week.

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VIRAL CONJUNCTIVITIS

Adeno Viral conjunctivitis often occurs as Epidemic Conjunctivitis with Conjunctival congestion, chemosis, watery discharge, conjunctival haemorrhages, preauricular lymphadenopathy.

TREATMENT


PHARMACOLOGICAL

1. Antibiotic eyedrops (as in mucopurulent conjunctivitis) prevent secondary infection.
2. Naphazoline 0.05% eyedrops 4 times a day.
3. Carboxy Methylcellulose eye drop 1 drop 6-8 times a day.
4. Antivirals not effective but oint. Acyclovir/Gancyclovir 5 times/day 7 days effected in Herpes.
5. Corticosteroids are contraindicated.

REFERRAL

Refer to an ophthalmologist, if no response in 7 to 10 days, corneal haze, uveitis develop

PATIENT EDUCATION

Same as for bacterial conjunctivitis. It is usually self-limiting. Not to share towels, handkerchief and other objects with other persons as highly contagious.

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II. CONJUNCTIVAL ALLERGIC DISORDERS

Bilateral allergic disorders include acute allergic conjunctivitis (Hay fever conjunctivitis- seasonal allergic conjunctivitis, perennial allergic conjunctivitis), atopic keratoconjunctivitis, vernal keratoconjunctivitis, phlyctenular keratoconjunctivitis, conjunctivitis medicamentosa, etc.

SYMPTOMS

Itching, watery/stringy discharge, conjunctival papillae

TREATMENT

NON-PHARMACOLOGICAL

Frequent instillation of tear substitutes and cold compresses to eye.

PHARMACOLOGICAL

1. Topical combination of antihistamine and vasoconstrictor (Naphazoline hydrochloride 0.05%) eyedrops QID, Ketorolac tromethamine 0.5% or Olopatadine eye drops 0.1% twice a day or 0.2% once daily till the resolution of symptoms.
2. Disodium cromoglycate 4% eyedrops BD or 2% eyedrops QID till resolution
3. If severe, systematic antihistaminic should be administered. Tab. Cetrizine hydrochloride 10mg once a day for duration of acute symptoms, in children, 5mg once a day.
4. Prednisolone sodium phosphate 1% eyedrops or Dexamethasone 0.1% or Betamethasone 0.1% QID for 3 days, twice daily for 3 days, once daily for next 3 days and then discontinue Or diluted steroids, dexamethasone
eyedrops 0.01% or Loteprednol eyedrops 0.2% four times a day till acute symptoms subside and then tapered over 2 weeks.

PATIENT EDUCATION (CAUTION): Avoid allergen or minimize exposure to allergens, wear protective goggles outdoor. Topical corticosteroids are contraindicated as a first line therapy. If required should only be advised by ophthalmologist, in low concentrations and monitoring.

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ACUTE PAINFUL RED EYE

ACUTE INFLAMMATION WITH LOCALIZED PAIN / TENDERNESS WITHOUT VISUAL LOSS
– dacryoadentitis / dacryocystitis, lid abscess, cellulitis, hordeolum, herpes Zoster ophthalmicus

ACUTE INFLAMMATION WITH DIFFUSE PAIN / TENDERNESS WITH MILD VISUAL LOSS - conjunctivitis, keratitis, scleritis, anterior uveitis, orbital cellulitis.

ACUTE INFLAMMATION WITH PROFOUND VISUAL LOSS - acute congestive glaucoma and secondary glaucoma, endophthalmitis

DIAGNOSTIC CRITERIA: -

Moderate to severe pain of sudden onset
Localised or diffuse tenderness, redness and swelling as in dacryoadentitis / dacryocystitis, lid abscess, hordeolum
Moderate to severe visual loss as in anterior uveitis, acute congestive glaucoma and secondary glaucoma, endophthalmitis

Proptosis and restricted movement -, orbital cellulitis
Eruptive skin lesions on forehead- herpes Zoster ophthalmicus
Circumcorneal congestion- keratitis, anterior uveitis, acute congestive and secondary glaucoma
Pupil- small, sluggish, irregular as in uveitis or dilated vertically oval, fixed as in glaucoma, CRAO

ALL PAINFUL RED EYE WITH VISUAL LOSS SHOULD BE REFERRED IMMEDIATELY TO A TERTIARY CARE LEVEL OR AN OPHTHALMOLOGIST.

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ORBITAL CELLULITIS

Occurs more frequently in children from paranasal sinuses (ethmoid sinuses commonest), dental abscess, ear, face and lid infection, panophthalmitis, dacryocystitis, dacryoadentitis, postoperative to any facial or ocular surgery, perforating injury and haematogenous spread etc

TREATMENT: -

1. Treat as for cellulitis – antibiotics are changed according to culture and sensitivity report and continue IV antibiotics for 1-2 weeks. Switch to oral therapy for 2-3 weeks. 2. Symptomatic therapy for pain: antipyretics and analgesics in usual doses. 3. Lubricating eye drops / artificial tears: 1-2 hourly or antibiotic eye ointment 5 times a day.
ANTERIOR UVEITIS / IRIDOCYCLITIS –
Inflammation of iris and ciliary body – small, sluggish, irregular pupil, KPs on endothelium.

TREATMENT:
1. Dexamethasone or Betamethasone 0.1% eyedrops or prednisolone phosphate/acetate 1% e/d 1-2 hourly tapered gradually based on slit lamp evidence of AC activity. If required long term, switch over to e/d Loteprednol 0.5% or fluoromethalone 0.1% over 6wks
2. Homatropine hydrobromide 2% e/d or Atropine sulphate 1% e/o/nt once or twice a day
3. If no response within 1 week, severe anterior uveitis, bilateral involvement & panuveitis – Tab Prednisolone 1mg / kg or 40-80 mg/day orally every day or on alternate days. Gradually taper depending upon satisfactory clinical response over 2-4 weeks and periocular corticosteroids – subconjunctival or posterior subtenon injection preferred.
4. If IOP elevated – Timolol maleate 0.5% e/d BD & / or tab Acetazlamide 250mg qid
5. Systemic immunosuppressives/immunomodulators if non responsive to maximal steroids
6. Pilocarpine, Latanoprost are contraindicated

PATIENT EDUCATION: Recurrent episodes of anterior uveitis & steroid therapy may lead to various complications particularly complicated cataract & steroid-induced glaucoma. Patients with history of uveitis, juvenile rheumatic arthritis, ankylosing spondylitis should be instructed to report to an ophthalmologist.

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CORNEAL ULCER (ULCERATIVE KERATITIS)

Superficial Suppurative inflammation of cornea frequently occurring in eyes with some predisposing factors is called corneal ulcer.

ETIOLOGY – Bacterial, fungal or mycotic, viral, Acanthamoeba.

DIAGNOSTIC CRITERIA: Acute inflammatory features with diffuse pain / tenderness, watering, photophobia with mild visual loss-ciliary congestion, corneal haziness / ulcer with slough/ abscess, hypopyon, iritis, secondary glaucoma, superficial corneal vascularisation.

COMPLICATIONS: corneal thinning, ectasia, descemetocoele, secondary glaucoma, perforation and its sequelae including endophthalmitis or panophthalmitis and loss of eye

TREATMENT (TO BE MANAGED BY AN OPHTHALMOLOGIST)

Corneal ulcer is stained green with fresh fluoroscein 2%. Perform corneal scrapings from base and edges of ulcer for Gram and Giemsa stains, KOH mount for fungi, culture and sensitivity test.

Initiate therapy based on clinical picture and findings obtained on smears of corneal scrapings.

As a first line therapy, broad-spectrum antibiotics are started in all cases, treatment is modified according to the clinical response and result of culture and sensitivity of microorganisms.
NON PHARMACOLOGICAL TREATMENT
i. Avoid patching. Maintain proper ocular hygiene by regular cleaning of discharge. Removal of contributory factors, e.g. trichiasis, foreign body, entropion, dacryocystitis,
ii. Debridement of slough, BCL or tissue adhesives (cyanoacrylate) if base is thin
iii. Prevention and treatment of complications – secondary glaucoma, uveitis. start empirical therapy and refer to an ophthalmologist.

BACTERIAL CORNEAL ULCER
- Cefazolin 5% eyedrops (50mg/ml) [mix 5ml of distilled water in 250mg of Cefazolin]. Solution or Topical fortified prepared fresh) tobramycin 14mg/ml (1.4%) instilled 1 drop every 30 minutes or 1 hourly round the clock for at least 24 hours.
- Gatifloxacin or moxifloxacin or tobramycin eye drops instilled 1 drop every 30 min – 1 hourly round the clock for at least 24 hours or ciprofloxacin or Ofloxacin 0.3% drop every 2 hours.
- Frequency of administration is reduced according to the response and continued for 2-3 weeks.
- Atropine sulphate 1% eye ointment to be applied 2 or 3 times per day.
- Parenteral antibiotics are indicated in perforated corneal ulcer, impending perforation, corneal ulcer following perforating injury and infections causes by Neisseria or Haemophilus

(Caution : Corticosteroids are contraindicated)

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 FUNGAL CORNEAL ULCER
(MYCOTIC KERATITIS)

Develops – 1-2 weeks following corneal injury with an organic or vegetative mater.

ETIOLOGY-Fungi in order of frequency are Aspergillus, fusarium, Candida, curvularia.

SALIENT FEATURES: Severe signs, less symptoms, dry rough appearance with feathery margins, satellite lesions, immune ring, endothelial plaque, thick immobile infected hypopyon.

TREATMENT:
1. Natamycin 5% suspension 1-2 hrl 24-48 hrs then 4-6 hourly for 2-3 weeks or till resolution of keratitis.
2. Amphotericin B 0.25% formulation prepared in distilled water, in case of immunocompromized patients, spreading ulcer, perforation or impending perforation instil every 15 to 30 minutes for 24-48 hours then 1-2 hourly continued for for 2-3 weeks or till resolution
3. Cap. Ketoconazole / Fluconazole 200mg or Itraconazole 150mg- 2 times a day for 2-3 weeks with liver function tests
4. Since superadded bacterial infection is common, add antibiotic eye drops.

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VIRAL CORNEAL ULCER (HERPES SIMPLEX KERTITIS)

It is characterized by unilateral or bilateral recurrent attacks of keratitis in the form of epithelial keratitis, stromal keratitis, or endothelitis, etc. The attack is often precipitated by trivial trauma; fever, cold, emotional stress, menstruation, etc.

TREATMENT (EPITHELIAL KERTITIS)
1. Acyclovir eye ointment 3%/Gancyclovir ophthalmic gel 0.15% five times a day for 2-3 weeks (not required for adenovirus as it is self-limiting). In refractory cases, tab Acyclovir 200/400/800mg five times a day till healing of ulcer, followed by 3 times daily for 7 days
2. Topical cycloplegics – Homatropine 2% eye drops 2 times a day.
3. Broad – spectrum antibiotic as treatment of mucopurulent conjunctivitis till ulcer heals
4. Artificial tear substitutes 3-4 times a day.

REFERRAL- if more than two recurrences occur. Tab. Acyclovir 400mg 2 times a day for 3-6 months for prevention of recurrence.

NON HEALING ULCER

PATIENT EDUCATION
Avoid corneal injury by protective eyewear. Avoid self medication. Avoid traditional remedies-cow’s urine, honey, juice, milk, kajal. Prompt visit for eye check up on fall of foreign body.

SURGICAL PROCEDURES
- cyanoacrylate glue with bandage contact lens (BCL)
- Conjunctival flap, amniotic membrane graft, therapeutic or tectonic keratoplasty

REFERRAL CRITERIA
- No improvement in signs & symptoms after 48 hours.
- Whole cornea is hazy, large ulcer, severe ocular trauma, other ocular structures affected
- Intractable pain, impeding perforation / perforated ulcer

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ACUTE ANGLE CLOSURE GLAUCOMA

SALIENT FEATURES
Acute pain, redness and blurring of vision in the affected eye, halos around lights along with headache and vomiting in some cases, corneal oedema (hazy cornea), shallow anterior chamber and vertically oval dilated pupils with very high IOP.

PHARMACOLOGICAL TREATMENT
1. Inj. Mannitol 20%, 1–2 g/kg, IV infusion over half an hour 2-3 times / day or Oral Glycerol 50%, 1-1.5 g/Kg in 50% solution orally, mixed with lemon or orange juice in 3-4 divided doses (Caution: It can cause hyperlycemia in diabetic patients. Do not drink water for 1 hour after; contraindications- dehydration, BHP or cardiac decompensation).
2. Pilocaprine 2% eyedrops every 15 min for 1 hour and thereafter 6 hourly started after IOP has been lowered by hyperosmotics as above.

3. Tab. Acetazolamide 500 mg stat followed by 250mg every 6 hours and maintained till the definitive treatment of laser peripheral iridotomy relieves the pupillary block.

4. Timolol 0.5% eyedrops 2 times a day (if pressure is still high) to be continued till surgery Or Betaxolol 0.5% eyedrops 2 times a day (preferred in asthmatics and patients with cardiac conduction defects). (Caution : all mydriatics / cycloplegics which dilate pupil are contraindicated)

REFERRAL

Once the IOP falls to early 20s by above treatment – usually in a day or so, evaluate it by gonioscopy, disc cupping and visual field charting. Definitive treatment is iridotomy by laser or surgery. Prophylactic laser PI on the fellow eyes as soon as possible.

PATIENT EDUCATION - Do not ignore headache and chronic ache in the eyes and report to eye specialist if coloured halos appear around light.

- Topical beta-blockers need to be used with caution in chronic obstructive pulmonary disease, myasthenia gravis, cardiac arrhythmias, diabetes mellitus, etc.

CHRONIC ANGLE CLOSURE GLAUCOMA

IOP is raised due to progressive angle closure or by repeated intermittent sub acute attacks secondary to pupillary block. Commonly asymptomatic, in spite of high IOP until significant visual loss. The presentation is thus more akin to open angle glaucoma.

PHARMACOLOGICAL TREATMENT

Timolol 0.25% - 0.5% or Betaxolol 0.25% - 0.5% eyedrops BD, Pilocarpine 2-4% eye drops 4 times a day usually required for life.

SURGICAL:

Laser / surgical PI in both eyes. If glaucoma still uncontrolled on maximal tolerable medical therapy (2 topical anti glaucoma medications), do filtering surgery or trabeculectomy.

GLAUCOMA

Glaucoma is an optic neuropathy related to intraocular pressure (IOP) which manifests as typical optic disc changes & visual field defects which are signs of progress of the disease and benchmark for assessing response to therapy. Width of the angle of anterior chamber differentiates the glaucoma into open or closed angle type as treatment modalities differ.

CONGENITAL GLAUCOMA/BUPHTHALMOS

Classic triad of manifestations, any one of which should arouse suspicion of glaucoma in an infant or a young child, includes epiphora, photophobia blepharospam and an enlarged cornea with haziness within the first few years. IOP is usually normal as sclera in children distends leading to increased corneal diameter.

PHARMACOLOGICAL TREATMENT

Aims is to control IOP till definitive treatment, i.e. surgery is performed. Timlolo/Betaxolol drops 0.25% - 0.5% eyedrop BD, Tab. Acetazolamide 12mg/kg in QID doses.
SURGICAL TREATMENT at a tertiary care centre includes trabeculotomy with trabeculectomy. Monitor corneal diameter, IOP, disc changes and refraction periodically.

PATIENT EDUCATION.

It is a slowly progressive disease, usually amenable to surgery. Regular follow-up lifelong is must for early detection of any failure or complications. Eye is vulnerable to trauma and thus contact sports may be restricted in these children. Screening of any child particularly the siblings who have a large cornea, photophobia or excessive watering.

PRIMARY OPEN ANGLE GLAUCOMA (POAG)

Sailent features: IOP is above 21 mm Hg, associated nerve head cupping and visual field defects. On Gonioscopy angle of AC is widely open. Usually asymptomatic, may complain of frequent change in spectacles, mild ache of the eyes.

PHARMACOLOGICAL TREATMENT:-

Timolol 0.5% or Betaxolol 0.5% or Brimonidine tartarte: 0.2% - 1 drop BD, If initial therapy fails, Latanoprost 0.005% drops at bed time, Bimatoprost 0.03% / Travaprost 0.004%.

REFERRAL

If he IOP is not controlled with 2 topical drops then consider laser trabeculoplasty or glaucoma filtering or trabeculectomy, Check IOP, Optic nerve head every 3 months and visual field at 6 monthly intervals.

PATIENT EDUCATION:

Avoid instillation of more than 1 drop of eye drop, Most drops especially beta blockers cause burning and stinging sensation, dry eyes. Punctual occlusion i.e. pressing medial end of the lower lid to increase drug time. In asthmatics/cardiac disease do not give timolol eye drop. High risk individuals i.e high myopia, Large cups, asymmetric cups, patient more than 40 years, with family history of glaucoma or diabetic should routinely get fundus and IOP evaluated annually.

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CATARACT

Cataract refers to opacification of crystalline lens or its capsule at any age, most common is senile cataract.

SALIENT FEATURES

Gradual painless progressive diminution of vision in one or both eyes. Excessive glare, monocular diplopia/polyopia, coloured halos around lights, diurnal variation in vision, fixed black spots before eyes. Torch light examination – greyish white or white lenticular opacity. No proven drug treatment exists, definitive treatment is lens extraction. In early cataract, decreased vision may be improved by accurate refraction and spectacles.

SURGICAL TREATMENT

PREOPERATIVE EVALUATION

GENERAL - examination by physician, FBS, PPBS, Hb, BT, CT, BP, HbSag, HIV
LOCAL - r/o focal sepsis, do LPI, IOP, SLIT LAMP examination, funduscopy, USG B scan to rule out posterior segment pathology. Biometry

Indications of lens extraction are visual handicap, interference in patient activities even if cataract is immature-glare, reduced contrast.

Urgent lens extraction in developmental, traumatic, mature / hypermatuare cataract to prevent further complication such as amblyopia, glaucoma, iritis, or drop of lens.

Standard extracapsular extraction (SICS) / phacoemulsification with PC IOL in capsular bag.

IMMEDIATE PREOPERATIVE TREATMENT
1. e/d NSAID- Flurbifen or Ketorolac with antibiotics 3-4times
2. lid scrubbing with 10% povidone iodine-3 times, instillation of 5% povidone iodine in conjunctiva
3. local peribulbar anaesthesia is preferred. May be done under GA for children, uncooperative pt and evaluated accordingly

POSTOPERATIVE TREATMENT
1. Ciprofloxacin 0.3% eye drops 4 times a day or Moxifloxacin 0.3% eye drops 1 drop QID – 7 days.
2. Dexamethasone 0.1% Prednisolone 1% eye drops 1 drop 2 hourly to 4 times a day depending on the inflammation with tapering over 4-6 weeks with declining frequency every week.
3. NSAID drops like Nepafenac, Bromefenac
4. Mydritics as per the operative procedure and advice of the surgeon.

PATIENT EDUCATION
Do not wait for maturation as complications may develop if total cataract remains un operated. Modern good quality Surgery is done free of cost in many centres with minimal hospital stay NdYAG laser for posterior capsulotomy done free of cost in many centres in OPD if needed for post op capsular opacification

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REFRACTIVE ERRORS & STRABISMUS (SQUINT)

Refractive errors (ametropia) includes – myopia / hypermetropia / astigmatism / presbyopia are the optical defects of eye in which the light entering the eye do not come to focus on fovea centralis.

CHARACTERIZED BY – blurring of vision / asthenopia (eye strain) / headache / latent or manifest squint.

TREATMENT & PATIENT EDUCATION
Refraction followed by glasses / contact lens / surgery – if spectacles are not used timely, patient may develop irreversible visual loss – amblyopia, squint –

Young patients (20 years and above) opting for laser correction should wait till the refraction is stable for at least 1 year.

Squint is not spontaneously correctable with age. Therefore, treatment should not be delayed.
REFERRAL
1. for funduscopy in pathological myopia –
2. for low vision / blind rehabilitation.
3. Any child presenting with strabismus should have refraction should under full cycloplegia
4. rule out cause of media opacity e.g. cataract / corneal opacity / retinoblastoma,
5. treat Amblyopia with occlusion therapy or other modality before surgery.

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VITAMIN A DEFICIENCY (XEROPHTHALMIA)

DIAGNOSTIC CRITERIA
Bilateral affection of night blindness followed by conjunctival xerosis, Bitot’s spots (triangular foamy white patches on interpalpebral conjunctival area), corneal dryness, keratomalacia-sterile melting of cornea which may perforate in severe cases. Mostly affects malnourished children, precipitated by acute illness like measles, severe diarrhoea, pneumonia

LAB TESTS - routine blood tests, stool for helminthiasis

TREATMENT
1. <6 months of age : 3 doses of oral vitamin A, 50000 IU each immediately on diagnosis, the next day and at least 2 weeks later.
2. 6 – 12 months : 3 doses of oral vitamin A, 100000 IU each immediately on diagnosis, the next day and at least 2 weeks later.
3. >12 months age : 3 doses of oral vitamin A, 200000 IU each immediately on diagnosis the next day and at least 2 weeks later.
4. All sick children, severe malnutrition, having measles given additional dose of vitamin A.
5. Women of reproductive age group having night blindness or Bitot’s spots <10000 IU daily or 25000 IU weekly for at least 3 months.
6. Water miscible vitamin A is given IM for children suffering from persistent vomiting, severe diarrhoea, and intestinal parasites, dose being half of oral dose.
7. Gentamicin / Tobramycin eyedrops 14mg/ml drop 4 hourly.
8. Artificial tears 6-8 times/day.
9. Regular stool examination and deworming.
10. Improve immunity, general and ocular hygiene

REFERRAL CRITERIA
1. If cornea becomes hazy, Keratomalacia is treated as corneal ulcer as medical emergency.
2. If night blindness is not reversed with treatment after 6m, rule out hereditary retinal diseases, retinitis pigmentosa.

PATIENT EDUCATION
1. Regular consumption of food rich in Vit A as fresh dark green leafy vegetables, yellow and orange fruits, milk, fish.
2. Exclusive breastfeeding for 6 months.

3. Vitamin A prophylaxis:
   - 100000 IU at 9 months with measles vaccine.
   - 200000 IU at 16-18 months with DPT booster.
   - 200000 IU every 6 months thereafter up to the age of 5 years.

4. Excessive consumption of vitamin A supplements can cause hypervitaminosis A.

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**DIABETIC RETINOPATHY**

Diabetic Retinopathy (DR) is the microangiopathy of retinal vasculature occurring in long-standing diabetes mellitus. It is classified into non-proliferative DR and proliferative DR;

**DIABETIC MACULAR OEDEMA** may be present at any of these stages – profound visual loss.

**TREATMENT**

**NON-PHARMACOLOGICAL**

Early diagnosis, proper diabetic control & follow-up fundus photography, FFA, OCT and timely intravitreal injections, laser photocoagulation or viteroctomy surgery or both.

**PHARMACOLOGICAL**

No time tested and proven pharmacological treatment exists which can delay, prevent or cure diabetic retinopathy.

**PATIENT EDUCATION:**

- All diabetics, irrespective of duration of diabetes should have funduscropy done yearly.
- 3 monthly or frequent fundus examination in proliferative DR, macular edema.
- Laser therapy can prevent deterioration of vision, cannot reverse vision deficit.
- Early diagnosis, proper diabetic control & follow-up.
- Daily Amsler Grid home testing and urgently report if black patch / distortion of vision in high risk patients.

(for details and prevention of complication of diabetes, see section on diabetes Mellitus).

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13. OBST & GYN
ANTENATAL CARE AND MANAGEMENT OF NORMAL PREGNANCY

Systematic supervision of a woman during pregnancy is called antenatal care.

Diagnostic criteria:
- Cessation of menses,
- Nausea, vomiting, frequency of micturition
- Breast enlargement and tingling sensation and secretion from nipples

a) First & early second trimester:
- Bluish discoloration of vagina (Jaquemier, Chadwick Sign)
- Softness of cervical lips (Goodell’s Sign)
- Pulsation in the lateral fornices (Osiander’s Sign)
- Enlarged uterine body feels separate from cervix and apposition of fingers in vagina with those placed in the abdomen at 6-10 weeks of pregnancy. (Hager’s Sign)
- Regular rhythmic contraction elicited by bimanual examination. (Palmer’s Sign)

b) Late II & III trimester:
- Gradual enlargement of abdomen with striae and pigmentation
- Fetal movements
- Palpable foetal parts
- Audible foetal heart sounds.

Laboratory tests:
- Urine test for hCG.
- Blood Grouping and Rh typing
- Blood Hb%
- Screening for HIV, HBSAg, HCV, Syphilis (VDRL Test)
- TSH- < 2.5 mIU/ml in 1st trimester, < 3 mIU/ml in 2nd & 3rd trimester.
- Diabetes screening with 2 hrs post 75g oral glucose (< 140 mg/dl).
- Urine analysis for albumin, sugar, pus cells and culture.

Ultrasoundography:
1. 1st Trimester - Gestation sac with foetal node seen. Location, viability & chorionicity determined.
2. 2nd Trimester- 18-20 wks to rule out congenital malformation.
3. 3rd Trimester- Foetal biophysical profile & placental location.

Management:
Non drug treatment:
Antenatal visits:
- Ideal: Monthly till 28 weeks, fortnightly till 36th week, weekly till EDD
Minimal: 3 Visits; with at least 2 visits in the 3rd trimester.

Antenatal check-ups: (What to see)

General: Pallor, oedema, weight gain (0.5-0.75 kg/wk), B.P. (< 130/90mm/Hg).
Obstetrical: Symphysio-fundal height in cm. (corresponding to week of gestation)
  Liquor status, uterine contour and irritability, feel of foetus,
  Lie, presentation, position of foetus  (Leopold’s maneuver),
  Head descent (Crichton’s fifths palpable)
  Auscultation of F.H.S (24 weeks onward)
  Pelvic assessment after 37th week.

Drug treatment:
- Inj.T. Toxid 0.5ml, 2 doses during ANC given 4-6 weeks apart, last dose at least 1 month prior to EDD.
- Tab. Ferrous sulphate 200mg (60mg elemental Iron) 1 tab daily from 20 weeks to end of lactation.
- Tab. Folic acid 5mg, 1 tab daily throughout 1st trimester.
- Tab. Calcium carbonate 500mg 2 tabs daily starting from 16th to 20th week, till end of lactation; 4 tabs daily in Hypertensive (P.I.H) women.

Patient Education:

Healthy Diet: Green leafy vegetables, pulses, soybeans, fruits and salads, milk, egg, fish juices 8 glasses per day (more in summer). No extra salt to hypertensives.

Exercise: Continue routine work, No heavy exercise or Work in last few weeks.
  Light exercises can be done.

Rest: Sleep for 8 hours at night, 2 hours of rest in the afternoon, lie on flanks after 20 wks.

Coitus: Prohibited in pregnancy with threatened abortion, placenta praevia, and preterm labor.

Addiction: Tobacco or alcohol intake should be stopped.

Medication: No drug should be used in the first 12 weeks unless absolutely necessary. (Anti – T.B., Anti – epileptics to continue, OHA should be changed to insulin, Anti-malarials now mandatory by Dept. of Health & F.W., Odisha, Directive.)

Danger Signals: Immediate medical attention to be sought in case of:
  Vaginal bleeding, persistent vomiting, fever, oliguria, swelling of face, fingers, persistent severe oedema of ankles, severe continuous headache, dizziness and blurred vision, abdominal pain, loss of fetal movements, watery vaginal discharge.

Referral Criteria:
  All high risk pregnancies like those associated with:
  Ante-partum hemorrhage, Severe Anemia, Severe Hypertension, Heart Disease, Diabetes, Bad Obstetric History, Previous caesarean Section, Contracted Pelvis, Multifoetal Pregnancy, Gross IUGR, Malpresentations, Gross Polyhydramnius, Previous V.V.F Repair, Prolapse Repair, Myomectomy, Repair of Uterus, Assisted Reproductive Techniques.
MANAGEMENT OF NORMAL LABOR

Introduction: A normal labor is one in which the live fetus presents by vertex, begins spontaneously at term and terminates naturally without any artificial aid and without complication.

Diagnostic criteria:
- Painful uterine contraction (at least 1 in 10 mins., lasting for 20 secs.)
- Blood stained mucoid discharge per vaginum.
- Progressive effacement & dilatation of the cervix.
- Formation of the bag of waters.

Stages of labor:
- Stage I  Cervical dilation 16-18 hrs in primi
  6-8 hrs in
  Multi-approximately
  Latent phase = 0-4 Cm
  Active phase = 4-10 Cm
- Stage II  Expulsion of Fetus. 1-2 hrs in primi
  20 mins in multi
- Stage III  Expulsion of after –births. 10-15 mins

Non–Drug management:
- Stage I  Exclusion of cephalo-pelvic disproportion.
  Assessment of feto – maternal condition at 0 hr.
  B.P., pulse recording 4 hrly.
  F.H.S. ½ hrly
  Cervical dilation 4 hrly.
  Decent of head (P/A) & station of head (P/V) 4 hrly.

Patient is advised to be ambulatory in latent phase.
In active phase –left – lateral position with I.V. line is advocated or any position in which the patient is comfortable.
Diet –only clear fluids.
Encourage to empty bladder frequently.
Reassurance.
Partographic monitoring of active phase of labor in which ‘Fetal condition and Progress of labor’ is monitored. Modified WHO Partograph is easy to use and very helpful as managerial and referral tool.

Stage II—Note—
Time of Onsent.
Descent of Head and Poles.
Stage III:
- Immediate administration of uterotonic agents wait till next contraction and signs of placenta separation
- Delivery of placenta by Modified Brandt Andrews Technique.
- Inspection of placenta for Cotyledons and Vessels leading to secundenturate lobe, if present remove them manually with full aseptic measures.
- Uterine massage.
- PPIUCD should be inserted after placental delivery as when required.

Transfer from Labor Room:
Check the vital parameters of the mother. Look for bleeding from vagina & inspect the genitalia.

Drug Treatment:

Stage I: Inj. Drotaverine 1amp. I.M once or Inj. Tramadol 1amp. I.M once
Inj. Diazepam 10 mg, 1 amp. Once.
Inj. Oxytocin 2.5 U in 500 ml of 5% Dextrose Soln. I.V drip at a rate of 15 dps/min.
(5 milliunits per min.) if acceleration of labor required.
No antibiotic routinely required unless indicated.

Stage II: Inj. Oxytocin 2.5 in 500 ml of 5% Dextrose Soln. I.V drip at a rate of 15 dps/min.
May increase drop rate if required but not go beyond 60 dps/min (16mU/min) prior to delivery foetus, otherwise uterus may rupture.

End of Stage II or Stage III: Inj. Oxytocin 10 IU I.M.
Inj. Ergometrine 0.2 mg, 1 amp. I.V. Not to be given to hypertensives.
2nd dose to be given in case of PPH. No 3rd dose before another 8hrs interval.
Inj. Carboprost tromethamine 250 U, 1 amp. I.M. only to hypertensives or in case of non-response to ergometrine, but contraindicated in asthmatics. A 2nd Dose may be repeated after an interval of 15 Mins.
Tab. Misoprostol 600mcg per rectal.

Patient education:
Attend the near –by hospital for delivery.
Throughout soap bath prior to labor.
Not to strain or bear-down before full dilatation.
During first part of labor take deep breathe out through mouth.
During labor, when bearing down stage arrives bear down as if defecating during contraction and take deep breath during relaxation.
Uterine massage should be done by the mother herself.
Exclusive Breast –feeding and burp after feeds. Care of New-born.
Neonate taken to warm, clean, dry place.
Nothing to be applied on cord. Handle baby with clean hands.

Referral Criteria:
Abnormal Presentation.
- Breech presentation.
- Oblique Lie etc.
Prolonged Labor (Stage I>18 hrs., Stage II>2hrs.)
Fetal Distress in Stage I.
Features of Obstructed Labor.
Shoulder Dystocia
Heart Disease.
Post–partum Haemorrhage.(when primary interventions fail to control)
   Morbidly adherent Placenta.
   Inversion of Uterus.
   Suspected Rupture of Uterus.
During the referral process on-going control measures like I.V. fluids, Indwelling Catheter, Uterine Massage.
Escalating Doses of Oxytocin (till 100 Units is reached) in case of Atonic P.P.H and Retained Placenta.
Vaginal pack in Traumatic P.P.H, must be instituted simultaneously.

**ANAEMIA IN PREGNANCY**

Hemoglobin concentration of a pregnant woman less than 11 gm % (W.H.O)

<table>
<thead>
<tr>
<th>Type</th>
<th>Hemoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild Anaemia</td>
<td>Hb 9-11 gm %</td>
</tr>
<tr>
<td>Moderate Anaemia</td>
<td>Hb 7-9 gm %</td>
</tr>
<tr>
<td>Severe Anaemia</td>
<td>Hb &lt; 7 gm %</td>
</tr>
</tbody>
</table>

Over 90 % of anaemias are due to Iron Deficiency.

**Diagnostic Features:** Weakness and Easy Fatiguability
- Breathless on Exertion
- Pallor ad Koilonychia
- Varying degrees of Oedema
- Tachycardia and Rapid thready pulse
- Sign of Heart Failure may be present.
  (Gallop rhythm, Enlarged Tender Liver, Basal Creps., Ascitis)

**Laboratory Investigation:**
- Blood Tests: Hb-less than 11 gm %
  - RBC Count.
  - M.P.
  - Peripheral smear examination
- Stool Exam: Ova or Cysts
  - Occult Blood

**Non Drug Treatment:** Rest
Diet rich in Iron-Fresh Vegetables and Fruits sprouted pulses, meat, cooking food in Iron Utensil.
Diet rich in Protein-pulses, milk & milk products, nuts.
Drug Treatment:

1. Tab. Ferrous Sulphate 200mg (60mg of Elemental Iron) 1 Tab. Twice daily for 3 months after anemia is cured and until breast feeding continues. Not to be taken with meals.
   
   In Severe anemia – Tab. Ferrous Sulphate 200 mg 1 Tab. Thrice daily till Hb% improves.

If Patient is Intolerant to Oral Iron and When Non-Compliance is suspected-

   I.V. Iron Preparations to be given.

   Total dose of Iron Requirement = [2.4 X Weight in Kg. X Hb. Deficit gm%] + 500mg for Iron Storage.

   I.V. iron preparation – Inj. Iron Sucrose. It is given as infusion of 200mg in 100ml of Normal Saline I.V. Infusion over 20 to 30 mins on alternate day. Max. 600 mg per week can be given. Oral Iron Therapy is suspended at least 24 hours prior to therapy to avoid reaction.

   Monitoring of adverse reaction like rigors, chest pain & hypotension should be done. If present stop the infusion.

2. Tab. Folic Acid 5 mg daily.

3. Tab. Albendazole 400mg, 1 tab chew at bedtime once during 2nd or 3rd trimester
   
   Repeat after 12 weeks in highly endemic areas of Hookworm Infection.

4. Tab. Chloroquin phosphate 300mg (150mg base) 2 Tab. Per week till delivery.

In Severe Anaemia

Hospitalization.

Blood Transfusion (Packed cells)

Oxygen Inhalation

Other Supportive Measures.

Frusemide 40 mg Tab./Inj. daily

Patient Education:

Correct Cooking Habits to avoid loss of Vit. C and Folate.

Not to defecate in fields.

Precaution against Malaria.

Counseling regarding Misconception in taking Iron during Pregnancy.

Oral Iron may cause Epigastric discomfort, Nausea Vomiting, constipation or Diarrhoea. Parenteral Iron may cause Staining of Skin, Fever, Allergy, Arthralgia.

Report for ante-natal Check-up to monitor improvement of Anaemia & Fetal well-being.

Plan for Institutional delivery.

Referral Criteria:

Severe Anaemia.

Heart Failure due to Anaemia.

Other varieties of Anaemia

   (other than Iron deficiency Anaemia)

Associated Obstetric Complications.

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HYPERTENSIVE DISORDERS OF PREGNANCY

Introduction:
Hypertension complicates 5 to 10% of all pregnancies. The spectrum of hypertensive disorders of pregnancy include:-

- **Chronic hypertension** (hypertension before 20 weeks of gestation),
- **Gestational hypertension** (development of new hypertension that is BP ≥ 140/90mmHg for the 1st time in pregnancy after 20 weeks of gestation without proteinuria);
- **Pre-eclampsia** (gestational hypertension plus proteinuria)
- **Eclampsia** (pre-eclampsia with convulsion).
- **Chronic hypertension with superimposed Pre-eclampsia & Eclampsia**

Mild Hypertension: ≥ 140-<150/ ≥ 90-<100 mmHg recorded at 4-6 hours interval.

Moderate hypertension: ≥ 150-<160/ ≥ 100-<110 mm Hg

Severe Hypertension: ≥ 160/110 mm Hg)

PRE ECLAMPSIA:

- More common in primigravida.
- Women with multifetal gestation, obesity, family history of hypertension, hydramnios & molar pregnancy are at increased risk.

Diagnostic Criteria:

- Hypertension (blood pressure > 140/90 mmHg recorded on two occasions at 4-6 hours interval or single reading of >160/110 mm Hg) with proteinuria and/or non-dependant oedema developing after 20 weeks of gestation in a previously Normotensive non-proteinuric patient.
- Proteinuria - presence of urinary protein in amounts exceeding 0.3 g in a 24-hour urine collection or more than 1 g/l (significant proteinuria) in random urine sample by dipstick. (Trace = 0.1 g/l, 1+ = 0.3 g/l, 2+ = 1 g/l: 3+ = 3 g/l, 4+ = 10 g/l.)
- Oedema is excluded from the diagnostic criteria unless pathological. However, presence of pitting oedema over the ankles after 12 hours of rest and/or rapid gain in weight of >0.5 kg/week or >2 kg/month may be early features.

Clinical Types:-

- **Mild Pre-eclampsia** when BP ≥ 140 - <150/ ≥ 90 - <100 mmHg recorded on two occasions at 4-6 hours interval with proteinuria trace.
- **Moderate Pre-eclampsia** when BP ≥ 150-<160/ ≥ 100-<110 mm Hg with proteinuria trace to 2+ with minimal elevation of liver enzymes.
- **Severe Pre-eclampsia** when BP ≥ 160/110 mm Hg, 24-hr urinary proteins 3+ on two occasions 4 hours apart, severe headache, visual disturbance, epigastric or right hypochondriac pain, oliguria (urine output <500 ml/24 h). Biochemical parameters: elevated serum Uric acid; thrombocytopenia (platelet count <50,000/mm³), microangiopathic hemolysis, raised liver enzymes and fetal growth restriction.
HELLP is considered a variant or complication of pre-eclampsia and has three main features of the syndrome: haemolysis, elevated liver enzymes and low platelet counts.

**Treatment:**

All cases should be hospitalized. Definitive therapy is to terminate pregnancy. The choice between immediate delivery and expectant management depends on:

1. Severity of disease
2. Condition of mother and fetus
3. Period of gestation (POG)

**Mild pre-eclampsia (BP >140/90 but <150/<100 mmHg)**

- Follow-up twice a week.
- **Refer and admit at CHC or tertiary care centre, if** significant proteinuria, pregnancy <32 weeks, unreliable patient, platelets <2 lakhs, elevated urine albumin, S/D ratio of umbilical artery >3, fetal compromise (IUGR, oligohydramnios).
- Consider delivery at 37 weeks.

**Pharmacological:**

**Antihypertensive** treatment is started if there is persistent hypertension > 150/100 mmHg. If already on ACE inhibitor or Angiotensin Receptor Blocker change to following antihypertensive. Aim of treatment is to achieve a diastolic BP around 90-100 mmHg.

1. **Tab. Labetalol** 100 mg twice daily increased up to 2400 mg/day.
   Or
   **Tab. Methyldopa** 250 mg twice a day; may be increased to 1 g 6 hourly depending upon the response.
2. If BP is not controlled in 72 hours with the above add any of the following:
   **Tab. Nifedipine retard** 10-20 mg 12 hourly (max 60-120 mg/day) (Caution: Do not give nifedipine by sublingual route.)
   (Caution: ARBs and ACE inhibitors are foetotoxic; thus are contraindicated in pregnancy.)
3. Low dose aspirin 75 mg/day, 12 weeks onwards if previous history of pre-eclampsia or gestational hypertension, pre-existing vascular disease or pre-existing kidney disease and stop at 34 weeks of pregnancy.
4. More than 1.5g Calcium helps to reduce severity of the disease if previous history of pre-eclampsia or gestational hypertension, pre-existing vascular disease and continue up to term.

**Monitoring:**

- Daily monitoring of weight gain, BP, urine albumin, urine output.
- **Weekly lab investigations** - haemogram with platelet count, liver and kidney function tests specially serum uric acid,
- Fundoscopy.
- Clotting profile in case of non-severe pre-eclampsia and gestational hypertension is not necessary if the platelet count is normal.
- Fetal monitoring by clinical and USG growth assessment, daily fetal movement count, non-stress test twice weekly and biophysical score weekly, Doppler studies in IUGR.
Pulsatility index (PI) > 1.7 of uterine arteries at 14 weeks scan predicts severity of disease, future fetal restriction, accidental hemorrhage which is prevented by low dose aspirin.

**Definitive management:**

Termination of pregnancy by labor induction/caesarean section should be done in the following conditions:
Gestational age e•37 weeks, fetal compromise like severe growth restriction, oligohydramnios, abnormal non-stress test or biophysical score, maternal compromise like development of features of severe pre-eclampsia, onset of labor, rupture of membranes or bleeding.

**B. Hypertensive emergency/crisis in pregnancy:**

Acute-onset, persistent (lasting 15 minutes or more), severe systolic (greater than or equal to 160 mm Hg) or severe diastolic hypertension (greater than or equal to 110 mmHg) or both in pregnant or postpartum women.

**Treatment (At PHC):**

**General measures** - Keep her in a quiet room in bed, position her on the left side.

**Start treatment immediately**

1. Cap Nifedipine 10 mg orally (not sub lingually), can be repeated after 30-60 minutes. Maximum dose 20 mg 4 hourly
2. Prophylactic anticonvulsant Inj. Magnesium Sulphate loading dose 50% of 4 g diluted to 20% (8 ml drug with 12 ml NS) to be given slowly IV in 5 minutes. 5 g IM (50%) each buttock (total 10 g).

Refer to CHC/higher centre once stable with a trained staff and record of treatment.

However, if a woman with a hypertensive disorder of pregnancy presents in the early first stage of labor, refer her to an FRU. If the woman is in the late first stage or second stage of labor, conduct the delivery and start medical treatment, stabilise and only then refer the woman for further management if needed along with a trained staff.

**Severe preeclampsia/hypertensive emergency (At referral hospital)**

Treatment is preferably done in a tertiary care centre or CHC.

- Observation in intensive care unit for 24 hours.
- Assessment of maternal and fetal conditions. BP monitoring 2-4 hourly, hourly urine output monitoring, watch for sign and symptoms of impending eclampsia and fetal distress.
- **Investigations:** Haemogram with platelet count, liver and kidney function tests, urinary proteins, coagulation profile, fundus examination, obstetric ultrasound with BPP.
- Patient be kept nil orally and in imminent eclampsia cases intravenous fluids like Ringer’s lactate at a rate of 80 ml/h to be given.

**Pharmacological:**

1. **Immediate management:**

   Goals of treatment - gradual lowering of blood pressure to target BP >150/90-100 mmHg (NOT to normalise), decreasing proteinuria.

   Tab. Nifedipine 10 mg orally can be repeated after 30-60 minutes (maximum dose 20 mg 4 hourly).

   **(Caution:** Side effects - tachycardia, headache, flushing, and aggravation of angina. Rapid fall in BP can cause fetal distress; Nifedipine should NOT be given sublingually to a woman with hypertension.)
If BP is not controlled with oral treatment, then IV drugs are started with intensive monitoring.

Inj. Labetalol 20 mg IV bolus over 2-minute period, followed by 40 mg after 10 min then 80 mg after further 10 min or until desired effect (max up to 220 mg)

Or

IV infusion - add 200 mg in NS so as to make total 200 ml infusion (1 mg/ml) start @ 20 mg/h (20 ml/h) (5-6 drops/min), double every 20 minutes until desired effect (BP < 150/90-100 mm ofHg), maintenance dose 40 mg/h (10-12 drops/min)

If injection is not available, give Tab. Labetalol 200 mg orally and can be repeated in 30 minutes if an appropriate improvement is not observed.

Or

If labetalol is not available or not effective, Inj. Hydralazine 5 mg IV every 30 minutes, maximum dose 30 mg.

2. Maintenance therapy. After initial control of acute hypertension, patient is started on maintenance therapy with antihypertensives as described in management of mild pre-eclampsia.

3. Prophylactic anticonvulsants in women with severe pre-eclampsia especially in cases with signs and symptoms of impending eclampsia. Dose is same as for eclampsia. Loading dose is Magnesium sulphate-4 g IV as 20% solution over 5minutes and 10g intramuscular as (50%) solution, 10 ml (5 g) in each buttock (total of 14 g) followed by second dose 5 g in alternate buttocks every 4 hours for maintenance. Before each dose monitor for presence of patellar reflex, respiratory rate> 16/min and urine output > 25 ml/h. It should be discontinued after 24 hours after BP is lowered if expectant management is planned.

4. Corticosteroids for fetal lung maturation: If birth is considered likely within 7 days in women with pre-eclampsia, give two doses of betamethasone 12 mg IM 24 hours apart in women between 24 and 34 weeks. Also give two doses of betamethasone 12 mg intramuscularly, 24 hours apart in women between 35 and 36 weeks. Dexamethasone or betamethasone may be used in HELLP syndrome.

After initial evaluation and stabilisation of the patient, definitive management is decided depending on fetal maturity and maternal response:

Expectant management. Considered if pregnancy is between 24 and 34 weeks and hypertension controlled with maximum of two drugs, urine output is normal, lab investigations are normal and no fetal compromise. Patient should be hospitalised till delivery.

Managed as mentioned in the mild pre-eclampsia with antihypertensives, bed rest and more frequent maternal and fetal monitoring.

Definitive management is termination of pregnancy:

- Pregnancy beyond 34 weeks - stabilise maternal condition and terminate pregnancy.
- Pregnancy less than 24 weeks - stabilise maternal condition and terminate pregnancy.
- Terminate Pregnancy if patient on expectant management develops following features: Uncontrolled hypertension despite maximum dose of 2 antihypertensive drugs, eclampsia, and raised liver enzyme >2 times with right upper quadrant pain and tenderness, pulmonary oedema, platelet <1 lac/mm3, creatinine> 1 mg/dl over baseline, persistent headache, vomiting and visual disturbance suggestive of impending eclampsia, papilloedema and fetal compromise.
Postpartum:
Measure BP at least 4 times a day, closely monitor for 48 hours, must measure BP up to 3rd to 5th day. Continue antihypertensive treatment if BP > 150/100 mmHg. Use labetalol, nifedipine, enalapril, captopril, atenolol, metoprolol. Do not use ARB, amlodipine, ACEIs other than enalapril and captopril. Reduce antihypertensives if BP < 130/80 mmHg for 48 hours. Avoid diuretics in breastfeeding mothers. Consider postpartum thromboprophylaxis (enoxaparin - low molecular weight heparin - 40-60 mg/day). Measure platelet count, transaminases and serum creatinine 48-72 hours after birth. Continue prophylactic anticonvulsant till 24 hours postpartum.

Patient education:
- Emphasize need for follow-up visit 1 week after discharge and again after six weeks; and finally 12 weeks postpartum for reclassification, necessary investigations and antihypertensive treatment if required.
- Advise on need for preconception counselling in cases of chronic hypertension.
- Advise on need of close antepartum monitoring in next pregnancy.
- Advise regarding Colour Doppler study of uterine arteries and prophylactic low-dose aspirin (75 mg) started early in pregnancy (14-16 weeks).
- Report immediately if alarming symptoms such as headache, nausea, oliguria and visual disturbances develop.

ECLAMPSIA:
Eclampsia is a life-threatening complication of severe preeclampsia. Eclampsia can occur during pregnancy (20th week of gestation), labor or in the postpartum period. Accordingly it is named as Antepartum, Intrapartum & Postpartum eclampsia.

Diagnostic Criteria:
- Occurrence of generalized convulsions associated with signs of pre-eclampsia during pregnancy, labor or within 7 days of delivery and not caused by epilepsy or other convulsive disorders. Key warning signs of developing eclampsia in a woman diagnosed with preeclampsia are severe headaches, blurred or double vision.
- Patient may develop aspiration pneumonia, acute left ventricular failure, cerebral haemorrhage, renal cortical necrosis, DIC, fetal distress, abruptio placentae, fetal death and even maternal death can occur.

Treatment (To be managed at a tertiary care level):
Eclampsia Patients should be managed at tertiary care level.

Principles of management are control and prevention of recurrence of convulsion and control of hypertension. Treat any complication that arises and deliver safely as soon as possible. Continue anticonvulsant therapy 24 hours after delivery or last fit whichever is latest.

Non-pharmacological:
- Place the patient in left lateral position in a separate quiet room with padded rails on sides of bed.
- Secure and maintain airway. Use mouth gag or airway to prevent tongue biting/tongue falling back. Intubate, if patient is deeply unconscious, poor arterial blood gases, extensive laryngeal oedema and extreme restlessness.
Suction to remove oropharyngeal secretions.

Oxygen by facemask at 6-8 l/min.

Set up IV access and start IV fluids - Ringer’s lactate at 80 ml/h.

Catheterize with indwelling catheter.

Monitor heart rate and respiration, BP, urine output hourly.

Lab. investigations: Haemogram with platelet count, liver and kidney function tests, serum Uric acid, urinary proteins, coagulation profile, serum electrolytes.

Fundus examination.

Fetal assessment: DFMC, daily NST, USG for FBPP.

Pharmacological:

1. Inj. Magnesium sulphate loading dose of 14 g of which 4 g as 20% W/V solution given slowly IV over 5-10 minutes and 5 g as 50% W/V solution given deep 1M upper outer quadrant in each buttock (total 10 g 1M). If fits are not controlled in 30 minutes, give another 2 g Magnesium sulphate as 20% solution slow IV over 5 minutes.

   Maintenance dose of 5 g magnesium sulphate as 50% solution deep 1M every 4 hours in alternate buttock or continuous IV regimen 4 g loading dose over 20 minutes followed by 1 g/h slow continuous IV infusion. Monitor for respiratory rate to be > 16/min, patellar reflex to be present and urine output >25 ml/h before giving magnesium sulphate. Continue till 24 hours after last fit/delivery whichever is earlier.

   (Caution: Side effects are respiratory depression and neuromuscular depression in mothers. Neonatal respiratory and neuromuscular depression.)

   If patellar reflex absent or urine output <25 ml/h, withhold magnesium sulphate and monitor hourly and restart maintenance dose if criteria fulfilled. If respiratory depression (RR <16/min), withhold magnesium sulphate and give calcium gluconate 1 g (10 ml) IV as 10% sol in 10 minutes. If respiratory arrest occurs, immediate endotracheal intubation and ventilation is to be done.

   If Magnesium sulphate is not available, Inj. Phenytoin loading dose of 15-25 mg/kg slow IV not exceeding 25 mg/min diluted in normal saline for first 750 mg and then 12.5 mg/min followed by 100 mg IV 8 hourly.

   ECG tracing to be taken every minute for 10 minutes during infusion of first 750 mg.

2. Fluid management should be closely monitored to prevent complications such as pulmonary oedema, left ventricular failure and adult respiratory distress syndrome.

3. Antihypertensives: As described in pre-eclampsia. Aim is to gradually lower the BP to 140-150/90-100 mm Hg.

Definitive management is termination of pregnancy irrespective of the fetal maturity. Termination is by labor induction and vaginal delivery or caesarean section.

Indications of caesarean section are:

✓ All deeply unconscious patients unless delivery is imminent,
✓ uncooperative patient due to restlessness,
✓ fetal distress,
✓ failed induction,
If vaginal delivery is unlikely to occur within 6-8 hours from the onset of 1st eclamptic seizure or eclamptic seizures are not controlled in 6-8 hours

Other obstetric indications.

Care after delivery:
- Patients of eclampsia and severe pre-eclampsia need intensive monitoring for at least initial 72 hours.
- Continue anticonvulsant till 24 hours after delivery or convulsion whichever occurs later.
- Gradually decrease the dose of antihypertensives.
- Patient is discharged after 10-14 days of delivery or earlier, if BP controlled without antihypertensives.
- Follow up after 6 weeks for re-evaluation.

Patient education:
- Delivery is the only definitive treatment. Underlying disease remains till delivery and complications can arise despite control of BP on treatment.
- Symptoms of severe pre-eclampsia/imminent eclampsia like headache, vomiting, epigastric pain, decreased urine output, blurring of vision should be immediately reported.
- Need for prolonged hospitalisation.
- Early booking in next pregnancy as there is 25-30% risk of recurrence.
- Prophylactic measures like low-dose aspirin can be started in early pregnancy.
- Need for re-evaluation at 6 weeks postpartum for reclassification and investigations of hypertension and need for long-term antihypertensive.
- High risk of development of chronic hypertension in later life.

Gestational hypertension:
- Expectant management - in cases of Gestational hypertension without fetal and maternal compromise, with gestational age <37 weeks and no proteinuria.

Non-pharmacological
- Extra rest preferably in left lateral position and regular diet adequate in proteins and calories with omission of extra table salt.

Follow up every week. At each visit:
- Check BP, urine for protein, weight and generalised body oedema.
- Exclude and counsel about symptoms of severe pre-eclampsia and danger signals
- Monitor fetal growth, ask the woman about fetal movements, check FHR.
- Ensure 100 g protein in diet/day.

If all observations remain stable, continue follow-up. Book the woman for delivery at the PHC/Hospital at term (do not allow to cross the expected date of delivery).

Refer to CHC if BP > 150/100 mmHg, GA <30 weeks, fetal affection (IUGR, oligohydramnios).

In case of mild gestational hypertension without proteinuria if after hospitalisation BP is controlled with rest without any antihypertensive drug, patient can be discharged after initial
evaluation if no maternal/fetal compromise is detected. It can be practiced only in reliable patients who will follow instructions for monitoring as above and also will report in case of ominous symptoms immediately (ominous symptoms are persistent severe headache, visual disturbances such as dimness of vision, double vision or blindness, epigastric pain, nausea, vomiting and oliguria)

In Gestational hypertension if BP remains persistently more than 150/100 mmHg, anti hypertensive therapy should be started as outline in management of mild pre eclampsia.

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**PRETERM PREMATURE RUPTURE OF MEMBRANES (PPROM)**

Now the preferred term is prelabor rupture of membranes.

**Definition:**
Spontaneous rupture of the membranes any time beyond 28th week of pregnancy but before the onset of labor. If after 37 weeks it is called term PROM and if before 37 weeks it is called preterm PROM.

**Management:**

- Patient should be put to bed and sterile vulval pad applied.
- Digital vaginal examination should be avoided due to the risk of introducing infection and stimulating release of prostaglandins.
- Speculum examination taking aseptic precautions should be done to established diagnosis by inspecting liquor escaping out through the cervix.
- If diagnosis of PROM cannot be confirmed visually, then pH testing by litmus paper or Nitrazine test, ferning methodcan be used.
- Evaluation for clinical evidence of chorio amnionitis such as maternal tachycardia , fever, uterine tenderness, offensive vaginal discharge, fetal tachycardia.
- Antibiotic prophylaxis - Inj. Ampicillin 2 g IV 6 hourly plus Erythromycin 250 mg IV 8 hourly for 48 hours, followed by Cap. Amoxicillin 250 mg orally Plus Tab. Erythromycin 333 mg PO 8 hourly for 5 days. If allergic to penicillin, Inj. Cefazolin 1g IV Plus Erythromycin 250 mg IV 8 hourly for 48 h followed by Cap. Cephalexin 500 mg Plus Erythromycin 333 mg PO 8 hourly for 5 days Or Inj. Vancomycin 1 g IV 12 hourly Plus Erythromycin 250 mg IV 8 hourly for 48 hrs followed by Tab. Clindamycin 300 mg PO 8 hourly

  Plus Erythromycin 333 mg PO 8 hourly for 5 days. (Note: Avoid prenatal Co-amoxiclav as it increases the risk of neonatal necrotising enterocolitis.)

- Cortcosteroids for fetal lung maturity. Inj. Betamethasone 12 mg 1M, 2 doses 24 hours apart.
  Or
- Inj. Dexamethasone 6 mg 1M four doses 12 hours apart or 12 mg 1M two doses 12 hours apart. (Caution: Contraindicated if clinical or laboratory evidence of chorioamnionitis is present.)
Investigations:
- CBC, CRP,
- Urine for routine analysis and culture
- High vaginal swab for culture.
- USG should be done in all cases to see for liquor volume, gestational age, fetal weight, to rule out major congenital malformation and cervical length.
- CTG

Delivery or conservative management should be planned according to the gestational age, fetal maturity and clinical & biochemical evidence of infection.

**Delivery at 34 weeks if possible**
- Tocolysis, amniinfusion, fibrin glue, progesterone are not found to be of much use in PROM so are not recommended.
- Monitor regularly for signs of intrauterine infection, i.e. chorioamnionitis (maternal pyrexia, tachycardia, leucocytosis, uterine tenderness, offensive vaginal discharge and fetal tachycardia).
- On rare occasion with bed rest, the leaking seals spontaneously & pregnancy continues. Hospitalize for at least 48 hours before a decision is made to allow her to go home. This method of management should be individualized and restricted to certain women. Women should be instructed to take regular temperature recordings at home every 4-8 hours.

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PRETERM LABOR

Introduction:
Onset of labor pain in pregnant women after 28 weeks and before 37 completed weeks of gestation associated with progressive dilatation and effacement of the cervix is known as preterm labor.

Women of low socioeconomic status, heavy manual labor, extremes of age (<20 years and >35 years), previous history of abortion or preterm delivery, cervical or vaginal infection, multifetal gestation or over distended uterus, cervical incompetence or other uterine malformations, cervical length on TVS< 25 mm, anaemia, heart disease, pre-eclampsia, IUGR, fetal congenital malformations and antepartum haemorrhage in present pregnancy are at increased risk.

Diagnostic Criteria:
Regular uterine contraction of at least one in every 10 minutes with duration of 30 seconds or more accompanied by cervical dilatation (>2 cm) and effacement (>80%), with or without leaking or bleeding per vaginum.

Antenatal Preventive Measures:
- Women with high risk factors should be monitored regularly with serial ultrasound examination for cervical length, funnelling of internal os, bulging of membrane in endocervical canal.
- Infections, if present should be treated.
- Coital abstinence should be advised.
- Avoid smoking & strenuous exercise.
- Educate women with high risk factors about warning signs for preterm labor – {menstruation-like cramps, low dull backache, pressure (feels like baby is pushing down), increase in vaginal discharge, leaking, uterine contractions < 10 min apart even if painless. } and to report to hospital immediately if any such symptom occur.

Management:
According to the clinical type:-
1. Preterm Contraction.
2. Established preterm labor.

1. Preterm Contraction:
- Presence of uterine contraction with no cervical changes.
- Admit the women for observation
- Offer analgesia.
- Reassess after 2 hours.
- If no/irregular infrequent contractions, no cervical change discharge and follow-up as outpatient after 7 days.

2. Established preterm labor:
- Hospitalisation with complete bed rest and hydration preferably in a centre with neonatal intensive care unit.
- Assess cervical dilatation by sterile digital vaginal examination unless contraindicated by ruptured membranes, suspected placenta praevia.
- Investigations- Haemogram, urine and endocervical swab for culture and sensitivity, C-reactive protein, USG for fetal well-being.
Drug Treatment:

1. **Tocolytics:**
   - Immediate tocolysis in pregnancies <34 weeks; if membranes are intact and labor is not advanced (cervical dilatation <4 cm); those needing transfer to a hospital for neonatal intensive care, those who have not yet completed a full course of corticosteroids; there is no indication for immediate delivery and no contraindication for tocolysis.

   A. **Cap. Nifedipine** 20 mg orally loading dose, repeat after 20 minutes for 3 doses, followed by 10-20 mg every 4-6 hours for 48 hours. (Caution: Do not administer along with magnesium sulphate; contraindicated in maternal hypotension [<90/50 mmHg], cardiac disease. Use with caution in renal disease; maternal side effects: flushing, headache, nausea, dizziness, hypotension.)

   B. If nifedipine is contraindicated, **Inj. Atosiban** 6.75 mg IV over 1 minute, followed by infusion containing 750 mcg/ml in 0.9% Normal saline at the rate of 24 ml/h for 3 hours, then 8 ml/h for a maximum of 48 hours.

   C. When gestational age <32 weeks, imminent preterm birth (cervical dilatation ≤4 cm) or planned preterm birth for fetal or maternal indications, **Inj. Magnesium sulphate** 4 g loading dose IV over 30 minutes followed by 1 g/h till delivery or max up to 24 hours, to buy time for corticosteroids to act.

   (Caution: Do not combine with nifedipine.)

   Monitoring during tocolysis:
   - Monitor Pulse, BP and cessation of the uterine contractions.
   - If pulse rate > 120/min and BP <90/50 mmHg, stop tocolysis.
   - Monitoring in magnesium sulphate therapy is as outlined in eclampsia.
   - Monitor for onset of chorioamnionitis (fever, maternal or fetal tachycardia, uterine tenderness, foul-smelling liquor, leucocytosis).

   *Tocolysis has no role in long-term therapy; it is needed for first 48 hours only to allow for action of steroids.*

2. **Antenatal Cortico steroid:**
   - In pregnancies at 28-34 weeks, steroids are given for fetal lung maturity.
   - Inj. Betamethasone 12 mg IM 2 doses 24 hours apart.
   - Or
   - Inj. Dexamethasone 6 mg IM four doses 12 hours apart or 12 mg IM two doses 12 hours apart.

   (Caution: Contraindicated if clinical or laboratory evidence of chorioamnionitis is present.)

3. **Antibiotics:**
   - Cap. Ampicillin 500 mg or Erythromycin 500 mg 4 times a day for 5-7 days, only if premature rupture of membranes (PROM).

4. **GBS Prophylaxis:**
   - In women in established pre-term labor if they have had group B streptococcal (GBS) found on HVS or MSU in that pregnancy or have had a previously affected baby.
   - Inj. Penicillin G, 5 Million units IV initial dose, then 2.5 million units IV every 4 hours till delivery Or Inj. Ampicillin 2 g IV initial dose, then 1 g IV every 8 hours till delivery.
If penicillin allergic — Inj. Cefazolin 2 g IV initial dose, then 1 g IV every 8 hours till delivery. If at high risk for anaphylaxis, Inj. Clindamycin 900 mg IV every 8 hours till delivery Or Inj. Erythromycin 500 mg IV every 6 hours until delivery Or Inj. Vancomycin 1 g IV every 12 hours till delivery.

5. **Progesterone Supplementation:**
   Progesterone supplementation is recommended to women with history of spontaneous preterm birth and in women with a shortened cervix ($\leq 1.5$ cm) on transvaginal sonography in the mid trimester. Micronized progesterone 200 mg vaginally or 17-OHPC 250 mg/weekly IM since early second trimester till 36 weeks. Neither progesterone nor 17-OHPC has been recommended as a preventive agent for asymptomatic women.

6. **Cervical cerclage:**
   Cervical cerclage is not routinely recommended; useful only in documented short, cervical insufficiency with history of 3 or more previous preterm births and/or second trimester loss.

**Indications for immediate delivery:**
- Advanced labor (cervical dilatation $> 4$ cm), major fetal malformation, ineffective tocolysis, chorioamnionitis, other maternal or fetal indications for delivery, fetal death, pregnancy $> 34$ weeks, fetal distress.
- Mode of delivery is decided as per obstetric indications.
- Careful fetal monitoring required throughout labor.
- **Inutero transfer of the baby to the center having good neonatal care facilities is ideal.**
- Ventouse should not be used at less than 36 weeks pregnancy.
- Liberal episiotomy and protection of preterm head by outlet forceps
- The neonatal team should be present at the time of delivery.
- Late cord clamping by 30-60 seconds to prevent intracranial hemorrhage
- If any sign of hypoxia, caesarean section is better but foetus should have a fairly good chance of survival depending on neonatal care facility.
  (If pain subsides, no evidence of infection the patient can be discharged after 1 week of tocolysis followed by regular antenatal surveillance with advice of -1. Restricted physical activity after discharge, 2. Sexual abstinence till at least 34 weeks of gestation.)

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**ANTEPARTUM HAEMORRHAGE**

Bleeding from or into the Genital Tract from 28 weeks of pregnancy upto birth of baby. 70% of such bleeding has a placenta aetiology. 25 to 30% patients are due to undetermined causes. It is usually of two types, both life-threatening to mother:

A. Placenta Praevia,
B. Abruptio Placentae.

**A. PLACENTA PRAEVIA:**
- Painless, Causeless, Recurrent, Revealed, bright-red bleeding, floating, presenting part, abnormal lie. Anaemia proportional to amount of visible blood loss.
Laboratory Investigations:


Ultrasonography: Placenta in Lower Uterine Segment. (To ascertain type of praevia)

Management: Hospitalization.

Assessment of Mother (Gen. & Adb. Exam.), Blood loss assessment, Fetal well-being. Keep adequate quantity of blood (Packed cell)

Resuscitation

(Establish I.V Line, Keep warm, Oxygen 8L/m, Foley’s Catheter)
Patient Education:
Incidence of A.P.H high with increasing age and parity.
Incidence is more in cases of anaemia so anaemia has to be corrected in antenatal period..

Referral Criteria:
All patients with A.P.H. should be treated in a well-equipped hospital having facility for Emergency C.S, Neonatal Care Unit, Feto-maternal assessment, Blood Transfusion, therefore have to be referred if such facilities are not available.

A. ABRUPTIO PLACENTAE:
Diagnostic Features:
- Painful, concealed/cevealed, dark-red bleeding.
- Often associated with pregnancy induced hypertension or trauma.
- Anaemia out of proportion of blood loss in concealed variety.
- Tense, tender, rigid uterus with fundal height more than period of gestation (concealed)
- F.H.S may be absent in concealed variety (80%).

Laboratory Investigation:
Blood Tests:  
- Hb%, Blood Grouping, Rh Typing,
- Periodic Coagulation Profile- B.T., C.T., A.P.T.T.

Ultrasoundography:  
- Placenta in Upper Uterine Segment. Look for retroplacental clot and measure the volume of clot..

Management:
- Hospitalization
- Gen. & Abdominal Examination of Mother
- Assessment of Fetal Status
- Assessment of Blood loss.

Referral Criteria:
All patients with A.P.H. should be treated in a well equipped hospital having facility for Emergency caesarian section. Neonatal Care Unit, Feto-maternal assessment, or else referred to a Hospital having such facility.

Patient Education:
Smoking, Drug abuse, Hypertension and high maternal age and parity predispose to it. Immediate Reporting to Local Hospital as mortality and morbidity is high.
POSTPARTUM HAEMORRHAGE

Excessive bleeding from the Genital Tract following birth of baby up to end of puerperium which adversely affects the general health of the patient (evidenced by increased pulse rate or fall of B.P.) Depending upon the amount of blood loss, PPH can be Minor < 1 L, Major 1-2 L, and Severe > 2 L.

Primary P.P.H. - Within 24 hrs.
   i) Third Stage haemoarrhage :- Before expulsion of placenta
   ii) True P.P.H. :- After expulsion of placenta up to 24 Hrs.

Secondary P.P.H. – Beyond 24 hrs up to end of puerperium.

Diagnostic Criteria:
- Excessive bleeding per vagina after birth of Baby
- Rapid Pulse rate, Low B.P
- Dryness of mouth, reeling of Head, mental Confusion.

Laboratory Tests:

Non Drug Treatment:
- If Uterus is Flabby and Atonic – Massage the Uterus till it become hard & continue till
  ✓ Adequate response.
  ✓ Controlled cord traction.
  ✓ Empty bladder / Catheterization.

MANAGEMENT OF TRUE PPH:
1. Call for help.
2. Established I.V line.
3. Arrange for blood.
4. Infuse crystalloid (2 Liters)
5. Continue uterine message.

1. Uterine Message
2. Explore & Surgical Management
3. 20 Units of Oxytocin in 500 ml R.S. / R.L
4. Increased up to 40 units per 500 ml NS / R.L @ max. up to 125 ml/minute till response.
5. Inf. Methergin 6.2 Mg I.V. It may be repeated I.M. every 1 to 4 hly max. up to 5 doses
   (Contraindicated in hypertensive, Heart patients).
**PUERPERAL PYREXIA**

Rise of temperature reaching 100.4° (38°C) or more on two separate occasions 24 hours apart within the 1st 10 days following delivery excluding the 1st 24 hrs after delivery is called puerperal pyrexia.

**Causes:**
- Urinary tract infection, Mastitis, Infection of caesarean Section wound, Pulmonary Infection, Septic Thrombophlebitis, Pelvic Organ Infection.

**Risk Factors:**
- Anaemia, malnutrition, PROM, Repeated vaginal examination, Traumatic operative delivery, Haemorrhage, Dehydration.

**Clinical Features:**
- Signs of Infection as per the site.

**Investigation:**
- Complete blood count, Blood for Malaria Parasite, Culture high vaginal Swab, urine & if required blood. Ultrasonography Of abdomen & pelvis.

**Treatment:**
- Supportive measures with correction of dehydration.
- Blood transfusion for correction of Anaemia.
- Inj. Gentamycin & Inj. Ampicillin if culture report is not available.
- Removal of Septic foci.
- Treatment of septic shock if appears.
CONTRACEPTION

A method or a system which allows intercourse and yet prevents conception is called a contraceptive method.

**Types of contraception:-**

- **Temporary** - the effect lasts only till the couple uses the method but the fertility returns after the use is discontinued.
- **Permanent contraceptive methods** - surgical and are aimed to achieve sterility after a surgical procedure.

A couple in the reproductive age group should be provided with all information about all the available methods of contraception and should be counseled and helped to choose a method most suitable for that couple.

Various contraceptive methods available

**1) Temporary methods**

a) **Hormonal**
   - Combined oral contraceptive pills
   - Injectable hormonal contraceptives (DMPA and NET-EN)
   - Progesterone only pill (desogestrel) 75 µgm once daily.

b) **Non-hormonal contraceptive pills**
   - Centchroman

c) **Intrauterine contraceptive device**
   - Multiload 250 and Multiload 375
   - CuT 380A
   - LNG IUD (Mirena)

d) **Barrier methods**
   - Male condoms
   - Vaginal diaphragms with spermicidal jelly
   - Contraceptive sponge

**2) Permanent methods**

a) **Female sterilization**
   - Postpartum sterilization
   - Interval ligation (Laparoscopic or minilap ligation)
   - Ligation concurrent with MTP

b) **Male Sterilization**
   - Vasectomy (traditional or non-scalpel)

**I. Temporary contraceptive methods**

A. (i) **Combined oral contraceptive pills**

Any of the low-dose combined oral contraceptive pill containing 30 mcg Ethinyl oestradiol and a Progestin (0.3 mg Norgestrel or 0.15 mg Levonorgestrel or 0.15 mg Desogestrel) can be prescribed. One tablet is to be taken daily with meals at a consistent time. It should be started on day 1 of the menstrual cycle. Pills should be taken for 3 weeks followed by 1 week of pill-free interval (irrespective of the day of onset of menses) during which placebo tablets are to be taken if pack contains 28 tablets.
Contraindications
Combined oral contraceptive pills are contraindicated in cases with current or history of:
✓ Thromboembolic disease, cerebrovascular disease, coronary artery disease.
✓ Complicated valvular heart disease.
✓ Active liver disease.
✓ Current or past breast cancer.
✓ Undiagnosed vaginal bleeding
✓ Pregnancy
✓ Heavy smokers over 35 years of age.
✓ Migraine with neurological symptoms and aura.
✓ Diabetes >20 years or with vascular disease.
✓ Current gallbladder disease.
✓ Uncontrolled hypertension (BP>160/100).

Follow-up:
First follow-up should be within 3 months and then annually. Follow-up involves history, blood pressure, urinalysis, breast examination, liver palpation and pelvic examination, pap smear.

Patient Education:
Common side effects are nausea, vomiting, GI upset, breast changes, weight gain, acne, breakthrough bleeding, amenorrhea, rash, vaginal candidiasis. Side effects decrease usually after 2-3 months of use.
✓ It is one of the most effective contraceptive methods with failure rate about 0.1-0.8%.
✓ It has non-contraceptive health benefits like regular periods with less pain and bleeding, improves premenstrual symptoms, and decreases functional ovarian cysts, pelvic inflammatory disease, ovarian, endometrial and colorectal cancer, endometriosis, protects against osteoporosis.
✓ Forgotten pill – if one active pill is forgotten, take as soon as remembered and next day take next pill and continue the schedule.
✓ If 2 active pills are forgotten take 2 pills and next day again take 2 pills, and then continue as per schedule. Condoms should be used or sex avoided until seven consecutive active pills have been taken.
✓ If 3 active pills are forgotten, this cycle is not protected, use back up method till next cycle and then restart with new pack.
✓ Medicines like rifampicin, barbiturates, phenytoin, carbamazepine, primidone, griseofulvin interfere with the effect of oral pills. So use additional method during their intake.
✓ Pills should be discontinued at least 4 weeks prior to any planned major surgery.

A. (ii) Injectable Hormonal Contraceptives:
It can be given in women where oestrogens are contraindicated like sickle cell disease, seizure disorders, age > 35 years who smoke; can be given in breastfeeding females after first 6 weeks. In non-breastfeeding females, injections can be safely given immediately postpartum. Depot Medroxyprogesterone Acetate – 150 mg injection to be given deep IM every three months. Next injection may be delayed up to 2 weeks. Norethisterone enanthate – 200 mg injection to be given deep IM every 2 months. Next injection may be delayed up to 1 week.

Absolute Contraindications:
✓ Unexplained genital bleeding
✓ Severe coagulation disorder
Previous sex steroid-induced liver adenoma, active liver disease.
During initial 6 weeks of delivery & Breastfeeding
Current or history of cerebrovascular disease, coronary artery disease, thromboembolic disease.
Current or past breast cancer
Diabetes > 20 yrs or with vascular disease.
Uncontrolled hypertension (BP>180/110 mmHg).

**Patient Education:**
- Common side effects are irregular break through bleeding, amenorrhoea, breast tenderness, weight gain, depression, headache, dizziness and abdominal pain.
- Fertility return is slightly delayed after discontinuation of use.
- Drug interactions are same as with oral hormonal pills.

**B. Non-hormonal oral contraceptive pills**

**Centchroman:**
30 mg tablet started on first day of periods. Take twice weekly for 3 months and then continue once weekly.

**Contraindications**
- Polycystic ovarian disease, cervical intraepithelial neoplasia, severe allergy, recent history of liver disease and first 6 months of lactation.

**Patient education:**
- If one tablet is missed take as soon as possible and resume scheduled intake. Add back up method till next period. If tablet missed for >7 days, start fresh regimen.
- It is a non-steroidal contraceptive pill. It has no hormonal effects.
- Delayed periods can occur in 6% cases. If delay is >15 days, perform urine pregnancy test.
- Failure rate is 1-2%.

**Intrauterine contraceptive devices (IUCD):**
Any of the following devices can be inserted inside uterus by trained health personnel at a healthcare facility under aseptic conditions after obtaining informed consent. IUCD should be inserted during or shortly after menstruation during the follicular phase of menstruation. The CuT 380A can be inserted immediately after counseling and obtaining informed consent (postpartum within 10 minutes after expulsion of the placenta following vaginal delivery on the same delivery table; intra-cesarean insertion after removal of placenta and before closure of the uterine incision) or within 48 hours after delivery, or post MTP (MVA, S&E), if there is no infection, bleeding or any other contraindication. The IUCD should not be inserted from 48 hours to 6 weeks following delivery because there is an increased risk of infection and expulsion. It can be inserted within 5 days of unprotected coitus. These devices need to be changed after the duration of their lifespan (Table 15.5)

<table>
<thead>
<tr>
<th></th>
<th>Lifespan</th>
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</thead>
<tbody>
<tr>
<td>Multiload 250</td>
<td>3 yrs</td>
</tr>
<tr>
<td>Multiload CuT 375</td>
<td>5 yrs</td>
</tr>
</tbody>
</table>

- Advise client to report any increase in vaginal bleeding or uterine cramping.
✓ Manage vaginal haemorrhage related to uterine atony as per the standard procedure (Immediate PPIUCD does not increase the risk of uterine atony).
✓ If severe uterine cramping occurs and persists after PPIUCD insertion, conduct a speculum or bimanual examination to check for partial or complete expulsion.

Follow-Up:
First follow-up should be after next menstrual period to check for complications and to rule out expulsion. After that, follow-up as and when required.

Contraindications:
Postpartum 48 hours to 6 weeks, septic abortion, distorted uterine cavity, uterine fibroids, current or past history (within 3 months) of PID or STD, increased risk of STD, AIDS, pelvic tuberculosis, unexplained vaginal bleeding, trophoblastic disease, genital tract malignancy.

Patient Education:
✓ Give a card mentioning type of IUCD inserted, date, along with month and year when IUCD needs to be replaced/removed and contact phone number, if she has any problems about IUCD.
✓ Common side effects are increased menstrual bleeding and dysmenorrhoea. These decrease after initial 2-3 months.
✓ Spontaneous expulsion can happen in some cases; can reinsert new IUCD.
✓ At 6 weeks postpartum, the IUCD strings can be felt by some women. If bothersome - the string can be cut short.
✓ Return for removal of the IUCD at any time she wants pregnancy and she will have almost immediate return of fertility.
✓ Failure rates are 0.5-3%. Ectopic pregnancy can still occur. Report if missed period or persistent irregular or heavy bleeding accompanied by severe pain abdomen.

D. Barrier methods:
These are inexpensive, available over-the-counter and coitus dependant methods. Male condoms, vaginal diaphragm, spermicidal jellies, vaginal sponge can be used.

Male condoms:
Any of the available condoms can be used. For each act of coitus, a new condom is to be used. If during intercourse, condom breaks or if there is spillage or leakage, woman should contact a clinician within 72 hours and emergency contraception should be provided to her.

Contraindication. Only contraindication is in cases with severe allergy to latex rubber.

Vaginal Diaphragm:
Available in different sizes. Proper size should be checked by a clinician by pelvic examination. It should be inserted only two hours before intercourse. About a tablespoonful of spermicidal cream or jelly should be placed in the dome of diaphragm prior to insertion. Should be left in place for approximately 6 hours (but not >24 hours) after coitus. Additional spermicide should be placed in vagina before each additional episode of sexual intercourse, without removing the diaphragm. After removal, wash with soap and water, rinse and dry.

Follow-up:
Annually to assess proper fitting of diaphragm.

Contraceptive sponge:
Contains spermicidal agent Nonoxynol 9. It should be removed at least 6 hours after sexual intercourse.
Maximal wear time is 30 hours.
Patient Education:
✓ Barrier methods provide protection against STDs and PID. Only condom has been proven to prevent HIV infection, other advantages are prevention of diseases like pelvic inflammatory disease and carcinoma cervix.
✓ It can be used soon after delivery.
✓ No hormonal side effects.
✓ Failure rates are very high. Typical failure rates are with condoms 14%, diaphragm with spermicidal jelly 20%, highest with sponge 28%.
✓ Side effects like vaginal dryness, itching, irritation, allergic reactions can occur

Permanent contraceptive methods:
Permanent contraception is provided by sterilization operation in male or female partners.

A. Female sterilization:
Sterilization can be done by laparoscopic ligation for interval ligation and by minilaparotomy for first trimester abortions or tubectomy.

Timing:
✓ Interval sterilization - within 7 days after menstrual period is over or after 6 weeks postpartum.
✓ Postpartum sterilization - after delivery till 7 days but preferably within 48 hours.
✓ MTP - concurrently.
✓ Spontaneous, abortion - concurrently but under antibiotic cover and in the absence of infection and anaemia.

Contraindications:
There are no absolute contraindications. Relative contraindications are psychiatric disorder, acute febrile illness, jaundice, Hb <8 g/dl, chronic systemic disease, malignancy, bleeding disorder, severe nutritional deficiency. Postpartum sterilisation is contraindicated in puerperal sepsis or fever, severe pre-eclampsia/eclampsia, premature rupture of membrane >24 hours, severe APH or PPH, genital tract trauma, Post-abortal in sepsis.

Follow-up:
First follow-up should be done 7 days after the surgery and after 1 month

A. Male sterilization:
There are no absolute contraindications. Relative contraindications include psychiatric and physical illness, local genital conditions, including large varicocele, hydrocele, inguinal hernia, filariasis, cryptorchidism, previous scrotal surgery, intrascrotal mass.

Follow-up:
First follow-up after 7 days of surgery for wound examination and stitch removal. Second follow-up after 3 months with semen analysis. Subsequent follow-ups required in cases of any complication or queries.

Patient education:
✓ It is a safe and simple procedure.
✓ It is a permanent method to prevent future pregnancies.
✓ It does not affect sexual pleasure, ability or performance.
✓ It has a small chance of failure even if performed under optimum circumstances.
✓ After vasectomy, it is necessary to use a back-up contraceptive method either for 20 ejaculations or for a period of 3 months.
Sterilisation does not provide protection against reproductive tract infections/STDs or HIV/AIDS.

Failure rates with female sterilisation are <0.5% and male sterilisation <0.1%.

**Emergency contraception:**

Method used to prevent pregnancy after a likely fertile unprotected act of sexual intercourse; can be used in cases of condom rupture, rape or other circumstances of unprotected sex.

1. First dose should be taken within 72 hours (the earlier the better) following unprotected sex and second dose 12 hours after the first dose. Any of the following can be used:
   - Levonorgestrel 0.75 mg 1 tablet 12 hourly for 2 doses or 1.5 mg single dose. Or
   - Combined oral contraceptive pills containing 50 mcg Ethinyl oestradiol with 0.3 mg Norgestrel 2 tablets 12 hourly for 2 doses.
   - Low dose combined oral contraceptive pills containing 30 mcg Ethinyl oestradiol with 0.3 mg Norgestrel 4 tablets 12 hourly for 2 doses.

IUCD insertion within 5 days of unprotected act provided there is no evidence of pelvic infection or rape.

**Patient education:**

- Side effects are nausea, vomiting, dizziness, fatigue, headache, lower abdominal pain, breast tenderness, vaginal bleeding.
- It must be used under medical supervision.
- It decreases risk of pregnancy by 70-90%. Earlier it is taken, better is the success rate. Emergency contraception does not protect against subsequent acts of coitus (except IUCD).
- If vomiting occurs within 2 hours of the dose, it must be repeated.
- There are no contraindications for emergency contraception.
- After this, use a barrier method for each act of intercourse until next menstrual period.
- If period delayed by >5 days, rule out pregnancy.
- Don’t use as a regular contraceptive method.

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PELVIC INFLAMMATORY DISEASE (PID)

INTRODUCTION:

PID is a spectrum of infections and inflammation involving the female upper genital tract- uterus, tubes, ovaries and pelvic peritoneum. Depending on the presentation, the disease may be acute or chronic. Mode of transmission: Most cases of acute PID are sexually transmitted diseases. Iatrogenic following procedures like dilatation and curettage, suction and evacuation, IUCD insertion and HSG.

DIAGNOSTIC CRITERIA:

- Minimum criteria include: Lower abdominal pain, cervical motion tenderness and adnexal tenderness.
- Tubo-ovarian mass, fever, Abnormalcervical or vaginal discharge and leucocytosis may be present.
- In severe cases, patient may be toxic with high-grade fever, vomiting, dehydration and abdominal distension.
- Long-term sequelae can be infertility, ectopic pregnancy, chronic pelvic pain and even mortality can occur in case of ruptured tubo-ovarian abscess.

RELEVANT INVESTIGATIONS:

Complete blood count, ESR, LFT, RFT, serum electrolytes, blood culture, endocervical swab culture, ultrasonography for adnexal mass.

Treatment (Acute PID): The patient can be treated as an outpatient or inpatient basis:

I. Outpatient treatment:

Criteria: Mild to moderately severe PID with slight pain and tenderness. Absence of high-grade fever, vomiting and abdominal distension.

Management of syndrome with the kits.

<table>
<thead>
<tr>
<th>Kit No</th>
<th>Syndrome</th>
<th>Colour</th>
<th>Contents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kit 1</td>
<td>Urethral discharge, anorectal discharge, cervical discharge</td>
<td>Grey</td>
<td>Tab Azithromycin 1g + Tab. Cefixime 400 mg</td>
</tr>
<tr>
<td>Kit 2</td>
<td>Vaginal discharge</td>
<td>Green</td>
<td>Tab. Secnidazole 2 g (1) + Tab. Fluconazole 150 mg (1)</td>
</tr>
<tr>
<td>Kit 3</td>
<td>Genital ulcer disease non-herpetic</td>
<td>White</td>
<td>Inj. Benzathine penicillin 2.4 MU (1) + Tab. Azithromycin 1 g (1) + Disposable syringe 10 ml with 21 gauge needle (1) + sterile water 10 ml (1)</td>
</tr>
<tr>
<td>Kit 4</td>
<td>Genital ulcer disease non-herpetic, for patients allergic to penicillin</td>
<td>Blue</td>
<td>Tab. Doxycycline 100 mg (30) + Tab. Azithromycin 1g (1)</td>
</tr>
<tr>
<td>Kit 5</td>
<td>Genital ulcer disease-herpetic</td>
<td>Red</td>
<td>Tab. Acyclovir 400 mg (21)</td>
</tr>
<tr>
<td>Kit 6</td>
<td>Lower abdominal pain</td>
<td>Yellow</td>
<td>Tab. Cefixime 400 mg (1) + Tab. Metronidazole 400 mg (28) + Cap. Doxycycline 100 mg (28)</td>
</tr>
<tr>
<td>Kit 7</td>
<td>Inguinal Bubo</td>
<td>Black</td>
<td>Tab. Doxycycline 100 mg (42) and Tab. Azithromycin 1 g (1)</td>
</tr>
</tbody>
</table>
Patient should be followed-up after 2-3 days of initiation of therapy for clinical response. If response is poor, patient is to be admitted for intravenous antibiotics.

II. Indoor treatment:
Criteria: Bed rest, IV fluids in cases of vomiting and dehydration and correction of electrolyte imbalance. Monitoring by clinical condition, vital monitoring, signs and symptoms of pelvic abscess and peritonitis.

Pharmacological:
Any of the following regimens may be instituted immediately following admission.

Regimen A
1. Inj. Cefoxitin 2 g IV every 6 hours for 2-4 days.
   Or
   Inj. Cefotetan 2 g IV every 12 hours.
2. Inj. Doxycycline 100 mg IV or orally every 12 hours.
3. Inj. Metronidazole 500 mg IV 8 hourly.

Regimen B
1. Inj. Clindamycin 900 mg IV every 8 hours.
2. Inj. Gentamicin 2 mg/kg IV followed by 1.5 mg/kg every 8 hours.
3. Inj. Metronidazole 500 mg IV 8 hourly.
4. In case of severe pain, Inj. Diclofenac sodium 75 mg deep IM 8 hourly.
   Or
   Inj. Paracetamol 500 mg IM SOS.

Duration for injectables should be atleast 48 hours after the patient demonstrates clinical improvement (becomes afebrile, decrease in lower abdomen and pelvic tenderness, sense of wellbeing). Then, Doxycycline 100 mg 2 times a day orally or Clindamycin 450 mg oral 4 times a day should be continued for a duration of 14 days.

Clinical improvement should be observed within 3 days of initiation of therapy.

If symptoms do not improve: diagnostic laparoscopy should be performed and differential diagnosis should be ruled out.

Following situations will require the necessary procedures:
✓ Pelvic abscess –colpotomy and drainage
✓ Retained products of conception in post-abortal sepsis: dilatation and evacuation.
✓ Pyoperitoneum, intestinal obstruction, ruptured tubo-ovarian abscess, enlarging pelvic mass despite medical therapy will necessitate laparotomy.

Treatment of male partner:
Asymptomatic male partner: Inj. Ceftriaxone 125 mg IM and oral Doxycycline 100 mg 2 times a day for 14 days or Azithromycin 1 g orally as a single dose.
Treatment (Chronic PID):

Treatment of chronic PID is surgical. Type of surgery is decided considering pathological lesion, patient’s age and desire for child bearing. Definitive surgery is total abdominal hysterectomy with bilateral salpingo-oophorectomy but in young females conservative surgery is preferred. Injection placentrex and pelvic diathermy may help. Chronic PID can also be caused by pelvic tuberculosis for which ATT should be instituted for 6 months.

Treatment (Pelvic tuberculosis):

Daily dose of the drugs is:

1. Tab. Isoniazid 10 mg/kg (maximum 300 mg).
2. Cap. Rifampicin 10-12 mg/kg (maximum 600 mg).
3. Tab. Pyrazinamide 15-30 mg/kg (maximum 2 g).
4. Cap. Ethambutol 15-25 mg/kg (maximum 1.5 g).

All these 4 drugs are given in the initial phase for 2 months followed by INH and rifampicin for 4 months. Indications of surgery are primary unresponsiveness, persistence or enlargement of adnexal mass after 6 months of treatment, persistence or recurrence of pelvic pain on treatment. Definitive surgery is total abdominal hysterectomy with bilateral salpingo-oophorectomy.

Patient education:

- Stress should be given to educate patients regarding behavioural and contraceptive methods to prevent STDs.
- Emphasis should be given regarding the importance to complete the full course of antibiotics, i.e. 14 days or ATT.
- Sexual abstinence should be advised till treatment is completed. Male partner of must be simultaneously treated to prevent reinfection.

Referral:

Referral should be made if: Uncertain diagnosis and surgical emergencies such as appendicitis and ectopic pregnancy cannot be ruled out, pregnancy, post-abortal or puerperal and if the patient is adolescent, severe PID, HIV positive, noncompliance or failure of outpatient treatment.
DYSMENORRHOEA

It is a condition of Painful Menstruation.
It can be – A. primary  B. Secondary.

A. Primary (Spasmodic) Dysmenorrhea:
No underlying organic pathology.

Diagnostic Criteria:
Suprapubic pain radiating to inner aspects of thigh, mostly in adolescents, which starts with menstruation and lasts for 1-2 days.

Non-Drug Treatment:
Improvement of General Health, Counselling.

Drug Treatment:
Tab. Ibuprofen 400mg + Dicyclomine 20mg. 1 tab. S.O.S/1 tab. 8 hrly 2 days during period. Must be taken after food to prevent g.i. upset.

Alternative Treatment:
If patient does not response to above treatment she may be treated with Oral Contraceptive Pills (Laevonorgestrel 0.15 mg + Ethinyl oestradiol 0.03 mg/Tab.) starting from 5th day of menstrual cycle for 21 days.

Patient Education:
Assurance to the patient that it is a normal phenomena.
Continue the day activities.
Exercises and yogasanas.

Referral Criteria:
Progressive Dysmenorrhea.
Co-menstrual Dysmenorrhea.

B. Secondary (Congestive) Dysmenorrhea
It is always associated with pelvic pathology such as Endometriosis.
Uterine mass, Tubo-ovarian mass or Presence of I.U.C.D.

Diagnostic Criteria:
Dull-aching pain in deep pelvis and sacral region, start 2-3 days prior to menstruation, not relieved with start of bleeding.
Pelvis examination may reveal tender fornices, uterine or Tubo-ovarian mass.

Laboratory Tests:
Routine Blood Count, Ultrasonography if available.

Non Drug Treatment:
Reassurance.
Removal of I.U.C.D.

Drug Treatment:
Tab. Ibuprofen 400mg + Dicyclomine 20 mg 1 tab. S.O.S /1 tab 8 hrly 2 days during period.
If associated with PID-a course of antibiotic may be tried.
Standard Treatment Guidelines of Odisha 2018

Cap. Doxycycline 100mg, 1 tab twice daily for 10-14 days.
Or Tab. Ofloxacin 200mg, 1 tab twice daily for 10-14 days.
And Tab. Metronidazole 500 mg, 1 tab twice daily for 10-14 days.
Associated Endometriosis may be treated with O.C Pills or Danazol.

Patients Education:
Doxycycline and Metronidazole may cause gastric upset.

Referral Criteria:
Patients not responding to above treatment.
Associated with Infertility, Tuberculosis, Fibroid uterus or other gross pelvic pathology.

***

ABNORMAL UTERINE BLEEDING

DEFINITION:
Abnormal uterine bleeding (AUB) is defined as any bleeding from the genital tract which is a deviation in the previously established menstrual pattern in terms of frequency, cyclicity or quantity and occurs in the absence of pregnancy.
Acute AUB may occur spontaneously or within the context of chronic AUB (abnormal uterine bleeding present for most of the previous 6 months).

Dysfunctional uterine bleeding (DUB) is defined as AUB without a demonstrable organic, systemic or iatrogenic cause. DUB is a diagnosis of exclusion after ruling out medical illness and pelvic pathology.

Diagnostic Features:
- Excessive amount of bleeding, long duration of flow, passage of clots (size and number), more consumption of sanitary pads.
- Patient looks pale and sick.

Laboratory Test:
Hb%, platelets, B.T, C.T., and Peripheral smear.
Serum TSH, T3, T4.
Ultrasonography of pelvis preferably TVS.
Patients above 35 years should have mandatory D&C and Histo-Pathological Study/Hysteroscopic guided biopsy.

Treatment:
Management protocol depends on the age of the patient.
Pregnancy and medical causes of AUB should be excluded.
Acute severe bleeding:
Outpatient management

1. Progesterone: Tab. Norethisterone acetate 5mg, 2 tabs 8hrly till bleeding stops then tapered. If not responding - Tab conjugated equine oestrogen 2.5 mg orally 6 hourly.
2. Tab. Tranexamic acid 500 mg 3 times a day for 5 days
3. Tab. Mefenamic acid 500 mg 3 times a day

Or
Combination of both Tranexamic acid and Mefenamic acid
If severe bleeding (> 1pad/h) continues, then Dilation and Curettage (D&C) is performed.

After severe bleeding stops:
Combined oral contraceptive pills from or 5th to 25th of cycle
Or
Cyclical progesterone treatment from 16th to 25th of cycle is given foe atleast 3 months.
Or
LNG IUS may be inserted.
Inpatient management:

Indications:
- Profuse active bleeding
- moderate-to-severe anaemia
- Haemodynamically unstable patient

1. Inj. Conjugated equine oestrogen 25 mg IV every 4 hours for 24 hours. (Contraindication: cardiovascular or thromboembolic risk factors.)
2. Inj. Tranexamic acid 1 g loading dose followed by 500 mg IV 8 hourly for 5 days.
3. Progesterone (Norethisterone acetate) 5 mg 2 tablets 3 times a day till bleeding stops followed by 5 mg 2 times a day for 3 days, then 5 mg once daily for 14 days.
4. PRBC to be transfused if haemoglobin <7.5 g/dl.
5. Once severe bleeding is controlled, OCPs/ progesterone/LNG IUS should be started.
6. D&C if no response to the above-mentioned medical management.

Suboptimal response: TVS/SIS should be performed. Management depends on the diagnosis-
- Polyps/submucous myomas-hysteroscopic resection
- Endometrial hyperplasia-following endometrial sampling, uterine artery embolisation, endometrial ablation or hysterectomy may be performed depending on the biopsy report.
- Adenomyosis- MRI is the gold standard. Progestin therapy, LNG IUS, leuprolide may be started but hysterectomy is the definitive treatment.
  - If TVS is normal or unresponsive to medical management, consider endometrial ablation or hysterectomy if child bearing complete.

Treatment of Chronic AUB :
1. Iron therapy: elemental iron maximum 60 mg 3 times a day depending on the degree of anaemia.
2. Levonorgestrel intrauterine system
   - Or
   - Oral contraceptive pills
   - Or
   - Progestin therapy.
3. Tab. Tranexamic acid 500 mg 3 times a day for 5 days
4. Tab. Mefenamic acid 500 mg 3 times a day
5. Histopathological diagnosis is mandatory before starting hormonal therapy in all cases except puberty menorrhagia.

Follow-up:
- Follow-up is done after 1,3,6 months of therapy. Treatment is stopped after 3-6 months.
- If symptoms recur, medical treatment is to be continued or surgery can be offered.

Role of surgery –

Endometrial curettage:
- Acute bleeding in haemodynamically unstable patient- therapeutic curettage to quickly control the bleeding.
- In women aged >35 years, premenstrual (Hysteroscopic guided dilatation and curettage for endometrial biopsy is a must to rule out endometrial pathology.
Surgical management:
If medical therapy is not effective, then endometrial ablation or hysterectomy is to be performed.

Patient Education:
- Emphasis must be given that medical management can cure most of the patients.
- Patients must be made aware of the:
  - Common side effects of high-dose oestrogens: nausea, vomiting, headache, depression and fluid retention.
  - Common side effects of progestogens: depression, fluid retention, fatigue, insomnia, dizziness, nausea and breast tenderness.
- Emphasize on maintaining menstrual calendar.
- Emphasize on correction of anaemia and a diet rich in iron and protein.
- Explain about maintaining an ideal BMI.
- Counseling on IUCD-related bleeding and its management.
- Provide information about medical management of DUB and uterus preserving surgeries like endometrial ablation.
- Explain risk of endometrial cancer.

REFERRAL:
- Hemodynamically unstable patient, acute heavy bleeding, nonresponsive to drugs.

******

POSTMENOPAUSAL BLEEDING

DEFINITION:
Postmenopausal bleeding (PMB) is bleeding per vaginum that occurs after menopause has been established for at least 1 year. Nearly one third of cases are due to malignancy hence each case must be evaluated.

COMMON CAUSES:
Genital malignancy, Senile endometritis, decubitus ulcer, urethral caruncle.

RELEVANT INVESTIGATIONS:
- Detailed history taking and clinical examination
- Paps smear with endocervical sampling should be taken if cervix appears healthy.
- If growth is found, punch biopsy should be taken.
- Pipelle endometrial sampling or hysteroscopy guided biopsy should be taken.
- Ultrasonography especially transvaginal for endometrial thickness.
- Saline infusion sonography
- CT scan and MRI in selected cases of postmenopausal bleeding.

Treatment:
Treatment depends on the cause.
Patient Education:
Patients should be educated regarding the significance of Pap smear and symptoms of cervical, endometrial and ovarian malignancy.

Indication for referral to a higher level of care:

Urgent referrals:
- Palpable pelvic mass or lesions suspicious of cancer of vulva, vagina, cervix or uterus on examination or on ultrasound.
- More than one or a single heavy episode of PMB in women aged >55 years (not on HRT).
- Prolonged or unexpected bleeding that persists for more than 4 weeks after stopping HRT.

Early referral: (within 4-6 weeks)
- Any other woman with PMB not on HRT who does not satisfy the criteria for ‘urgent referral’ of postmenopausal bleeding.
- Unexplained repeated postcoital bleeding.

Note: In women over 45 years with persistent abdominal distension or pain, ovarian cancer should be considered and, therefore, a pelvic examination should be performed.
- If excessive bleeding, give haemostatic drugs (oral or intravenous).
- Postmenopausal bleeding that is not due to cancer and cannot be controlled by any other treatment usually requires a hysterectomy.
14. DERMATOLOGY
SCABIES

A common skin infestation caused by arthropod mite (Sarcoptes scabiei) and transmitted by close personal contact after an incubation period of 3-4 weeks.

- Nocturnal itching, excoriated papules, papulovesicles, burrows and excoriation, lesions seen on interdigital clefts of hands, wrist, axillary folds, breasts, periumbilical region, medial side of thigh and genitals (inmates)
- Burrows are pathognomonic and a family/contact history of similar complaints invariably present
- Common complications are secondary pyoderma, eczematisation and glomerulonephritis (post-streptococcal).

Treatment

- Maintenance of adequate personal hygiene by daily bath
- Treatment of Secondary bacterial infection
- Permethrin cream 5% to be applied generously, after bath, at bedtime, covering surface of the body. In adults it should be applied below neck (except face) in from tip to toe. Minimum contact period 8-12 hours; single application required and is to be washed off next morning. Permethrin cream 5% as outlined above.

Or

- Gamma Benzene Hexachloride (GBHC) lotion 1%. Single overnight application below neck on entire body surface after a bath. Minimum contact period 8-12 hours, to be washed off next morning. Not to be used in the presence of extensive eczematisation.

Or

- Tab Ivermectin 200 mcg/kg as a single dose to be repeated after 2 weeks.

Supportive therapy

- Tab. Cetirizine 10 mg at night for 10-15 days.
- In children: 0.3 mg/kg/day single dose for 2 weeks
- Disinfestation of bedding and clothing by ordinary laundering and/or sun exposure is required.
- In lactating mothers – before feeding, areola should be washed thoroughly with soap and water. After the feed, permethrin cream should be reapplied on breasts and hands.

ONYCHOMYCOSIS

Invasion of the nail plate by Dermatophytes, Scytalidium or other non-dermatophyte moulds is called onychomycosis (Tinea unguium). Invasion of nail plate by Candida is regarded as candidal onychomycosis.

- The nail plate may appear to be discoloured (yellow, green or black), disfigured or, in extreme cases, might be totally destroyed. The nail folds may also show swelling and redness.
- Other causes of nail plate involvement should be ruled out, e.g. psoriasis, eczema, alopecia areata, lichen planus.
- It is prudent to determine the type of organism causing onychomycosis by doing potassium hydroxide mount or culture from nail clippings.
Candidiasis is an infection with protean clinical manifestations, caused by *Candida* species that are also part of normal skin/mucosal flora. The infections are usually confined to the skin, nails, mucous membrane and gastrointestinal tract but can be systemic and affect multiple internal organs.

- Various mechanical, nutritional, physiological, systemic and iatrogenic factors predispose to *Candida* infection. Extensive candidiasis resulting in internal organ involvement is an AIDS defining infection.

**Candidal paronychia**

- It is common in individuals whose hands are chronically involved in wet work, e.g. housewives, bakers, fishermen, *paan* vendors, etc.
- Redness, swelling and tenderness of the paronychial area with prominent retraction of cuticle towards the proximal nailbed. Occasionally, pus can be expressed from beneath this area. The nails might also be infected and discoloured.
- Treatment: Cap. Fluconazole 3-6 mg/kg (maximum 150 mg) orally once a week depending upon the area affected for 4-6 weeks.

**Oral candidiasis:** Topical 1% clotrimazole/2% miconazole nitrate or 1% ciclopirox cream gel or lotion twice daily for 14 days.

**Tinea corporis and cruris**

- Tinea corporis is a superficial dermatophyte infection characterised by either inflammatory or noninflammatory lesions on the glabrous skin (groin, palms and soles)
- Circular, sharply margined, itchy and scaly plaques with raised edges with papulovesicles at margins and central clearing.
- Laboratory diagnosis is made by KOH smear and culture.

**Treatment**

Topical treatment in localised disease (not for Tinea pedis)

- Ointment/cream/powder/spray Clotrimazole 1% twice daily for 4-6 weeks Or
- Miconazole 2% twice daily for 4-6 weeks Or
- Terbinafine 1% once daily for 2 weeks Or
- Butenafine 1% once daily for 2 weeks Or
- Ciclopirox olamine 1% twice daily for 4-6 weeks.

Systemic treatment (in extensive lesions) Tab. Griseofulvin 10 mg/kg for 4-6 weeks Or
• Tab. Fluconazole 3-6 mg/kg/week for 4-6 weeks Or
• Tab. Terbinafine 250 mg/day for 2 weeks Or
• Cap. Itraconazole 100 mg once daily for 4 weeks

ANTIFUNGAL POWDER CAN BE USED IN BETWEEN THE TOES AND GROIN

Tinea pedis/manuum and intertriginous tinea
• They are dermatophyte infection of the soles of the feet/hands and the interdigital spaces.
• SALIENT FEATURES include Peeling, maceration, fissuring affecting the lateral toe clefts.
• Hyperkeratotic plaque (affected areas are pink and covered with fine white scales).
• Vesiculobullous lesions, particularly at periphery.
• Treatment is same as that of tinea corporis.

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ECZEMAS AND DERMATITIS

Eczema is an inflammatory skin reaction characterised by itching, redness, scaling and clustered papulovesicles, induced by wide range of external or internal factors acting singly or in combination’ customarily the eczemas are divided into:

1. Endogenous (constitutional) – atopic dermatitis, seborrhoeic dermatitis, lichen simplex chronicus (LSC)
2. Exogenous (environmental) – contact allergic dermatitis, primary irritant dermatitis, photosensitive eczema, etc.

Salient features include:
Itching and vesicular eruptions on erythematous skin with erosion and exudation in acute cases or thickening, accentuated skin markings, fissuring with pigmentation (described as lichenification) in chronic cases.

1. Local treatment
• In acute exudative eczema:
  • Soak with dilute potassium permanganate solution (1:10,000) and 0.25% silver nitrate solution or 0.8% aluminium subacetate solution.
  • In long-standing situations:
    • Acute/subacute – appropriate topical steroid (Table 14.2) in lotion/gel or cream base for 2-4weeks.
    • Chronic long-standing and/or lichenified lesions – appropriate topical steroid in ointment/emollient base for 2-4weeks.

2. Systemic treatment
• Tab. Pheniramine maleate 25 mg 3 times a day till symptoms subside (about 7 days). In children: 0.5 mg/kg/day in 3 divided doses.
  • (Caution: Side effect – dry mouth)
  Or
• Tab. Cetirizine 10 mg at bedtime till symptoms subsides.
• In children: Syr. Promethazine 1 mg/kg/day 3 times a day till symptoms subside (about 7 days) or Syr. Cetirizine 0.3 mg/kg/day once daily till symptoms subside.
If there is no response with topical steroids and antihistamines or in case of extensive eczema (preferably under the supervision of a specialist) give,

- Tab. Prednisolone: up to 1 mg/kg (maximum 60 mg) as a single oral dose given in the morning after breakfast for 7-10 days. This should be tapered and withdrawn as early as possible after relief from symptoms and signs.

**Secondary bacterial infection**
- It should be treated in the acute stage with systemic antibiotics

**Patient education**
- Common skin irritants are overexposure to water or dry air, soaps and detergents, solvents, cleaning agents, chemicals, rubber gloves or ingredients in skin and personal care products.
- Following local side effects can occur due to misuse or overuse of corticosteroids: thinning of skin, striae distensae, increased facial redness and telangiectasia, purpura, tinea incognito, acneiform papules and increased hair growth.
- Systemic side effects can occur due to prolonged use of systemic corticosteroids or local applications on large surface area.

**ACNE VULGARIS**
- Chronic inflammatory condition of the pilosebaceous glands of the face, neck and upper back. Usually occurs in adolescents and young adults.
- Two types of lesions – non-inflammatory (comedones: blackheads or white heads) and inflammatory: pustules, nodules, cysts and abscesses.
- Acne can be secondary to mechanical friction/occlusion, detergents/chemicals, ultraviolet exposure, occupational, associated hirsutism/virilism exogenous etc

**Treatment**
- **Non-pharmacological**: Washing/cleaning of face to keep skin non-sticky, dry and dirt free; shampooing to keep scalp non-greasy.
- **Pharmacological**:
  - Non-inflammatory acne. Retinoic acid cream/gel (0.025%; 0.05%) usually applied once a day – at bedtime or alternate day. A therapeutic response appears characterised by redness and scaling within 3-6 weeks. Treatment is usually continued for at least 3 months.
  - Treatment is usually continued for at least 3 months.
  - Cream/gel Azelaic acid 20% applied once or twice a day after face-wash.
  - Inflammatory acne. Inflammatory acne treatment may need to be combined with treatment for non-inflammatory acne.
  - Mild cases. As above.
  - Clindamycin gel 1% to be applied twice a day (or more) for 4-6 weeks.
  - Or
  - Erythromycin gel/lotion 2%; 4% (safe in pregnancy) to be applied twice a day (or more) for 4-6 weeks. Begin with the lower strength.
  - Or
  - Benzoyl peroxide gel 2.5%, 5% (safe in pregnancy) to be applied to clean skin initially once daily on alternate days then twice a day (or more) for 4-6 weeks.
• Moderate-to-severe cases should be referred to a specialist preferably without treating with systemic antibiotics.
  1. Topical therapy as above (same drug should not be used topically as well as systematically as no extra-therapeutic benefit will result).
  2. Cap. Doxycycline 50-100 mg once daily for 4-12 weeks. The dosage can be reduced in accordance with the clinical response and discontinued.
• Tab. Minocycline 50-100 mg twice daily for 6-8 weeks. Or
• Tab. Azithromycin continuous or pulse therapy 250-500 mg daily for 6-8 weeks or 500 mg daily for 5-7 days every 4 weeks.

Treatment may need to be continued for up to 6 months. Severe and unresponsive cases should be referred to a tertiary care hospital.

Patient education

Redness and scaling with retinoic acid indicates a therapeutic response. Lasting benefit only after long duration use of comedolytic agents, usually for more than 6 months.

Avoid oil application and use of occlusive applications.

*****

URTICARIA

Urticaria is a heterogeneous group of disorders characterized by itchy wheals, which develop due to evanescent edema of dermis (and sometimes of subcutis).

Two main subtypes: Urticaria: Due to edema of dermis.

Angioedema: Due to edema of dermis and subcutis.

Etiology: Edema of dermis and subcutis due to mediators released from mast cells. Degranulation of mast cells mediated by IgE, complement, directly by drugs or idiopathic.

Triggers: Physical stimuli (scratching, cold, sunlight, pressure, etc.), dietary and inhaled allergens, and drugs. Often no cause.

Clinical features: Itchy evanescent wheals in urticaria. Less evanescent, not itchy in angioedema. Linear in dermographism; small wheals in cholinergic urticaria.

Complications: Laryngeal edema, anaphylaxis.


Immunosuppressives (methotrexate, azathioprine, and cyclosporine) in resistant disease.

CUTANEOUS ADVERSE DRUG REACTION

It is any noxious change on skin or mucous membrane which is due to drug intake.
Common ones include:

Fixed drug eruptions, Exanthematous eruptions and DRESS, Erythroderma, Steven Johnson syndrome and toxic epidermal necrolysis.

Appear as well-defined circular, deeply erythematous plaques, which sometimes develop central bullae. Subside leaving behind slate-gray hyperpigmentation, which persists in between acute episodes. Lesions recur at the same site, each time the drug is taken, usually 8–16 h after the intake of drug. Mucocutaneous junctions (lip and glans) most frequently involved presenting as large erosions.

Treatment

An early suspect of drug reaction and withdrawal of the drug is required. However, if it is an essential/life saving drug, one has to weigh the risks and benefits.

Short course of oral or injectable corticosteroids may be given and referred to higher centres.

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15. CARDIOLOGY
HYPERTENSION

Introduction

A condition of elevated BP above the normal value requiring antihypertensive therapy so as to reduce cardiovascular and renal complications.

Diagnostic criteria

If BP > 140/90 mmHg in normal adults or
>130/80 in diabetic or CKD.

Isolated systolic HTN (ISH) SBP > 160 & DBP < 90.

Usually asymptomatic and discovered on routine measurement of blood pressure. Secondary hypertension (HT) presents as a part of a symptom complex as in acromegaly, Cushing’s disease, renovascular or renal parenchymal disease, connective tissue disorders (SLE, scleroderma, etc.) or coarctation of aorta.

- Non-specific symptoms are fatigue, headache, epistaxis.
- Uncontrolled hypertension can lead to target organ damage (TOD) such as coronary artery disease (CAD), left ventricular hypertrophy (LVH), cerebrovascular accidents (CVA), transient ischaemic attacks (TIA), retinopathy, peripheral vascular diseases including dissecting aneurysm, renal disease.
- Associated risk factors are age >55 years in males and >65 years in females, smoking, diabetes mellitus, microalbuminuria or GFR <60 ml/min, hyperlipidaemia, family history, obesity, sedentary lifestyle and ethnic group.

Precautions to be taken while measuring BP

The auscultatory method of BP measurement with a properly calibrated and validated instrument should be used. BP should be measured using an appropriate size cuff in both upper limbs and at least one lower limb in both supine and erect posture. Patient should have been resting for at least 5 minutes and should not have consumed coffee or smoked during the last 30 minutes before measuring the blood pressure. At least 2 measurements should be made. Systolic BP is the point at which the first of 2 or more sounds are heard (phase 1) and diastolic BP is the point before the disappearance of sounds (phase 5).

Rational investigation

ECG, ECHO, Urine R/E & M/E, blood glucose, se Potasium, se cr., eGFR, & lipid profile.
Management of hypertension is shown in Fig. 3.1.

**If blood pressure fails to be maintained at goal, re-enter the algorithm where appropriate based on the current individual therapeutic plan.**

**Fig. 3.1.** Algorithm for treatment of hypertension. (Adapted from JNC 8 guidelines for treatment of hypertension.)

- **SBP** – Systolic blood pressure; **DBP** – diastolic blood pressure; **ACEI** – Angiotensin converting enzyme inhibitor; **ARB** – Angiotensin receptor blocker; **CCB** – Calcium channel blocker; **CKD** – Chronic kidney disease.

**Nondrug**
- Reduce dietary sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride).
- Adopt DASH eating plan—diet rich in fruits, vegetables, with low-fat dairy products with a reduced content of saturated and total fat.
- Lifestyle modification—exercise consisting of vigorous aerobic exercise like brisk walking, swimming or jogging at least 20-30 minutes on most days of the week with the heart rate reaching 65-70% of maximal heart rate.
- Weight control using a combination of dietary and exercise measures to maintain normal body weight (body mass index 18.5-24.9 kg/m²).
• Moderation of alcohol consumption – limit alcohol to no more than 2 drinks (1 oz or 30 ml ethanol; e.g. 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey) per day in most men and to no more than 1 drink per day in women and lighter weight persons.
• Cessation of smoking.
• Yoga.
• Control of other risk factors.

**Drug**

**Antihypertensive drug choices: Additional considerations**

(a) Diuretics – elderly, obese, congestive heart failure (CHF).
(b) Beta-blockers – young, coronary artery disease (CAD), vascular headache, associated atrial fibrillation (AF).
(c) Calcium channel blockers (CCB) – old age, CAD, atrial fibrillation (AF), paroxysmal supraventricular tachycardia (PSVT).
(d) Angiotensin converting enzyme inhibitors (ACEI) – young, left ventricular failure (LVF), diabetes.
(e) Angiotensin II receptor antagonists (ARB) – same as ACEI.
(f) Alpha-blockers – prostatism, diabetes, dyslipidaemia.
(g) Combined alpha and beta blockers – pregnancy.
(h) Old drugs – alpha-methyldopa (pregnancy), clonidine-refractory cases.

Thiazide diuretics should be used with caution in patients with gout or history of hyponatraemia.
Beta blockers should be avoided in patients with bronchial asthma, reactive airways disease, or second- or third-degree heart block.
ACEI should not be used in patients with a history of angioedema.
Aldosterone antagonists and potassium sparing diuretics can cause hyperkalaemia and should generally be avoided in patients who have serum potassium values of more than 5.0 mEq/l while not taking medications.

**Drug combination used for**

• Additive effect – use drugs acting by different mechanisms.
• Minimisation of the side effects:
  – Block opposing effects on homeostatic mechanism.
  – Block predictive side effects.
  – Permits use of lower dose (less side effects).

**Fully additive drug combinations**

• Diuretic + ACEI.
• CCB + ACEI.
• CCB+ARB

**Questionable drug combinations**

(a) Non-additive
  – beta-blocker + ACEI
  – ARB + ACEI.

(b) Side effects additive
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- beta blocker + Verapamil/Diltiazem (older CCB).
- alpha blocker + CCB.

**Drug combinations for patients with associated conditions**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Drug Combinations</th>
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</thead>
<tbody>
<tr>
<td>HT with angina</td>
<td>Beta blocker + CCB</td>
</tr>
<tr>
<td>HT with heart failure</td>
<td>Diuretic + ACEI</td>
</tr>
<tr>
<td>HT with diabetes mellitus</td>
<td>ACEI + CCB</td>
</tr>
<tr>
<td>HT with COAD</td>
<td>Diuretic + CCB</td>
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</tbody>
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**Isolated systolic hypertension (Systolic BP ≥ 160 mmHg)**

Drugs of choice in order of preference are:
- Diuretics.
- CCB
- Diuretic + CCB.
- ACEI + CCB.

**Hypertension in pregnancy**

Alpha-methyldopa, beta blockers and vasodilators are preferred medications for safety of the foetus. ACEI and Angiotensin II receptor blockers are contraindicated.

**Hypertension in children and adolescents**

In children and adolescents, hypertension is defined as BP that is, on repeated measurement, at the 95th percentile or greater adjusted for age, height and gender. The fifth Korotkoff sound is used to define diastolic BP. Be alert for possibility of identifiable causes of hypertension in younger children (i.e. kidney disease, coarctation of aorta). Lifestyle interventions are strongly recommended, with pharmacological therapy instituted for a higher levels of BP or if there is insufficient response to lifestyle modification. Choices of antihypertensive drugs are similar in children and adults, but effective doses for children are often smaller and should be adjusted carefully.

**Commonly used antihypertensive drugs**

- Tab. Hydrochlorothiazide 12.5-25 mg once daily
- Tab Chlorothalidone 12.5 mg OD
- Tab. Indapamide 2-5 mg once daily
- Tab. Atenolol 25-100 mg daily or Metoprolol 25-150 mg 2 times a day (Caution: Contraindicated in asthma, peripheral arterial disease, moderate-to-severe congestive heart failure, uncontrolled hypothyroidism, myocardial conduction defects) Or
  - Tab. Amlodipine 2.5-20 mg daily or Cap. Nifedipine 20-80 mg as sustained release daily (Caution: May cause peripheral oedema in some individuals)
- Tab. Enalapril 2.5-20 mg and may be increased to 20 mg daily or Lisinopril 2.5-20 mg daily or Ramipril 1.25-10 mg daily. (Caution: May cause dry cough in some individuals)
Tab. Losartan 25-50 mg once or twice daily
Or
Tab. Prazosin 1-10 mg/day first dose to be given at bedtime Or
Tab. Terazosin 1-10 mg daily in 2 divided doses. First dose to be given at bedtime
Or
Tab. Clonidine 0.05-0.6 mg 2 or 3 times a day
Or
Tab. Methyldopa 250-1000 mg 2 or 3 times a day

**Accelerated hypertension**
Patients presenting with BP 220/130 mmHg or more without papilloedema:
- Inj. Enalapril 1.25-5 mg IV 6 hourly
Or
- Inj. Nitroprusside 0.25-1.0 mcg/kg/min IV infusion (dose to be titrated with BP, maximum dose for 10 mcg only)
Or
- Inj. Nitroglycerin 5-100 mcg/min infusion
Or
- Inj. Hydralazine 10-20 mg IV or IM, if no response at 20 mg change the drug
Or
- Inj. Labetalol 20-80 mg IV every 5-10 min up to a total of 300 mg, infusion 0.5-2 mg/min, oral 100-600 mg 2 times a day Or
- Inj. Phentolamine 5-15 mg IV (specially useful in pheochromocytoma)

**Malignant hypertension**
Patients presenting with BP 220/130 mmHg and evidence of vascular damage like papilloedema, deranged renal function should be referred to a tertiary care centre.

N.B. - Do not use S/L nifedipine

**Resistant hypertension**
Resistant hypertension is the failure to reach goal BP in patients who are adhering to full doses of an appropriate 3-drug regimen that includes a diuretic. Refer these patients to a hypertension specialist.

**Follow-up**
1. Regular BP monitoring to ensure optimal or normal BP in young or middle aged or diabetic subjects (130/80 mmHg) and at least high normal BP in elderly patients (140/90 mmHg).
2. Monitor all risk factors for coronary artery disease.
3. Ensure compliance with lifestyle modifications.
4. Assess for complications and target organ damage.
5. In accelerated or malignant HT, avoid sudden fall in diastolic BP to below 100 mmHg.

**Patient education**
- Explain consequences of uncontrolled HT and the need for the long-term control with medication.
- Advise regarding control of other risk factors like diabetes, hyperlipidaemia, etc.
• Diuretics should be taken in the morning and if two doses a day are required second dose should be given before 4 PM.
• Manage target organ damage – referral to a higher centre for CAD (PTCA/CABG), nephropathy (prepare for dialysis or transplant), carotid Doppler to determine presence of atheroma and possibility of endarterectomy.

**Referral criteria**

All cases of secondary HTN for evaluation, accelerated HTN, malignant HTN, & resistant HTN should be referred to higher centres for effective control.

**References**


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**CORONARY ARTERY DISEASE (CAD)**

**ANGINA PECTORIS**

**Introduction**

Myocardial ischaemia is caused by critical obstruction in coronary arteries due to atherosclerosis, calcific aortic stenosis or rarely due to spasm/embolism manifests as angina pectoris.

**DIAGNOSTIC CRITERIA**

- Typical anginal pain is characterised by precordial or retrosternal discomfort or pressure or tight, squeezing, choking pain usually precipitated by exertion and relieved by rest and sublingual nitroglycerin. Pain may radiate to left shoulder, arm, neck or jaws.
- Atypical symptoms of acute coronary syndrome may be more common in the elderly, women and diabetic patients, but any patient may present with atypical signs and symptoms.

**Rational investigation** – ECG, TMT, ECHO, Blood sugar, lipid profile.

**Treatment**

**Non-drug**

Avoid heavy exertion and take rest during acute stage, lifestyle modification, smoking cessation, weight control.

**Drug**

1. For immediate relief, Tab. Isosorbide dinitrate 5-15 mg sublingual during the attack, dose repeated as required and tolerated. The onset of action is within 3 minutes.

Or

Tab. Nitroglycerin 0.3-0.6 mg sublingual during the attack, dose repeated as required.

2. If attacks are more than twice a week, regular drug therapy is required.
Tab. Isosorbide mononitrate 20 mg 2 times a day orally, may be increased to 120 mg per day, if required.
3. If no contraindications exist, Tab. Metoprolol 50 mg 2 times a day Or
4. If beta-blockers are contraindicated or angina persists, Tab. Diltiazem 60-120 mg/ day orally
5. Tab. Aspirin 100-150 mg per day orally Or
   If patient cannot tolerate Aspirin, Tab. Clopidogrel 75 mg/day
6. If still ischaemia is not controlled, Tab. Nicorandil 5- 10 mg 2 times a day can be started

Patient education
All patients with stable angina due to atherosclerotic disease should receive long-term aspirin and statin therapy. Life style therapy is a part of all CAD prescription.

Referral criteria
When adequate control of anginal symptoms is not achieved with beta-blockade, a calcium channel blocker should be added. Refer patients whose symptoms are not controlled on maximum therapeutic doses of two drugs to a cardiologist. Following coronary angiography and assessment of left ventricular function, patients may be considered for coronary revascularisation by PCI (percutaneous coronary intervention) or CABG (coronary artery bypass grafting). Revascularisation for prognostic and symptomatic benefit in patients is recommended with the following anatomy: significant LMS (left main stem) disease (>50% stenosis), or proximal 3-vessel disease, or 2-vessel disease involving the proximal left anterior descending (>70% stenosis).

ACUTE CORONARY SYNDROME (ACS)
Introduction –
Acute coronary syndrome (ACS) refers to any group of clinical symptoms compatible with acute myocardial ischaemia and covers the spectrum of clinical conditions ranging from unstable angina (UA) to non-ST segment elevation myocardial infarction (NSTEMI) to ST-segment elevation myocardial infarction (STEMI).

UNSTABLE ANGINA
Diagnostic criteria –
New onset (< 1 m) anginal pain (>20 min), or angina increasing in frequency, duration or intensity or angina occurring at rest without any precipitating factor, angina following AMI (2d-1m) or revascularisation (PTCA/CABG).

Treatment
Non drug - Patient should be hospitalised i.e absolute bed rest.

Drug -
1. Tab. Aspirin 300 mg stat. If aspirin is given before arrival at hospital, a note saying that it has been given should be sent with the patient.
2. Tab. Clopidogrel 300 mg stat followed by 75 mg/day, if available and no contraindications to it.
3. Inj. Nitroglycerin 5 mcg/min IV infusion, increase dose by 2.5-5 mcg every few minutes until pain is controlled (monitor BP).
4. Continue other drugs as above.
5. Inj. Heparin – unfractionated – 1000 units/h or low molecular weight heparin (Enoxaparin) 1 mg/kg (0.6 ml for 60 kg) 12 hourly.
6. Angioplasty/CABG may be done if found suitable or disease is progressive (left main artery occlusion or equivalent lesions, three-vessel disease or two-vessel disease including proximal left anterior descending artery disease, patients with diabetes mellitus and significant coronary disease and depressed LV function).

**Patient education** -
- Control the risk factors like diabetes, obesity, hyperlipidaemia, hypertension, smoking for prevention of future heart attacks.
- Correct anaemia, if present.
- Worsening angina/unstable angina requires further investigation and should report themselves to a higher centre.

**Referral criteria** –
All pts of UA should be referred for CAG & revascularisation if suitable.

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**MYOCARDIAL INFARCTION (MI)**

**Introduction** –
The term myocardial infarction is used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischaemia. Sudden death is first manifestation in significant number of patients. Some patients may be asymptomatic and may be detected on routine ECG.

**DIAGNOSTIC CRITERIA**
Any one of the following criteria meets the diagnosis:
- Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit, together with evidence of myocardial ischaemia with at least one of the following:
  - Symptoms of ischaemia
  - ECG changes indicative of new ischaemia; new ST-T changes (STEMI) or new left bundle branch block (LBBB). Development of pathological Q waves in the ECG.
  - ECG can be normal in the first 6 hours even in frank heart attacks. Cardiac biomarkers (trop-I, trop-T) rise in 4-6 hours with a gradual fall in up to 10 days and CK-MB rises in 4-6 hours with a rapid fall over 36-48 hours; serial evaluation every 6-8 hours for 24 hours.
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

**Reasonable investigation** –
ECG, cardiac biomarkers (Trop I/T, CKMB), ECHO, CAG (coronary angiography)

**Treatment** –
**Prehospital care**
- Administer 162-325 mg of aspirin (chewed) to chest pain patients suspected of having STEMI as soon as possible unless contraindicated or already taken by patient.
• Also give clopidogrel 300 mg orally.
• Perform and evaluate ECG
  Goal of transport time is to keep total ischaemic time within 120 minutes.

**Treatment (Preferably in ICCU)**

**Non-drug**
- Diagnosis should be made as soon as possible (within 20 minutes of arrival in hospital). Admit in CCU (if available), supplementary oxygen in patients with respiratory distress and oxygen saturation d"94%, bed rest, ensure IV access, continuous cardiac monitoring and pulse oximetry, avoid visitors and outside influences, e.g. radio, newspapers; therapeutic lifestyle changes, diet, restrict sodium intake.

**Drug**
1. Inj. Morphine 2-4 mg IV, repeated 4-6 hourly, if needed.
2. Tab. Aspirin 150-325 mg PO administered as soon as possible on admission, if not taken earlier.
3. Tab. Clopidogrel 300 mg stat and 75 mg/day for maintenance, if available.
4. Nitroglycerin (GTN) S/L spray 1-2 puffs or tab isosorbide dinitrate 5-10 mg sublingual.

**First 24 hours:**
5. Confirm MI by cardiac enzymes estimation and ECG.
6. Thrombolysis should be done within 6-12 hours with the following: Inj. Streptokinase 1.5 million units IV over 30-60 min Or inj Tenectaplace 0.5 mg/kg iv bolous or Inj. rt-PA 10 mg double bolous IV in 30 minutes apart.(exuberant cost may be a hindrance)
7. Primary percutaneous coronary intervention (PCI) may be done as an alternative to thrombolysis with a goal of door-to-balloon time <90 minutes, if fibrinolytics contraindicated, with failed fibrinolytics (rescue PCI), cardiogenic shock or if diagnosis of STEMI uncertain.
8. Close monitoring as mortality is maximum in the first 24 hours.
10. Limit physical activities at least for 12 hours.
11. Do not use prophylactic antiarrhythmics but should be readily available.
12. Inj. Enoxaparin 1 mg/kg subcutaneously 12 hourly or Inj. Unfractionated Heparin 5000 unit IV 6 hrly.
14. If no contraindication, Inj. Metoprolol 2 mg IV every 2 minutes for 3 injections; if well tolerated follow with 50 mg PO started 15 minutes after last IV dose and given every 12 hourly for 48 hours and then 100 mg once a day.
15. If no hypotension or contraindications and uncomplicated MI, give Tab. Enalapril 5 mg 2 times a day orally.
16. Measure serum lipids and electrolytes and magnesium levels.
17. Start with stanis tab atorovastatin 80 mg or rosuvastatin 40 mg OD.

**After first 24 hours:**
17. Continue aspirin, beta-blockers, nitroglycerine, heparin, ACEI, analgesia (if required).
18. Observe and treat any complication – high-dose aspirin if pericarditis; diuretics if congestive heart failure; defibrillation if haemodynamic compromise in atrial fibrillation; atropine if heart block/bradycardia; intra-aortic balloon if severe hypotension.
Indications for urgent angiography/angioplasty if facility available, with a goal door-to-balloon time of 90 minutes, with onset of chest pain within 12 hrs.

**Indications for urgent surgery**
- Failed PCI with persistent chest pain or haemodynamic instability.
- Persistent or refractory ischaemia who is not a candidate for catheter intervention.
- Cardiogenic shock and coronary anatomy not amenable to PCI.
- Mechanical abnormality leading to pulmonary oedema/hypotension, e.g. papillary muscle rupture or acute ventricular septal defect.
- Life-threatening ventricular arrhythmias in the presence of €50% left main stenosis and/or triple-vessel disease.

**Indications of temporary pacing**
- Sinus bradycardia unresponsive to drugs, Mobitz type II AV block, third degree AV block, bilateral bundle branch block (BBB), newly acquired BBB, and bifascicular or trifascicular block.

**Discharge from hospital**
- Stable patients with a low risk of complications may be candidates for early discharge such as STEMI patients managed with fibrinolysis, with an uncomplicated course after 72 hours of hospitalisation. However, the duration of hospitalisation in patients treated with reperfusion therapy may be determined by other needs, such as patient education or titration of medications to optimum doses. Non-invasive testing for ischaemia should be performed before discharge to assess the presence and extent of inducible ischaemia in patients with STEMI who have not had coronary angiography and do not have high-risk clinical features for which coronary angiography would be warranted. Exercise testing early after STEMI may also be performed to assess functional capacity and ability to perform work at home/work; evaluate efficacy of current medical regimen and titrate accordingly; risk stratification for future.

**Long-term management (Secondary prevention)**
- Complete smoking cessation—at every visit ask about smoking and assist patient in quitting.
- Initiate or maintain lifestyle modification—weight-control, increased physical activity, alcohol moderation, sodium reduction and emphasis on increased consumption of fresh fruits, vegetables and low-fat dairy products.
- BP control goal is <140/90 mmHg or 130/80 mmHg, if patient has diabetes or chronic kidney disease.
- Maintain LDL-C <100 mg/L (for very high-risk patients, an LDL-C <70 mg/dl); if TG are €200 mg/dl, non-HDL-C should be <130 mg/dl whereas non-HDL-C should be <100 mg/dl for very high risk with lifestyle modification, diet and hypolipidaemic agents.
- Physical activity 30 minutes 7 days/week (minimum 5 days per week).
- Weight management goal is to maintain body mass index 18.5-24.9 kg/m²; waist circumference: men < 40 inches, women < 35 inches.
- Aspirin, beta-blockers and ACEI (selected patients)—indefinite period. Those who do not tolerate aspirin give Tab. Clopidogrel 75 mg/day. For patients undergoing coronary artery bypass grafting, aspirin (110-325 mg) should be started within 6 hours after surgery to reduce saphenous vein graft closure.
- Warfarin for compelling indication for anticoagulant therapy, such as atrial fibrillation, prosthetic heart valve, left ventricular thrombus, or concomitant venous thromboembolic disease. Use of warfarin in conjunction with aspirin and/or clopidogrel is associated with increased risk of bleeding; therefore, monitor closely.
**Patient education**

- Counsel patient to comply with medication, to stop smoking, reduce weight, regular exercise (20 minutes brisk walk at least 3 times a week), restrict fat intake to control serum lipids.

- Family members of patients experiencing STEMI to immediately activate the Emergency Response Team when symptoms appear (unresponsive to nitroglycerin or new onset chest pain manifesting as heaviness, burning, discomfort, heaviness or pain often precipitated by physical or mental exertion; neurological changes – alteration in level of consciousness, seizures, symptoms of stroke; significant acute change in pain, fluid status or skin colour) and advise them to take CPR training and familiarise themselves with the use of an automated external defibrillator (AED).

**Referral criteria –**

All pts within 12 hrs if not thrombolysed, Refractory pain within 3-5 days of inf wall or 5-7 days of ant wall AMI to avoid complications, All AMI with complications (arrhythmia or mechanical or hemodynamic) Should be referred to centres with cardiology facility.

**References**


3. Focused Update of the Guidelines for the Management of Patients with Unstable Angina/Non-S


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**INFECTIVE ENDOCARDITIS**

**Introduction –**

Bacterial endocarditis is a life-threatening infectious disease that occurs in relatively young patients with underlying rheumatic and congenital heart diseases. The blood culture is negative in a high proportion of cases; the diagnosis is often delayed and the mortality is substantial. Infection of the endothelial surface of the heart with its attendant complications. Acute bacterial endocarditis (ABE) is usually caused by *S. aureus, Group A haemolytic Streptococci, Pneumococci* or *Gonococci*. Subacute bacterial endocarditis (SABE) is usually caused by *S. viridans* or other *streptococci*, rarely by other organisms. Prosthetic valvular endocarditis (PVE) develops in 2-3% of patients within 1 year, more in aortic valve than mitral and least with porcine valves. Early onset infections (<2 months) are caused by resistant *S. epidermidis, coliforms, candida*, etc. while late infections are caused by low virulence organisms. ABE may affect normal valves, especially in intravenous drug addicts while SABE complicates deformed/damaged valves or congenital heart disease (CHD).
DIAGNOSTIC CRITERIA

Modified DUKE criteria for diagnosis of IE

Major criteria-

1. positive blood culture
   - Typical micro-organism for IE from two separate blood cultures.
     Or
   - Persistently positive blood culture for micro-organism consistent with IE. eg.
     - 2 samples drawn >12 hrs apart
     - All 3 or majority of 4 samples, 1st and last drawn at least 1hr apart, or
     - Single +ve blood culture for Coxiella Brunti or phase Ig G Ab titre > 1:800.

1. Evidence of endocardial involvement
   - +ve echo for vegetation, abscess or new partial dehiscence of prosthetic valve, or
   - New valvular regurgitation.

Minor criteria –

- Predisposing heart condition/IV drug abuse
- Fever >100.4°F (38°C)
- Vascular phenomenon
- Immunologic phenomenon
- +ve blood culture not meeting major criteria/serologic evidence of active infection with an organism consistent with I.E.

Definitive diagnosis –

- 2 major, or
- 1 major +3 minor, or
- 5 minor criterias.

Rejected diagnosis –

- Alternate diagnosis established, or
- Symptoms resolve completely within < 4 days of antibiotic tt. Or
- Surgery or autopsy within < 4 days yields no evidence of endocarditis.

Possible diagnosis –

- Not meeting either definitive or rejected diagnosis.
  - Clinical manifestations of bacterial endocarditis are highly variable and frequently non-specific. The diagnosis of bacterial endocarditis is based on clinical, laboratory and echocardiographic criteria. Classification and definitions of infective endocarditis are given in Table 3.4.
  - Fever, toxaemia, clubbing, splenomegaly, anaemia, microscopic haematuria, a new-onset or changing murmur, evidence of immune phenomena and metastatic infection.
  - Complications such as congestive heart failure (CHF), mycotic aneurysm, embolic cardiovascular accident (CVA) or other phenomena may be the presenting features.
  - Definitive diagnosis requires positive blood cultures aided by positive echocardiogram (transoesophageal positive in >90%). Negative blood culture results from prior use of antibiotics.
Treatment (Infective endocarditis should be treated as a medical emergency)

Drug:

Treatment should be started on clinical suspicion. Subsequent changes in the antibiotic regimen should be based on the results of cultures and sensitivity testing. The choice of antibiotic therapy for bacterial endocarditis is determined by the identity and antibiotic susceptibility of the infecting organism, the type of cardiac valve involved (native or prosthetic) and characteristics of the patient, such as drug allergies.

Presumptive initial treatment for SABE should cover *S. viridans*, microaerophilic and anaerobic *streptococci*.

1. Inj. Crystalline Penicillin-G 12-18 million units/24 h after test dose in 6 divided doses + Inj. Gentamicin 3 mg/kg/day IV (or IM) 8 hourly for 2 weeks.
2. Inj ceftriaxone 2mg IV OD for 2 wks + inj gentamyci 3mg /kg iv per day 8hrly for 2wks.

Enterococci.

Inj. Crystalline Penicillin-G 18-30 million units/day after test dose (in 6 divided doses) + Gentamicin 1 mg/kg 8 hourly IV for 4-6 weeks.

Table 3.4. Classification and definitions of infective endocarditis (IE).

### IE according to localisation of infection and presence or absence of intracardiac material

- Left-sided native valve IE
- Left-sided prosthetic valve IE (PVE)
  - Early PVE: <1 year after valve surgery; late PVE: >1 year after valve surgery
  - Right-sided IE Device-related, i.e. permanent pacemaker or cardioverter-defibrillator

### IE according to the mode of acquisition:

- **Health care-associated IE:**
  - Nosocomial IE: Developing in a patient hospitalised >48 hours prior to the onset of signs/symptoms consistent with IE
  - Non-nosocomial IE: Signs and/or symptoms of IE starting <48 hours after admission in a patient with health care contact defined as:
    1. Home-based nursing or intravenous therapy, haemodialysis, or intravenous chemotherapy <30 days before the onset of IE; or
    2. Hospitalised in an acute care facility <90 days before the onset of IE
    3. Resident in a nursing home or long-term care facility
- **Community-acquired IE:** Signs and/or symptoms of IE starting <48 hours after admission in a patient not fulfilling the criteria for health care associated infection.
- **Intravenous drug abuse associated IE:** IE in an active injection drug user without alternative source of infection.

### Active IE

- IE with persistent fever and positive blood cultures or
- Active inflammatory morphology found at surgery or
- Patient still under antibiotic therapy or
- Histopathological evidence of active IE
Recurrence
• Relapse: Repeat episodes of IE caused by same microorganism <6 months after the initial episode
• Reinfection: Infection with different microorganism or repeat episodes of IE caused by the same microorganism >6 months after the initial episode

Or

Inj. Ampicillin 12 g/day, given 4 hourly may be substituted for crystalline penicillin-G

In acute bacterial endocarditis, cover for staphylococci (Native Valve):

Inj. Cloxacillin 2 g IV 4 hourly for 4-6 weeks.

In penicillin-sensitive individuals

Inj. Cefazolin (2gm)IV 8 hourly for 4-6 weeks.

Or

Inj. Vancomycin 15 mg/kg IV 12 hourly for 4-6 weeks. Methicillin-resistant Staphylococcus aureus (MRSA) in native valve

Inj. Vancomycin as above.

Prosthetic valve – cloxacillin as above 6-8wks +Inj. Gentamicin as above for 2 weeks + Rifampicin 300 mg orally 8 hourly for 6-8 weeks.

MRSA – vancomycin to replace cloxacillin above.

N.B. Although usually given for several wks longer, the basic regimens recommended is same for drug of PVT & NVE except caused by staphylococci.

Follow-up

Patients with bacterial endocarditis should be monitored carefully. Clinical response occurs in 3-7 days. Change the antibiotics as per culture/sensitivity report, if required. Blood cultures should be obtained to ensure eradication of the organism. Gentamicin blood levels should be monitored with dosage adjustments as indicated and renal function should be assessed frequently when an aminoglycoside is administered. If a prolonged course of gentamicin is planned, a hearing assessment should be performed. Fever usually resolves within several days of initiation of effective antibiotic treatment, although fever may persist longer with S. aureus infection. Persistent fever after the first week of treatment suggests a septic embolic complication or inadequate antibiotic therapy. The recurrence of fever after an initial defervescence suggests a septic or non-septic embolic event, a drug hypersensitivity reaction or the emergence of a resistant strain.

Cardiac surgery is required if:
1. No response to medical treatment (especially in prosthetic valve endocarditis).
2. Worsening heart failure and the lesion is correctable.
3. Acute onset cardiac complication due to infection, e.g. septal perforation/valvular damage/stroke perivalvular extension of infection.
4. Large (>1 cm diameter) hypermobile vegetation with increased risk of embolism.

Antibiotic prophylaxis in high-risk patients

Antibiotic prophylaxis should only be considered in patients at higher risk of IE:

• Prosthetic valve or a prosthetic material used for cardiac valve repair, patients with previous IE.
• History of infective endocarditis
• Patients with congenital heart disease, cardiac transplantation recipients with cardiac valve disease
• Dental procedures requiring manipulation of the gingival or periapical region of the teeth or perforation of the oral mucosa; however, prophylaxis is not recommended for local anaesthetic injections in non-infected tissue, removal of sutures, dental X-ray, placement or adjustment of removable prosthetics or orthodontic appliances or braces. Prophylaxis is also not recommended following the shedding of deciduous or trauma to the lips and oral mucosa.

Assess individual risk and develop an appropriate management plan with following:

• Cap. Amoxycillin 2 g (in children 50 mg/kg) orally 1 hour preoperatively or Cap. Clindamycin 600 mg (in children 20 mg/kg) Or Cap. Azithromycin Or Clarithromycin 500 mg (in children 15 mg/kg) 1 hour preoperatively. In case of hypersensitivity to Penicillin and unable to take oral medication, Inj. Cefazolin Or Ceftriaxone 1 g (in children 50 mg/kg) IM or IV Or Inj. Clindamycin 600 mg (in children 20 mg/kg) IM or IV.
• Higher risk patients with prosthetic valves, genitourinary procedures, Inj. Amoxycillin 2 g IV + Inj. Gentamicin 120 mg IV before procedure followed by Cap. Amoxycillin 1 g orally 6 hours postoperatively. Substitute Vancomycin 1 g IV infusion over 100 minutes in case the patient is allergic to penicillin.

**Patient education**

• Reinforce IE prophylaxis and dental hygiene at every contact with susceptible patients.
• Good oral hygiene, including daily flossing, is an important preventive measure.
• Discuss this potential risk with dentist or other healthcare providers that may be performing invasive procedures (dental extraction, upper respiratory tract) to take prophylactic treatment.

**Referral criteria –**

All patients need a proper microbiologic diagnosis if there is no response to empiric TT with in a wk.

However IE with a

• large vegetation(>10mm) which is very likely to embolize,
• prosthetic valve endocarditis,
• IE with mechanical complications, worsening HF, abscess, valve dehiscence, valve corporation, embolism, or chordal rupture needs referral to higher centre with cardiovascular back up.

**References**

RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

Introduction –
Rheumatic fever is a multi-system inflammatory disease that occurs as a delayed sequela (2-6 weeks) to group A beta haemolytic streptococcal pharyngitis. It is commonly a disease of childhood between the ages of 5 and 15 years. The disorder is largely self-limited and resolves without sequelae but chronic and progressive damage to heart valves lead to rheumatic heart disease (RHD).

REVISED JONE’S CRITERIA 2015 –

MAJOR CRITERIA –
Moderate & high risk population (India)
1. Carditis – clinical and/or subclinical (ECHO)
2. Arthritis –
a. monarthriis or polyarthritis. Or
b. Polyarthralgia.
3. Chorea
4. Erythema marginatum.
5. Subcutaneous nodules.

MINOR CRITERIA –
Moderate & high risk population -
1. Monoarthralgia.
2. Fever (>38°C).
3. ESR > 30 mm/hr and/or CRP >3 ng/dl.
4. Prolonged PR interval after accounting for age variability (unless carditis is a major criterion)

DIAGNOSTIC CRITERIA
Diagnosis is based on the presence of two major, or one major and two minor criteria in addition to evidence of recent streptococcal infection (raised ASO titre [>333 units for children and >250 units for adults, raised anti DNase B titre], positive throat swab or recent scarlet fever) is necessary for diagnosis of rheumatic fever.
RHD most frequently affects mitral and aortic valves. Isolated aortic valve involvement is rare and tricuspid and pulmonary valve involvement is unusual.
Complications of RHD are cardiac failure and infective endocarditis.

Rational investigation- ESR, CRP, ASO, Throat swab culture, ECG, ECHO.

Treatment (Acute rheumatic fever)
Hospitalisation is needed for moderate-to-severe carditis, severe arthritis or chorea.

Non-drug
Rest is individualised according to symptoms. For arthritis, rest for two weeks is adequate. Carditis without congestive heart failure (CHF) needs 4-6 weeks of rest. In cases of CHF, rest must be continued till the CHF is controlled.
Appropriate diet is a must for a growing child with cardiac involvement.
In severe CHF, salt restriction, fluid restriction, upright posture. Protect patient from getting injured in chorea.
Drug

1. **In arthritis and mild carditis without CHF.**

   Adult - Tab. Aspirin 6-8 gm/day in 4 divided doses for 2-3 weeks, taper doses once symptoms resolve.

   In children - 100mg/kg/day for 2-3 wks followed by 60-70mg/kg/day and for older children 50 mg/kg/day for 4 weeks to be given after meals.

   **(Caution:** Avoid gastric irritants, allow frequent feeding, medicine must not be taken on empty stomach and monitor for tinnitus, deafness, respiratory alkalosis/acidosis.)

   If no response in 4 days, rule out other conditions like chronic inflammatory, myeloproliferative disorders before switching over to steroids.

2. **In carditis with CHF.**

   Prednisolone: 2 mg/kg/day, maximum 80 mg/day till ESR normalises – usually 2 weeks and taper over 2-4 weeks, reduce dose by 2.5-5 mg every 3rd day. Start aspirin 50-75mg/kg/day simultaneously to complete total 12 weeks or (20-25% reduction of prednisolone per week)

   **(Caution:** Monitor blood pressure and blood sugar.)

   Duration of treatment for arthritis is 4-6 weeks and for carditis is 3-6 months.

   If no response to oral steroid therapy, start Inj. Methyl Prednisolone 30mg/kg/day for 3 days.

   For treatment of CHF, see section on cardiac failure.

3. **In chorea.**

   Mild chorea is treated with quite environment, and sedatives like oral phenobarbitone or diazepam. If there is no response, Tab. Haloperidol 0.25-0.5 mg/kg/day in 2-3 divided doses for 2-4 weeks after clinical improvement

   Or

   Tab. Sodium valproate 15 mg/kg/day for 2-4 weeks after clinical improvement Or

   Tab. Carbamazepine 7-20 mg/kg/day for 2-4 weeks after clinical improvement Resistant cases can be treated with plasmapheresis or pimozide.

   If there are laboratory features of rheumatic activity (ESR, CRP, ASO), anti-inflammatory drugs must also be given.

4. All patients with acute rheumatic fever should be treated as if they have group A streptococci infection whether or not the organism is actually recovered from culture:

   Inj. Benzathine penicillin 1.2 MU single IM after test dose

   In children: 1.2 MU (>27 kg), 0.6 MU (<27 kg) IM single injection.

   Or

   Oral penicillin V 500 mg twice daily for 10 days. In children: 125-250 mg twice daily for 10 days. Or

   Tab Azithromycin 12.5 mg/kg/day once daily for 5 days Or

   Cap Cephalexin 15-20 mg/kg/dose twice a day for 10 days.

   In acute rheumatic fever, observe for appearance of valvular lesions (most common in the first 4 weeks of disease; for details, see Chapter 3) and in RHD for effort in tolerance, signs and symptoms of CHF, echocardiographic studies of cardiac functions.

   Usually joint pains disappear within 24-48 hours, tachycardia settles, pericardial friction rub, if present, disappears and gradually ESR comes to normal. In established cardiac lesions with CHF not controlled by medical management, patient should be referred to a higher centre for surgical intervention.
**Monitoring**

1. Monitor blood levels of salicylates.
2. Watch for salicylate toxicity (ototoxicity, hyperventilation and metabolic acidosis).
3. Follow up for response to fever and decrease in acute phase reactants; to reduce salicylates or taper steroids.
4. Echocardiography for monitoring complications of carditis.
5. Termination of anti-inflammatory therapy may be followed by the appearance of clinical manifestation. Usually not treated unless clinical manifestations are severe; reinstate aspirin or steroids in such cases.

**For secondary prevention (For prevention of recurrences)**

- Inj. Benzathine penicillin 1,200,000 units (if weight >27 kg) or 600,000 units (if weight <27 kg) IM every 3 weeks
- Tab. Penicillin V 500 mg twice a day; in children 250 mg twice a day

If the patient is allergic to penicillin, Tab. Erythromycin 20 mg/kg/dose (max 500 mg) 2 times a day

(Warning: Contraindicated in liver disease)

**Duration of prophylaxis (IAP guideline 2008)**

Duration of secondary prevention is individualised.

1. Rheumatic fever with carditis and residual valvular involvement, for 10 years or until 40 years or lifelong may be needed.
2. Rheumatic fever with carditis and no residual valvular involvement, for 10 years or up to 25 years or whichever is later.
3. Rheumatic fever without carditis, for 5 years or until 18 years whichever is later.

**Patient/Parent education**

- Early and adequate treatment of sore throat.
- Patients with RHD should avoid contact with sore throat cases and if possible environmental modifications, e.g. avoid overcrowding.
- Explain consequences of rheumatic fever and ensure monitoring to rule out residual valvular involvement and compliance with prophylaxis.
- Patients with valvular involvement should report to cardiologist for evaluation and further management.
- Explain the importance of prophylaxis against rheumatic carditis as detailed earlier.

**Referral criteria**
- Carditis refractory to tt.
- Development of HF.
- Development of Infective endocarditis.

**References**


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16. TRAUMA AND EMERGENCY
CARE OF CRITICALLY ILL

A. CARDIO PULMONARY RESUSCITATION-CPR

Introduction

Sudden cardiac arrest is a leading cause of death among adults. CPR dramatically increases the chance of survival for victims suffering from sudden cardiac arrest. Although survival rates and neurologic outcomes are poor for patients with cardiac arrest, early appropriate resuscitation involving early defibrillation and appropriate implementation of post cardiac arrest care lead to improved survival and neurologic outcomes.

Cardiac Arrest

- Cardiac arrest is a sudden stop in effective blood circulation due to failure of the heart to contract effectively or at all.
- Also known as cardiopulmonary arrest or circulatory arrest.
- It is a medical emergency.

Cardiopulmonary Resuscitation (CPR)

- CPR consists of a series of manoeuvres by which oxygenated blood supply to brain and vital organs is maintained during cardiopulmonary arrest (CPA).
- In children, CPA is not sudden but end result of long period of hypoxacmia secondary to inadequate ventilation, oxygenation or circulation.
- Therefore, prompt management of these is essential to prevent CPA, the outcome of which is poor.

Diagnosis of cardiopulmonary arrest

Cardiac arrest

- Absence of pulse in major arteries (carotid or femoral in older children and femoral or brachial in infants as carotid is difficult to palpate due to short neck).
- Absence of heart sounds on auscultation.
- Asystole / ventricular fibrillation on ECG

Respiratory Arrest

- Seeing- Absence of respiration (absent chest movements)
- Listening- Absence of air flow by bringing ears close to mouth
- Feeling- Absence air flow by keeping hands in front of the mouth or nose.

Levels of CPR. There are two levels of CPR:

- BLS (Basic Life Support). The elements of CPR provided without additional equipment. Skill and speed are most essential.
- ACLS (Advanced Cardiac Life Support). Use of equipment and drugs for assisting ventilation or circulation.

Adult Chain of Survival

American Heart Association (AHA) Adult Chain of Survival

- Immediate recognition of cardiac arrest and activation of the emergency response system.
- Early CPR with an emphasis on chest compression
- Rapid defibrillation
• Effective advanced life support
• After revival from cardiac arrest, vital parameters of the patient are to be closely monitored and to be managed accordingly.

**Paediatric Chain Of Survival**

• Prevention
• Early CPR
• Prompt access to emergency response system (108 emergency ambulance service)
• Rapid Paediatric Advanced Life Support
• Care of child for vital parameters and further management

**Basic Life Support (BLS)**

• Provide CPR as a team.
• One rescuer activates the emergency response system.
• Second begins chest compressing.
• Third is either providing ventilations or retrieving the bag mask for rescue breathing.
• Fourth is retrieving and setting up a defibrillator.

**Call for help**

• Position the victim supine on a firm flat surface with the head level with the heart.
• To assess the need for CPR, the lay rescuer should assume that cardiac arrest is present if the victim is unresponsive and not breathing or only gasping.
• There has been a change in the recommended sequence for the lone rescuer to initiate chest compression before giving rescue breaths. (C-A-B rather than A-B-C).

**Check for response**

• Gently tap the victim and ask loudly, ‘Are you okay?’
• Call the victim’s name, if you know it.
• If the victim is responsive, he or she will answer, move, or moan.
• Quickly check to see if there are any injuries or if he or she needs medical assistance.
• If the victim is unresponsive and not breathing (or only gasping), begin CPR.

**Circulation**

• Initiate chest compressions before ventilation.
• ‘**Push hard and Fast**’ on the centre of the chest and continue chest compression until an AED (Automated External Defibrillator) arrives and is ready to use.
The lone rescuer should continue the cycle of 30 compressions and 2 breaths for approximately 2 minutes before leaving the victim to activate the emergency response system.

Obtain an automated external defibrillator (AED) if one is nearby.

Rescuer should stand or kneel at the side of the patient so that his hips are at level with the victim’s chest.

Newborn

- 2 thumbs are positioned side by side on the sternum just below the nipple line, with fingers encircling chest and supporting the back.
- Compress sternum by 1.2 cm (120/min).

Infant

- Compress the sternum with 2 fingers placed just below the intermammary line.
- 2 finger (index, middle) to compress sternum by at least 4 cm at the rate of at least 100/min and do not lift the finger when compression is released.
- Two thumb encircling hands technique can also be used.

Children (1 year up to the start of puberty)

- Use heel of hand on lower half sternum with long axis of heal same as long axis of sternum.
- Compress at least 2 inches (5cm)
- Maintain rate of at least 100/min.

Adults

- The heel of one hand is placed on the lower sternum and the other hand placed on top of the first. The elbows should be locked in position with the arms straight and the shoulders over the hands.
- Sternum should depress by at least 2 inches (5 cm)
- Rate of compression of at least 100/min.
- Allow for complete chest recoil after each compression and minimize interruptions in chest compressions. (Caution: Do not exert pressure on the ribs, costal cartilages or xiphoid.)

Combination of ventilation and cardiac massage

- If both cardiac and respiratory arrest – Compression : ventilation = 30:2 in adults, children and infants with one rescuer and 15:2 with 2 rescuers.
- Compression only (CPR if unable or unwilling to provide rescue breaths, although the best method of CPR is compression coordinated with ventilations.

Airway

(b) After delivery of 30 compressions, open the airway and deliver 2 rescue breaths in case of the lone rescuer.
(c) Clear airway by cleaning blood, secretions, foreign particles (suction, if available).
(d) Prevent posterior displacement of tongue due to muscle relaxation during CPA, by head tilt and chin lift or jaw thrust (may use an airway if available).

Head tilt: Put a hand at forehead and tilt head back to sniffing or neutral position in an infant and little more in older children and adults. (Caution: in a patient with suspected cervical spine injury, head tilt should be avoided.)
Chin lift: put finger of other hand under bony part of lower jaw at chin and lift chin upward.
Jaw thrust: Place 2-3 fingers under each side of lower jaw at its angle and lift jaw.
Breathing:
- While maintaining an open airway, look, listen and feel for breathing within 10 seconds.
- Rescue breaths be given at the rate of approximately 1 breath per every 6-8 seconds (about 8-10 breaths per minute).
- Avoid excessive ventilation, with enough volume to produce visible chest rise.
- It applies to all forms of ventilation during CPR, including mouth to mouth/nose/mask/airway breath (may use bag and mask, if available).
- The rescuer is to take a deep breath and has to blow the air into the nose and mouth of the child. However in adults the air may be blown into his/her mouth with the nose pinched.
- Avoid delivering breaths that are too large or too forceful.
- Once advanced airway is in place, continue chest compressions at the rate of at least 100/min.

Defibrillation:
- It remains the corner stone therapy for ventricular fibrillation and pulseless ventricular tachycardia.
- The automated external defibrillator (AED) analyses the rhythm and gives shock via 2 electrodes placed on the patient’s torso when required.
- Cardiac arrest can be caused by 4 different abnormal rhythms:
  A. Ventricular fibrillation (VF),
  B. Pulseless ventricular tachycardia (pVT),
  C. Pulseless electric activity (PEA), and
  D. Asystole
  The first two abnormal rhythms are shockable and require shock while last two are non shockable and dont require shock.
  
  ADVANCED CARDIAC LIFE SUPPORT (ACLS):
- If ACLS facility is available, shift the patient to ACLS as soon as possible.
- If this is not available, then continue cardiac massage till spontaneous HR is more than 60-80/ min and continue artificial breathing till adequate respiratory efforts are present (good chest movement, no cyanosis or shock).
- Carry on compressions while accessing vascular access, drug delivery, advanced airway placement.
  
  Adult Cardiac Arrest ACLS 2015 Update:
- When you witness a cardiac arrest, start CPR immediately, give oxygen, attach monitor and defibrillator
- Proceed to assess whether the rhythm is shockable or not.
  - If it is a shockable rhythm, ie VF or pVT,
  - Administer the first shock, 120-200J Biphasic or 360J Monophasic.
  - Resume the CPR, continue for 2 minutes.
  - Secure an IV/IO access.
  - But if the rhythm is Non shockable, ie Asystole or PEA.
  - Continue CPR for 2 minutes.
- Secure an IV/IO line
- Inj Epinephrine 1mg repeated every 3-5 minutes of resuscitation
- Consider securing an advanced airway and capnography monitoring.
- Then again proceed to check whether the rhythm is shockable or not.
- If shockable, give the second shock.
- The second shock should be equal or higher in voltage than the initial shock.
- Resume CPR, continue for 2 minutes.
- Administer Inj Epinephrine 1mg every 3-5 minutes of resuscitation.
- Consider securing an advanced airway and capnography monitoring.
- Assess the rhythm again, if shockable, give the third shock and resume CPR for 2 minutes.
- Give Inj Amiodarone 300 mg iv bolus followed by a second dose of 150 mg.
- Identify and treat reversible causes of cardiac arrest like hypovolemia, hypoxia, hydrogen ion (acidosis), Hypo/ hyperkalemia, hyperthermia, tension pneumothorax, cardiac tamponade, toxins, coronary and pulmonary thrombosis.
- If at any point of resuscitation, a shockable rhythm reverts to an unshockable rhythm, continue CPR for 2 minutes followed by Rhythm check every Two minutes for a total of 3 cycles and proceed accordingly.
- If at any point of resuscitation, an initially non shockable rhythm reverts to a shockable rhythm, continue the shock-CPR cycle till return of spontaneous circulation (ROSC) or a total of 3 cycles and proceed accordingly.

**NEUROSURGICAL TRAUMA**

**A. HEAD INJURY**

**B. SPINAL INJURY**

**A. HEAD INJURY**

The critical objective of management of traumatic brain injury (TBI) is to prevent secondary injury which is due to raised intracranial pressure and hypoxemia.

The initial management of any trauma patient, including the TBI patient, is based on Advanced Trauma Life Support (ATLS) guide lines.

**Diagnostic features**
- Level of consciousness assessed by Glasgow Coma Scale (GCS)
- Pulse, Blood pressure
- Respiration
- Pupillary size, symmetry and reaction
- Cranial nerve palsy
- Limb movements for paresis
Investigations
- Plain CT scan of brain is the primary neuroimaging technique. The indications are
  i) Failure to reach GCS of 15 within 2 hours
  ii) Suspected open skull fracture
  iii) Any sign of basal skull fracture
  iv) More than two episodes of vomiting
  v) Age older than 65 years
  vi) Amnesia before impact for longer than 30 minutes
  vii) Dangerous mechanism of injury
- X-ray of cervical spine (AP & Translateral)
- Chest X-ray PA view
- Routine haematological investigations

Non-drug treatment
- Maintain airway by lateral position, oropharyngeal airway
- Throat cleaning by suction of saliva, vomitus and blood
- Catheterization of urinary bladder
- Prevent hypothermia if exposed to cold for long time

Drug treatment
- Intravenous fluids
- Antibiotics
- Anti convulsants: Inj. Fosphenytoin 5 mg/kg IV
- Inj. Mannitol: 0.25 to 1.0 gm/kg 4 to 6 hourly, IV

Classification of TBI by GCS
  Mild TBI: GCS 13 & 14
  Moderate TBI: GCS 9-12
  Severe TBI: GCS 3-8

Investigations
- Plain CT scan of brain is the primary neuroimaging technique. The indications are
  i) Failure to reach GCS of 15 within 2 hours
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Classification of TBI by GCS
Mild TBI: GCS 13 & 14
Moderate TBI: GCS 9-12
Severe TBI: GCS 3-8

*Risk factors include ambiguous accident history, continued post-traumatic amnesia, retrograde amnesia more than 30 minutes, clinical signs of skull fracture, headache, vomiting, focal neurological deficit, seizure, age younger than 2 years and older than 60 years, coagulation disorder, or high-energy (speed) accident.

Management of moderate TBI

Patient education
- Unconscious patient should not be given oral drink or feed
- Frequent change of posture to prevent bed sores and chest infection.

Referral Criteria:
- Moderate and severe TBI
- Patient should be transported in lateral decubitus position.
- Bleeding scalp injuries must be stitched and limb fractures must be immobilized.

References:

B. SPINAL INJURY
Road traffic accident is the most common cause of spinal cord injury. The common levels of injury on admission are C4, C5 (most common), and C6, whereas the level for paraplegia is the thoracolumbar junction (T12).

The major causes of mortality in spinal cord injury are aspiration and shock. Initial survey should be done under ATLS protocol:
1. Airway assessment (A)
2. Breathing (B)
3. Circulation and control of haemorrhage (C)
4. Brief neurological examination
Diagnostic criteria
1. Trauma patient with limb numbness, weakness, paraplegia.
2. Trauma patient with abdominal breathing or priapism.
3. Trauma patients with neck/ back pain or tenderness.
4. All comatose patients of TBI are assumed to have spinal cord injury unless otherwise excluded

Clinical Evaluation
a) History:
   - Mechanism of injury (flexion, extension, axial loading??)
   - Loss of consciousness
   - Weakness/numbness following trauma.

b) Palpation of spine for tenderness, step-off, widened interspinous space.

c) Motor level assessment

d) Sensory level assessment

e) Evaluation of reflexes.

f) Examination of autonomic system.

Investigation
1. X ray of spine, AP and trans-lateral view
2. CT scan of spine with 3D reconstruction
3. MRI of spine

Non-drug treatment
Spine immobilization
- Place patient on back board
- Rigid cervical collar
- Sandbags on both sides of head with a 3 inch adhesive strap from one side of back board to other over the forehead.
- Air bed to prevent pressure sores
- Patient should be transported in this position

Drug treatment
1. Hypotension is due to interruption of sympathetics ‘! dopamine is agent of choice.
   - Careful hydration to prevent pulmonary edema.
   - Atropine for bradycardia associated with hypotension

2. Maintain oxygenation with or without intubation.

3. Indications for intubation in spinal cord injury are acute respiratory failure, decreased level of consciousness (Glasgow score < 9), increased respiratory rate with hypoxia, partial pressure of carbon dioxide (PCO₂) greater than 50 mm Hg, and vital capacity less than 10 ml/kg.

4. In the presence of autonomic disruption from cervical or high thoracic spinal cord injury, intubation may cause severe bradyarrhythmias from unopposed vagal stimulation. Simple oral suctioning can also cause significant bradycardia. Preoxygenation with 100% oxygen may be preventive. Atropine may be required as an adjunct. Topical lidocaine spray can minimize or prevent this reaction

5. Nasogastric tube and endotracheal tube suction

6. Indwelling catheterisation to monitor input and output and also to prevent urinary retention.
8. **Temperature regulation** by cooling blankets
9. **Maintain serum electrolytes** (Na⁺, K⁺), as SCI patients are more prone for hypokalemia

No **medical management** has shown efficacy in SCI. Methylprednisolone (MP) for treatment of acute SCI are not recommended (level 1 evidence). But the use of MP for SCI, (started within 8 hours of injury) is still prevalent in the following dose:
- 30mg/kg IV bolus over 15 minutes
- Followed by 45 minutes pause
- 5.4mg/kg/hr continuous infusion (maintain during surgery too) for 23 hrs.

**Patient education:**
- Maintain neutral position of neck by hard cervical collar
- Proper skin care to avoid pressure sores

**Referral criteria:**
- If the proper facilities are not available in the hospital
- If the patient is not showing proper improvement after following above mentioned protocol.
- The vital parameters should be stabilized before referral


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**FRACTURES (BONE AND JOINT INJURIES)**

**TRAUMA AND EMERGENCY**

Bone and joint injuries:

A) Fracture – i) close (simple)  
ii) open (compound)

B) Dislocation

**A) FRACTURE:**

a) Introduction- A break in continuity in bone is called fracture.
Green stick fracture in children is incomplete break in bone.
b) Diagnostic criteria:
Clinical: history of trauma followed by
- pain, swelling, tenderness, deformity at fracture site,
- Abnormal mobility,
- loss (partial or full) of function,
- bony crepitus (except greenstick and impacted fracture)
Radiology: X-ray of the injured part (preferably digital)
c) Rational investigation: X-Ray (plain or digital if available)
d) Non drug treatment:
- Closed fracture- plaster slab or splintage (crammer wire)
- Open fracture (compound)- clean and dress the wound,
  - Ligate any bleeding vessel,
  - never try to close the skin completely by suture,
  - plaster slab or splint.
  
* Never give tight bandage to prevent bleeding.

B) DISLOCATION:

a) Introduction:
A complete disruption of contact between articular surfaces.
Incomplete disruption is called Subluxation.

b) Diagnostic criteria:
Clinical: History of trauma (except habitual and some recurrent dislocation, eg shoulder, patella) followed by-
  - pain, swelling, deformity
  - loss of function of the joint.
Radiological: X-ray

c) Rational investigation: X-ray (plain or digital if available)

d) Non drug treatment:
- manipulation under analgesia of small joint (IP joints etc) & strapping.
- manipulation of large joints can be tried under analgesia, if fails refer to higher centres.

e) Drug treatment:
- acute pain: injectable analgesic (diclofenac, tramadol etc)
- tolerable pain: oral analgesic (diclofenac, tramadol etc)


g) Referral criteria -
Patient to be referred after fracture immobilazation to secondary centre (if orthopaedic specialist is available there), otherwise to tertiary centre.

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UROLOGICAL TRAUMA AND EMERGENCIES

A. ACUTE RENAL/URETERIC COLIC

B. ACUTE URINARY RETENTION

C. HEMATURIA

D. URETHRAL INJURY

A. ACUTE RENAL/URETERIC COLIC

Introduction
Renal / Ureteric colic is not uncommon emergency in medical practice. It is most commonly caused by the obstruction of the urinary tract by calculi.

Diagnostic Criteria
The classic presentation of a ureteric colic is acute, colicky flank pain radiating to the groin. Physical examination typically shows a patient who is often writhing in distress and pacing about trying to find a comfortable position; this is, in contrast to a patient with peritoneal irritation who remains motionless to minimise discomfort.

INVESTIGATIONS
Plain radiograph of the kidney, ureter and bladder, Ultrasonography, Intravenous urography, Non contrast enhanced computed tomography

MANAGEMENT

DRUG TREATMENT
Analgesics (NSAIDs & Opioids), Alfa- Blockers, Antibiotics, Corticosteroids, Calcium channel Blockers can be used

NON DRUG TREATMENT
General: Bed rest, plenty of fluids
Surgical: Ureteroscopy, percutaneous nephrolithotomy. Alternatively, open procedures like ureterolithotomy, pyelolithotomy can be considered

Patient education
- Take medicines as directed by physician
- Bed rest
- Drink plenty of fluids
- Strain the urine during each act of voiding
- Avoid foods like tomato, spinach, meat, dry nuts etc.

Referral
The criterion for referral remains, lack of appropriate infrastructure and expertise at primary level.
The indications for referral to tertiary care centre in managing stone disease are:

1) Complex calculi (multiple stones, staghorn calculi, stones with CKD, stones with obstructive uropathy) where in the opinion of the treating physician, the patient needs nephrolurological care and advanced surgical and medical care from a infrastructure standpoint

2) Special situations such as pediatric urolithiasis, stones in ectopic kidney.

B. ACUTE URINARY RETENTION (AUR)

Introduction
Acute urinary retention (AUR) is the sudden inability to pass urine. It is usually painful and requires emergency treatment with a urinary catheter.

Diagnostic criteria
Usually the diagnosis is self-evident. The patient is very uncomfortable and unable to pass urine, with a tender, distended bladder.

Investigations
Urine- Routine and microscopic for pus cell & RBC
CBC (Complete Blood Count)
PSA (Prostate Specific antigen)

USG KUB - commonly used, as it can provide a measure of post-void residual urine as well as looking for hydronephrosis and other structural abnormalities of the renal system

RGU (Retrograde urethrogram) - to look for stricture urethra

**Management**

**Initial management**

Immediate and complete bladder decompression by inserting a Foley catheter perurethrally - An alpha-blocker should be given before removal of the catheter.

In cases of failed Urethral Catheter placement, suprapubic cystostomy (SPC) should be considered

**Secondary management**

This is dependent on the cause of the AUR. For AUR caused by prostatic enlargement: Trial without catheter (TWOC) has become a standard practice worldwide for men with BPH and AUR. In most cases, an alpha-blocker is prescribed before commencing TWOC and significantly increases the chance of success.

**Non drug Treatment**

TURP (transurethral resection of Prostate) for refractory retention due to BPH. Urethroplasty/ internal urethrotomy for stricture urethra, Circumcision and Meatotomy for Phimosis and Meatal stenosis, respectively

**Patient education**

- Taking regular medicines for prostate gland enlargement.
- Women with genitourinary prolapse may prevent urinary retention by doing exercises to strengthen the pelvic muscles.
- Dietary and lifestyle changes will help prevent urinary retention.

**Referral**

In cases of failed catheterization/ SPC, patients should be referred to higher centre.

**C. HEMATURIA**

**Introduction**

Hematuria is defined as the presence of red blood cells in the urine. When visible to the patient, it is termed gross hematuria, while microscopic hematuria is not visible to the naked eye but rather detected by the microscopic examination of the urinary sediment.

**Diagnosis criteria**

Once a patient presents with history of hematuria, first step is detailed history and clinical examination.

1. History should include nature of hematuria whether intermittent / continuous, total / initial / terminal, or episodic.
2. Associated symptoms
3. Passage of stones, tissues, clots; shape of clots (tubular, small).
4. Lower urinary tract symptoms (poor stream, frequency, urgency, nocturia, incontinence, dysuria, etc.
5. Pain, location (flank, groin, suprapubic, other), nature and other characteristics.
6. History of fever, facial puffiness, pedal edema.
7. Medications (e.g. oral contraceptives, analgesics, anticoagulants, others)
8. Co-morbidity like tuberculosis, diabetes mellitus, hypertension,

Investigations
Urine analysis, urine culture and sensitivity should be done to rule out infection, Complete blood count, renal functions, blood sugar, Urine cytology for malignancy , Urine for AFB, Intravenous urography / CT urography.

Management:
Drug treatment
Antibiotics, tranexamic acid, IV fluids, 3 way saline irrigation

Non Drug Treatment
Cystoscopy & clot evacuation, TURBT

Patient education
Drink plenty of fluids, avoid strenuous exercise, stop smoking and alcohol intake

Referral
Refractory hematuria, hematuria secondary to malignancy should be referred to higher centre

D. URETHRAL INJURY

Introduction
Urethral injury results from straddle-type falls or pelvic fractures. For females, urethral injuries are rare.

Diagnostic criteria
Inability to void, blood at the meatus, palpable bladder

Investigations
X ray Pelvis, Retrograde Urethrogram, MCU

Drug treatment
Analgesics, tranexamic acid, antibiotics

Non drug treatment
SPC (suprapubic cystostomy), Urethroplasty, urethrostomy

BURN AND SOFT TISSUE INJURY

INTRODUCTION:
- Burns are burning problems having 7 million people affected by burn injuries all over the country and we have a major share of burn injuries in our state with more than 3000 cases per year. There has been mass burn casualties in the last two decades in different parts of the state. There is increase in electrical and lightning burns in the State enormously. Burn is preventable and requires public awareness. Morbidity and mortality can be reduced by developing infrastructure in the management of burn cases. Burn can be caused by thermal, electrical, chemical, lightning, radiation, blast injury & frostbite.
• Burns cause pain, anxiety, fluid loss and dehydration, oedema& infection leading to multi-organ failure and death.

• Burns involving face, nose, mouth with inhalation burn injuries of respiratory tract cause obstruction of airways and respiratory distress. Burn may give rise to inhalation of toxic gases with fatal outcome.

• Early complications in burn includes shock, septicaemia, toxaemia with acute renal failure.

• Late complications include post burn contractures hypertrophic scar with functional loss.

CLASSIFICATION OF BURNS

• Thermal
  – Flame burn - Due to dry heat
  – Scald burn - Due to hot liquid

• Chemical
  – Acid
  – Alkali

• Electric
  – Below 1000 volts
  – Above 1000 volts

• Lightening

• Others
  – Burn from fire cracker or bomb blast
  – Respiratory burn or inhalation burns

DIAGNOSIS CRITERIA

Calculation of percentage of body surface area in adults.

Rule of Nine (Wallace)

• Head & neck - 9%
• Upper Limb - 9% (Left), 9% (Right)
• Lower Limb - 18% (Left), 18% (Right)
• Chest & Abdomen - 18% (Front), 18% (Back)
• Perineum – 1%

1 fist (Gross estimation) – 1% TBSA

Schematic diagram for percentage of burn of total body surface area (TBSA) in adults

Consult urologist as early as possible for future management

CALCULATION OF PERCENT OF BODY SURFACE AREA BURN IN CHILDREN (Lund & Browder Chart)

• For Children’s (1 to 4 years)
  ➢ Head & neck - 19%
  ➢ Anterior trunk – 13%, Posterior trunk – 13%
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- Upper Limb – 9 ½ % (Left), 9 ½ % (Right)
- Lower Limb – 17 ½ % (Left), 17 ½ % (Right)
- Genetalia – 1%

**DEPTH OF BURNS**

- First Degree - Erythema, Severe pain
- Second Degree - Blister, Pain, Looks white
- Third Degree - Leathery, Waxy white, No pain
- Fourth Degree - Same as third + Exposed bone/muscle
- Respiratory burn - Associated with Facial burn with soot in nostrils & oral cavity, difficulty in respiration, tachypnoea

**MANAGEMENT**

- **First Aid**
  
  - **Thermal Burn**
    - Pour water till pain subsides
    - Do not remove the blisters, puncture it
    - Do not wrap with blankets
    - Do not remove all cloths, only which can be easily removed
    - Wrap with clean linens till the patient reaches hospital.
    - Use only silver sulphadiazine cream 1% or silver nitrate cream should be applied

  - **Electric Burn**
    - Do not pour water.
    - Use wooden materials to detach from the source of current.
    - Palpate the pulse & heart beat, if required cardiac massage.
    - Always refer the patient to nearest hospital for assessment.

  - **Chemical Burn**
    - Pour water repeatedly with sprayer till all chemical are washed off.
    - Use silver sulphadiazine or silver nitrate cream
    - Oral fluids ORS to be given in minor burns.

  - **Lightening Burn**
    - Asystole – Cardiac resuscitation
    - Arrhythmia – Debrillation

**INVESTIGATION**

Laboratory Test

- **Blood Test**
  
  - Hb%, DC, TLC
  - PCV, RBS
  - Na+, K+, Urea, Cr
Total Protein - Albumin & Globulin
- Grouping
- ECG
- Chest X-ray
- ABG

**Thermal Burn**
- Fluid Management
- IV Fluids - Through IV Cannula – 18 G (adult) / Venesection / Central line
- Crystalloids - Ringers Lactate
- Calculated by modified Parkland formula
  - 4 ml/kg/% TBSA (for 1st 24 hours)
  - 50% in 1st 8 hours
  - Rest 50% in rest 16 hours
  - 2nd 24 hours – 50% of 1st 24 hours + 20% human albumin
- Escharotomy – if required

**Chemical Burn**
- Same as above + Eyes, Nose to be dealt by concerned specialty

**Respiratory Burn**
- 100% oxygen inhalation
- Intubation within 4 to 6 hours
- Manage metabolic acidosis

**Electric Burn**
- Same as Thermal burn + Fasciotomy (to prevent gangrene)
- Cardiac & renal abnormality to be treated accordingly

**Drug Treatment**
- Tetanus prophylaxis – T.T. & ATS
- Analgesics – Diclofenac, Tramadol
- Oral sucralfate – 1 gm 4 times
- Antibiotics – 3rd generation Cephalosporin as prophylactic Antibiotics cover

**Referral Criteria**
- 20& 30 burns > 20% (any age group)
- 20& 30 burns > 10%
- Children > 10% of any degree
- 20& 30 burns involving face, hand, feet, perineum & major joints.
- 30 burns > 5% TBSA
- All electrical, chemical, lighting & inhalation burn
- Burn with comorbidity conditions / trauma

**PATIENT EDUCATION**
- Provide psychological support to the patient and relative about the extent of burns, possible outcome and complications.
• Educate parents about prevention of accidents and burns in future by taking necessary preventive steps at home.
• Transport of patient to healthcare centre should be done at the earliest.
• Inform the relatives about the medico legal aspects of the injury and importance of evidence and dying declaration by the patient in case of homicidal burns or suspected dowry deaths.

MAXILLOFACIAL & LIMB TRAUMA

• Associated with polytrauma, road traffic accident & fall from heights.
• Recognition of nature of injury for compromise of air way & head injury
• Clinical examination
  • Injury to soft tissue of face (Facial nerve, Lacrimal duct, parotid duct)
  • Any other fractures
  • Secretion inside oral cavity with loose tooth
  • Injury to mid facial area– Fractures of Mandible, Maxilla, Orbit, Zygoma, Nose etc.

Management
• Clinical diagnosis
• Investigation
  – X-ray, OPG,
• Immediate first aid
• Positioning of the patient (semi prone)
• Cleaning of oral cavity
• Removal loose tooth
• Prevent tongue fall
• Facial wound cleaning with normal saline & apply dressing
• Immediate tracheostomy if required

Drugs
• Analgesics
• Avoid over sedation
• GCS to be evaluated
• Neurological examination of head & spinal injuries
• Other fractures to be assessed & managed
• Refer the patient to nearest tertiary center for complete treatment of patient

Limb Amputation
• Amputated part to be transported in polythene bag to be kept inside ice box or thermos flask within 6 to 8 hours to tertiary center for reimplantation.

Bibliography

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17. NEPHROLOGICAL AND UROLOGICAL DISEASES
NEPHROLOGY SECTION
NEPHROTIC SYNDROME

A clinical complex characterized by profuse proteinuria (>3.5 .73 m2/241), oedema and hypoalbuminaemia. More than 90% of cases of nephrotic syndrome in adults are due to one of these - minimal change disease, membranous glomerulopathy, focal and segmental glomerulosclerosis, inembrano-proliferative glomerulonephritis, diabetic nephropathy and amyloidosis & fibrillary glomerulonephritis also.

KEY POINTS
• Periorbital and generalized pitting oedema, transudative pleural effusions, ascites, xanthomata. Hypercoagulability predisposes these individuals to peripheral arterial or venous thrombosis, renal vein thrombosis, and pulmonary embolism.
• Proteinuria of >3-3.5 g/24 h. Renal biopsy is indicated in all adults and children > 10 years with nephrotic syndrome for establishing a definitive diagnosis, guiding therapy and estimating prognosis.
• Other associated abnormalities are hyperlipidaemia, lipiduria and hypercoagulability.

Treatment
Non pharmacological
• Moderate salt restriction, usually 1-2 giday (no cooking salt) and low cholesterol diet.
• There is no consensus regarding the optimal protein in diet for these patients. High protein diet is not recommended, as it may hasten the progression of renal disease by increasing proteinuria. A protein intake of 0.8-1 g/kg/24 h of mainly first-class proteins is recommended.

Pharmacological
Nonspecific measures. To reduce proteinuria in patients with diabetic nephropathy
1. Tab. Enalapril 1.25 - 5 mg/day as a single dose Or
Tab. Losartan 25-50 mg/day as single dose. Ranipril 5 to 20 mg in divided doses
2. Tab. Frusemide 80-250 mg/day in two divided doses (8 AM, 2 PM), depending upon the severity of edema. The aim is to remove up to 1.0 kg/day of edematous fluid; Torsemide 10 to 100 mg / Metolazone 5-20mg can be used in case of refractory edema.
   In addition, if required Tab. Spironolactone 100 mg once daily may be added.
3. Only in-patients with symptomatic thrombosis
   Tab. Warfarin 2-4 mg/day (to titrate the dose to INR of 1.5-2.0).
   Patients may be relatively resistant to heparin.

Specific. Immunosuppression
Minimal change disease
   In adults prednisolone 1mg /kg (maximum 80mg 1 per dose) or 2mg /kg or alternate day. Treatment to be continued for 6 weeks after remission of proteinuria. During those 6 weeks daily prednisolone may be changed to alternate day or to a step wise reduction in the dose.
   In children prednisolone 60mg/m2 or 2mg /kg is given and changed to 40mg/m2 after remission. In adults if there is no remission steroid to be continued up to 12 weeks.
   In treatment relapse cyclophosphamide is to be given 2mg/kg for 12 weeks. Mycophenolate mofetyl (25-35 mg/kg/day ) can reduce treatment relapse by 50% can be given for extended period of time.
   In treatment relapse steroid dependent or toxicity, rituximab (1000 mg) in 2 doses with CNI (cyclosporin
or tacrolimus) or once weekly 4 doses can be given of Rituximab 375 mg/m2.

**Steroid resistance**

Cyclosporin (3-5mg/kg) in divided doses for at least one year can be given.

**FSGS**

Prednisolone 1mg/kg (in adults) for 4 months to be given. In steroid resistance CNI (cyclosporin 3-5/kg / day or Tacrolimus 0.05-0.3 mg/kg) with low dose steroid may be given for one year.

Mycophenolate moefetil (25-35mg/kg/day) can be given. Rituximab 375mg/m2 single or 4 weekly doses can be given.

**Membranous GN**

Treat underlying disease, control BP and use of ACE inhibitors in all cases. Idiopathic MGN may be divided into low risk (<4 gm proteinuria/day) has got excellent prognosis do not require immune suppressive medication or cytotoxic drugs are not indicated.

In medium risk patients (4-8 gm proteinuria/day) besides cyclophosphamide and steroid (ponticell’s regimen can be used, CNI, (cyclosporin (3-5 mg/kg) or Tacrolimus 0.25-0.3mg/kg with low dose steroid for 6-12 months can be given.

In high risk group cyclosporin or Tacrolimus to be given for 6-12 months. If above treatments fails, ACTH (80mg) subcutaneously twice weekly for six month can be given.

Rituximab weekly doses of 375 mg/m2 for 4 weeks or 1 gm on day 1 & 18 may be tried.

Over all 40% respond, 30 – 40% remit and relapse, 10 – 20% show progressive decline progress to ESRD in 10 -15 years.

**MPGN**

In type 1 mycophenolate and steroid may be tried. In type II (dense deposit disease) also.

Treatment depends on the cause of MPGN. Look for presence of hepatitis C, disorders of alternate complement pathway requiring specialized investigations. For Idiopathic MPGN with nephrotic syndrome and progressive decline in kidney function, initial treatment with cyclosporine plus low dose steroids is recommended. Usually progresses to ESRD over 5 – 10 years.

Rapidly progressive glomerulonephritis. Needs aggressive management preferably by a specialist

Inj. Methylprednisolone 0.75 – 1 g/day for 3 days followed by oral prednisolone and cyclophosphamide.

Anti-GBM disease needs plasmapheresis in combination with cyclophosphamide and steroids if serum creatinine is > 6 mg/ dl.

**References**

6. KDIGO guidelines 2012.

*****
ACUTE RENAL FAILURE (ARF)/ACUTE KIDNEY INJURY

A significant decline in the renal excretory function, mostly associated with decreased urine output (<500 ml/day), occurring over hours or days, detected clinically by a rise in plasma concentration of urea and creatinine. Most of the times, ARF is reversible.

**KEY POINTS**

- The clinical picture is usually dominated by the primary condition, which causes ARF. Manifestations of uraemia like anorexia, nausea, vomiting, muscular cramps and signs of encephalopathy may appear later.
- For purposes of diagnosis and management ARF is divided into three categories:— Pre-renal due to renal hypoperfusion (55%).— Renal due to disease which involve renal parenchyma (40%).— Post - renal due to diseases causing urinary obstruction
- Complications of ARF include hyperkalaemia, intravascular volume overload, hypotension, hypocalcaemia, hyperphosphatemia, metabolic acidosis, anaemia, coagulation abnormalities and infections; arrhythmias, pericardial effusion, pulmonary oedema, GI bleeding due to stress ulceration.

**Treatment**

Identify the causative factors and triggering event and treat accordingly for disease-specific therapy and prevention and management of uremic complications. Nutrition: Restrict dietary protein to 0.6 gm day and carbohydrate to 100 g/day.
**Pre-renal AKI**

1. Replacement fluids - Decided according to the composition of lost fluids *e.g.* in hemorrhage: blood transfusion/packed RBCs. Normal Saline (0.9%) in case of burns, pancreatitis, diabetic ketoacidosis. Hypotonic saline (0.45%) if increased urinary or GI losses. 
   (Fluid intake 500 ml + urine output + fluid loss from other sources)
2. Management of pulmonary edema, if present.
3. In patients with cirrhosis complicated by ARF, fluids should be administered slowly and adjusted JVP. Large volume paracentesis should be accompanied by IV albumin infusion.

**Intrinsic renal AKI**

It should be managed by a specialist. Approach for treatment depends upon the likely cause.

1. Acute glomerulonephritis, vasculitis - glucocorticosteroids, cyclophosphamide, and/or Plasmapheresis.
3. Malignant hypertension, toxaemia of pregnancy - aggressive control of blood pressures.

**Post-renal AKI**

1. Suprapubic catheterization.
2. Referral to an urologist for removal of obstruction.

**Other essential measures.**

1. Strict intake/output recording.
3. Reverse causative renal insult *e.g.* restore hemodynamic, eliminate nephrotoxins.
5. Volume overload should be avoided
   - Restrict salt (1-2 g/day- avoid all table/cooking salt and avoid food rich in sodium like milk) and water.
   - Inj. Frusemide IV dose depending upon extent of overload, usual dose is 40-200 mg/day as bolus or intravenous infusion.
   - Dialysis.

**Hyponatraemia**

1. Restrict free water intake (<1 liter/day).
2. Avoid hypotonic IV solutions. In chronic hyponatrimia Tolvaptan 15mg once/twice daily in hospitalized patients for 5 to 7 days may be tried.

**Hyperkalaemia**

1. Restrict dietary Potassium 40 mmol/d (*no food containing K*).
2. Inj. Glucose insulin drip- 50 ml of 50% of dextrose with 10 units of plain insulin over 10 min.
   And/Or
   Inj. Sodium bicarbonate 50-100 ml of 4.2% in IV fluid
   And/Or
   Inj. Calcium gluconate 10 ml of 10% solution over 10 (Preferable under cardiac monitor) min. (More than one steps taken if levels of serum $K^- > 6.5$).
3. Cation exchange resins e.g. Sodium or Calcium polystyrene sulphonate 15 g orally 6 hourly / may be used as retention enema in sorbitol.

**Metabolic acidosis**
1. Restrict dietary protein (0.6 g/kg/day).
2. Inj. Sodium bicarbonate IV to maintain an arterial pH o >7.2.

**Lypocalcaemia**
1. Tab. Calcium carbonate 1 g/day or
2. IV Calcium gluconate 10% 10-20 ml given over 20 minutes (if tetany).

**Hyperphosphataemia**
1. Restrict dietary phosphate intake (<800 mg/day).
2. Calcium carbonate as above.
3. Phosphate binders, Calcium acetate.

**Hyperuricaemia**
Treatment necessary only if uric acid is >10 mg/dl.
- Tab. Allopurinol 100 mg three times a day or Febuxostat 40-80 mg/day.

Dialysis is indicated for any of the following:
- Overt uremia manifesting as encephalopathy, pericarditis, uremic bleeding.
- Intractable fluid overload.
- Refractory hyperkalaemia.
- Rise in urea >150-180 mg% or creatinine>6-7 mg%.
- Severe acidosis producing circulatory compromise.
- Anuria for >48 hours.
Doses of all essential drugs for the underlying disease should be adjusted according to the degree of renal impairment.

**Prevention**
Since there is no specific therapy for ischemic or nephrotoxic ARF, prevention is important and includes:
1. Aggressive restoration of intravascular volume in case of losses e.g. during surgery, trauma, burns, gastroenteritis etc.
2. Avoid/reduce the dose of nephrotoxic drugs appropriately.
3. Hypovolaemia should be avoided in patients receiving nephrotoxic drugs.

**References**

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CHRONIC RENAL FAILURE (CKD)

Chronic kidney disease (CKD) is defined as abnormality of kidney structure or function present for 3 months with implications for health. GFR of less than 60 ml/min/1.73 m$^2$, an albumin excretion rate of 30 mg/24 h or greater and urine albumin creatinine ratio (ACR) of 30 mg/g or greater are diagnostic of CKD. End-stage renal disease (ESRD) GFR < 15 ml / min1.73 m$^2$ would result in death without renal replacement therapy. The important underlying causes are diabetes mellitus, hypertension, chronic glomerulonephritis, chronic pyelonephritis, analgesic nephropathy and polycystic disease.

KEY POINTS

1. The symptoms of uraemia develop gradually and late. Fatigue, dyspnoea, anorexia, nausea, vomiting, ankle oedema, pruritis, purpura, and neuromuscular disturbances. Nocturia may be present.
2. Examination may show the presence of pallor, nail dystrophy, purpura, hypertension, cardiomegaly, CHF, features of pulmonary oedema, pleural effusion and pericarditis with or without effusion. Fundus examination may show changes of hypertensive or diabetic retinopathy. The abnormalities in blood urea and creatinine may be detected during evaluation of anaemia.
3. Elevated blood urea and creatinine, hypocalcaemia, hyperphosphatemia, hyperkalaemia and a partially compensated metabolic acidosis. Peripheral smear shows a normocytic, normochromic anaemia and urinalysis usually reveals proteinuria and low fixed specific gravity. Presence of shrunken kidneys on ultrasound suggests end-stage renal disease. Kidney biopsy to be done if the kidneys are of normal size. A skeletal survey may show evidence of renal osteodystrophy. In certain diseases like diabetic nephropathy, polycystic kidney disease, kidney size may be normal.

Treatment

1. Identification and management of associated factors precipitating acute or chronic renal failure e.g., drugs, hypovolaemia, infections, obstructive uropathy, hypertension, CHF, pregnancy or presence of any life threatening emergency, requiring urgent treatment e.g. hyperkalaemia, pulmonary oedema, metabolic acidosis, uraemic encephalopathy or accelerated hypertension (Treat as mentioned under respective conditions).
2. Identification of specific cause of CRF and their treatment so as to delay the progress of CRF.
3. Modify loading and maintenance doses of drugs that are excreted through renal route.

Nonpharmacological

- Decrease protein intake to 0.6 g/kg/day of high quality protein.
- Phosphate restriction to 1000 g/day to reduce soft tissue calcification (avoid milk, egg etc.).
- Moderate sodium restriction to 60 mmol/day (low salt during cooking and avoiding foods rich in sodium) to control BP and edema.
- Potassium restriction if CRF is moderate to severe (foods rich in K. / include banana, citrus fruits, coconut water, papaya etc.)
- Sodium bicarbonate (baking powder) 600 mg 4 times a day if plasma HCO$_3^-$ is less than 20 mmol/liter
**Pharmacological Control of hypertension, cardiovascular and pulmonary abnormalities**

Target BP is 130/80-85 mmHg and in-patients with proteinuria >1 g/day, target BP is 125/75 mmHg.

The preferred drugs are:

Tab. Frusemide 40-160 mg per day./metolazone 5 to 10 mg

Or

Tab. Amlodipine 5-20 mg per day.

And/Or metoprolol 50 to 100 mg/day or Hydralazine dose 25 mg to 50 mg 3 to 4 times in a day, minoxidil 2.5 – 5 mg once or twice a day if BP remains higher than 180 mm of hg.

Tab Clonidine 100 µmg to 200 µmg 8 to 6 hourly can be added.

- Nifedipine retard 10 to 20 mg 6 hourly.
- In diabetic nephropathy or CKD with proteinuria - ACE inhibitor/ARB with or without diuretic are preferred in standard initial dose. If K⁺ is high or >30% drop in GFR then ACE & ARB should be stopped and avoided).

**Treatment of pericarditis**

Uremic pericarditis is an absolute indication for initiation or intensification of dialysis. Heparin free dialysate should be used.

**Treatment of anemia**

1. Look for common aggravating causes of anemia e.g. GI blood loss, iron deficiency and chronic infections and treat accordingly. Assess iron status of patient before Erythropoietin (EPO) therapy.

2. Iron supplementation to ensure adequate response to EPO. If required iv iron Sucrose dose – 100mg vials as infusion in 100 ml saline up to 5 doses or iron carboxymaltose 1000 mg ample in 100 ml infusion could be used.

3. Inj. EPO subcutaneous 80-120 units/kg/week (divided into 2-3 times a week) The target Hb should be 10-12 gm/ dl and optimal rate of correction should be to increase haematocrit by 4-6% over 4-week period. Long acting ESA such as darbapoitin 0.45 mcg/kg or CERA given per month S/C in 25 to 60 µgdosage

**Treatment of bleeding diathesis**

Usually problem arises when a patient of CRF needs to undergo some surgery. Inj. Vasopressin (DDAVP) 0.3 meg/kg in 100 ml of saline in 30 min, to be administered before surgery.

**Treatment of bone, phosphate and calcium abnormalities and acid base disturbances**

1. Phosphate restricted diet.

2. Calcium acetate - minimum of 1 g/day. Sevlamer in divided doses/calcium acetate-667-1334mg in devided doses

3. Vitamin D./Calcitriol - 0.25- 2 mcg/day. Sodabicab . 2gm in 3 to 4 divided doses if GFR < 60 ml; Folic acid 5 mg for hyperhomocystyenemia may be added.

Maintain serum calcium at about 10 mg% and phosphate at about 4.5 mg%.

**Treatment of hyperuricaemia (gout) if it is symptomatic**

Tab. Allopurinol 100-200 mg/day or febuxostat 40-80 mg preferably after food, then adjusted according to
plasma or urinary uric acid concentration. Management of metabolic acidosis should aim to maintain a near normal value of bicarbonate. Calcium carbonate is usually adequate. If needed sodium bicarbonate can also be added. For monitoring the progression of renal failure measure serum creatinine and creatinine clearance. ECG may show evidence of Left ventricular hypertrophy (LVH), pericarditis or hyperkalaemia.

**Absolute indications for dialysis**

Development of complications that cannot be controlled by conservative and pharmacological means i.e., fluid overload, severe hypertension, pericarditis, refractory hyperkalaemia, severe metabolic acidosis, encephalopathy and progressive neuropathy attributable to uremia.

**Renal replacement therapy (RRT)**

The choice of modality include - haemodialysis, continuous ambulatory peritoneal dialysis or renal transplantation; the choice depends on many factors - their availability, patient’s preference and availability of potential donors. Only kidney transplantation offers the potential for nearly complete rehabilitation.

**Renal Transplantation:**

- **Induction of Immunosuppression:**
  - Basimiximab 20 mg in 2 doses on day 0 and day 4 with 500 ml NS as infusion.
  - Or
  - Rabbit antithymocytic globulin 3 mg/kg on day 0 and day 3 or any other standard protocol; as infusion along with prior methyl prednisolone 5 – 6 mg/ kg BW.

- **Maintenance immunosuppression:**
  - Tacrolimus 0.12 – 0.2 mg/kg in 2 divided dosage/ Cyclosporin in case of Hepatitis C+ve.
  - Mycophenolate sodium 1440 mg/day or mycophenolate mofetyl 2 gm/day or Azathioprine 2.5 mg/kg BW day. And prednisolone 20-30 mg to be continued and tapered drug level estimation to be carried out as per institutional protocol.

**Patient education**

Counseling as must for these patients have varying reactions to illness from anger to depression. Prepare the patient physically and psychologically for renal replacement therapy, when ESRD is inevitable.

**References:**


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URINARY TRACT INFECTIONS (UTI)

UTI is defined as an infection of any part of the urinary tract. UTIs are common bacterial infections managed in general practice, particularly in sexually active women except in first year of life and in elderly, UTI predominantly affect females.

KEY POINTS

- Lower UTI includes infections of the urethra and bladder and can present with pain and burning during micturition, frequency of micturition, urgency, dysuria, pyuria or sometimes even haematuria. Fever is usually absent in lower UTI. Acute cystitis may present with suprapubic pain or discomfort.

- Upper UTI includes infections of the kidneys and ureters. The symptoms usually develop rapidly, sometimes within a few hours. In addition to symptoms of lower UTI, these patients may have high fever, chills, rigors and pain in the loins, nausea and vomiting.

- Sometimes UTIs may be asymptomatic, and are detected accidentally when the urine is tested. Asymptomatic infections are more common in pregnant women and the elderly. Untreated, asymptomatic bacteriuria in pregnancy can progress to upper UTI which can further lead to premature delivery and poor foetal outcome.

- Urine for pus cells - microscopic exam >10 pus cells/HPF and on culture bacterial growth i.e. >10^3 is diagnostic. In asymptomatic patients, two consecutive urine specimens should be examined and >10^5/mm^3 bacteria of a single species should be demonstrable in both specimens before therapy is instituted. The presence of bacteriuria of any degree in suprapubic aspirates or >10^2/mm^3 in urine obtained by catheterization indicates infection.

- Collection of urine sample. Collection of urine sample for culture should be from the midstream and should be preceded by adequate cleaning of external genitalia avoiding any antiseptic washes. Patients with recurrent UTIs should undergo ultrasonography of the genitourinary tract and micturatingcystourethrogram to detect underlying structural or functional abnormality.

Treatment

General principles

- Except in acute uncomplicated cystitis in women, diagnosis must be confirmed before treatment is begun and antimicrobial sensitivity should direct the therapy.
- Identify the predisposing factors and correct, if possible.
- Relief of clinical symptoms does not always mean bacteriologic cure.
- Each course of treatment should be defined as failure or cure on the basis of symptoms and eradication of bacteria.
- Uncomplicated lower UTIs generally respond to short courses of therapy while upper UTIs and complicated lower UTIs require longer treatment.
- Recurrences more than two weeks after cessation of therapy nearly always represent reinfection.
- Community acquired infections particularly the first infections are usually due to more antibiotic sensitive strains.
• In hospitalized patients, those requiring instrumentation and having recurrent infections, antibiotic resistant strains are the more likely causes of UTI.

**Non pharmacological** Plenty of oral fluids

**Pharmacological**

If symptoms are severe, antibiotics may be started empirically, after sending urine samples for culture & sensitivity. Otherwise, the antibiotics can be started as suggested by the culture and sensitivity report if symptoms are not severe.

The specific treatment regimens in Table.

1. Alkalining agents may be used with certain antibiotics like cotrimoxazole to prevent precipitation of crystals.
2. Tab. Pyridium up to 2 tablets 3 times a day for the first 2-3 days as a urinary analgesic to relieve dysuria.

**Prophylaxis of UTI**

Antibiotics for prevention are recommended to women who have two or more episodes of infection within six months or three or more infections within one year. The recommended antimicrobials include daily or thrice weekly administration of a single dose of Trimethoprim sulfamethoxazole (TMP-SMZ) (80/400 mg), TMP alone (100 mg) or Nitrofurantoin (50 mg). Prophylaxis should be initiated only after bacteriuria has been eradicated with a full dose treatment regimen. Same regimen can be used as a single dose after sexual intercourse in women in whom episodes of symptomatic UTIs are related to sexual intercourse. Frequent urine cultures are essential during this period.

**Suppressive antibiotics**

Indicated in patients with repeated infections with an underlying cause, till the underlying cause is removed or controlled e.g. to infants and children with vesico-ureteric reflux till 3-5 years of age. The recommended drugs are same as above.

Note: See also UTIs in paediatric section.

**Treatment of specific cases**

**UTIs in pregnancy.** Asymptomatic bacteriuria in pregnancy must be treated appropriately.

(Caution: Cotrimoxazole and quinolones are not recommended during pregnancy).

**UTIs in elderly.** Treatment is not recommended for asymptomatic infections among the elderly, particularly men.

**UTI in catheterized patients.** Need for treatment and optimal type and duration of treatment for such patients with asymptomatic bacteriuria have not been established. Removal of catheter with a short course of antibiotic may be appropriate: If catheter cannot be removed, bacteriuria should be ignored unless patient is symptomatic or at high risk of developing bacteriuria.

**Follow-up**

Patient should be re-evaluated within 3-5 days for relief of symptoms and urine microscopic examination should be repeated. In upper UTIs, fever and other symptoms normally subside within 2-3 days after starting the treatment. If symptoms do not subside within 5 days, underlying abnormalities like obstruction to the urinary tract, stones or collection of pus should be looked for. If the infection relapses after an initial treatment for 14
days, a six week treatment is recommended.

- Both the partners to clean the genitalia before and after sexual intercourse.
- Avoid diaphragm with spermicide as a contraceptive.
- Wipe from front to back during ablation.

### Regimen of UTI Table

<table>
<thead>
<tr>
<th>Condition</th>
<th>Characteristic pathogens</th>
<th>Mitigating circumstance</th>
<th>Recommended empirical treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute uncomplicated cystitis in women</td>
<td>Es. coli, S. saprophyticus</td>
<td>None</td>
<td>3-day regimen: oral TMP- SMZ</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>100/800 mg BD, TMP 100 mg BD,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Norfloxacin 400 mg BD, Ofloxacin</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>200 mg BD, Ciprofloxacin 500 mg BD</td>
</tr>
<tr>
<td>Acute uncomplicated pyelonephritis BD in women</td>
<td>E. coli, S. mirabilis, S. saprophyticus</td>
<td>Mild to moderate illness-outpatient therapy</td>
<td>Oral Ciprofloxacin 500 mg BD, Ofloxacin 400 mg BO, Amoxicillin 500 mg TDS, Ceftazidime 100 mg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe illness-hospitalization required</td>
<td>Parenteral Ceftriaxone 200-400 mg BD, Ofloxacin 1 mg/kg TDS, Ampicillin 1g QID, Ceftriaxone 1-2 g/day for 14 days</td>
</tr>
<tr>
<td>Complicated UTI in men and women</td>
<td>E. coli, P. mirabilis, K. pneumoniae, Staphylococcus</td>
<td>Mild to moderate illness-outpatients therapy</td>
<td>Oral Quinolone for 10-14 days (Doses as above)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe illness or possible hospitalization required</td>
<td>Parenteral, Ampicillin and Gentamicin, Quinolone, Ceftriaxone (Doses as above) until</td>
</tr>
</tbody>
</table>

**Patient education**

- To drink plenty of water, at least 10 glasses per day.
- Not to control the urge to pass urine. To empty the bladder completely and very often.
- To empty the bladder alter sexual intercourse.
REFERENCES


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BENIGN PROSTATIC HYPERPLASIA

DESCRIPTION

Benign prostatic hyperplasia is a noncancerous (benign) growth of the prostate gland. It usually occurs in men over 50 years of age. The cause is unknown and believed to be due to changes in hormone levels associated with ageing.

GENERAL MEASURES

Annual follow-up with digital rectal examination (DRE). For patients presenting with urinary retention, insert a urethral catheter as a temporary measure while the patient is transferred on referral.

Avoiding drugs that prevent urinary outflow e.g. tricyclics and neuroleptics.

DRUG TREATMENT

• Tamsulosin 0.4mg daily.

NON-DRUG TREATMENT

TURP (Trans Urethral Resection of Prostate) is the preferred surgical treatment.

REFERRAL

» Renal failure.
» For biopsy if associated with constitutional symptoms or weight loss.
» Hydronephrosis.
» Recurrent urinary tract infections.
» Urinary retention.
» Urge incontinence.
» Suspected prostate cancer on digital rectal examination.
» Suspected TB of prostate gland on biopsy.
» Haematuria.
» Bladder calculi.

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18. NEUROLOGY
EPILEPSY

DEFINITION:

A seizure is a paroxysmal event due to abnormal excessive or synchronous neuronal activity in the brain. Epilepsy describes a condition in which a person has recurrent unprovoked seizures due to a chronic, underlying process.

CLASSIFICATION:

1. **Focal seizures:** Jerky movements of a part of the body with normal consciousness (simple partial seizure) or impairment of consciousness with or without complex stereotyped movements (like smacking of lips) and a typical aura (complex partial seizures).

2. **Generalized Tonic-Clonic seizures:** Episodic involuntary jerky movements of the limbs, followed by loss of consciousness, lasting from a few seconds to a few minutes. Tongue biting causing bleeding, foaming at the mouth, urinary incontinence, a typical epileptic cry preceding the fits, and exhaustion and deep sleep after the fits, and occurrence during sleep all are features that confirm the diagnosis. Diagnosis is based only on a good history or observing the fits.

3. **Absence Seizures:** If there is just a momentary stare or jerk or fall, with quick recovery, occurring in children, often many times a day then one may suspect absence seizures.

4. **Status Epilepticus (SE):** Continuous seizures lasting for at least 30 minutes or two or more discrete seizures between which there is incomplete recovery of consciousness. Compensatory mechanisms start failing after 30 minutes resulting in hypotension, decreased cerebral blood flow, hypoglycaemia, hypoxia and hyperpyrexia.

INVESTIGATION-

Based on seizure semiology, EEG, Brain Imaging required to rule out structural defects.

Evaluation should be done to rule out metabolic derangements.

Consider differential diagnosis of vasovagal syncope, non-epileptic attack (pseudo seizures) and breath-holding spells, etc.

**DRUG TREATMENT-**

**simple partial and complex partial seizure**

**CARBAMAZEPINE:** In children 15-30 mg/kg body weight per day in 2 divided doses. Initially start with a low dose and dose should be gradually increased according to response. Maintenance dose of 0.8–1.2 g daily in divided doses;
PHENYTOIN SODIUM: 300 mg/kg daily as divided doses CHILD initially 5 mg/kg daily in 2 divided doses; usual dose range 4–8 mg/kg daily (maximum 300 mg).

Generalised seizure

PHENYTOIN SODIUM: 300 mg/kg daily in divided doses

CHILD - initially 5 mg/kg daily in 2 divided doses; usual dose range 4–8 mg/kg daily (maximum 300 mg).

SODIUM VALPROATE: initially 600 mg daily in 2 divided doses, preferably afterfood. Increased by 200 mg daily at 3-day intervals to maximum of 2.5g daily in divided doses; Usual maintenance dose 1–2 g daily (20–30 mg/kg daily);

Child up to 20 kg, initially 20 mg/kg daily in divided doses, may be increased cautiously. (monitor haematological parameters); Child over 20 kg, initially 400 mg daily in divided doses, increased until control (usually in range of 20–30 mg/kg daily); maximum 35 mg/kg daily.

LEVETIRACETAM:500-2000mg in divided doses.

CLOBAZAM-5-20 mg in two divided doses.

Absence Seizures:

Sodium valproate in above dose.

Febrile Convulsions

➢ Tepid water sponging
➢ Antipyretic - Paracetomol Syrup
➢ Diazepam : Per rectum as solution,

CHILD over 10 kg, 0.5 mg/kg (maximum 10 mg), with dose repeated if necessary.

CLOBAZAM-0.5 mg/kg in two divided doses for 3 days.

GENERAL MEASURES-

✦ Make sure the patient does not hurt himself by a fall or hitting objects nearby
✦ Loosen clothes, especially around the neck so that the patient does not choke.
✦ If tongue is being bitten and bleeding place a spoon or hard object wrapped around with a handkerchief in the mouth to prevent it. Do not try to insert anything into the mouth if the tongue is not obviously being bitten.
After the fits, place the patient in recovery position so that he or she does not choke on their own saliva.

Stay with the patient till he or she regains consciousness and help him to go home or to a known person as they may be confused and disoriented and even be behaving abnormally for some time after.

**REFERRAL CRITERIA:**

All patients of uncontrolled seizures should be evaluated by neurologists. All patients of Status epilepticus should be managed in a specialized unit with ICU facility.

**PATIENT EDUCATION**

- Help patient to take drugs regularly for at least three years – after the last episode of fits
- Educate to avoid stress factor that precipitate fits like lack of sleep or alcohol or excessive physical or mental stress
- Educate the family to relate to the sick child or adult as a normal person with a curable disease and not treat it as if he is crippled. To avoid going into traffic alone or swimming or going near exposed fire so that they do not hurt themselves if they have fits. But at the same time not to be afraid and over protect the patient. Particularly do not stop children from school and do not let misunderstand about the disease wreck a marriage.

**Status Epilepticus treatment protocol**

0-15 minute

- IV. Normal saline at TKO (TKO-To Keep Open)
- If blood glucose low : 1 amp D50 (50 ml IVP) and start second IVP with D5NS
- Thiamine 100 mg IVP if given D50 or if cachectic or manourished or alcoholic
- If actively seizing
- Lorazepam IV, 10 mg at <2 mg /min
- Fosphenytoin, max. delivery rate - 150mg. / min. total doses 20 mg /kg.
- * Do not use if status is due to a metabolic cause :
  unlikely to respond to phenytoin
- * Reduced delivery date if AV block or hypotension
- * Current treatment with phenytoin is not a contraindication.

15-60min.

- For patients with decreasing seizures after fosphenytoin load : additional 10mg/kg of fosphenytoin
- For patients continuing to seize who require intubation
- Thiopental ± succinylcholine (avoid succinylcholine if possible.)
- Consider additional dose of fosphenytoin 10mg/kg
- For continuing seizures post intubation :
  Midazolam : 0.3 mg/kg by slow IV. injection, may repeat at 5-min. intervals x 3 doses. Midazolam infusion starts at 2 microgram/kg/min; increase by microgram/kg/min. every 15 min. until seizure activity stops
PERIPHERAL NEUROPATHY

INTRODUCTION

Peripheral neuropathy (PN) is damage to or disease affecting nerves which may impair sensation, movement, gland or organ function, or other aspects of health, depending on the type of nerve affected. Common causes include systemic diseases (such as diabetes or leprosy), vitamin deficiency, medication (e.g. chemotherapy, or commonly prescribed antibiotics including Metronidazole and the fluoroquinolone class of antibiotics, traumatic injury including ischemia, radiation therapy, excessive alcohol consumption, immune system disease, Coeliac disease, or viral infection. It can also be genetic (present from birth) or idiopathic (no known cause).

DIAGNOSTIC CRITERIA AND RATIONAL INVESTIGATIONS

During physical examination, specifically a neurological examination, those with generalized peripheral neuropathies most commonly have sensory or motor deficit in various forms like Distal Symmetrical, Dermatomal, Mononeuropathy or Radiculopathy pattern. A physical examination will involve testing the Motor system for muscle power, Tone, Deep tendon reflexes as well as examining the Sensory for Touch, Temperature, Pain and Vibration. For large fiber neuropathy, an exam will usually show an abnormally decreased sensation to vibration, which is tested with a 128-Hz tuning fork, and decreased sensation of light touch when touched by a nylon monofilament.

Diagnostic tests include electromyography (EMG) and nerve conduction studies (NCSs). Diagnosis of small fiber involvement in peripheral neuropathy may involve a skin biopsy in which a 3 mm-thick section of skin is removed from the calf by a punch biopsy, and is used to measure the skin intraepidermal nerve fiber density (IENFD), the density of nerves in the outer layer of the skin. Reduced density of the small nerves in the epidermis supports a diagnosis of small-fiber peripheral neuropathy.

Laboratory tests include blood tests for vitamin B-12 levels, a complete blood count, measurement of thyroid stimulating hormone levels, a comprehensive metabolic panel screening for diabetes and pre-diabetes, and a serum immunofixation test, which tests for antibodies in the blood.

TREATMENT

NONPHARMACOLOGICAL

- Acupuncture: Inserting thin needles into various points on your body might reduce peripheral neuropathy symptoms.
- Herbs: Certain herbs, such as evening primrose oil, might help reduce neuropathy pain in people with diabetes.
- Transcutaneous electrical nerve stimulation (TENS).

PHARMACOLOGICAL TREATMENT

Treatment of cause
Relief of pain-

Today, many treatment options exist to alleviate symptoms, and aide in peripheral neuropathy pain relief, and it’s important to find the treatment option that will work best for you.

GABAPENTIN (300mg-900mg), PREGABALIN (75-300MG), AMYTRYPTILIN (10mg-50mg) with common NSAIDs are often prescribed medications for neuropathic pain.

REFERRAL CRITERIA:

Refer to higher centers if diagnosis could not be made. Usually can be managed at PHC level when cause is known.

PATIENT EDUCATION:

Proper care of skin if anesthesia. Report any skin ulcer.

Meningitis/ meningoencephalitis

INTRODUCTION

Meningitis: Fever with prominent headache, neck stiffness, photophobia, nausea, vomiting with or without altered mental status either acute or chronic.

Encephalitis: above features of meningitis with seizure, psychiatric manifestation, altered sensorium.

Chronic meningitis:

A characteristic neurologic syndrome due to chronic inflammation of meninges, that persists > 4 weeks & is associated with a persistent inflammatory response in the CSF with WBC>5/uL.

In adults and in children>1 year:

Fever, Intense headache, Neck stiffness, photophobia. Positive Brudzinski’s sign (patient supine, passive flexion the neck, elicits spontaneous flexion of knees) and Kernig’s signs (attempt to extend the flexed knee when the thigh is already flexed on abdomen in a supine patient elicits pain and resistance).

In children<1 year:

Classical meningeal signs often missing, fever, refusal to food, drowsiness, lethargy & altered behaviour are common. Infant may be hypotonic, neck is often not stiff, fontanelle is bulging even, when the child is not crying. Child may have diarrhoea, vomiting, convulsion.

DIAGNOSIS & INVESTIGATION:

Wherever facilities for neuroimaging is available CT scan of brain may be done.

Lumbar puncture and cerebrospinal fluid (CSF) examination.

Normal CSF is clear, and may have very few lymphocytes and proteins < 0.40 mg/dl, CSF/Plasma sugar > 0.4.
In bacterial meningitis: CSF may cloudy, opening pressure >180 mm H2O, WBC (10-10,000)/µL, protein >40MG/DL high, neutrophils predominate, glucose <40 mg/dl, ask for Gram staining and direct microscopy.

Viral meningitis: Usually Lymphocytic predominant pleocytosis with normal glucose level. Sometimes RBCs may be seen.

Chronic meningitis (tubercular meningitis): If the patient’s illness is long standing and the CSF shows high proteins and predominantly lymphocytes with low glucose, it may be tubercular meningitis. CSF ADA & TB PCR estimation may help in TBM.

Where malaria is endemic, it is important to consider cerebral malaria (rule out with blood smear examination using both thick and thin blood smears).

Enteric fever & pneumonia can also present with meningismus, fever, headache & should be ruled out when necessary especially in children.

TREATMENT:

Meningitis cases need hospitalisation. If microscopy and LP set is available, this level of diagnosis should be done at any PHC which has beds. However given present constraints all cases may need to be referred to and managed at CHC. CSF biochemistry is useful but not as essential as microscopy.

Symptomatic treatment with antipyretics should be started immediately with maintenance of hydration & correction of electrolytes.

(1) Empirical therapy should be initiated promptly whenever bacterial meningitis is a significant diagnostic consideration. Goal is to start antibiotic therapy within 60 minutes of patient’s arrival.

(2) All patients who have had recent head trauma, are immunocompromised, have known malignant lesions or CNS neoplasms, or have focal neurologic findings, papilledema or a depressed level of consciousness should undergo CT or MRI of the brain prior to lumbar puncture (LP) & Empirical antibiotic therapy should be started prior to neuroimaging and LP.

(3) A significantly depressed level of consciousness (e.g. Somnolence, coma), seizures, or focal neurologic deficits usually do not occur in meningitis; patients with these symptoms should be hospitalized for further evaluation and treated empirically for meningoencephalitis.

DRUG TREATMENT:

Empirical therapy of community acquired suspected bacterial meningitis should be as follows:

Preterm infants to infants <3 month – Ampicillin (200 mg/kg/day in 4 hourly divided dose) + cefotaxime (200mg/kg/day in 6 hourly divided doses).

Immunocompetent children >3 month & adults <55 – Cefotaxime (200mg/kg/day in 6 hourly divided doses maximum upto 12g/day), ceftriaxone (100mg/kg/day 12 hourly doses maximum upto 4 g/day) or cefepime (150 mg/kg/day in 8 hourly divided doses upto 6 g/day) + vancomycin (60 mg/kg/day in 6 hrly divided dose maximum upto 2 g/day in 12 hourly divided dose)
Adults >55 and adults of any age with alcoholism or other debilitating illnesses - Ampicillin + cefotaxime, OR ceftriaxone or ceftazidime + vancomycin (dose is same as above)

Hospital-acquired meningitis, posttraumatic meningitis, neutropenic patients, or patients with impaired cell-mediated immunity: Ampicillin + ceftriaxone (2GM 8HRLY) or meropenem (1 GM 8 HOURLY) + vancomycin

Tubercular meningitis
Treat as per RNTCP guideline.

Total duration of ATT regimen: - 2 + 10 months (induction phase: -2 months, continuation phase: - 10 - 16 months)

- In case of optic neuritis, ethambutol may be changed to streptomycin.
- Corticosteroids are indicated in all cases given for 4 wks then tapered over 2 wks.
- Inj Dexamethasone 12-16 mg in divided doses or Tab Prednisolone 1 mg/kg can be given.

Viral meningitis
Inj Acyclovir: 10 mg/kg, in 3 divided doses for at least 14 - 21 days.

REFERRAL CRITERIA:

PATIENT EDUCATION
- Patient attendant should be educated regarding left lateral position during convulsion, maintainace of hydration, temperature recording, cold sponging.

ACUTE TRANSVERSE MYELITIS

INTRODUCTION
Acute transverse myelitis is defined as the development of isolated spinal cord dysfunction over hours or days in patients in whom no evidence of a compressive or vascular lesion exists.

It can be categorized into 2 types:

1) Acute complete transverse myelitis (ACTM), idiopathic inflammation of the spinal cord causing symmetrical and moderate or severe loss of function relative to a distinct spinal cord level.

2) Acute partial transverse myelitis (APTM), asymmetric involvement or partial involvement of the spinal cord.

It is an acute/subacute onset (in hours to days) of back pain, progressive paraparesis/w girdle like sensation & zone of hyperesthesia often affects thoracic region. Bladder involvement is early. There is absence of root pain, spinal tenderness or spinal deformity. The plantar response is flexor. There is partial or complete sensory loss (all modalities) with a definite upper level.
DIAGNOSIS & INVESTIGATION:

Take a detailed history & do a complete neurological examination.

R/o compressive myelitis (h/o truma, Local tenderness, Gibbus, Radicular pain) & vascular cause of myelitis (h/o sickle cell disease, heart disease, a sensory level with dissociated loss sensation, not all type of sensation affected),

Look for optic nerve or focal cerebral involvement. Decide whether it is ACTM (usually monophasic & idiopathic) or APTM (more commonly progress to MS/NMO spectrum disorder).

INVESTIGATIONS

(should be selective based on history & physical examination)

1. MRI of spinal cord with and without contrast (also exclude compressive causes).
2. CSF studies: Cell count, protein, glucose, IgG index synthesis rate, oligoclonal bands, VDRL; Gram’s stain, acid-fast bacilli, and India ink stains;
3. Blood studies for infection: HIV; RPR; IgG and IgM enterovirus antibody
4. Immune-mediated disorders: ESR; ANA; ENA; dsDNA; rheumatoid factor;
6. Vascular causes: CT myelogram; spinal angiogram.

NONDRUG TREATMENT:

Bed rest with bladder (urinary catheterisation) & bowel care (stool softener)

Passive exercise of paralysed limbs

DVT prophylaxis with stockings & heparin when appropriate.

DRUG TREATMENT:

After ruling out specific causes of ATM, Inj. Methyle prednisolone 1gm qd for 5 days followed by 1mg/kg/day for several weeks & gradual tapering.

REFERRAL CRITERIA:

Patients with persistent areflexia.

Without sensory involvement or definite sensory level

Presence of incomplete myelopathy

Presence of visual signs & symptoms

H/O recurrent myelitis
PATIENT EDUCATION-

Patient & attendants should be educated regarding importance physical exercise, importance regular bladder & bowel habits & regarding prognosis & risks.

MIGRAINE

DEFINITION

Migraine is a benign and recurring syndrome of headache associated with other symptoms of neurologic dysfunction like Nausea, Photophobia, Lightheadedness, Scalp tenderness, Vomiting, Visual disturbances, Paresthesias, Vertigo, Photopsia, Alteration of consciousness, Diarrhea, Syncope, Seizure, Confusional state. Migraine can often be recognized by its activators, referred to as triggers. Migraine may also be preceded by focal neurological phenomenon called “aura” most commonly experienced as visual alteration (flickering lights, spots; loss of vision) but it may involve sensory symptoms (pins and needles numbers) or fully reversible dysphasic speech disturbance.

DIAGNOSTIC CRITERIA-

Repeated attacks of headache lasting 4–72 h in patients with a normal physical examination, no other reasonable cause for the headache, and:

AT LEAST 2 OF THE FOLLOWING FEATURES

PLUS

AT LEAST 1 OF THE FOLLOWING FEATURES:

- Unilateral pain
- Nausea/vomiting
- Throbbing pain
- Photophobia and phonophobia
- Aggravation by movement
- Moderate or severe intensity

INVESTIGATION-

Basically clinical. Brain imaging sometimes needed to exclude Space occupying lesion.

NON PHARMACOLOGICAL MANAGEMENT-

Identification and avoidance of specific headache triggers. A regulated lifestyle is helpful, including a healthful diet, regular exercise, regular sleep patterns, avoidance of excess caffeine and alcohol, and avoidance of acute changes in stress levels. Lessening one’s response to stress by various techniques like yoga, transcendental meditation, hypnosis, and conditioning techniques such as biofeedback.

PHARMACOLOGICAL MANAGEMENT-

1. In case of mild migraine, i.e. occasional throbbing headaches, no major impairment of functioning.

Tab. Paracetamol 1000 mg stat; if required can be repeated after 4 hours

Tab Naproxen 220-550 mg PO BD. Or
2. **In case of moderate to severe headache**, i.e. three severe attacks of headache a month with significant impairment of functioning and marked nausea or vomiting.

**ORAL**-

Tab. Ergotamine (1 mg) + Caffeine (100 mg) 1 or 2 tab at onset, then 1 tab half hourly (maximum 6/day, 10/week).

Or

Tab. Sumatriptan 25-100 mg orally at onset, may be repeated after 2 hr. Max dose -200 mg/day

**NASAL SPRAY**-

Dihydroergotamine Nasal Spray Prior to nasal spray, the pump must be primed 4 times; 1 spray (0.5 mg) is administered, followed in 15 min by a second spray.

Sumatriptan Nasal Spray 5–20 mg intranasal spray as 4 sprays of 5 mg or a single 20 mg spray (may repeat once after 2 h, not to exceed a dose of 40 mg/d)

**Hospital management of acute migraine**

1. Inj. Metoclopramide 10 mg IV.

2. Inj. Sumatriptan 6 mg SC (may repeat once in 24 hours).

(Caution: Contraindicated in ischaemic heart disease and hypertension).

Or

Inj. Prochlorperazine /Metoclopamide 10 mg in 25-50 ml saline slow push over 3-4 minutes. Can be given twice or thrice a day.

**Status migranous** (A debilitating migraine attack lasting for more than 72 hours)

Inj. Dexamethasone 4 mg/ml 2 ml IV 8 hourly for 2 days.

**Prophylaxis treatment**—To be considered if number of attacks is two or more attacks per month or each attack is very severe necessitating loss of work time. Frequent headaches (more than two a week) or a pattern of increasing attacks with risk of developing medication overuse headache or failure of, contraindication to, troublesome side effects from acute medications. Start the chosen drug at a low dose and increase it slowly until therapeutic effect or side effects develop. Give each treatment an adequate trial. A full therapeutic trial may take 2-6 months. If found to be effective, it should be continued for at least 6 months and then slowly tapered to assess its continued need. If headache recurs after discontinuation of prophylactic therapy, the medication regime should be reinstated for another 6 months trial. Drugs used are as follows-

Tab. Propranolol 40-120 mg BID daily. Or

Tab. Amitriptyline 10-75 mg at bed time. Or

Tab. Flunarizine 5-15 mg per day daily. Or

Tab. Topiramate 50-200 mg daily for obese patients Or

Tab. Sodium Valproate 400-600 mg BID for Non obese patients
REFERRAL CRITERIA

Refer patient with severe migraine to hospital, if attack is not controlled by above and if the patient is dehydrated. Also consider prophylactic medications.

PATIENT EDUCATION

- Avoid precipitating factors like lack of sleep, glare, anxiety, hunger, foods like processed cheese, banana, chocolates, wine, etc.
- Avoid overdose or frequent use of ergot preparation as these can cause hypertension and precipitate vascular disease.
- Drug for acute migraine should be taken as early as possible at the onset.
- Regular, more than twice a week, or increasing consumption of analgesics over time are alarms that require attention.
- Behavioural intervention relaxation techniques and stress management techniques.

PARKINSONS DISEASE

INTRODUCTION

Parkinson’s disease is a neurodegenerative condition due to the pathology in the basal ganglia. It occurs due to degeneration of the neurons in SubstantiaNigra in form of neuronal loss, gliosis, and the presence of extracellular pigment.

CLINICAL PRESENTATION AND DIAGNOSIS

It presents with a triad of features - rigidity, bradykinesia and tremors. The tremor is of pill rolling type in resting state. Other features may be reduced arm swing, slowness of speech as well as activities, abnormal short shuffling gait and forward stooped posture. Onset is often unilateral.

NONPHARMACOLOGICAL TREATMENT

Occupational and physiotherapy improves activities of daily living along with mood and mobility. A balanced diet with fibre supplement to be taken.

PHARMACOLOGICAL TREATMENT

Treatment to be started once the Parkinsonian symptoms start affecting daily activities. Treatment to be started with low dose and to be gradually increased until the benefit or side effects occur.

1. Tab. Trihexyphenidyl 6–20 mg in 3 divided doses Or Tab. Benztropine 1–6 mg in 2–3 divided doses. (For tremors predominant PD)
2. Tab. Levodopa plus carbidopa (100–250 mg Levodopa) in divided doses.
3. Tab. Bromocriptine 2.5-10 mg/day in 2-3 divided doses. Or Tab Pramipexol 0.375-4 mg/day in 3 divided doses. Or Tab. Ropinirole 6-24 mg/day in 3 divided doses. Or Tab. Peribidil 25-100 mg/day as single dose or 2 divided doses. Any of these drugs can be combined.

Selegiline, apomorphine and amantadine have limited role in treatment. Dopamine agonists, MAO-B inhibitors, co-enzyme Q10 and vitamin E are not recommended as neuroprotective therapy for PD as these are under trial. At present, there is no drug to retard the progression of PD. Most people with PD will develop, with time, motor and nonmotor (NMS) complications and will eventually require levodopa + Dopamine agonists / COMT inhibitors, triple combination—levodopa + carbidopa + entacapone and/or dopamine agonist.

REFERRAL CRITERIA

After diagnosis can be managed by specialist.

PATIENT EDUCATION

Family members should be explained and discussed about therapeutic expectations, lifestyle modification, motor disability and non-motor symptoms. Antiparkinsonian medication should not be withdrawn abruptly to avoid the probability of developing acute akinesia or neuroleptic malignant syndrome.

STROKE

INTRODUCTION

Stroke or Cerebrovascular accident (CVA) refers to acute onset clinical syndrome of focal or global loss of brain functions lasting for more than 24 hours or leading to death with no apparent cause other than that of vascular origin. Transient ischaemic attack (TIA) refers to the condition when focal or global cerebral dysfunction disappears within 24 hours. Stroke is pathologically and clinically heterogenous. It may be cerebral infarction, intracerebral haemorrhage or subarachnoid haemorrhage.

CLINICAL PRESENTATION

Clinical Presentation varies depending on the part of brain affected. Anterior and middle cerebral artery circulation stroke may present with hemiplegia and/or aphasia with or without facial nerve involvement. Posterior circulation stroke may present with vertigo, nausea, vomiting, dysphagia, dysarthria, diplopia, dizziness, unsteadiness, multiple cranial nerve involvement or coma.

Investigations

CT scan brain to rule out haemorrhagic stroke.

ECG if available to rule out coexisting ischemic heart disease and look for left ventricular hypertrophy suggestive of established hypertension.

Echocardiogram to rule out cardiac source of embolism.

If CT scan is normal and suspicion of SAH is very high, a lumbar puncture may be done after doing fundoscopy to look for crenate RBCs to detect subarachnoid bleed if there is strong suspicion.
**DRUG TREATMENT**

**Immediately following acute stroke**

ABC-airway, breathing and circulation to be established. Precautions to be taken to avoid aspiration. If available, urgent CT scan of brain should be done. Urgent neurosurgical assessment to be sought for patients with large cerebellar infarcts/bleed, hydrocephalus, subarachnoid haemorrhage and for selected cases of cerebral haemorrhage. In the history, time of symptom onset should be noted. BP, HR, RR, RBS to be measured. Acute elevation of BP may be transient. Excessive BP reduction to be avoided. Glucose levels should be kept below 150 mg% as hyperglycaemia is harmful. Dextrose containing fluids in euglycemics to be avoided. Excessive intravenous fluids not recommended.

**Anti-oedema measures** with Inj Mannitol 1-1.5 gm/Kg/day (20% one bottle IV 6 hrly) in divided doses can be started and continued for 3-5 days. Furosemide 20-40 mg can be given in case of signs of increase ICP.

**Antihypertensives** to be started if systolic BP >220 mmHg or diastolic BP >120 mmHg, or if systolic BP is 180-220 mmHg, diastolic BP is 105-120 mmHg; or mean arterial BP is >130 mmHg. (If rt-PA is to be given BP should be <185/110). If patient is eligible for acute reperfusion therapy except that blood pressure is >185/110 mmHg, Inj. Labetalol 10-20 mg IV over 1-2 minutes to be given, may repeat once. If systolic BP 180-230 mmHg or diastolic BP 105-120 mmHg: Inj. Labetalol 10 mg IV followed by continuous IV infusion 2-8 mg/min. If systolic BP is <180 mmHg and diastolic BP is <105 mmHg, defer antihypertensive therapy. Overzealous treatment can convert ischaemic penumbra into an infarct.

Monitor blood pressure every 15 minutes for 2 hours from the start of rt-PA therapy; then every 30 minutes for 6 hours; and then every hour for 16 hours.

For haemorrhagic stroke current recommendation is to lower BP to <150/90 mmHg at earliest.

**Reperfusion therapy for Cerebral infarction:**

Thrombolytic therapy with rt-PA (0.9 mg/kg/dose) with 10% as loading dose and rest over 1 hr if there is no contraindication in cerebral ischemia to be given. It demonstrates significant improvement in functional outcome in carefully selected patients treated in specialist units. **if used within 4.5 hours of stroke onset.**

**Stroke prevention**

Treat isolated systolic hypertension. Intensive cholesterol lowering (LDL-C goal level of <100 mg/dl) with statins reduces stroke risk. Low HDL-C may be treated with niacin or fenofibrate. Tab. Aspirin 50-325 mg once daily after ischaemic stroke/TIA or Tab. Clopidogrel 75 mg once daily or extended release Dipyridamole 200 mg added to Aspirin 50 mg twice daily. For patients who have an ischaemic cerebrovascular event while taking aspirin, there is no evidence that increasing the dose of aspirin provides additional benefit. In patients with non-valvular atrial fibrillation and also after cardioembolic stroke from valvular heart disease and recent myocardial infarction. Tab. Warfarin 2 to 7.5 mg/day to keep patients INR in 2-2.5. Urgent anticoagulation is not recommended.
NON PHARMACOLOGICAL TREATMENT

Bowel and bladder care.

Preventing bed sores and DVT prophylaxis

Start physiotherapy in hospital as soon as possible and it should be continued at home after discharge. He/she should be advised to practise Yoga, Pranayam and other relaxation techniques.

REFERRAL CRITERIA

All patients suspected of ischemic stroke with onset less than 4.5 hrs should be referred to nearby specialised stroke unit for early thrombolysis therapy. Patients with stroke need to be referred to specialist and can subsequently in most cases be followed up at the PHC level.

PATIENT EDUCATION

Prompt consultation with healthcare person after stroke improves the outcome. Modification of lifestyle including regular exercise, smoking cessation (active and passive), avoiding alcohol consumption, avoiding high fat and high salt diet, taking diet high in fruits and vegetables. Control of risk factors such as hypertension, diabetes mellitus, hyperlipidaemia and cessation of cigarette smoking.

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19. PHARMACOLOGY
USE OF DRUGS DURING PREGNANCY

Deformities can occur in the fetus if the pregnant mother is exposed to some drugs during the period of organogenesis (18 to 55 days of gestation).

**Teratogenic Risk Categories of Drugs (US-FDA)**

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
<th>Comments</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Controlled studies in women fail to demonstrate a risk to the fetus in the first trimester (and there is no evidence of a risk in late trimesters), and the possibility of fetal harm appears remote.</td>
<td>No risk</td>
<td>Inj. Magnesium. sulfate, thyroxine</td>
</tr>
<tr>
<td>B</td>
<td>Either animal-reproduction studies have not demonstrated a fetal risk, but there are no controlled studies in pregnant women, or animal-reproduction studies have shown an adverse effect (other than a decrease in fertility) that was not confirmed in controlled studies in women in the first trimester (and there is no evidence of a risk in later trimesters).</td>
<td>No evidence of risk in humans</td>
<td>Penicillin V, amoxicillin, erythromycin, paracetamol, lidocaine</td>
</tr>
<tr>
<td>C</td>
<td>Either studies in animals have revealed adverse effects on the fetus (teratogenic or embryocidal or other) and there are no controlled studies in women or studies in women and animals are not available. Drugs should be given only if the potential benefit justifies the potential risk to the fetus.</td>
<td>Risk cannot be ruled out</td>
<td>Morphine, codeine, atropine, corticosteroids, adrenaline, thiopentone, bupivacaine</td>
</tr>
<tr>
<td>D</td>
<td>There is positive evidence of human fetal risk, but the benefits from use in pregnant women may be acceptable despite the risk (eg, if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective).</td>
<td>Benefit may outweigh potential risk</td>
<td>Aspirin, phenytoin, carbamazepine, valproate, lorazepam, methotrexate</td>
</tr>
<tr>
<td>E</td>
<td>Studies in animals or human beings have demonstrated fetal abnormalities or there is evidence of fetal risk based on human experience or both, and the risk of the use of the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or may become pregnant.</td>
<td>Contraindicated</td>
<td>Estrogens, isotretinoin, ergometrine, thalidomide</td>
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</tbody>
</table>
Drugs found Safe to be used during pregnancy:

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<thead>
<tr>
<th>Drug class (condition)</th>
<th>Safe drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiemetics (morning sickness, other types of vomiting)</td>
<td>Promethazine, Doxylamine, Dicyclomine, Prochlorperazine, Metoclopramide</td>
</tr>
<tr>
<td>Laxative (constipation)</td>
<td>Dietary fibre, Ispaghula, Lactulose</td>
</tr>
<tr>
<td>Drugs for peptic ulcer</td>
<td>Ranitidine, Famotidine, Omeprazole, Pantoprazole</td>
</tr>
<tr>
<td>Allergy</td>
<td>Chlorpheniramine, Promethazine</td>
</tr>
<tr>
<td>Cold-cough</td>
<td>Nasal drops (Xylometazoline, Oxymetazoline, Budesonide)</td>
</tr>
<tr>
<td>Analgesics (headache, bodyache, joint pain, visceral pain)</td>
<td>Paracetamol, Ibuprofen (low dose)</td>
</tr>
<tr>
<td>Antiamebic</td>
<td></td>
</tr>
<tr>
<td>Anthelmintic</td>
<td></td>
</tr>
<tr>
<td>Antibacterial (systemic bacterial infections)</td>
<td>Diloxanide furoate, Paromomycin</td>
</tr>
<tr>
<td>Antituberular</td>
<td>Piperazine, Niclosamide, Praziquantel</td>
</tr>
<tr>
<td>Antimalarial</td>
<td>Penicillin G, Ampicillin, Amoxycillin-clavulanate, Cloxacillin,</td>
</tr>
<tr>
<td></td>
<td>Piperacllin, Cephalosporins, Erythromycin</td>
</tr>
<tr>
<td>Antifungal (superficial and deep mycosis)</td>
<td>Isoniazid, Rifampicin, Ethambutol,</td>
</tr>
<tr>
<td>Antihypertensive</td>
<td>Chloroquine, Mefloquine, Proguanil, Quinine (only in 1st trimester),</td>
</tr>
<tr>
<td></td>
<td>Pyrimethamine + Sulfadoxine (only single dose)</td>
</tr>
<tr>
<td>Anemia</td>
<td>Topical (Clotrimazole, Nystatin, Tolnaftate)</td>
</tr>
<tr>
<td>Antidepressant</td>
<td>Methyldopa, Hydralazine, Atenolol, Metoprolol, Pindolol,</td>
</tr>
<tr>
<td>Antipsychotic (schizophrenia)</td>
<td>Nifedipine, Prazosin, Clonidine</td>
</tr>
<tr>
<td>Antimanic (bipolar disease)</td>
<td>Iron salts (oral), Iron dextran (I.M), Vit. B12, Folic acid</td>
</tr>
<tr>
<td>Anti diabetic</td>
<td>Amitriptyline, Imipramine, Fluoxetine</td>
</tr>
<tr>
<td>Corticosteroid</td>
<td>Haloperidol, Trifluoperazine</td>
</tr>
<tr>
<td>Thyroid hormone (hypothyroidism)</td>
<td>Insulin (human insulin preferred)</td>
</tr>
<tr>
<td>Antithyroid drug (thyrotoxicosis)</td>
<td>Prednisolone (oral), Topical and inhalational corticosteroid</td>
</tr>
<tr>
<td>Bronchial asthma</td>
<td>Thyroxine</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>Propylthiouracil</td>
</tr>
<tr>
<td>Antiviral (other than HIV)</td>
<td>Inhaled Salbutamol/Salmeterol, Ipratropium bromide, Beclomethasone,</td>
</tr>
<tr>
<td></td>
<td>Budesonide, Sodium cromoglycate</td>
</tr>
<tr>
<td>Antiretroviral (HIV-AIDS)</td>
<td>Heparin (unfractionated / LMW)</td>
</tr>
<tr>
<td></td>
<td>Zidovudine, Lamivudine, Nevirapine, Nelfinavir, Saquinavir</td>
</tr>
</tbody>
</table>

Ref :
DRUG THERAPY IN RENAL DISEASE

1. Calculation of eGFR: \( \text{CL}_{\text{cr}} \) (creatinine clearance)
   - Recent recommendation is CKD-EPI (Chronic Kidney Disease Epidemiology collaboration formula).
   - However practically for bedside evaluation Cockcroft Gault Formula is used, i.e The Cockcroft and Gault formula (1973)
     \[
     \text{CL}_{\text{cr}} = \left(\frac{(140-\text{age}) \times \text{weight}}{72 \times \text{SCr}}\right) \times 0.85 \text{ (if female)}
     \]
   Abbreviations/Units- \( \text{CL}_{\text{cr}} \) (creatinine clearance) = mL/minute, Age = years, Weight = kg
   SCr (serum creatinine) = mg/dL

2. Renal impairment is divided into 5 stages (Kidney Disease improving global outcome) KDIGO.
   - **Grade**  **GFR**
     - G1: Normal or high \( \geq 90 \) with evidence of kidney injury.
     - G2: Mild decrease 60 – 89
     - G3a: Mild to moderate decrease 45-59.
     - G3b: Moderate to severe decrease 30-44.
     - G4: Severe
     - G5: Kidney failure < 15.

Dosing calculation:
- **Determining Loading Dose (LD)** –
  \[
  \text{LD} = \text{Vd} \times \text{IBW} \times \{C_p\}
  \]

- Ideal body weight: (Men) – 50kg + 2.5kg for every 2.5 cm over 152 cm.
- Ideal body weight: (Women) -45.5kg + 2.3 kg for every 2.5 cm over 152 cm.

- **Determining maintenance Dose**.
  - Dosing interval = Normal \( \text{CL}_{\text{cr}} \) x Normal interval / Patients\( \text{CL}_{\text{cr}} \)
  - maintenance Dose = Patients \( \text{CL}_{\text{cr}} \) x Normal Dose / Normal \( \text{CL}_{\text{cr}} \)

No Dose modification required:
- in drugs like
  - Doxycycline
  - TG cycline
  - Chloramphenicol
  - Clindamycin
  - Linezolid
  - Cefoperazone
  - Ceftriaxone

References:
**CLINICALLY IMPORTANT DRUG-DRUG INTERACTIONS**

Most patients are treated with more than one drug. A clinically relevant drug-drug interaction (DDI) occurs when the effectiveness or toxicity of one drug is decreased or increased due to prior or co-administration of another drug, leading to therapeutic failure or drug toxicity.

**Predisposing Factors for Clinically Significant Drug Interactions**

1. Drugs with Low Therapeutic Index.
2. Drugs with High Plasma Protein binding
3. Drugs with Saturable Kinetics
4. Drugs Inducing or Inhibiting CYP450 Enzymes
5. Patients with Hepatic or Renal Impairment.
6. Patients with extremes of age
7. Patients with long term drug therapy
8. Patients on Over the Counter drugs, herbal drugs and vitamins.

The Mechanisms underlying this could be either Pharmacokinetic (where the plasma conc. of one drug is increased or decreased in the presence of another) or Pharmacodynamic (where the similar effects gets added up).

<table>
<thead>
<tr>
<th>Object Drug</th>
<th>Precipitant Drug</th>
<th>Effect seen</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warfarin</td>
<td>Ceftriaxone, Cefoperazone, Aspirin, Clopidogrel</td>
<td>Bleeding</td>
<td>Prolongation of bleeding time</td>
</tr>
<tr>
<td>Carbenicillin</td>
<td>Aspirin, Ticlopidine, Clopidogrel</td>
<td>Bleeding</td>
<td>Excessive platelet inhibition</td>
</tr>
<tr>
<td>Sildenafil</td>
<td>Glyceryl trinitrate, Isosorbide dinitrate</td>
<td>Fall in BP, Myocardial ischaemia</td>
<td>Excessive increase in cGMP production</td>
</tr>
<tr>
<td>Levodopa</td>
<td>Metoclopramide, Chlorpromazine, Haloperidol</td>
<td>Loss of antiparkinsonian efficacy of Ldopa</td>
<td>Antidopaminergic action</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Alcohol, Opioid Analgesics</td>
<td>Enhanced Sedation</td>
<td>Increased CNS depression</td>
</tr>
<tr>
<td>Thiazides</td>
<td>NSAIDs</td>
<td>Blunt antihypertensive efficacy</td>
<td>Block PG mediated effects</td>
</tr>
<tr>
<td>Furosemide</td>
<td>Corticosteroids, Diuretics</td>
<td>Decrease hypoglycemic efficacy</td>
<td>Raise Bgl</td>
</tr>
<tr>
<td>Glipizide</td>
<td>Aminoglycoside</td>
<td>Increased nephrotoxicity</td>
<td>Additive effect</td>
</tr>
<tr>
<td>Glibenclamide</td>
<td>Antacid, Sucralfate, Iron, Calcium, Zinc</td>
<td>Reduced Efficacy of Object drug</td>
<td>Interference with Oral Absorption</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>Antacids (with Al, Ca, Mg)</td>
<td>Reduced Efficacy of Object drug</td>
<td>Interference with Oral Absorption</td>
</tr>
<tr>
<td>Doxycycline, Minocycline Tetracycline</td>
<td>Fluoroquinolones, Fluvoxamine</td>
<td>Toxicity of Object drug</td>
<td>CYP1A2 Inhibition</td>
</tr>
<tr>
<td>Theophylline, Imipramine, Clozapine</td>
<td>Ketoneazole</td>
<td>Reduced Efficacy of Object Drug</td>
<td>CYP2C9 Induction</td>
</tr>
<tr>
<td>Sulfonylurea</td>
<td>O C Pill</td>
<td>Antiplatelet action</td>
<td>CYP2C19 Inhibition</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>PPIs except Pantoprazole, Isoniazid, Ketoneazole</td>
<td>Toxicity of Object drug</td>
<td>CYP2D6 Inhibition</td>
</tr>
<tr>
<td>Diazepam, Phenytoin</td>
<td>Paxil, Haloperidol, Quinidine</td>
<td>Toxicity of Object drug</td>
<td>Enhanced Carbamazepine / Statins toxicity</td>
</tr>
</tbody>
</table>

Ref:


****
Drugs Safety in Indian Population & Pharmacovigilance Programme of India (PVPI)

Pharmacovigilance Programme of India (PvPI): The Central Drugs Standard Control Organisation (CDSCO), New Delhi has initiated a nation-wide pharmacovigilance programme under the aegis of Ministry of Health & Family Welfare, Government of India. The programme is coordinated by The Indian Pharmacopoeia Commission (IPC) located at Ghaziabad which is the National Coordinating Centre (NCC) for the above programme.

Pharmacovigilance: Pharmacovigilance, as defined by the World Health Organization, is the science and activities relating to the detection, assessment, understanding and prevention of adverse events or any other possible drug-related problems. Recent inclusions to this definition are: herbals, traditional and complementary medicines, blood products, biologicals, medical devices and vaccines.

Adverse Drug Reaction (ADR): It is a noxious response to a drug which is unintended and which occurs at doses normally used in man for prophylaxis, diagnosis, or therapy of disease or for modification of physiological function.

Reporting of ADRs through PvPI: If there is any suspicion that an adverse event or adverse reaction has occurred, the health care professional attending to the patient can fill up the suspected ADR form or patient suspects that he has experienced an ADR can report to the nearest ADRs Monitoring Centres (AMCs) under Pharmacovigilance Programme of India (PvPI). The details of AMCs are given in the website of IPC i.e. www.ipc.gov.in

Information/guidance about ADR reporting: One can directly go to the link www.ipc.gov.in/PvPI/pv_home.htm and get all the information about ADR reporting and also call at toll free (1800-180-3024) helpline to ask any query related to ADR reporting.

Information on Regulatory related issues: National Regulatory Agency (NRA) website www.cdsco.nic.in i.e. Drugs Controller General of India, Central Drugs Standard Control Organization (CDSCO) Head Quarter, FDA Bhawan, Kotla Road, New Delhi 110002. Phones: (Off.) 91-11-23236965 (D) Fax: 91-11-23236973. E-Mail: dci@nic.in
### SUSPECTED ADVERSE DRUG REACTION REPORTING FORM

**FOR AMC/NCC USE ONLY**

<table>
<thead>
<tr>
<th>Date of reaction started (dd/mm/yyyy)</th>
</tr>
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<tbody>
<tr>
<td>Date of recovery  (dd/mm/yyyy)</td>
</tr>
<tr>
<td>Describe reaction or problem</td>
</tr>
</tbody>
</table>

**C. SUSPECTED MEDICATION(S)**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name (Brand/Generic)</th>
<th>Manufacturer (if known)</th>
<th>Batch No / Lot No</th>
<th>Exp. Date (if known)</th>
<th>Dose used</th>
<th>Route used</th>
<th>Frequency (OD, BD etc.)</th>
<th>Therapy dates</th>
<th>Indication</th>
<th>Causality Assessment</th>
</tr>
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<tbody>
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</tr>
</tbody>
</table>

**D. REPORTER DETAILS**

<table>
<thead>
<tr>
<th>16. Name and Professional Address:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pin:</td>
</tr>
<tr>
<td>E-mail:</td>
</tr>
<tr>
<td>Tel. No. (with STD code):</td>
</tr>
<tr>
<td>Occupation:</td>
</tr>
<tr>
<td>Signature:</td>
</tr>
</tbody>
</table>

Confidentiality: The patient’s identity is held in strict confidence and protected to the fullest extent. Programme staff is not expected to and will not disclose the reporter’s identity in response to a request from the public. Submission of a report does not constitute an admission that medical personnel or manufacturer or the product caused or contributed to the reaction.
ADVICE ABOUT REPORTING

A. What to report

- Report serious adverse drug reactions. A reaction is serious when the patient outcome is:
  - Death
  - Life-threatening
  - Hospitalization (initial or prolonged)
  - Disability (significant, persistent or permanent)
  - Congenital anomaly
  - Required intervention to prevent permanent impairment or damage

- Report non-serious, known or unknown, frequent or rare adverse drug reactions due to Medicines, Vaccines and Herbal products.

Note: Adverse Event Following Immunization can also be reported in Serious AEFI case Notification Form available on http:// ipc.nic.in/showfile.aspx?id=650&EncHid=

B. Who can report

- All healthcare professionals (Clinicians, Dentists, Pharmacists and Nurses) can report adverse drug reactions

C. Where to report

- Duly filled Suspected Adverse Drug Reaction Reporting Form can be send to the nearest Adverse Drug Reaction Monitoring Centre (AMC) or directly to the National Co-ordination Centre (NCC).
- Call on Helpline (Toll Free) 1800 180 3024 to report ADRs.
- Or can directly mail this filled form to pvpi@ipcindia.net or pvpi.ipcindia@gmail.com
- A list of nationwide AMCs is available at:

D. What happens to the submitted information

- Information provided in this form is handled in strict confidence. The causality assessment is carried out at AMCs by using WHO-UMC scale. The analyzed forms are forwarded to the NCC through ADR database. Finally the data is analyzed and forwarded to the Global Pharmacovigilance Database managed by WHO Uppsala Monitoring Centre in Sweden.
- The reports are periodically reviewed by the NCC-PvPI. The information generated on the basis of these reports helps in continuous assessment of the benefit-risk ratio of medicines.
- The information is submitted to the Steering committee of PvPI constituted by the Ministry of Health & Family Welfare. The Committee is entrusted with the responsibility to review the data and suggest any interventions that may be required.

E. Mandatory field for suspected ADR reporting form

- Patient initials, age at onset of reaction, reaction term(s), date of onset of reaction, suspected medication(s) and reporter information.

For ADRs Reporting Call on PvPI Helpline (Toll Free)
1800 180 3024
(9:00 AM to 5:30 PM, Working Days)
# MEDICINES SIDE EFFECT REPORTING FORM (FOR CONSUMERS)

**Indian Pharmacopoeia Commission, National Coordination Centre- Pharmacovigilance Programme of India, Ministry of Health & Family Welfare, Government of India.**

<p>| | |</p>
<table>
<thead>
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## 1. Name of the medicine

- [ ] 
- [ ] 
- [ ] 
- [ ]

## 2. Date of the event:

- [ ]

## 3. Description of the event:

- [ ]

## 4. Event reported by:

- [ ]

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## 5. Other details:

- [ ]

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## 6. Treatment given

- [ ]

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## 7. Follow-up details:

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## 8. Other comments:

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**Note:**

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