Ministry of Higher Education Scientific Research
Foundation of technical Education
Technical Education Institute/ Al- Mosul
Department: Community Health

Subject
Medical & surgical for students of second class

Presented by
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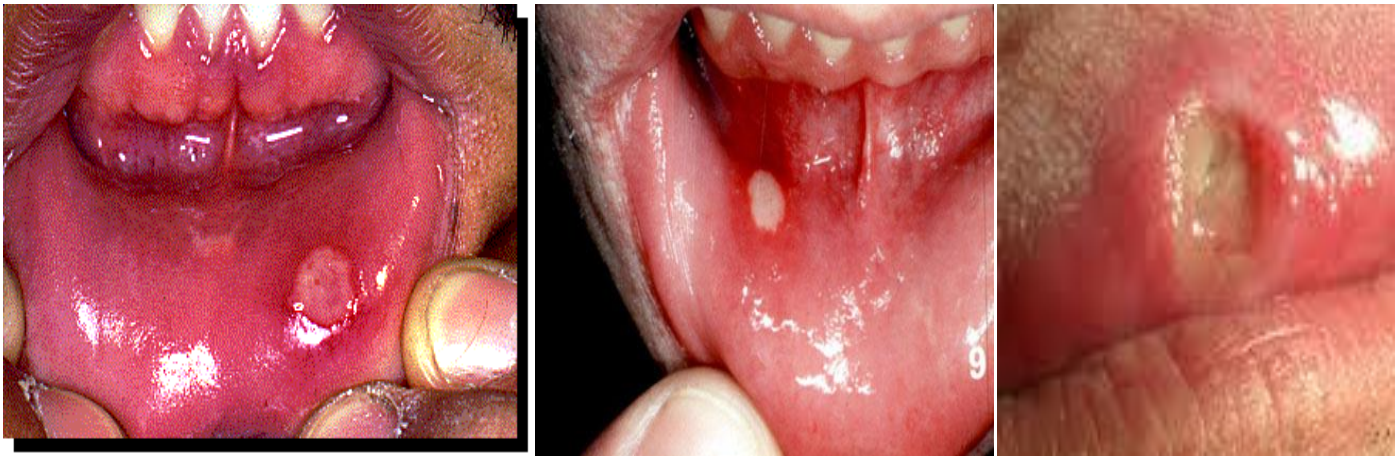
1st - 5th week

DISEASES OF THE MOUTH

APHTHOUS ULCERATION

Aphthous ulcers are superficial, painful ulcers which occur in any part of the mouth.

** Recurrent aphthous ulcers afflict up to 30%of the population and are particularly common in women prior to menstruation .



Aetiology

1- **Unknown.**
2- **In severe cases** other causes of oral ulceration must be considered.
   a-Infection; **Fungal** (candidiasis), **bacterial** (Vincentُ s angina, Syphilis),
   viral (herpes simplex).
   b- GIT diseases; Crohn's & Coeliac disease.
   c- Dermatological condition; Lichen planus, pemphigus.
   d- Drug; Hypersensitivity, cytotoxics.
   e- Systemic diseases; SLE, Behcet's syndrome.
   f- Neoplasia; Carcinoma, leukaemia.

****Occasionally ,biopsy is necessary to definite the diagnosis**

Treatment

1- **Topical steroid**(0.1% triamcinolon in orabase) gel or chlortetracycline **mouthwashes** can effect healing.
2- Symptomatic relief is achieved using local **anaesthetic mouthwashes** or **choline salicylate(8.7%).**
3- **Oral steroids** may be used for recurrent ulcers.

Vincent ́s angina

This characterized by **painful, deep, sloughing ulcers which principally affect the gum associated with fever & systemically unwell.**

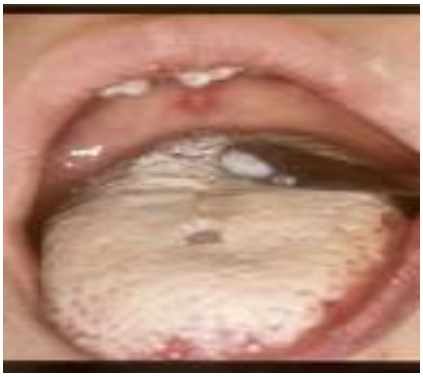
It is due to invasion of the mucous membranes by Borrelia vincenti organisms & other commensals, in case of **low resistance of host & poor oral hygiene.**

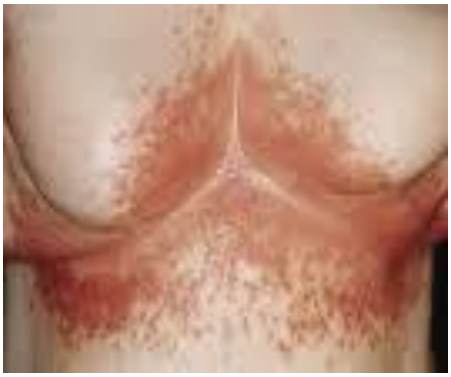
Predisposing factors; **Malnutrition , general debility & AIDS.**

Treatment

1- **Locally hydrogen peroxide mouthwashes.**
2- **Broad-spectrum antibiotic.**

Candidiasis





The fungus **candida albicans** is a normal mouth commensal but it may proliferate to cause **thrush.** This occurs in

1- Babies,

2-Debilitated patients,those receiving steroids or antibiotic therapy and

3-Immunosuppressed patients, especially those receiving cytotoxic therapy & patients with AIDS.

Clinical features

1- **White patches (thrush) on tongue & buccal mucosa** .
2- **Odynophagia** (painful swallowing) **or dysphagia**(difficult swalloing) suggest pharyngeal & oesophageal candidiasis.

****Sometimes brushings or biopsy can be obtained for mycological examination.**

Treatment

1- **Nystatin or amphotericin suspensions or lozenges**.
2- **Oral fluconazole** may required in resistant cases or immunosuppressed patients.

Infectious diseases

Diphtheria

****Infection with Coryne bacterium**,.

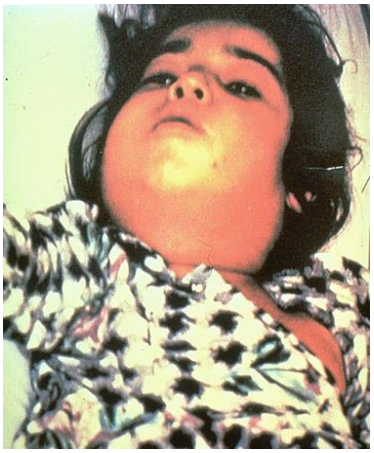
**** It occurs most commonly in the URT (upper respiratory tract)**, may occurs rarely in conjunctiva or genital tract or complicates wounds, abrasion or disease of skin (chronic lesion) or those who misuse alcohol.

**** Sore throat is frequently the presenting feature.**

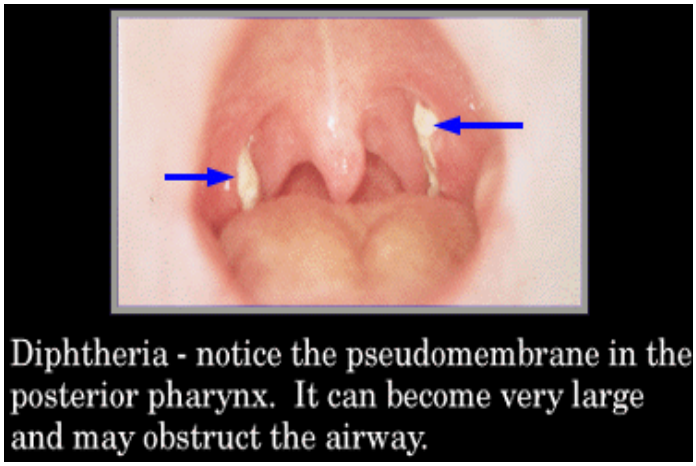
****Transmission by <u>droplets</u> from cases or carriers.**

**The organism remains localized at the site of infection & serious consequence result from the absorption of a soluble exotoxin which damage heart muscle & nervous system.

****<u>Incubation period; 2-4 days.</u>**



Child with Bullneck **Diphtheria**



Dipht.Medical Look

Coryne bacterium **diphtheria**

<u>Clinical features</u>

1-Infection begins insidiously.

2-<u>Tachycardia</u>(marked) & fever (seldom significant).

3- <u>Membranous tonsillitis</u>; Wash- leather elevated grayish- green membrane, with well-defined edge, firm & adherent & surrounded by a zone of inflammation on the tonsils(**diagnostic feature**). In mildest infection, membrane may never appear.

4-<u>Neck swelling (Bull neck) & tender enlarged lymph nodes.</u>

5-Nasal diphtheria; Nasal discharge & frequently blood- stained .

6-Laryngeal diphtheria; Husky voice, high pitched cough, may need urgent tracheaostomy.

7-Nasopharynx & uvula infection; gravely ill.

8-Skin, wound & conjunctiva infections are rare.

9-Death from <u>acute circulatory failure</u> may occur within the 1st 10 days.

Complications

1-Laryngeal obstruction or paralysis.

2-Cardiac complications; Myocarditis with arrhythmias or cardiac failure (25%).

3-Neurological complication; Palatal on about 10th day post-tonsillar or pharyngeal diphtheria(75%), paralysis of accommodation(difficulty in reading small print), generalized polyneuritis (weakness & parasthesia in limbs).

Management

1-Notification to the public health authorities and patient sent urgently to a hospital for infectious disease for isolation and treatment.

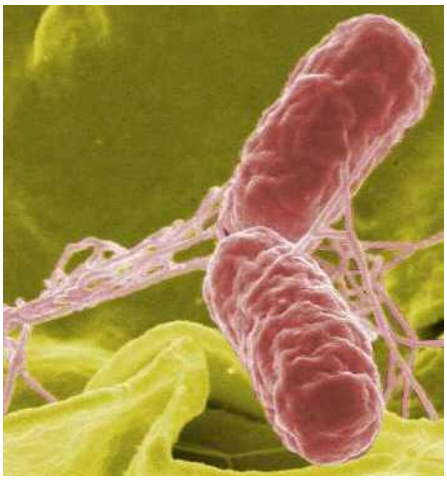
2-Treatment should begin once appropriate swabs have been taken before waiting the result, which includes;

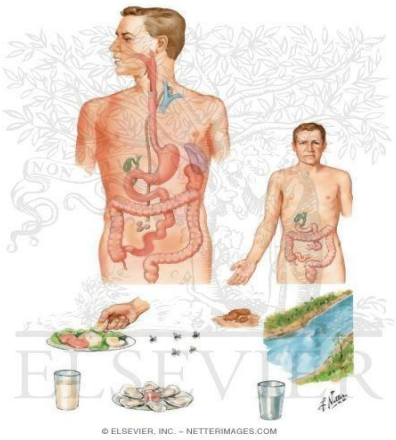
- **a-** **Administration of diphtheria antitoxin**(4000-32000 units i.m.-test dose first). Side effect anaphylactic reaction shock(prepare adrenalin 0.5-1 ml of 1/1000 solution i.m. & antihistamine) so small test injection is advised 1/2 hour before a full dose in every patient.
- **b-** **Administration of antibiotic**, penicillin 1200mg 6-hourly i.v. or amoxicillin 500mg i.v. 8-hourly for 2 weeks if allergy give erythromycin.
- **c-** **Treat complications**; like **tracheostomy** for respiratory obstruction).
- **d-** **All sufferers should immunize with diphtheria toxoid following recovery.**

Prevention

1. Active immunization should be given to all children.
2. Immunization or given booster dose of toxoid are required every 10 years to maintain immunity.
3. Protect close contacts; Erythromycin prophylaxis & booster dose of toxoid or immunization are given.

Typhoid & paratyphoid fevers





Aetiology

Salmonella typhi & S. paratyphi A&B.

****Transmission; fecal-oral rout.**

****Incubation period (I.P.); 10-14 days ,** but paratyphoid is somewhat shorter.

Pathology

After a few days of bacteraemia, the bacilli localize mainly in lymphoid tissues of small intestine (peyerُ s patches & follicles), which swell at first → ulcerate →heal, but during this sequence they may perforate or bleed.

Clinical features of typhoid fever

A-1st week

1-Fever; Stepladder fashion for 4-5 days.

2-Malaise & increasing headache, drowsiness,& aching limb(Myalgia).

3-Constipation (diarrhea & vomiting in children).

4-Relative bradycardia.

B- End of 1st week & 2nd week

1-Skin rash; Rose red spots on trunk, fade on pressure.

2-Cough & epistaxis.

3-Splenomegaly.

4-Abdominal distension, tenderness & diarrhea

(In severe diarrhea → HIV + Typhoid).

C-End of 2nd week & 3rd week

1-Bronchitis, delirium, then coma & death (due to toxaemia) but the disease is modified by antibiotic

2-Following recovery up to 5% of patients→ Chronic carriers & such patients have gall bladder disease(bacilli may live in G.B. of carriers for months or years & intermittently pass in the stool & less commonly in urine).

Complications

1- **Bowel; perforation & haemorrhage.**
2- Septicaemic foci; Bone & joint infection, meningitis, cholecystitis.
3- Toxic phenomena; Myocarditis & nephritis.

Investigations

1- **Leucopenia.**
2- **Blood culture (diagnostic method).**
3- **Stool examination; At 2nd & 3rd week feces contain the organism.**
4- **Widal reaction test; Not a reliable test**(detect antibody to the organism).

Management

A- Treatment for 14 days

1- **Fluoroquinolone(Drug of choice) e.g. Ciprofloxacin 500mg 12-hourly.**
2- **Azothromycin 500mg once daily in case of fluoroquinolone resistance.**
3- **Ceftriaxone & Cefotaxime.**
4- **Chloramphenicol 500mg 6- hourly.**
5- Ampicillin 750mg 6-hourly.
6- **Co-trimoxazole** 2 tablets 12-hourly.

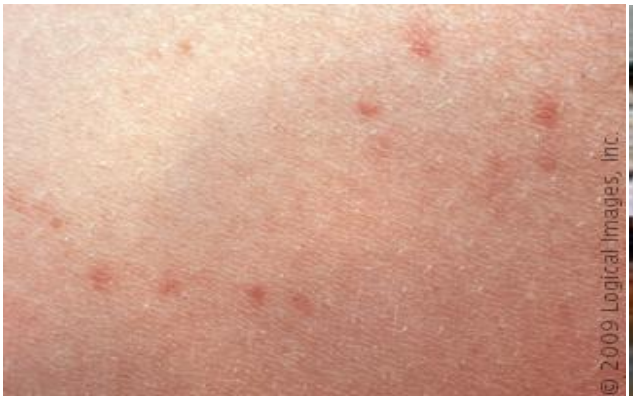
B- In chronic carrier the treatment for 4 weeks with Ciprofloxacine & cholecystectomy may be necessary in some patients.

Prevention

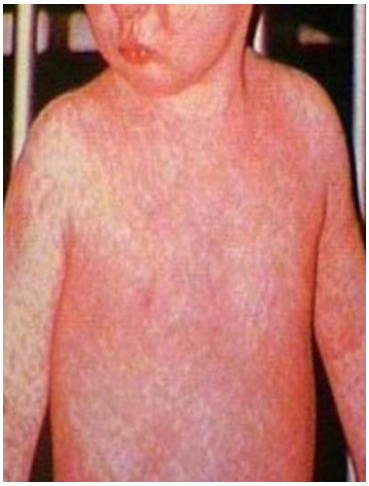
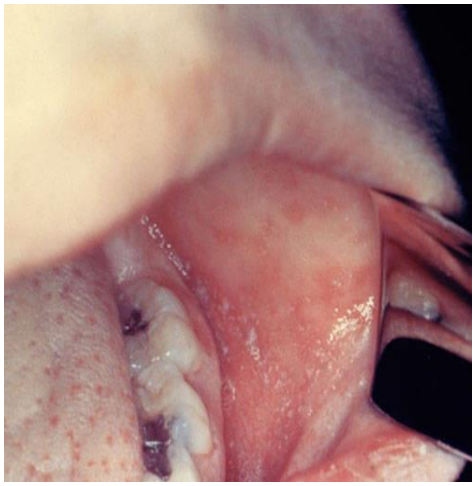
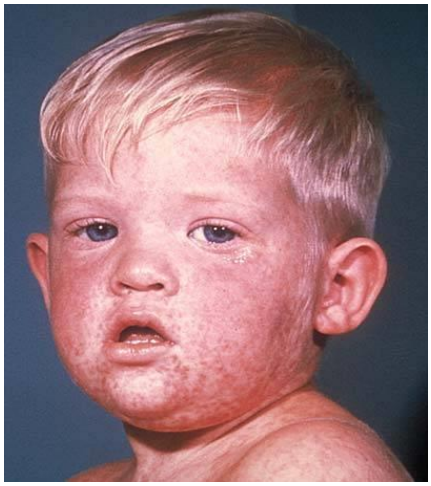
1- **Improved sanitation & living condition**→↓incidence of typhoid.
2- **Travellers to countries with endemic enteric infection, must inoculated with one of 3 typhoid vaccines** (2 inactivated injectable & one oral live attenuated).

Paratyphoid fever

1- **The course tend to be shorter & milder than that of typhoid fever & onset is often more abrupt with acute enteritis.**
2- **The rash may be more abundant & intestinal complications less frequent.**



Measles

**This paramyxo virus infection is endemic world-wide.

****It occurred in almost 100% of children.**

****Maternal antibody gives protection for the first 6 months of life.**

****Natural illness produces life-long immunity**, With live attenuated vaccine the condition is potentially almostly controlled.

****Transmission by droplet.**

****Incubation period ; 14 days to onset of rash.**

**Measles is a serious disease in malnourished, vitamine deficient or immunocompromised patients.

Mortality rate more in developing than in developed countries, **death result from bacterial super infection such as pneumonia, diarrhea disease.

Clinical features

1-Prodromal stage (Catarrhal)1-3 days before rash appearance;

- **a- Upper respiratory symptoms (fever, running nose, cough).**
- **b- Conjunctivitis(red, watery eyes, photophobia).**
- **c- Koplik's spots** (White spots surrounded by erythemia) **on buccal mucosa is a pathognomonic of measles.**
- **d- The patient mesirable & irritable**(due to viraemia).

2-Exanthematous stage; Rash appearance (due to natural antibody development)
lasting 5-6 days & gradually fading with staining in the pale skinned.

3-Generalized lymphadenopathy & diarrhea are common, bacterial pneumonia 4%, convulsion 1% of cases.

Management

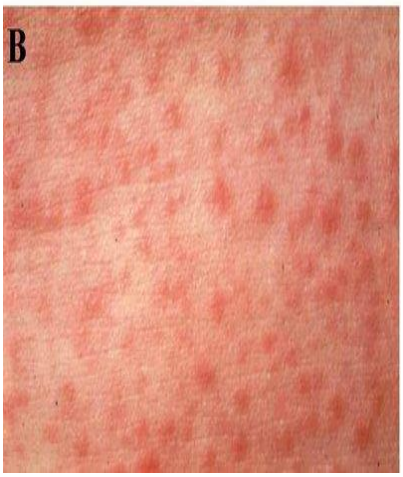
1- **Normal immunoglobulin** (I.M.) attenuate the disease in immunocompromised or in non immune pregnant women.
2- **The patient should be isolated if possible and excluded from school for 10 days from appearance of rash.**
3- **Vaccine can be used in outbreaks & vitamin A** may improve the outlook of uncomplicated disease.
4- **Antibiotic therapy only in super infection**.

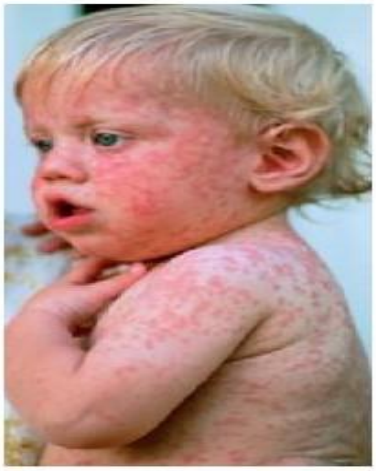
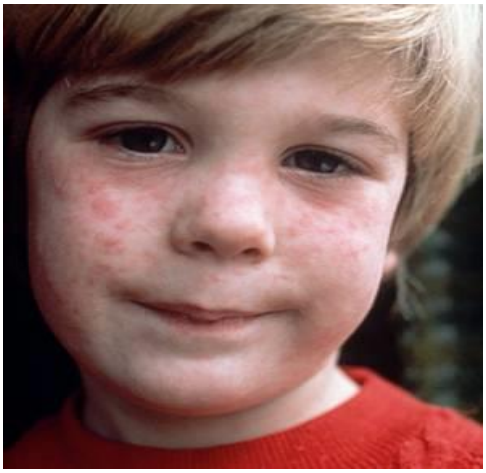
Prevention

1-Active immunization; MMR (measles, rubella, mumps) vaccine received in **two doses**; shortly after their first birthday prior to school entry.

2-Passive immunization; **Human normal immunoglobulin** I.M. is given to contacts under 18 months of age & non-immune debilitated children.

German measles(Rubella)



**Rubella(caused by toga virus) is endemic disease, & outbreak occurs in springs & early summer with epidemic every 7-10 years.

****Transmission ; by droplet** , **one attack confers a high degree of immunity**.

****Infectivity from up to 1 week before & 1week after the onset of rash.**

** Affect older children, adolescent & young adult.

****In children most cases are subclinical.**

Pathology

Initial infection via the upper respiratory tract & local lymph node is followed by viraemia to target organ such as skin, joint & placenta. If placenta infection take place in the 1st trimester, persistence of virus →severe congenital diseases.

Rubella & fetus

--Risk of congenital abnormality; 1st 4 weeks of pregnancy →80%.
16th week of pregnancy →less than 5%.
--Causes of congenital abnormalities; **Heart**(septa defect), **eye**(cataract),
brain(mental retardation), **ear**(deafness').

Clinical features

1-Lymphadenopathy, lasting several weeks (post auricle, post-cervical, suboccipital) & occasionally **splenomegaly.**

2-Maculopapular rash, start simultaneously on face & move to trunk.

3-Petechial lesion (Forchheimer spots), appears **on soft palate** associated with **mild coryza, conjunctivitis, fever occasionally on 1st day of rash.**

Complications

1-Arthritis(polyarthritis)& arthralgia 30% of female(fingers, wrist & knee joints).
2-Congenital abnormalities.
3-Encephalomylitis.
** Complete recovery from all of these complications is the rule.

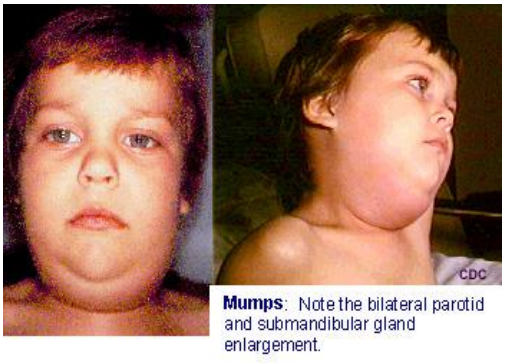
Diagnosis

Serological tests(rubella-specific IgG with absent IgM indicate previous infection, While specific IgG or rising IgM indicate recent infection, detect of maternal rubella specific IgG in early pregnancy indicate established immunity & allows the patient to be reassured that there is no serious risk of congenital disease) are necessary for definitive diagnosis.

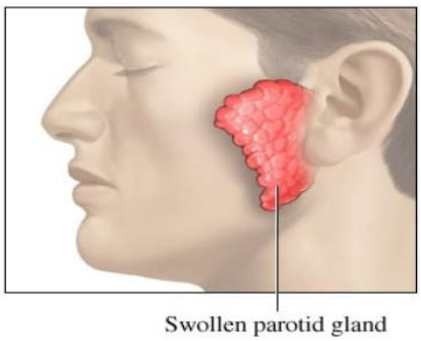
Management; **No treatment is available**

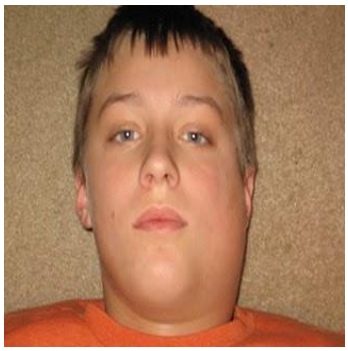
Prevention; **Give MMR vaccine like in measles**

Mumps



Mumps: Note the bilateral parotid and submandibular gland enlargement.


Swollen parotid gland



This paramyxovirus is endemic world-wide .

Primary infecting 5-9 years old children, may affect older teenagers.

****Transmission by <u>droplets or direct salivary spread</u> & initial infection is through the upper respiratory tract.**

****Incubation period ;<u>16-18 days</u> ends with 2-3 days of infectivity & a 2nd viraemia**.

Clinical features

1- **<u>Prodromal stage</u> of pyrexia & malaise.**
2- **<u>Classical tender parotid enlargement (75% bilateral)</u>**, other salivary glands are involved in approximately 10% of cases.

Complications

1- **<u>Oophoritis(5%) & orchitis (35%</u>** <u>& 33% of these cases bilateral)</u>, only occure post-pubertal, There is some degree of testicular atrophy but sterility is most unlikely .
2- **<u>Meningitis 50% of cases.</u>**
3- **Encephalitis**; acut & post-infectious.
4- **Transient hearing loss & labyrinthitis.**
5- **<u>Abortion</u> may occur if infection takes place in the 1st trimester of pregnancy.**

Diagnosis

Most cases diagnosed:

***<u>On clinical grounds</u>**, but can be confirmed by

***<u>Demonstration of specific antibodies</u>**, alternatively,

***<u>The virus may be cultured from the saliva or from CSF in meningitis</u>**.

Differential diagnosis

1-Salivary stone(unilateral).
2-Sarcoidosis(cause bilateral chronic parotitis).

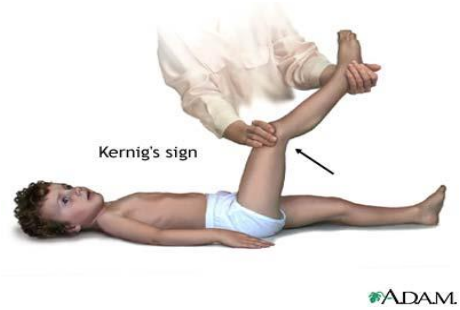
Management

1- **Symptomatic relief is important.**
2- **Prednisolon up to 40 mg/day orally for 4 days to relieve discomfort of orchitis**

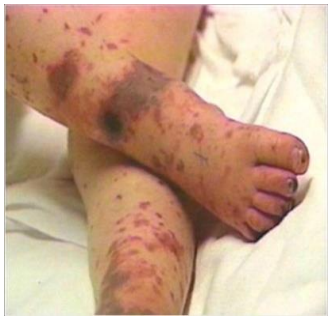
<u>Prevention</u>; Give MMR vaccine like in measles.

Meningitis

Meningococcal **Meningitis**



Kernig's sign of **meningitis**



Most cases of **meningitis**

An acute infection of the meninges presents with a characteristic combination of;

1-Pyrexia

2-Headache

3-Meningism;

-**Neck stiffness.**

-Kernig's sign(With hip joint flexed, extension at knee causes spasm in the hamstring muscles) .

- **Brudzinski's sign**(passive flexion of the neck causes flexion of the thighs &knee).

The severity of these features varies somewhat according to the causative organism.

Aetiology

1- Infective

- **a- Bacteria**; Brucella.
- **b- Viruses**; Enteroviruses, mumps, influenza, HIV etc.
- **c- Protozoa & parasites**; Toxoplasma, amoeba, etc .
- **d- Fungi;** Candida, Cryptococcus-neoformans..

2-Non-infective(sterile)

- **a- Malignant disease**; Breast cancer, bronchial ca., lymphoma…
- **b- Inflammatory diseases**; SLE, Behcet's disease, sarcoidosis..

Viral meningitis

The most common cause of meningitis , it is a benign & self-limiting illness & requiring no specific therapy.

Clinical features

Mainly occurs in children or young adults with acute onset

1. **More severe headache, irritability & rapid development of meningism.**
2. **May be high pyrexia, but focal neurological signs rare.**

Investigations

CSF(Cerebrospinal fluid) **examination**;

(Done in patient has not received antibiotic); ↑↑ **lymphocytes, normal glucose & protein.**

Management

No specific treatment, just symptomatically treated in a quiet environment.

Pyogenic bacterial meningitis

It is usually secondary to a bacteraemic illness, although it may result from direct spread from adjacent focus of infection in ear, skull fracture or sinus.

Aetiology

Streptococcus pneumonia, meningococcus (Neisseria meningitides), H. influenza….

The organism invades through the nasopharynx, producing septicaemia which lead to Pyogenic meningitis. In pneumococcal & H.influenzae infection there may be associated otitis media & pneumonia.

Clinical features

1-Headache, drowsiness, fever & neck stiffness is usual features.

2-In severe condition there may be comatose & focal neurological signs.

3-Purpuric rash 70% in meningococcal meningitis.

4-Circulatory collapse in septicaemia due to endotoxin &or cytotoxine release.

Investigations

1-CSF examination;--Cloudy (turbid) due to ↑↑neutrophils >10 billion cells/l.

--Significant ↑Protein &↓glucose.

--Gram film & culture to identify the organism.

2-Blood culture may be +ve.

3-PCR (polymerase chain reaction) techniques to identify bacterial DNA in blood & CSF.

4-CT-scan to exclude mass lesion, it is briefer done before lumbar puncture because of risk of coning(for patients with drowsiness, neurol. signs seizures).

Management

1-Empirical antibiotic therapy before waiting the results, then modified.

2-Benzyl penicillin i.v. immediately for meningococcal & hospital admission.

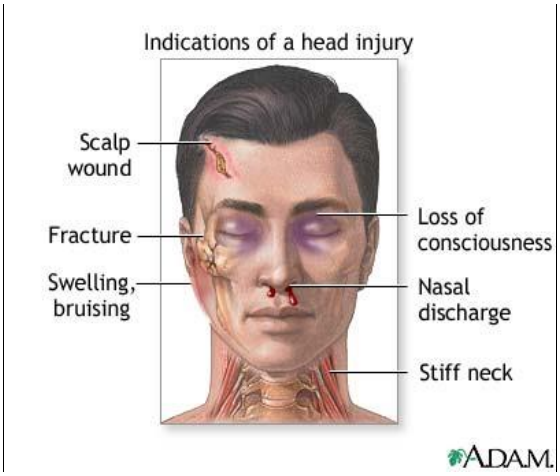
3-Adjunctive corticosteroid therapy is useful in children & adults.

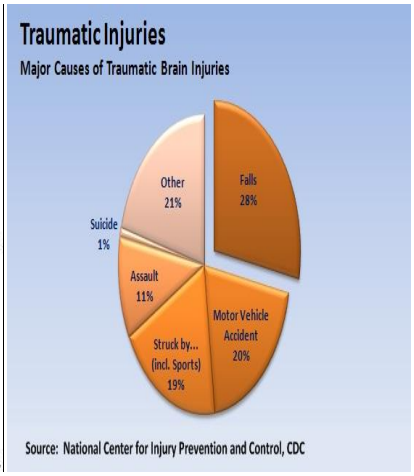
Prevention of meningococcal infection

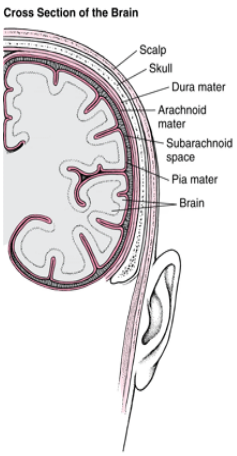
1-Houshold & other close contacts especially children should be given 2 days of rifampicin 5-10 mg/kg 12-hourly, & 600 mg 12-hourly for adults or ciprofloxacine 500mg single dose is alternative.

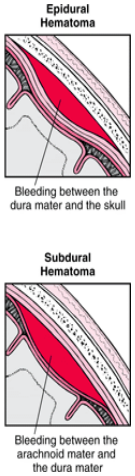
2-Vaccines are available for prevention of disease caused by meningococci of groups A & C only, for B is not available which is the most common serogroup .

Head injury









1- **<u>Scalp trauma</u>**; the wound is dealt with after shaving around the wound.
2- **<u>Cranium; Skull fracture</u>** (vault or base) mostly accompanying scalp wound.
It is either **linear or depressed** fracture (**may need elevation**).
3- **Extradural haematoma; Mostly associated with skull fracture** especially of temporal & parietal bones. The anterior or posterior branches of middle meningeal artery are injured→ bleeding & hematoma.

<u>Clinical features</u>

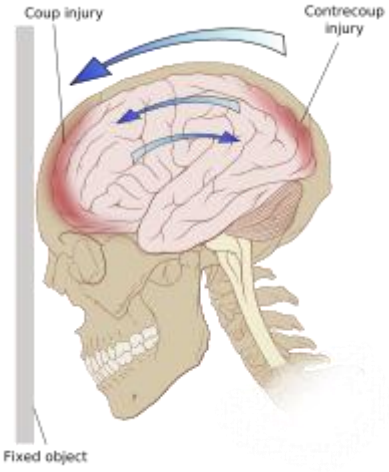
A- <u>Concussion</u>: Short initial period of loss of consciousness.
B- <u>Lucid interval</u>: The patient recovers but hematoma is getting more, accompanied by headache, drowsiness & tiredness.
C- <u>Change of level of consciousness</u>:
1- Confusion.
2- Irritability.
3- Drowsiness.
4- Contralateral hemiparesis due to direct pressure.
5- Pupil dilatation at same side of injury.
4- Subdural hematoma: Due to severe trauma & brain damage
a- Mostly on lucid interval.
b- Early loss of consciousness.
c- The hematoma may be at same side or on opposite side.
d- Hematoma is extensive & localized .

Diagnosis

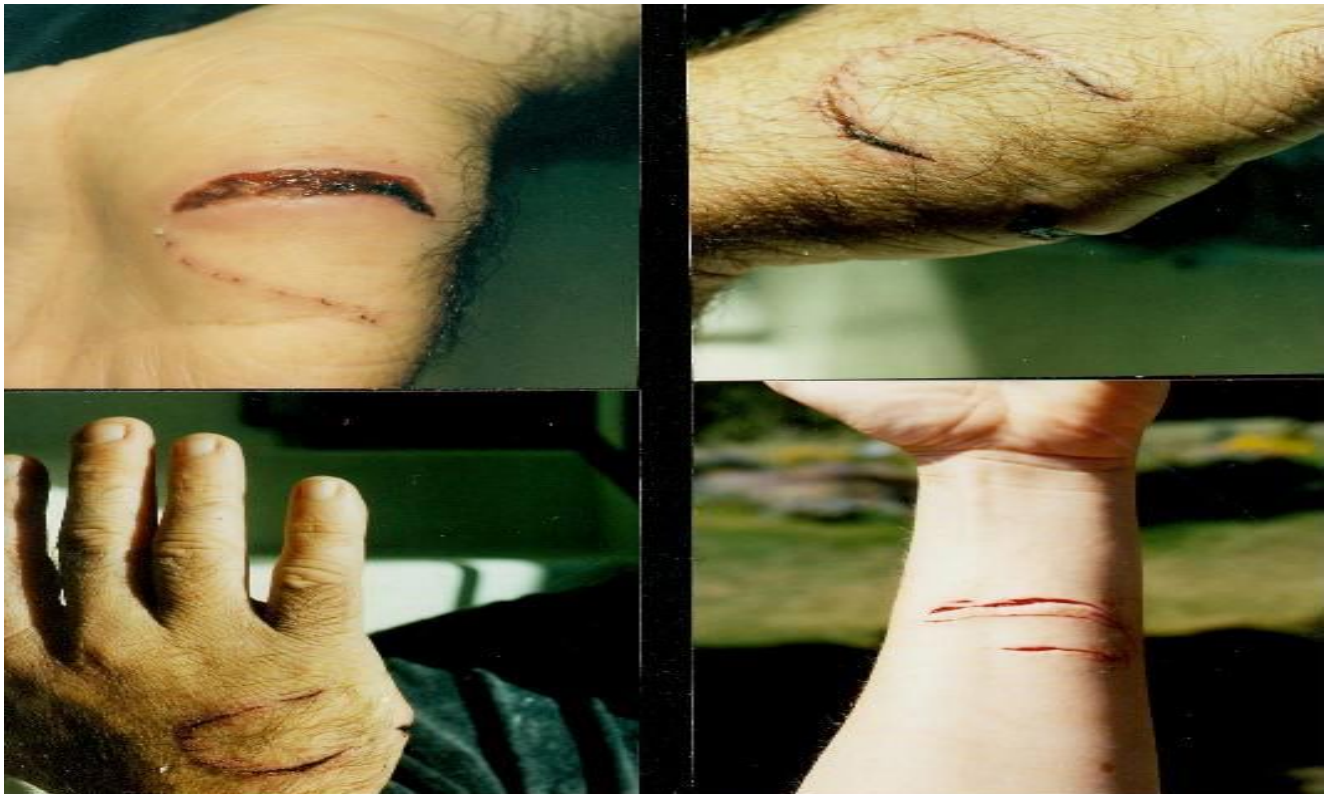
1- **Radiography.**
2- **Clinical examination.**
   a- **State of consciousness.**
   b- **Observe any wound in skull.**
   c- **State of pupil.**
   d- **Any paresis or plagia.**
   e- **History of injury & any bleeding from nose or ear mixed with CS (here the blood not clot rapidly if dropped on a sheet it leaves double rings).**
3- **CT scan.**

Management

1- **Clear the airway & make it free.**
2- **Stop bleeding & replace blood loss.**
3- **Treat intra-abdominal & intra-thoracic lesion firstly.**
4- **Assessment of level of consciousness, state of pupil , fits & recording vital signs repeatedly.**
5- **Signs of increasing intracranial pressure; slow pulse, high blood pressure & slow respiration.**



Wounds



1- **Incised wounds**: Caused by sharp knives or glasses(tendon, nerves, & arteries repaired).
2- **Penetrating w.**: Caused by sharp tips or dagger (Viscera are explored & dealt with).
3- **Perforating w.**: Gunshot (Viscera are explored & dealt with).
4- **Lacerated w.**: (need wound excision), delayed repaired of tendons & nerves because of infection. (due to traffic accidents).
5- **Crushed w.**: Due to severe road traffic accidents, industrial or car accidents (No closure of wound by primary intension).
6- **Blast injury (bombs).**

<u>Management of wound and injuries</u>

1- **Clear the airways.**
2- **Stop haemorrhage.**
3- **I.V. fluid.**
4- **Oxygenation administration.**
5- **Splint for fracture.**
6- **Clean wound & remove foreign bodies.**
7- Suture the wound if it is clean. During first 6 hours of injury, remove the dead tissues. Wound of chest, abdomen, & head should be explored.

Wound suturing;

a- Primary suture; if wound is clean &no loss of tissue during first 6 hours.
b- Delayed primary suture; 4-6 days after injury when oedema subsides & infection is controlled.
c- Secondary suture; 4-6 weeks after injury.
d- During suturing the wound, all cut tendons, nerves & arteries should be repaired.
e- If there is loss of skin, and suturing is impossible grafting is needed whether primary or delayed primary or secondary grafting.
f- The suture should not be tense otherwise blood supply will be impaired & leads to tissue necrosis.
8- Give antibiotics.
9- Antisera or vaccines against gas gangrene and tetanus in severe injuries and against rabies in case of dog bite.

Peptic ulcer (P.U.)

It refers to an ulcer in :

* **Lower oesophagus**

***Stomach or duodenum** (duodenal ulcer is more common)

***Jejunum** (after surgical anastomosis to stomach)

***Rarely in ileum** (adjacent to a meckel's diverticulum)

- Ulcer may be acute or chronic : in both penetrate muscularis mucosa .

- Ulcer also means necrosis but erosion do not penetrates muscularis mucosa.

Incidence and aetiology :

- The prevalence of P.U. is decreasing in many western communities as a result of wide spread use of helicobacter pylori as a result of widespread use of Helicobacter pylori eradication therapy but it remains high in developing countries .

- Male to female ratio for D.U. varies from 5:1 to 2:1 whilst that for G.U. is 2:1 or less .

- The main causes :

1- Helicobacter pylori infection : In general population raises steadily with age .

- Around 90% of D.U. patients & 70% of G.U. patients are infected with H.pylori . The remaining 30% of G.U. are to NSAIDs

- H-pylori spread by person to person contact via gastric refluxate or vomit .

- H-pylori acts by reducing gastric mucosal resistance to attack from acid & pepsin . Approximately 1% it causes a pan gastritis leading to gastric atrophy **&** hypochlorhydria which allows bacteria to proliferate with in the stomach , these may produce mutagenic nitrates from dietary nitrates , predisposing to development of G.cancer.

- Urea bath tests are best diagnostic test for H.pylori, also H.pylori can be tested by serology kit , faecal antigen test , endoscopy **&** histology of culture .

2- NSAIDs : (non – steroidal anti-inflammatory drugs) like : aspirin ,voltarin , ect :-

3- Smoking : more G.U less to D.U.

- it lead to more complicated ulcer **&** less likely to heal .

4- Hereditary (especially for D.U.)

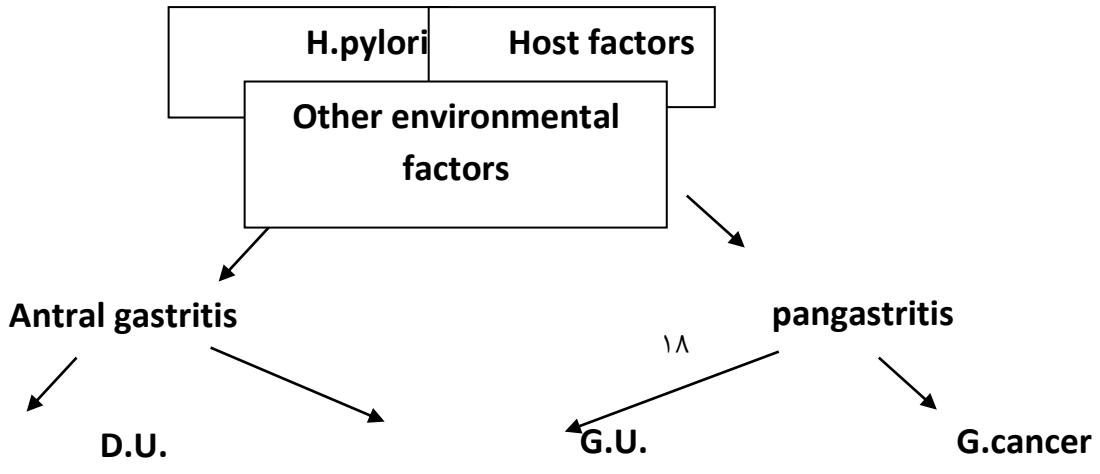
5- Psychological factor (stress) –> increase gastric acid secretion .

--- P.U. forms when there is an imblance between;

1- Aggressive factors : i.e. digestive power of acid**&** pepsin.

2- Defense factors : i.e. mucosal resistance (gastric mucosal barrier)

Ulcer only occur in presence of acid & pepsin so never occur in Achlorhydric patient (Pernicious anaemia) while more common in case of gastric hypersecretion (Zollinger-Ellison syndrome).



****Chronic G.U. : is usually single , 90% are situated on lesser curve** with in antrum or junction between body **&** antral mucosa .

****Chronic D.U. : is usually occurs in first part of duodenum & 50% on anterior wall .**

****G.U. & D.U. : coexist in 10% of patient** & more than one P.U. is found in 10-15% of patient .

Clinical features

P.U. is chronic condition with natural history of spontaneous relapse & remission lasting for decades , if not for life.

1- Recurrent abdominal pain :

It has 3 notable characteritics :

a- Localization : Epigastrium "pointing sign"

b- Episodic occurance : Last for 1-3 wks a time & 3-4 time a year .

c- Food relationship :

- Night pain : Wakes patient from sleep & relived by food eating .

- Day pain : (hunger pain) : stomach empty & relived by food eating

- Pain my be :

- discomfort in epigastrium .
- Radiate to back .

- **Heart burn** .

- associated with salivation **"water brush"** .

- Absent "silent ulcer" in elderly people .

2- Dyspepsia , loss of weight , loss of appetite (anorexia) , nausea , vomiting in 40% of patient if persistent daily suggest gastric out let obstruction .

3- Anaemia & melena " black stool" due to bleeding from ulcer & haemetamesis " vomiting blood or brown color vomitus "

4-Complicated ulcer → perforation " acute abdominal pain"

Diagnosis

Depend on investigation because diagnostic value of individual symptoms for P.U. is poor so the history is therefore a poor predictor of presence of P.U.

Investigation

1- Barium meal (double contrast barium meal) .

2- Endoscopy : is preferable & more accurate & biopsy can be taken especially in G.U. to exclude G. cancer .

Complications

1- Haemorrhage .

2- Perforation .

3- Caner of stomach .

4- Pyloric out let obstruction .

Management

The aim :

1- To relieve symptomes .

2- To induce ulcer healing .

3- To prevent relapse as long as possible, the ulcer heals within

(4-6 weeks) of treatment .

General measures

1- No smoking .

2- No NSAIDs as possible .

3- No alcohol .

4- Light & frequent meals .

Short term management of P.U. :

1- Antacids & alginates : aluminium hydroxide … ect .

2- H_2-receptor antagonist : ranitdine , cimetidin , nizatidin , famotidin ..ect .

3- Proton pump inhibtor (P.P.I) : omeprazol , rabeprazol , pantoprazol , lansoprazol , esomeprazol ..

4- Chelates : tripotassium , dicitra bismuthate .

5- Complex salt : sucralfate .

6- Prostaglandin analogues : "misoprostol"

H.pylori eradication is the corner stone of therapy for P.U. as this will successfully prevent relapse and eliminate the need for long therapy in the majority of patients :

1St line therapy for H.pylori eradication :

* P.P.I (12hrly) + clarithromycin (500 mg 12 hourly)

+ amoxicillin (1gm, 12 hourly) or metronidazol " flagyl" (400 mg, 12 hrly) For 7 days.

2nd line therapy : (quadruple therapy)

* P.P.I. (12hrly) + bismuth (120mg, 6hrly) + metronidazol (flagyi 400mg 12 hrly) + tetracyclin 500 mg (6 hrly) For 7 days.

Maintenance treatment : may be required for monitoring of patient by giving the lowest effective dose to prevent relapses & complications or if there is risk to do operation .This done by acid suppressing drugs.

Surgical treatment

Most ulcer cure by H.pylori eradication & potent acid suppressing drugs . but some need operation :

emergency : in case of perforation or haemorrhage elective in case of **" indications" :**

1- No healing of ulcer by drugs .

2- Relapse even with maintenance dose treatment .

3- Occurring of complication "gastric out obstruction " .

4- When patient cannot continue medical treatment .

5- Post surgical recurrence .

Surgical operation for G.U. :

*** partial gastrectomy with billroth 1anastomosis** " the ulcer itself & the ulcer bearing area of stomach are resected "

Surgical operation for D.U. :

*** vagotomy** "to reduce acid-secretory capacity of stomach "

With a drainage operation such as pyloroplasty or gastroenterostomy in order to overcome the retards of gastric emptying .

*** highly selective vagotomy without a drainage** " denervates only the acid-producing area of stomach & so no need to drainages .

Acute Appendicitis

Vermiform Appendix is a blind muscular tube with mucosal, submucosal, muscularis and serosal layers.

-Its location

-retrocaecal in distal end of large caecum (70%)

-pelvic 21%

-paracaecal 2% preileal & postileal 1.5%

Acute Appendicitis is of two types :

1-obstructive

2-non obstructive

Pathophysiology:

In many cases of early appendicitis the appendicular

lumen is patent despite the presence of mucosal

inflammation & lymphoid hyperplasia .In other cases lymphoid hyperplasia lead to narrowing the lumen and then obstruction with excessive mucus secretion &Inflammated exudate lead to increase intraluminal pressure which in turn lead to obstruct lymphatic drainage ,in this stage resolution may occur ,other wise further oedema &distention of appendix causing venous obstruction & ischemia of appendicular wall which lead to acute appendicitis & progressed to ischemic necrosis then gangrenous appendicitis which ultimately lead to free contamination of peritoneal cavity forming phlegmonous mass or paracaecal abscess.

Aetiology & incidence :

1- No unifying hypothesis regarding the aetiology but **decreased dietary fiber** and **increased consumption of refined carbohydrate** may be important so appendicitis more in highly civilized people than low classes "poor people".
2- More common in America than Asia & Africa .
3- Male more than female 3:2 at age of 25 but before puberty is equal 1:1 & peak incidence in teens & early 20years but
rare in infant & common in children.

4- Family susceptibility :
One third of children with acute appendicitis have

first degree relative with similar history.

5- Obstruction of appendix by :
faecolith , tumor , worms (pin worms) , foreign body

& stricture.

6- Infection a mixed growth of aerobic & anaerobic organism.

7-Abuse of purgatives.

Clinical features

1- Periumbilical pain

- Acute onset

- Colicky or may be vague (mid-gut visceral pain) then after a few hour- localized to right iliac fossa with more intensive (somatic pain)at Macburney's point (due to irritation of parietal peritoneum of right iliac fossa) - increasing during coughing or movement.

2- Anorexia (loss of appetite) , nausea & 1-2 episodes of vomiting also constipation

3- fever: during first 6 hour rarely increase of temperature but after that slightly rises (37.2 - 37.7C) if increased above 38.5C suspect another disease especially in children.

The classic visceral-somatic sequence of pain occur in half of patient , other may be atypical (i.e. somatic or visceral) especially in elderly patient .

Clinical signs:

1- pyrexia (fever)

2- localized tenderness in right iliac fossa, muscular guarding & rebound tenderness & if progressed rigidity occur (due to peritonitis)

Signs for elicit in appendicitis

1 - Pointing sign : Rebound tenderness at Mcburney's point.

2 - Rovsing sign : Deep palpation of left iliac fossa → pain in

right iliac fossa.

3 - Psoas sign : Right hip flexed to relieve pain.

4 – Obturator sign : pain in hypogastric region when you flex hip join with internal rotation.

****Obesity can obscure and diminish all local signs of acute appendicitis and also pregnancy so lead to delay diagnosis.**

Diagnosis:

1- **The diagnosis of acute appendicitis is essentially clinical** (more on clinical examination of abdomen than history or laboratory investigation)
2- **A-routine investigation** : -**full blood count, - Urinalysis**
B- Selective investigation:

- Pregnancy test.
- Urea and electrolytes.
- Supin abdominal radiography (K.U.B).
- Ultra sound & CT-scan of abdomen and pe

The Alvarado (maistrels) scores:

Symptoms:	- Migratory right iliac fossa pain	**1**
	- Anorexia	**1**
	- Nausea and vomiting	**1**
Signs	- Tenderness in right iliac fossa	**2**
	- Rebound tenderness	**1**
	- Elevated temperature	**1**
Laboratory*:*	- Leucocytosis	**2**
	- Shift to left	**1**

Total ***10***

****7 and above are strongly predictive of acute appendicitis**

Differential Diagnosis (D.D.)

In children :

1- **Gastro enteritis** .
2- **Mesenteric lymphadenitis** (following acute tonsillitis).
3- Meckel's diverticulitis
4- Intussusceptions :before 2 year old.
5- Henoch – schonlein purpura : preceded by sore throat or respiratory infection.
6- Lobar pneumonia and pleurisy: ChestX-Rayand pleural friction.

In adult male*:*

1- **Terminal ileitis (regional ileitis)** diarrhea and loss weight.
2- **Ureteric colick** : urinalysis and K.U.B .
3- Perforated P.U.
4- **Torsion of testis** : examine scrotum
5- Acute pancreatitis : serum amylase.
6- Rectus sheath haematoma : occur after strenuous exercise .

In adult female*:*

1- **Pelvic inflammatory diseases**:vaginal discharge and menstrual cycle.
2- **Pyelonephritis.**
3- **Ectopic pregnancy.**
4- Torsion and rupture of ovarian cyst.
5- Endometriosis.

In Elderly:

1- Sigmoid diverticulitis.
2- **Intestinal obstruction. 3- Colonic carcinoma**

Complications

per-operative complication:

1- perforation : risk factors :
- Extreme age.
- Immunosupperssion patient.
- D.M.
- Faecolith obstruction.
- Pelvic appendix position.
- Previous abdominal surgery.

2- Gangrene and appendicular abscess (need drainage).

3- Peritonitis → paralytic ileus.

4- Fistula.

5- Appendicular mass : need conservative treatment then appendicectomy after 3 month and use ultra sound for its diagnosis.

Post operation complication:

-Early: 1- Paralytic ileus.
2- Wound sepsis 5-10% .
3- Intra-abdominal abscess, fistula.
4- chest infection.
5- Venous thrombosis and embolism.

-Late: 1**-** Adhesive intestinal obstruction.

2- Incisional hernia. 3- Female sterility from frozen pelvis.

Management :

Pre operative:

1- **I.V.fluid** to establish adequate urine out put.
2- **Single dose of antibiotic** : to prevent post operation wound infection and sometimes need I.V. antibiotic for peritonitis
3- **Aspirin (analgesia)** for reliving fever in children.

Operation:

a- conventional appendicectomy : to prevent ↑ morbidity and mortality of peritonitis .

b- Laparoscopic appendicectomy .

Incision used in operation :

1- McBurney's grid iron incision.
2- Rutherford-morisons incision.
3- Right lower Para median incision.
4- Transverse or skin crease incision (Lanz).

Intestinal obstruction (I.O.)

There are 2 types of I.O.:

1- Dynamic (mechanical) : peristalsis working against mechanical obstruction like strangulation .

2- A dynamic (non-mechanical) : peristalsis may be absent as paralytic ileus or present in a non-propulsive form as pseudo-obstruction .

Pathophysiology :

In dynamic obstruction the proximal bowel dilates & develops an altered motility , below obstruction, bowel exhibit normal peristalsis & absorption until it becomes empty where contracts & becomes immobile .

Initially , proximal peristalsis is increased to overcome the obstruction , If obstruction is not relieved the bowel begin dilate causing reduction in peristaltic strength , Ultimately resulting in flaccidity & paralysis , this is a protection phenomenon to prevent vascular damage secondary to increased intralumenal pressure.

The distension proximal to an obstruction is produced by factors

1- Gases : which are increased significantly due to overgrowth of both aerobic & anaerobic organisms resulting gases production following O2 & CO2 reabsorption & nitrogen 90% & hydrogen sulphide .

2- fluid : is made up of the various digestive juices following obstruction fluid will accumulated within bowel wall & any excess is secreted into lumen while absorption from gut is retarded .

Dehydration & electrolyte are due to :

1- Reduced oral intake .

2- Defective intestinal absorption .

3- Vomiting .

4- Sequestration in the bowel lumen .

Causes of obstruction :

Dynamic I.O. :

1- Intraluminal :

- impaction .
- foreign body .
- bezoars (firm mass , undigested fruit or vegetable or hair)
- Worm (ascaris)
- Gall stone .

2- Intramural :

- Stricture (due to T.B. , Corhn's disease)
- Malignancy (Lymphoma)

3- Extramural :

- Bands (adhesions" following abdominal operation")(**common**)

- **Hernia .**
- **Volvulus .**
- **Intussusceptions .**

A dynamic I.O. :

- **Paralytic ileus** .
- Mesenteric vascular occlusion .
- Pseudo-obstruction .

Clinical features :

The clinical features or presentation vary according to :

1- Location of obstruction :

- Small intestine; (low & high) (acute type)

- Large intestine; (chronic type)

2- The age obstruction or time : **acute , sub acute , chronic , acute on chronic .**

3- The underlying pathology or causes .

4- The presence or absence of intestinal ischemia .

- Simple : blood supply is intact .

- Strangulation : blood supply is affected.

Clinical features of acute I.O. :

1- Abdominal pain :

- Sudden onset .

- Colicky in nature & intermittent .

- Centrally " umbilical" located if of small intestinal obstruction while lower abdomen if of large intestinal obstruction .

* In paralytic ileus → no pain .

2- Vomiting & dehydration : early in upper I.O. & late in lower I.O.

3- Abdominal distension : Early starts with **hyperborborygmi**

(increased bowel sounds) to overcome obstruction later on no sounds or reduced due to paralytic ileus .

4) Constipation :

It is of 2 types : **- Absolute (neither faeces or gases**

("complete I.O." are passed)

- Relative : (only gases are passed) .

(In complete I.O. = sub acute I.O) .

*** Distal I.O.:**

→ delayed vomiting , nausea & dehydration→ greater distension .

→ pain in lower abdomen & mild .

→ early constipation . and vice versa

Other delayed manifestation :

1- Oliguria .

2-↓K .

3- ↑ temperature .

4- Hypovolaemic shock & septicaemic shock (↓ temperature)

5- Abdominal tenderness → indicate ischemia .

Strangulated I.O. :

(The strangulated I.O. is a surgical emergency condition) .

- Strangulation is one type of I.O. when occurs , the viability of bowel is threatened secondary to a compromised blood supply

(venous & arterial blood supply) resulting into hemorrhagic infarction & markedly exposure to anaerobic organism with their toxin which transmitted locally & systemically "peritoneum & systemic blood vessels" .Onset of gangrene here is rapid .

The causes of strangulation are like that of extramural causes .

Strangulation either external (due to hernia ring)

Or internal " close – loop obstruction) due to carcinoma of colon. **Its clinical features are** :

1- Constant pain , severe , recur regularly & never completely absent.

2- Tenderness , rebound tenderness & finally rigidity .

3- Shock .

Diagnosis of I.O. depend on

1- Clinical features .

2- Radiological features :

- Gas shadow in erect posture .

- Fluid levels with gas above (step – ladder pattern) .

Differential Diagnosis :

1- **Acute gastroenteritis .**
2- Henoch – schoenlein purpura . 3- rectal prolapse .

Management

Surgical treatment is necessary for most cases of I.O. but should be delayed until resuscitation is complete provided there is no sign of strangulation or evidence of closed – loop obstruction .

1- Gastro duodenal suction pre & post operation .

2- I.V. fluid & electrolytes replacement .

3- Antibiotic .

4-Operation (-relief obstruction .- anastomosis .- colostomy).

Indication for early surgical intervention

1- external& internal strangulated I.O.

2- acute obstruction .

Gall Stones (Cholelithiasis)

* **G**all **S**tones :are the most common biliary pathology .

* 10-15% of adult population in U.S.A have **G.S .**

* Cholecystectomy one of the most common operation performed by general surgeons .

* **Types of Gall Stones (G.S) :**

1- Cholesterol : often solitary and large .
2- Mixed : multiple and facet
3- Pigment : **- Black** " common"
- Brown " rare "

***B**oth cholesterol **&** mixed stones are composed of 51-99% of pure cholesterol plus an admixture of calcium salt , bile acid , bile pigment **&** phospholipids while pigment stones are composed of insoluble bilirubin pigment **&** calcium phosphate **&** calcium bicarbonate .

***B**lack pigment stone , accompany haemolysis " hereditary spherocytosis , sickle cell anemia " **&** liver cirrhosis while brown stone , accompany bile stasis **&** infected bile **&** also associated with foreign body with in bile duct or parasites " ascariasis "

* Aetiology and incidence of G.S

* **I**n **U.S.A** and **Europe** : 80% are cholesterol or mixed G.S .

* **I**n **Asia** : 80% are pigment G.S .

***Classical sufferer (5F) (Fat , Fertile , Flatulent , Female of Fifty)**

* Cholesterol G.S causes

1- Increase cholesterol secretion : occurs in :-

- **O**besity (high colon diet) .
- **O**ld age .
- **F**emale gender.
- **R**apid weight loss .

2- Impaired gall bladder emptying : in case of :

- **P**regnancy .
- **G**all bladder stasis
- **F**asting
- **S**pinal cord injury

3- Decrease bile salt secretion : in case of **P**regnancy .

*** W**hen bile is supersaturated with cholesterol or bile acid concentration are low , unstable unilamellar phospholipid vesicles form , from which cholesterol crystal may nucleate **&** stones may form .

*** B**oth sexes and all ages may get gall stones .

* Clinical presentation

1- Asymptomatic (80% of patient)

2- Acute cholecystitic Feature .

3- Chronic cholecystitic feature .

4- Biliary colic : stone impacted in cystic duct (10-25% of patient)

Biliary colic

1- Abdominal pain : **severe right upper quadrant pain** or epigastric pain may last few minutes to several hours .

- may radiate to chest or interscapular .

- may start during night waking the patient .

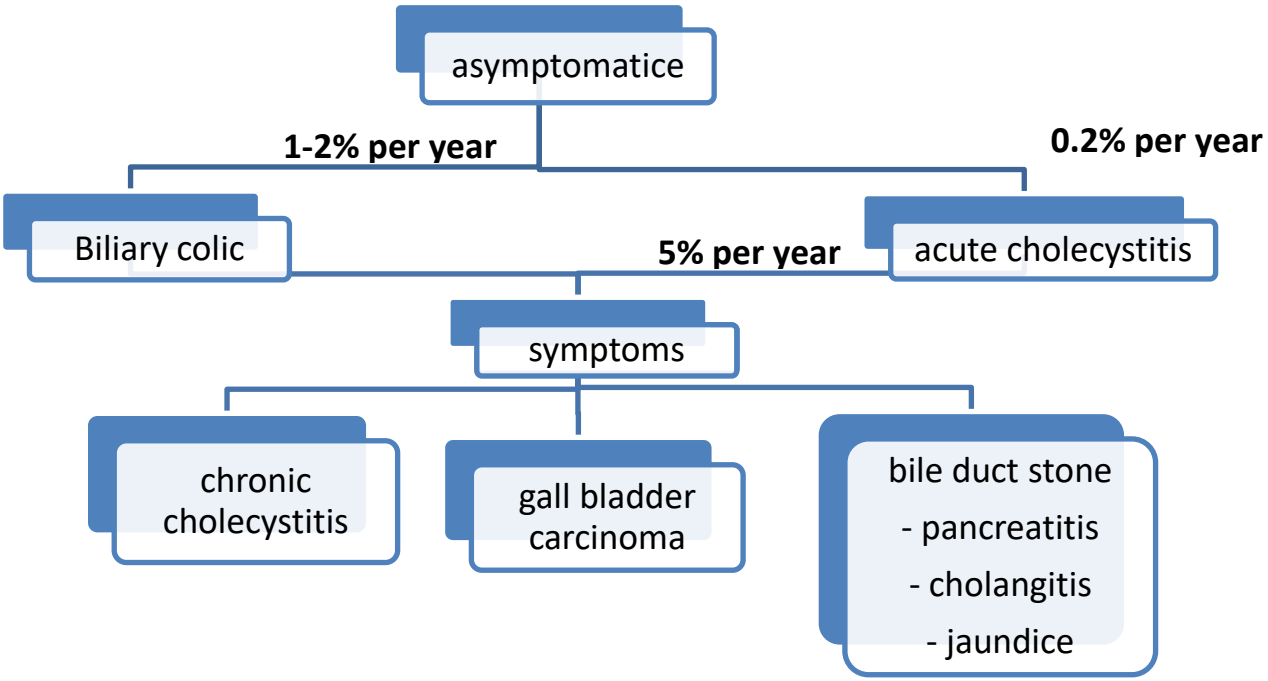
- Or sometimes minor episodes of discomfort during day time .

2- Nausea , anorexia , vomiting , sustained for 2 hrs .

Dyspepsia which worse after an attack and as the pain resolved the patient can eat or drink .

***** Patient may have several episodes over a period of few wks and then no more trouble for some months .

***** Natural history of gall stones :



* Diagnosis depend on

1- History

2- On examination : murphy's sign

3- Confirmatory by :

- **X**-ray
- **U**ltrasourd
- **R**adio nuclide scan

* Complication and effects of gallstones

In the gallbladder :

1- **B**iliary colic " cystic duct stone " .

2- **A**cute cholecystitis .

3- **C**hronic cholecystitis .

4- **E**mpyema of the gall bladder **&** gangrene .

5- **M**ucocele . 6- **P**erforation .

* In the bile ducts :

1- **B**iliary obstruction .

2- **A**cute cholangitis .

3- Acute pancreatis .

In the intestine :

- **I**ntestinal Obstruction " gallstone ileus "

* Treatment

1- Observation for asymptomatic G.S .

2- Cholecystectomy for those developing :

- Symptoms " Biliary colic and cholangitis "
- or Complication .

3- Prophylactic cholecystectomy in diabetes mellitus and congenital blood diseases .

Acute cholecystitis

* Aetiology

- **In 95% stones or stone present " impacted in cystic duct or neck of bladder .**
- **I**n other may be **bacteria** can be cultured from the he wall of gall bladder " E-coli , klelbsciella , strept faecalis (50% of cnlture of G.B content are sterile after surgery)
- **Occasionally obstruction may by mucus , worms or tumor** .

*** Pathogenesis :**

- **I**t is unclear , but initial inflammation is possibly chemicaly induced which lead to G.B .
- **M**ucosal damage which release phospholipase , converting biliary lecithin to lysolecithin " recogised mucosal toxin " .

* Clinical features

1- Abdominal pain (cardinal feature)

- Sudden onset .
- In RT. Upper quadrant but also in epigaitium , RT. shoulder tip or interscapular region .
- **L**asts for more than an hour
- **M**ay , be colicky or dull and constant .

2**- Fever and rigor may occur**

3- Nausea , anorexia , vomiting , dyspepsia and flatulence and food into tolerance especially to fat .

* On examination

1- **RT. Hypochondrial tenderness , rigidity which worse on inspiration " Murphy's sign .**

2- Gall bladder mass "palpable bladder" .

3- **Elevated temperature .**

4- **L**eucocytosis (is common except elderly) .

5- **J**aundice (20-25% of ratient) .

6- **M**inor increase of plasma trans aminase 8 amylase .

* mortality rate in elderly may reach 10% .

* Diagnosis depend on :

1- **H**istory

2- **P**lain X-ray of abdomen **&** chest { stone or sub diaphragmatic exclude lower lobe pneumonia } .

3- **U**lrasonogra phy .

4- **P**lasma amylase to exclude acute pancreatitis .

5- **L**eucocytosis .

* Complication

- **E**mpyema.
- **P**erforation .
- **P**eritonitis .

* Differential diagnosis :

Common :

- **Acute appendicitis .**
- **Perforated P.U .**
- **Acute pancreatitis** .

*** U**ncommon :

- **A**cute pyelonephritis.
- **M**yocardiac infarction.
- **P**neumonia .

* Management

- **Conservation medical treatment** followed by cholecystectomy medical treatment :
  1- **B**ed rest
  2-**P**ain relief "analgesic drugs ": voltarin , pethidin morphin, atropine
  3- **A**ntibiotic drugs " cephalosporin , metronidazol "
  4-**M**aintenance fluid balance and nasogastric aspiration just for persistent vomiting .

*** Surgical treatment :**

- **E**mergency surgery : when :
  1- **P**rogress cholecytitis inspite of medical therapy .
  2- **S**everely ill patient .
  3- **D**evelopment of complication .

- **Delayed surgery for 2-3 days of medical therapy .**

- Delayed surgery for 2-3 month is not favored .
- **S**urgery either open or laparoscopy .

Chronic cholecystitis

- **I**t is almost invariably associated with **G.S** .
  Or chemical irritation by or followed acute cholecystitis .
- May be a symptomatic .
- **I**f symptomatic usually present with : recurrent attacks of upper abdominal quadrant pain especially following a heavy meal with flatulence dyspepsia .
- **Si**milar to clinical feature of acute cholecystitis but milder .
- **I**n diagnosis same of acute cholecystitis in addition to oral cholestography or I.V cholecystograhy
- **P**atient may recovered spnotaneously or following analgesic and antibiotic even so we advice the patient to undergo a cholecystectomy sometimes we use dissolution of **G.S** by bile acid chenodeoxy chalic & ursodeoxy chalie acid will remove radiolucent **G.S** .

"Acute pancreatitis "

*The pancreas (head, body ,tail) is a spongy retroperitoneal gland posterior to the greater curvature of stomach.

*It accounts for 3% of all cases of abdominal pain to hospital

*It affect 2-28 per 100,000 of population

*May occur at any age with peak in young men (30-40y) &old women over 50.

Pathophysiology

Endocrine [pancreatic islents]produce insulin &glucagon hormon

Exocrine [acini] produce digestive enzyme (digest protein, CHO, fats). *When pancreas become severely damage or when the duct become blocked by gall stone, large quantities of pancreatic secretion sometimes become pooled in the damaged area and the trypsin inhibitor is often over whelmed and so pancreas secret rapidly become activate &digest entire pancreas within hours .
-**The severity of acute pancreatitis is dependent upon the balance between activity of released proteolytic enzymes &antiproteolytic factors** (trypsin-inhibitor protein ,circulating B2-macroglobin ,alph 1-antitrypsin &C1-esterase inhibitor).

Aetiology

Common (90% of causes):

1- **Gall stones** (biliary calculi) 50-70% .Reflux of infected bile or duodenal contents into pancreatic duct.
2- **Alcohol abuse** 25%.Hyperstimulation of pancreas 'also fat do that '
3- **Idiopathic**
4- **Post –ERCP**

Rare:

1- Post surgical (biliary , upper GIT surgery).
2- **Abdominal trauma (blow to abdomen).**
3- Obstruction of ampulla of vater due to peptic ulcer or tumour .
4- Drugs (thiazide ,estrogen ,corticosteriod ,etc..)
5- Metabolic (hypercalcaemia ,hypertriglyceridaemia).
6- Sphincter of oddi dysfunction
7- **infection (mumps**,coxsakie virus).
8- Hereditary &autoimmune pancreatitis ,hyoerparathyrodism
9- Renal failure
10-Organ transplantation "kidney ,liver "

11-Severe hypothermia

12-Petrochemical exposure

13-Scorpion bite

14-Malnutrition

Clinical Features :

1) Abdominal pain : (**Cardinal symptom**)

-Usually in **epigastrium** but may be in upper quadrant or felt diffusely throughout the abdomen.

-**Radiate to back in about 50%of patient**

-May relieved by sitting or leaning forwards.

-**Develops quickly ,reaching maximum intensity within minutes &persists for hours or days**.

-Frequently severe ,constant &refractory to the usual dose of analgsics.

2) Nausea ,anorexia ,repeated vomiting &retching are usually marked (which may persist despite the stomach kept empty by nasogastric aspiration) and also hiccough due to gastric distension and diaphragmatic irritation .

3) Fever in case of cholangitis

On Examination

1- Epigastric tenderness ;gaurding –rebound tenderness

*in early stages (in contrast to perforated P.U.)gaurding &rebound tenderness are absent due to the inflammuation is principally retroperitonial.

2-Bowal sounds quiet or absent (due to development of paralytic ileus which also cause distension.

3-Tachypnoea ,Tachycardia ,hypotension.

4-Normal or subnormal temperature **but raising in case of cholangitis**.

5-Mild icterus.

*In severe cases

1-Hypoxic &hypovolaemic shock with oliguria

2-Grey Turners sign [bluish discoloration of Flank region]

3-Cullens sign [bluish discoloration of periumbilical region]

These are due to bleeding fascial planes &neither sign is pathognomonic because may occur in ectopic pregnancy rupture .

4-Pleural effusion in 10-20% of patients .

5-Abdominal distension "due to paralytic ileus "and abdominal "epigastric" mass due to inflammation .

Differential diagnosis

1-Perforated peptic ulcer

2-Acute cholecystitis

3-Myocardial infarction .

Complications

Systemic ;

1-Systemic inflammatory syndrome.

2-Hypoxia –due to reduced ventilation due to pain, pancreatic oedema, elevation of diaphragm.

3-Hyperglycaemia.

4-Reduced albumin concentration.

Pancreatic ;

1-Necrosis causes abscess.

2-pancreatic ascites or pleural effusion.

Gastrointestinal ;

1-Upper gastrointestinal bleeding .

2-Duodenal obstruction.

3-Variceal haemorrhage.

4-Erousion into colon cause⟶ fibrosis ⟶ stricture of "transverse colon".

Investigation

The diagnosis is made on the basis of ;

1- **clinical presentation.**
2- **Serum amylase elevation (3 to 4 times above normal)is indicate for disease.**
3- **Serum lipase elevation** (slightly sensitive or specific)
4- **Contrast –enhanced CT scan** is the best single imaging investigation but used only in deteriorated case.
5- **Plain X-ray of abdomen** which show ;

-Dilated loop of small intestine over the Lt. upper quadrant of abdomen

-Moderate distension of duodenum with fluid air level

-Mild distension of transverse colon &collapse of descending colon "colon cut –off sign".

6- **Ultrasound** ; Not establish the diagnosis of acute pancreatitis but should be done with in 24hours in all patient to detect gall stone &swollen pancreas &to exclude acute cholecystitis &to determine common bile duct dilatation

7- **Cross-sectional MRI**

8- **ERCP** ;especially in gall stone pancreatitis to remove common bile duct stone.

******(**Some time diagnosis is made only at laporatomy**)

Management

***In mild acute pancreatitis attack**

1- Bed rest.

2- Observation & I.V. fluid.

3- Analgesic &antiemetic drugs.

4- No need of antibiotic.

***In severe acute pancreatitis**

1- Admission to high –dependency or intensive care unit{I.C.U}.

2- Analgesic drug.

3- Aggressive fluid rehydration .

4- Nasogastric drainge "suction ".

5- Oxygenation.

6- Invasive monitoring of vital signs ;

-central venous pressure -urine output -Blood gases

7- Frequent monitoring of haematological &biochemical parameters (liver &kidney function ,clotting ,serum Ca ,blood glucose).

8- Prophylactic antibiotic (imipenem ,cefuroxime ,flagyl ,ciprodar).

9- Nutrional support "nasogastric tube feeding " .

10- **ERCP with in 72hr for severe gall stone pancreatitis or signs of cholecystitis**.

11-Supporative therapy for organic failure [inotropic ,ventilation].

"Chronic pancreatitis "

***Chronic inflammation disease characterised by fibrosis &destruction of exocrine pancreatic tissue .**

***Diabetes mellitus occur in advanced cases**

***70-80% of cases result from alcohol abuse** &affect middle aged men.

clinical features

1- **Abdominal pain** 50%occurs as episodes of acute pancreatitis or slowly progressive chronic pain or no pain just diarrhea.
2- **Weight loss &steatorrhoea** caused by malabsorption & diabetes, avoidance of food due to pain ,anorexia.

On examination

1- Thin, malnouriched patient.

2- Epigastric tenderness.

3-Skin pigmentation over abdomen due to use hot water bottle to relief pain.

Treatment

1- **alcohol avoidance & fat restriction**
2- pain relief by NSAID
3- oral pancreatic enzyme supplements
4- surgical or endoscopy pancreatic therapy

"Carcinoma of Oesophagus"

- **It is the 6th most common cancer in the world.**

-It is a disease of mid to late adulthood with a poor survival rate only 5-10% of those diagnosis will survive for 5 years .

Types :

-**Squamous cell cancer = usually affect the upper 2/3(most common)**

-**Adenocarcinoma =usually affect the lower 1/3** (more in west &increasing in incidence)

The tumours can spread in 3 ways:

1- **Invasion directly through oesophageal wall (locally).**
2- **Via lymphatic**(common)→ caudal and cranial direction.
3- **blood stream** →liver, lung, brain, bone.

****Tumours is often well established before the diagnosis is made** .

****common aetiological factors are**: **Tobacco, alcohol and fungal contamination of food &nutritional deficient** especially in china.

Clinical features

In early disease may have;

1- Non-specific dyspeptic symptoms.

Or 2-Vague feeling of something that is not quite right during swallowing.

****Some are diagnosis during endoscopy &so do biopsy**.

A late features (Mechanical symptoms)

1- **Dysphagia.**
2- **Regurgitation.**
3- **Vomiting & anorexia & weight loss.**

In more advanced malignancy

*- Recrrent laryngeal palsy **"Hoarseness of voice** ".

*- Chronic spinal pain &diaphragmatic paralysis.

*- Palpable lymphadenopathy.

*- Cough when bronchi is involved.

Investigation

1- **Endoscopy is the first line investigation with cytological &histological specimens taken.**
2- **Barium –swallow** to detect filling defect and do **chest X-ray**.
3- **Ultrasound of abdomen** to detect spread to liver.
4- **Bronchoscopy** to detect spread to trachea or bronchi.
5- **laparoscopy** for diagnosis of intra abdominal &hepatic metastases and for biopsy.
6- **CT-scan** to identify haematogenous metastases.
7- **MRI scanning.**
8- **Endoscopic ultrasound "EUS"**

*the 2prinicpal prognostic factors for oesophageal cancer are the depth of tumour penetration through the oesophageal wall &regional lymph node spread and these are determined by EUS.

9- Positron emission tomography (PET) with CT-scan ,by using radiopharmaceutical agent ^{18}F-Fluorodeoxyglucose "FDG"

Treatment

At the time of diagnosis ,around 2/3 of all patients with oesophageal cancer will already have incurable disease .

1- **Radical oesophagectomy is the most important aspect of curative treatment**.
2- **Chemo-radiotherapy alone may cure selected patients "particularly squamous cell carcinoma ".**
3- **Useful palliation may be achieved for dysphagia relief by chemo/radiotherapy or endoscopic "photodynamic therapy "(PDT)** for those unfit or unwilling to undergo surgery ,by administration of photosensitive that is taken up by dysplastic &malignants cell followed an exposure to laser light.

Palliative therapy (for dysphagia relief in those with very advanced disease which should be simple and effective).

1- Endoscopic :

***Laser therapy** ,**Alcohol injection** ,bipolar diathermy ,argon-beam plasma coagulation .

***radiotherapy** (Brachy therapy)(intraluminal radiation). ***insertion of stents** or unblock stent .and rigid or rubber tubes placement **2- Chemotherapy** :use Cisplatinum &5-fluorouracil. **3- Radiotherapy**

4- Nutritional support :I.V. feeding , nasogastric tube feeding

5- Appropriate analgesia.

****Combination pre-operative radiotherapy ,chemotherapy &radical surgery is associated with 5-years survival of 30-50%in selected cases otherwise 6-9%.**

****90%of patient have extensive disease at presentation so mostly need palliative treatment to relieve dysphagia &pain .**

"Liver abscess "

It can be classificad as:

1- Pyogenic

2- hydatid

3- Amoebic

Pyogenic liver abscess

*Common in elderly, diabetics &immunosuppressed patient.

*Uncommon but important – because -potentially curable, Inevitably fatal if untreated& Readily over looked.

***single lesion are more common in Rt. Liver.**

Causes

Infection can reach the liver in several ways:-

***Biliary obstruction "cholangitis"**→multiple abscess

***Haematogenous** -portal vein "mesenteric infection"

-Hepatic artery "Bacteraemia"

***Direct extension** "empyema of gall bladder"

***Trauma** "penetrating or non-penetrating "

***infection of liver tumour or cyst.**

Microorganisme :

-**E-coli** .

-**streptococci** "especially strep.milleri "

-**anaerobes" streptococci & bacteriode"**

Clinical feature

A- Atypical presentation are common

B- 1- Abdominal pain –in Rt. upper quadrant sometimes radiate to Rt.Shoulder .

2- Fever sometimes rigor accompanied Rt. upper quadrant discomfort.

3- Wight loss.

4- Mild jaundice may present.

On examination

-Hepatomegaly ">1/2 of patients "

-Tenderness by gentle percussion over the organ .

Investigation

1- **Liver imaging** (CT.scan) is the most revealing investigation show 90% of symptomatic abscess.
2- **Needle aspiration** "percutaneous drainage" under ultrasound guidance confirm diagnosis &provides pus for culture &sensitivity.
3- **Leucocytosis.**
4- **Plasma alkaline phosphatase is usually increased.**
5- **Serum albumin is low.**
6- **Chest X-Ray** ; Raised Rt. Diaphragm &lung collapse or effusion at base of Rt. Lung.
7- **Blood culture** →may reveal causative organism.

Management

1- **Prolonged antibiotic therapy** :combination of ampicillin+gentamicin+metronidazol.
2- **Drainage of abscess or aspiration** by catheter under U/S guidance
3- Surgical drainage is rarely undertaken.
4- **Hepatic resection** may be indication for chronic persistant abscess "pseudotumour ".

Prognosis Mortality of liver abscess is 20-40% due to failure to make diagnosis, older patient &those with multiple abscess have higher mortilaty .

''Hydatid cysts ''

*It is caused by **Echincoccus granulosis infection**.

*May be **single or multiple** ,it is present in dog intestine and ova are ingested by humans &pass to liver via portal blood.

***Chronic cysts become calcified**.

***May be **asympatomatic but may present with symptoms** :

Upper abdominal pain or mass, or acute abdomen after minor trauma to abdomen due to rupture of cyst into peritoneal cavity.

***May be blood eosinophelia**.

***X-ray show calcification.**

***Serological test +ve in 50%of case by ELISA** (enzyme-linked immuno-sorbent assay).

***Liver imaging show the cysts by ultrasound and CT-scan.**

*__Rupture or secondary infection of cyst__ can occur &communication with intrahepatic biliary tree can result (__obstructive jaundice__)&if rupture in to diaphragm producing __.empyema__

Management

-__Medically by albendazol or mebendazol prior to definitive therapy__ .

-__Surgical__ :

*__a- Percutaneous aspiration of cyst follwed by injection of 100%ethanol into cyst &then re-aspiration of cyst content (PAIR)__{percutaneous treatment with hypertonic salin with alcohol}

*__b- Surgical removal of intact cyst__ when there is communicat with the biliary tree.

''Amoebic liver abscess''

*It is caused by **Entamoeba histolytica infection.**

*Up to 50% of case do not have a previous history of intestinal disease(dysentery).

*Most found in endemic area and it is spread by **faeco –oral route** .

*Abscess are usually large ,single ,&located in Rt.liver lobe.

*Multiple abscess may occur in advanced disease.

*__Fever & abdominal pain or swelling are the most common symptom__

*__Diagnosis depend on liver cyst aspiration to isolate the parasite &also from stool .__

Treatment

Metronidazol 750mg ''t.d.s.'' for 5-10 days'' &investigated further if they do not respond .

The amoebic cyst is ingested &develops in to trophozoite form in the colon &then passes through the bowel wall to the liver via portal blood, so two presentation either dysentery or liver abscess .

Liver Trauma ''injuries''

-It is uncommon because position of liver under diaphragm &protected by chest wall.

-But if occur it is serious.

-It divided in to **Blunt injuries &penetrating injuries** .

Blunt injuries produce :-contusion ,laceration &avulsion to liver

- **Often associated with splenic ,mesenteric or renal injuries.**
- **Need conservative treatment unless development signs of generalized peritonitis.**

Penetrating injuries ''as stab &gun shot wounds''

- **Often associated with chest or pericardial involvement.**
- **Need surgical exploration**

Diagnosis

1- **Chest X.ray and abdominal X-ray.**
2- **CT.scan** (for chest &abdomen)with contrast by oral or I.V.
3- **Laporatomy &thoracotomy &laparoscopy** {confirm diaphragmatic rupture }.
4- **Peritoneal lavage** to confirm haemoperitoneum.

Because liver is extremely well-vascularised organ , **blood loss is major complication &coagulopathy** ''lack of clotting factors &fibrinogen''other complication ;intrahepatic haematoma ,liver abscess ,bile collection, biliary fistula,A.V fistula,liver failure.

Management

1- Remember associated injuries
2- Resuscitate ; a- Air way patency
b- Breathing pattern –may need ventilation

c- Circulation ;O-ve blood or colloid I.V. through Cannula .

d- With C.B.P. ,renal function test ,liver function test, clotting ,glucose ,amylase test.

chest tube drainage in case of haemo or pneumothorax

3- Assessment of injury

- Spiral CT with contrast ''I.V. or oral ''

- Laparotomy

4- Treatment

1- **Correct coagulopathy** (may need fresh forzen plasma &cryo-precipitate).
2- Suture laceration.
3- **Resect if major vascular injury**.
4- Packing if diffuse parenchymal injury.

Jaundice

Definition: yellow discoloration of skin, sclera and mucous membrane due to increased bilirubin concentration in the body fluid. It is detectable when bilirubin exceeds 50 mmol/liter(3mg/100ml blood).All tissues are coloured except brain. Normal bilirubin is 2-17 mmol/liter.

Causes

1-Haemolysis.

2-Impaired hepatic bilirubin transport e.g Gilbert syndrome.

3-Hepatic cellular damage →hepatitis.

4-Impaired bile flow [cholestasis] stone, tumors, drugs, alcohol, cirrhosis.

Clinical features in cholestatic jaundice

1-J aundice.

2-Dark urine.

3-Pale stool.

4-Pruritus.

5-Later on: Xanthelasma, Steatorrhoea, Weight loss, Bleeding tendency.

Viral hepatitis

Aetiology

-A, B,S (delta) and C viruses.

-non A non B (E,C and sporadic).

I-A virus :

RNA virus spread by facecs which contaminate food.

****The infectivity; 2weeks before jaundice and one week after jaundice.**

****Incubation period 2-4 weeks.**

**prevalent more in ; Children, poor sanitation, overcrowding.

****Immune serum globulin can protect from disease about 3 months.**

II- B-virus:

DNA virus, incubation period 4-20 weeks spread by :

1.Sexual intercourse (mostly homosexual).

2.Sharing needles

3.Blood and its product transfusion.

4.Direct inoculation (wounds prick) in surgery and shaving and dentistry,

working in tattooing and acupuncture.

5.Handling blood.

6.Workers of hospitals and dentist.

7.Transplacental to the fetus.

-Hyper immune gamma globulin(passive immunization) i.m. or vaccine give protection 95% (active immunization i.m.)

III-S (Delta virus):

1-RNA virus, incubation period 6-9 weeks.

2-Incomplete (not independent) -Needs B virus for replication(causing infection).So it infest the person after infection or carrying B virus.

3-Routes of transmission as B virus.

IV-Virus C & E (Non A non B virus);

C – RNA virus:

1-Mode of transmission as B virus.

2-Cause 95% of post transfusion hepatitis and 25% of sporadic hepatitis.

Clinical features of acute hepatitis

1.Chill, headache &malaise.

2. Anorexia. Nausea. Vomiting. Diarrhea.

3.Upper abdominal pain., tender enlarged liver.

(These are prodromal sympotoms and signs before jaundice appears);

1.Dark urine.

2.Yellow sclera.

3.Sometimes the disease is subclinical.

Investigations

1- **Plasma amino tansferase exceed 400U/l.**
2- **Plasma bilirubin increase.**
3- **Prothrombin time increases.**
4- **Serological tests.**

Complications

1- Chronic hepatitis .
2- Liver cirrhosis.
3- Hepatic failure.
4- Carcinoma.
5- Bleeding tendency.

Management

1- **Bed rest.**
2- **High calorie diet .**
3- **No drugs.**
4- **Vitamines.**

Sometimes dexamethasone has been given

Tumours of the stomach

Gastric carcinoma

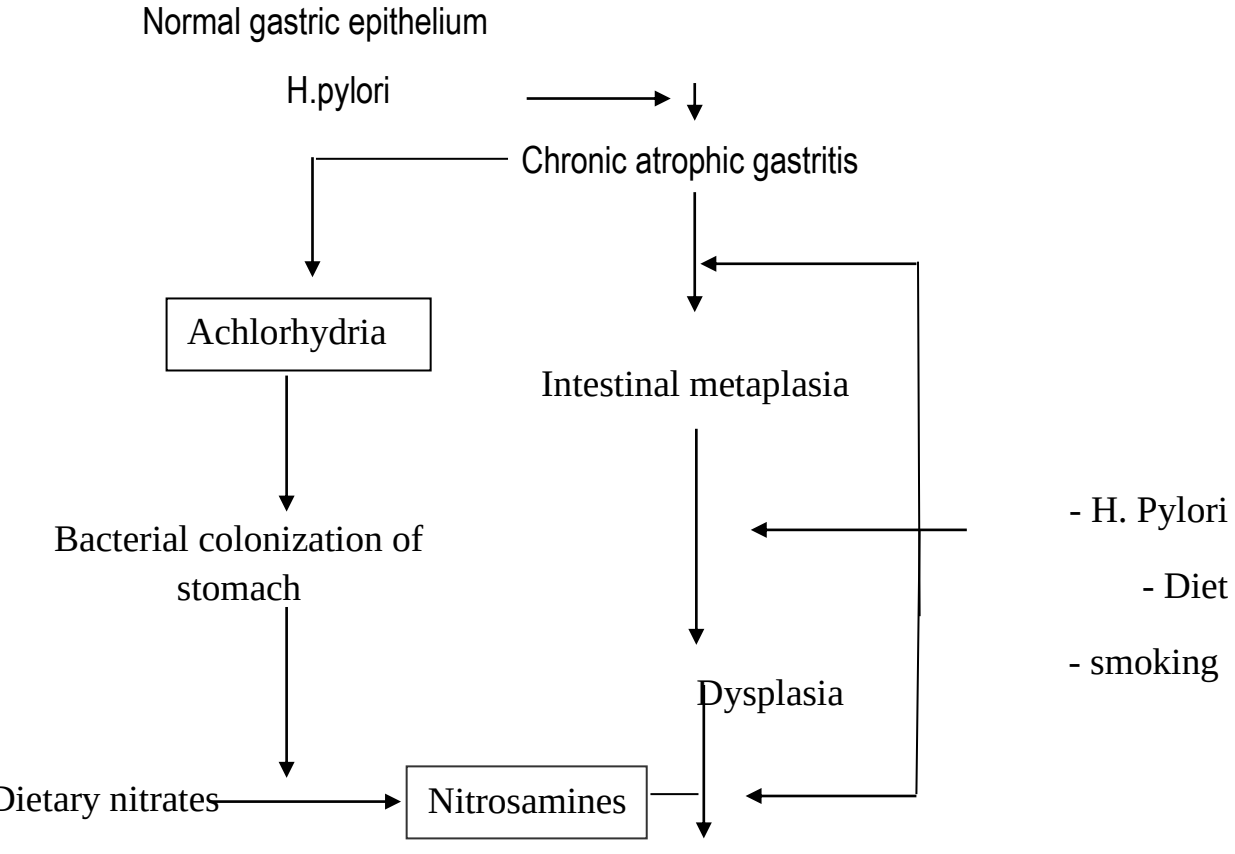
- It is the leading cause of cancer death world- wide.
- It is common in china, Japan & South America & its mortality rate 30-40 per 100000.
- It is more common in men & rises sharply after 50 years of age.
- **50% occurs in the antrum** & 20-30% in gastric body & 20% in cardia(great curvature).

Aetiology

1- **H. Pylori infection**; may be responsible for 60-70% of cases.
2- **Diets;**
   - Rich in salted, smoked or pickled foods & consumption nitrates & nitrites are associated with risk .
   - Diets lacking fresh fruit & vegetables & vitamins C& vitamins A.
3- **Smoking & dust ingestion from industrial process.**
4- **Heavy alcohol intake.**
5- **Pernicious anaemia** (failure of intrisie factor section by stomch no absorption of vit. B12 fumileum dreto no formation of complex of ingester B12 & intrensie factor in ilenm.
6- **Previous partial gastrectomy, Billroth II anastomosis**.
7- **Family history may be & those with group A.**
8- **Gastric atrophy** & poly & intestinal metaplasia.

Pathology

All tumours are **adenocarcinoma** develop upon a background of chronic atrophic gastritis with intestinal metaplasia & dysplasia.



Carcinoma

Clinical features

A- Early stage is usually asymptomatic, discovered during endoseopy.

B- 2**/3 of patient with advanced cancers have:**

1. **Weight loss.**
2. **Ulcer- like pain 50% of patient.**
3. **Anorexia, nausea 1/3 of patient.**
4. **Anemia from occult blood.**
5. **Early – haematemesis, malaena & dyspepsia are less common**
6. **Dysphagia: in case of cardia tumors.**

On examination

Reveal no abnormality but signs of:

1. **Weight loss**.
2. **Anaemia** is not infrequent.
3. **Palpable epigastric mass** is not infrequent.
4. **Jaundice & Ascites may signify metastatic spread of tumor to L.N:** (supraclavicular,Umbilical,Ovaries&also spread to liver, lungs, peritoneum)

.

Diagnosis

1. **Endoscopy**: the investigation of choice, with multiple biopsies must be taken for cytology.
2. **Barum meal**.
3. **Endoscopic ultrasound**: demonstrate penetration of submncosa & Lymph node affected.
4. **CT- Scan**: liver metastases.
5. **Laparoscopy**: to determine whether the tumour is respectable.

Management

Surgical resection (can be achieved in 80-90%& patient with early stage& preoperative neo-adjuvant chemotherapy are more encouraging.

1. **partial gastrectomy**: (for distal localized stomach tumour).
2. **Oesophagogastrectomy**: (for proximal body or cardia tumour)

* Extensive L.N. resection → survival rate but morbidity.

* post – operative radiotherapy is of no benefit.

* post – adjuvant chemotherapy also have disappointing.

Tumours of the small intestine

Small intestine is rarely affected by neoplasia & fewer than 5% of all G.I.T. tumours occur here.

1- Benign tumours:

-adenomas}(most common)

-lipomas

-hamartomas

***Asymptomatic or occult bleeding**

2-Malignant tumours :(rare)

-adenocarcinoma

-carcinoid tumour : most common inileum-spread to liver

-lymphoma : non-Hodgkin type.

*majority occur in middle age or later

*non-specific presentation & rarity lead to delay in diagnosis.

Investigation

1- **Barium follow-through & small bowel enema**

2- Enteroscopy, capsule endoscopy

3- mesenteric angiography

4- CT- scan

Treatment: surgical resection.

Colorectal cancer (Tumours of large intestine)

Although relatively rare in the developing world, colorectal cancer is the second most common internal malignancy & second leading cause of cancer death in western countries.

Aetiology

1- **Environmental factors(** Dietary risk factors): account for over 80% of all sporadic colorectal cancer.

****↑risk: - Red meat**

-Saturated animal fat }risk

****↓risk: - Dietary fibre**

- Firm & vegetables - Calcium - Folic acid

2- **Genetic factors**; Chromosomal instability & microstellite instability

** Most tumours arise from malignant transformation of a benign adenomatous polyp.

**Over 65% occur in rectosigmoid & 15% recur in the caecum or ascending colon.

**Majority of cancers are either polypoid (fungating) or annular & constricting.

**Spread locally, lymphatic, portal systemic circulation

Clinical features

1- Colicky lower abdominal pain in 2/3 of patient.

2- Anaemia due to occult bleeding in Rt. colonic cancer.

3- Altered bowel habit

4- Obstruction of intestines is late feature & also perforation & fistula formation

5- Mucus discharge & feeling of incomplete emptying(tenesmus) with rectal bleeding in carcinoma of rectum.

6- Palpable mass with signs of anaemia or hepatomegaly & by digital examination we can palpate low rectal tumour.

Investigation

1- **Rigid sigmiodoscopy: detect <1/3 of tumours**
2- **Colonoscopy: in inventigation of choice.**
3- **Barium enema**
4- **Endo anal ultrasound**
5- **pelvic MRI**
6- **CT colography**
7- **CEA (carcino embryonic antigen);** Raised its serum concentration is benefit for follow- up & detect early recurrence:

Management

A- **Adequate surgical resection** of tumour & pericolic Lymphnode.
B- **Palliative therapy; Chemotherapy** with 5- fluorouracil & folinica acid improve surviral.
C- **Pelvic radiotherapy**: useful for pain, bleeding & severe tenesmus .
D- **Endoscopic laser therapy** or **insertion of an expandable metal stent to relieve obstruction.**

Coronary heart diseases

1- **Stable angina**: Ischemia due to fixed atheromatous stenosis of one or more coronary arteries.
2- **Unstable angina**: Ischemia caused by dynamic obstruction of coronary artery due to plaque rupture with superimposed thrombosis and spasm.
3- **Myocardial infarction**: Myocardial necrosis caused by acute occlusion of coronary artery due to plaque rupture and thrombosis.
4- **Heart failure**: Myocardial dysfunction due to infarction or ischemia.
5- **Arrhythmia**: Altered conduction due to infarction or ischemia.
6- **Sudden death**: Ventricular arrhythmia, Asystole or massive MI.

Myocardial ischemia

Angina Pectoris

Definition: **Chest discomfort or pain due to transient myocardial ischemia.**

Aetiology:

1-Coronary atheroma (most common)

2-Aortic valve diseases(stenosis and regurgitation)

3- Hypertrophic cardiomyopathy

4-Coronary artery spasm(accompanied by ST-depression called also **prinzmetal** or **variant angina**)

*Angina may be unstable or stable

Unstable angina:(**crescendo angina**) which present with severe rapidly worsing angina at rest or prolonged and severe ischemic chest pain without ECG or enzymes evidence of significant MI.

Stable angina: may occurs whenever there is an imbalance between myocardial oxygen demand and supply which influenced by many factors (aggravating factors):

Oxygen demand;

Cardiac work, Heart rate, Blood pressure and Myocardial contractility.

Oxygen supply;

Coronary blood flow, Oxygenation(Hb and Oxygen saturation).

Clinical situation precipitating angina:

Common;**1 -** peripheral vasoconstrictor which lead to ↑Oxygen demand;

-Physical exertion and walking uphill

-Cold exposure and strong wind

-Heavy meal

2-Intensive emotion

3-Hypertension, Anaemia, Hyperthyroidism and Hypothyroidism

Rare; 1- Lying flat (Decubitus angina)

2- Violent(vivid) dreams (nocturnal angina)

Clinical features

1- **Chest pain;** --**Central**; may be **epigastric** or **interscapular** pain.

--**Relieved by rest or sublingual nitrate**

--May radiate to neck or jaw.

--Often accompanied by arms discomfort especially Lt., wrist and hands(heaviness and uselessness) and sometimes dyspnoea.

2- **Other presentations**;

--Sense of oppression or tightness in chest.

--Band around chest.

--The patient closes a hand the throat or clenched fist on Sternum or places both hands across the lower chest.

--**Sometimes pain come at start walking then disappears After that(start- up angina).**

-- **Sometimes pain come when lying flat(decubitus angina).**

-- **Sometimes pain awakened patient(nocturnal angina).**

Physical examination

It is frequently negative, but should include a careful search for evidence of risky factors and contributory diseases; like ; Hypertension, Hyperlipidaemia, Diabetes mellitus,

Myxoedema, Obesity, Anaemia, Thyrotoxicosis, Aortic valve disease, Nicotine stain and Lt. heart dysfunction.

Differential diagnosis

1- **Musculoskeletal pain**; provoked by specific movement rather than walking.
2- **Pericarditis**; provoked by changes in posture or deep breathing.
3- **Oesophagitis**; burning pain and relieved by antacid
4- **Oesophageal spasm**; difficult to differentiated from angina variant.
5- **Psychological chest pain**.

Investigation

1- **ECG:**---**normal in most patients.**
---Evidence of previous MI.
---**ST- segment depression or elevation with or without T- inversion at time of angina attack symptoms**(whether spontaneous or induced by exercise test).
2- **Exercise ECG; exercise tolerance test;(ETT):**
It shows planer or down sloping ST-depression of 1mm or more.
3- **Isotope scaning; Thallium 201(^{201}TI);** It shows perfusion defect either during stress(revesible myocardial ischemia) or during rest and stress(previous MI).
4- **Echocardiography** ; For ventricular function assessment.
5- **Coronary arteriography**; For the assessment of the nature and extent of coronary artery diseases.

Management

A- **General measurements(advices):**
   1- **Do not smoking**
   2- **Ideal body weight**
   3- **Take regular exercise** not beyond the point of chest pain to promot collateral vessels
   4- **Avoid severe unaccustomed exertion and vigorous exercise after a heavy meal**
B- **Prophylactic sublingual nitrates**
C- **Aspirin**(75-300mg) daily to reduce risk of adverse events such as MI
D- **Non-invasive Anti-anginal treatment**; To relieve angina symptoms:
   1- **Nitrates**

Vasodilator of vein and artery(↓preload, ↓ afterload and ↑myocardial O_2 supply) ;**---GTN**(glyceryl trinitrate); it is taken sublingually tablet, aerosol, oral tablet, percutaneously patch or paste, buccally, and lastly is used intravenously in case of unstable angina and aute heart failure.

Side effect; Headache with in 2-3 minutes of sublingually treatment.

---Isosorbide mononitrate and dinitrate; given orally.

2- β-Blockers

They lower myocardial O_2 demand by reducing heart rate, blood pressure and myocardial contractility.

---Cardio selective β-Blockers drug given once daily;

Atenolol –tenormine-(50-100mg/daily), **Bisoprolol**(5-10mg/daily).

---Cardio nonselective β-Blockers aggravate coronary vasospasm and Brochospasm, **Propranolol** (Inderal).

*** *We advice not to stop β-Blockers drugs suddenly to avoid danger of Arrhythmia,MI,angina and β-Blockers withdrawal syndrome(rebound HT).**

3- Calcium channel blockers(Ca^{++} antagonists)

They lower myocardial O_2 demand by reducing blood pressure and myocardial contractility, so may precipitate heart failure, flushing of face and dizziness.

---**Nifedipine**(adulate), **Nicardine**, **Amlodipine**; They are better to be used with β-Blockers to overcome reflex tachycardia.

---**Verapamil(Isoptine) or Diltiazem**; They are not better to be used with β-Blockers due to their effect of inhibiting conduction through AV-node so causing bradycardia.

4- Potassium- channel activators

Vasodilator of vein and artery---**Nicorandil**(10-30mg twice daily).

****Start with Aspirin + Sublingual GTN + β-Blocker and add Ca^{++} antagonists or Long – acting Nitrate later.**

E- Invasive anti-anginal treatment

1- Coronary angioplasty (Percutaneouse transluminal coronary angioplasty)

- PTCA - → 32% of angina is recurrent after 6 months of treatment.

2- Coronary artery bypass grafting (CABG).

--Reversed Saphenous vein bypass grafting.

-- Internal mammary artery grafting.

Myocardial infarction

Definition: Myocardial necrosis caused by acute occlusion of a coronary artery due to plaque rupture and thrombosis, it is aggravated by any situation(factors) that lead to imbalance between coronary blood supply and myocardial demand.

** the propagated thrombus in coronary artery lumen often undergoes spontaneous lysis over the course of next few days, although by this time irreversible myocardial damage has occurred.

**** The process of infarction takes place at least 8hours.**

**** Early death are due to ventricular fibrillation** but later on death determined by the extent of myocardial damage.

Clinical features

1- **Chest pain**; Like angina pain but more severe and longer(few hours)
2- **Dyspnoea** (may be only symptom)
3- **Nausea and vomiting**
4- **Collapse or syncope** (due to arrhythmia or hypotension) are common
5- **Anxiety** (fear of impending death) and sweating
6- **Silent or painless MI(common in elderly and D.M.)**

Signs;

1- **Signs of sympathetic activation(Pallor, Sweating, Tachycardia)**
2- **Signs of parasympathetic activation(Nausea, Vomiting,Bradycaria)**
3- **Signs of impaired myocardial function(Hypotension, Oliguria. Cold peripheries, Narrow pulse pressure, 3rd heart sound, Diffuse Apical impulse and Lung crepitation)**
4- **Signs of tissue damage(fever)**
5- **Signs of complication(Mitral regurgitation and Pericarditis)**

Complications

1- **Arrhythmias**(heart block ,Ventricular fibrillation)
2- **Murmurs**(mitral regurgitation)
3- **Acute circulatory failure**
4- **Heart failure and pulmonary oedema**
5- **Myocardial or septal rupture**
6- Cardiac tamponade
7- **Embolism** from endocardial thrombus
8- **Ventricular aneurysm**

Investigation

1- ECG(Electrocardiogram); Occasionally initially normal and diagnostic changes appear a few hours later.

1- **ST- elevation(within few minutes)**
2- R- wave diminished later on or loss
3- Q- wave developed(within hours)
4- Q- wave is established with T- wave inversion(houre→ few days)
5- **T- inversion persist after ST- segment retune to normal (for few weeks→months, i.e. old infarction)**

2-Plasma enzymes

A- **Troponin-T&I**; Released within 4-6hours and remain elevated for 2weeks.
B- **CK**(Creatine Kinase) and **CK-MB**(Massive assay); Rise at 4-6hours→peak 12hours→ 2-3days fall to normal.

C- LDH(Lactate dehydrogenase); Rise 12hours after MI→2-3days→return to normal for weeks.

D- AST (Aspartate amino-transferase); Rise 12hours after MI→ return to normal 3-4days.

3-Blood test

a- Leucocytosis **b-↑ESR** **c-↑ CRP(C-reactive protein)**

4-Chest X –ray; Demonstrate pulmonary oedema and cardiomegaly.

5-Echocardiography; To assess Lt. & Rt. Ventricular function, and to detect Important complications(mural thrombus, cardiac rupture, etc..).

Management:

(A)- Early management

1- Admission to ICU(intensive care unit)

2- Provide facilities for defibrillation(DC- shock)

3- Immediate measures;

- **High-flow oxygen**
- **I.V. access**
- **ECG monitoring and 12-lead ECG**
- **I.V. analgesia**(Opiate); Morphin sulphate 5-10mg
- **Antiemetic**(Metoclopramide 10mg i.v.) avoid i.m.
- **Aspirin** 300mg

4- Reperfusion;

A- Coronary thrombolytic drugs(↓hospital mortality of MI by 25-50% & survival to 10years especially if given 12hours of Onset of symptoms.

–Streptokinase (1.5 million units in 100ml of saline i.v. over one hour given within 1st few hours) S/E; bleeding, hypotension and serious allergy to drug.

_ Alteplase(15mg bolus dose, followed By 0.75mg/Kg. B. wt. over 30 minutes,then 0.5mg/Kg over 60 minutes.

_ Tenecteplase and **Reteplase**

C- PCI(Primary percutaneous. intervention (coronaryangioplasty)

Done within 3-6hours of onset of MI especially fo Cardiogenic shock .

5- **Aspirin;** 1st dose 300mg given within12hours→maintenance dose ****Clopidogrel** (75mg) accompanied with aspirin within 12hours .
6- **Anticoagulants; Heparin**(12500 units twice daily i.v.) should be given for 2-3 days following thrombolytic drugs. Sometimes give oral **Warfarin** if there is persistent atrial fibrillation or extensive anterior infarction.
7- **Adjunctive therapy; --- β-blockers** (Atenolol i.v. 5-10mg or Metoprolol 5-15mg over 5 minutes).then chronic oral β-blockers.
---Nitrate(sublingual GTN 300-500µg or i.v.nitrate).
8- **Detect and manage acute complications**; Arrhythmias, Ischemia and HF.

*****If there are no complications, the patient can be mobilized from the 2nd day and discharged from hospital on fifth or sixth day.**

(B)- Late management

1- **Lifestyle modification:--Stop smoking, --Regular exercise, --Diet(weight Control and lipid lowering)**
2- **Secondary prevention drug therapy:**
   - **Antiplatelat therapy(aspirin& or clopidogril for 4 weeks.**
   - **β-blockers.**
   - **ACE inhibitors**(captopril, enalapril etc..).
   - **Statin**(Atrovastatin 80mg)lipid lowering drug.
   - **Additional drug for control D.M. and HT.**

Prognosis

****Prognosis worse for anterior than inferior infarction**

- 25% of MI cases,death occurs within few minutes of onset
- Half the death from MI occurs within 24hours of onset
- 40% of MI cases,die within the 1st month

Of those who survive an acute attack

- >80% live for a further year
- 75% live for 5 years
- 50% live for 10 years
- 25% live for 20 years

Heart Failure

Definition: It is a state that develops when the heart cannot maintain an adequate cardiac output.

**Mildest form of HF ; Cardiac output is adequate at rest but inadequate when metabolic demand is increased(during exercise and other form of stress.

**Almost all forms of heart disease can lead to HF.

Causes of HF

1- **↓ventricular contractility;** like **MI.**
2- **Obstruction of ventricular outflow; Arterial Hypertension, Aortic and Pulmonary valves stenosis , Pulmonary Hypertension.**
3- **Obstruction of ventricular inflow; Mitral and tricuspid stenosis.**
4- **Ventricular volume overload; Mitral or Aortic regurgitation, Ventricular or atrial septal defect ,↑ Metabolic demand.**
5- **Arrhythmia;** Atrial fibrillation, Complete heart block.
6- **Diastolic dysfunction; Lt. ventricular hypertrophy**, Constrictive pericarditis, Cardiac tamponade, Restrictive

Pathophysiology

↑B.P. & cardiac output ← Myocyte loss → ↑B.P. & cardiac output

↑ ↓ ↑

↑ Afterload Heart failure (↑ Preload)

↑ (i.e. ↓ cardiac 0utput) ↑ Intravascular volume

↑ ↓ ↑

Vasoconstrictor ← Neurohormonal activity → Na & H_2O retention

(Renin-Angiotensine- Aldosterone system)
(ADH, Endotheline)

- The onset of pulmonary and peripheral oedema is caused by ↑atril pressure due to Na & H_2O retention due to impaired renal perfusion and secondary hyperaldosteronism.

Types of HF

1- **Left sided HF**; ↓ in Lt. ventricular output and or ↑Lt. atrial or pulmonary venous pressure.
2- **Right sided HF;** ↓ in Rt. Ventricular output for any given Rt. Atrial pressure or ↑ Pulmonary artery resistance (Cor-pulmonale), its causes chronic lung disease, pulmonary embolism and pulmonary valve stenosis.
3- **Biventricular HF;** Because the heart disease process is progressed(ischemic heart disease) lead to Lt. ventricular failure →↑Lt. atrial pressure → Pulmonary hypertension and Rt. Heart failure.

** **Acute HF**(MI)

****Chronic HF**(As progressive valvular heart disease)

****Forward HF;** Cardiac output is inadequate

** **Backward HF;** Cardiac output is normal or near normal but the problem is marked Na & H_2O retention causing pulmonary &systemic venous congestion.

Clinical assessment

A- Acute Lt. HF

-**Dyspnoea**; -Sudden onset at rest and rapidly progressed acute respiratory distress.

-**Orthopnoea** (**dyspnoea during lying down due to ↑venous Return**).

-**Prostration**

-**Agitation, pale, clammy, cold peripheries**

On examination

-Rapid pulse ,↑blood pressure or may be low in cardiogenic shock.

-↑Jugular venous pressure especially if accompanied with Rt. side HF.

-Basal lung crepitation and **murmur** of valve or septal rupture.

**Acute on chronic HF will have additional feature of long standing HF.

B- Chronic HF

Commonly experience of relapsing and remitting course and the features depend on the nature of underlying cause (heart disease),neural, endocrinal changes and types of HF.

-Low cardiac output→ Fatigue and weakness(blood diverted away from skeletal muscle).

→ **Listlessness and poor effort tolerance**

→ **↓blood pressure and Cold peripheries**

→ **Oliguria and uremia**(due to ↓renal perfusion)

→ **Weight loss-cardiac cachaxia-**(due to anorexia & gastrointestinal congestion→ ↓absorption)

-pulmonary oedema due to Lt. HF; present with;

-**Dyspnoea, Orthopnoea, Paroxysmal nocturnal dyspnoea**

(dyspnoea appear after 1-2 hours of lying down in bed).

-**Inspiratory crepitation over lung bases.**

- Rt. HF produce;

-↑jugular V. P.

- Hepatic congestion → cyanosis

-Dependent peripheral oedema; (Ankle in ambulant while Sacrum and Thigh in bed).(**Ascites and Pleural effusion in some cases).**

Symptoms (like **dyspnoea**) of chronic HF may first present on **moderately severe exertion**(walking up a steep hill) then progressed to be presented by **lesser exertion**(walking from room to room, washing or dressing), then other symptoms appear like **orthopnoea**, **Paroxysmal Nocturnal Dyspnoea** and **Cheyne-stokes respiration**(slowly diminishing respiration leading to apnoea followed by progressively increasing respiration and hyperventilation accompanied by a sensation of dyspnoea and apnoea during period of hyperventilation).

Complication

1- **Renal failure(Uremia)**; Due to low card. output→poor renal perfusion.
2- **↓K(hypokalaemia);** Due totreatment by diuretic or neuromechanical activation.
3- **↑ K(hyperkalaemia);** Due to renal dysfunction and treatment by ACE and Angiotensine blockers .
4- **↓Na(hyponatraemia);** Due to diuretic therapy and it is a poor prognostic sign.
5- **Impaired liver function;**→Jaundice; Due to hepatic venous congestion and poor arterial perfusion.
6- **Thrombo-embolism;** DVT, Pulmonary embolism ; Due to low cardiac output.
7- **Atria and ventricular arrhythmias;** frequent ventricular ectopic beat, ventricular tachycardia; Due to electrolytes changes.

Investigation

1- **Blood urea**
2- **Serum electrolytes(Na, k)**
3- **Hb**
4- **ECG**

5- **CXR;** show **enlarged hilar vessels**, **prominence upper lobe blood vessel**, **septal or" Kerley B " lines** ,**enlarged cardiac silhouette** and **alveolar oedema.**
6- **Electrocardiography**

Management

Management of acute pulmonary oedema (acute Lt. HF)

1- Site patient up(to reduce pulmonary congenstion)
2- Oxygen(high flow and concentration)
3- Nitrates(GTN i.v. 100-200µg/min. or buccal 2-5mg)
4- Loop diuretic(lasix 50-100mg i.v.)
5- Strict bed rest & monitoring continuously for cardiac rhythm & BP, pulse)
6- I.V.opiates be cautiously used(may cause respiratory depression & hypoxia)
7- Inotropic agent to improve cardiac output or insertion intra aortic ballon pump for those with acute cardiogenic pulmonary oedema 2ry toMI.

Management of chronic HF

1- General measures

- **Education**(explanation of nature of disease, therapy ..).
- **Diet**;*Good general nutrition weight,*Reduction for obese,*Avoid high salt.
- **Alcohol to be eliminated**.
- **Smoking to be stopped.**
- **Exercise;** regular moderate exercise within limits symptoms.
- **Vaccination;** Influenza & Pneumococcal .

2- Drug therapy

1- **Diuretic**;1st line of treatment (Bendroflumethiazide 5mg/day).

2- **Vasodilator**; **--Nitrate**→ ↓Preload & arterial dilatation.

--Hydralizine→ ↓Afterload.

3- **ACE inhibitor**; ↓Preload & afterload & modest ↑K^+(Captopril,Lansopril,..).

4- **Angiotensin receptor blocker** (losartan 50-100mg once daily).
5- **β- blocker** (Bisoprolol 1.25mg/day).
6- **Antiarrhythmic drug**(Amiodarone).

7- Digoxin; 1st line therapy for HF & atrial fibrillation

Angiotesinogen

↓ ←renin← **Kidney** **Bradykinin→** vasodilatation

Angiotensin I ↑

↓ ←←←←Angiotensin converting enzyme →→→↑

↓ (Inhibited by **ACE inhibitors**)

Angiotensin II

↓

↓

↓←←→→→→→→↓→→→→Enhance sympathetic activity ← ←**β-blocker**

↓ Vasoconstriction

Aldosterone

↓←←←Spironolacton(aldosteron receptor inhibitor)

Na & water retention←←**Diuretic**

8- Implantable cardiac defibrillators & resynchronization therapy for ventricular arrhythmias.

9- Revascularisation; **---Coronary artery Bypass surgery.**
---Coronary angioplasty(PCTA).

10- Heart transplantation.

Hypertension

Definition :- The level of blood pressure at which the benefit of treatment outweigh the costs & hazards .

-Or defined as an increase of blood pressure above the normal .

Types of systemic hypertension

1/ Primary or essential hypertension (more than 95%)

Its causes related to **environmental** & (40-60%) of **genetic factors** (more common in black people)

* Different investigators have proposed :

The kidney , the peripheral resistance vessel & the Sympathetic nervous systems as the seat of the primary abnormality .

* Environmental factors include :-

1- **High salt intake, Obesity .**
2- **Heavy alcohol consumption .**
3- **Lack of exercise, Stress .**
4- **Impaired intrauterine growth .**

2/ Secondary hypertension (about 5%)

It can be a consequence of a specific disease or abnormally leading to Na retention and or peripheral vasoconstriction :-

1- **Alcohol .**
2- **Obesity .**
3- **Pregnancy (pre-eclampsia)**
4- **Renal disease** : **polycystic kidney disease** , parenchymal renal disease (glomerulonephritis) & renal vascular disease.
5- **Endocrine disease**
   - **Pheochromocytoma** , thyrotoxicosis , Acromegaly,
   - **Cushing's syndrome** .
   - **Conn's syndrome** (1ry hyperaldosteronism)
6- **drugs**
   - **Oral contraceptive containing estrogen**
   - **Anabolic steroid , corticosteroid** .

- **NSAIDs** .

- Sympathomimetic agents .

7- coarctation of the aorta .

Clinical features

1. **mostly Asymptomatic** if there is no complication **but careful history** is important like : **family history , life style** (exercise , salt intake , smoking habit) drug history or risk factors should be record
2. **Headache .**
3. **features of Complications.**

On examination

If there is complication or causes of hypertension then we can detect :-

1- radio – femoral delay (coartation of aorta).
2- Enlarged Kidney (polycystic Kidney disease) .
3- Abdominal bruit (renal artery stenosis) .
4- Characteristic facies & habits of cushing's syndrome .
5- Central obesity &hyperlipidaemia .
6- Non-specific finding :

- LV hypertrophy

-4^{th} heart sound

-Optic fundi are often abnormal

-Generalized atheroma .

-Aortic aneurysm or peripheral vascular disease .

N.B.: The patient is said to be hypertensive when more than two successive reading are above normal & at resting state .

Complication of hypertension (Target organ damage)

1/ Blood vessels

- **Large arteries (over 1mm in diameter) dilate & tortuous & less compliant .**
- **Smaller arteries (less than 1mm) Hyaline arteriosclerosis** .
- **Wide spread atheroma coronary or cerebral vessel disease** .

2/ Central nervous system

- **Stroke** (due to cerebral hemorrhage or infarction)
- **Carotid atheroma & transient cerebral ischemic attack** .
- Subarachnoid haemorrhage **.**
- **Papilloedema (common) .**
- **Hypertensive encephalopathy (rare) .**

3/ Retina : (hypertensive retinopathy)

- cotton – wool exudates & hard exudates associated with **retinal ischaemia or infarction**
- **central retinal vein thrombosis**

4/ Heart :

- **LV hypertrophy**
- **4th heart sound**
- **Forceful apex beat .**
- **A trial fibrillation .**
- **Heart failure (Lt. side)**

5/ Kidney :

- **proteinuria .**
- **progressive renal failure .**

6/ Malignant or accelerated phase hypertensive(**rare condition**) characterized by accelerated micro vascular damage with necrosis in small arteries & arterioles wall (fibrinoid necrosis) & intravascular thrombosis .

Investigations

a- For all patient :

- **Urinalysis; for blood , protein & glucose** .
- **Blood urea , S. creatinine & S. electrolytes (Renal function test).**
- **Blood glucose .**
- **Lipid profile test .**
- **ECG (detect LV. Hypertrophy & coronary artery disease)**

b- For Selected patient :

- **C. X. R.** (detect cardiomegaly , HF. , ….)

- **Ambulatory Bl. Pr. recording** (assess borderline or white coat HT).
- **Echocardiography(**detect quantify LV hypertrophy)
- **Renal ultrasound** (detect renal disease)
- **Renal angiography** (detect renal artery stenosis)
- Urinary catecholamim (detect pheochromeocytoma)
- Urinary cortisol & dexamethasone (detect Cushing's syndrome)
- Plasma renin activity&aldosterone (detect1ry hyperaldosteronims).

Management

The aim :- - to reduce the risk of compilations .

- to improve survival of patient .

A- General measures

1/Diet – reduce alcohol .

- correcting obesity .

- very low salt diet

2/ Risk factor modification

- **Smoking should be strongly discouraged .**
- **Energetic treatment for hyperlipidaemia .**

3/ Exercise and relaxation ; Regular exercise to improve physical fitness.

B- Antihypertensive drug

**the choice of drugs will be determined; safety, convenience, freedom of side effect.May be single or combination of 2,3 or more .

1- **Diuretic :** Thiazide (S/E :- impotence , but good for HF.) .
2- **B-blocker :** (Lipid soluble) Propranalol across BBB→ bad dream, (water soluble) Atenolol. **Labetalol for malignant HT** (S/E : cold extremeties , but good for angina branchospams) .
3- **ACE(angiotensin – converting enzyme) inhibitors** :
- good for HF, but should be careful with those with renal failure & renal artery stenosis & may cause proteinuria , cough , rash & unpleasant metalic taste .(Captopril enalapril,lansipril) .

4- **Ca++ antagonist :** S/E palpitation , flushing face & fluid retention (Nifidipin,Amlodipib, verapamel).
5- **Some vasodilator :**
- Prazosin (α□ - antagonist)

-Hydralazine & minoxidil(S/E;↑hair growth)

-Methyldopal (centrally acting drug)

The emergency treatment of accelerated phase of hypertension

It is unwise to lower Bl.Pr. too quickly because this may compromise tissue perfusion (due to altered auto regulation) & so can cause cerebral damage including occipital blindness & precipitate coronary & renal insufficiency .

- The controlled reduction to level of about 150/90mmHg over a period of 24-36 hours is ideal .

* **I.V. Labetalol** 2mg/min to maximum 200mg

* **I.V. Nitroglyccim** (0.6-1.2 mg/hour)

* **Hydralazine** (5-10 mg repeated half hourly intervals)

***I.V. Na-nitroprusside** (0.3-1.0 mg/kg/min) .

Congenital heart disease

CHD usually manifests in childhood but may pass unrecognized and not present until adult life.

Aetiology and incidence

The incidence of haemodynamically significant CHD is about 0.8%.

Lesion	% of CHD defect
1- Ventricular septal defect	**30**
2- Atrial septal defect	**10**
3- Patent ductus arteriosus	**10**
4- Pulmonary stenosis	7
5- Coarctation of aorta	7
6- Aortic stenosis	6
7- Tetralogy of Fallot	6
8- Complete transposition of great arteries	4
9- Others	20

Causes

1- **Maternal infection**(Rubella infection) or exposure to drug or toxins.

2- **Maternal alcohol misuse.**
3- **Maternal Lupus erythematosus.**
4- **Genetic or chromosomal abnormalities**(Down's Syndrome).

Clinical features

1- **Symptoms may be absent.**
2- **Central cyanosis and digital clubbing.**
3- **Breathlessness.**
4- **Growth retardation and learning difficulties.**
5- **Murmurs, thrills or signs of cardiomegaly.**
6- **Radio-femoral delay of pulsation.**
7- **CVA and cerebral abscesses.**
8- **Arrhythmias and syncope.**
9- **Pulmonary hypertension with its changes appear on chest X-Ray and ECG.**

Persistent ductus arteriosus

When the ductus fail to close soon after birth, which is a connection between main pulmonary artery and aorta.

**More common in females.

Since pressure in aorta is a higher than that in pulmonary lead to continuous arteriovenous shunt(Lt. to Rt. Shunt)** and 50% of Lt. ventricular output may be recirculated to lungs with a consequent increase in heart work.

Clinical features

1- **Small shunt(ductus)→ No symptoms for years.**
2- **Large shunt(ductus)→Growth and development retardation.**
3- Dyspnoea
4- No disability but heart failure eventually ensue.
5- **A continuous machinery murmur (&thrill);** maximal heard in the aortic area.
6- ↑ Pulse volume.
7- Chest X-Ray; Enlarged pulmonary artery (pulmonary hypertension).
8- ECG ; Normal or Rt. Ventricular hypertrophy.
9- **Central cyanosis; More in feet and toes than upper body, this occurs when pulmonary artery pressure exceeds aortic pressure(Rt. to Lt. shunt).**

Management

1- Surgical intervention.
2- Cardiac Catheterisation with an implantable occlusion device done in infancy but may delayed until later childhood.
3- Medical therapy; Indomethacine or Ibuprofen in 1st week of life.
4- Long acting penicillin to prevent endocarditi

Coarctation of aorta

**Narrowing of the aorta most commonly occurs in the region where the ductus arteriosus joins the aorta(at isthmus just below origin of subclavian artery).

**More common in males 2:1 and occurs in1 in 4000 childrenand it is associated with other abnormalities(bicuspid aortic valve&berry aneurysm of cerebral vessel)

**Sometimes acquired coarctation follow trauma or progressive arteritis.

Clinical features

1- Symptoms are often absent when detected in older children or adults.
2- Important cause heart failure in newborn.
3- **Headache due to hypertension proximal to narrowing.**
4- Weakness or cramps in legs due to ↓circulation in lower part of body.
5- **↑Blood pressure in upper body but ↓ or normal in legs.**
6- **Radio-femoral delay of pulsation.**
7- Systolic murmur over coarctation posteriorly.
8- Dilated and tortuous collateral arteries formed in periscapular,internal mammary and intercostals arteries→ bruits.

Investigation

1- Chest X –Ray; Indentation of descending aorta″3 sign″ and notching of ribs inferiorly due to collateral (these changes appear at later age).
2- ECG ;Lt. ventricular hypertrophy.
3- MRI.

Management

1- Surgical correction in early childhood.

2- Treat hypertension.
3- Follow up for long term due coexist other abnormalities.

Atrial septal defect

**Females to males 2:1.

Large volume of blood shunts through defect (persistent open foramen ovale) from Lt. to Rt.atrium(Lt. to Rt. shunt)**, lead gradual **enlargement of Rt. side of heart and pulmonary hypertension** may complicate atrial septal defect.

Clinical features

1- **Most children are free of symptoms for years and detected by routine examination.**
2- Other presentation; Dyspnoea, chest infection, cardiac failure & arrhythmias.

Physical signs;

1- Wide fixed splitting of second heart sound.
2- Systolic flow murmur over pulmonary valve.
3- Diastolic flow murmur over tricuspid valve in children with a large shunt.

Investigation

1- Chest X-Ray; Enlarged heart and pulmonary arteries.
2- ECG; Incomplete Rt. bundle branch block.
3- Echocardiography; Rt. ventricular dilatation and hypertrophy and pulmonary arteries dilatation.
4- Transoesophageal echocardiography; Show size and location of defect.

Management

Closed surgically or at cardiac catheterization using implantable closure devices.

**** Sever pulmonary hypertension & shunt reversal are contraindication to surgery.**

Ventricular septal defect

Aetiology

Due to incomplete septation of ventricle.

**It is the most common congenital cardiac defect, occurs 1:500 live birth.

**Sometimes is acquired from rupture as a complication of MI and from trauma.

Clinical features

1- **Plan systolic murmur**(Small defect → Loud murmur)

(Large defect→ Soft murmur)

2- Cardiac failure in infant.
3- **Prominent parasternal pulsation .**
4- Tachycardia and in drawing of lower ribs on respiration.

Investigation

1- Chest X-Ray; Pulmonary plethora(congestion).
2- ECG; Bilateral ventricular hypertrophy.
3- Doppler Echocardiography; Detect small defect.

Management

1- Spontaneous closure if small .
2- Cardiac failure initially treated with digoxin & Diuretic.
3- Surgical repair for persisting cardiac failure.

Tetralogy of Fallot

It is composed of 4 components

1- **Pulmonary stenosis**
2- **Ventricular septal defect**
3- **Rt. ventricular hypertrophy**
4- **Overriding aorta**

Clinical features

1- **Cyanosis in children not in neonate; ↑Cyanosis after feeding or crying associated with apnoeic or unconscious(fallot's spells).**
2- Stunting (retard) of growth.

3- Digital clubbing.
4- Polycythemia
5- Loud ejection systolic in pulmonary area.

Investigation

1- ECG; Rt. ventricular hypertrophy.
2- Chest X-Ray; Small pulmonary artery and boot shaped heart.
3- **Echocardiography; Is diagnostic.**

Management

Total correction of defect by surgery before age of 5.

"Oropharynx"

Tonsillitis

Tonsil; lymphatic distributed tissue situate at entrance of pharynx which is composed of crypts &pyramids .

Acute tonsillitis

Aetiology

*Half the cause are bacterial "pyogenic group A streptococcus

*the remainder are viral "infectious mononucleosis

*predisposing factors ;-cold weather, cold drinks, dryness of mouth ,

mouth &teeth infection, bad mouth hygiene.

Clinical feature

Incubation period: 36-48 hours

1-sore throat

2-Fever(sometimes with rigor)&general malaise & arthralgia

3-dysphagia

On Examination

1- swollen, erythematous &yellow or white pustules has been seen on palatine tonsils "follicular tonsillitis "

2- some times we see Quinzy "abscess in the peritonsillar region "that cause severe pain & trismus

3- bad smell

4- enlarged tonsils and secondary enlargement of cervical lymph nodes

Investigation

A throat swab should be taken at the time of examination

Treatment

1-Analgesia ;Aspirin or paracetamol

2-Gargles of glycerol-thymol are soothing

3-Benzyl or phenyoxy methyl penicillin (**penicillin V**) these given until antibiotic sensitivities are established

Quinsy : -an **abscess in peritonsillar region** cause severe pain &trismus

-inspection reveals a diffuse swelling of soft palate ,displacing uvula medially and sometimes pus underneath mucosa

Treated by *I.V .broad –spectrum antibiotic

*Or may need to incision & drainage of pus under local anesthesia and in children need general anesthesia.

Chronic tonsillitis

It result from repeated attacks of acute tonsillitis in which the tonsils become progressively damaged &provide a reservoir for infective organisms .

-Common in children, adolescents & young adults

Treatment

Tonsillectomy under general anesthesia

-**"Ideally should done when tonsils are not acutely infected "**

-Following surgery the patient is kept under close observation for any bleeding systemically or locally

Absolute indications of operation are ;

1-Enlarged tonsil contributing to air way obstruction

2-Suspected tonsillar malignancy

Relative indications

1-Recurrent acute tonsillitis especilly lead to convulsion

2-Chronic tonsillitis

3- Quinsy

4-Systemic disease caused by B-haemolytic streptococcus "nephritis, rheumatic fever".

Obstruction of larynx and trachea

Obstruction of larynx

Aetiology

1- **Impaction of foreign body;** Mostly in children(due to smaller size of glottis) as a piece of food, bone, seeds, coin, toy, (denture in adult)**.**
2- **Infections;** as diphtheria, viruses, influenza(moostly cause acute epiclottis).
3- **Bilateral vocal cord paralysis** after operation (Thyroidectomy)**.**
4- **Allergy**(Angioneurotic oedema).
5- **Irradiation.**
6- **Irritants** as corrosives, scalds and noxious gases.
7- **Trauma** as car accident.
8- **Tumours** as enlarged metastatic mediastinal lymph nodes from bronchial cancer.
9- **Retrosternal goiter**.

Signs and symptoms

1- Hoarseness of voice or whisper.
2- Dyspnoea.
3- Paradoxical respiration.
4- Stridor.

Diagnosis and investigation

1- **Laryngoscopy**; To determinate the size, degree & nature of obstruction.
2- **Chest X- ray.**

Treatment

1- **Foreign body**
   A- Direct removl or through laryngoscopy.

   B- Turning the patient head upside down & Squeezing the chest vigorously.IN adult a sudden forceful compression of upper abdomen (Heimlich maneuver) be effective.

   C- Tracheostomy.

2- **Inflammatory condition, allergic cause, irritation**
   A- Steam inhalation.
   B- Adrenalin spray or epinephrine 0.5-1mg i.m.
   C- Antibiotic.
   D- Anti-inflammatory drugs(Hydrocortisone).
   E- Anti-toxin in case of diphtheria.
   F- Anti- allergic drugs in case of allergy(Chlorphenarmine maleate 10-20mg slowly i.v.).
   G- Oxygen,
   H- Intubation and tracheostomy may be needed**.**
3- **Bilateral vocal cord paralysis treated by tracheostomy for life.**
4- **Trauma treated by intubation & tracheostomy.**
5- **Tumour.**
   A- Surgery (localized tumour can be resected).
   B- Tracheostomy.
   C- Deep radiotherapy.
   D- Chemotherapy.

Tracheal-bronchial obstruction

****Mostly in children.**

**** mostly in right side bronchus(due to more vertical).**

Causes

1- **Extraluminal**
   A- Enlarged thyroid due to any causes.
   B- Aortic aneurysm.
   C- Enlarged metastatic mediastinal lymph nodes from bronchial cancer.
2- **Intramural**
   A- Tuberculous mediastinal lymph nodes.
   B- Post tracheostomy fibrosis and stenosis.
   C- Tumours.
3- **Intraluminal**
   A- Foreign body.

B- Tumours.

Clinical features

1- **Wheezing& stridor.**
2- **Irritating cough.**
3- **Signs of unilateral obstructive emphysema.**
4- **Atelectasis & suppuration (cough, sputum & fever)**

Investigation

1- **Chest X-ray**
2- **Endoscopy (Bronchoscopy).**

Treatment

1- **Early bronchoscopy and removal of foreign body .**
2- **Localized tumours of trachea can be resected.**
3- **Benign tracheal stricture can be dilated but may have to be resected.**
4- **Endobronchial laser therapy.**
5- **Bronchoscopically placed tracheal stent.**
6- **Chemo radiotherapy are alternative to surgery.**

Peumonia

Defined as an acute respiratory illness associated with recently developed radiological pulmonary shadowing which may be segmental, lobar, multilobar.

Types

1- **Community- acquired pneumonia(CAP).**
2- **Hospital- acquired pneumonia(Nosocomial).**
3- **Supportive and aspirational pneumonia**(destruction of lung parenchyma lead to pulmonary abscess.
4- **Pneumonia in the immunocompromised patients**(those with cellular & humoral immune mechanism defect receiving immunosuppressive drugs, like acute leukaemia, lymphoma& multiple myeloma).

Community- acquired pneumonia(CAP)

**Pneumonia account for almost 1/5th of childhood deaths world wide, with approximately 2 million children under 5 dying each year.

**Most patients may be safely managed at home, but hospital admission is necessary in 20-40% of patients.

CAP spread by **droplet infection and most cases occur in previously healthy individuals.

Factors predispose to pneumonia(Impair the effectiveness f local defence)

1- **Cigarette smoking.**
2- **Upper respiratory tract infection.**
3- **Alcohol.**
4- **Corticosteroid therapy.**
5- **Old age.**
6- **Recent influenza.**
7- **Pre-existing lung disease.**

Once the organisms settle in the alveoli an inflammatory response ensues.

The majority of CAP cases are due to infection with **streptococcal pneumonia**.

The main microorganisms of causing Pneumonia

1- **Strept. Pneumoniae**.
2- **Mycoplasma & Chlamydia Pneumoniae** are common in young adult.
3- **Haemophilus influenza** are common in the elderly.
4- **Viral infections** predominante in young children.
5- **Logionella pneumophilia** in patiens with history of foreign travel.
6- **Staph. Aureu**s in patient with history of recent influenza.

Common microorganism;Strep. Pn. , Chlamydia & Mycoplasma Pn. ,Log. Pn. .

Uncommon microorganism;**;** Haemophilus infl. , Staph. Au. , Chlamydia psittaci,1ry viral pn.(measles,influenza. Parainfluenza, varicella,respiratort syncytial virus seen in infancy.

Clinical features

It presents as an **acute illness** in which:

1- **Systemic features**
   - **A- fever, rigor, shivering, herpes labialis, dry hot skin &flush face.**
   - **B- Vomiting after predominant headache & loss of appetite.**
2- **Pulmonary symptoms**
   - **A- Cough,** in the **beginning** short, painful& **dry but later** accompanied by expectorated of **mucopurulent sputum(rust-colored in strept.Pn.).**
   - **B- Haemoptysis** is occasionally.
   - **C- Pleuritic chest pain** may be referred to shoulder or anterior abdomen.
   - **D- Upper abdominal tenderness** (lower lobe pn. With hepatitis.
   - **E- Rapid shallow breathing→ cyanosis.**

** Less typical presentation may be seen in very young and elderly.

Physical signs

1- **↓respiratory movement.**
2- **On percussion : dullness (stony dullness in pl. effusion without breathing sound).**
3- **On auscultation: Bronchial breathing, coarse crepitation, & pleural rub.**

Investigation

The aims

a. To obtain radiological confirmation of diagnosis.
b. To exclude other conditions.
c. To obtain microbiological diagnosis.
d. To identify the development of complications.

1- **Chest X ray**; A **homogeneous opacity** localized to the affected lobe or segment usually appears within12-18 hours from onset of disease.
   Also we can assess the severity and complications as **empyema** & **intrapulmonary abscess.**
2- **Microbiological investigations**
   - **A- Sputum; Direct smear & culture with drug sensitivity.**
   - **B- Serological titers** to detect mycop. chlam. & pneumo.antigen.
   - **C- Throat, nasopharyngeal swabs(**In children or influenza epidemic).

D- Pleural fluid sample with ultrasound guidance.

3- General blood tests

A- ↑WBC.-↑Neutrophil >15 x10^9/l bacterial cause .>20x10^9/l→sever pn.

B- Urea, electrolytes & liver function test.

C- ↑C-reactive protein.

Complication of pneumonia

1- **Para pneumonic effusion(common).**
2- **Empyema(accumulation of pus in pleural cavity).**
3- **Retention of sputum causing lobar pneumonia.**
4- **Development of thromboembolic diseases.**
5- **Pneumothorax (staph. Au.).**
6- **Supportive pn. , lung abscess.**
7- **Ectopic abscess, renal failure & multiorgan failure.**
8- **Hepatitis, pericarditis, myocarditis.**
9- **Pyrexia due to drug hypersensitivity.**

Differential diagnosis

1- **Pulmonary infarction**(No fever, no response to antibiotic, cough is not common & haemoptysis is common).
2- **Tuberculous pleurisy & effusion.**
3- **Pulmonary tuberculosis.**
4- **Pulmonary oedema.**

Management

A- Rest & avoidance of smoking.

B- Attention is paid to ;

1- **Oxygenation;** Given to all patients with tachypnoea, hypoxemia, hypotension and acidosis, with aim to maintain **PaO_2=60mm Hg**, and **high concentration>35% &humidified**. May need ventilation if hypoxaemia remain.
2- **Fluid balance;** Adequate oral intake of fluid should be encouraged but I.V. fluid should be considered in those with severe illness, elderly & recurrent vomiting.
3- **Antibiotic(it is favored to do culture specimen initially).**

***Uncomplicated**

Amoxilline 500mg 8hourly for 7-10 days.

If allergy give **clarithromycine** 500mg 12 hourly or erythrmycine 500mg 6hourly.

If staph. Au.→ Flucloxacillin 1-2gm 6hourly I.V.+ clarithromycin.

If mycoplasm & log.→ Clarithromycin 500mg 12hourly(orally or I.V.) or

Erythromycin 500mg 6hourly(orally or I.V.).

Rifampicin 600mg 12hourly I.V. in severe cases.

***Complicated and severe**

Clarithromycin 500mg 12hourly I.V. or

Erythromycin12 hourly I,V.**+ Clarithromycin.**

Co-amoxiclav 1.2gm /day I.V. or

Ceftriaxone 1-2gm/day I.V. or

Cefuroxime 1.5gm8hourly I.V. or

Amoxicillin 1gm 6hourly **+ flucloxacillin** 2gm 6 hourly I.V.

C- Treatment of pleural pain;
To allow the patient to breath normally and cough efficiently by mild analgesic (paracetamol) but rarely adequate so opiate must be used with extreme caution in patient with poor respiratory function.

D- Physiotherapy.
**Fever for several days & chest X ray take several weeks or months to resolve especially in elderly.

Diseases of pleura

Pleurisy

**It is a result (term)of any disease process involving the pleura and giving rise to pleuritic pain or evidence of pleural friction.
**It is a common feature of pulmonary infection and infarction and also occur in malignancy.

Clinical features

1- **Pleural pain** .
2- **Clinical features of underlying causes.**
3- **On examination**;- **The rib movement is restricted** and
- **pleural rub** may be heard in deep inspiration or near the pericardium (pleura pericardial rub).

** Loss of pleural rub and diminution in the chest pain may indicate recovery or herald the development of a pleural effusion.

Every patient should have a **chest X ray, but normal chest X ray does not exclude a pulmonary causes of pleurisy.

Management

The primary causes of pleurisy must be treated.

Pleural effusion

It is an accumulation of serous fluid within the pleural space.

Accumulation of frank pus = Empyema

Accumulation of blood = Haemothorax(due to injuries, malignancy, pleural infarction and traumatic tap).

In general pleural fluid accumulate as a result of :

Either transdative effusion due to ↑hydrostatic pressure or ↓osmotic pressure as seen in cardiac, hepatic or renal failure.

Or exudative effusion due to ↑micro vascular pressure resulting from disease of pleural surface or injury in adjacent lung.

Clinical features

1- **Signs and symptoms of pleurisy**; Often precede the development of effusion especially in patient with underlying pneumonia, pulmonary Infarction or connective tissue disease.
2- **The onset may be insidious**.
3- **Breathlessness** is the only symptom related to effusion and its severity depend on size and rate of accumulation .
4- **Signs and symptoms of underlying causes.**
5- **Physical signs includes;**

A- **Inspection→ Tachypnoea.**
B- **Palpation→↓Expansion on Rt. lung& trachea with apex moved to Lt.**
C- **Percussion→ Stony dullness in Rt. mid & lower zone.**
D- **Auscultation→ Absent breath sounds, vocal resonance in Rt. base and crack above effusion.**

Investigation

1- **Chest X ray;** On erect PA chest film is a curved shado at lung base blunting the costophrenic angle and ascending towards the axilla. Around 200cc of fluid is required to be detected on PA chest film
2- **Ulrasound or CT- scanning** to identify smaller size of fluid& display benign from malignant.

3- **Safe needle aspiration & guides pleural biopsy;**To establish the diagnosis for colour, texture of fluid, biochemical analysis, cytology & bacteriology.
4- **Video- assisted thoracoscopy give the best result.**

Management

1- **Therapeutic aspiration** may be required to palliate breathlessness but **removing more than 1.5 liter in one episode is inadvisable** as there is a small risk of re-expantion pulmonary oedema. So effusion should never be drained to dryness before establishing a diagnosis as further biopsy.
2- **Treatment of underlying causes.**

Asthma

It is characterized by chronic airway inflammation and increasing airway hyper-responsiveness leading to symptoms of wheeze, cough, chest tightness and dyspnoea.

Epidemiology

**Estimation of 300 million people world-wide suffer from asthma and an additional 100 million may be diagnosed by 2025.

**Inn childhood is more common especially in boys but following puberty females are more common.

Aetiology

It is a complex and multiple environmental and genetic determinants are implicated.

Clinical features

Asthma is of two type

1- **Early onset asthma**(appeare in **childhood**,in **atopic** individual & antigen is extrinsic).
2- **Late(adult) onset**(non atopic)asthma(affect adult and antigen(allergen) is intrinsic).

Typical symptoms

1- **Recurrent episodes of wheeze, cough, chest tightness and dyspnoea**.The attack may last hours or days.
2- **The attack worse in the early morning and may disturb sleep (nocturnal asthma).**

3- **Precipitated by many factors like**; **Exercise, cold weather, exposure to airborne allergens or pollutant and viral upper respiratory tract infection.**

****Atopic individual & smokers appear to be at ↑risk.**

****Early diagnosis and removal from exposure lead to a significantly improve prognosis→ cure.

****In mild intermittent asthma**; Asthma are usually asymptomatic between an exacerbations which occur during viral respiratory tract infection or after exposure to allergens.

****In persistent asthma**; The pattern is one of chronic wheeze and dyspnoea.

****Cough-variant asthma;** Cough is the dominant symptom with no wheeze & dyspnoea so lead to delayed diagnosis.

****Aspirin-sensitive asthma**; It is often associated with rhinosinsinusitis and nasal polyp.

Occupational asthma; Most common form of occupational respiratory disorder , it form 5% of adult – onset asthma.

****Acute severe asthma(status asthmaticus);**

1- More common in females.
2- All respirator muscles involved
3- Unproductive cough.
4- Pulsus paradoxus.
5- Sweating and central cyanosis in severe cases.
6-Tachycardia.

Physical signs

1- **Rhonchi.& Vesicular breathing sound.**
2- **Prolonged expiration.**
3- **Accessory muscles work.**
4- **Pigeon chest in atopic one may present.**

Investigation

The diagnosis of asthma is made on basis of a compatible clinical history combined withthe demonstration of variable airway obstruction.

1- **Pulmonary function test;↓FEV_1**(forced expiratory volume at 1st second).
2- **Chest X ray**; May be normal or in acute asthma is accompanied by hyperinflation& lobar collapse I f mucus has occluded a large bronchus.
3- **Measurement of allergic states**;
   - **A- An elevated sputum or peripheral blood esinophil count .**
   - **B- An elevated serum total IgE in atopic asthma.**
   - **C- Skin prick tests are simple & provid rapid assessment of atopy**.
4- Assessment of airway inflammation by induced sputum & exhaled breath.

Management

The goals of the management of asthma are

1- Achieve and maintain control of symptoms.
2- Prevent asthmatic exacerbation.
3- Maintain pulmonary function as close to normal as possible.
4- Avoid adverse effects from asthma medications.
5- Prevent development of irreversible airflow limitation.
6- Prevent asthma mortality.

A- Patient education;

1- Encouraging patient to take responsibility for control this Disease.

2-Revisited in subsequent consultation.

B- Avoidance of aggravating factors.

C- A stepwise approachs to the management of asthma;

Step 1: Occasional use of inhaled short-acting β2-agonist**(inhaled SABA**) **bronchodilator-salbutamol-**(for mild intermittent asthma).

---------(Inhaled SABAs) ---------

Step 2: Regular anti-inflammatory therapy; inhaled corticosteroid**(ICS)** (400μg beclometasone dipropionate/day)**+ inhaled SABAs .**

----------(Low dose ICS + Inhaled SABAS)---------

Step3 :ICS(800 μg beclometasone dipropionate/day)+**LABAs**(long acting β2-agonist-**salmeterol**-).there is fixed combination inhaler of ICS+LABAs.Also can be added **leukotriene antagonist**(motelukast 10mg/day orally)& **theophyline.**

-Low to moderate doseICS+LABAS+ Leukotriene antagonist+Theophylline--

Step4: Step3 drugs+ Itraconazole + Monoclonal antibodies agonist(IgE).

Step 5:Additon of Continous or frequent use of oral steroids

(prednisolone single dose in the morning).

****Step-down therapy:** Once asthma control is established the dose of inhaled or oral corticosteroid should be titrated to the lowest dose at which effective control of asthma is achived.

Management of mild –Moderat exacerbation

Doubling the dose of ICS, also short courses of (recure) oral coticosteriod

(prednisolon 30-60mg/day tapering is not necessary unless given for more than 3weeks.

Management of acute severe asthma

1- **Oxygen;**High concentration& humidified (maintainO_2 saturation>92%).
2- **SABAs in high dose via nebulizer** driven of multiple dose of salbutamol&O_2. Add **Ipratropium bromide to SABAs**.
3- **Systemic corticosteroid; oral or I.V.** (in case ofvomiting or dysphagia).
4- **I.V. fluid;** Hydration therapy due to excessive sweating during attack.
5- **Potassium supplements may be necessary because repeated dose of sulbutamol can lower serum K.**
6- **Subsequent management;**
   1- **I.V. Mg** may provid addition bronchodilator.
   2- **I.V. Aminophilline** with careful monitoring.
   3- **I.V. Leukotriene Antagonist.**
   4- **Endotracheal tubation & intermittent +ve pressure ventilation.**

Bronchitis

Acute bronchitis

It is an acute inflammation of trachea and bronchi.

Aetiology

1- Virus as influenza.
2- Bacteria as strept.pn., hemophilus influenza& rarely staph. Au. Or bacillus anthracis.The condition is commonly followed course of coryza(common cold), influenza, measles and whooping cough.
3- **Precipitating factors; Cold, damp&dusty atmosphere, cigarette smoking.**

Clinical features

1- **Irritating dry cough.**

2- **Retrosternal discomfort or pain** due to tracheitis.
3- **Tightness of chest & rhonchi** is heard(bronchi are affected).
4- **Dyspnoea & wheezes.**
5- Respiratory distress in presence of chronic bronchitis or emphysema.
6- **Small amount of sputum, sometimes with blood, then becomes copious& mucopurulent.**
7- **Pyrexia.**
8- **Neutrophil leukocytosis .**

**The condition may subside spontaneously within 4-8 days.

****If bronchiolitis established then these features appears;**

Breathlessness, cyanosis& crepitation is heard.

Management

1- **In early stage** when cough is painful & dry ,mucus is tough & viscid**, steam inhalation 3-4times/ day** to losses this viscid mucus.
2- **Cough suppressor**(pholcodeine 5-10mg 6hourly).
3- **Bronchodilators** in case of airflow obstruction(Salbutamol, terbutaline).
4- **Antibiotic** are not always needed.
5- **Oxygen** sometimes is needed.

Chronic bronchitis

Definition; A state of productive cough on most days or at least 3 consecutive months for more than 2 successive years if other causes have been excluded.

Aetiology

1- Chronic irritants; Smoking, dust, fumes.
2- Viral or bacterial infection.
3- Dampness.
4- Sudden change of temperature.
5- Fog.
6- Middle & late adults are more affected.

Pathology

1- Hypertrophy & oedema of mucous membrane.
2- Hyperplasia of goblet cells & other mucous .
3- Damage of ciliated cells.
4- Narrowing of the lumen of bronchi.

Clinical features

1- **Repeated productive cough** which increase steadily in its severity with successive years, then become persistent all the year round.
2- Usually occurs **after cold exposure**(winter month).
3- **Wheezes, dyspnoea &chest tightness mostly in the morning.**
4- **On examination**; **Rhonchi, crepitation may present, & hyper resonant lung if emphysema is accompanied it.**

Investigation

1- **Chest X ray**; May show emphysema.
2- **Pulmonary function tests;**
   - **a-** ↓FEV_1.
   - **b-** ↑Residual volume & total lung capacity.
   - **c-** ↓Vital capacity.
   - **d-** Arterial blood gases measurement ;↓PaO_2 & ↑ PaO_2.
   - **e-** ↓Exercise tolerance test.

Complications

1- Emphysema.
2- Respiratory failure.
3- Secondary polycythemia.
4- Pulmonary hypertension & Rt. ventricular hypertrophy.
5- Cor pulmonale.

Management

1- **Reduction or avoidance of irritants.**
2- **Treatment of infection by antibiotic.**
3- **Bronchodilators.**
4- **Symptomatic treatment**(steam inhalation, warm room& hot drinks).
5- **Oxygen if needed**.
6- **Diuretics for pulmonary oedema.**
7- **Physiotherapy as assisted expectoration, forced expiration.**

Tumours of trachea, bronchi and lungs

**90- 95% are malignant.

****Smoking is the main aetiological factors.**

****Benign tumours**; Carciniod , adenoma, fibroma, chondroma, lipoma and angioma.

Malignant tumours

Primary; Mostly carcinoma(Squamous and adenocarcinoma).The great majority of tumour of lung.

Secondary; Mostly sarcoma, teratoma of testis, carcinoma from breast, bowel, thyroid and kidney.

Carcinoma of lung

****It is the commonest cancer world –wide**, accounting for 1.2 million new cases annually in 2000, causing 18% of all cancer death.

**More in men over age of 40, and more in urban than rural(due to atmospheric pollution.

Aetiology

1- **Smoking(90% of lung carcinoma)**.

2- **Other irritants** as asbestos, radiation, exhaust fumes&chromium.

Common cell types of bronchial carcinoma

1- 35% are **squamous cell carcinoma**.
2- 30% **Adenocarcinoma.**
3- 20% **Small cell carcinoma.**
4- 15% **Large cell carcinoma.**

Signs and symptoms

1- **Due to primary tumour(local invasion)**

a- **Cough;** Most common early symptom, often dry but;
b- **Sputum** may purulent if there is secondary infection.
c- **Hemoptysis**; Repeated episodes of scanty or blood streaking of sputum.
d- **Dyspnoea**; Reflect occlusion of large bronchus→ Collapse of lobe or lung→ pleural effusion.
e- **Bronchial obstruction**; may be **complete or partial**, which may lead to **pneumonia→ lung abscess.**
f- **Chest pleural pain**; reflect invasion of pleura or infection or involvement of intercostals nerves.
g- **Hoarseness of voice** due to pressure on recurrent laryngeal nerve.
h- **Strider** (aharsh inspiratory noise), due to lower trachea, carina or main bronchi are narrowed by 1ry tumour or enlarged L.N. .
i- **Dysphagia** due to metastinal spread & pressure on oesophagus.

2- **Due to secondary deposits(blood borne metastases)**
   a- **Neurological features**; headache, paralysis, vomiting& epilepsy.
   b- **Bone pain**.
   c- **Skin nodules**.
   d- **Clubbing of fingers.**
   e- **Lassitude, anorexia, weight loss.**
3- **Non- metastatic extra pulmonary manifestations**
   a- **Endocrine syndrome**(**inappropriate ADH secretion).**
   b- **Hypercalcaemia**(parathyroid hormone –like peptides secretion.

Investigation

1- **Chest X-ray**;
   a- Awel defind shadow, peripheral pulmonary opacity.
   b- Collapsed segment, lobe or lung.
   c- Pleural effusion.
   d- Unilateral hilar enlargement, cardiac enlargement , rib destruction & elevated hemidaiphragm.
2- **Bronchoscopy and biopsy.**
3- **Sputum for culture and cytology.**
4- **Barium swallow bronchography.**
5- **Ct-scan of whole body and chest tomography.**
6- **Supraclavicular lymph nodes biopsy.**
7- **Mediastinoscopy and biopsy.**

Treatment

1- Surgery.
2- Radiotherapy.
3- Chemotherapy.

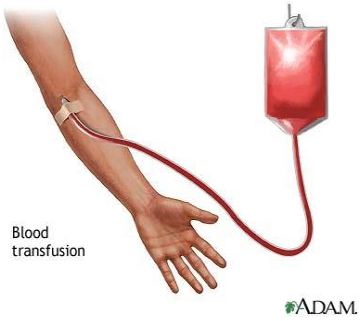
Preparation of blood for transfusion

Donor should be

1- **Healthy** (**No** heart disease, cancer, severe asthma, bleeding disorders, or convulsion).
2- **Normal blood pressure(100-150/60-90).**
3- **Not of an extreme age(at least 18years and above).**
4- **Normal PCV(male ≥ 40%, female ≥35%) and normal Hb.**
5- **Normal pulse(50- 100 beat/minute).**
6- **Good weight(≥ 50kg).**
7- **Normal temperature(≤ 37 ° C).**
8- **Free of infectious diseases**(hepatitis, syphilis, and AIDS).
9- **No pregnancy or recent major surgery.**

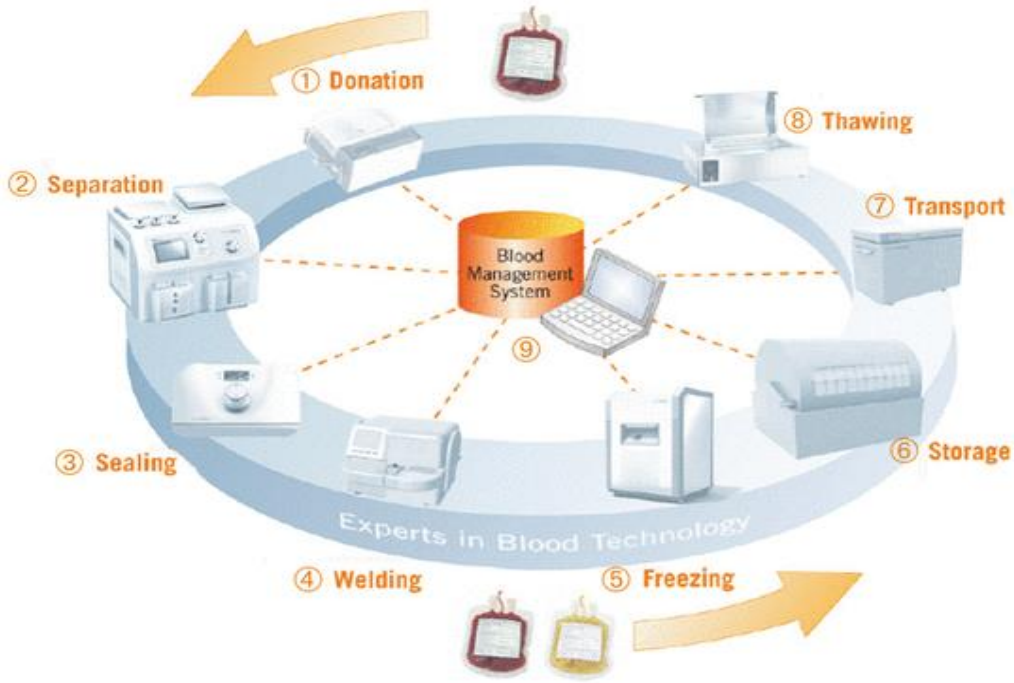
Collecting blood from the donor

1- Donor is lying down.
2- Sphygmomanometer cuff is applied to the upper arm and inflated to a pressure of 70mm Hg.
3- Puncture the median cubital vein by a needle of transfusion set.
4- The other needle is introduced into a plastic bag(which is sterile).
   Containing **anticoagulant** citrate-phosphate-dextrose solution(75ml).
5- 410ml of blood is allowed to run into the bag.
6- During collection mix the blood with the anticoagulant solution gently.
7- The needle is removed from the donor and the bag after clamping the tube
8- If the bag is with a tube, part of tube is clamped at two points, to allow part of blood remains in it which is used for blood grouping and cross match.
9- Let donor gradually gets up and goes after checking his blood pressure.



Blood storage

1- The blood stored in a special blood bank and refrigerator at special temperature according to types of blood products, and if stored at high temperature more than two hours. The chance of infection increase.
2- A fresh blood is better than stored blood.
3- W.B.C. are rapidly destroyed in stored blood.
4- Platelets reduced too much after 24 hours.
5- Quick fall down of clotting factors.



Blood and blood products

1- **Whole blood**; Rich in coagulation factors if fresh. 450ml of donor blood collected into 63ml of anticoagulants(preservative solution).
Stored in 2- 6 ◌̊ C , with shelf life to 5weeks.
2- **Packed red cells**; Cells that are spun down and concentrated, each unit contains 330ml which has haematocrit of 50-70%.
Stored in SAG-M solution(saline-adenine-glucose-manitol) **at 2-6 ◌̊ C for 5weeks.**
3- **Fresh frozen plasma (FFP)**; Rich in coagulation factors , removed from fresh blood and **stored at -40 to -50 ◌̊ C with shelf life of 2 years.** It is **used for treatment of coagulopathic haemorrhage.**150-300ml plasma is obtained from one donation of whole blood.
4- **Human albumin solution of two strength 5% and 20% , used for nephrotic syndrome.**
5- **Platelets;** They are supplied as a pooled platelet concentrate containing about $250\times10_9$ cells/liters. **Stored 20-24 ◌̊ C, with shelf life 5days**. It is **used for thrombocytopenia or platelet dysfunction who are bleeding or undergoing surgery.**
6- **Cryoprecipitate;** supernatant precipitate of FFP, rich in **factors VIII and factor IX Stored -30 ◌̊ C with 2years shelf life. Used for hemophilia.**
7- **I.V. immunoglobulin (IV IgG); used to replace IgG.**
8- **Prothrombin complex concentrates(PCCs);** They contain **II**, **IX, X** and **VII factors. Used for emergency reversal of anticoagulant(warfarin)** therapy in uncontrolled haemorrhage.

Category	Type	Used for
Blood transfusion products	Whole blood products	Blood transfusion for excessive bleeding due to operations, accidents, etc.
	Red blood cell products	Chronic anemia and subacute hemorrhagic anemia
	Plasma products	Severe hepatopathy, disseminated intravascular coagulation (DIC), etc.
	Platelet products	Thrombocytopenia, etc.
Plasma derivatives	Albumin products	Hemorrhagic/traumatic shock, burn, etc.
	Immunoglobulin products	Hypoglobulinemia or aglobulinemia, severe infection, etc.
	Blood coagulant factor products	Supplementing blood coagulant factor to hemophiliacs

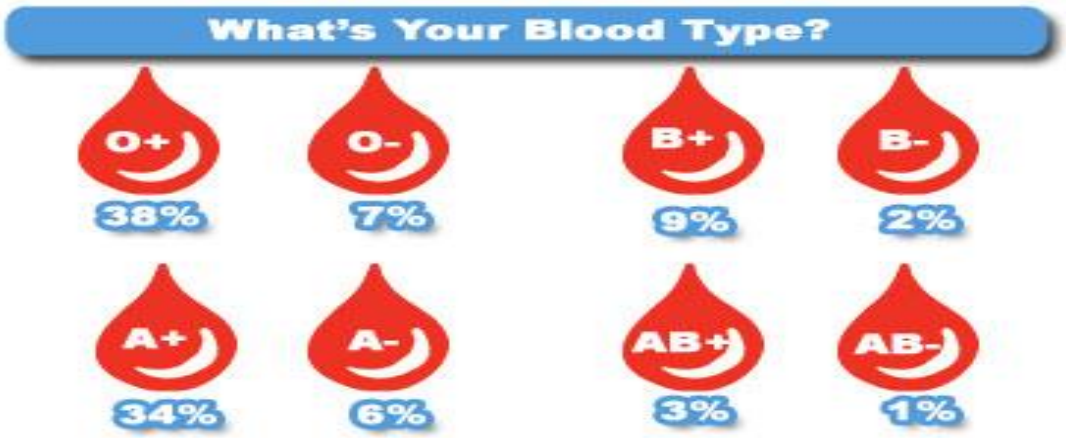
Indications for blood transfusion

1- **Acute blood loss due to any cause(**trauma, severe burn, major operation, severe postpartum hemorrhage, hemophilia).
2- **Perioperative anaemia.**
3- **Symptomatic chronic anaemia.**
4- **Blood exchange(infant Rh incompatibility).**

Blood grouping and cross match

Human R.B.C. have many different **antigens on their cell surface**, two groups of major importance in surgical practice are **ABO and Rhesus** systems. On other hand the **antibodies(mostly IgM) present in plasma.**

Group(phenotype)	Antigen(Aglutinogen)	Frequency	Antibody(Aglutinine)
A	A	42%	Anti-B
B	B	9%	Anti-A
AB	A,B	3%	None
O	None	46%	Anti-A&B
Rh+	Rh	85%	No anti-Rh
Rh -ve	No Rh antigen	15%	No antiRh but could be acquired by Rh+ transfusion

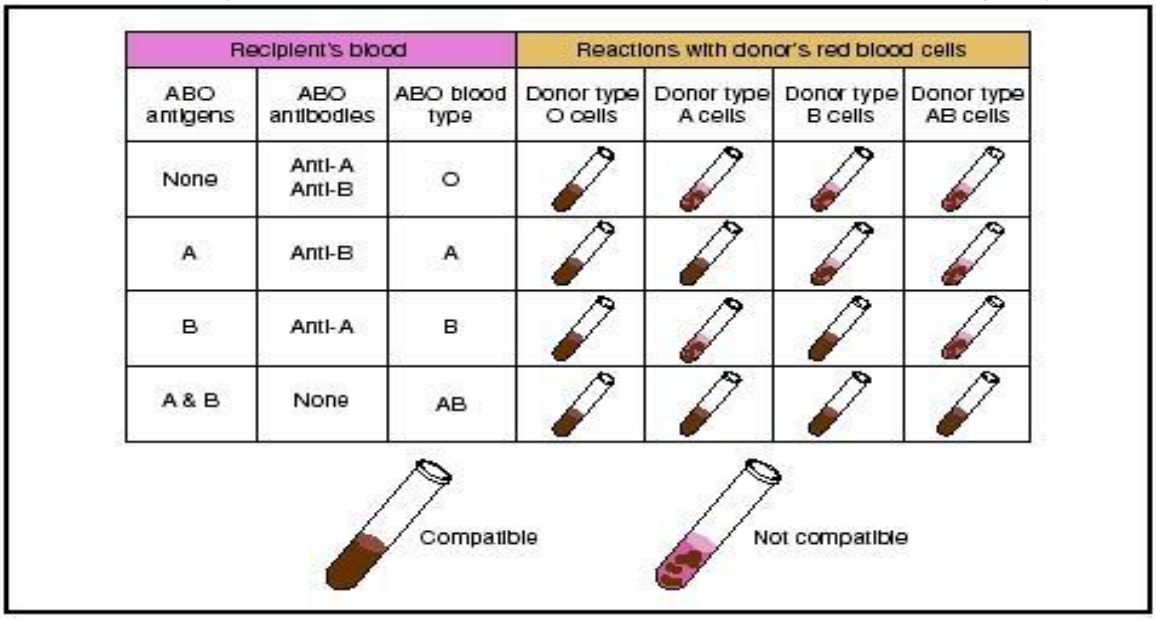


Transfusion incompatibility

When incompatible blood given to a person there may be renal failure to be occurred due to haemolysis of donated RBC by recipient's serum antibodies(IgM)

Cross match

1- **Direct matching of donor 's RBC with recipient's serum.**
2- **If no agglutination occur the blood is compatible.**
3- Blood of type O Rh-ve Should be present in blood bank for emergency.



Blood grouping

Mix the RBC of a person(**donor**) with anti-A, B and Rh.(solution drop) on slide If;

1- No reaction in all groups mean→ Group O Rh-
2- Reaction occurs in anti A means→ Group A.
3- Reaction occurs in anti B means→ Group B.
4- Reaction occurs in both anti A & B means→ Group AB.
5- Reaction occurs in anti Rh means→ Group Rh+.

In collection of blood you have to;

1- Write the name of donor and recipient.
2- Blood groups of both.
3- Ward's name of recipient, hospital and bed number.
4- Date of collection.

In giving blood you have to;

1- Check names of both donor and recipient.
2- Check blood groups of both donor and recipient.
3- The ward's name of recipient, hospital and bed number.
4- Select proper site and canula should be inserted.
5- Gently shake blood bag by your hands or warm water or warming unit.
6- Rate of blood given is determinate by drops/minute.
7- Apply pressure cuff on container in case of rapid transfusion.

Complications of blood transfusion

Complications from single transfusion

1- **Incompatibility hemolytic transfusion reaction.**
2- **Febrile transfusion reaction(1%).**
3- **Allergic reaction(1%).**
4- **Infection; Bacterial, viral(hepatitis& HIV) and malaria.**
5- **Air embolism and thrombophlibitis.**
6- **Transfusion-related acute lung injury.**
7- **Congestive heart failure especially in heart diseases.**

Complication from massive transfusion

1- **Coagulopathy(due to dilution of clotting factors).**
2- **↓Ca++, ↓K+, ↑ K+.**
3- **↓Temperature.**
4- **Iron over load (thalassaemia) due to repeated transfusion.**

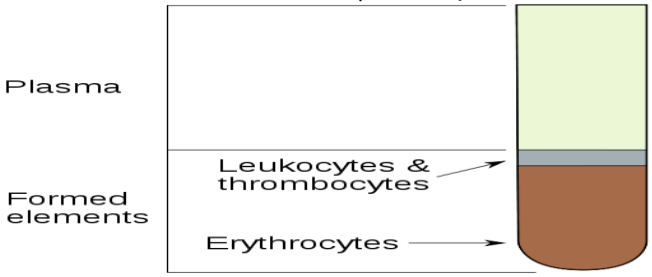
Blood diseases

Blood Is a highly specialized connective tissue. The normal total circulating blood volume is 8% of the body weight(i.e. 5600 ml for 70kg body weight person).

Composition of blood

1- **Plasma;** Fluid portion (55%).It is composed of water(91%), ions(2%) and plasma proteins(7%)mainly albumins, globulins(α, β, δ) and fibrinogen.
2- **Blood cells**; Solid portion (45%):
    1- **Specialized cells (erythrocytes, leukocytes).**
    2- **Cell fragments (platelets or thrombocytes).**

Serum=plasma−clotting factors I,VIII, V,II+ serotonin(due to platelet destruction).



Erythrocytes(RBC);**Composed of65% water + 35% solid(95% is Hb).**
--MCV(mean red cell volume)=**87cubic micrometer**(fomtoliter-FL-).

1- **↑MCV**(↑size of RBC)=**Macrocytic** (**Megaloblastic anaemia, infant Blood & Diseases lead to ↑ plasma lipid)**.
2- **↓MCV**(↓size of RBC)=**Microcytic**(**Iron deficiency anaemia**).

MCH(mean corpuscular haemoglobin in single RBC)=**29pg(pigogram).**
MCHC(mean corpuscular haemoglobin cocentration in single RBC)=**34gm/dl.**
PCV(Haematocrit), i.e. volume of RBCs present in100ml(dl) of blood**=male47% =female42%**

Life span of RBC=120 in male while 110 in female.
Total blood volume= 5-6liters in male while 4-5liters in female.

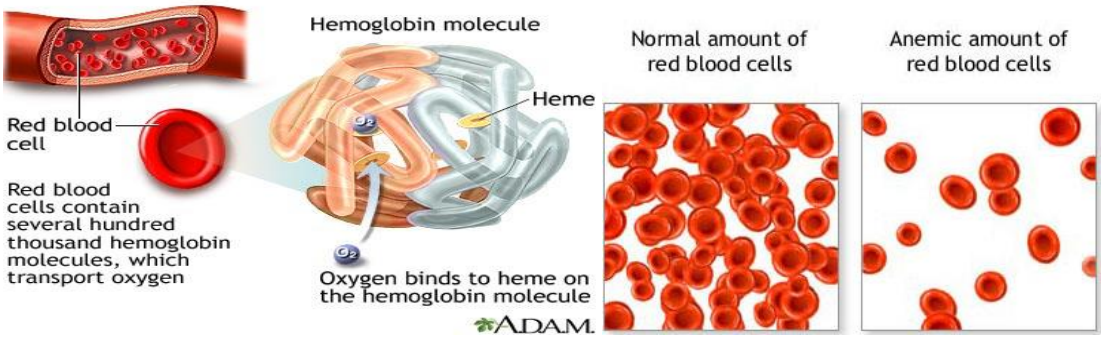
Hemoglobin(Hb)

It made of 4 subunits; each unit contains heme conjugated to globin.
Hb = 4hemes + 4protein chains (**2α & 2 β chains**).

Types of Hb

1- **Embryonic Hb(**occurs early in gestation)(Gower1,2 &Portland)**.**
2- **Fetal Hb (HbF); consists of 2α & 2γ chains)** (before birth which gradually replaced by adult Hb).
3- **Adult Hb (HbA); HbA1=97.5% of total HbA . HbA2=2.5% of total HbA.**
4- **Glycosylated Hb(HbA1c);** when glucose attached to N-terminal valine of β–chains, this test done in person suspected of having D.M.,its normal level in adult is **5% of HbA1,** while in diabetics the level is elevated.

Anaemias



Anaemia refers to a state in which **the <u>level of hemoglobin in blood is below the normal range</u>** appropriate for age & sex.

Or <u>deficiency in total number of RBC.</u>

Causes of anaemia

A- <u>Central causes:</u> <u>Decreased or ineffective</u> <u>bone marrow production</u>

1- Defective(↓)Hemoglobinization(Normal DNA synthesis)(Microcytosis)
   1- **Iron deficiency anaemia.**
   2- **Thalassaemia anaemia .**
   3- **Sideroblastic anaemia.**

2- Defective(↓)DNA synthesis (Normal Hemoglobinization)(Macrocytosis)
   1- **↓B_{12}.**
   2- **↓Folate.**
   3- **Cytotoxic drugs.**
   4- **Mylodysplasia.**

3- Invasion by malignant cells

4- Renal failure → ↓Erythropoietin hormone.

B- <u>Peripheral causes:</u>
   1- **Blood loss.**
   2- **Haemolysis.**
   3- **Hypersplenism.**

C- <u>Diseases lead to ↑ plasma lipid (Macrocytosis)</u>

1- **Liver diseases.**
2- **Hypothyroidism.**
3- **Alcohol abuse.**
4- **Hyperlipidaemia.**
5- **Pregnancy.**

Clinical feature

The features reflect ↓oxygen supply to tissues and depend on;

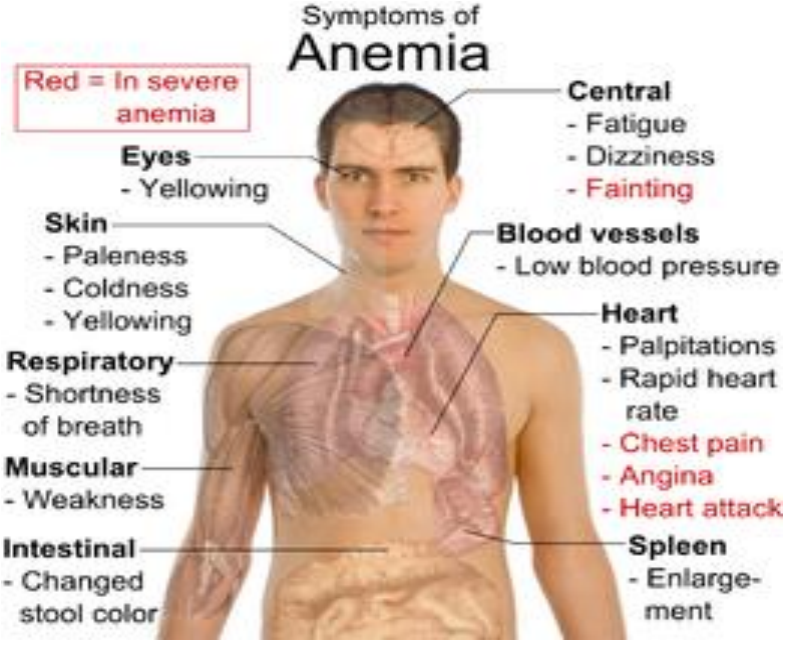
1- Degree of anaemia.
2- Rapidity of its development of anaemia(i.e. acute blood loss).
3- The presence of cardio respiratory diseases.

Symptoms

1- **Tiredness and fatigue.**
2- **Light headedness.**
3- **Breathlessness and palpitation.**
4- **Ankle swelling.**
5- **Worsening of any previous coexisting diseases (such as angina).**
6- **Paraesthesia (numbness) in fingers and toes.**

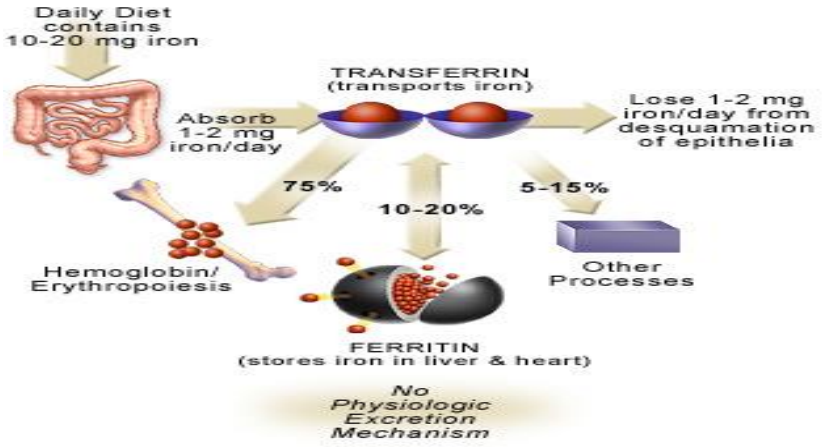
Signs

1- **Pallor of skin and mucous membrane.**
2- **Tachypnoea and tachycardia.**
3- **Raised jugular venous pressure.**
4- **Systolic flow murmurs.**
5- **Ankle oedema.**
6- **Postural hypotension.**
7- **Cracking and brittle nails.**



Around 30% of total world population is anaemic and half of these(i.e. 600 million people) have iron deficiency anaemia.

Iron deficiency anaemia

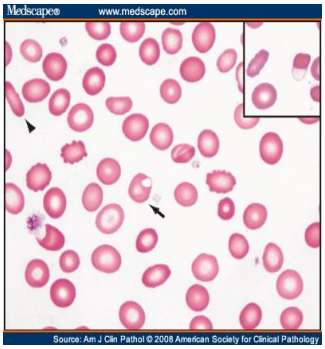


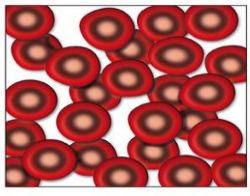
Causes

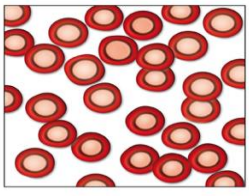
1- **Iron loss; Due to blood loss** through; GIT& UTI bleeding of any causes, menstruation and child birth.
2- **↑Demand of iron(depleting iron storage);**Like in breast feeding, pregnancy and rapid growth state(infant &puberty).
3- **Malabsorption;** Coeliac disease and hypochlorhydria(elderly & PPI drugs).
4- **↓Iron intake**; most of vegetable food.

Investigation

1- **Plasma ferritin is subnormal;** To measure iron stores, it is the best single test to confirm iron deficiency.
2- **Plasma iron & total iron binding capacity(TIBC);**It is **low** during acute phase **but raised** in liver disease and haemolysis.
3- **Transferrin level; low** in acute phase & liver disease**.**
4- **Bone marrow aspirate for iron stores.**
5- **Blood film; Microcytic (↓MCV) hypochromic anaemia.**




Normal erythrocytes


Hypochromic erythrocytes containing less haemoglobin

Management

1- **Oral iron supplementation;**
-**Ferrous sulphate** 200mg 8 hourly for 3-6 months.
-**Frrous gluconate** 300mg12hourly(given in dyspepsia &altered bowel habit.
2- **Parenteral iron** with deep i.m. injection of **iron sorbito**1.5mg/kg.
side effect is brown discoloration at site of injection.

Megaloblastic anaemia

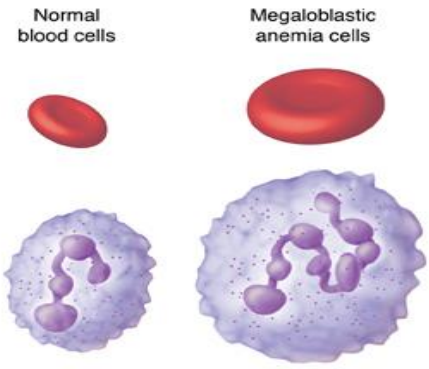
This result from; **1- Folic acid & vitamin B_{12} deficiency.**
2-Disturbance in folic acid metabolism.

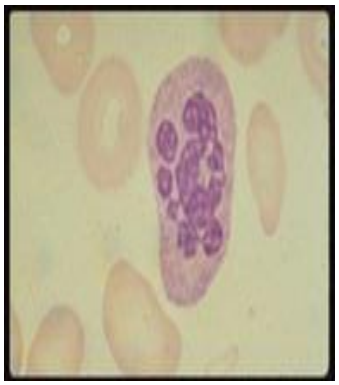
Clinical features

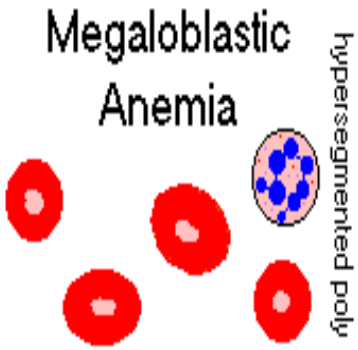
1- **Malaise (90%).**
2- **Sore throat.**
3- **Altered skin pigmentation (vetiligo & skin pigmentation).**
4- **Poor memory, depression& impotence.**

Diagnostic features;

1- **↑↑Hb.**
2- **↑MCV (above 120 fl).**
3- **↓RBC, ↓Reticylocyte count,↓WBC or normal &↓platelet or normal.**
4- **↑Serum ferritin**
5- **↑↑LDH.**
6- **B. marrow; ↑Cellularity** & other changes of megalplastic anaemia.
7- **Blood film**; **Oval macrocytosis**& other changes of megalplastic anaemia.







Treatment;

Vitamin B_{12} deficiency;

Hydroxocobalamine1000µg parenterally single dose then every 2-3 days further dose& **maintainance dose is 1000µg every 3 months**. It is advised to **add 200mg/day ferrus sulphate** to prevent depletion of iron reserves due to rapid regeneration of blood.

Folate deficiency;

Folic acid 5 mg/day orally is sufficient. **5mg/week as a maitainance therapy**.
Never give it with vitamin B12 in vitamin B12 deficiency anaemia because the risk of aggravating neurological features of vitamin B12 depletion.
Give folate supplementation 350 µg/day to all pregnant women.

Hemolytic anaemia

Haemolytic anaemia means an **accelerated destruction of peripheral RBC** (accelerated haemolysis) =life span is short. The bone marrow may ↑ its output of RBC 6-8 folds by many changes. If the rate of destruction exceed this increased production rate, then anaemia will develop(or when output no longer compensate).Liberated Hb in plasma (after destruction of RBC) bound to haptoglobin to be taken up by liver & degraded, if all haptoglobin is consumed→ free Hb lost in urine →Damage the kidney(acute renal failure).

Types of haemolysis

1- **Intravascular;** Malaria infection.
2- **Extravascular;** The most common like physiological RBC destruction in liver & spleen.

Types of haemolytic anaemia according to the cause

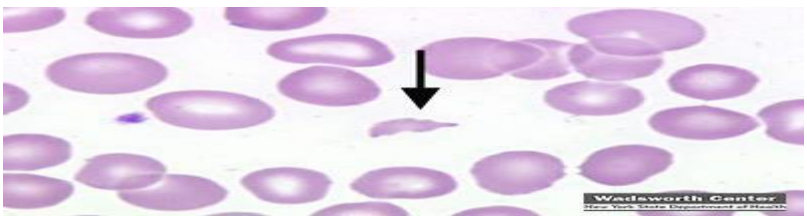
1- **Congenital;(In hereditary RBC defects of structure or metabolism)**
Hereditary spherocytosis, glucose-6—phosphate dehydrogenase (G6PD) and haemoglobinopathy(Thalassemia α,β & sickle cell anaemia).
2- **Acquired ; -- Autoimmune haemolytic anaemia**(Warm80% & Cold20%)**.**
-- Non- immune haemolytic anaemia
-Physical trauma; mechanical heart valve, vigorous exercise, thermal injuries.
& -Infection like malaria, clostridium septicemia).

Clinical features

1- **Those of anaemia.**
2- **Splenomegaly.**
3- **Mild jaundice.**
4- **Dark yellow urine(black water).**
5- **Hb in urine.**
6- **Bone pain (sickle cell anaemia).**

Investigation

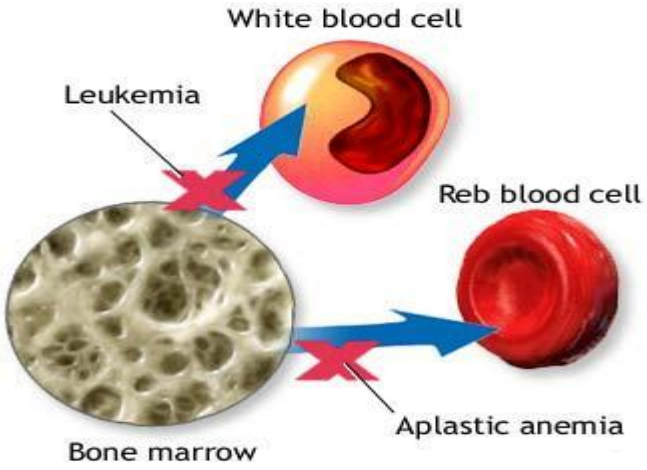
1- **Blood film(Thalassemia).**
2- **Hb electrophoresis.**
3- **Sickling test.**



Treatment

1- **Splenectomy** (sphyrocytosis & thalassemia)**.**
2- **Bone marrow transplantation** (sickle cell anaemia& thalassemia).
3- **Disferrioxamine therapy**(No- iron).
4- **Folic acid (5mg/day) & penicillin-**V against infection.
5- **Blood transfusion.**

Aplastic anaemia



It means **lack of functioning bone marrow** (i.e.**no blood cell production**).
Or failure of the stem cells to a varying degree, producing **hypoplasia of bone marrow elements.**
It is of **two types**; **1- Idiopathic acquired anaemia.**
2- Secondary aplasia.

Causes

1- **Drugs;** Cytotoxic drug, chloramphenicol, sulphonamides, indomethacin,etc..
2- **Chemicals;** Insecticides, organophosphate & carbonates.
3- **Radiation**.
4- **Pregnancy.**
5- **Renal failure;** ↓production of erythropoietin.

Investigation

1- Full blood count→ Pancytopenia (anaemia of normocytic & normochromic)**.**
2- Bone marrow examination.

Management

1- Blood product support & aggressive management of infection.
2- Allogeneic **bone marrow transplantation**(< 20years).
3- Immunosuppressive therapy.

Prognosis; 50% of patient die after supportive therapy, but **60% are survival after bone marrow transplantation.**

Anaemia of chronic diseases(CDA)

**** It occurs in the sitting of chronic infections, chronic inflammations or neoplasia.**
****It is not related to bleeding, haemolysis or marrow infiltration.**
****The anaemia is mild(85-115g/l), normochromic normocytic** may be↓MCV(25%).
****↓Serum iron** but iron stores are normal or ↑ as indicated by ↑ferritin.
**If ferritin↓or normal→ Iron deficiency anaemia in the sitting of disorders associated with CDA.

Hemorrhagic anaemia

The loss of blood either; **1- Acute(Large volume in short period)**.
Or **2- Chronic(small loss over long period).**

Haemorrhage lead to a state of hypovolaemic shock which lead to physiological exhaustion (coagulopathy, acidosis and hypothermia)--→ subsequently death.
So every effort must be made to rapidly identify & stop haemorrhage and to avoid or limit physiological exhaustion.

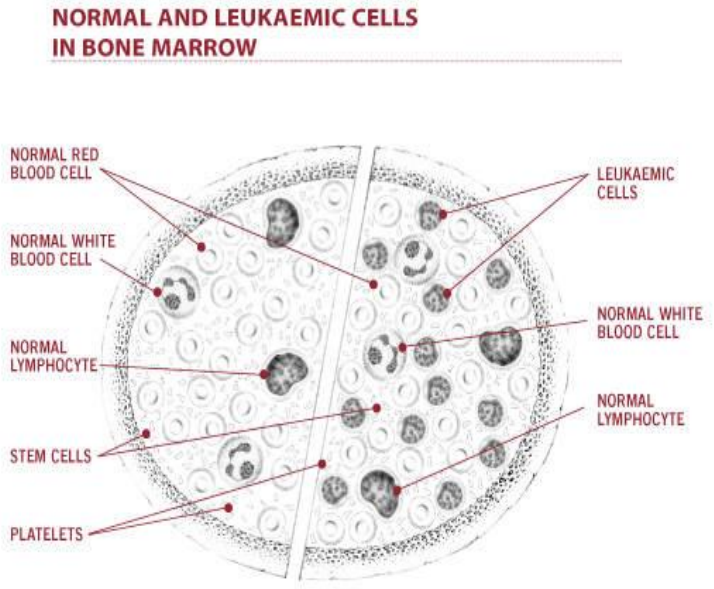
Types of Haemorrhage

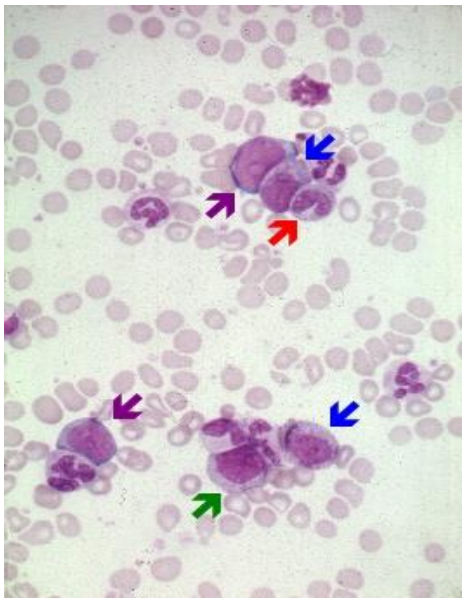
1- **Revealed (External).**
2- **Concealed (Internal) .**
3- **Primary hemorrhage(occurs immediately after injury).**
4- **Reactionary hemorrhage(**occurs within 24hours –dislodgement of clot-).
5- **Secondary hemorrhage**(occurs 7-14 days after injury).
6- **Surgical & non-surgical hemorrhage**(due to coagulopathy).

Management

1- **Identify the hemorrhage & its site.**
2- **Immediate resuscitation maneuvers.**

Leukaemias





Leukaemias are a group of malignant disorders of hematopoietic tissues characterized by **increased number of primitive WBC(blast) in bone marrow**, the course of leukemia vary from few days to many years depending on types.

Epidemiology

1- About 10/100000 of population are affected.
2- Males are more affected(3:2 in acute & 2:1 in chronic).
3- **Acute leukaemia occurs at all age**(acute lymphoblastic leukaemia affect mostly 1-5years), **while chronic leukaemia affect adult & old age**.

Aetiology

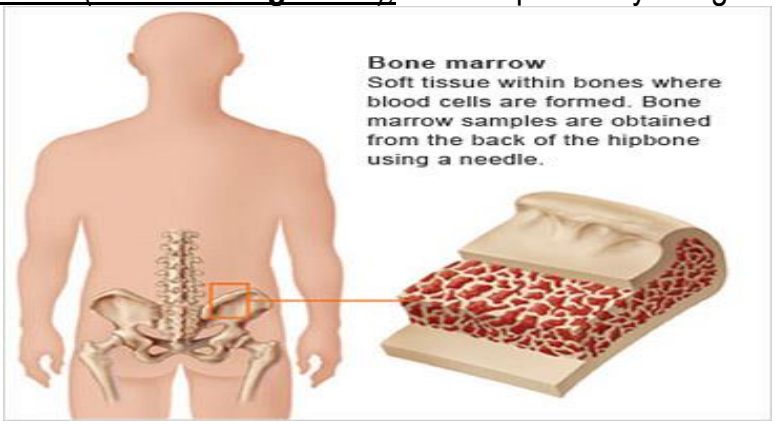
1- **Mostly unknown.**
2- **Ionizing radiation**(Japanes cities by atom bombing&X-ray of i.uterine fetus)
3- **Exposure to benzene in industry**.
4- **Viruses**(retroviruses) & **cytotoxic drugs**.
5- **Genetic and hereditary causes (down's syndrome).**
6- **Acquired Immune Deficiency Syndrome(AIDS).**

Classification of leukemia

1- **Acute lymphoblastic leukaemia(survival rate 30 months).**
2- **Acute myeloid leukaemia(survival rate 5years).**
3- **Chronic lymphatic leukaemia(most common).**
4- **Chronic myeloid leukaemia**

Investigation

1- **Blood film; A-leukocytes** may be normal, subnormal or very higher(1000-500000/cu.mm).
B-Immature cells(blast)(is diagnostic).
C-Anaemia normal or ↑MCV.
D-Thrombocytopenia is severe.
2- **Bone marrow aspiration(valuable diagnostic)**;Which replaced by malignant blast cells.



3- **Coagulation time and bleeding time are increased.**
4- **Renal function test (urea& creatinine) .**
5- **Hepatic function test** (total protein, albumin, bilirubine, alkaline phosphatase**).**
6- **↑γ-globulin.**
7- **↑Serum enzymes (plasma LDH & plasma urate).**
8- **↑ ESR.**

Clinical features(features of bone marrow failure)

1- **Low grade fever (**acute leukaemia **) & pallor.**
2- **Malaise& sweating.**
3- **Bleeding & purpura, gingivitis & mouth ulcers** acute leukaemia**.**
4- **Hepatosplenomegaly & lymphadenopathy(mostly in chronic).**
5- **Bone pain (in chronic).**

Management

The first decision must be whether or not to give specific treatment.
Group of patients is no need for specific treatment

1- Very elderly patients (above 80years).
2- Patients with other serious disorders.
3- Patient who decline specific therapy.
4- Types of leukaemia to be very unresponsive to specific treatment.

In these groups supportive therapy only should be offered.

Treatment of acute leukaemia

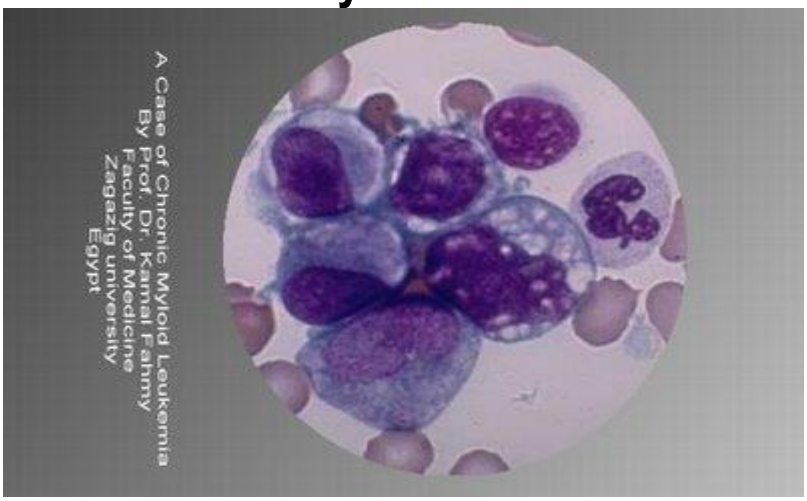
1- Specific treatment

Lymphoblastic Leukaemia	**Myeloblastic Leukaemia**
Phase1(Induction)	
Vieristine (I.V.)	Daunorubicin (I.V.)
Prednisolone (oral)	Cytosine arabinosisd (I.V.)
L-asparaginase (I.V.)	Etoposide (I.V. and oral)
Daunorubicin (I.V.)	Thioguanine (oral)
Methotrexate (intrathecal)	
Etoposid (I.V. & oral)	
Cytosine arabinoside(I.V.)	
Phase 2(Consolidation)	
Mercaptopurine (oral)	Cytosine arabinosisd (I.V.)
Methotrexate (intrathecal)	Amsacrine (I.V.)
	Etoposide (I.V. and oral)
	Mitozantrone (I.V.)
Phase 3(Maintenance)	
Prednisolone (oral)	Mercaptopurine (oral)
Vincristin (I.V.)	Cytosine arabinosisd (I.V. or S.C.)
Mercaptopurine (oral)	Or no drug
Methotrexate (oral)	

2- Bone marrow Transplantation.

3- **Radiotherapy**
4- **Supportive therapy**
    1- Treat complication; Anaemia, Bleeding(due to thrombocytopenia)and infection(UTI, oral candidacies. Gingival ,skin infection) .
    2- Protected from environments**.**
    3- Supportive observation and continuous monitoring of renal, hepatic & haemostatic functions are necessary with fluid balance.
    4- Psychological support.

Chronic myeloid leukaemia



**The peak incidence at 55 years.
Most patients have **Philadelphia chromosome.

Clinical features

1- **Asymptomatic.**
2- **Tiredness, malaise, lethargy & sweating.**
3- **Dyspnoea.**
4- **Abdominal pain, abdominal fullness, anorexia & weight loss.**
5- **Bruising.**
6- **Hepatosplenomegaly (90%).**

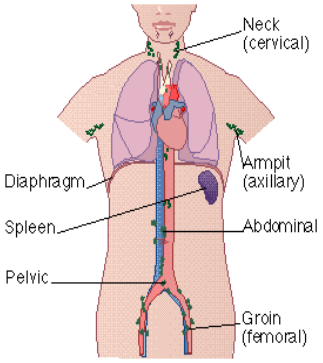
Management

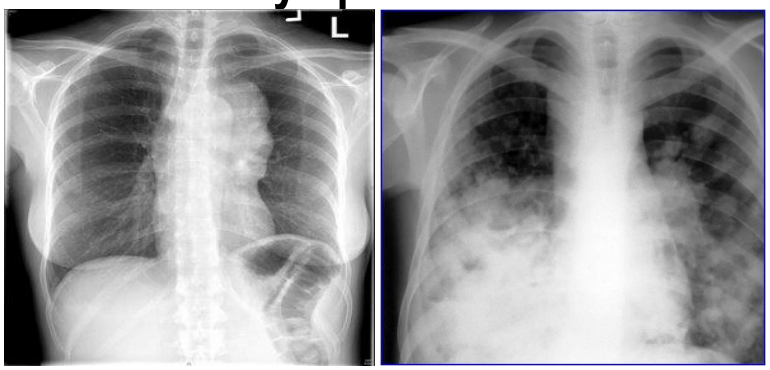
1- Sometimes no treatment.
2- **Chemotherapy mostly are needed;**
   --Busulphan 4mg daily, --Hydroxyurea 2mg/day, --Melphalan 4-5mg/day.
3- **α- interferone**
4- **Splenectomy.**
5- **Treatment of thrombocytopenia and bleeding.**

Prognosis

Median survival is about 3 years.

Lymphoma

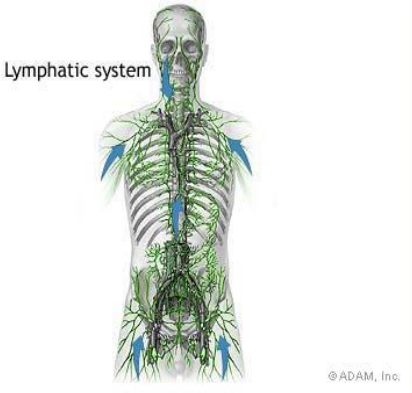


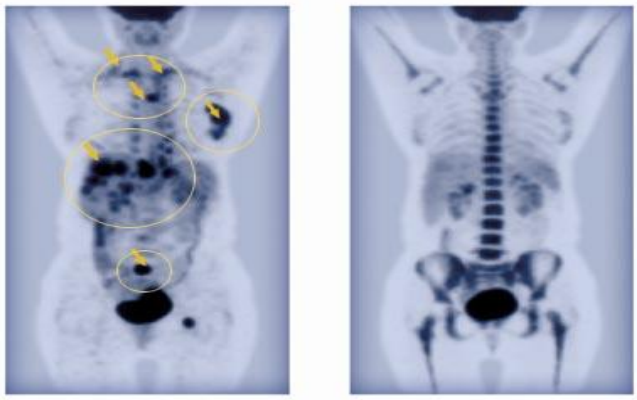


Non Hodgkin lymphoma

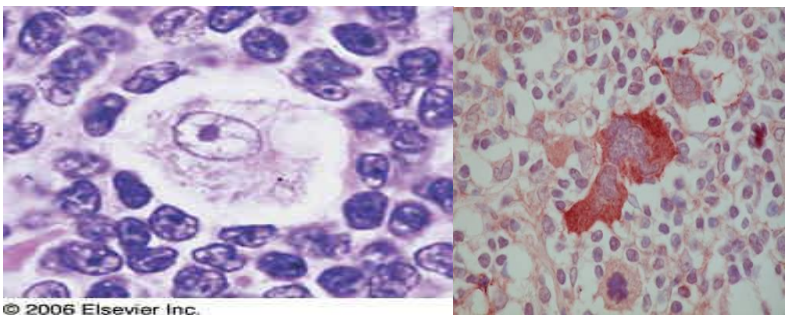
1- **High grade tumours→ <u>Divide rapidly, few week to be diagnosed.</u>**
2- **Low grade tumours→ <u>Divide slowly need months.</u>**

(This classification according to its proliferation)





Hodgkin lymphoma



****Characterized by <u>progressive painless enlargement of lymphatic tissue</u> usually in neck and supraclavicular fossa.**

** It is more common in adolescence male **.**

** **Reed Sternberg (RS) cells seen,** they are a histological hallmarks (which are large malignant lymphoid cell of B- cell origin).

Clinical features

1- **Lymph node** **1-Solitary enlarged lymph node or groups of nodes.**
2- Mostly in neck, mediastinum & supraclavicular fossa.
3-Rubbery in consistency & discrete.
4-Painless, freely mobile not attached to skin, fluctuate in size.

2- **Dysphagia, dyspnoea & dry cough(in mediastinal type).**
3- Weakness, **loss of weight** & sweating(at night).
4- Splenomegaly may present.
5- Low grade fever & pruritis.
6- Spread of disease to other lymphatic & extra lymphatic tissues(liver, bone marrow).

Investigation

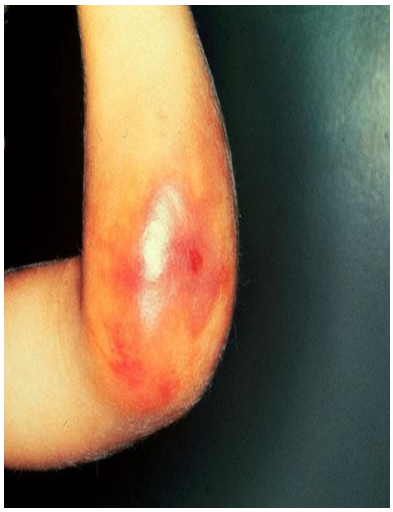
1- **Lymph node biopsy(for histopathplogy)**.
2- **Full blood count**; may be normal, anaemia, lymphopenia.
3- **↑ESR.**
4- Renal function test.
5- ↑LDH.
6- **Chest X-ray; mediastinal mass**.
7- CT-scan of chest & abdomen.

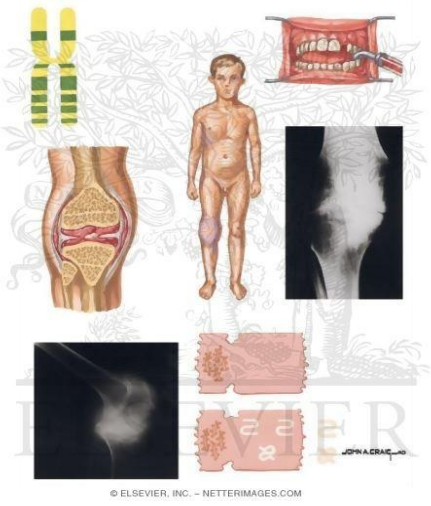
Treatment

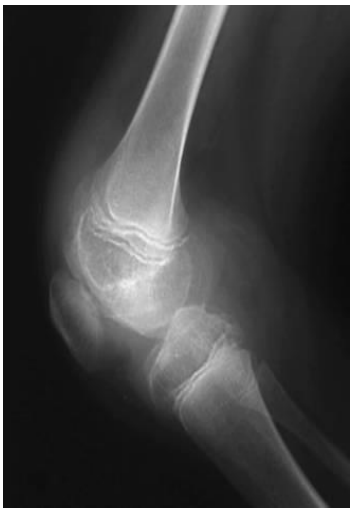
It depend on stage at presentation, and it is mostly curable.

1- **Radiotherapy**.
2- **Chemotherapy**; 1- Chloamphenicol 6mg /m² orally for days1-4.
2- Vinblastine 6mg /m² I.V. days 1 & 8.
3-Prednisolone 40mg /m² orally 14 days.
4-Procarbazine 100mg/m² orally 1-14days.
3- **Combined radio-chemotherapy.**

Hemophilia







Types

1- **Hemophilia A =deficiency factor VIII (factor 8).**
2- **Hemophilia B =deficiency factor IX (factor 9)(Christmas disease).**

** Both are X linked chromosome; males are affected & females are carrier.

Signs and symptoms

1- **Bleeding in joints (haemarthrosis)(bleeding is unusual until 6months of age due to inactivity of babies)** & **superficial bruising.**
2- **Bleeding in muscles (haematoma)**.
3- **Bleeding after minor trauma** as tooth extraction, injection or circumcision.
4- **Prolonged bleeding time.**

The severity of Hemophilia

Degree of severity	Level of F.8 or 9	Clinical presentation
Severe	< 2%	Spontaneous haemarthrosis& haematoma
Moderate	2-10%	Mild trauma or surgery causes haematom
Mild	10-50%	Major injury or surgery results↑ bleeding

****The severe degree is diagnosed with in 1st 2 years of life.**

****Mild & moderate may escape diagnosis until adulthood.**

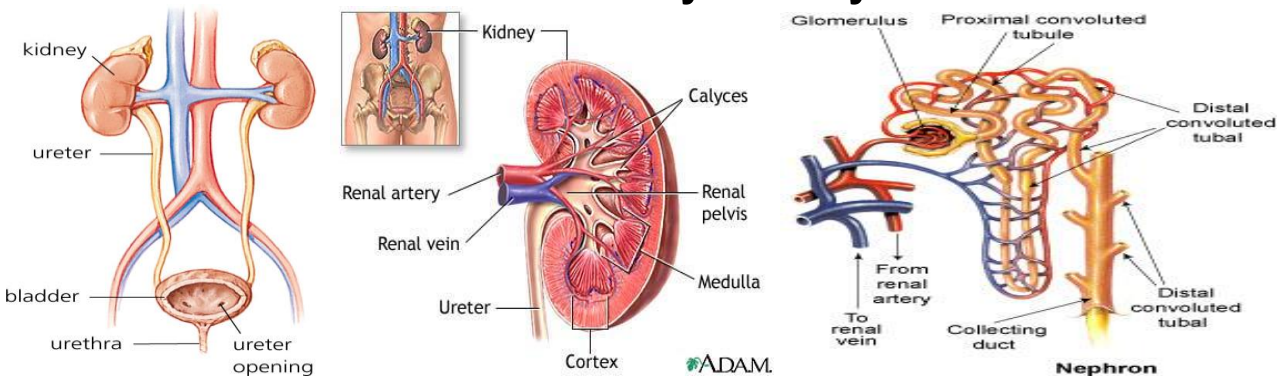
Treatment

1- Resting of the bleeding site by bed rest or splint to↓ continuing bleeding.
2- **Infusion of deficient factor or fresh blood.**
3- Infusion of desmopressin for mild haemorrhage.
4- The treatment is repeated before surgery or tooth extraction.
5- Once the bleeding has settled, the patient should be mobilized & physiotherapy is done to restore strength of surrounding muscles.

Complication

1- **Stiffness of joints.**
2- **Hepatitis(A,B,C,D virus).**
3- **Atrophy of muscle secondary to hematoma.**
4- **Arthropathy of large joints(knee, elbow).**
5- **Mononeuropathy resulting from pressure by hematomas.**
6- **Sudden death in intracranial haemorrhage.**

Urinary tract system



** It consists of **Kidney, Ureter, urinary bladder & urethra**

** **Adult length of kidney= 11-14 cm (3 vertebral bodies)**,**their weight about 300gm**, they are located retroperitoneal on either side of aorta & inferior vena cava**. Rt. Kidney is usually a few centimeters lower** because the liver lies above it. Each kidney contain approximately 1 million nephrons(functional unit of kidney). There is a rich blood supply composes about 20-25% of cardiac output.

** Blood urea is a poor guide to renal function as it vary with protein intake.

** **Serum creatinine is more reliable guide** as it is produced from muscle at a constant rate & almost completely filtered at the glomerulus by tubular cells.

** **Creatinine clearance provide a reasonable approximation of glomerular filtration rate(GFR).**

** Urine volume give a poor guide to renal function.

** **Between 300- 500 cc /day are needed to excrete solutes at maximum concentration on a normal diet.**

** **Complete anuria suggests either acute vascular event or total urinary obstruction** even in severe intrinsic renal disorders some urine is usually still produced.

** **Polyuria is referred to production of an excessive volume of urine** & have many causes.

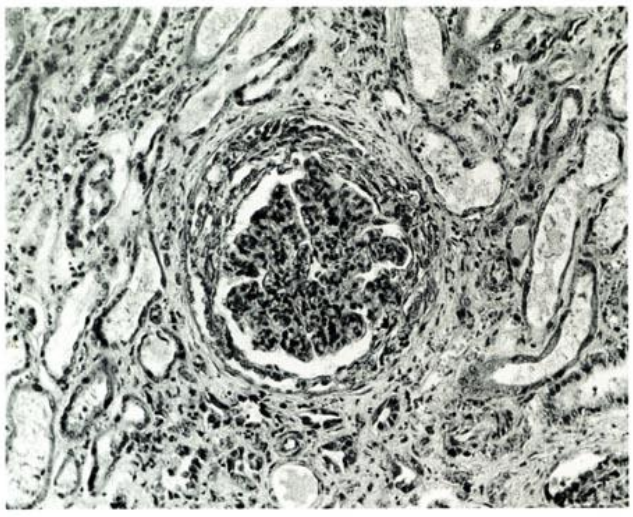
Imaging techniques

1- **Plain x-ray of abdomen (KUB);** for opaque stone in urinary system**.**
2- **Renal ultrasound (non invasive);** for renal size, position, dilation of collecting system, tumour or cyst.
3- **Intravenous urography(IVU);** By giving iodine containing compound, an early image (1 min) after injection→ Nephrogram phase. Side effects include **allergy, nephrotoxicity**, and **time need injection** & depend on adequate renal function.
4- **Pylography**; Need needle insertion of contrast medium into pelvicalyceal system under ultrasound or radiography control.
5- **Micturating cystourethrography**(**MCU**); For diagnosis of **vesico-ureteric reflux** by using urinary catheter to **fill urinary bladder with contrast medium & take film while patient voiding.**
6- **Renal arteriography & venography**; For renal arterial stenosis or haemorrhage by entrance of catheter via femoral vein.

7- **CT- scan (Computered tomography).**
8- **MRI (Magnatic resonance imaging).**
9- **Radionuclide studies (^{99m}Te-DTPA).**
10- **Renal biopsy;** To establish nature & extent of renal diseases to judge prognosis & treatment.

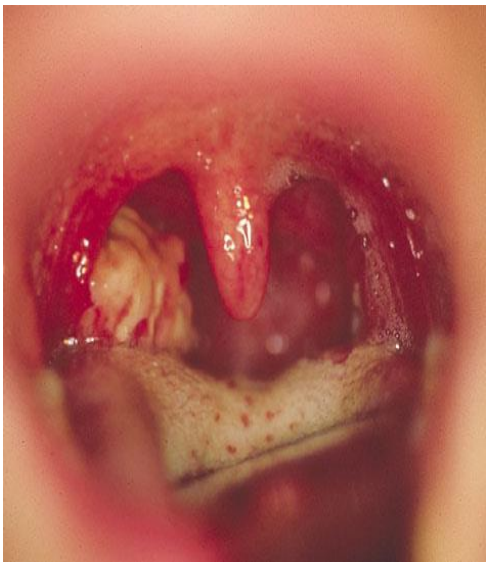
Urinary system diseases

Acute glomerulonephritis (G.N.)



PBBH–A40–24

FIGURE 40.—Photomicrograph, acute glomerulonephritis. Note epithelial crescent, erythrocytes in Bowman's space, and slightly increased cellularity of the tuft. There is protein precipitate in the tubules with some cells and cell debris. There are also interstitial edema and increased cellularity of the interstitial tissue. [Description, courtesy Dr. G. J. Dammin.]



It mean <u>inflammation of glomeruli</u>, it occurs following infection with certain straine of **streptococcal (β-haemolytic type)** so called **<u>post- streptococcal nephritis</u>** (it can following other infections). Much more common in **children** than adults, **10 days after a throat infection or longer <u>after skin infection</u>** suggesting an immune mechanism than direct infection. Most types of G.N. seen to be immunologically mediated (deposition of **antibody-immunoglobulin** or anti GBM- **against glomerular antigen**).

Clinical features

An acute nephritis of varying severity occurs;

1- **<u>↓GFR & Na retention</u> → <u>Hypertension.</u>**
2- **<u>Oedema(mostly eyelids).</u>**
3- **<u>Proteinuria & Haematuria.</u>**
4- **<u>Slight fever.</u>**
5- **<u>↓urine volume(oliguria).</u>**
6- **<u>Discoloration of urine(smoky, red).</u>**
7- **<u>Loin pain or discomfort.</u>**

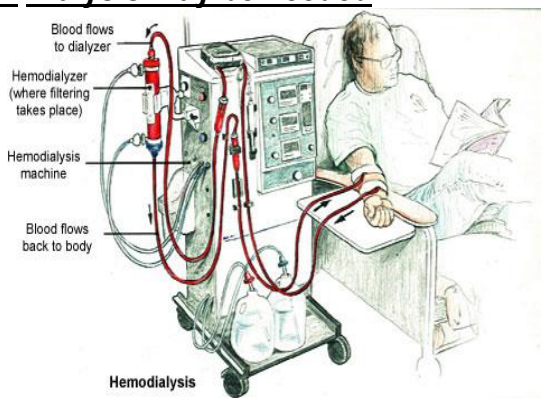
Investigation

1- **<u>General urine examination(protein, cast, RBC, Epithelial cell & WBC).</u>**
2- **↑serum urea.**
3- **<u>↑Serum ASO titer.</u>**
4- **<u>Culture or swab from skin or throat.</u>**

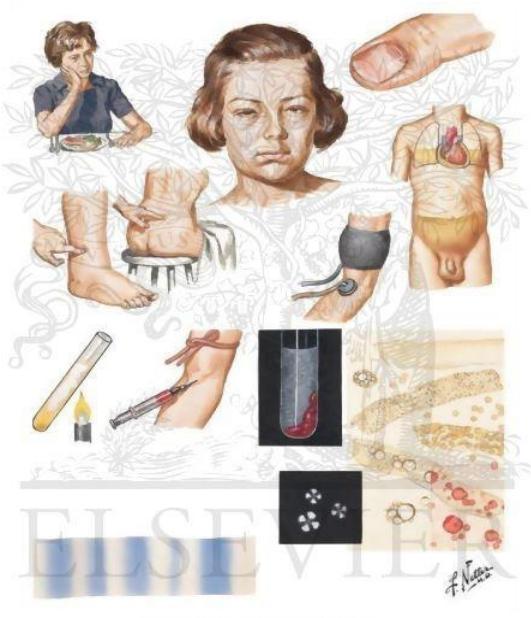
Management

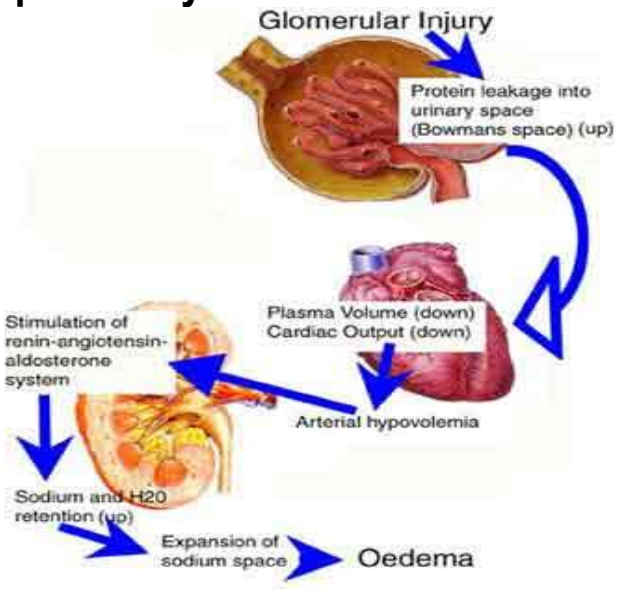
**Renal function begins to improve spontaneously within 10-14 days.

1- **Complete rest.**
2- **Restriction of salt and fluid and use diuretic drugs.**
3- **High calorie diet(Carbohydrates).**
4- **Treatment of strep. Foci.**
5- **Treatment of hypertension. Dialysis may be needed..**



Nephrotic syndrome





****It refers to the secondary phenomena that occurs when substantial amount of protein are lost in the urine.**

Aetiology

1- Nephritis(glomerulo or pyelonphritis).
2- Unknown.
3- Hereditary.
4- Systemic diseases (D.M.→ Diabetic nephropathy).

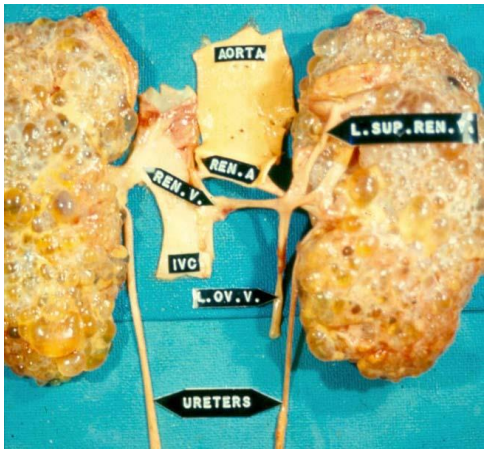
Clinical features

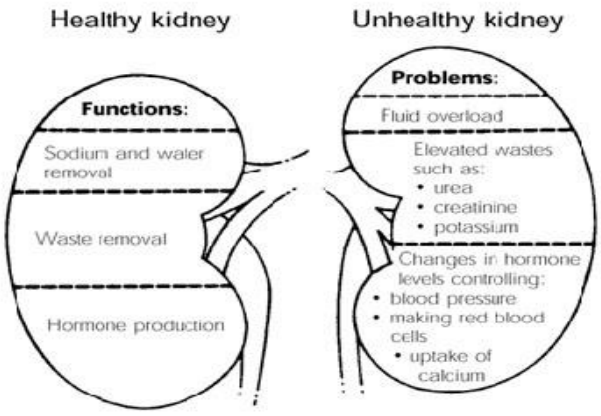
1- **Dependent oedema** accumulates predominantly **lower limb** in adult extending to **genitalia & lower abdomen as it become more severe**, in the morning the upper limbs & face may be more affected. **In children** ascitis occurs early & oedema is often seen only in the **face.**
2- **Blood volume normal or ↑ or ↓.**
3- **Avid renal Na retention** is an early and universal feature**,** leading to ↓oncotic pressure, ↓intrvascular volume, secondary hyperaldosteronism & 1ry defect in renal Na execration.
4- **↑↑ proteinuria (>3.5gm/day –frothy urine).**
5- **↓Albumine in blood(Hypoalbominaemia) <30 gm/l**; due to urinary protein loss exceed synthesis capacity of liver→↓ oncotic pressure →oedema.
6- **↑Lipid in blood(Hypercholesterolemia);** due to ↑lipoprotein synthesis by liver in response to↓ oncotic pressure→↑ **atherosclerosis rate**
7- **↑ coagulation(Hypercoacgulability**); Due to relative loss of inhibitors of coagulation, leading to **venous thromboembolism**.
8- **Infection(pneumococcal);** due to **hypogammaglobulinaemia.**
9- **Anaemia → Pale & puffy face**.

Management

1- **Establish and treat the cause.**
2- **Treat the symptoms and prevent complications;**
   - **A- Diuretic for oedema .**
   - **B- ↓ Na diet.**
   - **C- ↑protein diet .**
   - **D- ↓ Lipid diet.**
   - **E- Anticoagulant & vaccination in children.**
   - **F- Sometimes high dose of corticosteroid for children with minimal change nephropathy.**

Renal failure





Acute renal failure

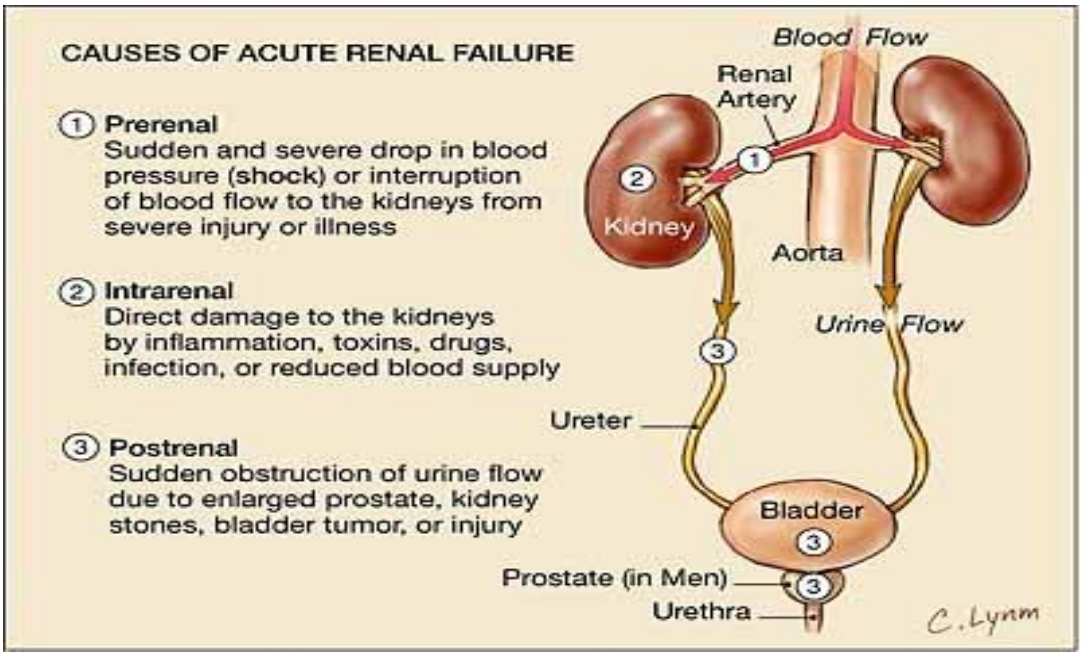
It refers to **sudden** & usually **reversible loss of renal function which develops over period of days or weeks** & **usually accompanied by ↓in urine volume**.

Aetiology

1- **Pre renal causes**
   - **A- Systemic**; Heart failure & blood or fluid loss.
   - **B- Local;** Renal artery occlusion or stenosis& diseases affecting arterioles.

   **Under perfusion initially causes reversible changes. Subsequently → acute tubular necrosis or temporary intrinsic renal failure.

2- **Post renal; Obstruction** e.g. stones, tumour, prostatic enlargement.
3- **Intrinsic renal disease**
   - **A-** Acute tubular necrosis/ toxic/ septic renal failure. 85%.
   - **B-** Glomerular diseases(1ry or component of systemic diseases). 5%.
   - **C-** Interstitial disease. 10%.



Clinical features

They are often dominated by underlying condition

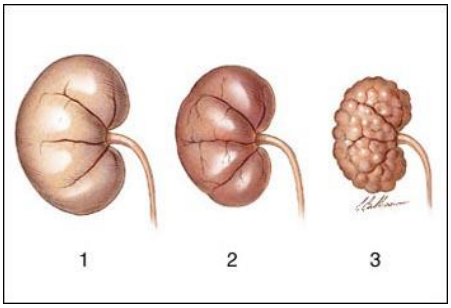
1- **Pulmonary oedema.**
2- **Lethargy.**
3- **↓serum *Na***
4- ***↓urine volume (Oliguria).***
5- **↓ serum *Ca*++.**
6- **↑ serum K+.**
7- **↑ blood urea.**
8- **↑plasma creatinine(>200µmol/l).**

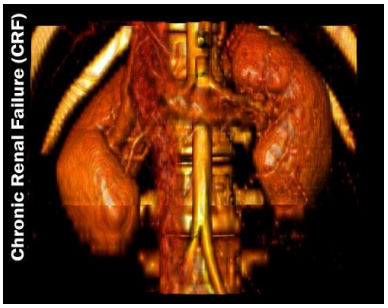
Treatment

1- **Rapidly treat the cause**, otherwise temporary renal replacement therapy may required.
2- **Dialysis if uremia is high**.
3- **Maintain renal flow** by restoring blood volume in shock due to haemorrhage.

4- **Correct hyperkalaemia** by hypertonic solution.
5- **Correct hypocalcaemia** by giving calcium.
6- **Correct acidosis** by I.V. Na bicarbonate.
7- **Treat pulmonary oedema.**
8- **Measuring urine output continuously.**

Chronic renal failure





It refers to an irreversible deterioration in renal function which classically develops over a period of years.

Aetiology

		Proportion of ESRF
1-	Congenital & inherited(polycystic kidney dis.)	5%
2-	**Renal artery stenosis**	5%
3-	**Hypertension**	**5-25%**
4-	**Glomerular diseases(G.N., Pyelonephritis)**	**10-20%**
5-	Interstitial diseases	5-15%
6-	Systemic inflammatory diseases(SLE, Vasculitis)	5%
7-	**Diabetes mellitus**	**20-40%**
8-	Unknown	5-20%
9-	Post renal obstruction.	

Stages of renal failure

Stage 1:Kidney damage with **normal or high GFR** ≥90ml/min/1.73m² .
Stage2: Kidney damage with **slightly low GFR** 60-89 ml/min/1.73 m².
Stage 3: Kidney damage with **moderately low GFR** 30-59 ml/min/1.73 m².
Stage 4: **Severe low GFR** (prepare renal transplantation) 15-29 ml/min/1.73 m² .
Stage 5: **Kidney failure** (prepare renal transplantation) <15 ml/min/1.73 m².

Clinical features

Disturbance in water,electrolyte &acid-base balance contribute to clinical features of CRF.

1- No sign& symptoms in early stage; Especially when renal function deteriorates slowly, patient may remain asymptomatic until GFR falls below 30ml/min/ min/1.73 m²(**stage 4or 5**).

2- Nocturia due to ↓ability to concentrate urine.

3- Symptoms due to widespread effect of CRF to almost every body system;

- **a- Anaemia& vague ill health.**
- **b- Breathlessness on exertion & tiredness.**

c- **Hypertension &↑JVP**(fluid overload).
d- **Headache, pallor& yellow complex(earthy color).**
e- **Pruritus & excoriation of skin with bruising easily.**
f- **Loss of libido.**
g- **Pericardial friction rub & pleural effusion.**
h- **↓Sensation. Paraesthesia, restless leg.**

4- Symptoms of end stage renal failure(ESRF-Stage 5); Hiccough, deep respiration(metabolic acidosis), anorexia, nausea, vomiting, muscle twitching, drowsiness, fit & coma→ death.

Adverse effect of CRF

1- **Anaemia** (Deficiency of erythropoietin hormone).
2- **Fluid & electrolyte imbalance.**
3- **Cardiovascular diseases**(occlusive CV Diseases, HT ,Hypercholesterolemia).
4- **Infection** (Hepatitis due to impairment of cellular& humoral immunity) .
5- **Bleeding.**
6- **Renal osteodystrophy** (due to poor nutrition, vit. D deficiency& electrolyte metabolism disturbance, hyperparathyroidism).
7- **Myopathy.**
8- **Neuropathy.**

Investigation

1- **Hematology; Full blood count.**
2- **Biochemistry**
   a- **↑ Serum urea, electrolyte(↓Na, ↑K),↑creatinine.**
   b- **↓ Ca++,↑phosphate& albumin.**
   c- **↑Parathyroid hormone .**
3- **Microbiology; HIV & hepatitis serology test.**
4- **Imaging**
   a- **Renal ultrasound.**
   b- **Chest X ray.**
   c- **ECG.**
   d- **Renal artery imaging.**
5- **Renal biopsy.**
6- **Immunology for renal transplantation.**

Management

1- **Control fluid & electrolyte balance .**
2- **Maintain nutrition**
   a- **High carbohydrate & fat.**
   b- **Low protein diet** to suppress endogenous protein catabolism(60gm/day).
   c- **High fluid intake** (3 liters/day) because kidney cannot concentrate urine .
   d- **Parenteral nutrition** may be required because of vomiting & diarrhoea.
3- **Control biochemical abnormality**
   a- **Low K+** (70mmol/day).
   b- **High NaCl** (5-10gm) if no hypertension& heart failure, or give **Na bicarbonate** when acidosis is present.

c- High Ca++ intake.

d- ↓Lipid by HMG-CoA reductase inhibitor**.**

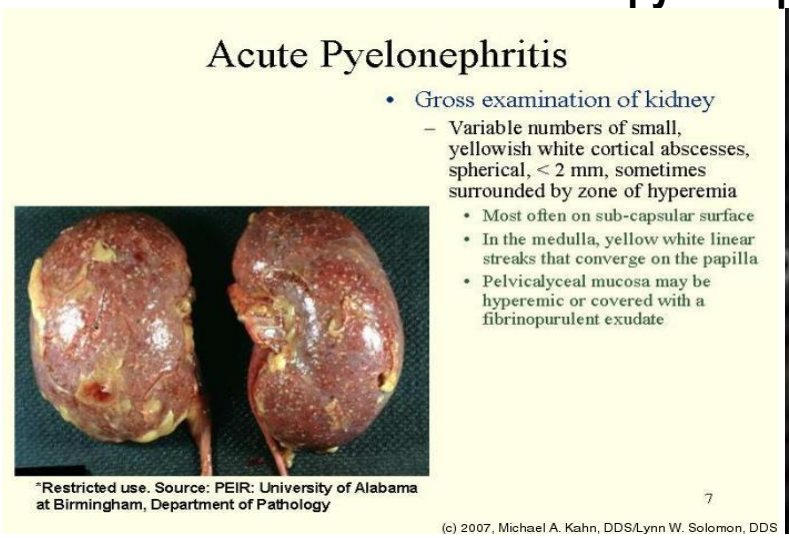
4- Dialysis (↓bleeding tendency).

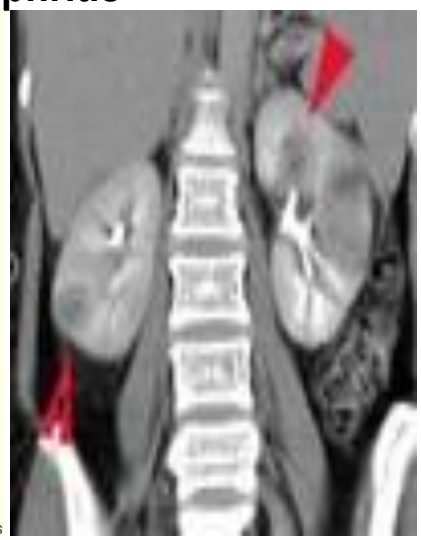
5- Protect patient from infection by appropriate antibiotic drugs.

6- Renal transplantation.

Pyelonephritis

Acute pyelonephritis





It refers to the renal pelvis inflammation and small abscesses in the renal parenchyma.

**** More common in females**(after marriage as complication during honey moon cystitis & during pregnancy & menopause) and **in childhood.**

****Frequently bilateral but if unilateral , Rt. Side is more common.**

Aetiology

The causative micro organism reachs kidney by;

1- Haematogenous route from any infective focus in the body.

2- Ascending route from urinary bladder.

The commonest micro organisms

1- E.coli. & other gram –ve organism. (make acidic urine).

2- Streptococcal fecalis. (make acidic urine).

3- Proteus spp. . (make alkaline urine).

4- Staphylococcus. (make alkaline urine).

5- Mycobacterium.

Clinical features

1- In infant & children; **Fever without localizing symptoms**& there may be convulsion, abdominal distention & diarrhea.

2- In adult; **Fever with localizing symptoms.(≥39° C).**

3- Rigor(due to septicaemia**) with headache& vomiting.**

4- Sudden onset of loin pain(unilateral or bilateral) **radiate to groin region.**

5- Tenderness & guarding in flanks (lumbar region).

6- Cystitis features(Urinary frequency , urgency & scalding dysuria 30%).

7- **Rarely** in severe bilateral pyelonephritis especially. associated with obstruction lead to renal dysfunction and uraemia.

Investigations

1- **General urine examination**(**GUE**);**Cloudy, pus cells, bacteria, red cells**, **WBC are plentiful** &epithelial cells.
2- **Culture of mid stream urine**(**MSU culture & drug sensitivity**).
3- **Pyelography (urography).**
4- **Renal biopsy**; Show focal infiltration by polymorphonuclear leucocytes & presence of these cells within tubules.
5- **Full blood count; ↑↑WBC.**
6- **Renal function test (** Serum urea, creatinine & electrolytes**).**
7- **Blood culture.**

****Diagnosis depends on clinical features & urine culture result.**

Differential diagnosis

1- **Acute appendicitis.** 2- Diverticulitis.
3- **Cholecystitis.** 4- Salpingitis.
5- **Perinephric abscess**(blood culture +ve, no pus or bacteria in urine).

Complications

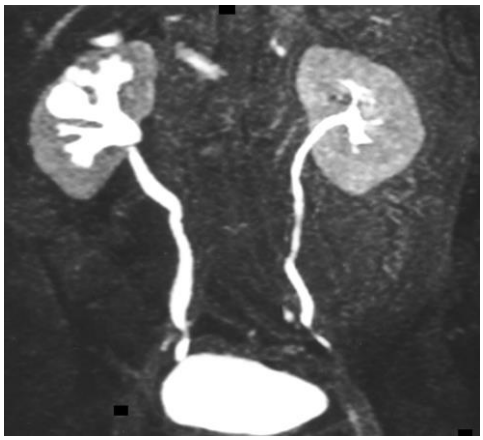
1- **Chronic pyelonephritis.**
2- **Urinary obstruction.**
3- **Hydronephrosis.**
4- Pyonephrosis.
5- Acute papillary necrosis(fragment of papillae are excreted in urine).
6- **Renal failure**(especially in D.M..& chronic urinary obstruction).
7- **Septicaemia & hypotension.**

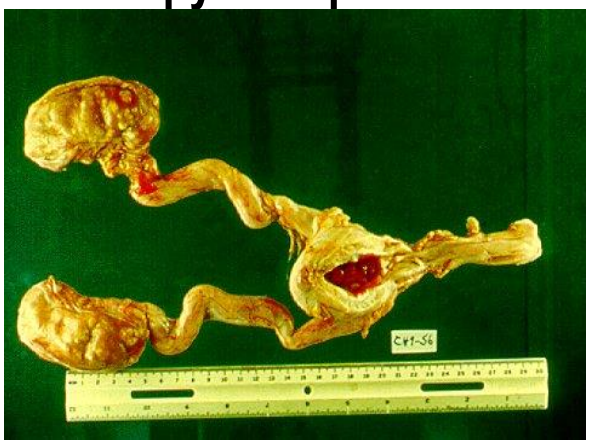
Management

**Early correction & prolonged treatment is necessary with careful follow up.

1- (**MSU culture) & drug sensitivity**
2- **Proper antibiotic**. In waiting the result of culture should start broad spectrum antibiotic as; Co-trimethoxasole(metheprim), Cephalosporine, Amoxycillin Ciprofloxacune .
In severe case use I.V. therapy.
3- **Treatment is continued for 10 days then replaced by another drugs for 2-3 weeks. Urine culture should be repeated during course ,7th & 21 day after treatment.**

Chronic pyelonephritis





****It is more dangerous than acute, it is associated with vesico-ureteric reflux(reflux nephropathy) →(common cause of ESRF).**

****2/3 of female patients are under 40years of age, 60% of male are over 40.**

Aetiology

1- Mostly secondary to acute one.
2- Anomalies of urinary tract.

Clinical features

1- **<u>May be symptomatically silent</u>** lead to progressive renal scarring.
2- **<u>Lumbar pain</u>(or low back pain), dull**, **non specific** i**n 60% of cases**.
3- **<u>Cystitis features</u>**(Urinary frequency , urgency & scalding dysuria).
4- **Constitutional symptom;** Malaise, headache, nausea & lassitude**(30%).**
5- **Pyrexia**.
6- **Anaemia**.
7- **<u>Hypertension in 40% of cases</u>**, may be of malignant type.

Investigations

Same like acute pyelonephritis (proteinuria is less marked than G.N.)(<3gm/day).

Complications

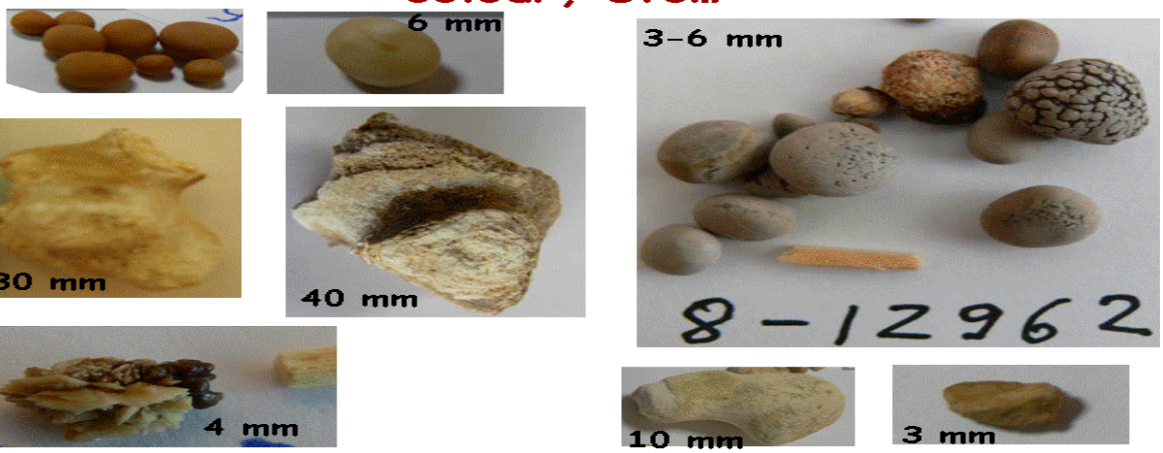
1- Pyonephritis. 2- Hydronephrosis.
3- Nephrocalcinosis(deposition of Ca++ throughout renal parenchyma).

Treatment

1- **Treat the cause.**
2- **Repeated MSU culture, drug sensitivity & proper antibiotic for infection.**
3- **Nephrectomy (or partial) if unilateral Pyonephritis developed**.
4- **Renal transplantation in case of ESRF**.

Urinary calculi

Urinary stones can vary in size, shape, colour, etc…

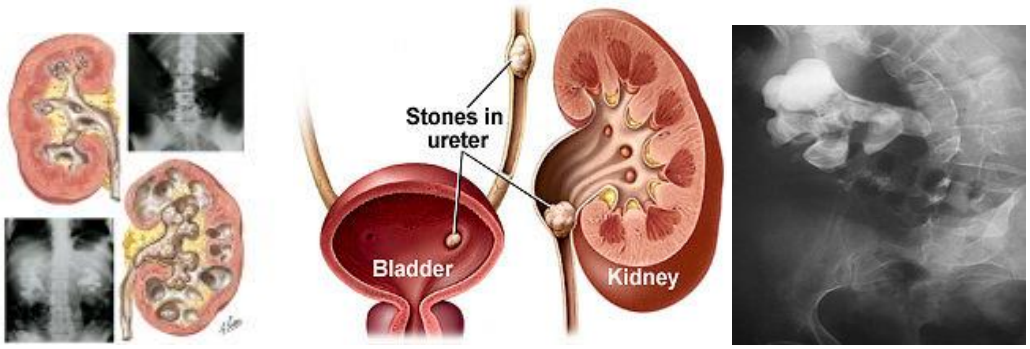


**They consist of aggregates of crystals & small amount of protein & glycoprotein but their genesis are poorly understood.

Calculus may be in renal calyces or pelvis, ureter , bladder or urethra.

Mostly is formed in renal pelvis & urinary bladder. The ureteric & urethral stones one or more due to descending of renal or vesicle stones, most of these stones are mixed.

Renal calculi



Aetiology

1- **Dietetic; Vitamin A deficiency**→ Desquamation of epithelium, the cells form a nidus on which a stone is deposited.

2- **Altered urinary solutes & colloids and PH of urine**

--**Dehydration** →↑ concentration of urinary solutes until they liable to precipitate.

--**↓urinary colloids** (which adsorb solutes), or mucoprotein (which chelate calcium)→might result in atendency for crystal & stone formation.

--**Alkaline urine** →↑ precipitation of calcium, phosphate & may responsible of calcium phosphate stone in renal tubular acidosis.

--**Acid urine** → comfortable for Cystine & uric acid stone.

3- **Renal infection** ; Common in urea-splitting strep., staph. especially proteus species,

and E, coli..

4- ↓ **urinary citrate**; urinary excretion of citrate under hormonal control & decreased during menstruation.
5- **Inadequate urinary drainage & urinary stasis** → stone liable to form.
6- **Prolonged immobilization** (Parplegia)→ Skeletal decalcification→↑ urinary calcium → calcium phosphate calculus.
7- **Hyperparathyroidism**→ Hypercalcaemia & hypercalciuria (Pass their skeletons in their urine) so Parathyroid adenoma should be removed.

Types of calculi

1- **Oxalate calculus(Calcium oxalate); (39%);** Irregular, covered with sharp projection→ **Bleeding**. Ca++ oxalate monohydrate is hard & **radio dense**.
2- **Phosphate calculus(mostly calcium phosphate);(13%);** Smooth, dirty white, **grow in alkaline urine** especially in presence of urea-splitting proteus organism. May enlarge to fill all collecting system forming **Stag horn calculus** (Silent for years then → Haematuria & urinary infection & renal failure. It is easy to see on radiographic film.
3- **Uric acid & urate calculi;** Hard ,smooth, often multiple & yellow to reddish brown. Pure acid stones are **radiolucent**(appear in excretion urogram as filling defect).
4- **Cystine calculus;** Uncommon, **appear in acid urine**, very hard,often multiple & grow to form a cast of collecting system. Yellow or pinck change to a greenish colour when exposed to air. It radio opaque because contain sulphur
5- **Xanthine calculus**; Extremely rare, smooth, round, red in color.

Clinical features

**50% of patients present between the ages of 30 & 50 years.
**Male: female ratio 4:3.

1- **Silent calculus**; Even large stag horn may cause no symptoms for long period during which time there is progressive destruction of renal parenchyma, uremia may be the first indication of bilateral stones although secondary infection usually produce symptom first.
2- **Pain**; **It is the leading symptom in 75%** of patient with urinary stones.
   A- **Fixed renal pain**, located posteriorly in the renal angle, anteriorly in the hypochondrium or in both. It may **worse on movement, particulary on climbing upstairs**
   B- **Ureteric colic**; It is due to stone entering ureter or lodged in pelvi-ureteric junction. It is an **agonizing pain passing from the loin to the groin, penis, scrotum or labium. It starts suddenly causing patient to writhe to find comfort** (pattern of severe exacerbation on a background of continuing pain). The pain rarely last more than 8 hours in absence of infection.
3- **No pyrexia but pulse rate is increased** due to severe pain.
4- **Pallor, sweating & vomiting.**
5- **Haematuria**; **Is leading symptom to stone.**
6- **Pyuria**; Due to infection→ septicemia(Rigor).
7- **Abdominal examination**
   A- **Rigidity of lateral abdominal muscle during attack of ureteric colic**.
   B- **A stab of pain** if percussion over kidney, with **tenderness** on gentle palpation.
   C- **Palpable loin swelling in case** of hydronephrosis or pyonephrosis(rare).

Investigation

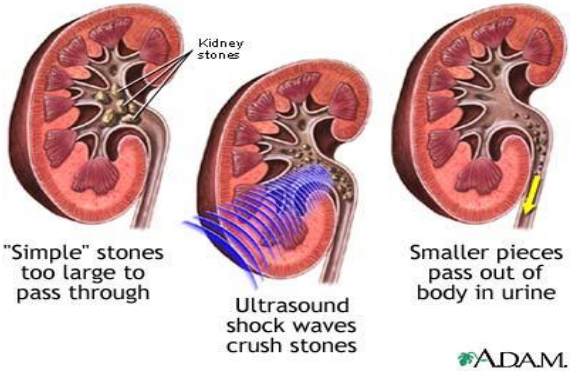
1- **Urinary tract investigations**; **GUE, MSU culture & radiography** ;
   a- **KUB**(An opacity that maintains its position relative to the urinary tract during respiration is likely to be a calculus.
      Differential diagnosis:
      a- Calcified mesenteric L.N.
      b- Gall stone.
      c- Tablet or F.B. in alimentary tract.
      d- Ossified tip of 12th rib.
      e- Calcified T.B. lesion in kidney.
      f- Calcified adrenal gland.
   b- **IVU (IVP)**; show stone, obstruction & abnormality of urinary tract.
2- **Renal function test** ; Blood urea, serum creatinine &creatinine clearance.
3- **Investigations to determine underlying cause**; By **chemical analysis of stone**, plasma Ca++, phosphate, plasma parathyroid hormone
4- **Contrast- enhanced CT scan.**
5- **Excertion urograph.**
6- **Ultrasound scaning.**

Treatment

1- **Most small stones(≤5mm) will pass spontaneously** & can be treated **conservatively:**
2- **Bed rest, warmth to site of pain.**
3- **Treat the colic** by I.V. hyoscine or voltarin or pethidine or morphin (10-20mg + atropine sulphate 0.8-1.2mg).
4- **Treat the infection** pre & post operatively.
5- **Drink 2-3 liters of fluid/day.**

Presence of infection in upper urinary tract obstruction by stone is danger & need urgent **surgical operation which include;

A- **Open operation**; Pyelolithotomy, nephrolithotomy, partial nephrectomy when stone in lower calyx & complete nephrectomy when kidney is damaged & other kidney is work.
B- **Lithotripter**; Extracorporeal shock wave lithotripsy**(ESWL**) or by nephroscopy & erasing stone by ultrasound lithotripter or electrohydraulic probe.



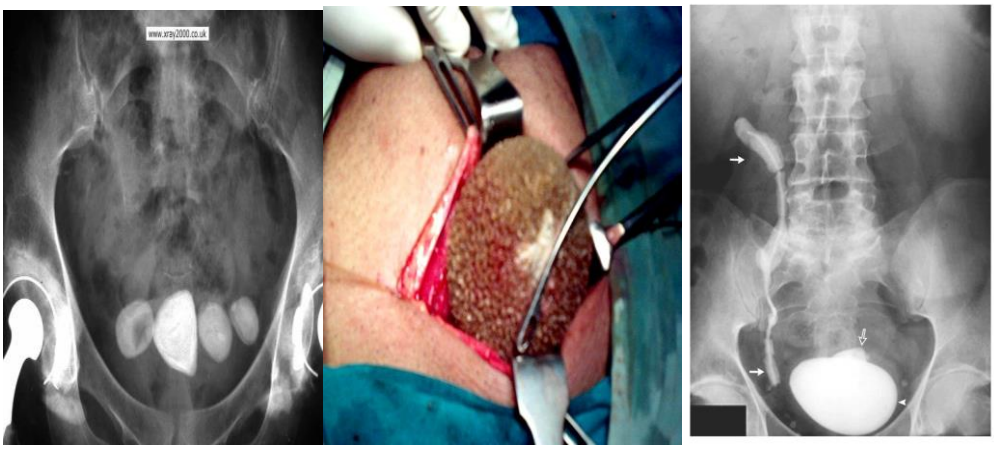
C- Endoscopy; Percutaneous nephrostomy in renal stone, ureteric stone & also can use endoscope via bladder(**Cystoscopy**).

**** In bilateral stones remove the stone of better functioning kidney.**

Prevention of recurrence

1- **Treat hyperthyroidism& gout**.
2- **Diet**; High fluid intake, low purine diet, low calcium oxalate diet as rhubarb, spinach &asparagus acid. **Lemon juice**→↑ **citrate excretion**, while hypokalaemia→↓ citrate excretion.
3- **Alkalinization of** urine is benefit in case of uric, urate & cystine stone.
4- **Acidify urine is benefit in phosphate calcium calculi**(by giving ammonium chloride).
5- **Avoid any preparation containing vitamin D,** also reduce Na especially for idiopathic hypercalciuria & use Thiazid.
6- **Allopurinol** 100-300mg/day is useful in uric acid or urate stone & also for calcium oxalate.

Vesicle stone(Stone in urinary bladder)



** The stone usually comes from the kidney (**secondary**), or due to infection of bladder(**primary**)

Clinical features

1- **No symptom.**
2- **<u>Frequency</u> & <u>dysuria.</u>**
3- **<u>Haematuria.</u>**
4- **<u>Urine retention.</u>**

Treatment

1- **<u>Endoscopy</u>**(Cystoscopy).
2- **<u>Cystotomy</u>** if large stone(Suprapubic lithotomy).

3- Electro hydraulic **lithotripsy.**

Treatment of ureteric stone

1- Treat of colic.
2- Treat of stone by;
   A- Expectant to descend by itself.
   ****Removal of stone in case of large, not descending, obstructing & associated with infection**.
   B- By **ureterolithitomy** or by **endoscopy via bladder** or **Percutaneous nephrostomy** or pushed into pelvis & treat it like pelvic stone.

Lower urinary tract infection(LUTI)

**Imply multiplication of organism in the urinary tract & is defined by presence of more than 100,000 organisms/ml in MSU.
****More common in women** about 1/3 of females (due to short urethra & absence of bactericidal prostatic secretion). 3% at age 20 & about 1% in each subsequent decade.
**In male is uncommon exception 1st year of life & over 60 year due to prostatic hypertrophy.
**Urinary tract infection cause considerable morbidity & small minority lead to renal damage & failure.
**LUTI takes two stage to develop ; 1st is colonization. 2nd multiplication which depend on size of the inoculums & virulence of bacteria.
** **LUTI include; 1- Cystitis. 2- Urethritis. 3- Prostatitis**

Causes & mode of infection

1- **Faecal E. coli (75%),** strept. & staph. Proteus one is the most.
2- **Coitus**(cause minor urethral trauma & transfer bacteria from perineum to bladder).
3- **Obstruction** due to any causes.
4- **Low urine outflow rate.**
5- **Residual urine**(due to bladder outflow obstruction, pelvic flow weakness, neurological problem & gynecological abnormality) which interfere with mucosal defense mechanism.
6- **Instrumentation of bladder**
7- **Haematogenous.**
8- **Ascending.**

Clinical features

1- **Cystitis features(Frequency & dysuria).**
2- **Urethral scalding.**
3- **Suprapubic pain**
4- **Urgent desire to remain & pass urine after voiding.**
5- **Pyrexia & rigor.**
6- **Haematuria**

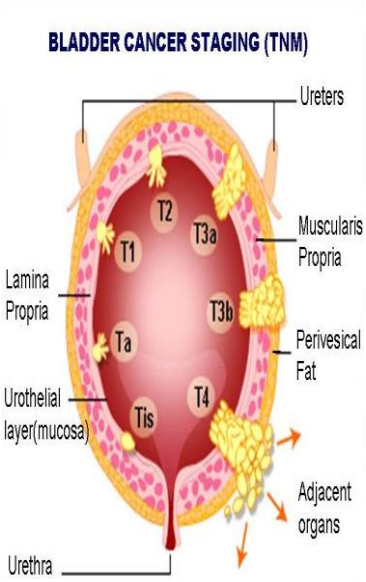
Investigation

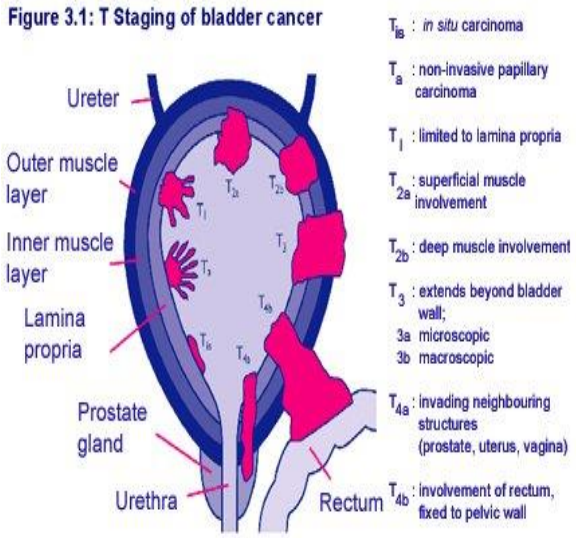
Bacteria more than 100,000/ml in MSU.

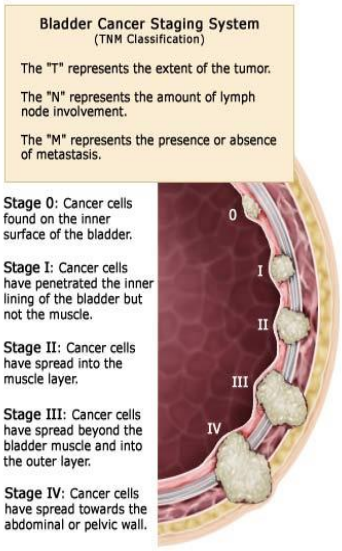
Management

1- **MSU culture & drug sensitivity.**
2- **Start with broad spectrum antibiotic**, metheprim & amoxyicillin, **then** change according to result of culture.
3- **High fluid intake.**
4- **Proper emptying of bladder & frequent emptying before retiring, before & after intercourse**.
5- **Treatment of both couple.**

Carcinoma of bladder







Histologically include
1- **Urothelial (bladder lining)(90%).**
2- Squamous (5%).
3- Adenocarcinoma(Or mixed as result of metaplasia in a transitional cell carcinoma) (1%).

It is 4th most common non-dermatological malignancy in men (male: female=3:1). It is an **industrial disease.

Aetiology

1- **Cigarette smoking** is main aetiological factor (**40% of cancer**).
2- **Occupational chemical exposure** as; 2,naphthylamine , benzidine, etc.. And the workers with high risk are; Textile, dye, tyre rubber, cable, chemical, sewage, rodent exterminator, petrol, leather workers. Also shoe manufacturers, cleaner, painter. Hair dresser, lorry drivers, drill press operators .
3- **Genetic event** has been clearly implicated in cancer formation.

4- **Strongly associated with schistosomiasis haematobium** infection in regions where the parasite is endemic.
5- **Following chronic inflammation or irritation due to stone.**

Staging & grading

1- **Non- muscle invasion** (**pTa**)(no invasion of lamina propria) ,(**pT1**)(invasion of lamina propria) account for 70% of new cases . Excellent prognosis.
2- **Muscle- invasion** disease, account for 25% of new cases .
3- **Flat, non- invasion(1ry Cis**), account for 5% of new cases..

Clinical features

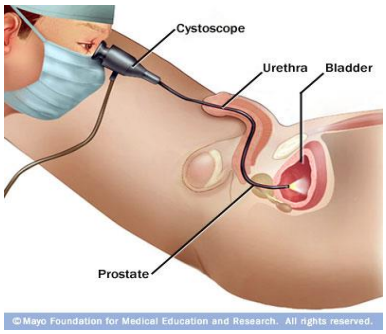
1- **Haematuria;>80%, Gross, painless**& there may be clot formation& retention.
2- **Constant pain in pelvis**.
3- **Obstructive symptom(loin pain)** due to ureteric obstruction& pyelonephritis.
4- **Frequency & discomfort** associated with urination.
5- **Late feature, nerve involvement, causing pain in suprapubic, groin,anus**.

Investigation

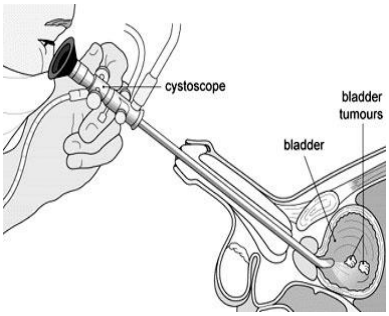
1- **Urine; Culture & cytological examination for malignant cell**.
New test depend on presence of **antigens NMP22**(nuclear matrix protein) orMCM protein, which may able to detect new or recurrent tumour.
2- **Blood; Hb & level of serum electrolyte & urea.**
3- **IVU or ultrasound scanning;** Should performed in all patients with painless haematuria .The film show **filling defect or indentation in wall of bladder.**
4- **Ct-scan.**
5- **Cystourethroscopy**; It is the **mainstay of diagnosis** which done under local anesthesia(use flexible instrument) or general anesthesia(use rigid one).
6- Bimanual examination with full relaxation of patient under general anesthesia, before & after endoscopocal surgical treatment with empty bladder.

Management

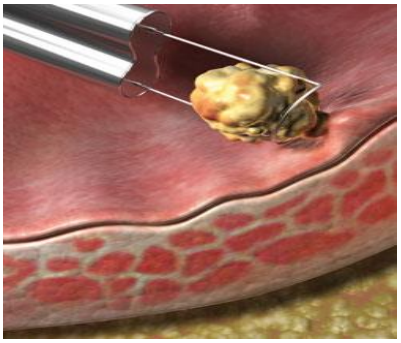
1- **Endoscopically by transurethral resection of tumour(TURT).**For Small, large, multiple bladder tumour**.**
2- **Intravesical chemotherapy** (e.g. eprubicin, mitomycin C) used for multiple low grade tumour.
3- **Regular check cystscopies are required & recurrence can usually be controlled by diathermy** & raely cystectomy be required for superficial disease.
4- **Intravesical BCG**(bacilli Calmette-Guerin);For carcinoma in situ-Cis-, with well respond.
5- **Radical Cystectomy** for invasion bladder tumour with urinary diversion into an incontinent ileal coduit .



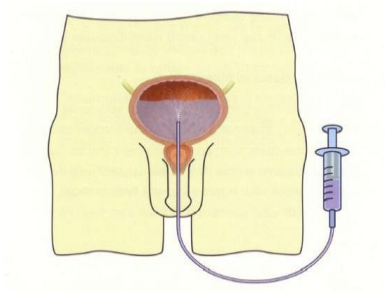
flexible Cystourethroscopy



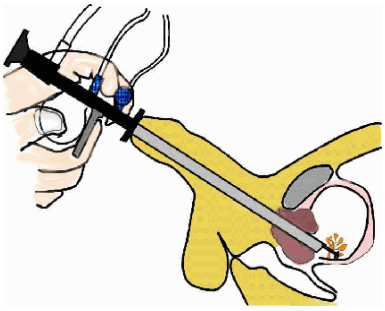
rigid Cystourethroscopy



transurethral resection of tumour

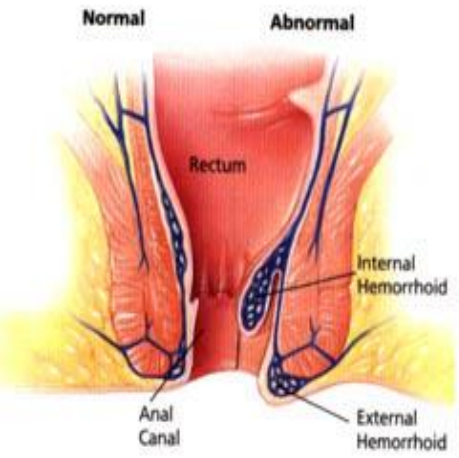


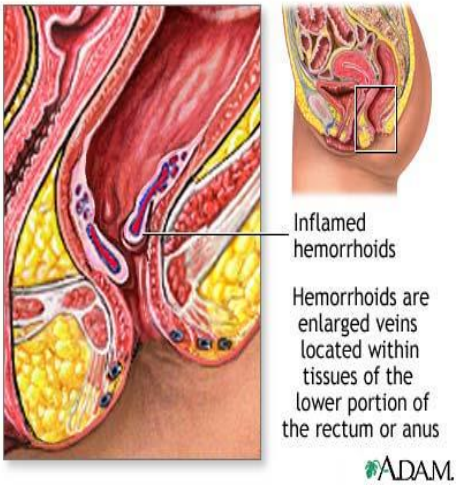
Intravesical BCG

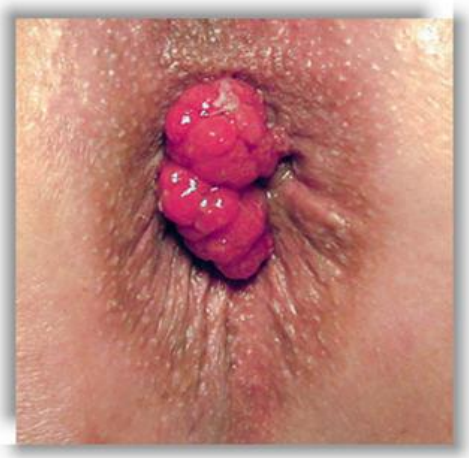


Intravesical chemotherapy

Hemorrhoids(Piles)







Definition; Dilated veins within anal canal(Varicosity), in subepithelial region formed by radicals of superior, middle &inferior rectal veins.

Classification of piles

A- According to site or region

1- **Internal**;(**Varicosity** of internal hemorrhoid plexus), within internal canal & **internal to anal orifice**, covered by mucous membrane, bright red in colour.

2- **External;** (Varicosity of external hemorrhoid plexus), at anal verge, **outside anal orifice**, covered by **skin**, firm, blue, or purple & painful.
3- **Internoexternal** ;(Varicosity of both hemorrhoid plexus).

B- According to pathological anatomy

1- **Primary;** Three numbers; 3,7 & 11 O'clock positions.
2- **Secondary;** Additional piles between primary piles.

C- According to prolapsed

1- **First degree;** Vein congested& bleeding(splash of pan) during defecation **(not prolapsed)**.
2- **Second degree**; Piles come out (**prolapsed**) only during defecation & is **reduced spontaneously** after defecation.
3- **Third degree;** Piles come out (**prolapsed**) during defecation & **reduced manually**.
4- **Fourth degree ; Permanently prolapsed** & feeling of heaviness & discomfort in rectum.

Aetiology

1- **Primary(idiopathic) causes**
   a- **Hereditary.**
   b- **Morphological(anatomical) factors**; Absence of venous valves in rectal vein, gravity, loose submucosa, constriction of smooth muscle.
2- **Secondary causes**(precipitating factors→↑ **intra abdominal pressure**).

a- **Chronic constipation.**
b- **Tenismus.**
c- **Pregnancy.**
d- **Abdominal tumour.**
e- **Lifting heavy weight & coughing.**
f- **Straining during micturation & defecation.**
g- **Portal obstruction.**
h- **Diet & sedentary habits**.

Clinical features

1- **Bleeding (painless)→ anaemia.**
2- **Prolapsed.**
3- **Pruritis** due to mucous discharge.
4- **Pain may present due to complications** as anal fissure or carcinoma.
5- **Proctoscopy show bulging of piles into lumen of proctoscope.**

Laboratory studies

1- Haematocrit; Total blood count, differential count, Hb., ESR, Bleeding time, clotting time, blood sugar, blood urea, etc ..

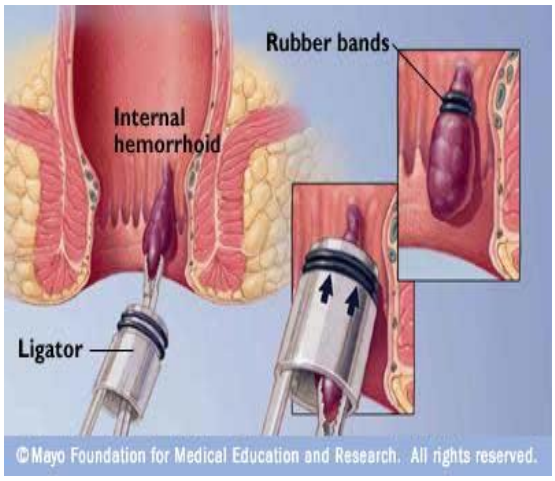
2- Urine; Routine & microscopically.
3- Stool; Occult blood, ova & cyst.

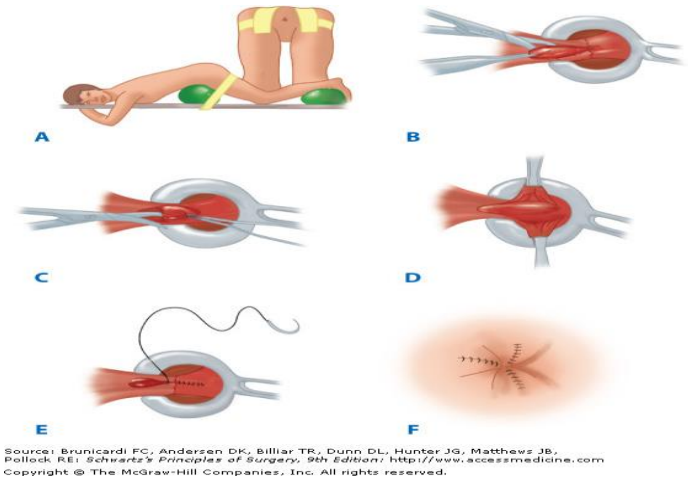
Complications

1- **Profuse bleeding.**
2- **Thrombosis.**
3- **Strangulation→ Gangrene.**
4- **Fibrosis & anal stricture.**
5- **Anal fissure, ulceration & suppuration.**

Management

1- **Preventive**; Avoid& treat precipitating factors.
2- **Curative**
   a- **Medical;** Use glycerin & tannic acid ointment or suppositories or hot sponge with analgesia.
   b- **SURGERY:**
      1- **Non- operative treatment;** Injection of 5% phenol in almond oil, plastic bandage ligation, cryosurgery, photo coagulation.
      2- **Operation; Hemorrhoidectomy & anal sphincter dilatation.**

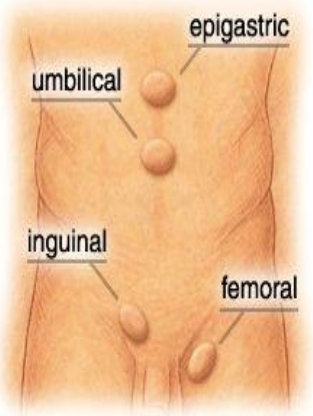


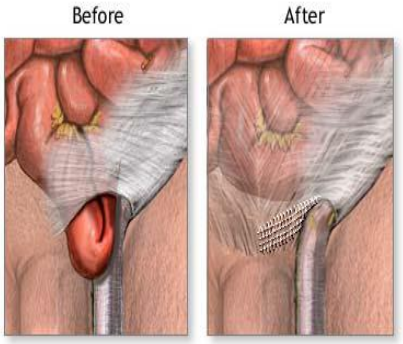


Post operative complication

1- Pain.
2- Bleeding.
3- Urine retention.
4- Anal stricture.
5- Anal fissure.

Hernia





Definition; **Protrusion of viscus or part of viscus through an abnormal(defect) opening in the wall of its containing cavity**.

Aetiology

1- **Hereditary**; As an indirect hernia occure in congenital performed sac (the remains of the processus vaginalis).
2- ↑ **Intra-abdominal pressure** due to any causes;
   a- **Straining** on micturation or defecation(in adult).
   b- **Coughing**(Whooping cough in children) or chronic cough.
   c- **Jumping** from height or lifting heavy weight.
3- **Weakness(stretching) of the abdominal musculature** as in;
   a- **Obesity.**
   b- **Pregnancy.**
   c- **Intra-abdominal malignancy.**
   d- **Post traumatic hernia after operation(incisional hernia).**
   Male to female 20:1.

Composition of hernia

1- **The sac**; **Diverticulum of peritoneium , consisting of mouth, neck, body, & fundus.** Neck diameter is important because strangulation of bowel is related to it as in femoral & paraumbilical hernia.
2- **Covering of the sac**; Derived from **layer of abdominal wall** through which the sac passes.
3- **Contents of the sac**; Omentum (omentocele), Intestine(enterocele), portion of circumference of intestine, a portion of bladder(part or sole content of direct inguinal hernia), ovary, or fluid of ascitis.

Classification of hernias

1- **Reducible**; **Content of hernia can be returned to abdomen**(by lying down or manually).

2- **Irreducible; Content of hernia cannot be returned** but there are no other complication(Due to adhesion between the sac & its contents or overcrowding).
3- **Obstructed; Bowel(Content) in the hernia has good blood supply but bowel is obstructe**d(colicky abdominal pain, tenderness over hernia site & less severe).
4- **Strangulated**; **Blood supply of bowel in hernia is obstructed** (Blood supply of its contents is seriously impaired→ **ischemia**→ **gangrene** as early as 5-6 hours after onset of 1st symptom.
5- **Inflamed; Content of sac here become inflamed**.

The types of hernia according to sites

1- **Inguinal (Direct & indirect-is the most common hernia of all especially in young while direct hernia more common in elderly-) hernia.**
2- **Umbilical hernia.**
3- **Paraumilical hernia.**
4- **Epigastric hernia.**
5- **Femoral hernia.**
6- **Incisional hernia.**
7- **Hiatus hernia.**

Clinical features of hernias(Reducible)

1- **Swelling with pain on groin or refered to testicle when performing heavy work or taking strenuous exercise.** In large hernia a sensation of weight & dragging on the mesentery 7 this may produce epigastric pain.
2- **Expansible impulse on coughing**.
3- **Reduced on lying down or manually.**
4- **Audible gurgle on reduction**.
5- **Doughy sensation on pressure** on sac(sometimes lumen of that portion of colon occupied a hernia sac is blocked with feces→ capable of being indented with finger, like putty).

Clinical features of strangulated hernia

a- **Pain; Sudden, initially situated over hernia then followed by generalized abdominal pain, colicky** in character often locates mainly at umbilicus.
b- **Nausea & subsequently vomiting.**
c- Patient may complain of an ↑ **in size of hernia.**
d- **On examination; hernia is tense & extremely tender & irreducible with no expansile cough impulse.**

e- **Unless relieved by operation→ Spasm of pain** continuing until peristaltic, contraction cease with the onset of **ischemia**, when paralytic ileus & **septicaemia** developed. Spontaneous cessation of pain must be viewed with caution as this may be a sign of **perforation**.

f- **Required urgent operation.**

Preoperative treatment of strangulated hernia include

1- **Resuscitation with <u>adequate fluids</u>,**
2- **<u>Empty stomach</u> with nasogastric tube,**
3- **<u>Give antibiotic</u> to contain infection,**
4- **<u>Catheterize</u> to monitor haemodynamic state, <u>empty bladder</u> if necessary by catheterization.**

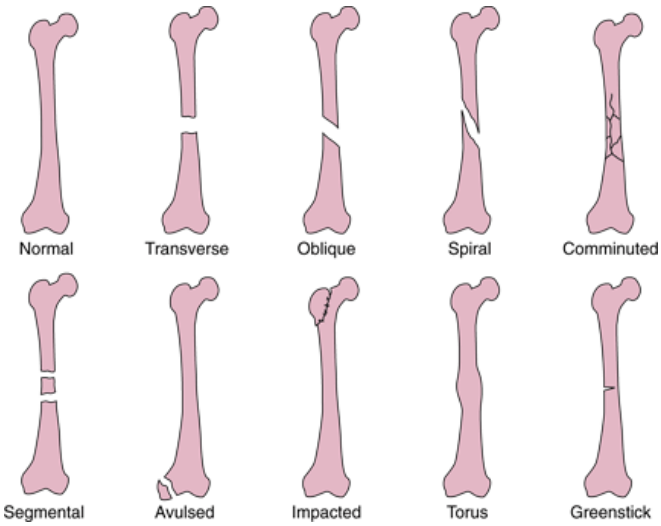
Differential diagnosis of inguinal hernia

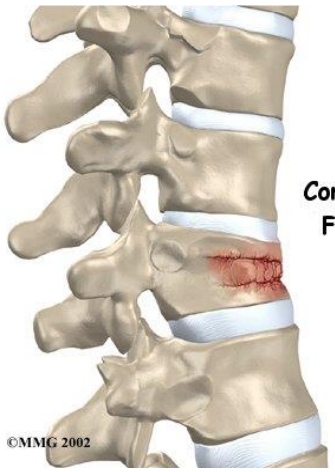
1- **Vaginal hydrocele**.
2- Encysted hydrocele of cord.
3- Spermatocele.
4- **Femoral hernia**.
5- **Incompletly descended testis in inguinal canal.**
6- Lipoma of cord.

Treatment

1- **Surgery** is treatment of choice; herniotomy, herniotomy & herniorrhaphy or hernioplasty.
2- **Supportive treatment**; After operation no smoking, no heavy weight, no obesity & treat constipation & urinary tract obstruction.
3- **Conservative treatment by Truss** when operation is contraindication & only for reducible.

Fractures





Compression Fracture

Definition; **Break in the normal continuity of bone**

Types of fracture (#)

1- **Simple** #; Bone is broken into 2 pieces.
2- **Comminuted** # ; Bone is broken into more than 2 pieces.
3- **Compound** #; Fracture with open wound.
4- **Compression #;**Vertebral fracture due to fall from hight on buttock or heel.
5- **Traction #;** Traction, twisting or vigorous muscles contraction →avulsion of pieces of bone.
6- **Impacted #** ;Direct or indirect trauma to cancellous bones.
7- **Pathological #** ; Due to bone diseases as tumour, osteoporosis.
8- **Stress # .**
9- **Depressed #**; As skull #.
10- **Green stick #;** In children.

Aetiology

1- **Direct trauma.**
2- **Indirect trauma**(# of vertebrae, scphoid, & lower radius).
3- **Vigorous muscle contraction.**
4- **Sudden twisting.**
5- **Bone diseases;** Tumour, osteoporosis, multiple myeloma.
6- **Stress.**

Clinical features

1- **Local pain.**
2- **Tenderness on # site.**
3- **Later on swelling & bruising.**
4- **Impairment of function.**

5- **Deformity as angulations, twisting & over riding of # parts.**
6- **Abnormal movement with crepitus.**

Diagnosis; By radiology in 2 views.

Management

A- General management

1- **Analgesic f**or pain & **splintage**.
2- **Blood transfusion** if needed.
3- **Vital signs should be assessed** .
4- **Treatment of associated injuries.**
5- **Vaccination of tetanus.**
6- **Antibiotic.**

B- Local management

1- **Treatment of wounds, tissue, veins or nerves injuries.**
2- **Reduction** if needed by;
   a- **Closed reduction manipulation.**
   b- **Open reduction(surgery).**
   c- **Gravity**; --Collar & cuff, --Banging cast.
3- **Fixation(immobilization)**

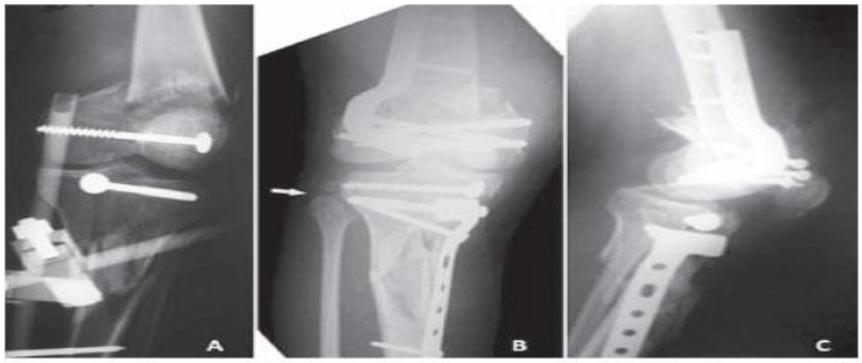


***Figure 1** – X-ray controls for clinical case nr. 2: (1-A) X-ray image at AP plane: temporary early stabilization with tubular external fixator and minimal osteosynthesis with spongy screws and washers for reducing femoral and tibial joint surfaces. (1-B) X-ray image at AP plane: permanent synthesis and detail of the fibular head bone avulsion (arrow) and (1-C) X-ray image at lateral plane: permanent synthesis.*

   a- **External splintage**(plaster, plastic cast external fixator).
   b- **Internal**(operation) by screw, plate, nail, wire.

C- Rehabilitation.

Reduction is needed if there is displacement of # bone.

During immobilization healing depend on ;

Age, Type of #, Area of #, Patient nutrition, Complication.

Indications for operation

1- **Compound #.**

2- **Reduction of # if needed internally.**
3- **For stabilization.**
4- **For shortening the time.**
5- **Soft tissue management.**
6- **Complication management**.

Complications

1- **Immediate complications**
   a- **Skin, vessel, nerve, muscles & visceral injuries.**
   b- **Haemorrhage→ Shock.**
2- **Early complications**

Local

   a- Volkmann's ischemia.
   b- Venous thrombosis.
   c- Infection & # blister.

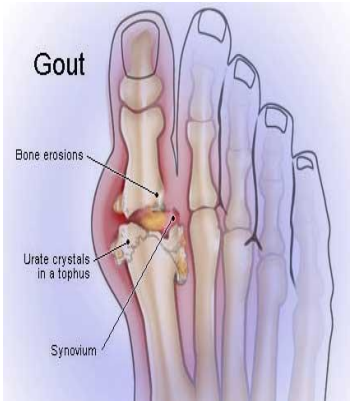
General

   a- Fat embolism.
   b- Pneumonia.
   c- Tetanus.
   d- Bed sores.

3- **Late complication**
   a- Non union & false joint formation.
   b- Shortening.
   c- Refracture.
   d- Chronic infection of bone.
   e- Joint stiffness.
   f- Muscle fibrosis & atrophy.

.

Gout



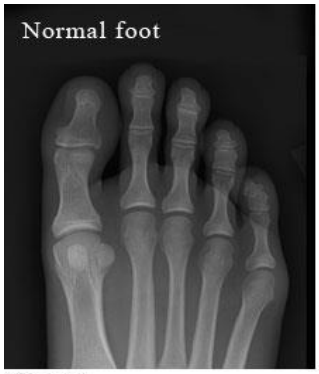


Figure 1

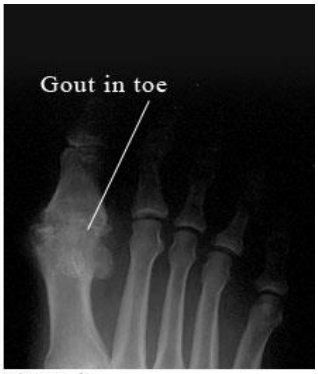


Figure 2

The disease is due to <u>increase body uric acid</u> <u>leading to inflammatory reactions in the joints, synovial membranes, bursa and other tissues.</u>

Epidemiology and aetiology

There is positive correlation between serum uric acid level (Normal level of serum uric acid is 0.36-0.42mmol/l)and ; Urban, Hb, Weight, Intelligence, Social classes and high protein diet. So;

1- **Seldom is seen in women before menopause.**
2- **Appears after puberty.**
3- **More common in males.**
4- **<u>More in urban than rural areas.</u>**
5- **More in intelligent.**
6- **More in obese.**
7- **More in high intake protein person (more in rich people).**
8- **More in hot climate (10% of patients).**

The concentration of uric acid in body fluid depend on a balance between purine synthesis plus ingestion and uric acid elimination through kidney(2/3 = 600mg/day) and intestine(1/3= 200mg/day). Various **genetic and environmental factors** lead to **hyperuricaemia** and gout by ↓the excretion of uric acid &/ or increasing its production(75% genetically determined defect in excretion of uric acid & 20-25% due to ↑production of uric acid).

Clinical features

1- **The onset may be insidious or explosively sudden**, often waking the patient from sleep
2- **Excruciatingly painful joint, tender, swollen, redness, hot with shiny overlying skin and dilated veins (<u>signs of inflammation</u>).**
3- **In 70% of patients the <u>metatarsophalangeal joint of the big toe</u> is affected, and it is the site of 1st attack of acute gouty arthritis,** then ankle, knee, small joints of feet and hand, wrist and elbow joint.
4- **Fever in very acute attack** with ↑WBC & ESR **and preceded by prodromal symptoms(nausea, anorexia & chang in mode).**
5- **The acute attack may precipitated by**
   a- **Trauma.**
   b- **Exercise.**
   c- **Diuretic drugs.**

d- **Dietary excess or severe dietary restriction.**
e- **Alcohol.**
f- **Severe systemic illness.**
g- **Surgery.**

6- **Urate stone** (urolithiasis).
7- **Nephropathy.**
8- **Tophi on joints tendons, ear pinna & bursae.**
9- If the attack is untreated , it lasts for days or weeks, but eventually subsides spontaneously. (**Resolution** of attack may accompanied by itching (pruritis) & desequamation).
Recurrence of attack after months or years while may lead to progressive cartilage & bone erosion & deposition of tophi & secondary degeneration changes, severe function impairment & joint deformity.

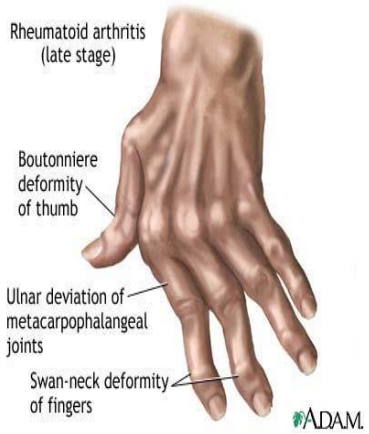
Diagnosis

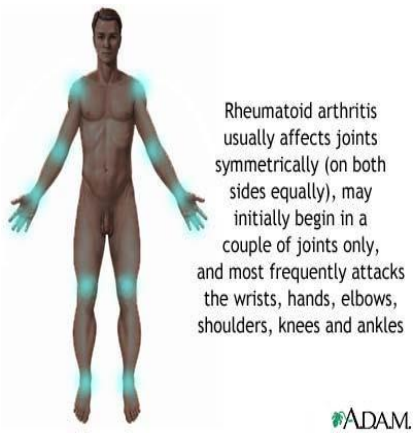
1- ↑**Serum urate level**, but not diagnostic because asymptomatic hyperuricaemia is very common.
2- **Synovial fluid examination** under polarizing light microscope(show needle shaped crystal of monosodium urate-MSU-).
3- **X-ray of joint** (show erosion & soft tissue swelling of urate tophi).

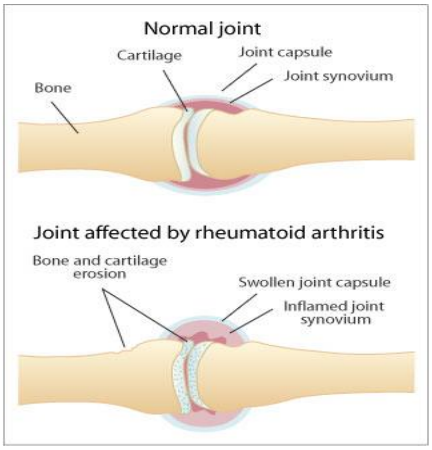
Management

1- **Avoid salicylates**(antagonist to uricosuric drug) **& diuretic**.
2- **NSAIDs** ; **Indocid** 50mg 6 hourly as early as possible or **naproxen** 500mg 8 hourly, continue the treatment until the acute attack disappear then **continue for 7-10 days.**
3- **Colchicines** 1mg at once followed by 0.5mg 2 hourly later could be enough, but may cause vomiting & diarrhea.
4- **Uricosuric drugs** (lowering s. urate level, ↓ frequency of attack &↓ size of tophi) are used after subsidence of acute attack as;
Probencide 0.5-1gm twice daily, contraindication in renal failure & urate stone (**benzbromarone** 100mg/day in case of moderate renal failure) .
5- **Allopurinol** (300mg initially then increased) for long term prophylaxis (lowering serum urate level), it is better to be given after many weeks of the last attack & is given with colchicines (0.5mg twice daily)for several months.
6- **Diet**; **↓purine, no alcohol, ↓weight gradually, severe calorie restriction must be avoided because it leads to lactic acidosis & ↑serum urate.**
7- **Surgery of large, painful, or ulcerating tophi.**

Rheumatoid arthritis







It is the most common inflammatory arthritis in females & hence an important cause of potentially preventable disability, it is a **destructive joint process leading to deformity.**

Aetiology

1- **More in females** .Before age 45, 6:1 prevalence, ↑ with age.
2- **Peripheral joints are more common affected.**
3- **More common in adults.**
4- **The autoimmune plays a major rule**, frequency of disease is↑ in **1st –degree relatives of patient with RA.**
5- 50% of genetic contribution is due to genes in the HLA region.

Pathology

RA is characterized by persistent cellular activation, autoimmunity and the presence of immune complexes at sites of articular and extra-articular lesions, so this leads to chronic inflammation, garrulous formation and joint destruction.

Clinical features

The diagnosis of RA can only be established by an accurate and careful **history and physical examination.** Only limited help is provided by laboratory test.

The typical clinical phenotype of RA is;

a- **Symmetrical.**
b- **Deforming.**
c- **Small and large joints polyarthritis.**
d- **Often associated with systemic disturbance and extra-articular diseases.**

****The clinical course is usually life-long, with intermittent exacerbations and remissions and highly variable severity.**

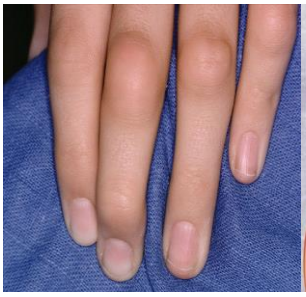
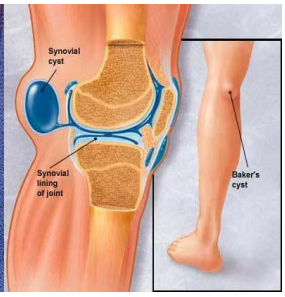
**The clinical hallmark of inflammatory joint disease is persistent synovitis.

Criteria for diagnosis of RA

Diagnosis of RA is made with 4 or more of the following

1- **Morning stiffness (>1hr).**
2- **Arthritis of 3 or more joint areas**
3- **Arthritis of hand joints.**
4- **Symmetrical arthritis.**

5- **Rheumatoid nodules.**
6- **Rheumatoid factor.**
7- **Radiological changes.**
8- **Duration of 6 weeks or more**.

Pattern of presentation

A- **<u>Gradual onset</u> of symmetrical arthralgia and synovitis of small joints of hands, feet and wrist.**
B- **A <u>dramatic acute onset</u>**, sometimes over **just a few days,** with florid morning stiffness, polyarthritis and pitting oedema more commonly in elderly.
C- **<u>Recurrent symmetrical acute episodes</u> of joint pain**, **tender on pressure and swelling which last only for few hours or days**. Most patients have evidence of morning and inactivity stiffness and stress pain on passive movement (symmetrical swelling of metacarpophalangeal(MCP) and proximal interphalangeal(PIP) joints).

Extra-articular features

RA is systemic disease ; Anorexia, weight loss, fatigue, generalized osteoporosis and muscle wasting . Most features are due to serositis, granuloma, nodule formation or vasculitis;

1- **Systemic**; Fever, Anorexia, weight loss, fatigue, susceptibility to infection.
2- Muscloskeletal; Muscle wasting, tenosynovitis, bursitis, osteoporosis.
3- Haematological; Anaemia(iron deficiency), thrombocytosis, eosinophilia.
4- Lymphatic; splenomegaly.
5- Nodules; Sinuses, Fistula.
6- Ocular; Episcleritis, scleritis.
7- Vasculitis; Digital arteritis, ulcer.
8- Cardiac; Pancarditis, conductive defect coronary vasculitis.
9- Neurological; Cervical cord compression, peripheral neuropathy, amyloidosis.
10- Pulmonary bronchiolitis, pleural effusion.
11- Cyst formation(Baker's popliteal cyst).

Diagnosis

To establish diagnosis

1- **Clinical criteria**
2- Acute phase response
3- **Serology**; **--Antinuclear factor**,
--↑C- reactive protein
--Rose Waaler & Latex fixation test.

4- **High ESR.**
5- **X- ray & arthrography** & functional assessment.

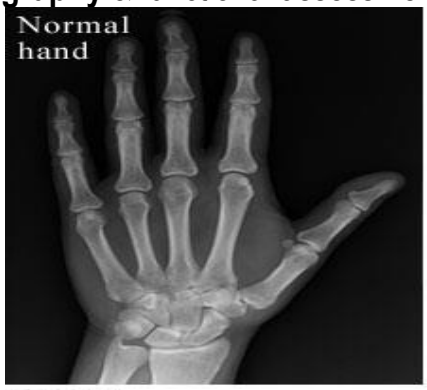


Figure 1

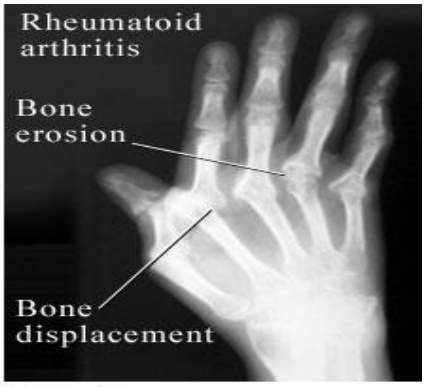


Figure 2

6- Biopsy.
7- **Ultrasound.**
8- CT- scan.
9- **Arthroscopy & biopsy.**
10- MRI.

Management

The main stay of treatment for acute RA

1- **Physical rest.**
2- **Targeted anti-inflammatory therapy.**
3- **Passive exercise.**

Hospital admission for

1- **multiple intra-articular injection.**
2- **Joint splinting.**
3- **Regular hydrotherapy.**
4- **Physiotherapy and education**.

Outside hospital

1- **I.M. or intra-articular corticosteroid.**
2- **Oral analgesic & NSAIDs, & adjustment of DMARDs**(Disease modifying antirheumatiod drugs) (Slow acting antirheumatoid drugs).
3- **Periodic assessment of disease activity, progression**(damage)& disability is required.

Drug therapy

1- **Promote introduction of DMARDs** either singly or in combination.
Methotraxate & sulfasalazine are currently 1st choice of DMARDs for RA.
2- **Targeted anticytokine treatment** (If DMARDs are failed); Anti- TNF-α (anti-tumour necrosis factor); **Infliximab, Etanerecept, or Adalimumb**.
More effective with combination with methotrexate.
3- **Prednisolon (**7.5mg/day)+NSAIDs+ DMARDs therapy may slow rate of radiological progression over 2years in patient with early RA).
4- **Symptomatic management with continued use of NSAIDs & analgesic** may also be required.

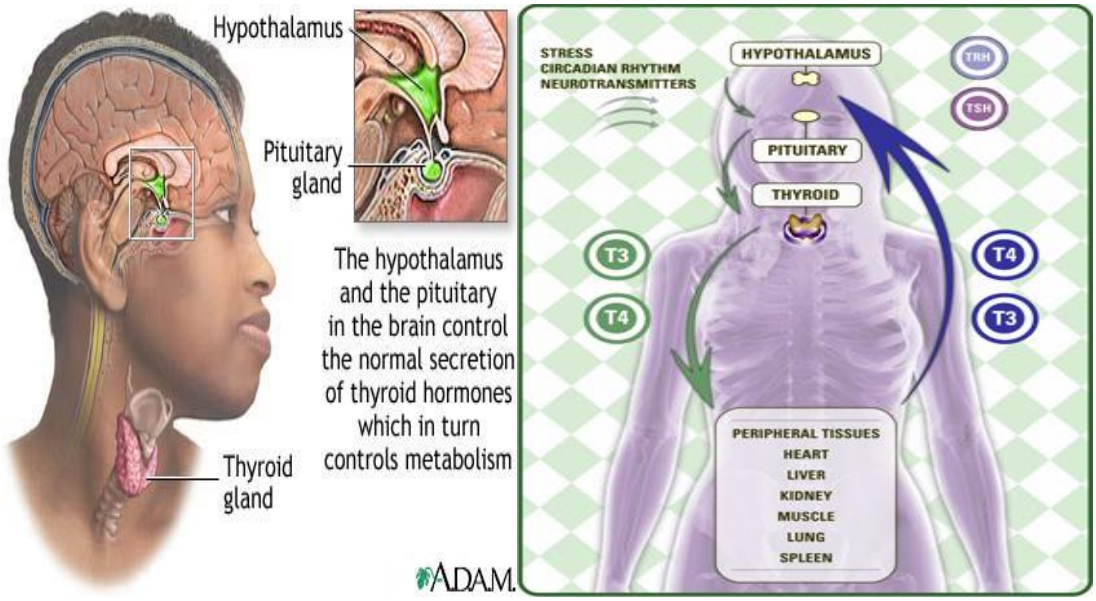
Progression & prognosis

1- Average life span is ↓by 8-15 year by RA.5 years survival for patient with severe disease is only 50%.
2- 40% of patient will be disabled within 3 years.
3- 80% of patient will be moderately to severely disable within 20years & 25% have required large joint replacement.

DMARDs include

1- **Hydroxychloroquine** (anti- malarial drug) 200-400mg/day.
2- **Sulfasalazine** 2-3gm/day.
3- **D-penicillamine** 250-750mg/day.
4- **Gold** 50mg/month by i.m. injection.
5- **Methotrexate** 5-25mg/week.
6- Azothioprine 50-150mg/day.
7- Leflunomide 20mg/day.
8- Cyclophosphamide 0.5-1gm by i.v. 1-4 weekly.
9- Chlorambucil 4-8mg/day.
10- Ciclosporin 150-300mg/day.

Thyroid Diseases



Thyroid physiology

The parafollicular C-cells secret **calcitonin**(play a rule in calcium metabolism).

The follicular epithelial cells synthesis thyroid hormones by incorporating iodine into the amino acid tyrosine on surface of thyroglobulin (Tg) protein secreted into colloid of the follicle. Iodine is a key substrate for thyroid hormone synthesis, dietary intake in excess of 100μg/day is required to maintain thyroid function in adult. Thyroid secrets predominantly **thyroxine (T_4)** and only a small amount of **triiodothronine (T_3)** , approximately 85% of T_3 in blood is produced from T_4 by a family of monodeiodinase enzymes. Production of thyroid hormones is stimulated by TSH from anterior pituitary gland.

Thyroid gland diseases are classified into

1-Hormone excess(Hyperthyriodism)

A-Primary;

1- Graves'disease.

2- Multinodular goitre.

3-Adenoma.

4-Subacute thyroiditis.

B-Secondary; Pituitary TSHoma (**hyperpituitarism**)

2-Hormone deficiency (Hypothyriodism)

A-Primary; 1-Hashimoto's thyroiditis. 2- Atrophic hypothyroidism.

B-Secondary; Hypopituitarism.

3-Hormone hypersensitivity.

4-Hormone resistance; Thyroid hormone resistance syndrome.

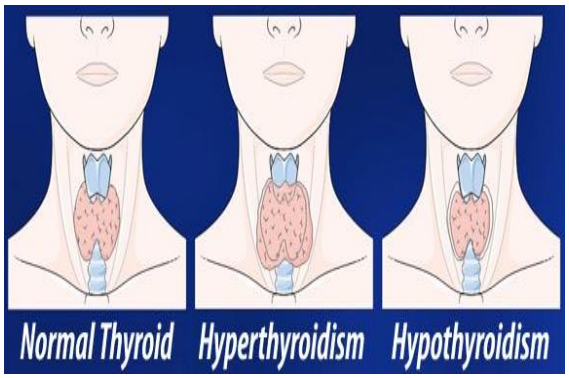
5-Non-functioning tumours; 1-Differentiated carcinoma.2- Medullary carcinoma.
3-Lymphoma.

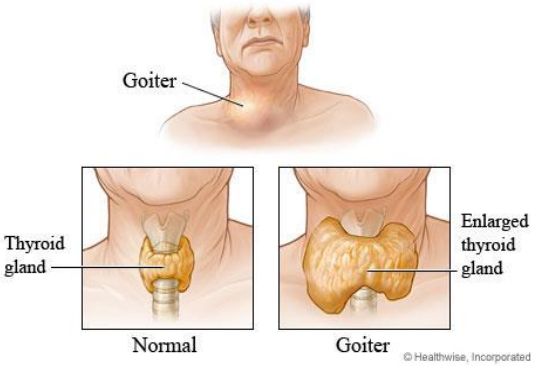
****The most common presentations of thyroid diseases are ;**

1- Hypertyroidism (Thyrotoxicosis).

2-Hypothyroidism.

3-Goitre (Enlargement of thyroid gland).





Hyperthyroidism(Thyrotoxicosis)

Aetiology

Disease	Frequency
1-Graves'disease	**76%**
2-Multinodular goitre	**14%**
3- Toxic adenoma	**5%**
4-**Thyroiditis-viral infection-; Subacute,**	**3%**
post-partum	0.5%
5-Extrathyroidal source of thyroid hormone	0.2%
6-TSH-induced(TSH-secreting pituitary adenoma)	0.2%
7-Follicular carcinoma± metastases	0.1%

Clinical features

Symptoms

1-General; **a- <u>Weight loss despite normal or ↑appetite.</u>**
b- <u>Heat intolerance.</u>
c- Fatigue, apathy(in elderly).
d-Osteoporosis.

2-Gastrointestinal; **a- Diarrhoea, steatorrhoea.**
b-<u>Hyperdefecation.</u>
c-Anorexia & vomiting.

3-Cardiorespiratory features; **a-<u>Palpitation</u> & exacerbation of asthma.**
b-<u>Dyspnoea</u> on exertion & angina.

c-Ankle swelling.

4-Neuromuscular; a-Anxiety, irritability & psychosis.
b-Tremor, muscle weakness & periodic paralysis.

5-Dermatological; a- sweating, pruritis.
b-Alopecia.

6-Reproductive; a-Amenorrhoea/ oligomenorrhoea.
b-Infertility, spontaneous abortion.
c-Loss of libido & impotence.

7-Ocular; a-Red eyes.
b-Excessive lacrimation.
c-Diplopia.
d-Loss of acuity.

Signs

1-Weight loss, goitre with bruit .

2-Signus tachycardia, atrial fibrillation, systolic hypertension,↑pulse pressure, & cardiac failure.

3-Lymphadenopathy.

4-Tremor, hyperreflexia, & myopathy.

5- Palmar erythema, pretibial myxoedema, digital clubbing,pigmentation & **vetiligo.**

6-Gynaecomastia.

7-Lid retraction, lid lag, exophthalmos, corneal ulceration, periorbital oedema, ophthaloplegia & papilloedema**.**

Investigations

1-↑Serum T_3 & T_4.

2-TSH is undetectable.

3-↑TSH receptor antibodies(in Graves'disease).

4-Isotope scanning (99m technetium scintigraphy) of thyroid gland.

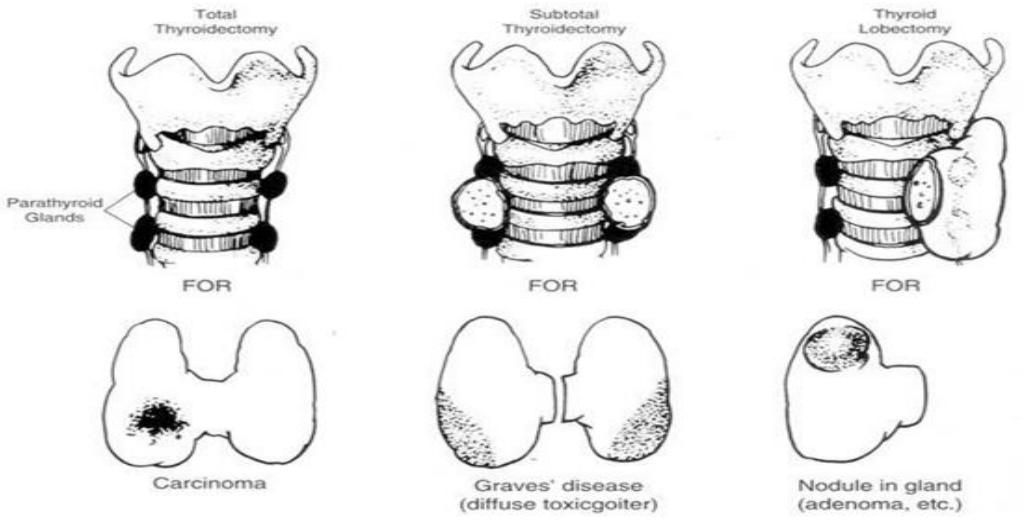
5-Non-specific laboratory abnormalities; -↑Serum enzymes (Alanine aminotransferase, alkaline phosphatase) -↑bilirubin & mild ↑Ca ++ & glycosuria.

Management

Treatment depend on underlying cause;

1-Antithyroid drugs; (Indicated in patients<40 years); Carbimazol (C/I in hypersensitivity & breast feeding) & its S/E is more than 50% relapse rate usually with 2 years of stopping treatment. Propylthiouracil suitable for breast feeding.

2-Subtotal thyroidectomy ; Indicated in large goiter, poor drug compliance especially in young patients, recurrent thyrotoxicosis after course of antithyroid drugs.



3-<u>Radio-iodine; Indicated in patients> 40years</u> , recurrence following surgery. C/I pregnancy.

4-β-blocker propranolol 160 mg/day or nadolol 40-80 mg/day will alleviate but not abolish symptoms within 24-48 hours, so it is recommended for short term treatment & for atrial fibrillation or following ^{131}I therapy.

5-Warfarin is required in thrombo-embolic vascular complication.

Thyroid crisis(thyroid strom)

It is a life-threatening increase in severity of clinical features of thyrotoxicosis(fever, confusion, tachycardia or atrial fibrillation & even heart failure in elderly) **the mortality rate is 10%, it is most commonly precipitated by infection.**

Treatment

1-Rehydration.

2-Broad spectrum antibiotic.

3-Propranolol is rapidly effective orally 80mg 6-hourly or i.v. 1-5 mg 6-hourly.

4-Na ipodate 500 mg/day orally will restore serum T_3 levels to normal in 48-72 hours.

5-Dexamethasone 2 mg 6-hourly & amiodarone.

6-Carbimazol orally 40-60 mg/day or rectal.

Hypothyroidism

****female to male ratio is 6:1.**

**The prevalence of primary hypothyroidism is 1:100.

Aetiology

****Autoimmune disease & thyroid failure following ^{131}I or surgical treatment of thyrotoxicosis account of >90% of cases.**

1-Autoimmune disease; -**Hashimoto's thyroiditis**(Graves' disease with TSH receptor-blocking antibodies).

2-Iatrogenic; -Radioactive iodine ablation thyroidectomy. **-Drug(carbimazol Lithium, amiodarone).**

3-Transient thyroiditis; -- Subacute thyroiditis. --Post-partum thyroiditis.

4-Iodine deficiency ; in mountainous region.

5-Congenital.

6-Infiltrative; -Amyloidosis. –Sarcoidoosis.

7-Secondary hypothyroidism; TSH deficiency.

Clinical features

1-General; a- Weight gain &cold intolerance.

b- Fatigue & somnolence.

c- Hoarsness of voice(low-pitched) , **slurred speech**(large tongue) & **poor hearing**.

d-Goitre as a sign.

2-GIT; Paralytic ileus & ascites(rare).

3-Cardiorespiratory ; **a-Bradycardia & Hypertension. b-**Pericardial & pleural effusion(rare).

4-Haematological; Macrocytosis,& iron deficiency anaemia.

5-Neuromascular; a-Carpal tunnel syndrome with aches & pains.

b-Muscle stiffness, deafness, & depression.

c-Myxodema madness(rare).

d**- Delayed relaxation of tendon reflexes** (**hyporeflexia**)& cerebellar ataxia.

6-Dermatological; a-Dry skin & hair , alopecia.

b-Myxodema(infiltration of dermis) →**Non-pitting oedema** involve skin of head, feet, eyelids(periorbital puffiness).

c-Purplish lips, malar flush, **facial pallor**(vasoconstrictor &anaemia), **lemon-yellow tint** to skin(Carotenaemia).

7-Ocular; Periorbital oedema & loss of lateral eyebrow.

Investigations

In primary hypothyroidism due to intrinsic disorder of thyroid gland;

1-↓Serum T_4. **2-↑TSH in excess of 20mU/l.**

3-Serum T_3 cocentration do not discriminate reliably between eythroid & hypothyroidism, & should not be measured.

4-ECG; Sinus bradycardia, low voltage &complex, ST-segment & T wave Abnormalities.

5-Thyroid peroxidase antibodies helpful.

Management

1-Life long Thyroxine therapy; Customary to start slowly single 50µg/day for 3 Weeks, then increasing to 100µg/day for further 3 weeks & finally to maintenance dose(100-150µg/day). Treatmentis given 6 weeks to repeat thyroid function test & adjusting the dose.The correct dose of thyroxine is to restores serum TSH to within the reference range. It is important to measure thyroid function every 1-2 years once the dose of throxine is stabilized .

In pregnancy; **↑ the dose of thyroxine of ~µg/day to maintain normal TSH levels, & serum TSH & freeT_4 should be measured during each trimester.**

****Thyroxine replacement in ischemic heart disease should be introduced at low dose & ↑ very slowly under specialist supervision.**

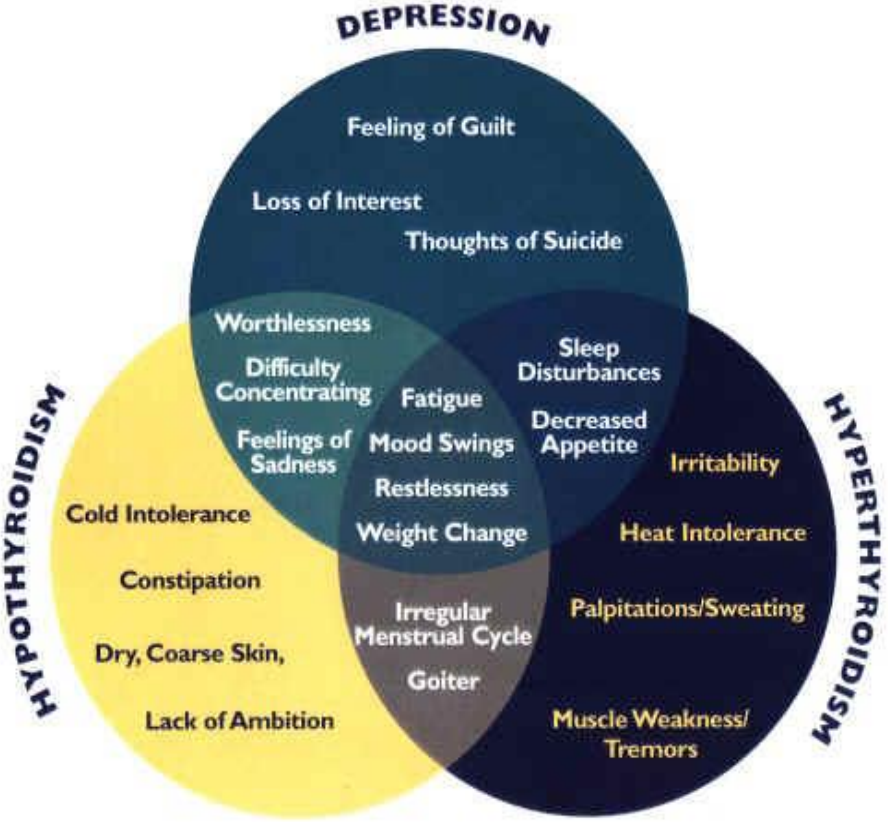
****Myxodema coma(rare case)**

Treated by T_3 i.v. bolus dose of 20µg followed by 20µg 8-hourly until sustained clinica improvement, then substituted by oral thyroxine 50µg/day

****Subclinical thyrotoxicosis; Serum TSH undetectable, ↑Serum T_3 &T_4.**

****Subclinical hypothyroidism; ↑serum TSH, ↓Serum T_3 &T_4.**

Symptoms of Hyperthyroidism and Hypothyroidism



Thyroid enlargement

Palpable thyroid enlargement is common affecting 5% of population.

Aetiology

1-Diffuse goiter; Simple goiter occurs in younger age, causes;
Hashimoto's thyroditis, Graves'disease, drug(Iodine, lithium & amiodaron),
Iodine deficiency, suppurative thyroiditis,& Transient thyroditis.

2-Multinodular goiter; causes; Multiple adenoma, Focal hyperplasia, Cancer.

3-Solitary nodule; Simple cyst; causes; Benign colloid nodule, Follicular adenoma, Papillary carcinoma, Follicular carcinoma, Medullary cell carcinoma, Anaplastic carcinoma, Lymphoma metastasis.

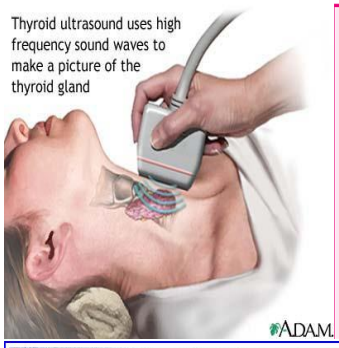
Investigations

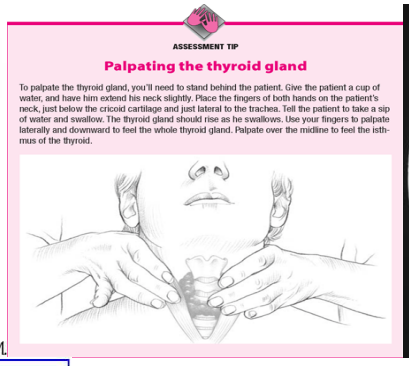
A-Serum T_3 ,T_4 & TSH should be measured in all patients with solitary thyroid nodule.

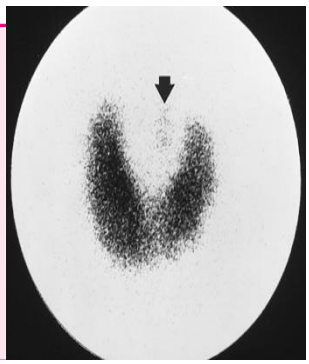
**** TSH undetectable is suggestion of autonomously function thyroid follicular adenoma.**

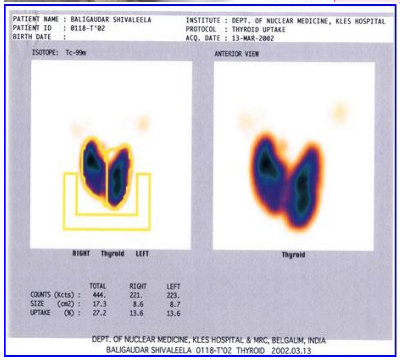
B-Thyroid isotope scanning(99mTc scintigraphy); Cold nodule may be malignant.

C-Ultrasonography.









Management

****In diffuse goiter**; in absence of thyrotoxicosis & hypothyroidism need no treatment unless causing cosmotic symptom or compression on other local structures(Strider or dysphagia)

****Sometimes need thyroxine to shrink goiter.**

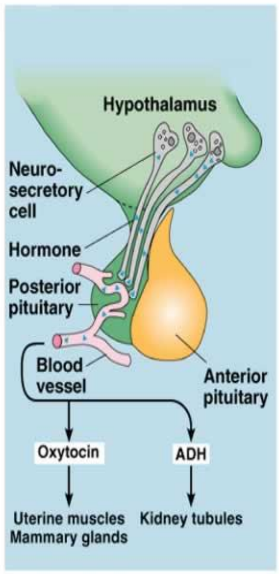
****Needle aspiration of nodule for therapeutic & diagnostic(cytological examination) purposes.**

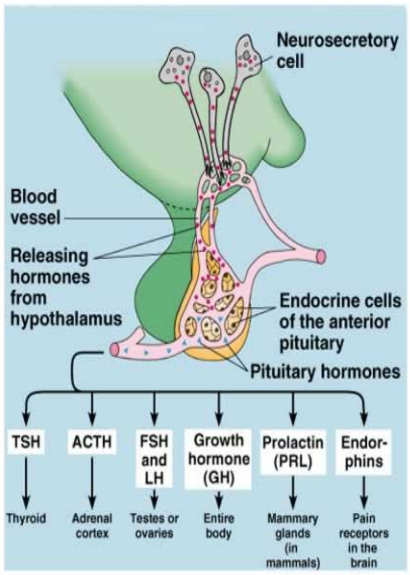
<u>Solitary nodules with solid component are treated by surgical excision.</u>

**Benign lesion are sometimes excised.

<u>Carcinoma are treated by total thyroidectomy & followed by large dose of ^{131}I with long-term treatment of thyroxine in dose sufficient to suppress TSH .</u>

Pituitary gland diseases





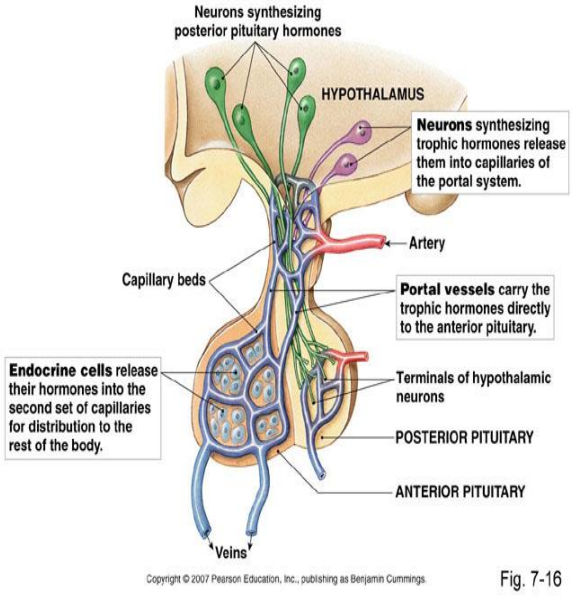


Fig. 7-16

Pituitary gland anatomy & physiology

It consists of two lobes, **anterior lobe glandular tissue & posterior nervous tissue lobe**. The pituitary gland is connected to the hypothalamus by pituitary stalk (infundibulum), which has portal vessels carrying blood from median eminence of hypothalamus to anterior lobe & nerve fibres to posterior lobe.

**** The pituitary under the control of hypothalamus by substances produced in hypothalamus & released into portal blood which either stimulate or inhibited anterior pituitary hormone secretion.**

Anterior lobe secretes

1- **Growth hormone**→ Liver(The target).
2- **Prolactin hormone**→ Breast.

3- **ACTH** (Adrenocorticotrophic hormone)→ Adrenal cortex.
4- **TSH** (Thyroid stimulating hormone)→ Thyroid gland.
5- **FSH**(Follicle stimulating hormone) & **LH** (Luteinisinghormone)→ Gonads.
6- **β-LPH** (β-Lipotrophic hormone) → Melanocyte.

Posterior lobe secretes

** Their hormones are synthesized in hypothalamus & transported down nerve axons to be released from posterior pituitary lobe.

1- **ADH** (Antidiuretic hormone) –Vasopressin-→Distal nephron of kidney.
2- **Oxytocine** (pituitarin) hormone→ Uterus & breast.

Hypopituitarism

Causes

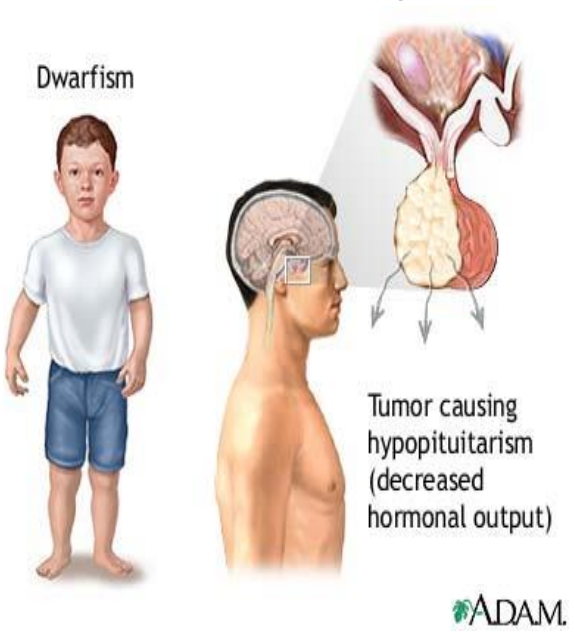
1- Diseases of hypothalamus (congenital or acquired).
2- Tumour of anterior pituitary (Usually benign adenoma, while primary carcinoma is rare).
3- Surgery & radiotherapy.
4- **Postpartum necrosis of pituitary.**
5- **Head injury.**
6- Auto-immune.

Clinical features

It depends on the hormone affected

A- Anterior lobe

1- Growth hormone deficiency → Dwarfism (short stature).



2- Stimulating hormone deficiency leads to multi endocrine hypofunction.

Investigations

1- **Measurments of the hormonal levels (Hormonal Assays).**
2- Skull X ray.
3- CT scan.
4- **MRI.** **5-Radioactive isotopes**.

B- Posterior lobe

↓ADH→ Polyuria, polydipsia & dehydration (i.e. diadetes insipidus)
Which treated by giving decompression intra-nasally ,metered dose spray 5µg in morning &10µg at night, it is also available as tablet or i.m. injection for sick patient.

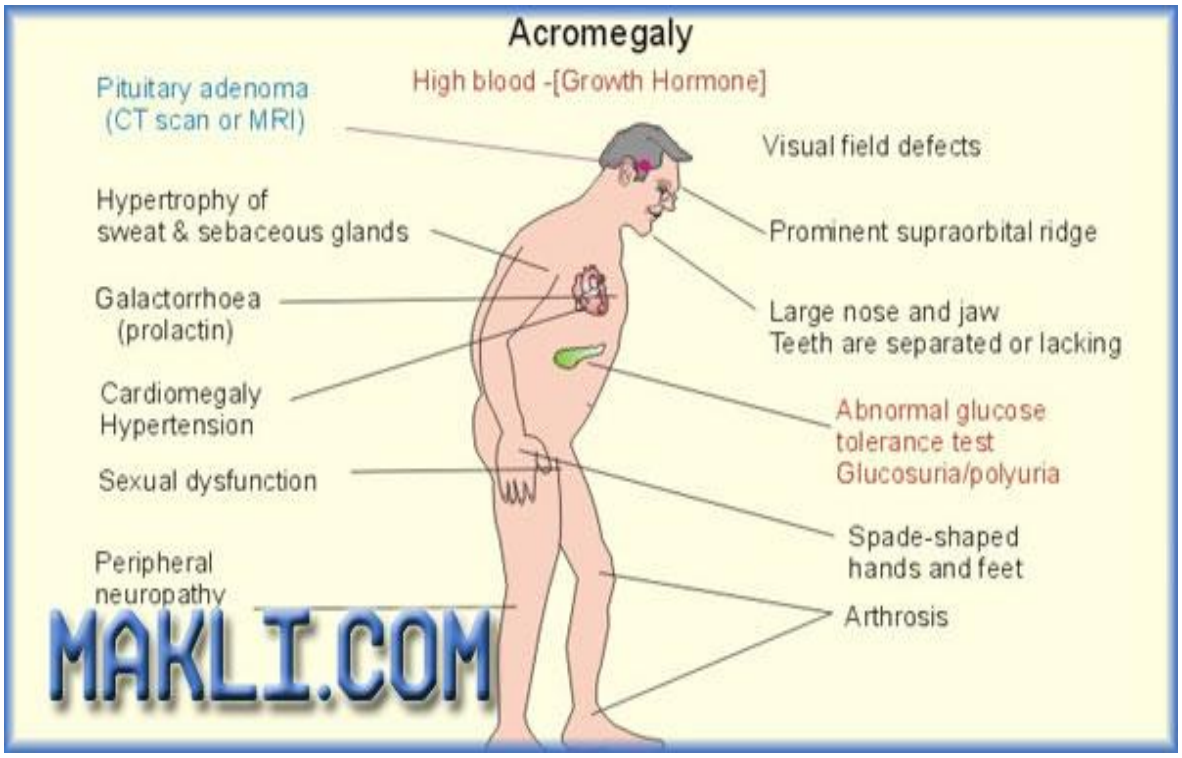
Treatment ; By further assessment & treat the causes

Pituitary hyperfunction

Growth hormone

A- Before puberty → Giantism (Long stature)(rare).
B- After puberty→ Acromegaly(more common).

Acromegaly



Clinical features

1- **↑ connective tissue → enlarged hands, lips, & feet.**
2- **Thickening of vocal cords & hoarseness of voice.**

3- **Enlarged skull & lower jaw.**
4- **Muscular atrophy, weakness and sexual dysfunction.**
5- **Thickening of skin.**
6- **Enlarged liver and heart.**
7- **Hypertension.**

Treatment

A- Medical treatment

1- **Bromocriptine**(long acting dopamine agonist).
2- Somatostatin (Octerotide) s.c. injection 3 time /day.

B- Surgical removal of tumour.

C- Radiotherapy.