

Surgery

Final year case templates

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Acute pancreatitis

Investigations

- Confirm diagnosis
 - S. amylase ↑ in 1st 24 hours (3 or 4 times above normal)
 - Urine amylase – better than serum level because remains high for longer duration.
 - S. Lipase – Better than the amylase. Most specific.
- FBC
 - Raised WBC
 - Low PCV - Due to hypovoleamia
- U & E, Blood glucose, LDH, LFT
- CRP – Assess progression
- ABG – Metabolic Acidosis
- S. calcium (levels are low)
- CXR – can exclude other DD perforated peptic ulcer(Air under the diaphragm), sympathetic pleural effusion, atelectasis, ARDS
- X-ray abdomen – Sentinel loop of jejunum (generalized or local ileus), colon cut-off sign, pancreatic calcifications in chronic pancreatitis)
- USS abdomen –ultrasonography should be performed within 24 hours in *all* patients
 - To detect gallstones as a potential cause
 - Rule out acute cholecystitis as a differential diagnosis
 - To determine whether the common bile duct is dilated
 - Can visualise swollen pancreas
 - Can exclude pancreatic pseudocysts
- CECT Abdomen – **Indications**
 - If there is diagnostic uncertainty
 - In patients with severe acute pancreatitis, to distinguish interstitial from necrotising pancreatitis
 - In patients with organ failure, signs of sepsis or progressive clinical deterioration
 - When a localised complication is suspected, such as fluid collection, pseudocyst or a pseudo-aneurysm

Assessment of severity in acute pancreatitis

Table 64.3 Scoring systems to predict the severity of acute pancreatitis: in both systems, disease is classified as severe when three or more factors are present

Ranson score	Glasgow scale
On admission	On admission
Age > 55 years	Age > 55 years
White blood cell count > 16 × 10 ⁹ l ⁻¹	White blood cell count > 15 × 10 ⁹ l ⁻¹
Blood glucose > 10 mmol l ⁻¹	Blood glucose > 10 mmol l ⁻¹ (no history of diabetes) - 180mg/dl
LDH > 700 units l ⁻¹	Serum urea > 16 mmol l ⁻¹ (no response to intravenous fluids)
AST > 250 Sigma Frankel units per cent	Arterial oxygen saturation (PaO ₂) < 8 kPa (60 mmHg)
Within 48 hours	Within 48 hours
Blood urea nitrogen rise > 5 mg%	Serum calcium < 2.0 mmol l ⁻¹
Arterial oxygen saturation (PaO ₂) < 8 kPa (60 mmHg)	Serum albumin < 32 g l ⁻¹
Serum calcium < 2.0 mmol l ⁻¹	LDH > 600 units l ⁻¹
Base deficit > 4 mmol l ⁻¹	AST/ALT > 600 units l ⁻¹
Fluid sequestration > 6 litres	

- At 48 hours after the onset of symptoms
 - Glasgow score of 3 or more
 - C-reactive protein level greater than 150 mg l⁻¹
 - worsening clinical state with sepsis or persisting organ failure
- Severity stratification should be performed in all patients within 48 hours of diagnosis.
- Patients with a body mass index over 30 are at higher risk of developing complications

} Indicate a severe attack

Management

- 80% recover without any complication
- Keep NBM to Rest the pancreas
- NG suction - Vomiting
- Obtain IV access
- Start volume expanders (Colloid/plasma substitutes/N.saline) until SBP>100mmHg & UOP>30ml/hour
- Catheterize the patient and monitor urine output
- Analgesia –give IM Pethidine 75 mg IM 6 hrly
- Anti-emetics
- Antibiotics – Cefuroxime 750mg 8 hourly to prevent 2ry infections
- Monitor blood glucose 2 hourly and treat with Insulin, if high
- Heparin – Prone to DVT
- Operative
 - Pancreatic enzyme inhibitors – expensive
 - Pancreatic drainage if abscess formation
 - Pancreatic debridement
 - Peritoneal lavage
 - Remove gall bladder – recurrent pancreatitis
- Monitor for complications and manage

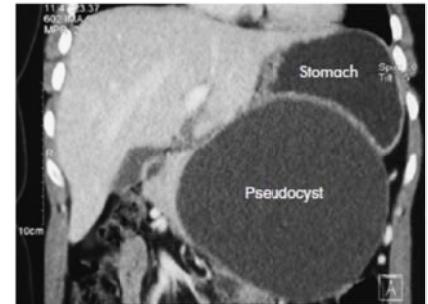
- Common complications**
- ARDS
 - ARF
 - DIC
 - Sepsis
 - Hypocalcaemia

Table 64.5 Complications of acute pancreatitis

Systemic	Local
(More common in the first week)	(Usually develop after the first week)
Cardiovascular	Acute fluid collection
Shock	Sterile pancreatic necrosis
Arrhythmias	Infected pancreatic necrosis
Pulmonary	Pancreatic abscess
ARDS	Pseudocyst
Renal failure	Pancreatic ascites
Haematological	Pleural effusion
DIC	Portal/splenic vein thrombosis
Metabolic	Pseudoaneurysm
Hypocalcaemia	
Hyperglycaemia	
Hyperlipidaemia	
Gastrointestinal	
Ileus	
Neurological	
Visual disturbances	
Confusion, irritability	
Encephalopathy	
Miscellaneous	
Subcutaneous fat necrosis	
Arthralgia	

Prognosis of acute pancreatitis

- The overall mortality from acute pancreatitis has remained at 10–15%.
- The aetiology of the attack of pancreatitis should be determined before the discharge.
- Failure to remove a predisposing factor could lead to a second attack of pancreatitis, which could be fatal.
- In a patient who has gallstone pancreatitis, the gallstones should be removed as soon as the patient is fit to undergo surgery.



Pancreatic pseudo cyst

- Collection of amylase-rich fluid enclosed in a wall of fibrous or granulation tissue.
- Pseudocysts typically arise following an attack of acute pancreatitis, but can develop in chronic pancreatitis or after pancreatic trauma.
- Formation of a pseudocyst requires 4 weeks or more from the onset of acute pancreatitis.
- They are often single but, occasionally, patients will develop multiple pseudocysts.

Investigations

- Elevated S. amylase
- Aspirated cystic fluid has low CEA level (in a cystic tumor of pancreas → High CEA)
- USS, CT, MRI
- Therapeutic interventions are advised
 - Only if the pseudocyst causes symptoms
 - If complications develop
 - Distinction has to be made between a pseudocyst and a tumour.
- Pseudocysts will resolve spontaneously in most instances, but complications can develop.

Table 64.6 Possible complications of a pancreatic pseudocyst

Process	Outcomes
Infection	Abscess Systemic sepsis
Rupture	
Into the gut	Gastrointestinal bleeding Internal fistula
Into the peritoneum	Peritonitis
Enlargement	
Pressure effects	Obstructive jaundice from biliary compression Bowel obstruction
Pain	
Erosion into a vessel	Haemorrhage into the cyst Haemoperitoneum

- Small cysts – observation
- Drainage - If symptomatic
 - External –Fistula formation is a complication
 - Internal –UGI endoscopic drainage through stomach (endoscopic transgastric cystgastrostomy)
 - ✓ Surgical - drain into stomach or jejunal lumen internally
 - ✓ Need a mature cyst wall of 5mm thickness. So wait 6 weeks to gain this. Check the thickness by imaging

Chronic pancreatitis

➤ Chronic pancreatitis

- Chronic inflammatory disease with progressive destruction of its endocrine & exocrine tissue by replacement with fibrous tissue
- Causes
 - Alcohol – Heavy and prolonged consumption
 - Ductal obstruction
 - Gall stones
 - Strictures
 - Tumours
 - Hypercalcemic states (hyperparathyroidism)
 - Hereditary pancreatitis(AD)
 - Idiopathic malnutrition
 - Cystic fibrosis
 - Malnutrition
- Clinical features
 - Recurrent abdominal pain radiating through to back
 - Relieved by leaning forward
 - Evidence of chronic liver disease may also be apparent due to alcoholism
- Complications
 - Recent onset DM
 - Steatorrhoea
 - Fat soluble vitamin deficiency (Vit D →Osteomalacia)
 - Wasting
 - Anorexia
 - Obstructive jaundice
 - Pancreatic CA
 - Immunodeficiency
 - Portal vein thrombosis and portal HT
 - Pseudocyst formation

- o Investigations

- Serum Amylase → may be normal even during the exacerbation because of severe exocrine failure.
- Radiology is the mainstay of the diagnosis
 - ✓ Abdominal x-ray – pancreatic calcification in L1 level[30– 50 %]
 - ✓ USS abdomen – pancreatic tumors, duct dilatation, pseudocyst, Cirrhosis
 - ✓ CECT- enlargement , irregular consistency, calcifications and ductal changes
- ERCP / MRCP
 - ✓ “Chain of lakes” appearance
 - ✓ Distal bile duct stenosis

- o Treatment

Remove the aetiology	<ul style="list-style-type: none"> • \$, 1-° 1#2æ # °" 1^ 1# • 6-10#æ 1CE1A#
Medical	<ul style="list-style-type: none"> • Pain relief – Opiate analgesia(According to analgesic ladder), Coeliac plexus block • Pancreatic insufficiency – enzyme tablets during meals (Enteric coated tablets. Given with PPI or H2 receptor antagonists to prevent inactivation by acid in the stomach) • Treat for DM • Psychological support
Supportive	<ul style="list-style-type: none"> • Diet – low fat, stop intake of wheat flour, frequent small meals enriched with vitamins and minerals
Surgical	<ul style="list-style-type: none"> • Resection <ul style="list-style-type: none"> - Total pancreatectomy - Distal pancreatectomy • Drainage – Duct decompression(Puestow procedure) –Side to side anastomosis of the pancreatic duct and the jejunum • Obstructive jaundice may be relieved by using Roux-en-Y reconstruction • Endoscopic stenting of the pancreatic duct to relieve obstruction

DISCUSSION

- Acute pancreatitis

- o Acute inflammation

- Rapid onset of pain & tenderness previously well patient suddenly develop symptoms
- With raised pancreatic enzymes in blood or urine

The underlying mechanism of injury in pancreatitis is thought to be premature activation of pancreatic enzymes within the pancreas, leading to a process of autodigestion..

- After cellular injury inflammatory process lead to
 - Pancreatic edema
 - Haemorrhage
 - Necrosis

- Haemodynamic instability
- Bacteremia
- Acute respiratory distress syndrome
- Pleural effusion
- GI haemorrhage
- Renal failure
- DIC
- Mild acute pancreatitis
 - Interstitial edema
 - Minimal organ dysfunction
- Severe acute pancreatitis
 - Pancreatic necrosis
 - Severe systemic inflammatory reaction
 - Multi organ failure
 - CAUSES

Summary box 64.5

Possible causes of acute pancreatitis

- Gallstones
- Alcoholism
- Post ERCP
- Abdominal trauma
- Following biliary, upper gastrointestinal or cardiothoracic surgery
- Ampullary tumour
- Drugs (corticosteroids, azathioprine, asparaginase, valproic acid, thiazides, oestrogens)
- Hyperparathyroidism
- Hypercalcaemia
- Pancreas divisum
- Autoimmune pancreatitis
- Hereditary pancreatitis
- Viral infections (mumps, coxsackie B)
- Malnutrition
- Scorpion bite
- Idiopathic

Summary box 64.2

Causes of raised serum amylase level other than acute pancreatitis

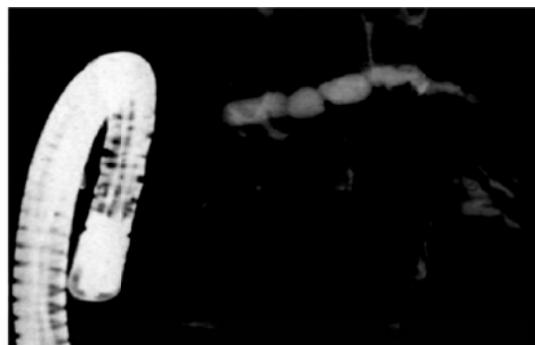
- Upper gastrointestinal tract perforation
- Mesenteric infarction
- Torsion of an intra-abdominal viscus
- Retroperitoneal haematoma
- Ectopic pregnancy
- Macroamylasaemia
- Renal failure
- Salivary gland inflammation

- Calcifications in X-ray



Chain of lakes appearance

(a)



(b)

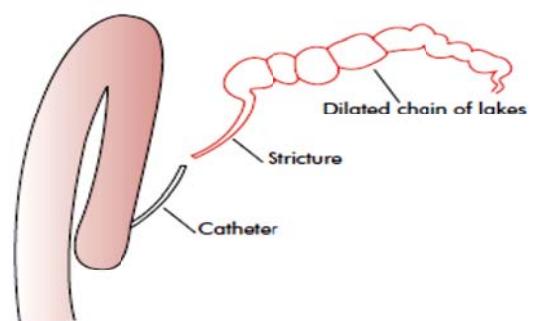


Figure 64.17 (a) Endoscopic retrograde cholangiopancreatography: chronic pancreatitis. Long stricture of the pancreatic duct in the head; distal pancreatic duct shows sacculation with intervening short strictures, 'chain of lakes'. (b) Diagrammatic outline of (a).

Bladder Outflow Obstruction

Presenting complaint

1. Rapid deterioration of already existing features(Duration-for the last one month time)
Voiding LUTS - Hesitancy, Poor stream, intermittent stream, terminal dribbling
BPH→presents commonly with voiding symptoms

Usually Storage LUTS comes with Infection, overactive bladder eg:-Frequency, Urgency, Urge incontinence, Nocturia
Not commonly with BOO.
2. Intractable back pain-Back pain during night refractory to ordinary analgesia, refractory to home remedies
3. LOW, LOA

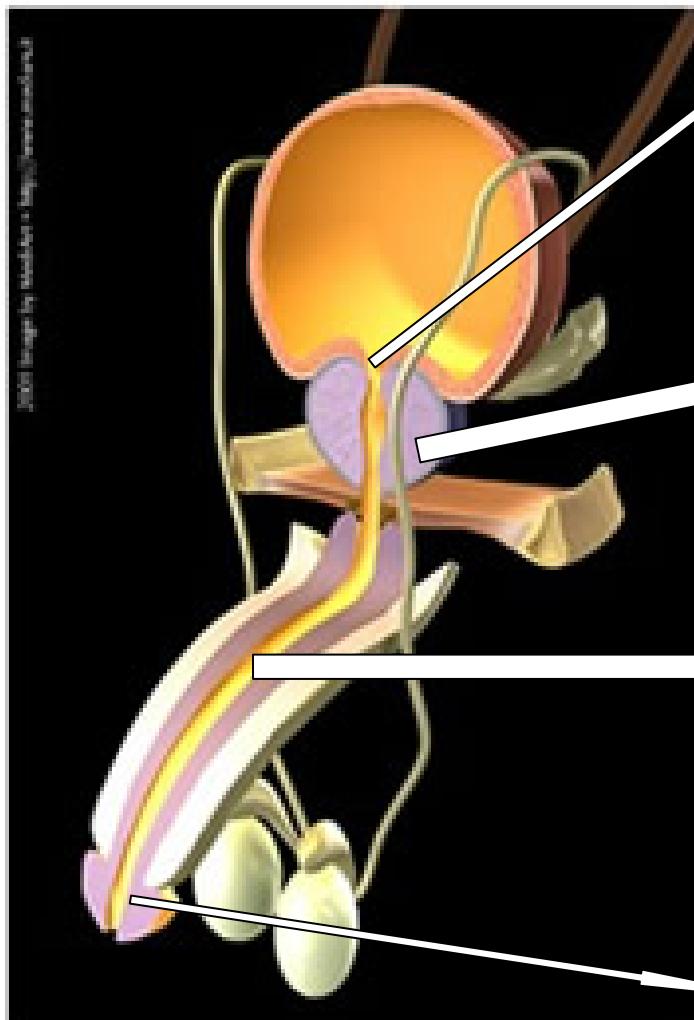
Middle aged

- Think of a stone
- Think of a stricture→Gonococcal urethritis
- Prostatitis
- Think of trauma
- After a pelvic surgery→Post operative acute retention of urine

Young age

- Posterior urethral valve
- Phimosis
- Balanitis
- Meatal stenosis

Bladder outflow obstruction Sites and causes of obstruction



Bladder neck

- Bladder neck hypertrophy (Neuromuscular incoordination)
- Stenosis following prostatectomy
- Stones

Prostate

- Benign prostatic hyperplasia (BPH)
- Prostatic CA
- Prostatitis

Urethra

- Urethral strictures
- Post traumatic-Accidental, iatrogenic,(Instrumentation, catheterization)
- Inflammatory → Gonococcal urethritis
- Neoplastic
- BXO-Balanitis xerotica obliterans

External meatal

- External meatal stenosis
- Tight phimosis
- Urethral stones
- BXO- Balanitis Xerotica Obliterans

At the exam.....

In elderly

- **Majority of BOO → BPH(99%),**
- **Minority → Ca of prostate(By the time it give symptoms most of the time disease is advanced)**
- **Occasionally → strictures, stones**

What is the issue in exam??

- ✓ If you miss a malignancy → Disaster
- ✓ If taken benign as malignancy and done unnecessary investigations → Not so disaster as above

History of presenting complaint

Features of outflow tract obstruction

Two groups of symptoms

- a. Voiding symptoms
 1. Hesitancy-*Difficult in initiating micturition after the individual is ready to pass urine*
 2. Poor stream- *A slow stream reduced urine flow rate*
 3. Terminal dribbling-*Prolonged fine part of micturition when the flow has slowed to a trickle or dribble.*
 4. Intermittent stream-*Urine flow stops and starts on one or more occasions during voiding*
 - b. Storage(Irritative) symptoms (Due to detrusor instability or detrusor dysinergia)
 1. Frequency-*Increased daytime frequency, self explanatory*
 2. Nocturia-*waking up at night more than once to void urine*
 3. Urgency-*Sudden compelling desire to pass urine which is difficult to postpone*
 4. Precipitancy-*Passing urine without his knowledge*
 5. Recent onset nocturnal enuresis in adults- *involuntary passage of urine during sleep*
-
- Some patients have obstructive symptoms only
 - Some patients have irritative symptoms only
 - Some have both obstructive and irritative symptoms both

BOO cause	History	Examination
Benign Prostatic hyperplasia	<p>Majority of BOO due to BPH (99%)</p> <ul style="list-style-type: none"> ✓ Cardinal symptoms → voiding symptoms ✓ Irritative symptoms also present in BPH ✓ Acute retention of urine ✓ Haematuria from ruptured dilated bladder neck veins <p>Signs of uraemia → Fatigue, Peripheral neuropathy, alteration of consciousness, Seizures, Anorexia, Nausea' Decreased taste and smell' Cramps' Restless legs, potentially due to decreased muscle membrane potential' Sleep disturbance' Coma</p>	<ul style="list-style-type: none"> ✓ Palpable bladder, Tender abdomen ✓ Occasionally smell of stale urine on patient ✓ Enlargement of kidney (Hydronephrosis) ✓ PR Ex-Smooth enlarged prostate, median groove palpable, enlarged lateral lobes, rectal mucosa is freely mobile • Signs of uraemia → Alteration of consciousness
Prostate CA	<ul style="list-style-type: none"> • Rapidly progressive symptoms of bladder outflow obstruction • Anorexia and wasting- Genuine anorexia <p>Features of Disseminated disease:-</p> <ul style="list-style-type: none"> • Anaemia → Lethargy, dizziness and undue exertional dyspnoea due to anaemia because of bone marrow infiltration • Scalp lump, a limp, Intractable backache • Impotence • Haematuria • Lower limb and genital oedema (Renal failure) • Back pain-Mets in vertebral column • Pathological fractures-Mets • Evidence of spinal cord compression → Lower limb weakness • Symptoms of Uraemia-In RF 	PR ex <ul style="list-style-type: none"> ✓ Prostate hard in consistency ✓ Surface irregular or nodular ✓ Median groove is obliterated ✓ Rectal mucosa is fixed ✓ Sometimes ulcerated ✓ (Median lobe is enlarged into the bladder, hence in PR feel the lateral lobes)

Prostatitis	<p>Acute bacterial prostatitis Fever, Low back pain, perineal pain, bladder irritation, Outflow obstruction,</p> <p>Chronic prostatitis Symptoms of UTI & dull perineal ache.</p>	<p>Acute prostatitis Febrile, tender abdomen PR Ex:- Enlarged tender prostate</p> <p>Chronic prostatitis Febrile or afebrile/tender abdomen PR Ex:- Normal or indurated irregular prostate</p>
Bladder stones	<ul style="list-style-type: none"> • Most are asymptomatic • Supra-pubic pain • Pain induced by movement • Pain referred to the tip of penis • Intermittent attacks of retention of urine or intermittent flow • Acute onset painful agonizing Painful haematuria • Responds to Pethidine • Associated with Nausea, vomiting, Dysuria & frequency • Rocking movements pain relief <p>Aetiology PMHx of Urinary stones</p>	Scars of previous removal of stones. Supra-pubic tenderness
Urethral stones	<ul style="list-style-type: none"> • The narrowest part of the urethra is the external urethral meatus • Often the stone can be seen protruding through the meatus • Otherwise, patient develops acute retention • The stone will be felt along the ventral surface of the shaft of the penis <p>Tip of penis</p>	On palpation of ventral surface of shaft of penis can feel the stone
Balanitis xerotica obliterans (BXO)	<ul style="list-style-type: none"> • A cause of tight phimosis • Common in Sri Lanka • A premalignant condition • It is a clear indication for circumcision and histopathological evaluation 	

Urethral strictures	<ul style="list-style-type: none"> • Hx of urethral instrumentation Post traumatic-Accidental, iatrogenic (Instrumentation/ catheterization) • Hx of urethral discharge <ul style="list-style-type: none"> - Inflammatory → Gonococcal urethritis 	If patient is catheterized it is rational to exclude tight phimosis and urethral strictures
Neurological	Exclude following conditions <ul style="list-style-type: none"> ✓ Spinal cord disease ✓ Stroke-Weakness in limbs, paralysis of body ✓ DM-Autonomic neuropathy → already diagnosed pt, on treatment ✓ Degenerative neurological disease 	
Stenosis following prostatectomy	Previous surgery for prostate removal(TURP)	
Prolapsed intervertebral disc	<ul style="list-style-type: none"> • Sudden onset of acute back pain, shooting type pain which radiates from back of thigh on walking • Spinal metastases <ul style="list-style-type: none"> ✓ Any CA of breast, lung, prostate previously ✓ Breast-any breast lumps ✓ Lung-any recurrent chest infections with unresolving pneumonia features • Spinal cord injury-Trauma to back, accident (Transection of cord in early phase) • Diabetic autonomic neuropathy- Hx of DM, whether on treatment for DM • Meningo-myelocoele → lump at back and was operated in the past 	<ul style="list-style-type: none"> • Ex of the back • SLRT • Meningo-myelocoele operated scars • Nervous system examination
Causes of neurogenic bladder	<ul style="list-style-type: none"> • Cauda equine syndrome- Spinal or root pain, leg weakness, sensory loss, saddle anaesthesia, (Due to 2ry metastasis-breast, lung, Prostate) • Infection • Cervical disc prolapse • Haematoma (Warfarin) 	

Post operative acute retention of urine	After a pelvic surgery Eg:-Fistulectomy, perianal abscess drainage, Haemorrhoidectomy	
Clot retention	Patient already in a catheter following TURP	
Post traumatic retention of urine	With a hx of accident, trauma	Blood in the urethral meatus
Phimosis	Ballooning of prepuce on urination	Cant retract the prepuce
Balanitis		
External urethral Meatal stenosis	Ballooning of prepuce on urination	
Posterior urethral valves	<ul style="list-style-type: none"> • Hx of recurrent UTI • Ix done MCUG and found Posterior urethral valves • Dose the child on antibiotics 	
Renal cell ca, Bladder ca	<ul style="list-style-type: none"> • Haematuria • LOW 	Renal cell ca → abdominal mass

- ✓ Old people get LOA, LOW → Genuine anorexia (Real anorexia-Aversion to even favourite food)
- ✓ Other causes of old people LOA
 - With bladder outflow obstruction → Obstructive uropathy (Block in VUJ)
 - Infections

Usually old people disease present not like young people → there common manifestation of old age is LOA

As far as your history is concern, what is your diagnosis?
Lower urinary tract symptoms due to bladder outflow tract obstruction. Very likely it can be due to prostatic CA.

Why do you say so?

Examination

General examination

- Built
- Pale
- Scalp lumps(bone metastasis)
- Left supraclavicular lymph node enlargement(Virchow's node, Troisier's sign)
- Ankle oedema → CRF
- Bone tenderness → patella

Abdominal examination

Inspection

- Supra pubic fullness (Due to a distended bladder)
- Is there any scar due to prostatectomy or due to inguinal herniotomy
- It is mandatory to examine the external genitalia→
 - ✓ Examination of the Penis-feel the ventral area for stones
 - ✓ In phimosis-cant retract prepuce, there can be hardened indurated area
 - ✓ If there is no phimosis- Check for external meatal stenosis
 - ✓ An indurated area along the ventral surface of the shaft of the penis is indicative of a urethral stricture
 - ✓ If the patient is catheterized it is rational to exclude tight phimosis, external meatal stenosis and urethral stricture
 - ✓ Check whether the testicles are swollen and tender (epididymo orchitis) whether there are cysts of the epididymis
 - ✓ Straining results in bilateral inguinal herniae. If the patient is having herniae and out flow tract obstruction. First relieve the obstruction eg:-prostatectomy, then the herniotomy
- If the patient catheterize supra-pubic tenderness indicates cystitis
- In an old man with haematuria and a loin lump exclude renal cell carcinoma

Cardiovascular system

- Examine the cardiovascular system for any hypertension, cardiomegaly with a heaving apex (CRF due to obstructive uropathy)

Digital rectal examination

- The anal sphincter tone is lost in cauda equine syndrome, here patulous anus

Benign prostate:-

- Is firm in consistency, the surface is irregular or nodular, The median groove is palpable, the rectal mucosa is freely mobile, the gland is enlarged

Malignant prostate

- Is hard inconsistency, the surface is irregular or nodular, the median groove is obliterated and the rectal mucosa is fixed

At the end of examination examiner will ask, is more favour of a benign or malignant?

Why do you say so?

Is the CA advanced or early?

How will you prove prostate is benign or malignant?

Investigations

General investigations in BOO/Benign prostatic hyperplasia

Initially how will you investigate for BPH?

- I will do a Pelvic Ultra sound scan
 - ✓ Can detect prostatomegaly, prostate volume, nodularity- Trans rectal endosonography
 - ✓ Post voidal bladder volume
 - ✓ Hydroureters, hydronephrosis(USS of abdomen)
 - ✓ Bladder trabeculae can be demonstrated

How to confirm benign prostatomegaly?

- Cytological confirmation and histopathological confirmation

Cytological and histopathology confirmation

- Ramson's needle → aspirate blindly. You see the nodularity and prick it
- True cut biopsy needle → Ultra sound guided biopsy

Cyto-urethroscopy

Looks for strictures, visualizes the urethra and bladder

Uro-flowmetry and pressure flow urodynamic

Studies are not routinely performed in Sri lanka

- ✚ Blood investigations → FBC- Anaemia, renal function tests, PSA(Non specific)
- ✚ Urine investigations → UFR and culture to look for features of a urinary tract infection

Investigation of Prostatic CA

What are the principles of management of Prostate CA?

- The first step is confirmation of the diagnosis. The following investigations are performed

PSA (Prostate specific antigen)

- ✓ <5ng/dl: Definitely benign
- ✓ >20ng/l: Disseminated
- ✓ This is organ specific , not tumour specific
- ✓ Equivocal results necessitate prostatic biopsy

- Next staging of the disease is performed
 - Consider → CT scan of abdomen, bone scan

The subsequent management depends on the stage of the disease

- **Trans-rectal ultrasound guided biopsy**
 - ✓ This visualizes the prostate gland and looks for focal lesions. Histological confirmation is done.
 - ✓ Transrectal ultrasonography is not freely available
 - ✓ To assess the size of the prostate
 - ✓ For guided biopsy

Staging

- **X ray KUB** (Kidney,Ureter,Bladder)
After bowel preparation with 4 tablets of Bisacodyl for 3 nights to see any → stones, Osteo-sclerotic deposits(Ca of Prostate)
- **CT abdomen**
- **Bone scan**

Underlying complicating conditions

UFR

- Pus cells(Infection, stones, Malignancy)
- Proteins(Infection, stones, Malignancy, CRF)

Urine for culture and ABST

To assess fitness for surgery

- **Hb, PCV**
- **Blood urea and serum electrolytes** (S.Creatinine if the Blood urea remains high after hydration)
- **FBS**
- **ECG**
- **Chest X ray**
- **Echocardiogram** → If there are ECG changes, abnormalities in the chest X ray, If the history and the examination warrant
- **Exercise tolerance** → Is the best way to assess the cardiac and pulmonary functions and the Hb level

Management

Acute retention of Urine

Post operative acute retention of urine

- Mechanism → May be due to spasms of bladder neck muscles due to unbearable pain
- Avoid catheterization- As it will introduce infections (Do not invite trouble)
- Give a painkiller → Pethidine, Morphine
- Provide privacy to urinate (As there is central inhibition when urinating in-front of public need to eliminate it)
- Let the patient stand up
Or take the patient to the toilet and open the tap (causes central stimulation)

Acute retention of urine of an old man

- It is a mechanical obstruction → Can try catheterize here not like earlier acute retention in post operative patient.

Post traumatic retention of urine

- You see little bit of blood in urethral meatus
- Never catheterize the patient
- Do temporary urine diversion by a means of Supra-pubic catheter

Spinal cord injury acute retention

In spinal shock bladder is full but patient will not complain of pain.

Retention with overflow → bladder fills and due to over stretch urine pass. No reflexes working

If suspect stricture

- Dilate stricture or cut stricture → Optical urethrotomy
- Stenosis in the meatus & can't pass a catheter - What are the measures you will take?
 - ✓ Use a small catheter size
 - ✓ Dilate
 - ✓ If fail, do supra-pubic catheterization

Stone → Push stone in to bladder , and destroy

Bladder tumour → Look Mx

An already catheterized patient - No UOP → How will you manage that patient?

- Check any block in catheter → Clot retention (Flush it with bladder syringe)
- See IV drip is working
- See the colour of urine → If dark colour urine that means kidneys function to its maximum & no ARF due to ATN. (In ATN urine like watery)
- But can be a pre renal cause
- Scanty urine
- If it's due to pre renal failure → Give N/S or Hartman IV drip. (Furosemide)

In a patient with chronic retention of urine, sudden decompression should not do. It causes,

1. Severe haematuria (Torrential bleeding) – Due rupture of the bladder vessels following sudden decompression
2. Hyponatraemia

To avoid that → Gradual intermittent decompression need to do

Benign Prostatic Hyperplasia

- New trend is moving away from surgery
- Earlier managed as an open surgery → transurethral, transrectal, retropubic
- Then endoscopic surgery
- Finally found medical management is better → Drugs
- Drugs
 - ✓ Specific α_1 -adrenergic blockers → Prazosin (bd), Terazocin once a day (Inhibit contraction of prostatic smooth muscles)
A/E – 1st dose hypotension – within 2 hours from the 1st dose (should be told not to stand up too quickly), Nasal congestion, priapism(rare)
 - ✓ Finasteride (5 α reductase inhibitor) – Blocks the conversion of Testosteron to DHT (Decrease size of prostate)
 - ✓ Combination therapy
 - ✓ Anti-cholinergics

Drugs	Mechanism of action	Side effects
α_{1a} -selective alpha blocker → Tamsulosin	Inhibit the contraction of prostatic smooth muscle	Postural hypotension and dizziness
5 α reductase inhibitors → Finasteride	Decrease the size of the prostate	Sexual dysfunction

Tamsulosin (Urimax™, Flomax™)

Tamsulosin is a selective α_1 receptor antagonist that has preferential selectivity for the α_{1A} receptor in the prostate versus the α_{1B} receptor in the blood vessels.

When α 1 receptors in the bladder neck and the prostate are blocked, this causes a relaxation in smooth muscle and therefore less resistance to urinary flow. Due to this the pain associated with BPH can be reduced.

A/E – Retrograde ejaculation, postural hypotension (Dizziness), headache, nasal congestion & palpitation

Complicated BPH (Retention, Calculi, Infection, Diverticulae)

- Transurethral surgery → TURP, TUIP, Laser
 - ✓ TUIP-Trans Urethral Incision of prostate
 - ✓ Laser
 - ✓ TURP(Transurethral resection of prostate)
- Open surgery → Retropubic, Tranvesical prostatectomy

Surgical management of prostatectomy

The indications for prostatectomy. The decision for surgery should be made by a urological surgeon

1. Severe symptoms with low flow rates and increased post void residue
2. Episodes of acute retention
3. Chronic retention and renal impairment
4. Complications of BOO- Stones, recurrent infection, bladder diverticuli

Prostatic CA

Mx of CA of prostate

- Androgen dependent
- Adenocarcinoma mainly in the peripheral zone of the prostate.
- Spread
 - Local : Involve the seminal vesicles, the bladder neck, trigone and the distal sphincter .Further upward extension obstructs the lower end of one or both ureters. The direct spread of the rectum is rare.
 - Blood stream: Most commonly metastasise to bone. Most frequently involved are the pelvic bones and the lower lumbar vertebrae. The femoral head, rib cage and skull are the other common sites.
Spread to breast, the kidney, the bronchus and the thyroid gland.
 - Lymphatic :Internal iliac nodes , external iliac nodes, retroperitoneal nodes , supraclavicular nodes
- Investigations
 - PSA levels
 - X-Ray lumbosacral spine- osteosclerotic deposits
 - Bone scan
 - Trans-rectal ultrasound guided biopsy
 - CT pelvis and abdomen(for staging)

➤ Staging (Clinical staging)

○ TNM

- ✓ T_{1a,b} – Subclinical. Rectal examination is normal. But PSA ↑
- ✓ T_{2a,b} – Palpable prostate. No capsular invasion
- ✓ T_{3a,b} – Locally advanced. Extend through capsule & extend to seminal vesicles
- ✓ T₄ – Invade adjacent structures other than seminal vesicles (Rectum & pelvic wall)

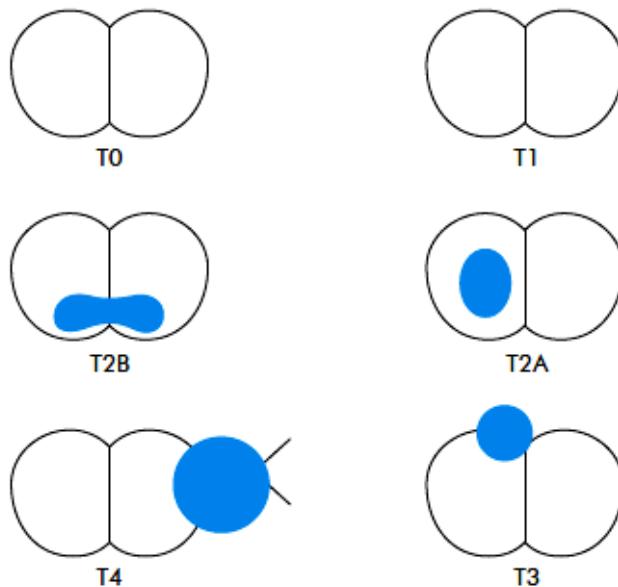


Figure 73.16 Tumour, node, metastasis (TNM) staging system for prostate cancer.

- Treatment options - Depend on stage of the disease, life expectancy of the patient and patient preference
- Localized cancer(T1 & T2)
 - Radical prostatectomy (Done only if the life expectancy is >10years)
OR
 - Radical radiotherapy
 - External beam radiation
 - Brachytherapy - Under transrectal ultrasound guidance, radioactive seeds are permanently implanted into the prostate
OR
 - Watchful waiting – If life expectancy less than 10 years

- Advanced disease (T3, T4 and metastatic disease)
 - Palliative
 - Hormone ablation (first-line therapy)
 - Surgical
 - Bilateral orchidectomy (Remove androgen stimulation)
 - Medical treatment
 - LHRH agonists
 - Initially stimulate hypothalamic LHRH receptors but, because of their constant presence (rather than the normal diurnal rhythm), they then down-regulate them, resulting in cessation of pituitary LH production and, hence, a decrease in testosterone production.
- T4 stage in a young male
 - Can't do orchidectomy
 - Can't inhibit testosterone
 - Can use testosterone receptor blockers
 - ✓ Give in intermittent doses

Chemotherapy is not useful

Radical radiotherapy

The complications are:-

- ✓ Radiation cystitis
- ✓ Dysuria
- ✓ Frequency
- ✓ Haematuria
- ✓ Contracted fibrotic bladder
- ✓ Radiation proctitis- Blood and mucous diarrhea, Tenesmus
- ✓ Impotence
- ✓ Anorexia, Nausea
- ✓ Malaise, Lassitude

Disucussion

What is the most common cause of BOO?

Benign prostatic hypertrophy

BPH is a diagnosis made on? Histology

Complications of out flow tract obstruction

1. Acute retention of urine
 - ✓ Patient has an intense desire to micturate with inability to do so
 - ✓ There is suprapubic pain
 - ✓ A palpable bladder
2. Chronic retention-
3. Urine infection-Fever,
4. Stone formation-painful haematuria
5. Obstructive uropathy-

Pre operative preparation of prostatectomy

- Informed written consent
- Steam inhalation and chest physiotherapy
- Oral hygiene
- Investigations to assess the fitness for surgery and anaesthesia
- Pre anaesthetic medication
- A shower
- Overnight fasting

Post operative care

- Monitoring → PR, BP, RR, Temperature chart, Drains
- External bladder irrigation → Using a three way Foley's catheter with normal saline
- When the efflux is clear this can be removed and can offer trial without catheter for 6-8 hours
- Prop up
- Steam inhalation
- Chest physiotherapy
- Sips on demand
- A light diet vespa
- Gentamicin 80mg IV 8 hrly 3 doses
- Pethidine 75mg IM
- Early mobilization

What are the complications of Prostatectomy?

1. Bleeding → Primary, Reactionary, Secondary

Primary bleeding

Occur at the time of prostatectomy. Diathermy coagulation and fluid/blood replacement will be helpful.

Reactionary bleeding

- ⊕ During the first 24 hrs
- ⊕ There will be → Tachycardia, Pallor, Tachypnoea, Hypotension, Intense thirst, Dizziness, Cold and clammy extremities
- ⊕ If the external catheter is not blocked the bleeding will be obvious. However torrential bleeding can result in clot retention

Clot retention

- ⊕ External irrigation is not functioning
- ⊕ There is severe supra-pubic pain and features of internal bleeding
- ⊕ Treatment
 - ✓ Flush the catheter with a bladder syringe
 - ✓ If there is no response, change the catheter
 - ✓ Some patients need a return to theatre and arrest of bleeding
 - ✓ Rapid infusion of normal saline
 - ✓ Pethidine 75mg IM
 - ✓ Tranexamic acid is useful

2. Secondary bleeding

- ⊕ Bleeding within first two weeks of surgery
- ⊕ It is usually secondary to infection
- ⊕ The features and the treatment will be the same as above

3. Perforation of bladder

- ⊕ Intraperitoneal
- ⊕ Extraperitoneal

4. Perforation of the capsule of the prostate

- ✓ The fluid get accumulated in the scrotum, penis, lower part of the abdomen and upper thigh
- 5. Sepsis**
- ✓ At the time of induction prophylactic antibiotics should be started. Eg:-gentamicin 80mg IV
- ✓ Otherwise, prostatectomy can result in septicaemia, wound infection, epididymo orchitis and bacterial endocarditis

6. Impotence

- ✓ Erectile dysfunction

7. Retrograde ejaculation

- ✓ When the bladder neck is incised there is damage to the internal sphincter urethrae
- ✓ Urinary incontinence:-Due to damage to the external sphincter urethrae. It improves with time with pelvic floor exercise.

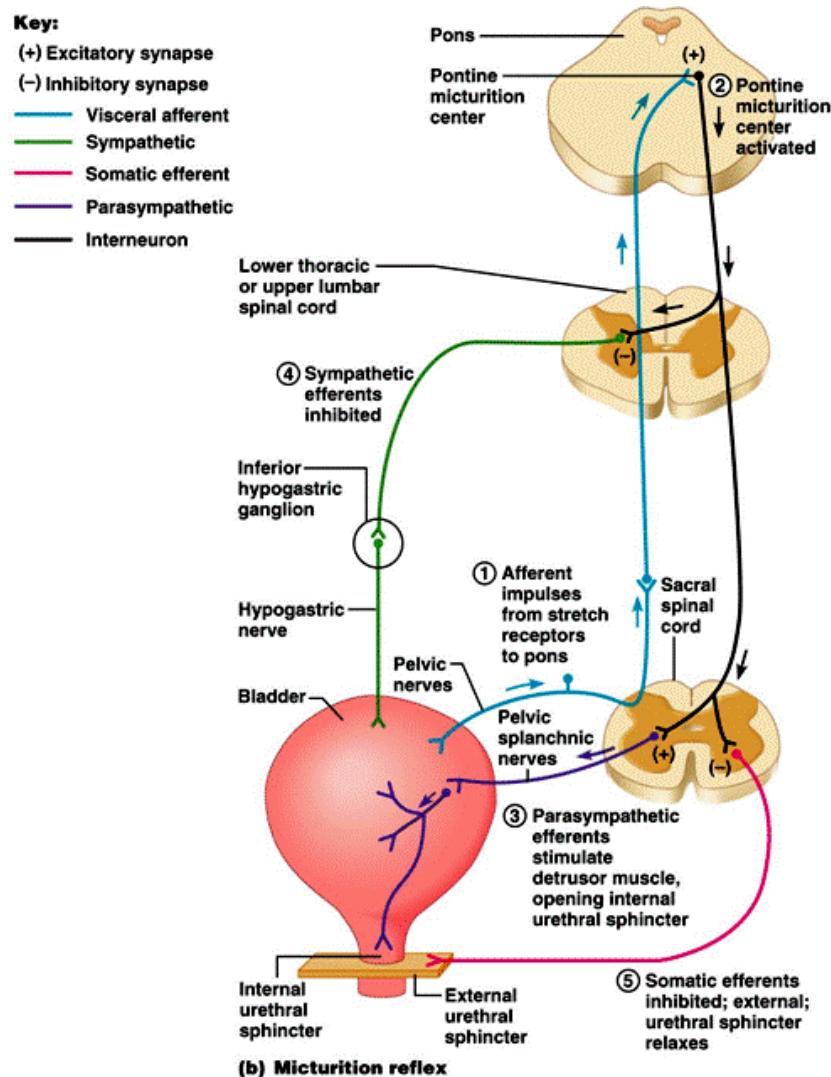
8. Urinary fistula

It generally responds to catheter drainage of the bladder

9. Bladder neck stenosis

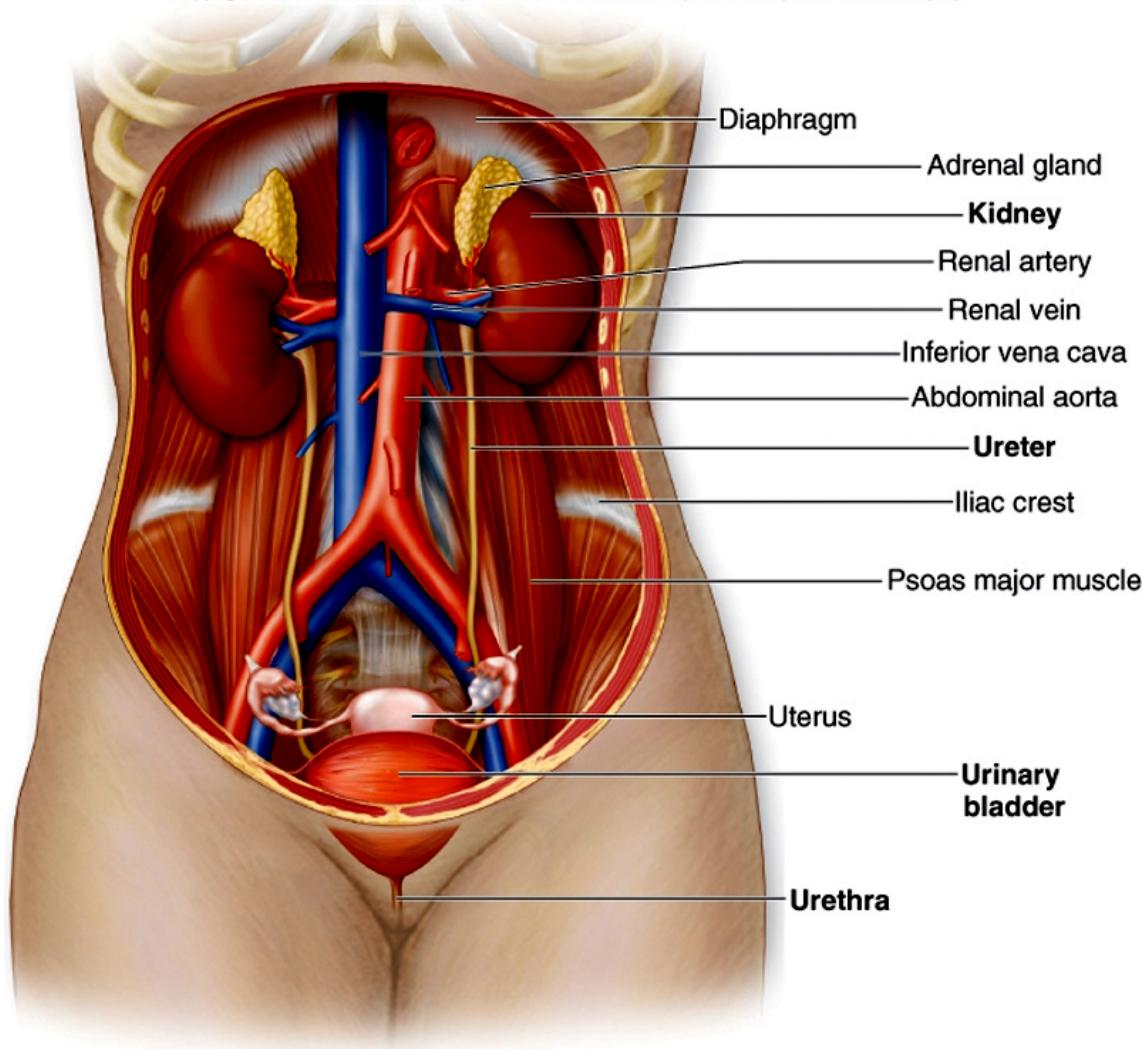
10. Urethral strictures
11. Recurrence
12. Wound related complications of open prostatectomy
 - I. Wound infection
 - II. Haematoma
 - III. Incisional hernia
 - IV. Urinary fistula
 - V. Keloid and hypertrophic scars
13. General complications
 - I. Deep vein thrombosis
 - II. Pulmonary embolism
 - III. Myocardial infarction
 - IV. Hypostatic pneumonia

Micturition reflex

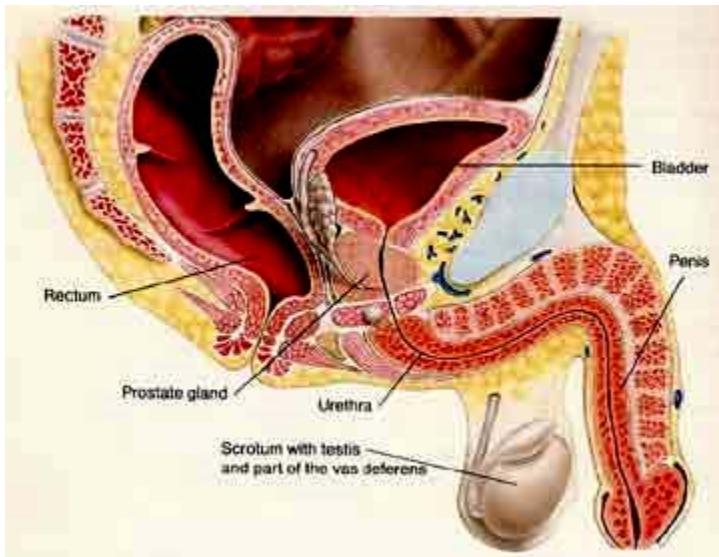
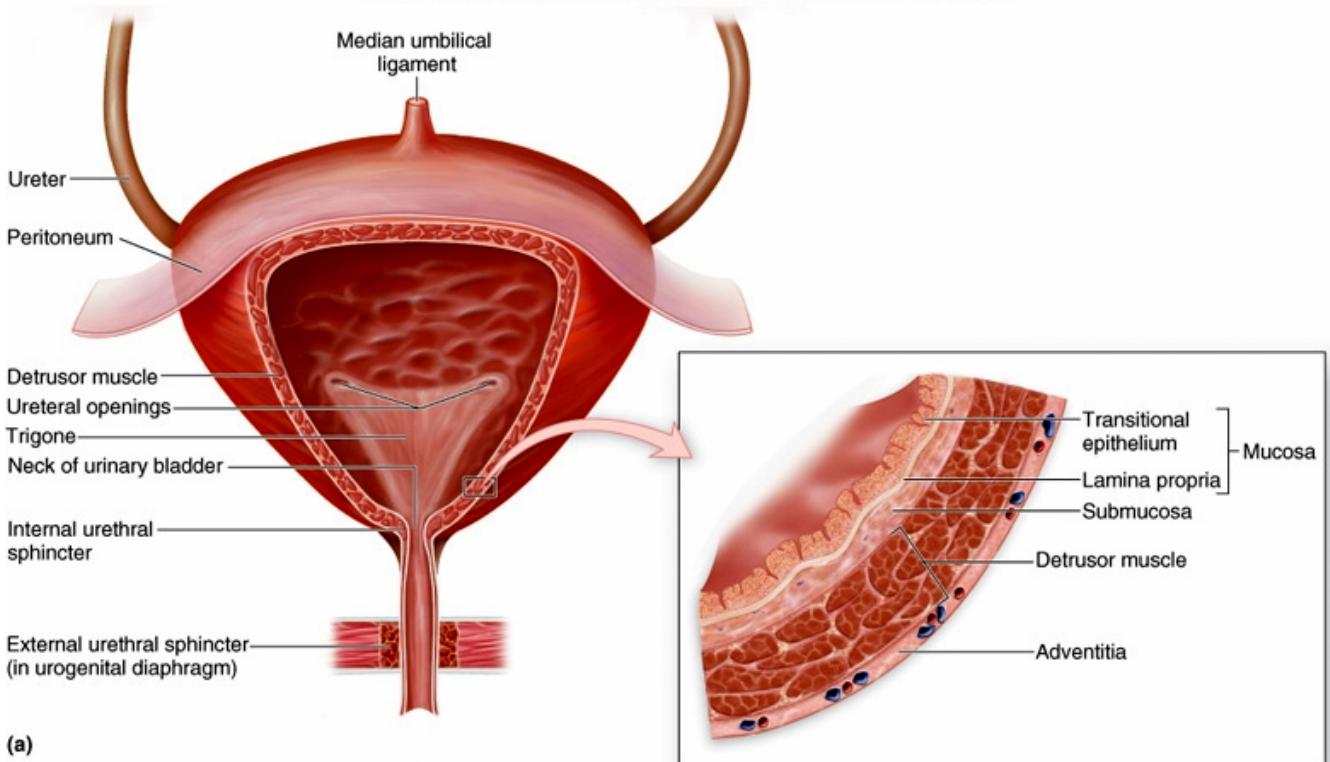


Anatomy of urinary tract

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Breast pathologies

DD :

1. Painless breast lump

- ✓ Fibroadenoma
- ✓ Carcinoma of breast
- ✓ Cyst
- ✓ Fibroadenosis (fibrocystic disease)

2. Painfull breast lump

- ✓ Fibroadenosis (fibrocystic disease)
- ✓ Cyst
- ✓ Abscess (Lactational, Post partum)
- ✓ Carcinoma
- ✓ Periductal mastitis

3. Pain in the breast w/o a breast lump

- ✓ Cyclical breast pain
- ✓ Non cyclical breast pain
- ✓ CA (rare)

4. Nipple discharge

- ✓ Duct ectasia – Multicoloured (yellow, brown, green)
- ✓ Intraduct papilloma – Blood stained / Clear
- ✓ Ductal CA in situ/ intraduct CA – Blood stained
- ✓ Breast abscess – Purulent discharge
- ✓ Lactation, galactorrhoea - Milk

5. Distortion or skin changes over nipple and areola

- ✓ Duct ectasia CA
- ✓ Paget's disease
- ✓ Eczema

6. Changes in breast size and shape

- ✓ Pregnancy
- ✓ Carcinoma
- ✓ Benign hypertrophy
- ✓ Rarely large tumors (phylloides tumour)

Age, marital status, number of children

Age - At young age after menarche (**Fibroadenoma**)
Obese elderly (**Traumatic fat necrosis**)
Lactating females (**Breast abscess**)
At any age, but rare in <30 yrs (**Breast CA**)

Sex: Common among females

Prevalence of breast CA in males is < 1% - mcq

PC

- A lump in breast for ___ duration
- Pain in the breast for ___ duration
- Nipple discharge for ___ duration
- Change in overlying skin for ___ duration
- Change in breast size and shape for ___ duration

HPC

History of the lump

How and when 1st noticed the lump

Progression of the lump over time (Any recent rapid enlargement)

Associated symptoms of the breast lump - Pain, alterations of the nipple or skin, any discharge from the nipple (if present its colour) – with duration

Whether noted any other lumps

Exclude benign breast diseases

History of trauma to breast - **fat necrosis**

Relationship of the size of the lump to the menstrual cycle - **Breast cyst**

Contact history of TB - **Tuberculous mastitis**

Lactating mother – **abscess**

Periodic B/L diffuse mastalgia, breast engorgement & lumpiness during menstruation –

Fibroadenosis/ Fibrocystic disease

Suggestive of breast CA

Hardness in the breast

Recent rapid enlargement

Pain

Overlying skin changes

Blood stained nipple discharge

LOA & LOW

History

1. **History of the breast lump**
2. **Exclude other breast conditions**
 - a. **Fat necrosis**
 - b. **Benign breast cyst**
 - c. **TB mastitis**
3. **Features of distant metastases**
4. **Aetiology/risk factors**
 - a. **Early menarche & late menopause**
 - b. **Parity**
 - c. **Age at 1st child birth**
 - d. **Breast feeding**
 - e. **OCP**
 - f. **HRT**

Hx for metastasis of breast CA

LN: Whether noticed any swellings in the armpits

Lung: Chronic cough, haemoptysis, chest pain, SOB

Liver: Right hypochondrial pain, LOA, LOW

Bone: Bone pain, intractable backache, features of hypercalcaemia-Confusion, Constipation, Polyuria, pathological fractures

Brain: Early morning headache with associated vomiting, adult onset seizures, weakness of the limbs

Ix and Rx done up to now

Any pathology in the other breast

Menstrual and lactating history

1. Age at menarche and menopause [early menarche (<11yrs) and late menopause (>51 yrs) have a high risk]
2. Parity - high risk in nulliparous
3. Age at first child birth – Low risk if young at the 1st child birth
4. Number of children and duration of breast feeding – Breast feeding is a protective factor

PMHx

Hx of breast CA, ovarian CA, endometrial CA, colorectal CA

Exposure to ionizing radiation during adolescence or early adulthood

Other comorbidities

PSHx

Hx of breast surgery

DHx

- OCP for > 5yrs before the 1st pregnancy
 - Postmenopausal HRT for > 5 yrs
- } Dose, frequency, duration

FHx

Breast CA, Ovarian CA, endometrial CA, Colorectal CA

SHx

Alcohol, smoking → Active / Passive

High cholesterol diet – rich in saturated fat

Concerns about the disease and about the surgery

Knowledge on self-breast Ex

Examination

O/E

Obesity - Risk factor in post-menopausal women (Increase conversion of steroids hormones to oestradiol in body fat)

Pallor

Icterus

Clubbing

Oedema of upper limb

Dilated veins – UL, neck

Sensory loss, muscle wasting

Breast Ex

Position - Patient seated, fully undressed to the waist, hands over waist

❖ Inspection

Enlargement - over the upper outer/ upper inner/ lower outer/ lower inner quadrants.

Overlying skin changes – discolouration, ulceration, peau d' orange, puckering

Nipples (Ask the pt to evert or squeeze for any discharge)

- Inverted / everted
- Discharge – colour
- Deviation
- Destruction

Accessory nipples, ectopic breasts

Ask to raise hands above the head

Peau d' orange, skin tethering become prominent

Axillary enlargements or skin changes

Support pt's hands and palpate axilla

Axillary and supraclavicular nodes – whether tender

❖ Palpation

When pt is in supine position

Palpate with the flat of hand (involve fingers and palm), press circularly

Start from normal breast and axilla

Go anticlockwise

Site

- ✓ In quadrants - if far from nipple (upper outer/ upper inner/ lower outer/ lower inner quadrants)
 - ✓ In clock position – if closer to nipple

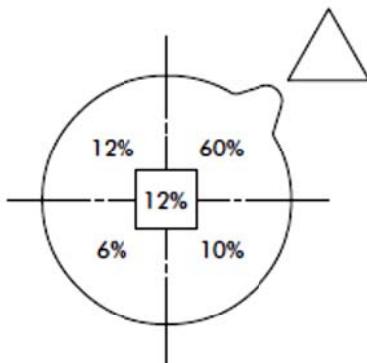


Figure 50.24 The relationship of carcinoma of the breast to the quadrants of the breast.

Shape

Size (length, width)

Surface – smooth/ irregular (nodular)

Consistency - soft/ firm/ hard

Margins – well defined, ill defined

Tenderness

Mobility (*In 2 planes*)

Attachment to skin

Attachment to underlying muscle (**If attached to pectoralis muscle become more apparent, tethered and less mobile with the hands pressed firmly against waist**)

Cardinal signs of CA of breast

- ✓ Common in upper outer quadrant (**60% of breast CA**)
- ✓ Overlying skin changes
- ✓ Hard/ firm
- ✓ Non tender
- ✓ Irregular lump
- ✓ Ill defined
- ✓ Blood stained nipple discharge
- ✓ Tethering or fixation of the lump
- ✓ Palpable axillary LN

Abdominal Ex

Hepatomegaly

Ascites

DRE

Nodules in pouch of Douglas (Blummer shelf)

VE

Ovarian lump – Krukenberg's tumor

Scalp

Lumps

Lumbar spine

Tenderness

Restricted movements

RS

Pleural effusion

Consolidations

Diagnosis – R/L sided benign/ malignant/ suspicious breast lump complicated with _____ & the TNM classification is _____.

How do you diagnose a breast lump

By the triple assessment

1. Clinical assessment – Hx & Ex
2. Radiological assessment – USS / Mammography
3. Pathological assessment – FNAC/ true cut biopsy/ open surgical biopsy

Investigations

❖ To confirm the diagnosis

- USS
 - In < 35 yr pts
 - In > 35 yr pts for further assessment in addition to mammography
 - Identifies the depth
 - Detect whether a cystic/ solid/ mixed lump
 - Identify invisible small lumps
 - Detects - *irregularity, ↑ echogenicity, Axillary LN, local invasion*
 - Advantages – less expensive, less invasive & freely available
Pt preparation is not required
 - Disadvantage – less accurate than a mammogram
- Mammography
 - Done only in pts > 35 yrs (Breast is more dense when < 35 years)
 - B/L 2 way mammography is used (Cranio-caudal section & medio-lateral oblique views)
 - Problems –
 - ⊕ Difficult to visualize the lump when the breast is large and pendulous
 - ⊕ Cannot perform when pt has mastalgia
 - Characteristic features of a breast CA on mammography
 - ⊕ Micro calcifications & spicular appearance
 - ⊕ Dermal oedema
 - ⊕ Distortion of architecture
- FNAC
 - Provide relevant clinical details & a diagram on the request form
 - Using a blue/ green(23G) needle into a **10ml syringe**
 - Need **3 glass slides** – 2 slides to make smears and one is as a spreader
 - Immediately fix by **95% ethanol** to prevent air drying
 - **OPD** procedure & cheap
 - Anaesthesia not required
 - Quick to perform, process & interpret
 - No complications except for bruising
 - Sensitive – minimum number of false negatives
 - Specific – minimum number of false positives
 - Is a cytological diagnosis
 - Results
 - ⊕ **C1** = inadequate
 - ⊕ **C2** = benign breast cells
 - ⊕ **C3** = indeterminate cytology
 - ⊕ **C4** = suspicious of malignancy
 - ⊕ **C5** = diagnostic of malignancy

- Tru cut biopsy (core biopsy)
 - Under local anaesthesia taken using trucut biopsy needle
 - Is a tissue diagnosis
 - Helpful when FNAC is non diagnostic
 - Can also assess oestrogen, progesterone and human epithelial receptor status
- ❖ To detect metastatic spread (To stage the disease)
 - CXR
 - Cannon ball appearance – Metastatic deposits
 - Military mottling (multiple deposits)
 - Pleural effusion
 - Solitary deposits
 - Osteolytic lesions involving ribs & thoracic vertebrae
 - Mediastinal lymph node enlargement
 - Assesses the heart and any other comorbidities of the lung
 - USS – abdomen
 - Liver secondaries
 - Ascites
 - Lymph nodes
 - Krukenberg's tumor (2^{ry} ovarian tumor)
 - CT scan
 - Bone scan (T^{99}) – Not indicated routinely
 - Skeletal survey – also not routinely indicated.
Only the relevant x-rays are requested if there is any hip pain/ skull lump/ intractable backache
 - Liver function test
- ❖ Other Ix
 - Oestrogen receptor status – on a core biopsy sample
 - Tumor markers – CA 15.3, steroid hormone assay
- ❖ To assess the fitness for Sx
 - FBC – Hb, PCV, Plt
 - CXR & echocardiogram in older patients & patients with risk factors
 - ECG
 - FBS
 - S. creatinine
 - UFR

Management

Mx of a fibroadenoma

- < 3 cm → No surgical intervention
- 3- 5 cm → controversial, discuss with the pt
- > 5 cm → Giant fibroadenoma – Excisional biopsy with a rim of normal tissue.
- Indications for surgery in a fibroadenoma
 - Giant fibroadenoma
 - Lump increasing in size
 - Symptomatic lump
 - Pt preference
 - If presenting for the 1st time after 40yrs.
- Surgery – lumpectomy under GA

Mx of breast CA

- Staging
 - **TNM classification**
 - Colombia classification
 - Manchester classification

TNM staging

Tumor grade

T0 – no evidence of 1st tumor

T1 – Tumor < 2cm

T2 – Tumor 2 to 5 cm

T3 – Tumor > 5 cm

T4 – Tumor of any size with skin or chest wall involvement

Node grade

N0 – no regional lymph node metastasis

N1 – mobile ipsilateral axillary nodes

N2 – fixed ipsilateral axillary nodes

N3 – Ipsilateral internal thoracic (internal mammary or parasternal nodes) – ***Not palpable***

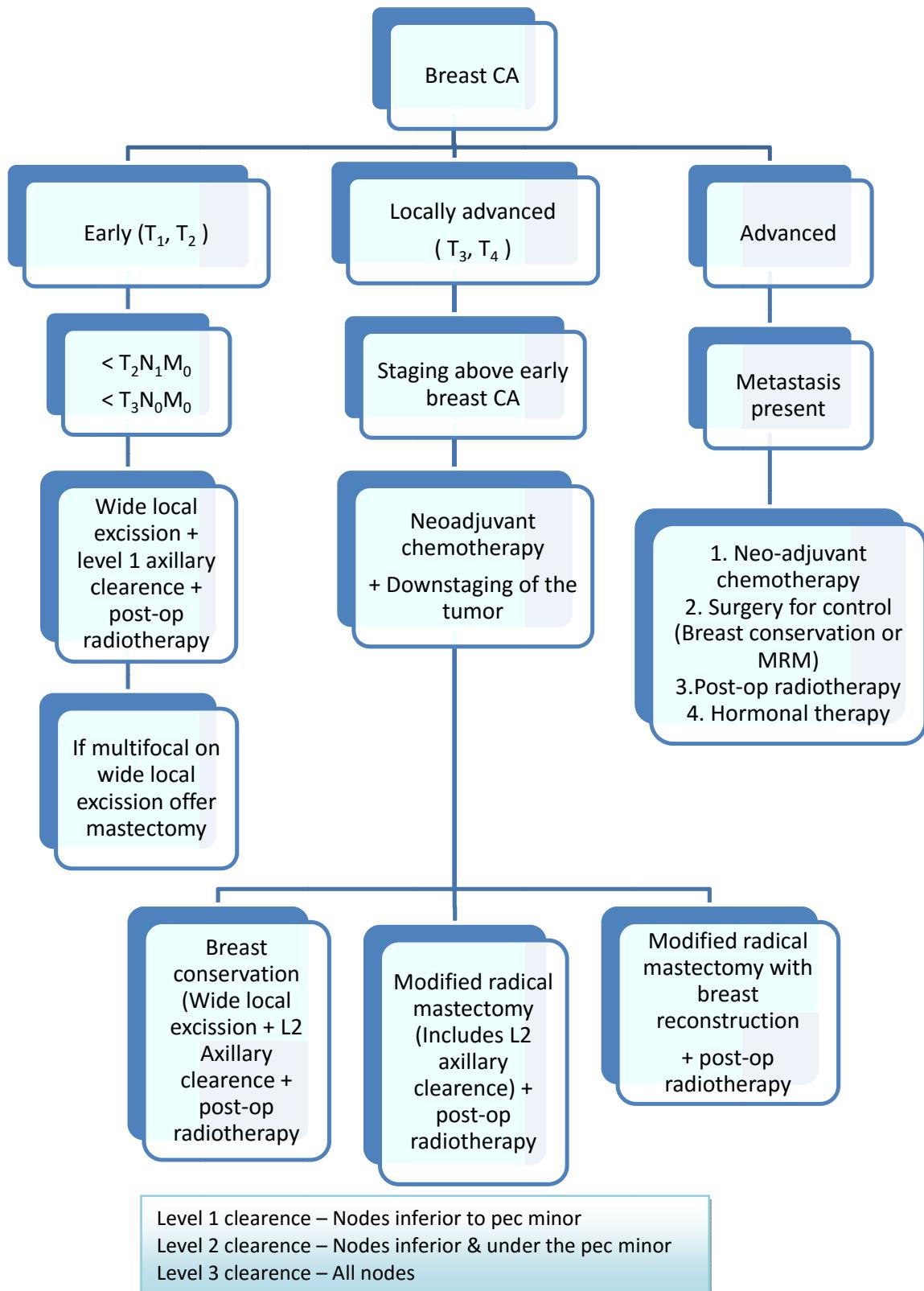
Distant metastasis

M0 – no metastasis

M1 – distant metastasis

Tx / Nx / Mx → x = cannot be assessed

➤ How does the M_x change according to the TNM staging



Surgery for early breast CA

- ✚ Wide local excision
 - Removal of the lump with a margin of the normal breast tissue (**2 cm margin**)
 - It's combined with sentinel node biopsy, axillary node sampling or axillary clearance (**level II axillary clearance usually**)
- ✚ Simple mastectomy & axillary node sampling or axillary clearance
- ✚ Modified radical mastectomy (excision of whole breast, large portion of skin – centre which overlies the tumour always include, all of the fat, fascia & level 2 axillary clearance. Pec major is preserved. Pec minor is either divided or retracted)
- ✚ Systemic adjuvant treatment = chemotherapy &/ or anti oestrogen therapy
 - Oestrogen blockade – Tamoxifen
 - Oestrogen deprivation therapy – anastrozole, letrozole, exemestane

Surgery for locally advanced breast CA

- ✚ Neoadjuvant chemotherapy or radiotherapy (chemotherapy or radiotherapy prior to surgery)
- ✚ Salvage mastectomy

Surgery for advanced breast CA

- ✚ Local palliation – Radiotherapy for fungating lesions
- ✚ Radiotherapy to localized bony metastases
- ✚ Aspiration of pleural effusion & instillation of cytotoxic agents
- ✚ Hormonal manipulation – Tamoxifen
- ✚ Chemotherapy
- ✚ Extensive surgery for chest wall defects requiring grafting with myocutaneous flaps
Eg: latissimus dorsi flaps

Immunohistochemistry

- a. Oestrogen receptors
 - b. Pregesterone receptors
 - c. Her-2-Neu receptors
- ✚ Best combination → a +ve, b +ve, c -ve → consider hormonal therapy
 - ✚ Worst combination → a -ve, b -ve, c +ve → No hormonal therapy

Recurrent disease

- ✚ Radiotherapy
- ✚ Hormonal manipulation or chemotherapy – for systemic disease

Follow up

- ✚ Routine self examination of the operation site & other breast
- ✚ Follow up for 5 years - pt's who are disease free can be discharged after 5 years
- ✚ Mammography to contralateral breast every other year & to ipsilateral breast every year if conserved.

Prognosis (staging according to the Manchester classification)

- ✚ Stage I – 80% 5 year survival
- ✚ Stage II – 50% 5 year survival
- ✚ Stage III – 15% 5 year survival
- ✚ Stage IV – 5% 5 year survival

➤ Pre-operative preparation for mastectomy

- Informed written consent
- Pre-op Staging
- ECG, urine sugar, FBS, FBC(Hb & platelet), PT/INR, S.creatinine
- CXR & USS of the abdomen including the liver
- Previous biopsy report – FNAC or true cut biopsy
- Side of the mastectomy should be noted and pt's wrist label should be labeled correctly
- Reservation of cross matched blood
- Advice the pt to have a bath on the day prior to the Sx and shave the axilla on the same side
- BHT with the relevant ix should be sent to the theatre with the pt
- Keep fasting for 6 hrs
- Steam inhalation
- Pre op antibiotics – Cefuroxime (At induction)

➤ Post operative Mx

- Keep the pt supine on bed with the arms by the side of the pt
- Administer adequate post op. analgesia – SC Morphine 5 mg
- Watch for bleeding & the functioning of the surgical site drain
- Monitor pulse, BP & RR
- IV fluids until oral is started
- Start oral fluids in 2hrs once the peristalsis has returned
- 2 more doses of Cefuroxime at 6hrs & 12 hrs from the time of the op.
- Steam inhalation
- Limb & chest physiotherapy
- Make the pt sit the next day
- Early ambulation
- A shower the following day
- Remove the drain when the drain is reducing & when the daily output is < 25ml
[important properties of a surgical drain – closed drainage system, with a patent tube, draining due to negative pressure]
- Suture removal after 7-10 days
- Review the pt in the clinic with the histopathology report
- Refer the pt to oncologist to decide on adjuvant therapy

Complications of mastectomy and axillary clearance

1. Due to mastectomy

Immediate

- Bleeding
- Haematoma
- Seroma
- Wound infection & suppuration (after 3-4 days)
- Breast cellulitis

Late

- Flap necrosis (rare, but specific for mastectomy)
- Keloid
- Hypertrophic scar

2. Due to axillary clearance

- Lymphoedema of the ipsilateral upper limb – high risk erysipelas & cellulitis
Mx – limb elevation, massaging, compression garment, prophylactic penicillin therapy for cellulitis
- Frozen shoulder
- Axillary vein thrombosis
- Neuropraxia – resolve spontaneously in about 6/12
 - a) **Long thoracic nerve** – serratus anterior
Ask the pt press the straightened hands against the wall firmly & observe the pt from the back for any winging of the scapula.
 - b) **Intercostobrachial nerve** – axillary sensation
Check the axillary sensations
 - c) **Thoracodorsal nerve** – latissimus dorsi
Ask the pt to press his hands firmly on the bed while the arms are on either side of the body & observe from the back for the contraction of the muscles.

➤ Chemotherapy

For the Rx of systemic disease, specially with micro metastatic disease.

- Cyclophosphamide
- Methotrexate
- 5 – Fluorouracil

SE –

- ✓ LOA, N, V
- ✓ Alopecia (temporary)
- ✓ Bone marrow suppression & immunodeficiency
- ✓ Chemical thrombophlebitis

➤ **Radiotherapy**

Used for the loco-regional disease with infiltration of the chest wall.

SE –

- LOA, N, V
- Somnolence
- Lethargy
- Exfoliation, erythema, blister
- Late onset coronary artery disease, MI, cardiac tamponade & constrictive pericarditis
- Osteonecrosis of ribs
- Fibrosing alveolitis

➤ **Hormonal therapy**

Effect & the prognosis is good in oestrogen receptor positive and node positive pts.

- Tamoxifen

Rx duration – 5 years

Uses –

- ✚ As an adjuvant therapy
- ✚ Also reduce the incident of contralateral breast CA
- ✚ Protective against MI and osteoporosis

AE –

- ✓ N, V
- ✓ Hot flushes
- ✓ Weight gain
- ✓ Vaginal dryness
- ✓ Eye problems
- ✓ High risk of endometrial CA

Other hormonal therapies -

- ✚ Aromatase inhibitors - anastrozole, letrozole and exemestane

➤ **What is a sentinel node biopsy**

- A sentinel node is a lymph node which initially receives lymphatic drainage from a site of pathological process.
- A dye is injected to the breast lump (Methylene blue) after the induction of anaesthesia
- Then the lymph node which first takes up the dye is detected
- Then a frozen section is taken within 1/2hr and examined for malignant cells.
- If this node is negative for malignant cells; it is presumed that the axillary nodes are devoid of metastasis. Hence an axillary clearance is not done.

Mx of benign breast diseases

1. Fibroadenosis/ Fibrocystic disease

- ✚ Evening Primrose oil
 - ✚ Danazole
 - ✚ Bromocriptine
 - ✚ Tamoxifen
- } For intractable symptoms

2. Breast cyst

- ✚ Aspiration to dryness

3. Acute bacterial mastitis

- ✚ Antibiotics
- ✚ Aspiration
- ✚ Incision & drainage

4. Breast abscess

- ✚ PCM & resting
- ✚ Antibiotics
- ✚ Aspiration
- ✚ Incision & drainage

5. Galactocele

- ✚ If < 3 cm – No Rx
- ✚ If > 3 cm – Excision or drainage under GA

6. Intraduct papilloma

- ✚ Excision of a single duct (microdochectomy)

7. Phyllodes tumor

- ✚ If small – Wide excision to achieve clear margin around the tumor
- ✚ If large or if recur – Mastectomy

Discussion

BREAST CA

What are the risk factors for breast CA

- ❖ High risk
 - Female sex
 - PHx of breast CA
 - FHx of breast, ovarian, endometrial, colorectal CA
- BRCA 1 – Breast CA , Ovarian CA
- BRCA 2 – Male breast CA , Prostatic CA
- Lynch type 2 – Breast CA, Colorectal CA, Ovarian CA, Endometrial CA

- ❖ Moderate risk
 - Early menarche
 - Late menopause
 - Nulliparity
 - Less breast feeding
 - Atypical lobular breast disease
 - Breast disease with ductal hyperplasia
 - Old age at the 1st pregnancy
 - Exposure to ionizing radiation during adolescence or early adulthood
- ❖ Low risk
 - OCP - >5 yrs before the 1st child birth
 - Hormonal replacement therapy
 - Alcohol
 - Smoking
 - High cholesterol/ saturated fat diet

Why do we need to Rx breast CA urgently

Because malignant cells enter the blood stream from day 1

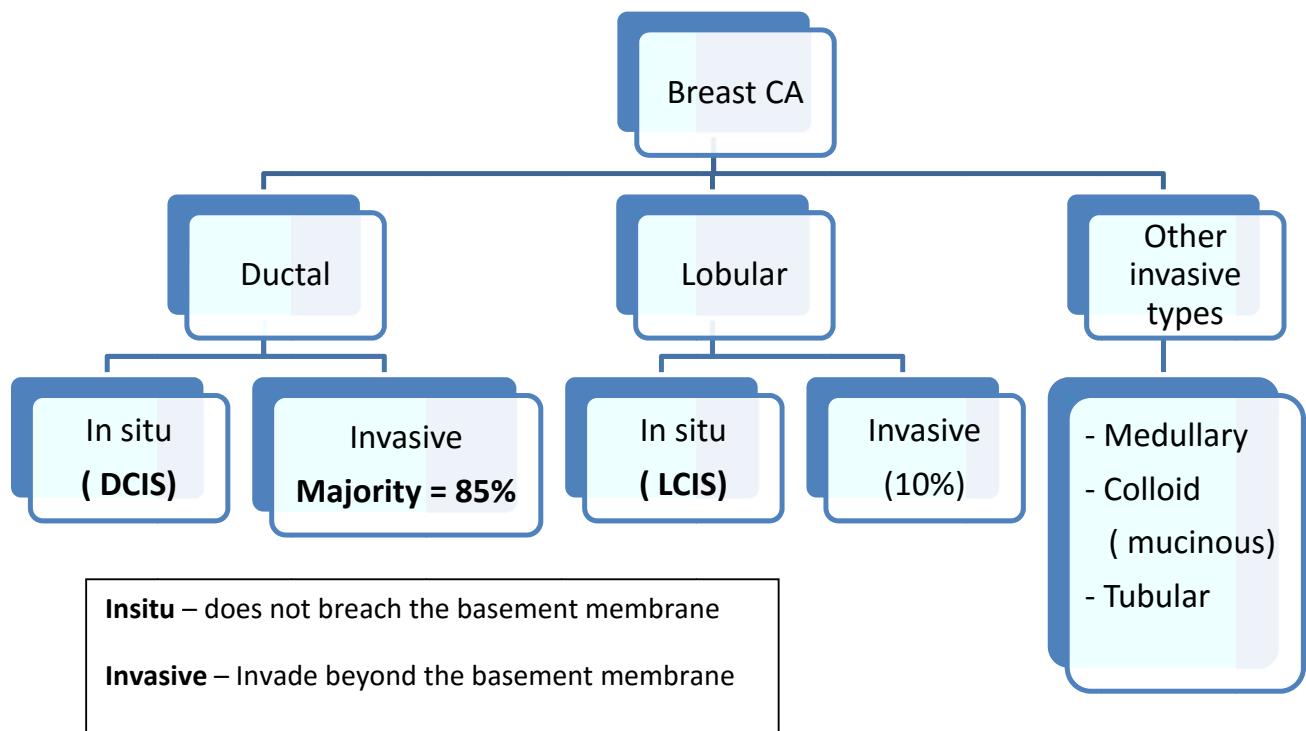
Incidence of breast CA in SL (2005)

18.5 per 100,000 females

Prognosis of breast CA

- Depend on;
 - ✓ Tumor size
 - ✓ Lymph node status
 - ✓ Chronological age of the tumor
 - ✓ Invasive and metastatic potential
 - ✓ Hormone receptor status
 - Oestrogen & progesterone receptors – **Good prognosis**
 - HER 2 / NEU receptor – **Poor prognosis**
- To determine the prognosis – Nottingham prognostic index

Pathology of breast CA



Clinical presentation of breast CA

- 1) As local disease
 - ✓ Breast lump
 - ✓ Nipple discharge (blood stained, unilateral, single duct)
 - ✓ Breast pain
 - ✓ Altered contour of breast
 - ✓ Ulcer
- 2) As regional spread
 - ✓ Palpable axillary or supraclavicular lymph nodes
- 3) As systemic symptoms
 - ✓ Shortness of breath, haemoptysis
 - ✓ Back ache
 - ✓ Severe loss of appetite and loss of weight
 - ✓ Jaundice
 - ✓ pathological fractures

How does breast CA spread

- A. Local spread – direct invasion
 - ✓ Involvement of skin
 - Tethering
 - Peaud O'range
 - Ulceration

- ✓ Enroachment of the nipple
 - Nipple retraction
 - Nipple deviation
- ✓ Infiltration into muscles and bone
 - Pectoralis major / serratus anterior
 - Chest wall / ribs

B. Lymphatic spread (regional)

- ✓ Axillary nodes
- ✓ Supraclavicular nodes
- ✓ Internal mammary chain

C. Haematogenous spread

- ✓ Opposite breast
- ✓ Lung & pleura
- ✓ Liver
- ✓ Brain
- ✓ Bone

D. Trans coelomic spread

- ✓ Krukenberg tumor of the ovary

E. Infiltration of bone marrow

- ✓ Leukoerythroblastic blood picture
- ✓ Hypercalcaemia
- ✓ Increase alkaline phosphatase

What are the bones which mainly get involved in the metastasis of breast CA

Skull, spine, ribs, long bones, pelvis

- ✓ Due to the presence of more bone marrow

FIBROADENOMA

- The commonest benign tumor
- Common among young, peak in 3rd decade
- Solitary / multiple
- Discrete, soft/firm, mobile lump
- Not attached to skin or underlying structures
- If size is > 5 cm = Giant fibroadenoma
- Pathology –
 - Macroscopy
 - Well defined and encapsulated
 - Firm mass
 - Cut surface – uniformly whitish and firm
 - Microscopy
 - Proliferation of both glandular & stromal elements
 - Stroma – loose and fibroblastic

Anatomy of the breast

❖ Breast is a **modified sweat gland**.

❖ **Lymphatic drainage of the breast**

- ⊕ Axillary nodes - 75%
- ⊕ Parasternal nodes
- ⊕ Postintercostal nodes
- ⊕ Infraclavicular nodes
- ⊕ Interpectoral nodes
- ⊕ Lymphatics within the breast communicate with a subareolar plexus of lymphatics
- ⊕ Superficial lymphatics of the breast have connections with those of the opposite breast & ant. abdominal wall

20- 30 lymph nodes arranged in 5 groups.

❖ **What are the levels of axillary lymph nodes**

Level 1 – Inferior to the pectoralis minor

Level 2 – Behind the pectoralis minor

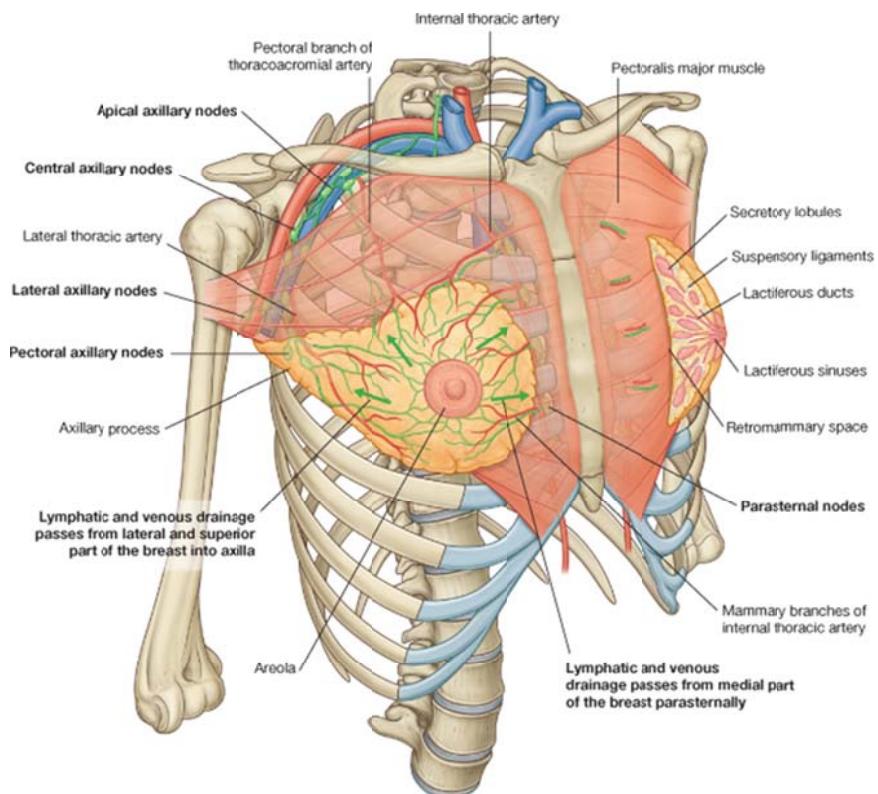
Level 3 – Superior to the pectoralis minor

❖ **Blood supply of the breast**

- ⊕ Lateral thoracic artery (from axillary artery)
- ⊕ Internal thoracic artery branches of 2nd and 3rd intercostal spaces
- ⊕ Branches from posterior intercostal arteries
- ⊕ Pectoral branches of the thoracoacromial arteries
- ⊕ Venous drainage – internal thoracic vein and axillary vein.

]

Mainly

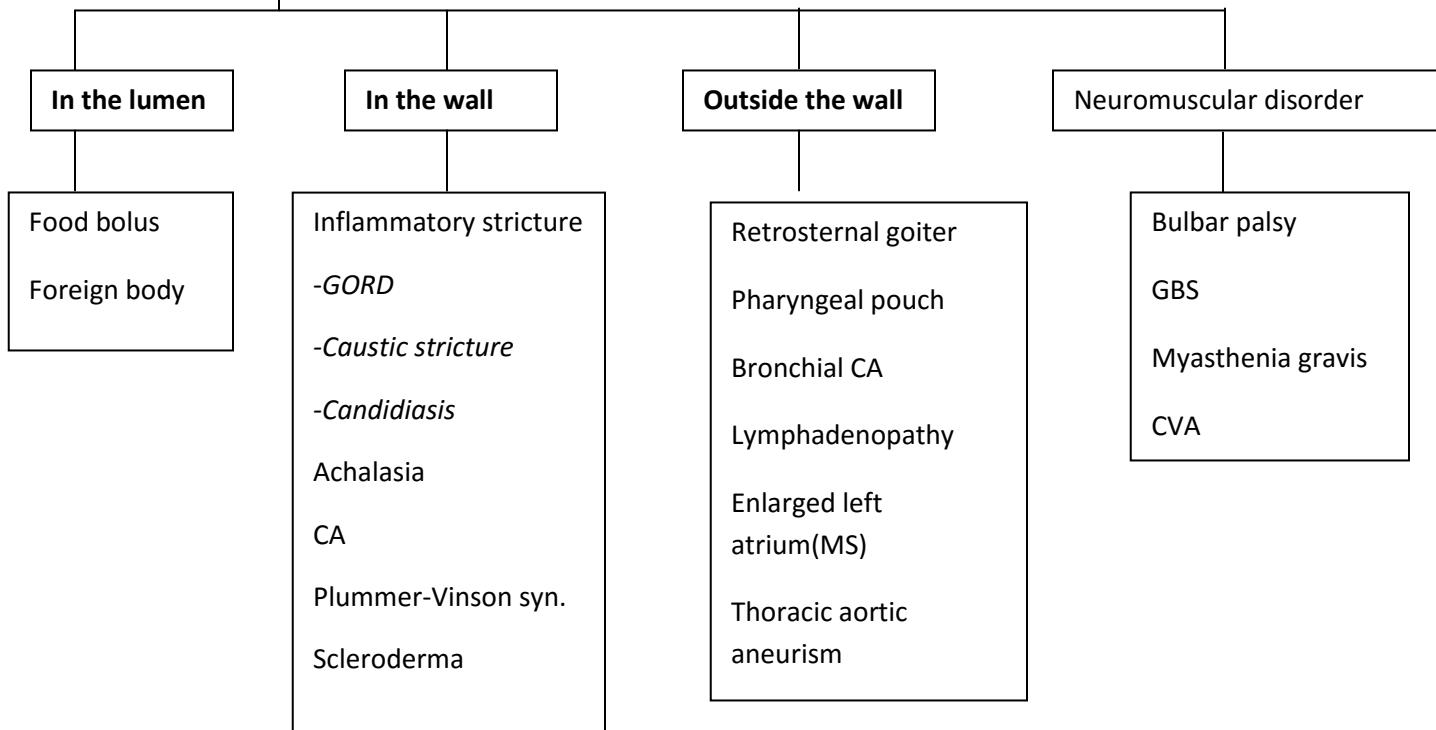


DYSPHAGIA

D.D

1) Congenital – Oesophageal atresia

2) Acquired



History

Age

- Newborn : inability to swallow feeds, Dribbling , choking attacks , chest infections
- 30 – 50 : achalasia
- 60 -70 : CA

Sex

CA M: F female common in SL 11.8 prevalence)

Occupation

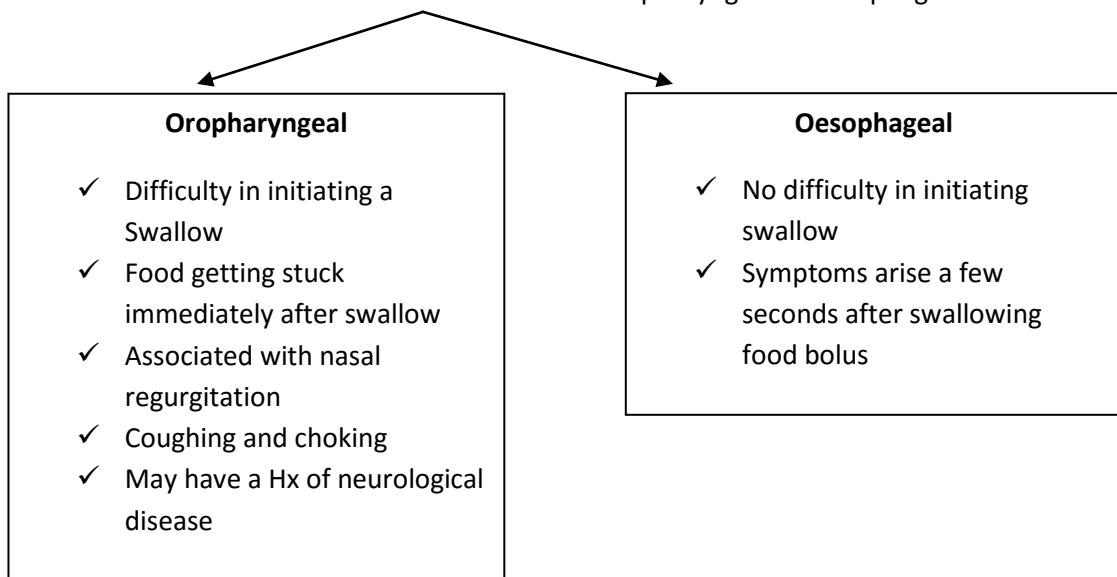


PC

- Difficulty in swallowing (progressive)
- Duration of symptoms
 - ✓ Long Hx of symptoms – benign causes more likely
 - ✓ Weeks – months – CA

HPC

- Onset –
 - Acute : Foreign body
- Progression of symptoms
- Dysphagia is for liquids or solids
- Locate the anatomical site of the lesion as oropharyngeal or oesophageal



- Exclude the oropharyngeal causes of Dysphagia.
- Locate the anatomical site to the oesophagus.
- Pattern of dysphasia
 - Progressive dysphagia
 - ✓ Benign or malignant stricture or external compression compression of the oesophagus
 - Intermittent
 - ✓ This indicate neuromuscular cause (achalasia) or peptic stricture
- Next describe the dysphagia is
 - ✓ Solid= liquid ,solid> liquid , liquid> solid
 - ✓ Level at which difficulty is encountered can be identified by the patient -CA
- Associated pain when food passing – CA, achalasia, oesophagitis

Ask the following points to come to a diagnosis

carcinoma of the oesophagus

- progressive dysphagia initially or solids then for liquids
- Associated regurgitation of undigested food
- LOW in spite of good appetite
- Initially LOW then develop LOA may indicate to lower oesophageal CA spreading to Stomach
- Features of local spread -**haematemesis** and **melaena, hoarseness of voice**(CA of upper oesp.) , **interscapular pain**
- Hx of hiccups - Diaphragm irritation
- Features of distant spread
 - : Neck lumps
 - : Liver - jaundice, RHC pain
 - : Lungs - persistant dry cough, SOB, coughing up of blood
 - : Brain - Early morning headache vomiting, adult onset seizure
 - : Bone - intractable backache, bonepain

Benign oesophageal strictures

- Peptic
 - intermittent progressive dysphagia
 - long Hx of symptoms
 - Hx of burning pain which is worse in recumbency / bending down (**GORD**)
- Other
 - ingestion of corrosive substances
 - Exposure to radiation (radiotherapy)

Oesophageal webs or rings

- Ask for long standing anaemia

Disorder of motility

Achalasia

- Dysphagia for solids and liquids both.
- longer duration of symptoms.
- liquid >solids, dysphagia relieved by changing posture.
- Can have intermittent dysphagia which later become progressive
- LOW
- Halitosis
- Regurgitation of undigested food specially at night when lying flat.
- Ask for episodes of nocturnal cough and aspiration
- High fever, recurrent chest infections ,

Scleroderma

- Hx of changes in skin, around lips, in fingers , past Hx of renaud's phenomina

After establishing the possible cause for the symptoms of the patient describe the impact they cause for the patient's life.

Past medical history

- Neurological disease- stroke/bulbar/pseudo bulbar palsy
- GORD
- Recurrent aspiration pneumonia
- TB – lymphadenopathy
- DM, HT, BA – fitness for surgery
- Exercise tolerance- to assess the fitness for surgery

Past surgical history

- Thoracic surgery
- Radiotherapy
- UGIE (stricture)
- Bronchoscopy (CA bronchus)

Drug history

- Anti-gastritis drugs
- Steroids (candidiasis)

Dietary history

- To assess the nutrition of the patient
- Intake of fresh fruits and vegetables

Family history

- Malignancy ,achalasia

Social history

- Smoking, alcohol, beetle chewing – Risk factors
- High contents of canned food – nitrosoamines
- Social circumstances- estate sector (eating rotti ,drinking hot tea, sooth mixed rice)
- Family support
- Income
- Psychological status of the patient

EXAMINATION

General

- Ill looking / well looking
- **Weight, BMI**
- Emaciated (weight loss)
- Pallor
- Icterus
- Glossitis, angular stomatitis, koilonychia- anaemia, plummer winson syn
- Lymphadenopathies (supraclavicular LN, cervical)
- Neck lumps –goitre
- Scleroderma – beaked nose, tight skin, small mouth ,reynaud's
- Nicotine stains
- Tylosis

Tylosis palmoplantar is an autosomal dominant disorder characterized by hyperkeratosis of palms and soles. Lesions start during childhood and are more evident in areas of pressure. Familial tylosis palmoplantar comprises two forms: epidermolytic and non-epidermolytic. Patients with the epidermolytic variant have up to 40% higher chance of developing squamous cell carcinoma of the esophagus. The association of tylosis palmoplantar with esophageal cancer is called Howel-Evans syndrome.



Abdomen

- Sister Mary Joseph nodule (if the tumor had spread to stomach)
- Lumps in the epigastric region (CA stomach)
- Irregularly enlarged liver (by 2ry deposits)
- Digital rectal examination – **MUST**
 - ✓ Trans- coelomic spread of tumor and deposits in POD

CVS

- Features of Mitral stenosis
 - ✓ Mid diastolic murmur
 - ✓ Previous surgical scars
 - ✓ Tapping apex
 - ✓ Malar flush
 - ✓ Peripheral cyanosis

Respiratory

- Features of bronchial CA – haemoptysis
- Pleural effusion

CNS

- Features of neuro- muscular disorder
- Horner's Xn

DISCUSSION

What are the investigations you would like to perform?

Investigations should be performed for 3 basic objectives in this patient.

- ✓ Confirming the diagnosis(histological diagnosis)
- ✓ Staging of the disease
- ✓ Assessment of the Fitness for surgery

1) Confirming the diagnosis

- UGIE – to visualize the probable lesion and take biopsy
 - Assess the length of the lesion
 - can be used therapeutically to dilate, so improving nutrition before a definitive operative intervention
 - Informed written consent before the procedure
 - Overnight fasting



- Barium swallow :
 - * used when the endoscopy is difficult to perform due to the obstruction.
 - the first-line investigation after the history of dysphagia
 - characteristic image of an irregular stricture with shouldered margins, 4-10 cm long and often tortuous 'rat tail appearance'
 - a tracheo-oesophageal fistula may also be demonstrated



2) Staging

To assess the 'T' stage

- Contrast CT of the thorax / endoscopic ultrasound
 - Assess the depth of the lesion
 - *To assess the direct invasion of the descending aorta. (To prevent death on table)*
 - In CT thorax can see different levels of oesophagus
 - Local infiltration
 - lymphnode spread (hilar nodes)

To assess the 'N' stage

- Difficult to assess the lymphatic spread of the tumor.
- Supraclavicular, coeliac, mediastinal LN are affected.

To assess the M stage

- USS liver – liver deposits, coeliac LN , Ascites)
- CECT abdomen and pelvis
- Bronchoscopy – if tracheo- bronchial involvement is suspected

3) Assess the fitness for surgery

- Blood
 - FBC: low Hb- anaemia, Plummer Vinson Syn.
 - ESR : malignancy & scleroderma
 - Grouping and cross matching at least 4 units
 - FBS
- Heart
 - ECG (Left atrial hypertrophy)
 - Echocardiogram
- Lungs
 - CXR : Foreign body –radio opaque, air –fluid level in achalasia
 - Lung function tests (this is most important as the surgery may involve thoracotomy)
- Renal
 - BU & SE
 - S.creatinine

- Liver
 - LFT : increased ALT – liver secondaries
 - Serum albumin :extremely important as it may reflect nutritional status

What are the principals of management of this patient?

- Mx depends mainly on the stage of the disease and fitness of the patient to undergo a major surgery.
- Therefore pre-operative status and co-morbidities of the patient are extremely important.
- Curative resection should be attempted in patients without distant metastasis and limited local lymph node spread.
- Tumor length < 5cm is considered as an operable tumor.
- If the tumor is not resectable palliative measures such as ablation of the tumor and stenting can be performed.

Management

- Upper third lesion:
High dose radiotherapy is indicated for lesions up to 5 cm long. Vital structures in the mediastinum closely related to the upper third make surgical clearance and resection very difficult.
- Middle third lesion :
Early tumours are resectable.
Again, radical radiotherapy may be indicated
- Lower third lesion :
Most accessible surgically; adenocarcinomas are radio resistant

How would you prepare this patient for the surgery?

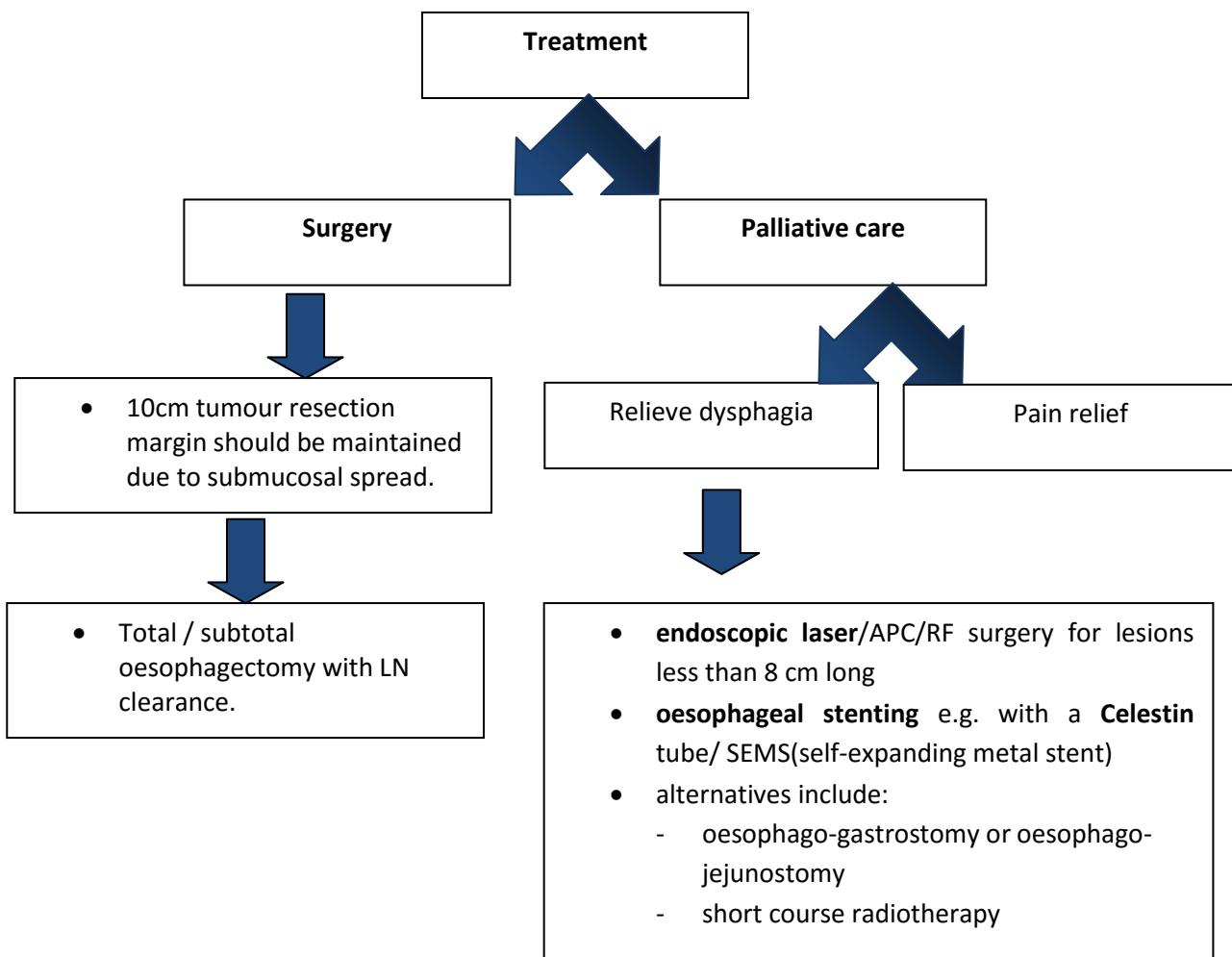
- Breaking the bad news and explain the future cause of treatment
- Oncology referral for chemotherapy to downstage the tumor prior to surgery.
- Cardiology referral if necessary & anaesthetic referral.
- Optimize the patient for surgery.
- Special attention should be made on the pre-op nutritional status of the patient.
- Assess nutritional status
 - ✓ Anthropometric measurements
 - Height ,weight ,BMI(<18)
 - Weight for height
 - ✓ Biochemical parameters
 - Serum protein
 - Hb level, MCV
 - Serum ferritin
- Why nutrition is important?
 - ✓ Heal the anastomotic site
 - ✓ Patients are usually elderly

ASA grading (Physical status classification) To assess the fitness for surgery

- Grade I – Normal healthy patient
- Grade II – Mild systemic disease
- Grade III – Severe systemic disease
- Grade IV – Severe systemic disease that is a constant threat to life

- Assess the immune status
 - ✓ Lymphocyte count
- Do lung function tests (spirometry)
 - ✓ Why? – Usually they are elderly people suffering from COPD.
 - ✓ During the surgery have to deflate one lung.
- Enteral nutrition is preferred over parenteral nutrition. Consider feeding jejunostomy if unable to take oral feeds.
- Prior to surgery patient should be well hydrated & should be in electrolyte balance.
- Commence pre-op chest physiotherapy. (incentive spirometry, steam inhalation)
- Arrange for epidural before surgery
- Inform the theater and anesthetist.
- Arrange for an ICU bed before finalizing the theater list

What are the surgical options available for the management?



Methods of accessing the oesophagus

- Target of the surgery is to remove the tumor with a clear resection margin (usually 5-10 cm).

1) Ivor-lewis

- Abdomen is opened first and construction of a gastric tube.
- Followed by a right thoracotomy to excise the tumour and create an oesophagogastric anastomosis.

2) McKeown's

- Used for lesions above carina.
- 3 stage oesophagectomy
 - o Thoracotomy
 - o Laparotomy
 - o Cervical incision
- Anastomosis in the neck.
- Near total oesophagectomy with en-bloc lymphadenectomy is feasible.

3) Orringer's transhiatal blunt oesophagectomy (for lower tumours)

- Laparotomy and cervical incision
- No thoracotomy
- Posterior mediastinum is dissected bluntly using a finger.
- This approach can provide an adequate removal of the tumour and lymph nodes in the lower mediastinum, but it is not possible to remove the nodes in the middle or upper mediastinum.

4) Median sternotomy (Akiyama)

- Curative resection with en-bloc lymphadenectomy

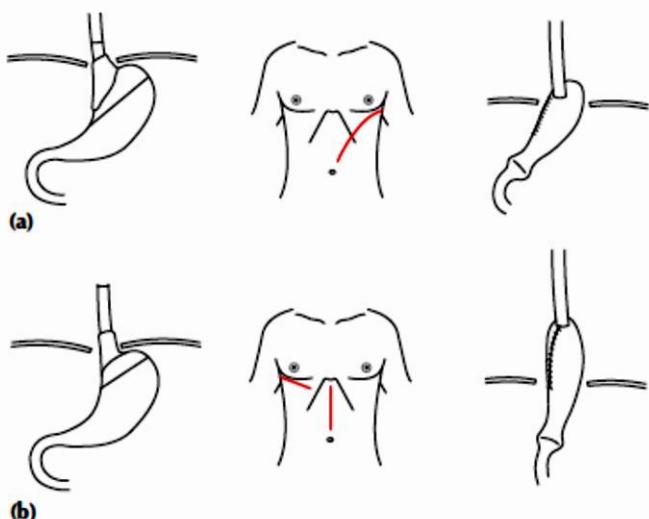


Figure 59.53 The two usual approaches for surgery of the oesophagus are the thoracoabdominal (a), which opens the abdominal and thoracic cavities together, and the two-stage Ivor Lewis approach (b), in which the abdomen is opened first, closed and then the thoracotomy is performed. In the McKeown operation, a third incision in the neck is made to complete the cervical anastomosis.

Principles of post operative management

- Principles as for any major surgery
- ICU Mx is required
- Observation and monitoring of vital parameters
- Monitor drains including IC tube and NG
- Analgesics via epidural catheter
- NBM
- Fluid Mx
- Pulmonary care – Chest physiotherapy
- DVT prophylaxis (LMW heparin 60mg SC daily/Unfractionate heparin 5000IU IV), graduated compression stockings
- Monitor for complications
- **Complications related to anastomosis**
- **Respiratory complications**
- Damage to recurrent laryngeal nerve
- Trachea –bronchial injury
- Pneumonia
- Pleural effusion
- Chylothorax
- **GI care**
 - **NBM**
 - About 2nd day start feeding via feeding jejunostomy tube
 - Confirmation of intact anastomosis by water soluble contrast study on the 10th day. (gastrographin study)
 - After confirming that there's no anastomotic leakage, start on oral feeds. Start with clear fluids(oral sips) and move on to semi solid and solids
 - Small volume frequent meals (6- 8 times per day)
 - Advice on discharge.

8 tubes after oesophagectomy

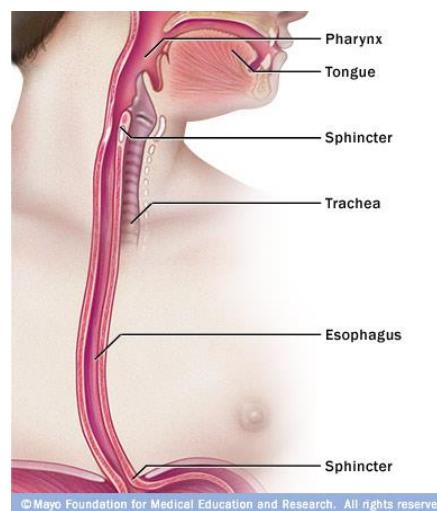
1. 2 IC tubes
2. Laparotomy drain
3. NG tube (To drain the secretions from the anastomotic site)
4. Jejunostomy tube
5. Urinary catheter
6. Epidural catheter
7. IV Cannula

Follow-up

- For histology report (for staging)
- Complications (reflux may be common)
- Medications
- Recurrence.

Anatomy of the oesophagus

- ✓ Esophagus is a tubular structure
- ✓ About 10 inches
- ✓ Continuous above with laryngopharynx opposite 6th cervical vertebra
- ✓ Passes through diaphragm at the level of 10th thoracic vertebra where it joins the stomach in the abdomen.



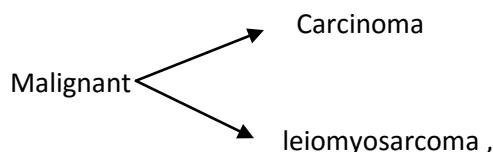
Blood supply of the oesophagus

- Upper 1/3rd of oesophagus
 - Arterial : inferior thyroid artery
 - Venous : inferior thyroid vein
- Middle 1/3rd
 - Descending thoracic aorta
 - Azygous vein
- Lower 1/3rd
 - Left gastric artery
 - Left gastric vein

Areas of narrowing in oesophagus:

- The aortic arch
- The left main bronchus
- The diaphragm

PATHOLOGY



- ✓ Carcinoma of the upper 2/3 of the oesophagus are usually **squamous** in origin while CA of lower 1/3 is usually **adenocarcinoma** in origin.
- ✓ Adenocarcinoma arises in a region of specialised columnar epithelium (SCE) metaplasia in the lower 1/3 of the oesophagus - Barrett's oesophagus.
- ✓ Carcinoma is commonest in the middle 1/3 of oesophagus.

Risk factors

- ✓ Age- >55
- ✓ Sex – male sex is a risk factor for squamous cell CA an gastro-oesophageal junction tumors
- ✓ Smoking and alcohol – squamous cell CA
- ✓ BMI – risk factor for adenocarcinoma
- ✓ Diet – low fiber diet, rich in meat, hot drinks
- ✓ Existing conditions – Barret's oesophagus, achalasia cardia
- ✓ Inheritance – rare

Spread

Local	Lymphatic	Haematogenous(late)
Trachea	Para- oesophageal	Liver
Aorta	Tracheo-bronchial	lung
Pleura	Supraclavicular	
Lungs	Sub diaphragmatic	

Achalasia of the cardia

Definition

Primary esophageal motility disorder characterized by:

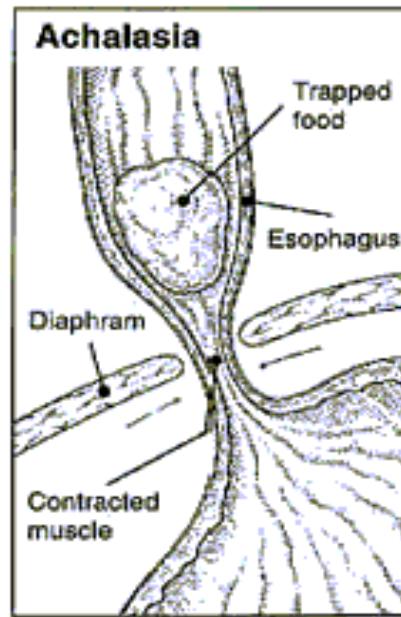
- 1) Absence of peristalsis
- 2) Elevated pressure of the Lower oesophageal sphincter (LES)
- 3) Failure of the LES to relax during swallowing

Causing functional obstruction at the gastro-esophageal junction

- 25-50% report episodes of **retrosternal chest pain**.
- 80-90% experience **spontaneous regurgitation**.
- Some patients may present with signs or symptoms of **pneumonia or pneumonitis**.
- Patients with achalasia are at **increased risk for esophageal cancer**
- Physical examination is non contributory.

Pathology

- Progressive dilatation of upper segment of the oesophagus.
- Wall thickened (hypertrophy)/ thinned (Dilatation)
- Ganglia - absent / varying degrees of degeneration in Ayerbach plexus.
- Ulcero-inflammatory lining.



Investigations

Lab Studies

- Laboratory studies are non contributory.

Imaging Studies

- Barium swallow : Bird's Beak.
- EGD: Normal or dilated esophagus.
- Manometry

The **radiological examination of choice** in the **diagnosis** of achalasia is a **barium swallow study** performed under fluoroscopic guidance.

Diagnosis of achalasia supported by the results of radiologic studies must always be confirmed by

- 1) Performing (EGD) to rule out cancer of the gastro-esophageal junction or fundus, and
- 2) Esophageal manometry. (Reveal elevated LES pressure, abnormal relaxation of sphincter,)



Bird's Beak



Normal Barium Swallow

Management

- The goal of therapy for achalasia is to relieve symptoms by eliminating the outflow resistance caused by the hypertensive and non relaxing LES.
 - 1) Medical Management
 - 2) Surgical Management

Medical management

- 1) **Botulinum toxin:** intra-sphincteric injection
 - Only 30% of patients treated endoscopically still have relief of dysphagia 1 year after treatment.
 - (50% are re-treated in 9 months where it is 10% in balloon dilatation)

2) Pharmacological therapy: relax the smooth muscle of the LES.

- Calcium channel blockers - Nifedipine and verapamil
- Anti-cholinergic agents - Cimetropium bromide
- Nitrates - Isosorbide dinitrate
- Opioids - Loperamide

Advantages of medical treatment

- ✓ primarily in elderly patients who have contraindications to either pneumatic dilatation or surgery or as a temporary measure while other treatments are considered

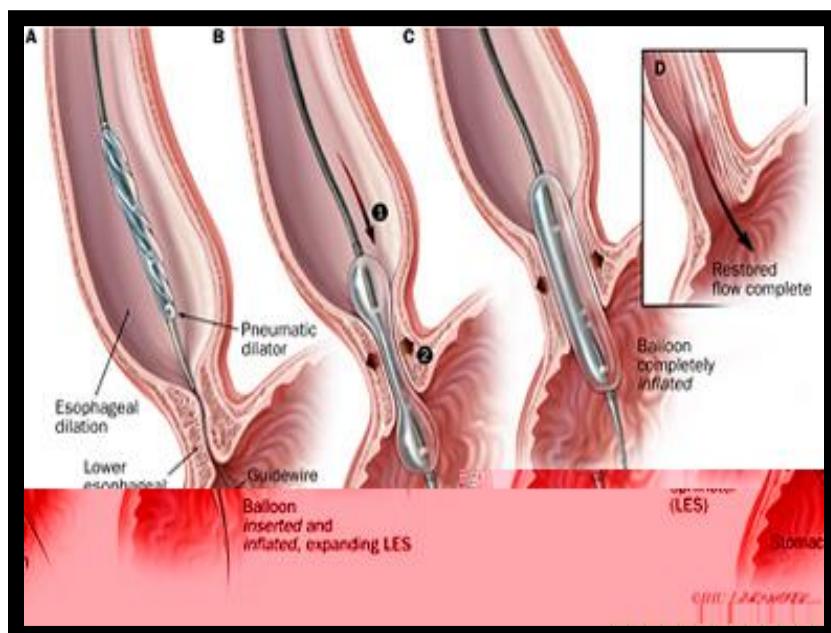
Disadvantages

- ✓ These agents all have demonstrated effectiveness in decreasing LES pressure however, they frequently fail to relieve symptoms, or they are associated with significant adverse effects.

Surgical treatment

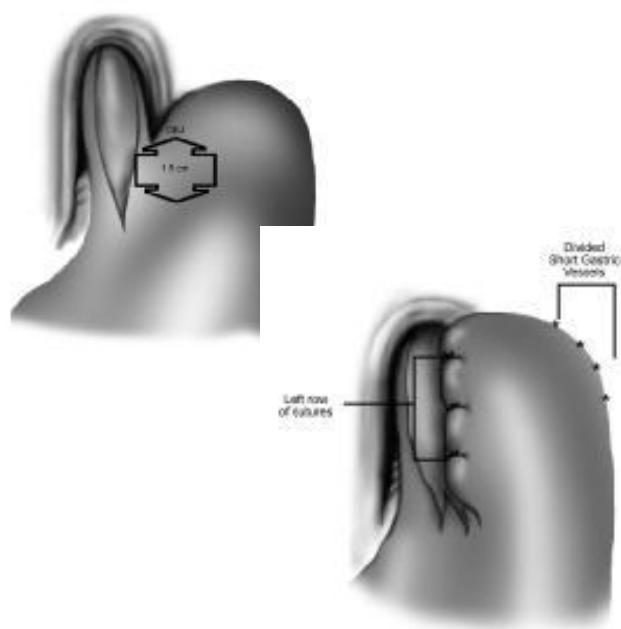
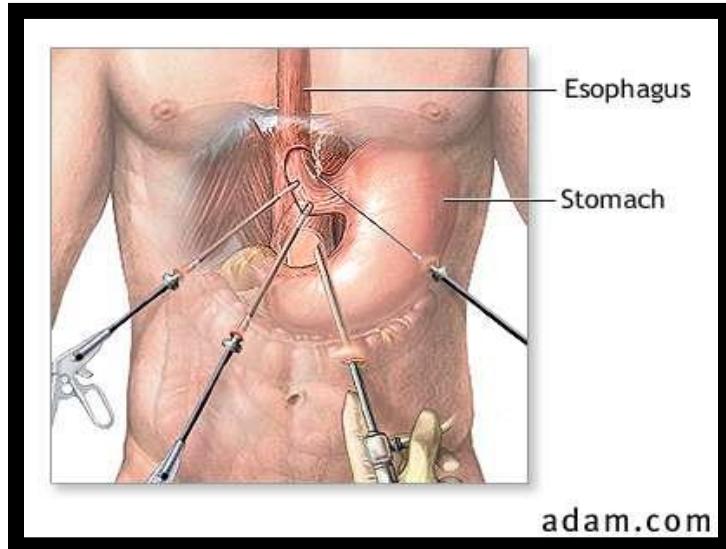
1) Pneumatic dilatation:

- Pneumatic dilatation by a qualified gastroenterologist is the recommended treatment.
- Dilation of LES with a bougie (flexible cylindrical instrument)
- Most commonly performed by using pneumatic balloons.
- The success rate is 70-80%, and the perforation rate is approximately 5%.
- Approximately one half of patients experience recurrent symptoms within 5 years.
- If a perforation occurs, emergency surgery is needed to close the perforation and perform a myotomy.
- As many as 50% of patients may require more than 1 dilatation.
- The incidence of abnormal gastroesophageal reflux after the procedure is approximately 25%.



2) Esophageal (Heller) myotomy (procedure)

- Heller myotomy – cardiomyotomy and dividing the muscle of the lower end of the esophagus and the upper stomach down the mucosa.
- Heller myotomy is the appropriate treatment for patients in whom pneumatic dilatation fails.
- The laparoscopic approach appears to be most appropriate. The results are as durable as those with an open approach.
- Several series of laparoscopic esophageal myotomy with concomitant partial fundoplication have been reported.
- Under general anesthesia, make a controlled division of the muscle fibers (myotomy) of the lower esophagus (5 cm) and proximal stomach (1.5 cm) followed by a partial fundoplication to prevent reflux .
- Patients remain hospitalized for 24-48 hours and return to regular activities in about 2 weeks.
- The operation relieves symptoms in 85-95% of patients, and the incidence of postoperative reflux is 10-15%.
- Patients in whom surgery fails may need a dilatation, a second operation, or removal of the esophagus (ie, oesophagectomy).



Benign oesophageal stricture

Acute management

- Assessment and resuscitation.(ABC)
- Avoid:
 - Emesis
 - Gastric lavage
 - Neutralizing agents
 - NG suction
- Offer supportive care
 - NBM
 - IV fluids
 - Continuous monitoring : Airway, evidence of perforation, peritonitis
 - Pain relief
 - Antibiotics : 3G cephalosporins
 - IV proton pump inhibitors
- Endoscopy by an experienced person to see the extent of the disease.
 - If severe injury : monitor perforation for 1 week + supportive care
 - Less severe : oral feeds in 24-48 hrs
- Psychiatric referral
- Follow up
- Barium swallow in 4 weeks.
- Management stricture
 - Dilatation
 - Stenting
 - resection

Lymph Drainage of the Esophagus

Lymph vessels from the upper third of the esophagus drain into the deep cervical nodes, from the middle third into the superior and posterior mediastinal nodes, and from the lower third into nodes along the left gastric blood vessels and the celiac nodes (Fig. 3-27).

- ❖ In addition to this there is sub mucosal lymphatic plexus in which lymph spread through out.

Upper GI endoscopy (Oesophagogastroduodenoscopy –OGD)

Length

110cm up to the 2nd part of the duodenum

Indications

- *Diagnostic indications*
 - Haematemesis & malaena
 - Dyspepsia in a more than 55yrs old patient
 - Unexplained anaemia
 - Persistant vomiting
 - Dysphagia
 - Odynophagia
 - Surveillance of Barrete's oesophagus
 - Surveillance of gastric ulcers or duodenal ulcers
- *Therapeutic indications*
 - Treatment of bleeding lesions (variceal banding & sclerotherapy)
 - Stricture dilatation
 - Palliative methods(Stent insersion, lazar therapy)

Instruments in endoscopy probe

- Biopsy forceps
- Snares (Use for polypectomy)
- Injecting needles

Procedure

- Consent (Informed written)
- NBM for 6 hours
- Spray 5ml of 10% lignocaine to oropharynx (Xylocaine)
- Make the patient lie on his left with the head resting on pillow. A mouth guard should be used. Usually sedatives are not given(Because patient has to swallow the endoscope)
- Ask the patient to swallow the tube when it comes to throat.
- Have to visualize up to the 2nd part of the duodenum. When returning back at the stomach have to visualize the cardia of the stomach using the "J" maneuver.

Complications

- Aspiration – Asp pneumonia
- Bleeding
- Perforation
- Cardiopulmonary problems

Contraindications

- *Relative contraindications*
 - COPD
 - Recent MI
 - Instability of atlanto-axial joints

CHRONIC EPIGASTRIC PAIN AND LUMP

Disease	Features
Peptic ulcer disease	<p>Burning or aching type pain in the epigastrium Nausea, vomiting <ul style="list-style-type: none"> • <u>Gastric ulcers</u> pain↑with meals (within 30 min)↓ with vomiting No diurnal variation Episodic Radiates to back-posterior in penetrating ulcer <ul style="list-style-type: none"> • Duodenal ulcer Burning type pain↓with meals, nocturnal pain(pain small hours) Wakes the pt in early morning has a wt gain due to↑take of meals </p>
Gastric carcinoma	<p>Local symptoms Epigastric pain and discomfort Anorexia Anaemic symptoms - lethargy, palpitation, faintishness Asthenia (Loss of strength) Early satiety, bloating, abdominal distension</p> <p>Obstructive features Gastro-esophageal junction → Dysphagia Antral/pylorus involvement → Projectile non-bilious vomiting, abdominal fullness Radiates to back if –pancreas involved Melena/hematemesis-perforation or h'rrhage</p> <p>Symptoms due to secondaries Liver - ascites, RHC pain, +/-jaundice Lung – Cough, haemoptysis Brain – sinister headache Bone – Intractable backache at night, pathological fractures</p> <p>Para neoplastic syndromes Jaundice -haemolytic anemia Membranous GN features-heamaturia, HT, edema DIC</p> <p>Aetiology Cigarette smoking Heavy alcohol consumption Salty foods (Baken, sausages, ham) Smoked food High cholesterol diet Hereditary non polyposis colorectal CA (Family history of colorectal CA)</p>

EPGASTRIC PAIN

DDs

1. Peptic ulcer disease
2. Gastric CA
3. Acute pancreatitis
4. Chronic pancreatitis
5. Oesophagitis

4 As in gastric CA

1. Anorexia
2. Anemia
3. Asthenia
4. Blood Group A

Acute pancreatitis	<p>Sudden onset severe constant epigastric pain radiating to the back (Sharp, stabbing pain) Relieved in leaning forward Worse with fatty meals Early and profuse vomiting Frequent retching Fever, jaundice</p> <p>Causes for A. pancreatitis = GET SMASH'D</p> <ul style="list-style-type: none"> G-allstones E-thanol T-rauma S-teroids M-umps A-utoimmune (Poly Arteritis Nodosa) S-corion bites H-yperlipidemia D-rugs (azathioprine) D-diuretics 	<p>DDs</p> <ol style="list-style-type: none"> 1. Pancreatic CA 2. Pancreatic pseudocyst 3. Gastric CA 4. Peri ampullary CA 5. Aortic aneurism 6. Epigastric hernia
Chronic pancreatitis	<ul style="list-style-type: none"> • Asymptomatic/recurrent abdominal pain • Radiating to back • ↓ by bending forward • Abdominal pain worse with food or alcohol • Steatorrhoea, weight loss • Features of fat soluble vitamin malabsorption <p>A-bitot spots, night blindness D-fractures E- K-bleeding problems</p> <ul style="list-style-type: none"> • Symptoms of DM • +/- Obstructive jaundice <p>Aetiology</p> <p>Heavy alcohol consumption Gall stones obstructing pancreatic duct Hyperlipidaemia Hypercalcaemia Hereditary Idiopathic</p>	
Oesophagitis	<p>Night pain, radiate to back(inter-scapular region) Nocturnal onset dysphagia pain worsen in bending forward Associated GORD</p>	

If pain ass with epigastric lump

PC- Epigastric pain and lump (duration)

HPC

- a. Describe the pain
- b. Site
- c. Onset-sudden/gradual
- d. Character-colicky/constant
- e. Radiation
- f. Severity
- g. Exacerbation and relieving factors
- h. Timing
- i. Associated symptoms
- j. 1st episode or previous history
- k. Chronological order of the events

PMHx DM - *pancreatic CA*

Chronic peptic ulcer disease- *Gastric CA*

H.Pylori infection

Pernicious anemia - *gastric CA (P)*

Mumps, hypercholesterolemia, hyperparathyroidism- *A.pancreatitis*

PSHx

Gastric resection-gastric CA
Sx in opened abdominal cavity
ERCP
Trauma to abdomen

]
pancreatitis

DHx

NSAIDS- *peptic ulcer disease*
Steroids, Na valproate, OCP – *Acute pancreatitis*

FHx

Colonic CA-gastric Ca

SHx

Smoking-
Alcohol-
Stress-
]
Peptic ulcer, esophagitis, A.pancreatitis, C.pancreatitis

Salty food, smoked food, high cholesterol diet-*gastric CA*
Low socioeconomic status
Blood group A
Attitude towards the disease, pain

Pancreatic CA

- Epigastric dull pain-constant, aching type
- Painless progressive jaundice and features of obstructive jaundice - periampullary/pancreatic head
- Radiating to back
- Severe LOW with anorexia
- Recent onset DM
- Steatorrhoea, epigastric bloating
- Abdominal distension

Pancreatic pseudo cyst

- Epigastric pain & fullness
- Pain relieved by bending forward
- Nausea, vomiting
- Infected cyst – rigors, sweating, severe pain
- Exacerbate symptoms with meals
- History of acute pancreatitis [usually 2 weeks ago]

Pyloric stenosis

- Episodic projectile vomiting in large amount.
- Containing undigested or partly digested 1-2 days old food, non-bilious.
- Constipation-due to dehydration.
- Weakness due to electrolyte disturbance.

Aortic aneurism

- Usually asymptomatic,
- Back pain, abdominal bloating and pulsatile swelling

Epigastric hernia

- Lump get prominent with coughing and straining

Examination

General

Emaciated, pale, jaundice, hydration

B/L ankle edema,

Fx of Fe deficiency anemia-angular stomatitis, glossitis

Fx of CLD - Palmer erythema, deputryrens contracture, gynecomastia, parotid swelling (b/l)

Supra clavicular lymphadenopathy (left) – Virchow's node (Troisier's sign)

Abdomen

Distension, scars - upper midline scar

Grey turner's sign
Cullen's sign } Acute pancreatitis

Guarding, epigastric tenderness

Umbilical deposits - Sister joseph's nodules - Gastric CA

Hepatomegaly

Ascites

DRE



LUMP

Epigastric lump- Intra abdominal lump/not

Inspection

Pulsatile

Visible peristalsis

Palpation

Tenderness

Consistency

Succision splash

Discussion

Gastric CA

I ncidence
A ge of onset
S ex of predilection
G eopgraphy
Predisposing factors
M acroscopy
M icroscopy
S pread (route of)
C linical features
P rognosis

I-5th commonest of cancer deaths in SL
A-50-70yrs
S- M=F
G-Japan, Finland, Portugal, Scotland
P-low vit C, blood group A, post gastrectomy, pernicious anemia, hypogammaglobulinemia
M-ulcerative, polypoidal, linitis plastica(diffusely infiltrative)
M-adenocarcinoma
S- Direct, lymphatic, blood stream & transperitoneal
C-anorexia, wt loss, projectile vomit, succession splash, C.LN, sister joseph
Prognosis-5 year -5%
Stage 1 - 70%
Stage 4 - 3%

Gastric CA Pathology

Macroscopy

4 macroscopic appearances

1. Malignant Ulcer with raised everted edges
2. Polypoidal tumour – May results in gastric outlet obstruction
3. Colloid tumour – Massive gelatinous growth
4. Linitis Plastica – Leather bottle appearance due to submucosal infiltration of tumour with marked fibrous reaction

Spread of gastric CA

- **Local:** direct spread to oesophagus, duodenum, greater & lesser omentum, liver, pancreas, spleen and diaphragm.
- **Transcoelomic:** small tumour nodules in the peritoneum, ovaries- Krukenberg tumour.
- **Lymphatic:** paragastric, omental, mesenteric.
- **Blood:** liver, lung, bone & brain.

Classification (Laurens')

INTESTINAL

- Polypoid:fugating
- Glands, well differen.
- Limited mucin seen in gland lumen.
- Growth expansile
- Metaplasia ++
- M>F (2:1) ~ 55yrs.

DIFFUSE

- Ulcerative:infiltrate
- Signet cells, poorly differen.
- Extensive mucin, around glands stroma (colloid ca.)
- Metaplasia less seen
- M=F ~48yrs.

Ix-

- 1) UGIE+ biopsy –key investigation - CONFIRMS THE DIAGNOSIS
Biopsies taken multiple at least 8 sites
Follow up with biopsy report
- 2) Blood investigations - FBC, BU/SE, LFT
- 3) Endoscopic ultrasound-to assess the intramural tumor penetration/LN spread
- 4) CT To asses nodal spread & metastases
- 5) USS To asses nodal spread & metastases
- 6) CXR – Canon ball deposits
- 7) Laparoscopy-identify peritoneal seeding's
- 8) Peritoneal lavage-identify tumor cells
- 9) Barium meal & follow through (Xray)

Staging – Birmingham staging

Clinico pathological

Stage 1- disease confined to muscularis propria

Stage 2- muscularis and serosal involvement

Stage 3- gastric and nodal involvement

Stage 4a- residual diseases

Stage 4b- metastatic diseases

Table 60.5 International Union Against Cancer (UICC) staging of gastric cancer

T1	Tumour involves lamina propria
T2	Tumour invades muscularis or subserosa
T3	Tumour involves serosa
T4	Tumour invades adjacent organs
N0	No lymph nodes
N1	Metastasis in 1–6 regional nodes
N2	Metastasis in 7–15 regional nodes
N3	Metastasis in more than 15 regional nodes
M0	No distant metastasis
M1	Distant metastasis (this includes peritoneum and distant lymph nodes)

Complications of gastric Carcinoma

- 1) Perforation of and peritonitis
- 2) Gastric outlet obstruction due to pyloric stenosis
- 3) Torrential hemorrhage when a major blood vessel eroded

Tumor Markers & Oncogenes

CA 19-9, CA 72-4

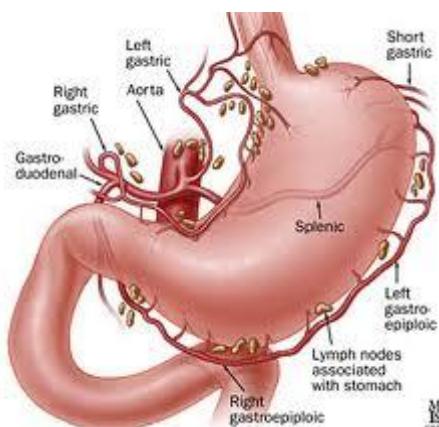
CEA

Intracellular Estradiol(hCG)

C-erbB-2

Purpose of tumor markers

1. Screen healthy or high risk population for the presence of tumor
2. Make specific diagnosis
3. Determine the prognosis
4. Monitor the course, remission effectiveness of the treatment



TNM staging

T1-Mucosal & Sub mucosal

T2-Subserosa

T3-Serosa

T4-Contiguous Structures

N0

N1-Regional

N2-Perivascular

N3-Para Aortic, Retro pancreatic, Hepatoduodenal, Mesenteric

M0

M1-metastasis

Management

- Surgery is the only prospective of cure
- **Antral tumours** may be suitable for a **partial gastrectomy** usually **with Polya reconstruction**
- **Other tumours** will need a **total gastrectomy** with **oesophagojejunal anastomosis and Roux-en-Y biliary diversion**
- A tumour is considered resectable if confined to stomach or N1 or N2 nodes involved
- Nodes less than 3 cm from tumour = N1 nodes
- Nodes greater than 3 cm from tumour = N2 nodes
- If tumour and N1 nodes resected = D1 gastrectomy
- If tumour and N2 nodes resected = D2 gastrectomy
- Evidence to support the use of D2 gastrectomy is incomplete
- D2 gastrectomy associated with increased post-operative mortality
- May be associated with improved long-term survival
- Even in patients with incurable disease surgery may palliate symptoms
- Results from adjuvant chemotherapy post-surgery are disappointing
- Chemo radiotherapy may reduce relapse and improve survival

D₁-stomach, perigastric LN, omenta
D₂-stomach, omental bursa, extensive
LN dissections

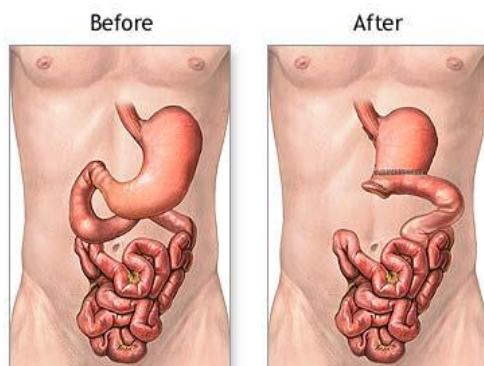
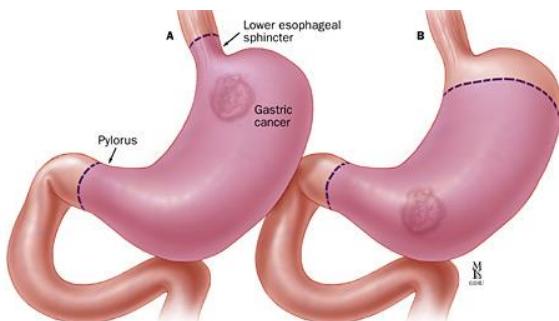
Palliative procedures

Gastro jejunostomy

Exclusion gastrectomy

Feeding jejunostomy

Laser resection



ADAM.

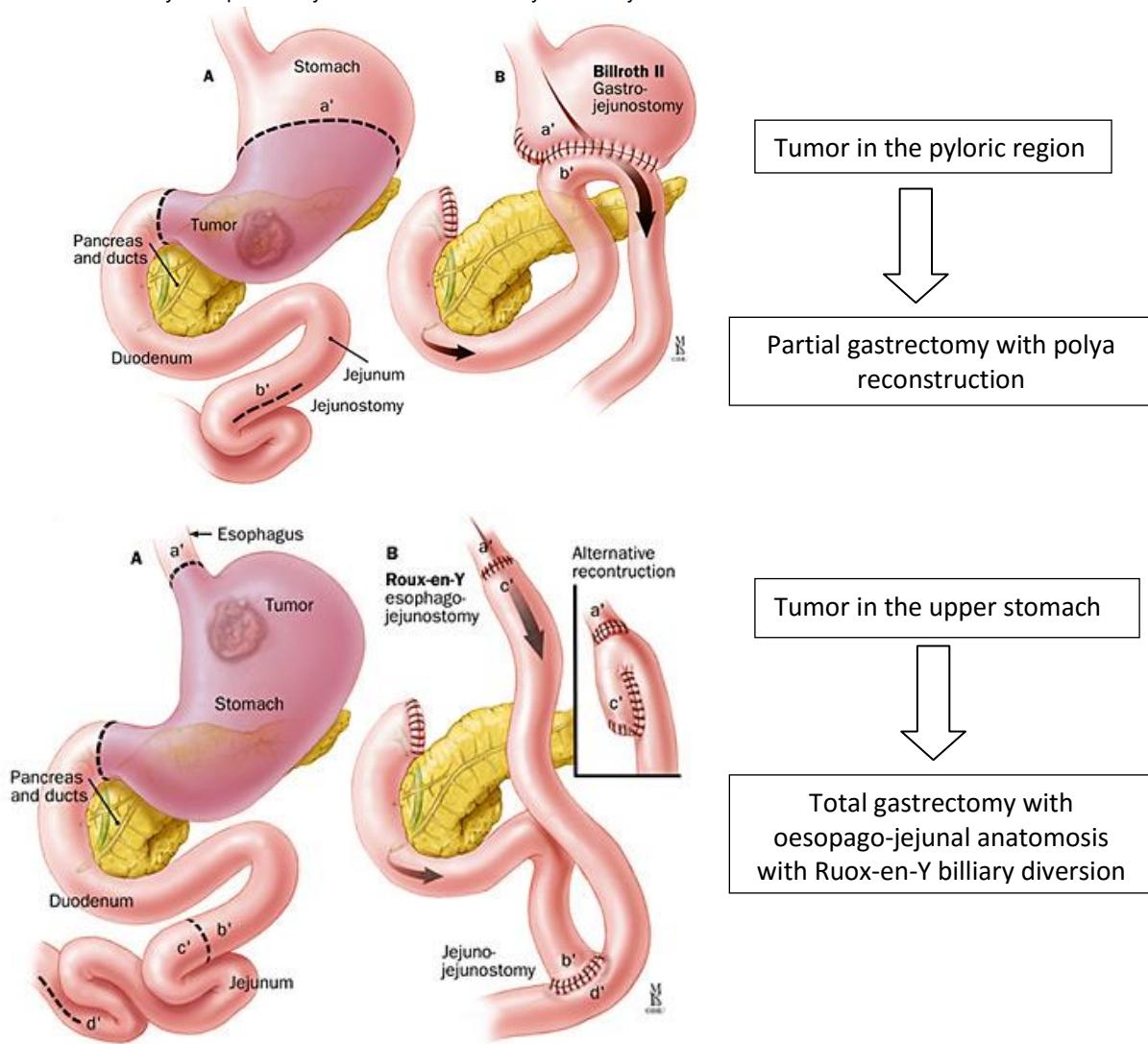
Radiation Therapy

Not effective

Chemotherapy

5 Fluorouracil, Adriamycin, Mitomycin

5 Fluorouracil, Epirubicin, Cisplatin



Pre op work up before gastrectomy

- Explain the condition and Tx options to pt and relatives
- Oncology referral
- Nutritional assessment
 - Anthropometric assessment
 - Biochemical assessment
- Nutritional supplement
 - High protein diet, high calorie diet
 - Fe, vit B₁₂,
- Anaesthetic referral
- Hb level-if anaemic blood Transfusion 2 days prior to Sx to achieve 10g/dl
- Pre-op Ix – FBC, BU/SE, S.creatinine, S.protein, PT/INR, ECG, CXR, 2D echo, Lung function tests
- Grouping & DT 2 units of blood
- Chest physiotherapy
- Steam inhalation
- Breathing exercise(Insentive spirometer)
- If abnormality in PT/INR correct with Vit K & FFP
- Overnight fasting
- Graduated compression stockings or DVT prophylaxis

Post-op

- Monitoring – PR, RR, UOP, Bleeding, temperature
- Pain relief – epidural analgesia or SC morphine 5mg
- IV fluids
- Metronidazole 500mg 8/H & cefuroxime 750mg 8/H
- Steam inhalation
- Chest physiotherapy
- Jejunostomy feeding from 2nd post-op day
- Gastrographin study from 10th post-op day to see the anastomotic healing
- If no leaking → start oral fluids little by little

Advices after gastrectomy

- Take small frequent meals with large amount of water
- Meals should be consistent with high protein, high in vitamins
- Nutritional supplements should be taken

Complications after gastrectomy

Immediate

General immediate

Mnemonic: **PROBS**

P Primary hemorrhage/Pain

R Reactive hemorrhage

O Oliguria – acute urinary retention

S Shock/Sepsis

early

Mnemonic: **ABCDE**

A Analgesia- or Anesthetic-related nausea + vomiting

B Breakdown of wound or anastomotic dehiscence –

Gastric fistula, duodenal fistula, peritonitis, intra-abdominal abscess, seroma , heamatoma

C Confusion – acute

D DVT leading possibly to PE

E Elevated temperature – pyrexia

late

Mnemonic: **RIB**

R Recurrence of malignancy

I Incisional hernia

B Bowel obstruction

- Other- Small (no stomach syndrome)

- early satiety and Inability to enjoy a large meals

- Early morning bilious vomiting

- After polygastrectomy

- Bile and pancreatic juice are get collected in the afferent loop.

- It get distended leading to epigastric pain vomiting relieves the pain

- Pt learn to induce vomiting to relieve early morning pain

- Dumping syndrome

Early

- Hypovolemic, tachycardia and dizziness -

Due to loss of controlled release of food in to the intestine from the stomach leading to increased intraluminal osmolality. Then vascular fluid diffuses to intestinal lumen causing hypovolemia.

Late

- Hypoglycemia-dizziness, sweating, palpitations -

This occurs 1-2 hrs after meals. Rapid absorption of glucose in to the body causes hyperglycaemia. Resultant hyperinsulinaemia. That leads to reactive hypoglycemia. It responds to oral glucose.

- Intestinal hurry
 - Diarrhea and malnutrition
Borborygmi, abdominal cramps cause poor absorption of food.
- Nutritional deficiencies
 - Iron deficiency anemia
Lack of HCl and bypass of duodenum (the main site of Fe absorption)
 - Vitamin B₁₂ deficiency
Lack of intrinsic factor
 - Fat malabsorption
 - Fat soluble vitamin deficiency
Mainly vit D-osteomalacia
 - Protein maldigestion and malabsorption –wasting
 - Immunodeficiency-reactivation of TB

Peptic ulcer disease

Investigations

- Fiber optic endoscopy
 - To visualize oesophagus, and duodenum
 - Take biopsy
- H. pylori detection
 - Non-invasive-urea breath test (inject radio labeled urea)
Stool tests (fecal occult blood)
Serum IgG Ab
 - Invasive-endoscopy and biopsy
 - Serological testing-Ab

Treatment

- Medical-
 - H. pylori eradication therapy
 - PPI+ metronidazole+ amoxicillin
 - PPI+ metronidazole+ clarithromycin
 - PPI+ metronidazole+ bismuth+ tetracycline
 - Acid reduction-
 - Mg tricilicate
 - Al₂(OH)₃
 - other agents
 - misoprostol(synthetic PG)
- Non pharmacological
 - Avoid alcohol
 - Smoking
 - Stress
 - Aspirin/NSAID

- surgery
 - bilroth 1 gastrectomy
 - bilroth 2 gastrectomy
 - duodenostomy and pyloroplasty
 - polya gastrectomy
 - truncal vagotomy and drainage, highly selective vagotomy
- } Gastric ulcers
} Duodenal ulcers

Perforated Peptic Ulcer Management

M>females

Hx of peptic ulcers

Hx of NSAID, steroids

Mortality rate-5-10%

- Perforated appendix
- Acute cholecystitis
- Acute pancreatitis
- MI
- Acute/sub-acute intestinal obstruction

Clinical Fx

Acute severe pain

Pain ↑ with movement

Patient lies still

Nausea, vomiting occasionally-if so haematemesis and melena

Examination

Pt in pain.

Cold peripheries, severe sweating-features of shock

Shallow breathing

Abdomen-silent, rigid, occasional bowel sounds

Liver dullness-↓In ½ of the cases

DRE-pelvic tenderness

Delayed cases

Generalized peritonitis,

Paralytic ileus

Abdomen distended

Effortless vomiting

Ix-

Erect chest X ray.

70% shows air under diaphragm

CT-to exclude pancreatitis

Treatment-

Nil by mouth

Put NG,IV cannula

IV fluids

Antibiotics

IV H₂ receptor blocker or PPI

Surgical treatment

Suturing an omental plug to seal the perforation

Management of gastric CA in our ward setting

Op note

- Total gastrectomy
 - Under GA
 - Midline incision
 - Peritoneal cavity opened into,
 - Liver seen
 - Large tumor involving body and pylorus
 - Gastrocolic ligament divided from transverse colon
 - Gastrohepatic ligament divided
 - Lymph neck? Dissected at pylorus and body of stomach
 - Oesophagus transected at GOJ
 - Oesophagojejunal anastomosis done
 - Feeding jejunostomy
 - Washed out the region with warm water
 - Post op plan-
 - NBM
 - IVF 100cc/hr
 - IV AB
 - ceftazidime1g(forum)
 - flagyl 500mg
 - IV KCl 60g daily(40ml/24hrs)
 - If pt admit to ICU
 - Prop up-O₂ via facemask
 - Insert CVP line and monitor CVP
 - Monitor PR,RR,BP-1/4-2hrs,1/2-4hrs,1-24 hours
 - NBM
 - IVF crystalloid 50ml/hr
 - Morphine IV infusion
 - SE,FBC,blood transfusions if needed
 - Daily Mx
 - Jejunal feed,5% dextrose→70cc/hr
 - s/c enoxaparin 40mg daily?
 - Chest physiotherapy
 - Steam inhalation
 - Nebulise with salbutamol 6hrly
 - When to start oral feeding???
 - After about 10 days if no problem with wound healing
 - Pt c/o discharge of the wound site at 10th day what do u do?
 - Stop oral
 - Start jejunal feed again
- You shouldn't remove the jejunal feeding tube after starting oral b'coz of the above complication

HAEMATURIA

HISTORY

Presenting complaint - Haematuria

History of presenting complaint

- Whether that is truly haematuria
- If it is haematuria site of bleeding
- Progression of bleeding
- History of similar episodes
- Presence of urinary symptoms
- Aetiology for haematuria

1. Enumerate the presenting complaint

- Onset
- Duration
- Progression
- History of similar episodes
- Colour of urine & passage of clots or fleshy pieces

2. Is it truly haematuria?

- Causes for red colored urine other than haematuria
 - Foods- beet root/black berries/rhubarb
 - Drugs – Rifampicin/Sulfasalazine/phenytoin/phenothiazines/metronidazole
- Causes for tea colour urine mimicking haematuria
 - Myoglobinuria (leptospirosis)
 - Haemoglobinuria
 - Recent surgery with general anaesthesia - Propofol

3. Site of bleeding

- Blood at start of voiding & after that urine become clear – **Urethral causes**
- Blood diffusely present throughout void - **Bladder & above**
- Blood only at the end of voiding - **Prostate gland and bladder neck**

4. Whether haematuria associated with pain

Differential diagnosis for painful haematuria

1. Pyelonephritis

- High fever
- Toxic features
- Loin pain

2. UTI

- Dysuria
- Frequency
- Urgency
- Fever

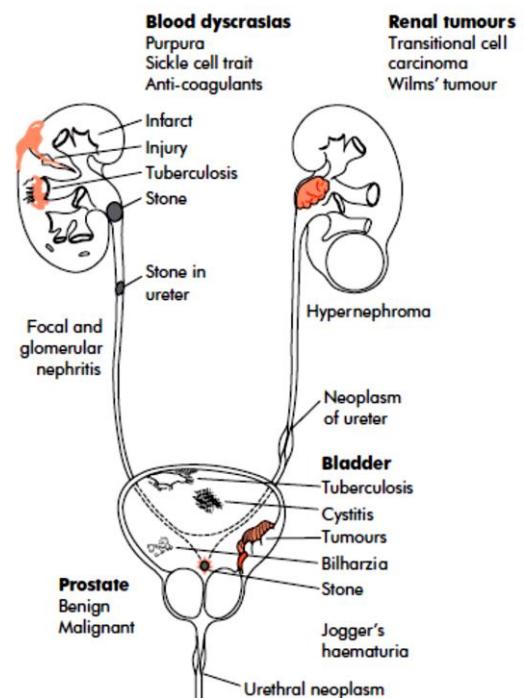


Figure 70.1 The more common causes of haematuria.

3. Urerteric colic (passage of calculi or a tumour particle or blood clots from a tumour)
 - Colicky abdominal pain
 - Radiate from loin to groin – Upper ureter
 - Radiate to the tip of the penis – Lower ureter
 - Autonomic symptoms – Associated nausea, vomiting, sweating
4. Obstruction
 - Lower urinary tract symptoms (LUTS)
 - Voiding symptoms (experienced during the voiding phase)
 - Hesitancy, slow stream, intermittency
 - Storage symptoms (experienced during the storage phase)
 - Increased frequency, nocturia, urgency, urge incontinence
 - Post micturition symptoms (experienced immediately after micturition)
 - Feeling of incomplete emptying, post micturition dribble
 - Prostatic CA
 - Symptoms
 - LUTS – rapidly progressive
 - Anorexia
 - LOW
 - Anaemia – Lethargy, dizziness, palpitation, undue exertional dyspnoea
 - Features of metastasis
 - Back pain
 - Limping
 - Scalp lump
 - Aetiology
 - High cholesterol diet
 - Family history
 - Complications
 - Urinary stasis → Pyelonephritis, Recurrent UTI
 - Acute urinary retention
 - Hydronephrosis → Renal failure
5. Infarction – Polycythaemia, hypertension, hypercholesterolemia, smoking

Painless haematuria differential diagnosis

1. Glomerulonephritis
 - **PSGN** : Recent history of sore throat or skin infection
 - Hypertension (headache , visual blurring, fits)
 - Oedema
 - Oliguria (urine output)
 - **HSP** : Joint pain, skin rash, abdominal pain
 - **SLE** : Arthralgia, skin rash, fever
 - **IgA nephropathy** : Recent Hx of URTI or tonsillitis
 - **Infective endocarditis** : Congenital heart defects, Rheumatic fever
2. Acute tubular nephritis
3. Interstitial nephritis

4. Tumours - **Renal cell carcinoma**

- Symptoms
 - Intermittent Haematuria
 - Dragging loin ache
 - Dull loin pain
 - Mass in the flank
 - PUO
 - Malaise, anorexia, weight loss (30%)
- Hypertension
- 5% polycythaemia
- Anaemic features
- Hypercalcaemia (Due to ↑ PTH secretion)

5. Bladder CA

- Intermittent attacks of haematuria
- Recurrent attacks of UTI (necrotic center of the bladder tumor is a good culture medium for bacterial growth. The patient experiences recurrent attacks of dysuria frequency and nocturia).
- Strangury- intense desire to urinate which results in a passage of a clot and sense of incomplete evacuation
- Acute urine retention
- BOO symptoms.

6. Bleeding disorders

- Past history of easy bruising, petechial haemorrhage, Family history

7. Tuberculosis

- Low grade fever, weight loss, night sweating, haematuria
- Increased frequency, mild lumbar pain
- Previous Hx of chronic cough +/- haemoptysis
- Contact History of TB

8. Trauma

- Trauma to the back / loin

PMHx :

- Previous episodes of renal stones
- Chronic abdominal pain (enlargement of cyst)
- Bleeding disorders

PSHx :

- Catheterization
- Urological surgeries
- Pelvic irradiation
- Fragmentation of stones

Menstrual History: LMP & regularity of periods

Drug Hx : drugs causing haematuria

- Cyclophosphamide
- Aspirin , Clopidogrel
- Warfarin /heparin

Family history

- Polycystic kidney disease/medullary cystic disease
- Familial haematuria/deafness- (Alport Xn) → family Hx in maternal side
- FHx of similar presentation

Social history

- Smoking
- Work place heavy metal (mercury/ copper)/ Dye exposure – Bladder CA
- Foreign travel (schistosomiasis →Africa)

EXAMINATION

General examination

- Eyes- conjunctivitis, sub conjunctival haemorrhage
- Pallor/ plethoric (in RCC), jaundice (leptospirosis)
- Scalp – alopecia
- Enlarged left supra-clavicular lymph node
- swollen joints in SLE, HSP
- Rashes :
 - Petechial rash specially in lower limbs around joints and buttocks- HSP
 - Malar rash, discoid rash (SLE)
 - Bruises/echimosis (bleeding tendency)
- Mouth – oral ulcers/ poor dental hygiene
- Hands –
 - Nail fold infarcts(SLE)
 - Peripheral stigmata of infective endocarditis

Cardio vascular system

- BP
- Features of heart failure: Tachycardia, gallop rhythm
- New onset murmur (IE)

Respiratory system

- Tracheal deviation due to pulmonary fibrosis in TB)
- B/L crepitations (heart failure due to hypertension)

Abdominal examination

- Renal angle tenderness
- Renal masses B/L or unilateral
- Ballotable kidney
- Pelvic masses
- Bladder dullness (urine retention)

Genitalia

- Urethra and vagina in females
- Penile urethra for lumps / external meatus
- Left sided varicocele- renal cell carcinoma

Rectal examination (prostatic lumps)

- Hard nodular(irregular) prostate with fixed rectal mucosa
- Obliteration of median groove

CNS

- GCS

DIGITAL RECTAL EXAMINATION

The examination should be done with consent, with the patient in lateral position.

It is necessary to inspect for haemorrhoids, anal fissures and then feel the prostate and the seminal vesicles if possible.

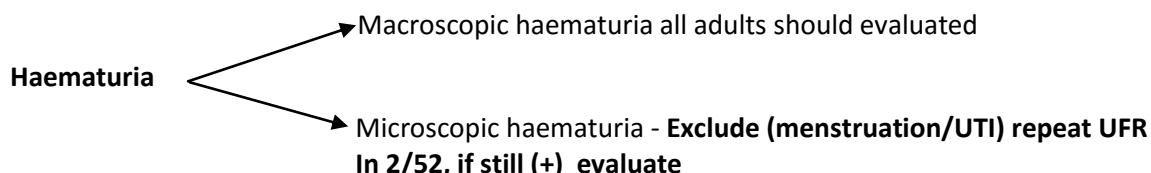
He should be reassured that it is not painful, asked to open the mouth and breath deeply to get him to relax, and a well lubricated gloved finger inserted with the nail of the finger parallel to the bed.

Once it is in the anus, gently move the finger to feel the prostate. It has two lobes and a groove. The consistency is like the tip of the nose. If it is unusually tender and the consistency is like the cheek, you maybe dealing with Prostatitis.

If it is hard as the bridge of the nose and irregular, there may be cancer. Obliteration of the median groove is also suspicious. The seminal vesicles are above the prostate and not usually palpable. If they are, and continuous with the prostate growth, it is probably a prostatic cancer.

These patients will need a referral to surgeon/urological surgeon

DISCUSSION



Investigations

1. Confirm haematuria – UFR
 - Haematuria + pus cells – infection
 - Haematuria
 - granular casts → tubular disease
 - red cell casts → glomerular bleeding
 - Dysmorphic red cells – To find-out Glomerular cause
2. To find out the aetiology
 - Urine culture and ABST – exclude urinary tract infection
 - FBC
 - WBC ↑: in infection
 - Hb ↓ : gross haematuria , malignancy
 - Hb ↑: Polycythaemia due to RCC
 - platelets reduced in (dengue, bleeding disorders)
 - CRP elevated in infection
 - ESR: ↑ in Malignancy or TB
 - 24 hr urinary protein measurement
 - S. creatinine and electrolytes : to assess the renal function

- ECG – hypertensive changes
- PSA in males>50 – CA prostate suspected
- CXR- pulmonary oedema(LVF)
 - TB
 - Cannon ball deposits in RCC
 - Pleural effusions
 - Mediastinal lymph nodes
- Coagulation profile – exclude coagulopathies
 - PT/INR

3. Imaging studies

X ray KUB

- Show 90% of calculi, 10% calculi radiolucent (uric acid stones)
- Osteosclerotic deposits (in CA prostate)
- Osteolytic lesions (in RCC)

USS KUB

- Kidney changes, size, surface, cortico-medullary demarcation
 - Detect SOL in kidneys, bladder – renal tumors >2cm
 - Detect the size of prostate
 - Any residual urine
 - Any hydronephrosis
- } If Haematuria is associated with LUTS
(Ultrasonography has been found to be more sensitive than intravenous urography in detecting renal cell carcinoma but less so in detecting urothelial transitional cell carcinoma)

IVU

- Visualize entire urinary tract (function and anatomy)
- Calculi/pelvic & ureteric, urothelial tumors

(Intravenous urography is superior to CT in detecting transitional cell carcinoma involving the kidney or ureter but has limited application in the evaluation of the bladder and urethra.)

- Patients undergoing intravenous urography are exposed to contrast media that is potentially nephrotoxic, especially to patients with renal insufficiency.

Preparation

- High fluid intake to maintain a UOP of at least 2L for 24 hours
- Take bisacodyl 3 tablets nocte for 3 days before the procedure. If no satisfactory bowel opening take bisacodyl suppository in the morning of the day of investigation
- Low fibre diet during 3 days before the procedure and avoid green leaves & fruits
- Overnight fasting (From 10pm)
- Should have to admit on the day before the procedure with other medical records & investigations
- If any history of drug & food allergy or eczema take prednisolone 2 tablets tds for 3 days before the surgery
- If previous generalized contrast medium reaction → give IV hydrocortisone 200mg 30mins prior to administration of contrast medium
- Should have to come to the procedure with empty bladder
- If patient has the following, serum creatinine (if high hydrate well & give NAC) should be provided (< 6 months old)
 - Renal disease/surgery
 - Proteinuria
 - DM (And if on metformin)
 - HT
 - Gout
 - Neurotoxic Rx use

CT scan

- Determines nature and effects of renal masses
- CECT : for Renal tumors
- Non contrast spiral CT more sensitive than IVU to detect calculi
(Microscopic hematuria associated with renal colic is best evaluated with CT in light of its high sensitivity for identifying renal calculi. Unenhanced helical CT is more accurate for evaluating patients with renal colic compared with ultrasonography, intravenous urography, or plain X-ray)

Cystoscopy

- Any patient >40 yrs with haematuria is assumed to have CA bladder until proven otherwise – investigation is mandatory
- If >40 yrs & X-Ray KUB shows stone – cystoscopy still mandatory
- May be →flexible (no GA/Spinal needed) or rigid
- Must be done for –
 - all patients >40 yrs
 - patients <40 yrs with negative imaging or imaging shows bladder/upper UT tumour(Cystoscopy is the only reliable method of detecting transitional cell carcinoma of the bladder and the urethra. The primary disadvantages of cystoscopy are patient discomfort with this invasive procedure and its limited ability to detect carcinoma in situ of the bladder.)

Renal biopsy

Glomerular disease

Biopsy is not done for RCC due to needle tract seeding

+ Practical box 11.3

Transcutaneous renal biopsy

Before biopsy

- 1 A coagulation screen is performed. It must be normal.
- 2 The serum is grouped and saved for crossmatching.
- 3 The patient is given a full explanation of what is involved.

During biopsy

- 1 The patient lies prone with a hard pillow under the abdomen.
- 2 The kidney is localized by ultrasound.
- 3 Local anaesthetic is injected along the biopsy track.
- 4 The patient holds a breath when the biopsy is performed.

After biopsy

- 1 A pressure dressing is applied to the biopsy site and the patient rests in bed for 24 hours.
- 2 The fluid intake is maximized to prevent clot colic.
- 3 The pulse and blood pressure are checked regularly.
- 4 The patient is advised to avoid heavy lifting or gardening for 2 weeks.

Other specific investigations

1. ASOT/throat swab/discharge from ear/ skin lesion culture – PSGN
2. C3 levels-AGN
3. ANCA- vasculitis
4. ANA/dsDNA –SLE
5. Renal biopsy-light microscopy/electron microscopy/immunofluorescence
6. PSA male >50 yrs if suspected prostatic CA
7. Anti GBM Ab- Good pastures Xn
8. Leptospiral Ab
9. Blood picture – DIC/CRF
10. Coagulation studies- APTT/PT

DISCUSSION

Microscopic Haematuria definition

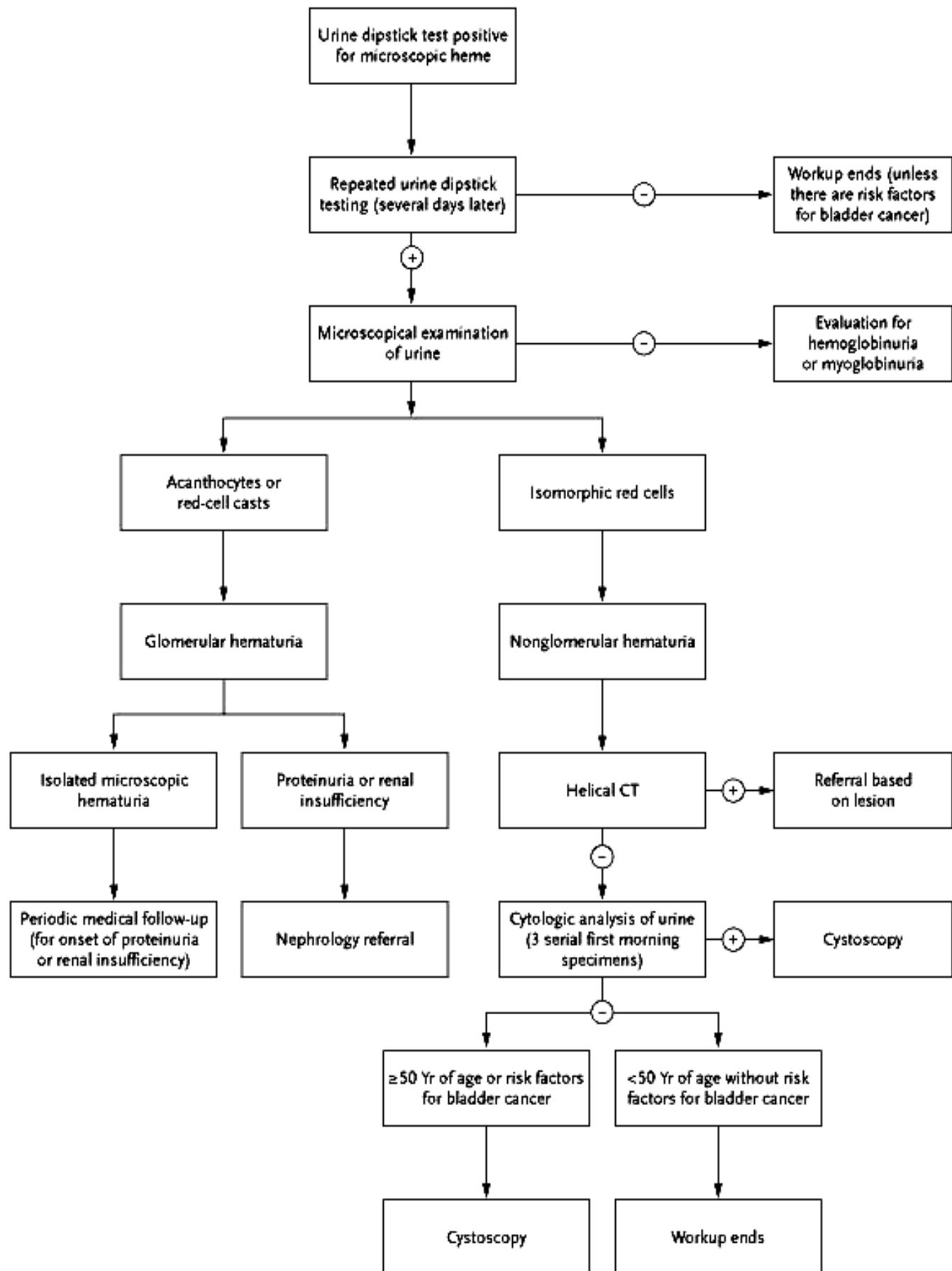
The American Urological Association (AUA) defines clinically significant microscopic hematuria as **three or more red blood cells per high-power field on microscopic evaluation** of urinary sediment from two of three properly collected urinalysis specimens.

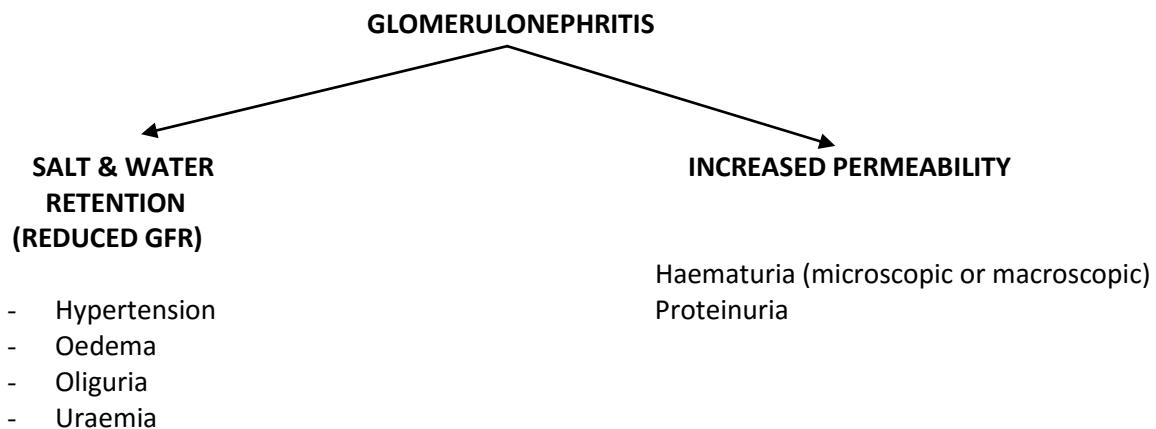
General management of haematuria

- Fluid restriction
- Low salt diet
- Monitor fluid input output
- BP tds and protein
- Daily body weight
- Education
- Daily serum electrolytes
- Diet, if hyperkalaemia restrict K+ containing foods
- Strict bed rest in high BP patients
- Likelihood of tumor within 2-5 yrs after completely negative haematuria evaluation is up to 3% → must be followed up
- Urinalysis & BP at 6, 12, 24 & 36 months
- Need immediate urological evaluation if –
 - Gross haematuria
 - Persistent storage type LUTS without UTI

When to refer to a nephrology

- Significant proteinuria (>1g/24 hrs or >0.5g/24 hrs if persistent or increasing)
- Renal impairment on biochemistry (s.creatinine elevated)
- Evidence of glomerular bleeding (dysmorphic RBC or RBC casts)
- Other evidence of nephrological disease, hypertension or diabetes





GLOMERULAR CAUSES

1. Glomerular nephritis
 - IgA nephropathy
 - HSP
 - Lupus nephritis
(membrano proliferative/mesangeal proliferative)
 - Post streptococcal GN
 - Good pastures
 - Post infectious GN
2. Haemolytic uraemic syndrome
3. Thin basement membrane nephropathy(benign familial haematuria)
4. Alport syndrome

IgA Nephropathy (commonest form worldwide)	Previous episodes of asymptomatic haematuria Most following upper respiratory tract or gastrointestinal viral infection
HSP	Syndrome characterized by petechial purpuric skin rash GI symptoms such as abdominal cramps (persistent or paroxysmal) Joint pain Haematuria
Lupus nephritis	SLE features, symmetrical small joint arthritis Malar rash, photosensitivity rash, oral ulcers
PSGN	Streptococcal skin infection (cellulitis) Throat infection Otitis media 1-3 weeks prior to onset of haematuria
Infections associated GN	IE (IV drug users, peripheral stigmata of infective endocarditis, chest pain), new murmurs
Haemolytic uraemic Xn	Previous febrile illness with acute gastroenteritis Followed by mucosal bleeding and haematuria and Oliguria Or similar clinical picture following taking following medications (OCP, ciclosporins, tacrolimus, heparin)

TUBULO – INTERSTITIAL CAUSES

1. Acute tubular necrosis
2. Familial – hereditary nephritis
 - Polycystic disease
3. Infection – TB, pyelonephritis
4. Interstitial nephritis – Drug induced(penicillins, cephalosporins)
5. Renal cell carcinoma
6. Vascular diseases – AV malformation/ malignant HT/renal artery embolism or Thrombosis/renal venous thrombosis

HSP

- Characteristic skin rash/abdominal colic/joint pain/ GN
- 30-70% haematuria and /proteinuria
- Renal disease usually mild but nephritic Xn and ARF can occur
- Pathology – focal segmental proliferative GN
- Sometimes with mesangeal hypercellularity
- Severe cases epithelial crescents +
- Immunoglobulin deposits mainly Ig M
- Management supportive therapy
- But crescentic GN aggressive immunosuppression

Lupus Nephritis

- Kidney is most commonly involved organ
- 50% develop clinically evident renal disease
- Among these 25% reach ESRF within 10 yrs
- Can develop acute nephritic disease – HT, Haematuria/Nephrotic Xn/ARF , CRF/uraemia fluid overload
- Classification of lupus nephritis

Post streptococcal GN

Caused by Lancefield group A, beta haemolytic streptococcus of a nephritogenic type

Pathology – diffuse acute inflammation of glomerulus without necrosis

But occasional crescent formation

With neutrophils and deposition of immunoglobulin IgG + compliments

Specific management

- Frusemide oral/IV – 40-80mg/day
- Anti hypertensives-nifedipine (beta blockers and captopril avoided)
- Penicillin IV/oral (if allergy Erythromycin)
- If patient develop ARF dialysis

Haemolytic uraemic syndrome

- ✓ Characterized by **intravenous haemolysis** (micro-angiopathic haemolysis)
- ✓ **Thrombocytopenia** and **ARF** due to thrombosis of small arteries and arterioles
- ✓ Similar picture like DIC but **clotting profile normal in HUS**
- ✓ Etiology
 - Diarrhea associated HUS (bloody diarrhea)
 - Xn follows a febrile illness (particularly gastroenteritis)
 - E-coli (shiga toxin) verocytotoxin inhibits protein synthesis and initiate vascular Endothelial damage
 - this can also associated with pregnancy/SLE/Scleroderma/malignant HTN/metastatic CA/HIV infection
 - Drugs as OCP/tacrolimus/therapeutic agents
- Mx- supportive care-maintenance fluid and electrolyte balance
 - Anti hypertensive medication
 - Nutritional support
- Antibiotics and antimotility agents for diarrhea increase risk of HUS and its Complications

Renal TB

- The kidney is usually infected by hematogenous spread of bacilli from a focus of infection in the lungs and/or bowel.
- Clinically, renal tuberculosis usually presents unilaterally
- The healing process results in fibrous tissue and calcium salts being deposited, producing the classic calcified lesion. The occurrence of renal calcification is common in TB and may require surgical excision
- Hypertension may occur as a complication of severe unilateral TB and reduced renal blood flow.
- Presentation as Especially in urinary tuberculosis, voiding problems and chronic urgency non-responding to antibacterial drug regimens, are indicative of genitourinary TB. In men, chronic epididymitis is the typical manifestation of tuberculosis of the male genital tract, mostly combined with scrotal fistulas.
- Other symptoms that sometimes occur include suprapubic pain, hematuria, frequency, and nocturia. Renal colic is uncommon.
- Diagnosis-A microbiologic diagnosis of tuberculosis is usually made by isolation of the causative organism from urine or biopsy material.
- Treatment as for pulmonary TB and renal Ultrasonography and IVU 2-3 month after initiating treatment (ureteric strictures develop 1st in healing phase)

Acute Pyelonephritis

- Acute pyelonephritis is an infection of the upper urinary tract, specifically the renal parenchyma and renal pelvis.
- Acute pyelonephritis is considered uncomplicated if the infection is caused by a typical pathogen in an immune-competent patient who has normal urinary tract anatomy and renal function.
- Misdiagnosis can lead to sepsis, renal abscesses, and chronic pyelonephritis that may cause secondary hypertension and renal failure.

Predisposing factors

- Either an ascending infection or haematogenous spread.
- Organisms: *E. coli* 60%, *Proteus* 20%, *S. faecalis* 10%, *Klebsiella* 5%.
- Female (short urethra)
- Renal stones
- Bladder catheter
- Chronic liver disease
- Structural abnormality of renal tract
- Pregnancy
- Diabetes mellitus
- Intravenous drug abuse
- Infective endocarditis
- Gyne Hx : Fibroids, Cystocoele

Pathogenesis

- Most renal parenchymal infections occur secondary to bacterial ascent through the urethra and urinary bladder.
- In men, prostatitis and prostatic hypertrophy causing urethral obstruction predispose to bacteriuria
- Hematogenous acute pyelonephritis occurs most often in debilitated, chronically ill patients and those receiving immunosuppressive therapy.
- In more than 80% of cases of acute pyelonephritis, the etiologic agent is *Escherichia coli*
- Other etiologic causes include *Proteus*, *Klebsiella*, *Enterococcus faecalis*, *Staphylococcus epidermidis*.

- In elderly patients, *E. coli* is a less common (60%) cause of acute pyelonephritis. The increased use of catheters and instruments among these patients predisposes them to infections with other gram-negative organisms such as *Proteus*, *Klebsiella*, *Serratia*, or *Pseudomonas*.
- Patients who have diabetes mellitus tend to have infections caused by *Klebsiella*, *Enterobacter*, *Clostridium*, or *Candida*. They also are at an increased risk of developing emphysematous pyelonephritis and papillary necrosis, leading to shock and renal failure.

Presentation

- Loin pain, fever, and rigors.
- Nausea, vomiting, anorexia, malaise, confusion, or weakness.
- Lower urinary tract symptoms (frequency, dysuria, haematuria)
- A preceding history of intermittent loin pain may imply intermittent obstruction with pyonephrosis. Renal parenchymal abscesses are seen with IV drug use, endocarditis, or skin infections.
- Ex : Febrile, Renal angle tenderness, Cystocoele, Urethral strictures, Prostate hypertrophy(DRE), BP

Investigations

- **UFR:** shows blood and protein. White cells, bacteria, WBC casts may be seen on microscopy.
- **Urine Culture & ABST**
 - Urinalysis and urine culture confirm the diagnosis of acute pyelonephritis (> 5 WBCs/HPF) (*identification of $> 10^5$ colony-forming units per ml.*)
 - Urine specimens generally are obtained by a midstream clean-catch technique,
 - Pyuria is present in almost all patients with acute pyelonephritis and can be detected rapidly with the leukocyte esterase test or the nitrite test.
- **U&E, FBC (anaemia, leukocytosis), FBS(Exclude DM)**
- **Blood cultures**
- **Abdominal XRay :** Stones
- **USS-Abd :** Perinephric abscess and renal size, Hydronephrosis
- **CT** (if surgery is planned)
- **IVU, MCUG(in children), Cystoscopy**

Management

- Stabilize the patient : IV fluids \pm inotropes
- IV antibiotics
 - Gentamycin
 - Ciprofloxacin
 - Ceftazidime/Ceftriaxone
 - Coamoxyclav

} 7 – 14 days
- Fluid balance: maintain high fluid intake (e.g. 3L/24h)
- Monitor fluid balance and urine output
- Antiemetics (Metoclopramide 10mg IV) & Antipyretics
- Analgesia: NSAID if renal function is normal.(e.g. diclofenac sodium 75mg im). Alternatively try IM pethidine 50-75mg 3hrly.
- Urgent Urology Referral (Pyonephrosis, renal/perinephric abscess)
- Investigate for any underlying cause: IVU, DMSA, and DTPA scans will determine anatomy, extent of renal damage, and remaining function.

Indications for Hospitalization in Patients with Acute Pyelonephritis

Absolute indications

Persistent vomiting
Progression of uncomplicated UTI
Suspected sepsis
Uncertain diagnosis
Urinary tract obstruction

Relative indications

Age > 60 year
Anatomic urinary tract abnormality
Immunocompromised (diabetes mellitus, cancer, sickle cell disease, organ transplant)
Inadequate access to follow-up
Frailty, poor social support

Complications

- Septicaemia
- Perinephric abscess
- Pyonephrosis
- ARF
- Acute haemorrhagic necrotising papillitis

Renal tract calculi

Aetiology- Metabolic

Eg:- Idiopathic Hypercalciuria

Hormonal imbalances

Eg:- Hyperparathyroidism

Environmental

Diet (increase intake of fluoride in water)

Inadequate water intake

Infection

Eg:- urea splitting Proteus (favour calcium phosphate stone formation)

Pseudomonas

Urinary stasis

Benign prostatic hyperplasia

Urethral stricture

External meatal stenosis

Ureteric colic

- comes from a hollow viscous
- Sudden onset
- Sharp
- Suddenly increase in intensity to a plateau
- Intensity decreases
- Cycle repeats
- Not completely pain free
Between attacks

Composition-

- Calcium oxalate – commonest (60%)
- Uric acid – (radio-lucent)-rare
- Triple stones / Struvite stones – Magnesium, ammonium and calcium
- Cysteine -rare

Oxalate stones – Radio-opaque

- commonest 60%
- Radio opaque
- <1cm usually-irregular , spiky surface
- Produce symptoms at early stage-U.colic
- Idiopathic hypercalciuria
- hypercalcaemic states - hyperparathyroidism, sarcoidosis

Uric acid stones (5%) - Radiolucent

- Associated with hyperuricaemia
- Rapid cell turnover states and gout -due to uric acid increase
- Associated with hyperuricosuria
- Acidic pH
- Idiopathic

Cysteine stones (1%)

Defect in transport of amino acid - cysteine

Triple stones – “struvite stones”- 33%

- usually bilateral and large
- Magnesium, ammonium, calcium phosphate stones
- Radio opaque
- Following infections by urea splitting bacteria (**Proteus mirabilis** and **staphylococcus**)
- leading to **alkaline urine**
- Formation of staghorn calculi
- Smooth surface
- Asymptomatic
- Attain in large dimension
- Acquire the shape of the renal pelvis major and minor calyceal system, hence the shape of a stag horn
- Often results CRF-B/L, large

Ureteric colic

Clinical features

- Sudden onset
- Episodic : Waxing & weining
- Unbearable pain
- Colicky type
- Radiating
 - Upper ureter – loin to groin
 - Lower ureter – loin to testis / tip of the penis
- Associated nausea, vomiting, sweating
- +/- haematuria, Dysuria, frequency
- Fever with chills & rigors
- Restless
- Past history of similar episodes
- Area of living : North central province
- Some occupations - ↑ dehydration

Examination-

- Tachycardia
- Palpitation
- Urethral stones will be felt along the ventral surface of the shaft of the penis

Investigations-

- Urine culture & ABST - UTI
- UFR – Red cells, pus cells
- FBC – Infection, Hb
- X- ray KUB
 - Bowel preparation with bisacodyl 4 tabs nocte, overnight fasting
 - 90% of the Ureteric stones are radio-opaque
 - Cystein and urate stones are translucent(10%)
- USS KUB - Hydro ureter, hydronephrosis, urinary stones, cortico-medullary pattern of the kidney
- IVU (Intravenous urogram) – Hydroureter, Radio-lucent stone identification, to locate the obstruction, diagnosis of PUJ obstruction with stones.
Before PCNL to get knowledge about the exact calyceal anatomy.
- Non excreting kidney in IVU with a stone may need direct contrast studies and Isotope studies for its structural and functional details

Best investigation for stones –Non contrast spiral CT

Stones in distal ureter

- < 4mm 90% pass spontaneously
- 4-6mm 50% ”
- > 6mm 20% ”

Stones in the proximal ureter are less likely to pass

Management

Acute –

- Adequate analgesics
 - Eg:- NSAID's or Opiates- Diclofenac sodium 50mg suppository, Pethidine 75mg IM
 - Pain relief
 - Inhibit prostaglandin mediated renal vasodilatation → ↓ GFR
- Antiemetics – Metoclopramide IV 10mg tds / Promethazine 25mg IM
- IV fluids – to prevent dehydration
 - IV N. saline (1/3 is retained in the intravascular compartment). Can combine with 5% dextrose
- Anti spasmodic : Hyoscine butylbromide (antimuscarinic) – Oral 20mg qds / IM or slow IV 20mg

Post acute

- Increase fluid intake is essential - UOP >2L/24hr
- Encourage citric acid containing food - lemon, lime, oranges
- Avoidance of oxalate containing food - spinach, tomato
- Optimization of calcium intake
- Diclofinac sodium 50mg bd for pain

Specific Management

- Empirical antibiotics
 - IV cefuroxime 750mg 8 hrly
 - IV ciprofloxacin 400mg bd
 - Can give IV gentamycin in young people. It may cause nephrotoxicity & ototoxicity in extremities of life. If renal angle tenderness is present → Have to give 3rd generation cephalosporins
 - Change the antibiotics according to the ABST
- Treatment
 - Urgent decompression by percutaneous nephrostomy or cystoscopic JJ stenting.
 - To relieve acute obstruction due to calculi specially when associated with infection.
 - **Watchful waiting**
 - Small ureteric calculi with minimal hydronephrosis
 - Uncomplicated small calculi in minor calyces.
 - Large stag-horn calculi should not be managed conservatively.
 - **Cystoscopic, uretero-renoscopic (URS) intra-corporeal lithotripsy(ICL)**
 - Mainstay of bladder and ureteric stone management.
 - Rigid and semi-rigid scopes → bladder and upper ureter
 - Flexible URS → reach calyceal system
 - Stones can either be removed by
 - ✓ Domia basket – Extraction using a cystoscope for lower Ureteric stones
 - ✓ Disintegration(ICL)- pneumatic , ultrasonic, electro-hydrolic, laser
 - Complications
 - ✓ Bladder/ureteric perforation
 - ✓ Ureteric avulsion by baskets leading to extravasation of urine.

- **Extra-corporeal shock wave lithotripsy – ESWL**
 - Non-invasive method of stone disintegration in renal, upper/mid ureteric calculi.
 - High frequency sound waves generated by various methods (Eg: under water spark gap) are focused to the stone without harming the soft tissue.
 - Sound shock waves are coupled to the body by a warm bath, pool.
 - Relatively painless but may need sedation under midazolam.
 - Contraindications to ESWL
 - ✓ Too small (< 5mm)
 - ✓ Too large (> 2cm/staghorn)
 - ✓ Too hard (cysteine, oxalate stones)
 - ✓ Too soft (matrix, multiple small calculi)
 - ✓ Pregnancy, bleeding disorder, acute infection
 - Relative contraindications- cardiac pacemakers, spinal deformities,
 - Urinary infection and obstruction should be treated prior to ESWL.
 - Lower ureteric /bladder calculi respond poorly to ESWL.
 - Complication
 - ✓ Lower ureteric obstruction due to multiple stone fragments.
 - ✓ Renal haematoma
 - ✓ Parenchymal injury leading to renal hypertension
 - It is advisable to limit the maximum number of ESWL sessions to 3 and avoid in chronic renal disease.
- **Percutaneous nephrolithotomy(PCNL)**
 - For removal of larger renal and upper ureteric calculi.
 - Under image guidance
 - Done after disintegration with intra-corporeal lithotripsy.
 - Complications – renal trauma → perinephric haematoma, severe haematuria, stricture formation
 - Contraindications – clotting disorders, gross spinal deformity, obesity
- **Open conventional sx-**
 - **Nephro/ pyo/urolithotomy** has a limited place in very large staghorn calculi, associated with obstruction which need open correction.
 - Removal of bladder stone – Vesicolithotomy
 - ✓ For large stones → Pfannenstiel incision
 - ✓ Need a 10 day Foley catheter drainage for healing of bladder

Kidney stone

- acute onset agonizing loin pain
- Stag horn calculi are asymptomatic-may have vague loin pain

Bladder stones

- Most are symptomatic
- Suprapubic pain
- Pain induced by movements
- Pain refers to tip of the penis
- Intermittent attacks of retention of urine or intermittent flow

Urethral stones

- Patient developed acute retention of urine, narrowest part of the urethra is external urethral meatus

Renal cell carcinoma

Malignant-

- RCC
- Nephroblastoma
- Transitional cell carcinoma of the renal pelvis
- Sarcomas of the Kidney
- Renal lymphoma
- Metastatic tumours Eg:-Lung, Breast, GI, Melanoma

Benign

- Angiomyolipoma
- Oncocytoma
- Renal cortical Adenoma

RCC can present as:

- Incidental finding
- The classical triad of pain, mass, haematuria
- Metastatic symptoms such as dyspnoea, cough , haemoptysis, convulsions, bone pain
- Paraneoplastic syndrome – anaemia, polycythaemia, PUO, hypercalcaemia, HT

Investigations-

- Confirmed by USS (tumor and abdominal metastases)
- CT scanning for staging
 - CT scanning - identify the renal lesion & involvement of the renal vein or inferior vena cava.
 - MRI better than CT for tumour staging.
- Chest X ray for lung metastases
- Biopsy not done due to needle tract seeding
- Urine cytology for malignant cells is of no value.
- Liver biochemistry may be abnormal

Management

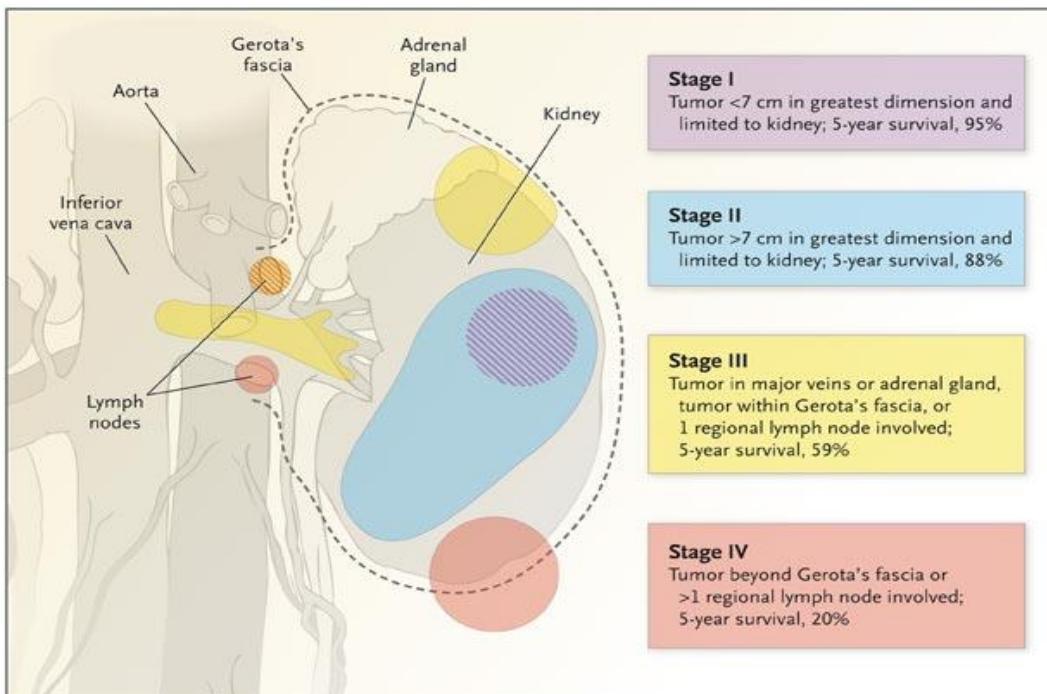
- Radical Nephrectomy
- Nephron sparing surgery

CHEMO AND RADIO INSENSETIVE

Adenocarcinoma of the kidney

- Often presents with haematuria
- May be associated with pyrexia of unknown origin, polycythaemia, hypercalcaemia, anaemia and other paraneoplastic symptoms
- Metastasises via the bloodstream to bone, liver and lung (cannonball secondaries)
- Renal vein extension may embolise to the pulmonary circulation during nephrectomy

- ✚ A **nephrectomy** is performed unless bilateral tumours are present or the contralateral kidney functions poorly in which case conservative surgery such as partial nephrectomy may be indicated. If metastases are present, nephrectomy may still be warranted since regression of metastases has been reported after removal of the main tumour mass.



Robsen's staging

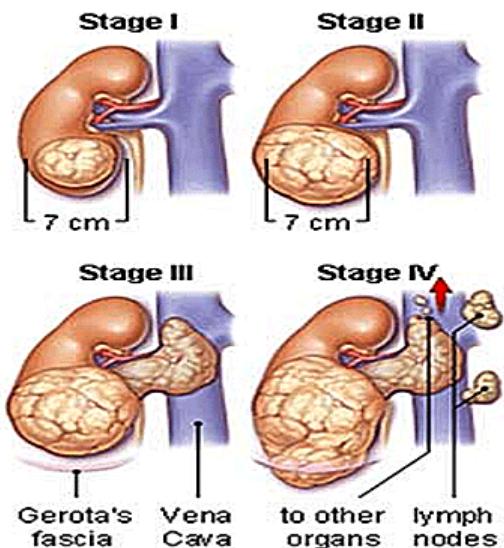
- Stage I – Confined to the kidney
- Stage II – Infiltration of peri-nephric fat but remains limited to Gerota's fascia
- Stage III - Infiltration beyond the fascia of Gerota(renal vein, LN)
- Stage IV - Direct invasion to adjacent organs and Metastasis

Rx - Radical Nephrectomy

Partial nephrectomy (when contralateral kidney is weak)

Pre-op preparation

- Informed written consent
- Hb, PCV, FBS
- Recheck X-Ray KUB, USS IVU are ready
- Renal functions to asses contralateral kidney
- ECG, CXR
- If necessary 2D echocardiogram
- Grouping & DT – 2 units of blood
- Steam inhalation & chest physiotherapy
- Optimize the nutritional condition of the patient. Eg : Calories, Proteins, Iron, Folic acid, Vitamin C & B complex, zinc
- Informed the anaesthetist & theatre
- Pre-operative shower
- Shave the loin and groin
- 6 hours fasting
- Anxiolytic – Diazepam 5mg, or Lorazepam 2mg, or Midazolam 7.5mg
- Preoperative antibiotics-
 - ✓ Clean surgeries such as radical nephrectomy, varicocelectomy,
 - ✓ Contaminated Surgeries such as pyeloplasty and all stone operations need antibiotics.
- Mark the side of operation (kidney/ureter) with indelible ink away from the incision site.



Procedure

- A midline laparotomy
- Trans-abdominal approach
- The renal vein should be ligated first to prevent dissemination. Soon the renal artery has to be ligated to prevent congestion & bleeding
- Kidney with the adrenal gland, perinephric fat & fascia of Gerota with EN-BLOC LYMPHADINECTOMY
- The specimen should be sent for histopathological evaluation

Post-op care

- Monitoring – PR, RR, BP, temperature, any bleeding, any bleeding, drains
- Prop-up
- Steam inhalation
- Chest physiotherapy
- Sips on demand
- Pethidine 75 mg IM SOS
- IV fluids until the oral intake is acquired
- Early mobilization

Complications of nephrectomy

- Local
 - Bleeding – primary & reactionary
 - Haematoma
 - Wound infection
 - Burst abdomen
 - Incisional hernia
 - Keloids
- General
 - Aspiration pneumonitis
 - DVT
 - Pulmonary embolism
 - Myocardial infarction

Refer to an oncologist

- Oncologist colour
- Radiotherapy
- Chemotherapy
- Immunotherapy

Follow up-

- 5 – 10 Years
 - With CXR
 - USS of the Abdomen
- } 6 monthly

Renal cell carcinoma

- Renal cell carcinomas (RCC) arise from proximal tubular epithelium.
- They are the most common renal tumour in adults.
- They rarely present before the age of 40 years, the average age of presentation being 55 years

Bladder CA

Types

- Transitional Cell Carcinoma is commonest (90%)
- Squamus cell CA
- Adeno CA

Risk Factors

- Age (6th decade)
- Smoking (4X greater risk)
- Chemical exposure – Aniline dye, Aromatic amines, Rubber, Tar, Printing, Cable, Heavy metal industry)
- Cyclophosphamide therapy

Tumour spread

1. Local
 - a. Prostate
 - b. Pelvic wall
 - c. Vagina
 - d. Rectum
2. Distal
 - a. Nodes
 - b. Lung
 - c. Liver
 - d. Bone

Symptoms of bladder cancer

- Abdominal pain
 - Blood in the urine
 - Bone pain or tenderness if the cancer spreads to the bone
 - Fatigue
 - Painful urination
 - Urinary frequency
 - Urinary urgency
 - Urine incontinence
 - Weight loss
- } Recurrent attacks of UTI

Examination

- Supra-pubic tenderness(due to cystitis) or dullness(due to retention)
- Ballotable kidneys – Bilateral or unilateral hydronephrosis

Investigations

- UFR
 - Urine culture & ABST
 - Bladder tumour antigen
 - Nuclear matrix protein (NMP22)
 - Telomerase level
 - USS abdomen (See the bladder growth, Renal pathology, Lymph node enlargement)
 - CT/MRI
 - IVU (to detect synchronous tumours in upper tracts)
 - Cystoscopy – Used for **resection & biopsy** of tumour with **examination under anesthesia for TNM staging**. It **decides the treatment & prognosis**
- } Local invasion & metastases

Pre-op preparation for cystoscopy

- Informed written consent
- Hb, PCV, FBS, BU/SE
- ECG if >40Yrs
- Chest X-ray
- Overnight fasting
- Pre-anaesthetic medication

Grading (According to histology)

1. Grade I – Well differentiate
2. Grade II – Moderately differentiate
3. Grade III – Poorly differentiate (Anaplastic)

Staging

1. Stage 0a (Ta, N0, M0) : **Mucosa**

- Non-invasive papillary carcinoma
- **Grown toward the hollow center of the bladder** but has not grown into the connective tissue or muscle of the bladder wall
- N0 & M0

2. Stage 0is (Tis, N0, M0) : **Mucosa**

- Flat, non-invasive carcinoma, also known as flat carcinoma in situ (CIS)
- **Growing in the inner lining layer of the bladder only.** It has neither grown inward toward the hollow part of the bladder nor has it invaded the connective tissue or muscle of the bladder wall
- N0 & M0

3. Stage I (T1, N0, M0) : **Mucosa**

- **Grown into the layer of connective tissue under the lining layer of the bladder** but has not reached the layer of muscle in the bladder wall
- N0 & M0

4. Stage II (T2a or T2b, N0, M0) : **Muscle**

- **Grown into the thick muscle layer of the bladder wall**, but it has not passed completely through the muscle to reach the layer of fatty tissue that surrounds the bladder
- N0 & M0

5. Stage III (T3a, T3b, or T4a, N0, M0) : **Local spread**

- **Grown completely through the bladder into the layer of fatty tissue that surrounds the bladder (T3)**
- **It may have spread into the prostate, uterus, or vagina (T4a)**
- It is not growing into the pelvic or abdominal wall
- N0 & M0

6. Stage IV : **Spread to pelvic or abdominal wall +/- Nodes +/- Distant metastases**

One of the following applies:

- T4b, N0, M0: The cancer has **grown through the bladder wall and into the pelvic or abdominal wall** (T4b). The cancer has not spread to lymph nodes or to distant sites.
OR
- Any T, N1 to N3, M0: The cancer has spread to nearby lymph nodes (N1-N3) but not to distant sites.
OR
- Any T, any N, M1: The cancer has spread to distant sites such as bones, liver, or lungs (M1).

Treatment

Stage 0a, 0is, I (TNM – Ta, T1)

- Loco regional endoscopic resection
- Intravesical chemotherapy or immunotherapy (with BCG) – Prevents tumour recurrence

Stage II (T2) & Stage III (T3)

- Radical cystectomy with urinary diversion (Uretero sigmoidostomy, ileal conduit neo bladder formation)

OR

- Radical external beam radiotherapy (Later by palliative cystectomy if it fails)

👉 Cystectomy : Risk of major surgery

👉 Radiation : Haemorrhagic(radiation) cystitis, enteritis & pelvis fibrosis

Stage III (T4 – locally advanced) and Stage IV (Metastatic disease)

- Local radiotherapy and systemic chemotherapy with MVAC (Methotrexate, Vinblastin, Adriomycin, Cis/carboplatin)
- And palliative cystectomy in case of severe bleeding or intractable pain

Follow up

- For rest of life with regular urine cytology, check cystoscopy & annual IVU especially when the bladder is intact

Bladder irrigation

It is commonly used (though not essential) after any operation that causes blood accumulate within the bladder or the prostatic cavity. (Objective is to remove blood before it becomes a clot)

It is useful following both transurethral & open surgery.

Irrigating fluid should be (Glycine 1.5%)

- Isotonic
 - Use of hypotonic fluid → Get absorbed through the venous plexus in it & around the prostatic capsule → Intravascular lysis of blood
- Non conductive
 - To prevent electrical conduction during the surgery (So N.Saline can use during the post operative period)

Irrigation is done through a 3 way latex urethral catheter (To make the inflow & outflow at the same time through the same catheter). Alternatively can use a suprapubic catheter with 2 way urethral catheter (Wider flow lumen than 3 way catheter)

Should carefully monitor the process of irrigation

- Urine bags must be emptied frequently (Because large volumes of fluid used to maintain effective & continuous irrigation)
- Irrigation fluid should be pre-warmed to approximately body temperature (To prevent thermal loss from the body)

If the irrigation fluid suddenly becomes quite clear after haemorrhagic operation, usually suggests

- Fluid is not circulating sufficiently to washout the blood
- Heding haemorrhagic shock (Low BP) & temporary cessation of bleeding

Trans-urethral resection of the prostate (TURP) and bladder tumour (TURBT)

Now at a decline due

- wide usage drug therapy for BPH
- Other minimally invasive procedures such as lasor prostatectomy & prostatic thermotherapy

Procedure

- At present it remains gold standard of prostatic resection
- Done under spinal or general anaesthesia
- The resectoscope is inserted into the urethra and under direct vision central adenomatous part of the prostate gland (Surrounds the prostatic urethra)
- Cut it into small pieces (Paratactic chips)
- And evacuate with the help of an Elliks evacuator.
- Haemostasis is achieved with coagulation ball diathermy & special care is taken not to cut the pseudo capsule and the external urethral sphincter which is situated just distal to the verumontanum (Prostatic utricle)

Post-op

- 3 way catheter is left for 24-48 hours and bladder irrigation with saline until the effluent is clear
- Thereafter trial without catheter (TWOC) is given for 6-8 hours.
- Monitor & observe for
 - Haemorrhage
 - Infection
 - Fibrosis with stricture formation
 - Other specific complications of TURP
 - Irrigant absorption
 - Capsular & bladder perforation (rare with current use of video camera with magnification)

TURP syndrome

(This is extremely rare in TURBT as the bladder doesn't absorb irrigant fluid)

Pathogenesis

Irrigant absorption via prostatic veins → haemodilution, hyponatremia, cerebral/pulmonary oedema

Symptoms

- Headache
- Dyspnoea
- Confusion
- Convulsion
- Death

Warning signs

- Immediate serum Na⁺ levels of <120mmol/L

Prevention

- Restrict the resection time to <1 hour
- Keeping the irrigant reservoir height <90cm
- Performing open prostatectomy for >80g prostates
- Use of isotonic non electrolyte irrigants (1.5% glycine/mannitol) and adding ethanol to the irrigant with regular checking of ethanol levels in the exhaled air

Management

- Early detection
- Stop the surgery
- IV frusemide
- Hypertonic saline(1.8% NaCL)
- TURP syndrome can occur during or after surgery in about 1% of cases.

Procedures and Ix-

IVU-

Contrast media inject usually into a vein in the antecubital fossa, it is filtered from the blood by the glomeruli and does not undergo tubular absorption. As a result, it rapidly passes through the renal parenchyma into the urine, which it renders radio-opaque.

Preparation-

It is usual to give a laxative to clear faeces that might otherwise obscure details of urinary tract anatomy. Modest fluid restriction is permissible, but dehydration is dangerous because it may precipitate acute renal failure.

- Plain film-Looking for calcification overlying the region of the kidneys, ureters, and bladder
- Nephrogram phase-First phase of IVU; film taken immediately following intravenous administration of contrast (peak nephrogram density). The nephrogram is produced by filtered contrast within the lumen of the proximal convoluted tubule (it is a proximal tubular, rather than distal tubular phenomenon).
- Pyelogram phase-As the contrast passes along the renal tubule (into the distal tubule) it is concentrated (as water is absorbed, but the contrast agent is not). As a consequence, the contrast medium is concentrated in the pelvicalyceal system, and thus this pyelogram phase is much denser than the nephrogram phase. The pyelogram phase can be made denser by dehydrating the patient prior to contrast administration. Pelvic compression can be used to distend the pelvicalyceal system and demonstrate their anatomy more precisely. Compression is released and a film taken (20-30 min).

Uses of the IVU

- Investigation of haematuria, detection of renal masses, filling defects within the collecting system of the kidney and within the ureters (stones, TCCs)
- Localization of calcification overlying the urinary tract (i.e. is it a stone or not?)
- Investigation of patients with loin pain (e.g. suspected ureteric colic). Increasingly being replaced with CTU, which has superior sensitivity and specificity
- Very good for identification of congenital urinary tract abnormalities (e.g. ureteric anatomy in duplex systems) (malrotation; horseshoe kidneys)
- Used for follow-up post-ureteric surgery to identify strictures

OBSTRUCTIVE JAUNDICE

History

During history following questions to be answered.....

1. Is this obstructive jaundice
2. What is the cause for obstruction
3. Is it malignant or benign
4. Are there complications

Presenting complaint: jaundice

History of presenting complain

1. Is it obstructive jaundice

- Deep jaundice
- Dark urine
- Pale stools
- Generalized pruritus (without evidence of a skin rash)

2. Symptoms

- Jaundice

- Onset
 - Sudden- gallstones, viral hepatitis
 - Gradual- cirrhosis, pancreatic CA, porta-hepatitis metastases
- Progressive – malignant obstruction by pancreatic CA or cholangiocarcinoma
- Intermittent jaundice
 - Stones in the CBD
 - Peri-ampullary carcinoma(intermittent necrosis of the tumour may relieve the obstruction and cause reduction in jaundice)
- Pain
 - Pain – Viral hepatitis (may have dragging type subcostal pain due to liver enlargement), CBD stones
 - Painless – Carcinoma of the head of the pancreas, peri-ampullary CA, Porta hepatis LNs or metastatic deposits

3. Is it due to intrahepatic cholestasis or extra hepatic cholestasis?

Pre-hepatic causes

- Haemolytic anaemia
 - Hereditary
 - Past history of similar illness
 - History of splenectomy
 - History of thalasseamia & consanguineous anaemia
 - Acquired
 - Auto-immune haemolytic anaemia – Other associated AI diseases (Type 1 diabetes, vitiligo, pre-mature graying)
 - Infection – Malaria, Mycoplasma pneumonia

Causes for extra hepatic cholestasis

- Common
 - common bile duct stones
 - CA head of pancreas
 - Malignant porta hepatis lymph nodes
- Infrequent
 - Ampullary CA
 - Pancreatitis
- Rare
 - Benign stricture (iatrogenic, trauma)
 - Recurrent colangitis
 - Mirrizi's syndrome
 - Sclerosing colangitis
 - Colangiocarcinoma
 - Biliary atresia
 - Choledochal cysts

Intra-hepatic cholestasis

- Acute hepatitis
 - Fever, LOA followed by jaundice
 - Hx of
 - Blood transfusions
 - Sexual promiscuous behaviour within last 6 months
 - Drugs – Anti-psychotics(chlorpromazine)
Anti-TB drugs (Rifampicin)
 - Leptospirosis – Mud contact

- Chronic liver disease
 - Ankle swelling, body swelling, bleeding
 - Impotence
 - Chronic alcohol consumption

- Haemochromatosis
 - Recent darkening of skin
 - Features of heart failure
 - Diabetes mellitus

- Wilson's disease
 - Family history of involuntary movements of the limbs, Jaundice

Extra-hepatic cholestasis

Cause	Important points in the history
Carcinoma of the head of the pancreas	<ul style="list-style-type: none"> - Old age - Painless progressive jaundice - Associated dull epigastric pain radiating to the Back and may worse during night - LOW, LOA - Back pain - Recently diagnosed DM (without FHx & above 50yr) - Ask for features of local spread <ul style="list-style-type: none"> ✓ Gastric outlet obstruction cause vomiting of Undigested food without bile ✓ Profuse upper GI bleeding (vascular invasion) - Ask for features of distant spread (Liver, lung, bone, brain)
Peri-ampullary CA (intermittent jaundice)	<ul style="list-style-type: none"> - Typically presents with fluctuating jaundice & intermittent melaena (silver streaked stools) - LOA, LOW - Ask about features of local & distant spread
Chronic pancreatitis	<ul style="list-style-type: none"> - Recurrent episodes of epigastric pain - Radiates to the back and relive when the patient leaning forward - Associated nausea and vomiting - Altered bowel habits and steatorrhoea - Heavy alcohol consumption - Features of malabsorption

Common bile duct stones (intermittent jaundice)	<ul style="list-style-type: none"> - Previous history of dyspeptic symptoms and other presenting symptoms of gall stones - Billiary colic with Intermittent jaundice - May have features of ascending cholangitis <ul style="list-style-type: none"> ✓ Right hypochondrial pain + Fever + Obstructive Chills jaundice Rigors <p style="text-align: center;">charcot's triad of ascending cholangitis</p>
Acute cholecystitis (Inflammation → swollen GB → compress CBD → causing tinge of jaundice)	<ul style="list-style-type: none"> - Biliary colic & other features of acute cholecystitis - With tinge of jaundice
Mirzzi's syndrome (Gall stone become impact in the cystic duct or neck of the gall bladder causing compression of CBD – RARE)	<ul style="list-style-type: none"> - Biliary colic & other features of acute cholecystitis - Jaundice
Sclerosing cholangitis	<ul style="list-style-type: none"> - Blood and mucous diarrhoea (associated with IBD constitutional symptoms Such as fever chills, and rigors)
Porta hepatis lymph node enlargement	<ul style="list-style-type: none"> - Night sweats - Loss of weight and loss of appetite - Epigastric pain after meals (Gastric CA) - Haematemesis and melaena
Liver flukes	
<i>Clonorchis sinensis</i> (Chinese liver fluke)	<ul style="list-style-type: none"> - Travel history to china - Eating raw fish - Diarrhoea, tender hepatomegaly, cholecystitis, recurrent biliary colic, jaundice -
<i>Fasciola hepatica</i> (S.America, Europe, North Africa)	<ul style="list-style-type: none"> - Eating raw plants

4. Complications of obstructive jaundice

- Coagulopathy due to deficiency of Vit-K dependant clotting factors – Haematuria, Gum bleeding
- Hepatic encephalopathy – Inverted sleep pattern(1st sign), confusion
- Ascending cholangitis – Fever with chills & rigors, RHC pain & worsening jaundice
- Hepato-renal syndrome - ↓ Urine output

PMHx

- Previous billiary surgeries (traumatic strictures CBD)
- Previous Hx of haemolytic anaemia (pigment gall stones)
- DM
- Alcoholic liver disease and chronic pancreatitis

DHx

- OCP
- Octreotide
- Ceftriaxone
- Antipsychotics- chlorpromazine (intra hepatic cholestasis)
- Methyl testosterone

FHx

- Similar symptoms
- Family history of gall stones
- Pancreatic cancer
- Familial cancer syndromes which are associated with pancreatic CA
 - Hereditary breast and ovarian cancer syndrome
 - Familial melanoma
 - Familial pancreatitis
 - Hereditary non Polypoidal colorectal CA
 - Von hippel-landau Xn (increase cancer risk of ampulla of water)

Social history

- Alcohol consumption (Increase pancreatic cancer risk)
- Smoking (Increase pancreatic CA risk)
- Occupation exposure to pesticides, metals (increase pancreatic CA risk)
- Diet- consumption of red meat/processed meat (bacon sausages) (risk of pancreatic CA)
- Fruits and vegetables reduce the risk of CA pancreas
- High cholesterol and low fat diet increase risk of gall stones
- Physical activity
- Sexual promiscuity for other types of jaundice

Examination

General Examination

- Weight/Height/BMI
 - Obese → Gall stone disease
 - Weight loss → CA Gall bladder
- Febrile (Ascending cholangitis, Acute cholecystitis)
- Icterus
 - Lemon tinge jaundice – haemolytic anaemia
 - Obstructive jaundice
 - Intermittant – Chledocholithiasis (obstructive jaundice lasts till the small stone pass through the sphincter of oddi or it disimpacts and falls back to the dilated CBD)
 - Cholidocholithiasis → Ascending infection → Inflamed duct system → Obstructive jaundice)
 - CA Gall bladder – At the late stage
 - Cholidocholithiasis → Chronic back pressure → 2ry biliary cirrhosis or liver failure
 - In acute cholecystitis → swollen GB → Press on CBD → Tinge of jaundice
- Pallor (CA gallbladder)
- Look for signs of chronic liver disease (Jaundice, clubbing, leukonychia, ascites, B/L ankle oedema)
- Signs of hepatic encephalopathy – Constructional apraxia, disorientation, flapping tremors
- Skin scratch marks
- Virchow's node (CA pancreas) → If +ve → M₁ disease → No loco-regional control

Abdominal Examination

Inspection

- Previous surgical scars – Removal of ileum due to IBD (Can result in cholesterol stones)
- Dilated veins
- Ascites
- Visible mass over right hypochondrium – Dilated gall bladder
- Sister Mary Joseph nodule

Palpation

- Palpable gall bladder
- Acute cholecystitis
 - Tenderness & guarding in the right hypochondrium (Distended, inflamed GB wrapped in inflammatory adhesions to adjacent organs, especially the omentum)
 - Murphy's sign – palpate the abdomen just below the tip of the 9th costal cartilage & ask the pt to take a deep breath. When the liver & the attached gallbladder descend & strike the palpating hand, the pt will experience a sharp pain which prevents further inspiration
 - Palpable enlarged GB, beneath the right costal margin

 Courvoisier's law states that if the patient with obstructive jaundice has a palpable gall bladder the cause for the jaundice is unlikely to be due to gall stones

- Hepatitis – Enlarged Liver +/- tenderness

Percussion

- Free fluid – ascites (Peritonitis, Chronic liver disease)

DRE

- Rectal CA → 2ndry deposits in porta hepatis → Obstructive jaundice
- 2ndry mets in pouch of douglas
- Can confirm pale stools

Respiratory system

- Look for evidence of pleural effusions

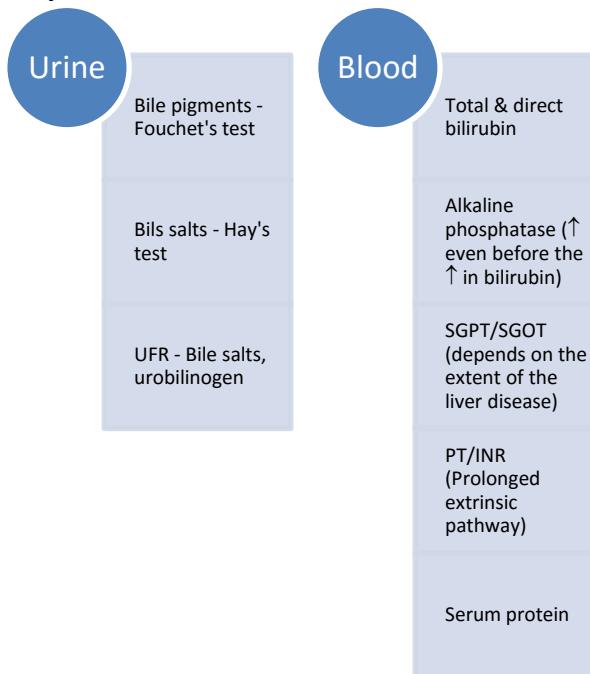
DISCUSSION

Where is the lesion?

Obstructive jaundice with a palpable gallbladder	Obstructive jaundice with an impalpable gallbladder
Obstruction distal to the entry of the cystic duct (There may be an exception to Courvoisier's law with 2 stones, one in the CBD and one in the cystic duct leading to a mucocele)	Obstruction proximal to the entry of the cystic duct Gall stones in the common bile duct (produced in the GB)

What are the investigations you would perform?

1. To confirm the clinical suspicion



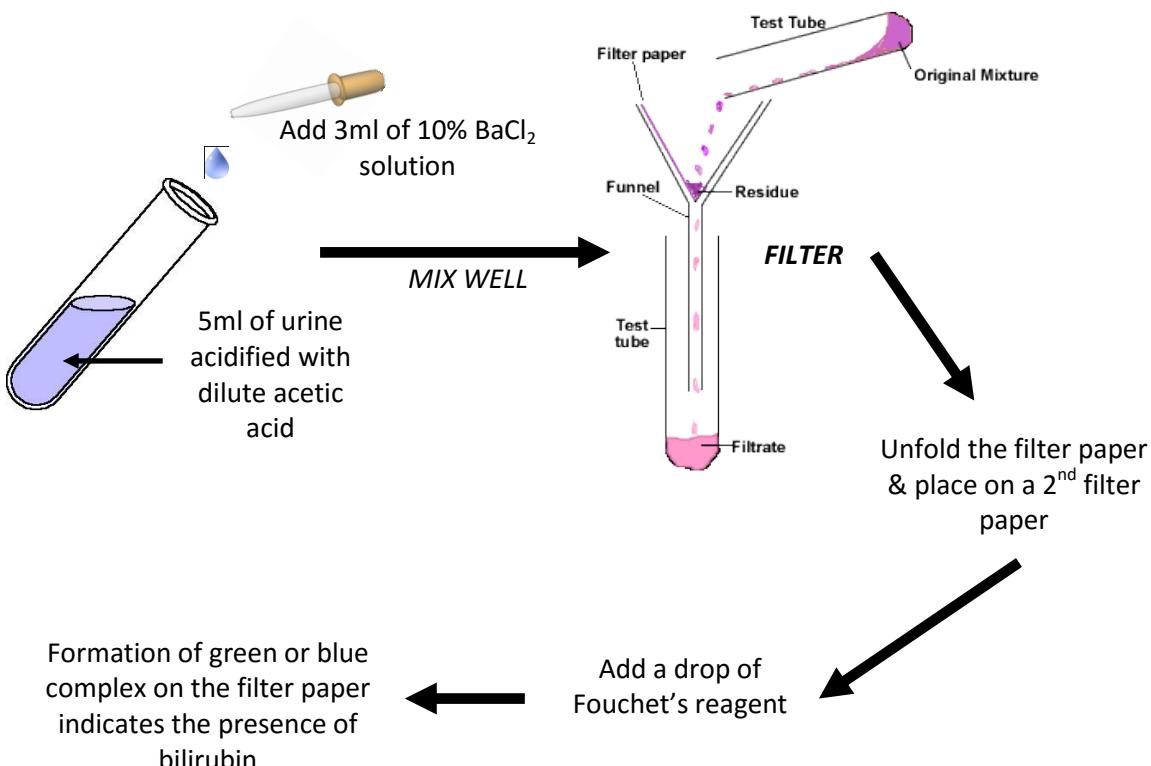
UFR	Haemolysis	Obstruction	Hepatocellular
Conjugated bilirubin	Normal	Increased	Normal
Urobilinogen	Increased	Nil	Normal

Hay's test for bile salts

Sprinkle a dash(a minute quantity to the tip of the spatula) of sulphur on the surface of the sample of urine(5ml). Bile salts reduce the surface tension thus facilitates the sulphur particles to sink and settle at the bottom of the tube.

NOTE : As the test depends on the surface tension reducing property of bile salts, it is important not to contaminate the urine sample with soap or detergents.

Fouchet's test for bile pigments



2. To exclude intra-hepatic cholestasis

Screen for viral hepatitis

- Hep A – IgM
- Hep B – HBsAg → if +ve → HBeAg

3. Diagnose the problem

USS abdomen (keep the patient 6 hours fasting to distend the gallbladder)

- Dilatation of the intra hepatic & extra hepatic ducts (CBD diameter normally $\leq 6\text{mm}$)
- If CBD $\geq 1\text{cm}$ → Definite obstruction
- GB stones (intensely echogenic foci)/GB carcinoma
- Thickened wall of the gall bladder in acute / chronic infection
- Liver mets
- Ascites
- Sensitivity 70-95% and specificity 80-100%

Dilatation of both IH & EH ducts	Only IH duct dilatation	No duct dilatation
Pancreatic head mass	Hilar cholangiocarcinoma	Medical (intra hepatic cholestasis)
Stone in the common bile duct	Gall bladder pathology Mirrizi's syndrome Porta hepatis lymphadenopathy	

4. To determine the site & level of the obstruction

- Contrast enhanced CT abdomen – To stage the disease
 - Useful in obesity or excessive bowel gas
 - Better at imaging lower end of CBD
 - Staging
 - Demonstrate pancreatic lesions
- ERCP (Best option)
 - Invasive – Use side viewing endoscope
 - Need higher skills
 - Has a therapeutic arm
 - Dynamic
 - If the dye accidentally injected to pancreatic duct → Can give rise to acute pancreatitis
- PTCA (Percutaneous transhepatic cholangiography)
 - If ERCP not available
 - Use chiba needle
- MRCP
 - No therapeutic arm

5. Supportive investigations

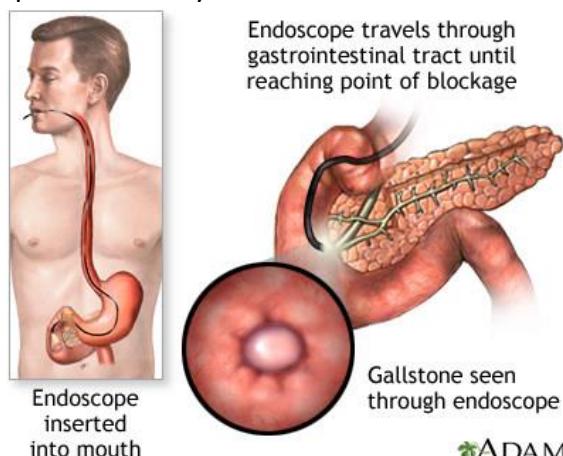
- FBC – Cholangitis, Hb
- Renal function tests- BU /SE (hepatorenal syndrome)
- Plain X-Ray abdomen – Pancreatic calcifications in chronic pancreatitis

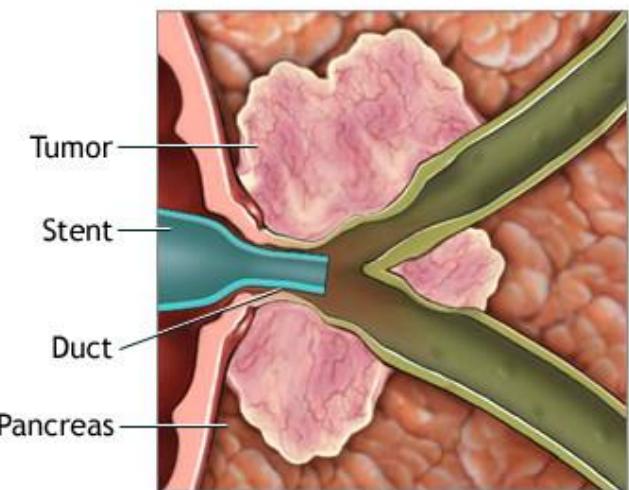
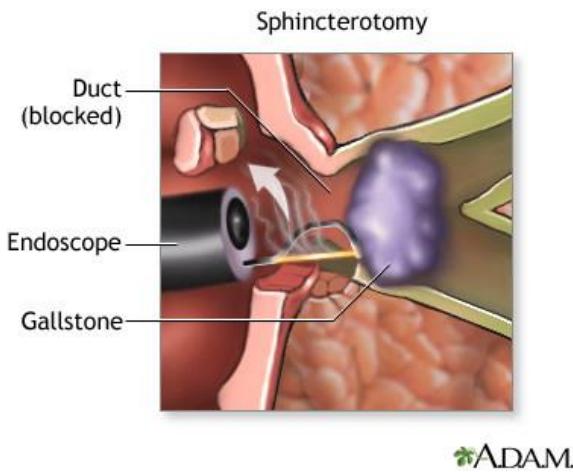
ERCP (endoscopic retrograde cholangiogram)

A dye is injected into the bile and pancreatic ducts using a flexible, fiber-optic endoscope. Then x-rays are taken to outline the bile ducts and pancreas.

Equipment

- The flexible endoscope
- ERCP is used for therapeutic treatment of:
 - a. Stone extraction
 - b. Internal biliary stent placement
 - c. Endoscopic sphincterotomy





Pre op

- Admit 2 days prior to the procedure
- Investigate – BT / PT
- If PT abnormal
 - Vit K 10mg/d IM for 3days and repeat PT
 - If not corrected by Vit K → Give FFP and repeat PT
- Maintain hydration
- Keep fasting 6 hrs
- 1hr prior to ERCP – IV Cefuroxime 750mg (cont for 24hrs)
- Send to endoscopic room with BHT and medications

Procedure

- IV cannula (right hand)
- Pre medication with – IM medozolam and IV hyoscine butylbromide
- Put patient in to left lateral position
- Local anesthesia spray 10% lignocaine (xylocaine)
- Upper GI endoscope with side view
- Cannulate ampulla
- Insert water soluble contrast medium and serial x-rays to visualize
 - Pancreatic duct
 - CBD
 - Extra hepatic duct
- Take bile for cytology and culture/ABST
- CBD brushing for cytology
- If any obstruction - sphincterotomy and stenting
- Removal of stones- sphincterotomy
 - Stenting
 - Balloon sweeps for microliths
 - Microliths extraction with basket extraction
- Remove the scope and keep patient for ½ hr in left lateral position

Post op

- NBM for 24 hrs
- IV fluid 2 pints - Normal saline or 3 pints of 5% dextrose for 24 hrs
- Continue antibiotics for 1 day
- Diclofenac suppository 10mg bd
- Observe for complications

Complications

- Acute pancreatitis
- Acute cholangitis

If acute pancreatitis occurs (10 R's)

1. Resuscitate with IV fluids
2. Relieve pain with opiates
3. Rest pancreas by NBM and IV fluids
4. Rest bowel → NG Tube
5. Resist → enzymal activity → peritoneal lavage
6. Resist infection – IV cefuroxime 750mg tds
7. Reassess and resuscitate
8. Re investigate after 48 hrs
 - S. Ca
 - S. Albumin
 - LDH
 - ALT/AST
9. Renal out put → 0.5-1ml/kg/hour
10. Respiratory support for ARDS

PCT (per cutaneous trans hepatic cholangiography)

Pre op

- Assess past history of hypersensitivity
- Ix- USS Intrahepatic ducts dilated
Coagulation profile
- IV antibiotics (prophylactic)- IV cefuroxime 750 mg tds
1hr prior and cont for 24 hrs
- Maintain good hydration
- 4-6 hrs fasting

Procedure

- Under local anesthesia
- Pliable needle in to liver through mid axillary line and inject water soluble contrast medium
- Take serial Xrays
 - I. And visualize
 - II. Percutaneous insertion of endo-prosthesis/stent
 - III. Demonstrate anatomy above extra hepatic duct obstruction

MANAGEMENT OF PANCREATIC CA

Pre-op

1. Breaking bad news
2. Informed written consent
3. Electrolyte and fluid balance to prevent hepato-renal syndrome
 - IV fluid and adequate hydration
 - Maintain fluid balance chart → Maintain UOP 100cc/hour
4. Correction of coagulopathy → IV Vit K 10mg/day for 3 days → Check PT/INR after 3 days → If no increase → Give FFP
5. Prophylactic Antibiotics (To prevent hepato-renal syndrome)
 - IV cefuroxime 750mg
 - IV Metronidazole 500mg
6. Correction of serum protein
7. Oral lactulose 20ml/bd to prevent endotoxin absorption (If constipation)
8. Anaesthetic referral
9. Grouping & DT – Reserve 3 packs of blood
10. Chest physiotherapy – Incentive spirometer
11. Steam inhalation
12. Reserve an ICU bed

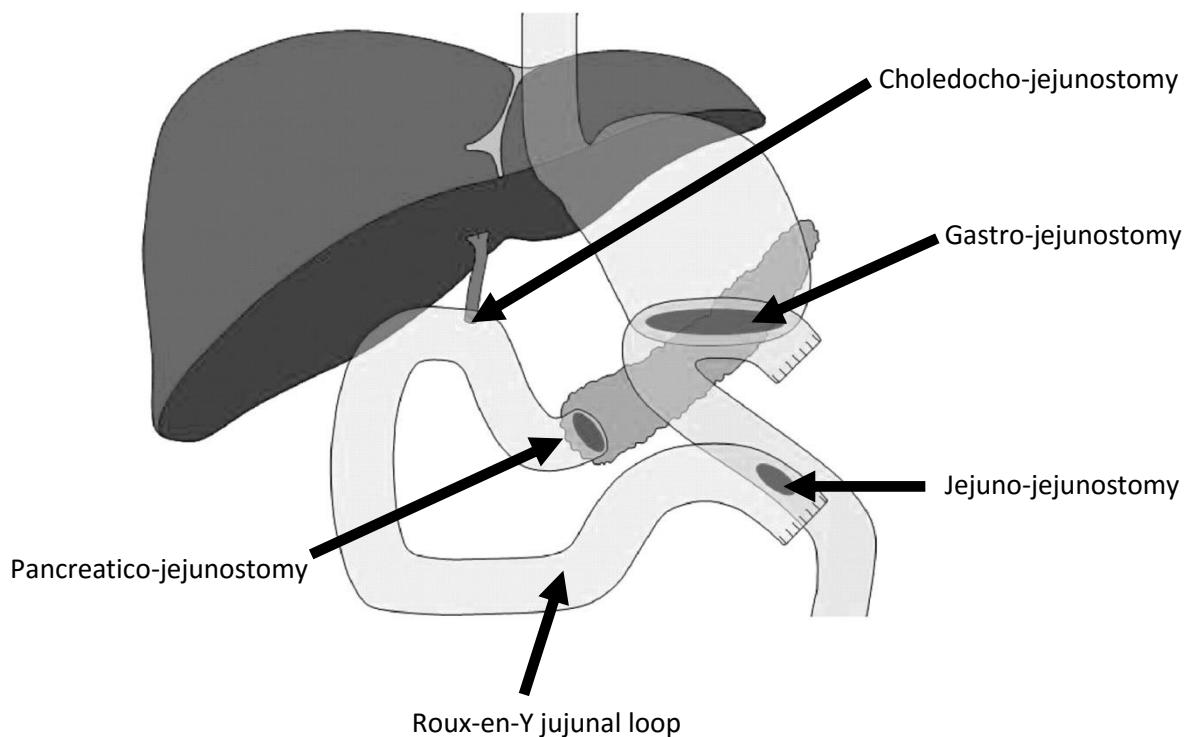
Assess the fitness for surgery

- ECG
- CXR
- 2D Echo
- S. Creatinine
- BU/SE
- LFT
- FBS

IN CASE OF DELAY OF SURGERY OR AS PALLIATION, SYMPTOMATIC RELIEF BY ERCP & STENTING HAS TO BE DONE

- 10 French gauge needle (ERCP needle) & self expanding metal stent

Surgery of choice for CARCINOMA OF THE PANCREAS is PANCREATICODUODENECTOMY (WHIPPLE'S PROCEDURE)



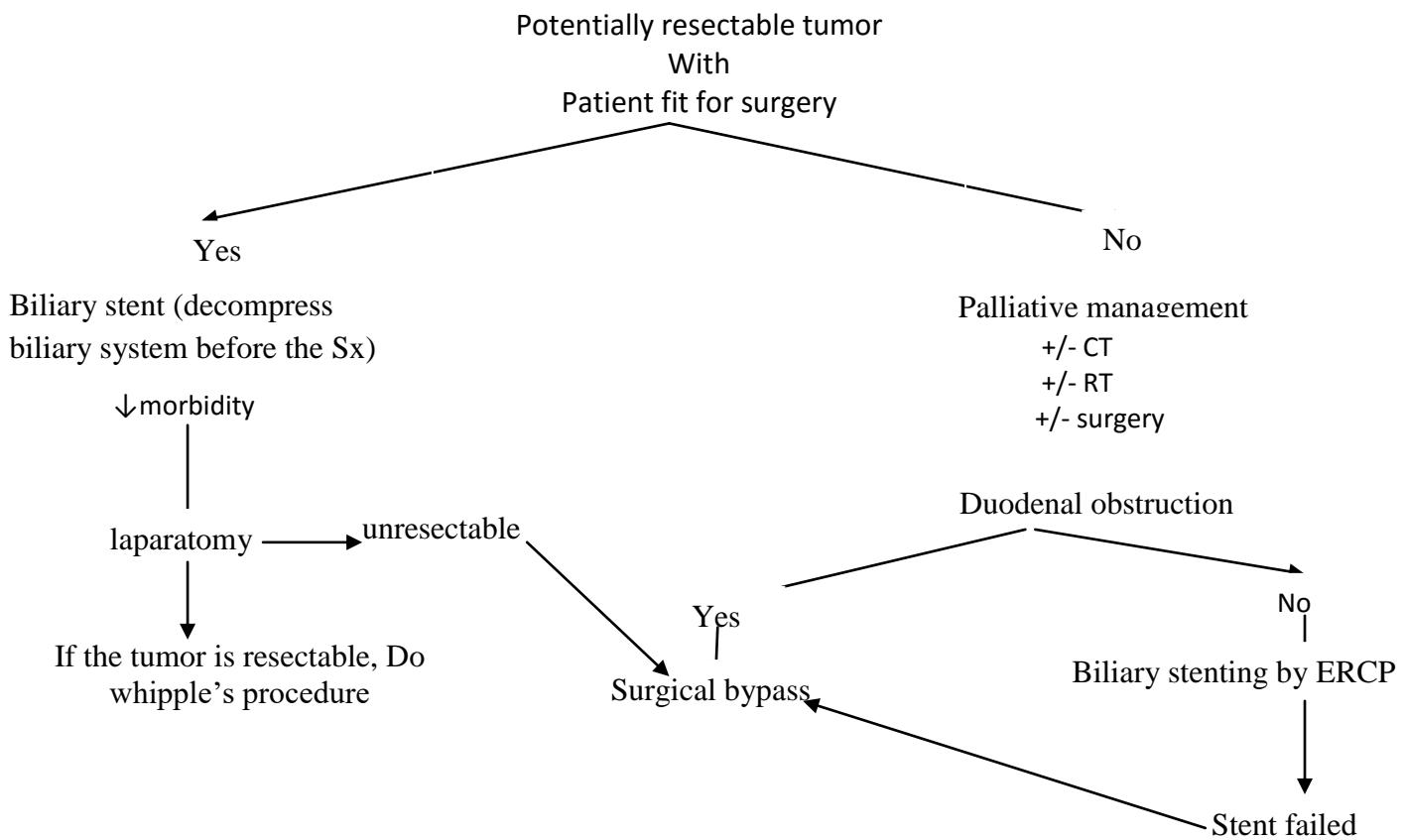
- Midline laparotomy. Look for liver secondaries, ascites LN enlargement, fixity and resectability of the tumor.
- Gall bladder. Common bile duct, distal stomach and duodenum with the tumor and head of the pancreas are removed.
- Reconstruction of gastrointestinal continuity by creating Roux –en- Y loop with the above illustrated anastomosis.
- Feeding jejunostomy & drain are inserted.

Post-op

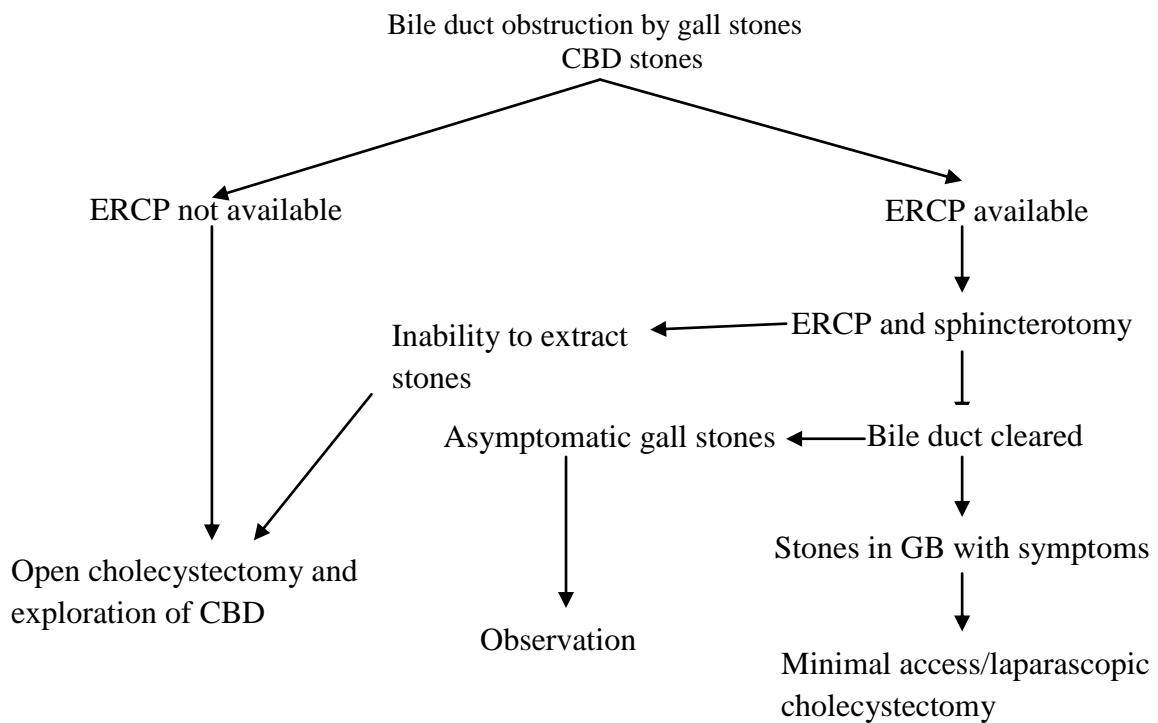
- ICU care
- Monitor PR, BP, RR. Temperature, UOP
- IV fluids – N. Saline, 5% dextrose, KCL if hypokaleamia develops
- Pain relief – Epidural analgesia (Light bupivacaine)
- Antibiotics
 - Cefuroxime 750mg IV 8H
 - Metronidazole 500mg 8H
- Feeding
 - No jejunostomy feeding till bowel sounds appear
 - Then start jejunostomy feeding from the 2nd day
- After 10 days do gastrographin study to exclude anatomic leakage. If no leakage start oral sips of fluid. Then increase gradually.
- Post op chest physiotherapy, steam inhalation
- Sutures are removed on the 10th post-op day.
- Follow up the patient at clinic with histology report.
- Oncology referral

 **Modified whipple's surgery – Preserve the pylorus to maintain controlled gastric emptying.**

Rx options for malignant bile duct obstruction



Management of CBD stones



JAUNDICE

Excess circulatory bile → Yellowish discoloration of skin/mucous membranes/sclera

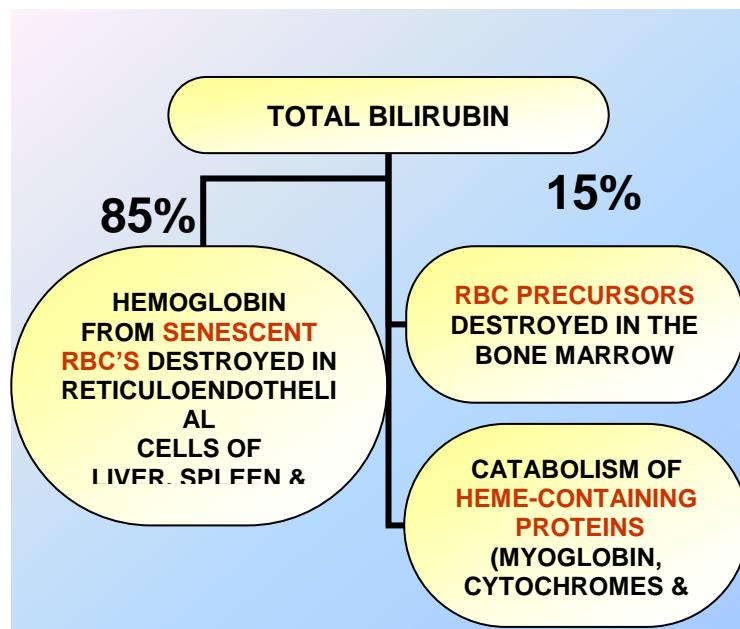
Normal → total bile → 5-17 µmol/l (<1mg/dl)

Jaundice → total bile → >40 µmol/l (>2mg/dl)

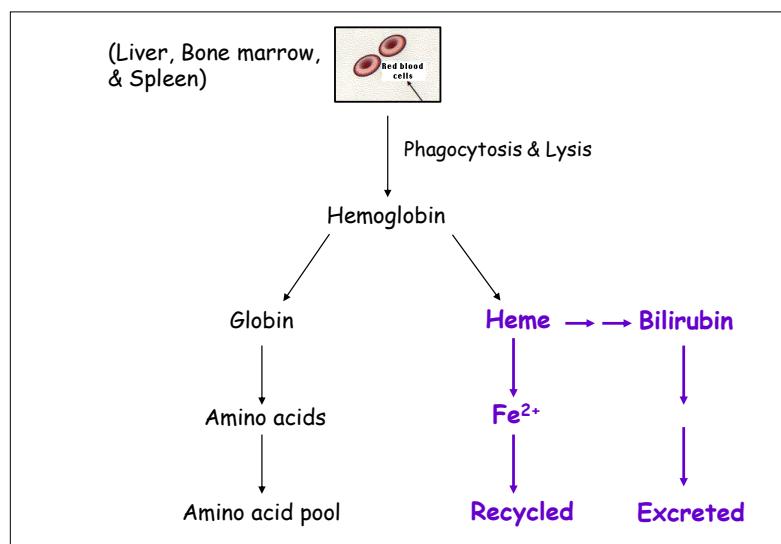


Bile pigment metabolism and defects

- An average person produces about 4 mg/kg of bilirubin per day.
- The daily bilirubin production from all sources in man averages from 250 to 300 mg.



Extravascular Pathway for RBC Destruction

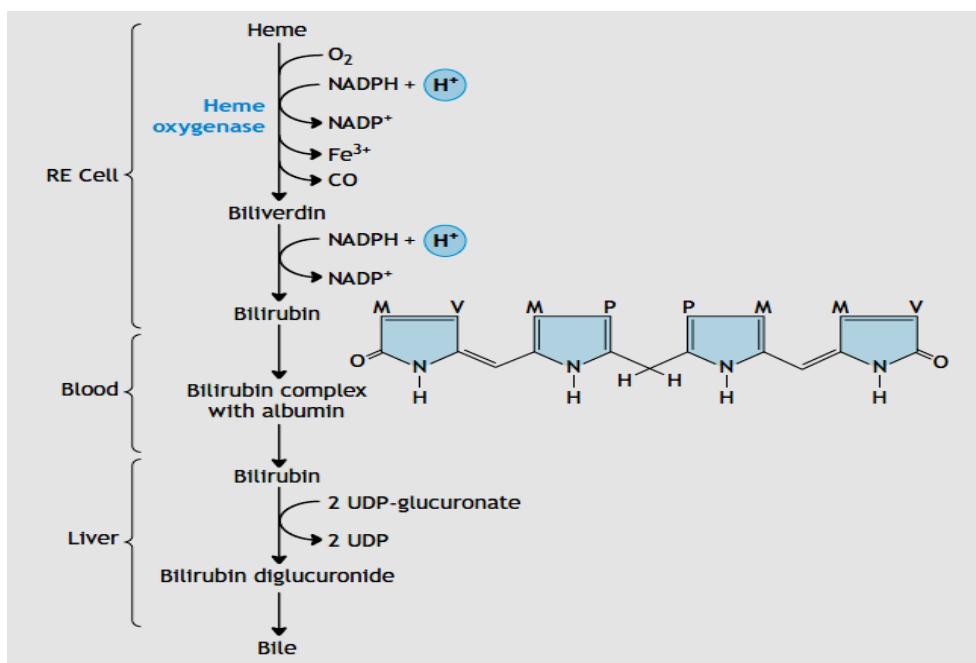


- 85% is derived from RBCs

- In normal adults this results in a daily load of 250-300 mg of bilirubin
- Normal plasma concentrations are less than 1 mg/dL
- Hydrophobic – transported by albumin to the liver for further metabolism prior to its excretion
- Uptake of bilirubin by the liver is mediated by a carrier protein (receptor)
- Uptake may be competitively inhibited by other organic anions
- On the smooth ER, bilirubin is conjugated with glucuronic acid, xylose, or ribose
- Glucuronic acid is the major conjugate - catalyzed by UDP glucuronyl transferase
- “Conjugated” bilirubin is water soluble and is secreted by the hepatocytes into the biliary canaliculi

Unconjugated bilirubin

- Lipid soluble
- : limits excretion
- 1 gm albumin binds 8.5 mg bilirubin
- Fatty acids & drugs can displace bilirubin
- Indirect positive reaction in van den Bergh test



In the Intestine

- In the small intestine, conjugated bilirubins are poorly reabsorbed, but are partly hydrolyzed back to unconjugated bilirubin by catalytic action of bacterial β -glucuronidases.
- In the distal ileum and colon, anaerobic flora mediate further catabolism of bile pigments:
 - a) hydrolysis of conjugated bilirubin to unconjugated bilirubin by bacterial β -glucuronidases;
 - b) multistep hydrogenation (reduction) of unconjugated bilirubin to form colorless urobilinogens(stercobilinogens); and oxidation of unconjugated bilirubin to brown colored mesobilifuscins

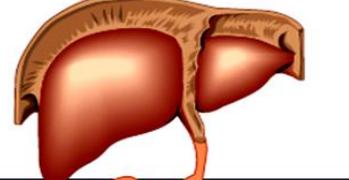
- Urobilinogens is a collective term for a group of 3 tetrapyrroles;

- Stercobilinogen (6H)
- Mesobilinogen (8H)&
- Urobilinogen (12H)

Up to 20 % of urobilinogen produced daily is reabsorbed from the intestine & enters the entero-hepatic circulation

Different Causes of Jaundice

- Excessive Production of Bilirubin
- Reduced Hepatocyte Uptake
- Impaired Bilirubin conjugation
- Impaired Bile Flow

Types	HAEMOGLOBIN ↓ BILIRUBIN	Causes
Prehepatic		Haemolysis
Cholestatic HEPATIC	CONJUGATION 	Viral hepatitis Drugs Alcoholic hepatitis Cirrhosis – any type Pregnancy Recurrent idiopathic cholestasis Some congenital disorders Infiltrations
POST HEPATIC	GALL BLADDER PANCREAS	Common duct stones Carcinoma <ul style="list-style-type: none"> bile duct head of pancreas ampulla Biliary stricture Sclerosing cholangitis Pancreatic pseudocyst

	Pre-hepatic	Hepatic	Post hepatic
cause	Excessive break down Of RBC's Malaria, HS Gilbert Syndrome	Infective Liver Damage	Bile Duct Obstruction
Serum Bilirubin	unconjugated	Both conj+unconj.	conjugated
Urine bilirubin	Absent Achloric jaundice	Bilirubinuria + Deep yellow urine	As in hepatic jaundice ++
Urine urobilinogen	Increases Because of increased stercobilinogen	Decreases Because of decreased stercobilinogen	Absent(-)
Fecal stercobilinogen 20-250mg/day	Markedly increased Dark brown stool	Reduced Pale coloured stool	Absent clay colored stool
Fecal fat 5-6%	normal	Increased 40-50% Bulky, pale greasy foul smelling faeces	As hepatic jaundice
Liver functions	normal	Impaired SGOT/SGPT	Normal Alkaline phosphatase++
Von derburg test	Indirect+	biphasic	Direct+

Inherited Disorders of Bilirubin Metabolism

Gilbert's Syndrome

Crigler-Najjar (Type I)

Crigler-Najjar (Type II)

Lucey-Driscoll

Dubin-Johnson

Rotor's Syndrome

Algorithm for differentiating the familial causes of Hyperbilirubinemia

Crigler-Najjar Syndrome (Type I) is a rare genetic disorder caused by complete absence of UDP-glucuronyltransferase and manifested by very high levels of unconjugated bilirubin.

It is inherited as an autosomal recessive trait.

Most patients die of severe brain damage caused by kernicterus within the first year of life.

Early liver transplantation is the only effective therapy.

Crigler-Najjar Syndrome (Type II)

This is a rare autosomal dominant disorder.

It is characterized by partial deficiency of UDP-glucuronyltransferase.

Unconjugated bilirubin is usually 5 – 20 mg/dl.

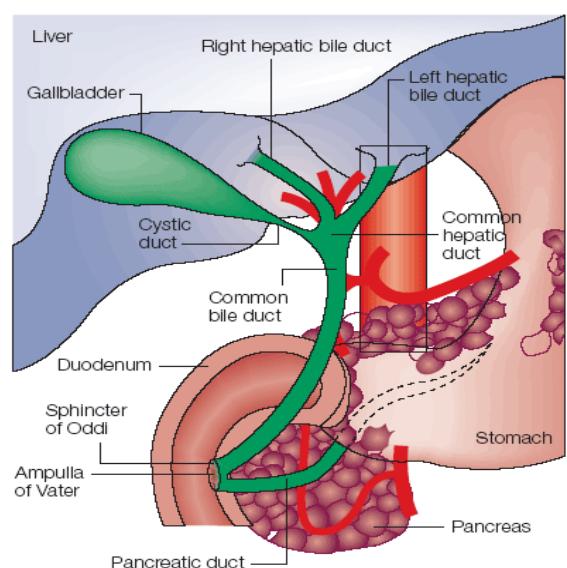
Unlike Crigler-Najjar Type I, Type II responds dramatically to Phenobarbital & a normal life can be expected.

- Gilbert's syndrome is also called as familial non-hemolytic non-obstructive jaundice.
- mild unconjugated Hyperbilirubinemia.
- It affects 3% – 5% of the population. It is often misdiagnosed as chronic Hepatitis.
- The concentration of Bilirubin in serum fluctuates between 1.5 & 3 mg/dl.
- In this condition the activity of hepatic glucuronyltransferase is low as a result of mutation in the bilirubin-UDP-glucuronyltransferase gene(UGT1A1).

Dubin-Johnson Syndrome

- It is a benign, autosomal recessive condition characterized by jaundice predominantly elevated conjugated bilirubin and a minor elevation unconjugated bilirubin.

- Excretion of various conjugated and bilirubin into bile is impaired reflecting the underlying defect canalicular excretion.



- The Liver has a characteristic gray-black appearance and liver biopsy reveals a dark brown melanin-like pigment in hepatocytes and kupffer cells.

Fatal consequences of obstructive jaundice

Hepato renal syndrome

Rapid deterioration of the kidney function

Fatal condition

2types – rapid condition

- slow progressive

Diagnostic criteria

Major

- Liver disease and portal hypertension
- Renal failure in the absence of shock
- Absence of improvement renal function despite Rx with 1.5l of IV N/S
- Absence of renal disease or obstruction to renal flow in USS

Minor

- reduced urine output
- low Na⁺ in urine <107μmol/l
- low serum Na⁺ <130mmol/l
- urine osmolality > blood
- absence of RBC in urine

Mx

Terlipressin (vasopressin analogue)

Sepsis

Due to bile stasis and colangitis

Bacterial colonization and over growth

Septicemia

Mx prophylactic antibiotics covering coliforms

Lactulose

Hypocoagulability

Bile stasis → ↓ absorption of fat soluble vitamins (vit K) → impaired γ carboxylation of produced Clotting factors (ii, vii, ix ,x)

Prolong PT (32+/- 7)

Mx

Monitor clotting profile

Pre op vit K (6-8hr)

Emergency FFP

Per rectal bleeding

DD

- Haemorrhoids
- Anal fissure
- Colorectal CA
 - CA Rectum
 - CA Sigmoid colon
 - CA of Descending colon
- IBD
- Amoebiasis

- Striking symptoms ass. With bleeding
- Probable diagnosis
- Complications
- Predisposing factors
- Exclude other DD

Right colonic carcinoma will not present as bleeding per rectum. Yet discussed here.

PC – bleeding PR – duration

HPC

- ♥ Onset
- ♥ Duration
- ♥ Type – dark (rectal) bleeding
 - Bright red/ fresh blood – anal canal
 - Altered blood – upper GI (malaena)
- ♥ Mixed with stools – bleeding higher than sigmoid colon
 - Streaking the stools – surface bleeding
 - Bleeding at the beginning and end
 - Lower sigmoid colon, rectum and anus
 - Separate from faeces
- ♥ Ass. Mucus – lesion in the large bowel
- ♥ Pain – perianal/ lower abdominal
- ♥ Tenesmus – painful desire to defecate w/o resultant evacuation
- ♥ Feeling of incomplete evacuation
- ♥ Altered bowel habits – alternative episodes of constipation and diarrhoea
- ♥ Spurious diarrhoea – mucoid diarrhoea early in the morning
- ♥ Progression – volume (drop by drop → discolouring the pan)
 - Frequency
- ♥ Any discharge, pruritus
 - Mucus secretion → watery diarrhoea
 - Hypokalemia
 - Mucus irritating anal mucosa → pruritus ani

Condition	Points in the Hx							
Haemorrhoids	<p>Bleeding after defecation</p> <p>Fresh blood streaking the stools – surface bleeding Drop by drop/ flash in pan (fill up whole pan) Lump/mucosal tag coming out of anus Perianal discomfort – prolapsed and thrombosed haemorrhoids</p> <p>Aetiology – chronic constipation</p> <table style="margin-left: 200px;"> <tr> <td>Pregnancy</td> <td rowspan="3" style="vertical-align: middle; font-size: 2em;">}</td> <td>Congested</td> </tr> <tr> <td>Rectal CA</td> <td>sup. rectal</td> </tr> <tr> <td>Pelvic tumour</td> <td>V.s</td> </tr> </table> <p>Co-existing ano-rectal varices (portal HTN)</p> <p>Complications – prolapse → mucus discharge, pruritus, faecal soiling Thrombosis – strangulation of prolapsed piles by anal Sphincter → obstruct venous return, +/- ulcerate</p>	Pregnancy	}	Congested	Rectal CA	sup. rectal	Pelvic tumour	V.s
Pregnancy	}	Congested						
Rectal CA		sup. rectal						
Pelvic tumour		V.s						
Anal fissures	<p>Fresh blood streaking stools Spurting of blood</p> <p>Painful, pain is sharp and severe Pain starts during defecation and lasts long after defecation (~1hr)</p> <p>Aetiology – chronic constipation and hard stools Crohn disease, herpes simplex infection – HIV pts.</p>							
CA of the rectum	<p>Age – 50-70years</p> <p>Dark rectal bleeding</p> <p>Blood streaking stools / (mixed with stools – Hasanjaya sir) Altered bowel habits</p> <p>Tenesmus Sense of incomplete evacuation Spurious diarrhoea – mucoid diarrhoea early in the morning (95%)</p> <p>Complications –</p> <p>1) subacute intestinal obstruction</p> <ul style="list-style-type: none"> - Episodes of colicky lower abdominal pain with absolute constipation & abdominal distension - Spontaneously resolved <p>2) local spread</p> <ul style="list-style-type: none"> - sacral plexus → intractable backpain - bladder → formation of recto-vesical fistula (faecaluria, pneumaturia & Haematuria) <p style="text-align: center;">Recurrent UTI</p> <ul style="list-style-type: none"> - vagina → recto-vaginal fistula (faecal incontinence) - Infiltration of the anal sphincter → faecal incontinence - Ureters → hydronephrosis (loin pain) - perforation → peritonitis <p>3) Distant metastasis</p> <ul style="list-style-type: none"> - Liver → LOA, LOW - Lung → chronic cough, pleuritic type chest pain - Brain → early morning headache with vomiting, late onset fits - Bone → bone pain, backache (sacrum) 							

	4) Anaemia Aetiology – FHx of colorectal, uterine, ovarian and gastric CA – 1 st degree FHx of longstanding blood and mucous diarrhoea – UC Diet – poor in fibre High protein & high fat
CA of sigmoid colon	Altered blood Blood streaking/ mixed with stools Same features as above Complications – colovesical fistula instead of rectovesical
CA of descending colon	Altered blood mixed with stools No tenesmus, sense of incomplete evacuation
IBD	Blood and mucus diarrhoea Systemic features - Low grade fever Extra- GI → large joint pain and swelling, red eye, cholangitis skin rashes, oral ulcers
RARE Colitis Diverticular disease	- blood and mucus diarrhoea Eg:- Amoebiasis – gradual onset, Abd pain - elderly > 80years - diarrhoea/ constipation - diverticulitis – acute appendicitis like, blood and mucus diarrhoea Complications – fistula, bowel obstruction, perforation, per rectal bleeding Aetiology – low fibre diet, genetic
Angiodysplasia of colon Ischemic colitis	- elderly → rectal bleeding - Hx of chronic lung disease

DD – according to symptoms

Rectal bleeding, mucous diarrhoea,
tenesmus

- ♥ Rectal CA
- ♥ CA of sigmoid colon
- ♥ Ulcerative colitis
- ♥ Diverticular disease
- ♥ Bacillary dysentery
- ♥ Amoebiasis
- ♥ Radiation proctitis

Painful PR bleeding

- ♥ Anal fissure
- ♥ CA of anal canal

Mucus diarrhoea

- ♥ Villous adenoma
- ♥ VIPoma

PMHx – Hx of bleeding tendency

IBD – relapses and remissions, serial colonoscopy

Exposed to radiation – Radiation proctitis

PSHx – uretersigmoidostomy – after bladder removal reconstruction Sx

↑ risk of colorectal CA (Rare)

Cholecystectomy – trivial ↑ in risk of colon CA 30years after Sx

DHx – blood thinners – aspirin, clopidogrel, anti coagulants

SHx - Smoking

Dietary Hx – low fibre diet, red meat, processed food

Personal hygiene, food hygiene

Family support to the Pt

Psychological status of the Pt

Impact on occupation

Knowledge on disease and impending Sx, its complications Eg:- colostomy

Examination

General

Haemodynamically stable – lying comfortably on bed/ dyspnoeic

If blood is transfused / normal saline – indicate hypovolaemia

Pallor

Icterus – 2ry deposits in porta hepatis

LN – Virchow's node (spread via thoracic duct)

Inguinal LN – with anal invasion

Evidence of subcutaneous/ mucous membrane petechial haemorrhages – bleeding tendency

Clubbing - IBD

Peripheral oedema

Peripheral stigmata of IBD – skin – Erythema nodosum, Pyoderma gangrenosum

Eye – Scleritis , Uveitis

CVS Ex

Look for hypovolemia – tachycardia, hypotension

Abdominal Ex

Inspection

Abdominal distension, dilated veins, surgical scars

Palpation

Palpable masses

Liver – enlarged, what lobes affected, surface, nodules

Kidney – hydronephrosis (balatable)

Para aortic LN

Ascites

Liver dullness (if absent – presence of air b/w- indicate bowel perforation)

DRE

Inspection - Prolapsed haemorrhoids, mucosal tags

Perianal fistula – multiple with PR bleeding – CD

Anal fissures, lumps protruding from anus



Palpation - Sphincter tone (ask Pt to tighten over the inserted finger)

Mucosal growth – distance from anal verge

Upper margin reachable or not

Consistency, surface – smooth/ nodular

Mobility of the mucosa overlying the lump and elsewhere

Prostate – size, median groove, tender, mucosa mobile over gland surface

Faeces - +/-, consistency, contact bleeding, malaena , mucous discharge

How to do →

- 1) Inform the patient of what you intend to do.
- 2) Require disposable gloves and lubricant gel (lignocaine – local anaesthetic gel if anal fissure)
- 3) The patient made to lie in the left lateral position with both knees drawn up to the chest
- 4) The buttock at the edge of the examination couch.
- 5) Inspect the anus after separating the buttock.
 - *Anal scars suggestive of previous operation or perineal tears in parous female*
 - ***a patulous anus due to reduced anal sphincter tone/ resisting anus***
 - *erythema, skin excoriation, skin tags – posterior (anal fissures common)
anterior (fissure difficult to heal)*
 - *prolapsed haemorrhoids,*
 - *genital and peri-anal warts*
 - *The external opening/s of an anal fistula or sinus.*
- 6) Apply gel to the peri-anal area and, to the tip of your examining index finger.
- 7) Gently **introduce the finger** into the anal canal whilst asking the patient to breathe comfortably **and bear down**, which helps reduce internal anal sphincter tone and facilitates anal canal opening.
- 8) If there is severe pain, likely that the internal sphincter is in spasm. It's Suggestive of a fissure.
- 9) Abandon the procedure → examine UNDER MAJOR ANALGESIA/ EUA
- 10) Once the finger is within the anal canal, palpate
 - *Mass, polyp, ulcers*
 - *3,7,11 'o' clock positions - haemorrhoids*
 - *Check the anterior posterior and lateral walls,*
 - *The prostate gland in males and the pouch of Douglas in female patients respectively.*
- 11) Wipe the perineum dry

Summary

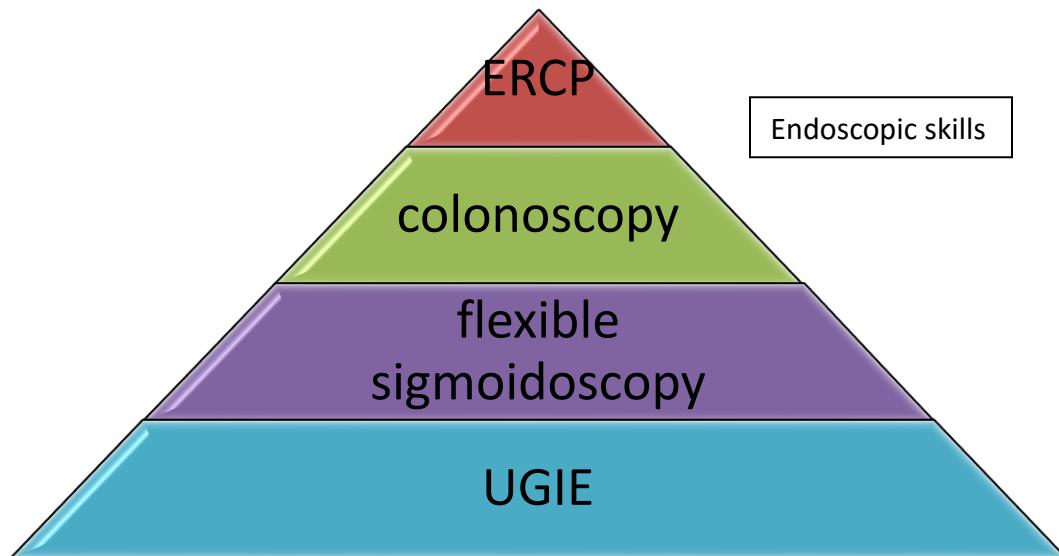
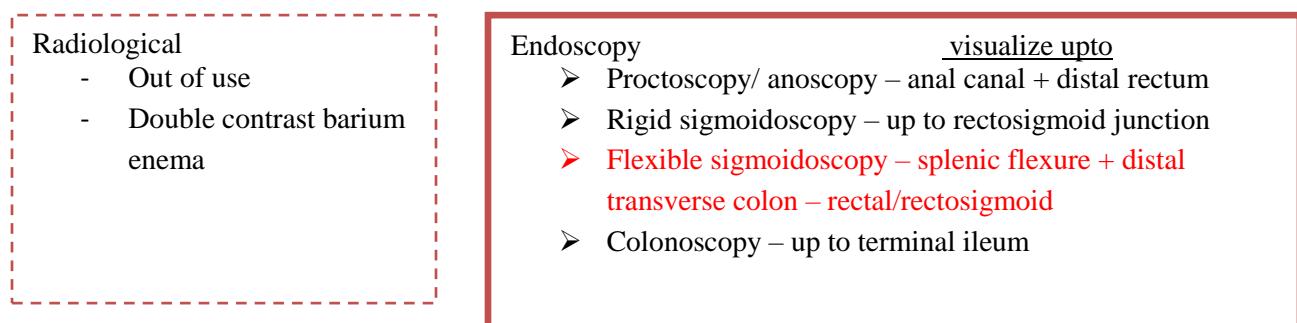
Discussion

DD – colorectal CA possible site is (rectum/rectosigmoid junction/ sigmoid colon/descending colon) and staging (evidence of distant metastasis/local spread)

How will you manage?

1) Ix – confirm the diagnosis

⊕ Visualize the lesion



- After visualizing the lesion biopsies taken for histological diagnosis
- Next line of Ix based on the biopsy report.

Proctoscopy

Length – 13cm

Adv. → 1) no bowel preparation needed

2) Does not require much skill

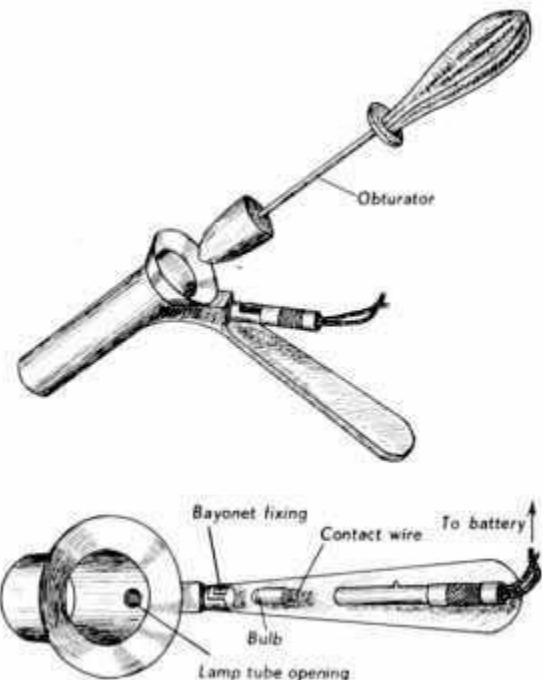
Visualize haemorrhoids & lumps

(haemorrhoids are not palpable unless prolapsed & thrombosed. Seen as purple cushions at 3, 7& 11 'o' clock positions)

Able to perform banding and injection of sclerotherapy

Take biopsy

Disadv – can only visualize anal canal



Rigid sigmoidoscopy

Length – 25cm - 30cm

Adv. – 1) no bowel preparation needed – can be done as an outpt. Procedure (microenema bowel preparation)

2) Lesser skills needed

3) Can take a Bx from a rectal growth

Disadv. - Can visualize only the full length of the rectum

Flexible sigmoidoscopy

Length – 60cm (70-80cm)

Can visualize up to splenic flexure and distal transverse colon

Adv. – 1) able to identify 70% of colorectal CA – as 70% arises in the region up to the proximal transverse colon from the recto-anal junction

2) Can take Bx

Disadv. – 1) require bowel preparation

Bowel cleansing – bisacodyl 3 tablets for 3 consecutive nights before Ix →

Phosphate enema/ bisacodyl suppository 2/ kleen enema 2 bottles on the morning of the examination

2) More skills

Colonoscopy

Length – 160cm

- Adv. – 1) full colonic evaluation
2) Take Bx
3) Primary prevention of colorectal carcinoma via colonoscopy and polypectomy
4) Palliative procedures in those not fit for Sx – stenting

- Disadv. – 1) full bowel preparation
2) Require skilled person
3) Require sedation – midazolam and IM pethidine

Dose – IV midazolam 5mg stat. + IM pethidine 25mg stat

Midazolam vial – 5mg Pethidine vial – 50mg

Pethidine given with IV metoclopramide 10mg stat

- 4) Since risk of respiratory depression – require pulseoximeter monitoring
5) Availability of O₂ and drug antidotes
Eg: midazolam – Flumazenil (1vial 0.5mg/5ml)
Pethidine - Naloxone
6) Need a recovery area

Full bowel preparation

- ❖ Low fibre diet for 3 days prior to the Ix
- ❖ Light diet in the morning before the Ix
- ❖ If elderly >70years – start N/S drip before commencing
- ❖ 11am – 12noon – 1 sachet of polyethylene glycol dissolved in 1L of boiled cooled H₂O
- ❖ Next hour – take clear fluid (strained king coconut water, diluted plain tea, sprite etc)
- ❖ 1-2pm – next sachet
- ❖ 2-3pm – clear fluids, avoid dark coloured fluids → onset of diarrhoea 2-3x
- ❖ 3-4pm – 3rd sachet → 10-15x diarrhoea
- ❖ 4-5pm – clear fluids
- ❖ 5-6pm – 4th sachet → watery diarrhoea
- ❖ up to 12MN continue clear fluid → only water until Ix

- 7) Require admission and informed written consent (explain the procedure, outcomes and risks)
8) Stop all anti-coagulants Eg: Aspirin - clopidogrel

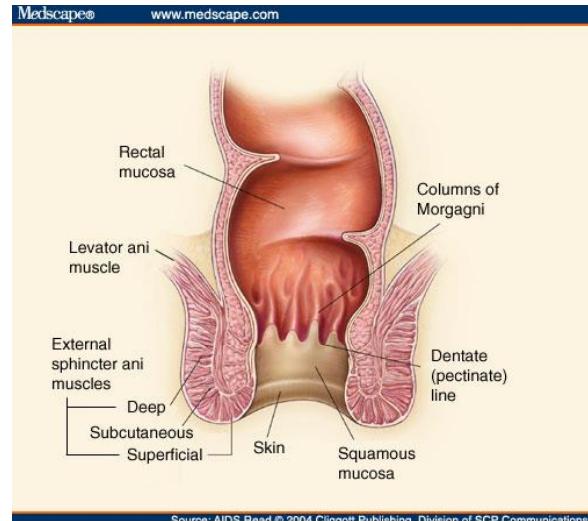
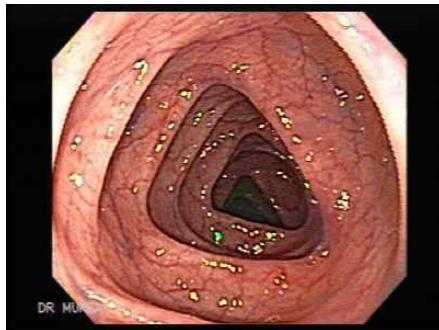
Complications

- 1) Electrolyte and fluid imbalance – due to bowel preparation
KUO for 6hrs, resume normal diet

2) Bowel perforation – if undue abdominal pain + tachycardia → send immediately for exploration & repair

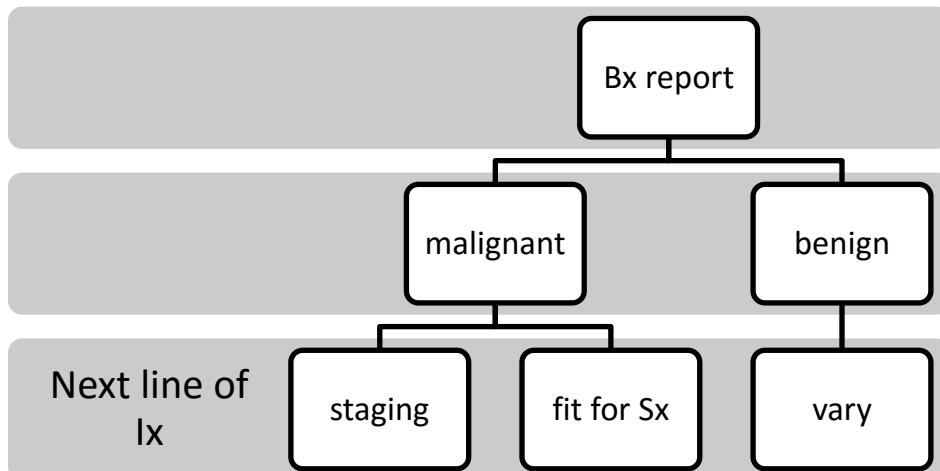
Anatomic landmarks in lower GI endoscopy

- ❖ Dentate line at anorectal junction
- ❖ voluminous organ with upper, middle and lower shelves, upper shelf taken
- ❖ Rectosigmoid junction – cephalic end of rectum with an acute bend in large bowel resulting in loss of view of the lumen
- ❖ Sigmoid colon – concentric folds within lumen, Variable length
- ❖ Descending colon – sigmoid-descending colon demarcation not clear cut. It's a straight tube allowing full view of its lumen unlike in sigmoid colon
- ❖ Splenic flexure – acute bend at the proximal descending colon
Transilluminated view of blue-black spleen
- ❖ Proximal to splenic flexure – triangular folds



General IX

1. FBC – Hb - ↓ - due to rectal bleeding
WBCC - ↑ - IBD, ischemic colitis
Platelets - ↓ - contribute to bleeding Bx report will take around 2weeks



rectal tumours - Malignant <ul style="list-style-type: none"> • primary <ul style="list-style-type: none"> • AdenoCA 90% • colloid 9% • squamous CA of lower anal canal • melanoma • carcinoid tumour • lymphoma • secondary <ul style="list-style-type: none"> • prostate CA • uterus • pelvic peritoneal deposits 	rectal tumours - Benign <ul style="list-style-type: none"> • Adenoma <ul style="list-style-type: none"> • villous adenoma <10% • Papilloma • Lipoma • Endometrioma 	rectal polyps <ul style="list-style-type: none"> • ALWAYS BENIGN <ul style="list-style-type: none"> • hyperplastic polyp • pseudopolyp (UC) • Juvenile polyp • MALIG. TRANSFORMATION <ul style="list-style-type: none"> • adenomatous polyp • vilous adenoma
colon -malignant <ul style="list-style-type: none"> • primary <ul style="list-style-type: none"> • adenocarcinoma • mucinous carcinoma • lymphoma • carcinoid tumour • GIST • secondary <ul style="list-style-type: none"> • inv. from stomach, bladder, uterus,ovary 	colon - benign <ul style="list-style-type: none"> • adenoma <ul style="list-style-type: none"> • tubular adenoma >90% • papilloma • lipoma • neurofibroma • haemangioma 	colon polyps <ul style="list-style-type: none"> • Benign <ul style="list-style-type: none"> • peutz jeghers polyp • Malig. transformtion <ul style="list-style-type: none"> • adenomatous polyp

Second line IX

1) Staging of disease –

- Contrast enhanced HRCT of thorax, abdomen and pelvis – high resolution (64 slices)
Detect 5mm size mets
- (Some – Abd-USS – liver mets., hydronephrosis, LN
CXR – lung mets., pleural effusion, osteolytic lesions on ribs, Mediastinal LN, co-morbidities)
- Trans rectal USS – local invasion of tumour } pick up 10mm size tumours
- Endoluminal USS (EUS)

2) Extent of colonic involvement – colonoscopy to look for synchronous lesions and polyps (in 5%)

- If unable to pass the colonoscope – CT colonogram
- Alternative to colonoscopy – Double contrast barium enema
 - Pathognomonic feature – apple core stricture

3) Fitness for Sx and anaesthesia

Haematological – FBC – Hb, WBCC, PCV, Plt

Biochemical - FBS, BU/SE, PT/INR (if Hx of heavy alcohol consumption), LFT

ECG → if changes present/ hx of reduced exercise tolerance → 2D echo

Spirometry – if any evidence of pulmonary compromise

4) Tumour markers – CEA (carcino-embryonic antigen) – as a baseline

Normal – 5ng/ml

CT COLONOGRAM (CTC)

- I° - 1) Examination of patients with incomplete colonoscopy – failure to intubate caecum
- 2) Detection of synchronous lesions in obstructing colon cancers
- 3) Superior localization of lesions before surgery
- 4) CI to colonoscopy – pt. who need anti-coagulants, medical CI for sedation, Pt. preference, Frail immobile Pt, Pt with severe co-morbidities
- 5) Post surgical evaluation of recurrence, metachronous disease etc. (not widely used)

Procedure –

CO₂ insufflation per rectum via flexible tube passed in 2inches up the rectum → CT scan → creates 2D image of the colon + other structures in abdomen and pelvis

Adv. – 1) Minimally invasive

2) Requires no sedation

3) Less time consuming than an endoscopic examination.

4) Allow evaluation of extra luminal and remote findings – distant mets, tumour invasion and L

Disadv. – 1) Requires full bowel preparation

2) Radiation exposure

3) CI - abdominal wall hernia with entrapment of colonic loops, abdominal and pelvic Sx
acute inflammatory conditions - acute diverticulitis, acute active stage of UC & CD
toxic megacolon

4) CT contraindicated conditions

Barium Studies

Double contrast – barium enema → air insufflated into rectum and colon

I° - To determine level of obstruction (single contrast .Eg: dilute/ water soluble)

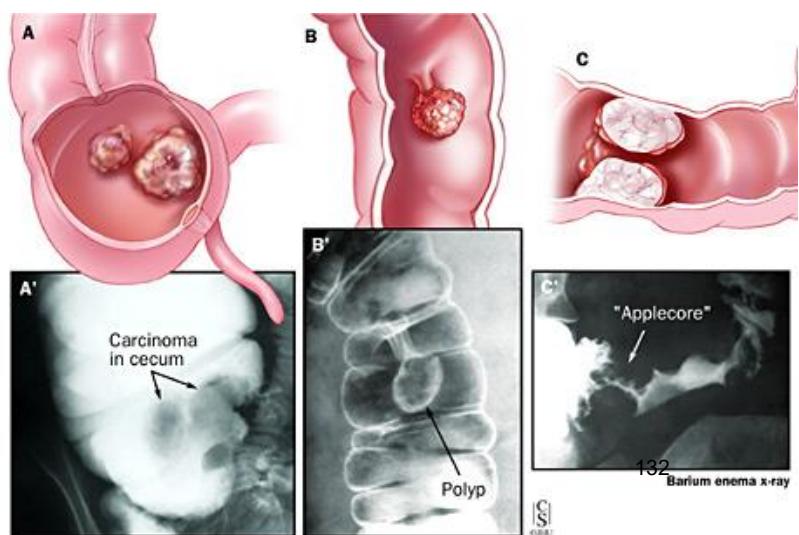
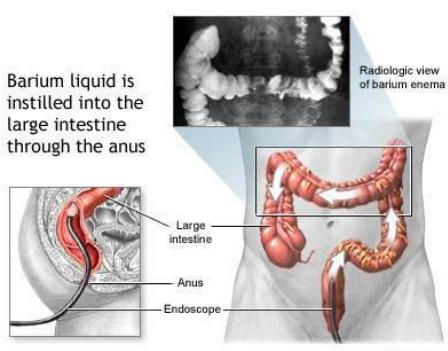
Carcinoma of the colon

Ulcerative colitis

Diverticular disease of the colon

Familial adenomatous polyposis

Assessment of anastomotic leak /
perforation (water soluble contrast)



Disadv. –

- 1) Incomplete assessment of the rectum and, therefore, done after endoscopic assessment
- 2) Limited to evaluation of large bowel obstruction only – usually by water soluble contrast
- 3) Undertaken by trained personnel, the risk of perforation is around 1 per 2000.

How would you manage this patient?

Further management depends on the;

- ◆ staging of the disease,
- ◆ grading of the tumour - histology
- ◆ site of the lesion

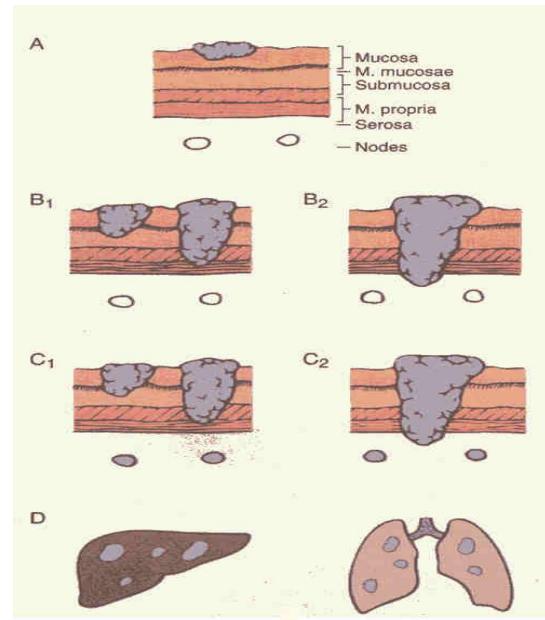
Staging of tumour – 1) (Cuthbert) Dukes classification (pathological)
2) TNM classification

Duke A – involving bowel wall but not pericolic fat/serosa

Duke B – infiltrating through bowel wall into pericolic fat & serosa

Duke C - LN

Stage	Prognosis	Mx
Duke A	90% 5yr survival	Sx
Duke B/ T _{3/4}	60-70%	Pre-op chemoradiation
Duke C/D	<50%	Post-op adjuvant chemorad.



- ❖ assess the fitness for surgery
- ❖ oncology referral needs to be done – opinion on neoadjuvant chemoradiation prior to Sx (in locally advanced /high grade tumours → to shrink the tumour)

❖ Surgery aimed at

1) Loco-regional control – removal of the local tumour + draining loco regional LN with adequate resection margin (based on the distribution of blood vessels and adequate resection margin)

Adequate resection margin – definition

- ◆ Colon cancers – require 5cm resection margin on either side of the lesion
- ◆ Rectal carcinoma – 2cm (previously 5cm)

2) Palliative – relieve obstruction/ stenting

❖ Plan the surgery

If lesion is resectable following surgeries are carried out

Site of lesion	Surgical procedure
Carcinoma of the rectum	<ul style="list-style-type: none"> ❖ Sphincter sparing – anterior resection I° - tumour in upper rectum ❖ Sphincter loosing – APR + permanent colostomy Above combined with Total mesorectal excision
Carcinoma of the sigmoid colon	Sigmoid colectomy
Carcinoma of splenic flexure and desc. colon	Left hemicolectomy
Carcinoma of transverse colon	Transverse colectomy/ extended right hemicolectomy
Carcinoma of hepatic flexure	Extended right hemicolectomy
CA of caecum and asc. colon	Right hemicolectomy

- ❖ Admit the patient day prior to the surgery
- ❖ Preparing the patient for surgery
 - ◆ Counsel the patient regarding the disease condition
 - ◆ Informed written consent
 - Type of Sx – major Sx (what parts are going to be removed)
 - Type of anaesthesia - GA
 - Risks
 - Post-op outcomes – Eg:- colostomy
 - Liberty bestowed to the surgeon to make decisions during surgery
 - ◆ Counsel regarding the possible placement of a stoma and educate on stoma care
 - ◆ ensure stoma site is correctly marked
 - ◆ Anaesthetic referral and complete pre-op assessment
 - ◆ Optimize the patient
 - Correct Hb → 9g/dL
 - ◆ Cross match and reserve blood –

Surgery	Amount to be reserved
APR	4pints
Anterior resection	2 pints
Hartmann's procedure	2 pints
Left hemicolectomy	2 pints
Right hemicolectomy	2 pints

- ◆ Prepare theatre list
- ◆ Reserve ICU bed
- ◆ Full bowel preparation with polyethylene glycol day before surgery

12 noon – low fibre diet lunch
 1-3pm → kleen prep, 1 litre
 4-6pm → kleen prep 2nd sachet
 7-9pm → 3rd sachet
 Drink clear fluids until fasting

- ◆ Premedication –
 - preop-AB → co-amoxiclav 1.2g + metronidazole 500mg – 2 doses at home
 1 dose intra-op
 - anti- reflux Rx – IV Ranitidine 50mg bd + metoclopramide
 - anxiolytic – o. diazepam 10mg
- ◆ Kept fasting from 12midnight on previous day
- ◆ Shave abdomen and perineal region
- ◆ NG decompression
- ◆ Catheterization
- ◆ Hartmann solution at 10pm on the night before Sx – at 80ml/hr overnight
- ◆ Shower with hibiscrub before Sx
- ◆ Anti – DVT stockings before Sx

- ❖ Post –operative management
- ❖ Keep NBM until BS appear
- ❖ Monitor – PR, RR, BP closely in the 1st 6hrs
 - 1/4hly → 2hr
 - 1/2hly → 2hrs
 - 1hly → 2hrs
 - IP/OP chart, quarterly hour temperature
- ❖ Hydrate – IV fluids (0.9% Normal saline and 5% dextrose alternatively) at 80cc/hr
- ❖ Analgesia – epidural if one sited
 - s/c morphine 0.1mg/kg every 6-8hrs / IM pethidine 100mg 4hly
- ❖ Drain – observe the amount drained each day and note the trend (declining/ ascending)
 - Colour of drain fluid – blood/serous/ serosanguinous
 - Drain removal – with gradual declining trend with ≤25ml left (not necessary until dry)
- ❖ IV AB – cefuroxime + metronidazole – continued (since GI Sx → commonly cause lung infec.)
 - Co- amoxiclav 1.2g + metronidazole 500mg tds
- ❖ Stoma care
- ❖ Chest physiotherapy
- ❖ DVT prophylaxis – in all
 - 1.5mg/kg LMWH subcutaneously x 5days
 - Early mobilization – until then anti – DVT stockings

Monitoring

Hydration

Analgesia

AB

Complication Mx

Feeding

Wound care

Stoma care

Observe for complications

Anaesthetic → hypotension – venodilatation due to drugs, inadequate fluid input, bleeding

Hypertension – pain, pre-existing HTN

Bradycardia

Desaturation – due to hypoventilation, residual NM block/ sedative action

Electrolyte abnormalities – K⁺, Ca, Mg – K⁺ maintainance 40μmol/24hrs (or 60mg daily)

In order to raise K⁺ by 1mmol add 10μmol

Maximum 20μmol in 1pint N/S

Immediate – surgical → 1) damage to local structures – ureter, bladder
2) Primary haemorrhage

Early –surgical → infection – anastomotic site, intra-abdominal abscess

Reactionary/secondary bleeding

Anastomotic leakage

Sepsis

Stoma problems – high output, sloughing, necrosis, retraction

Paralytic ileus

Late – surgical → short bowel syndrome

Sexual dysfunction

Small bowel obstruction

Feeding – D1 – IV fluids (80cc/hr)

D2 – start oral sips (20cc/hr)

D3 – gradually increase oral intake (30-50cc/hr)

D4 – complete liquid diet, omit IV fluids

D5 – semisolids (yoghurt, jelly, biscuit)

D6 – high protein diet (2 eggs, 2yoghurt, 2 oranges, fish 125g, 1 jelly pkt)

Wound care – inspect suture site (soaked bloody) → daily dressing if soaked

Suture removal – 10-14days

Lung care – steam inhalation

Incentive spirometer – 15x/1hr

❖ Post-op – adjuvant chemoradiation

- I^o - LN disease + mets
- Usually post-op since LN involvement cannot be predicted pre-op
- Minimum of 6 cycles
- Colon CA – **FolFOX** → Folinic acid + 5-flurouracil + oxanic acid
- For metasatatic disease → **FolFIri** → Folinic acid + 5-flurouracil + Irinotecan
- IV Rx
- Oral chemo – capecitabine – taken at home, 3tablets per dose x tds. , 3000/- per tablet
- 2nd line Rx – non responders
 - Biological agents – VEGF, Anti epith GF
- Radiation – rectum
 - Shrinks the tumour in 2 ways – 1) macro – tumour apoptosis
 - 2) micro – reduce size of the tumour – Downsizing
 - Reduce the stage of tumour - Downstaging

Requirements for good wound healing

Hb >10g/dL

Alb > 3.5g/dL - if low Alb replacement

Albumin 50mg bd, CPP

Ix – Hb, WBCC (FBC), SE (K+) – daily

S. Albumin - EOD

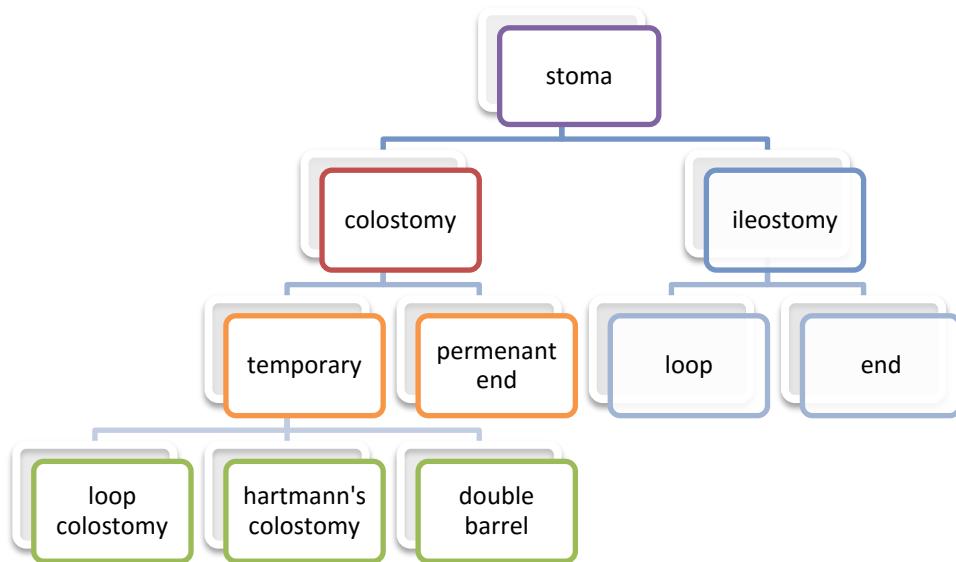
Follow up

- ✓ Emphasize that 75% of recurrences occur during the 1st 3years of surgery
- ✓ Therefore need comprehensive follow up
- ✓ Tumour recurrence may be identified by
 - Clinically
 - Biochemically (CEA)
 - Radiologically
- ✓ Be watchful regarding the following symptoms that may develop in tumour recurrence → consult
 - Rectal bleeding
 - Unintentional wt. loss
 - Abdominal pain
 - Unexplained diarrhoea/ constipation
 - Fatigue due to anaemia
- ✓ During clinic visits look for evidence of recurrence
 - General well being
 - Virchow's LN
 - DRE – growth in rectum
- ✓ CEA – should come down to normal after Sx
 - Advise to come to clinic with CEA report → 3monthly – for the 1st 3yrs
 - 6monthly – from 3rd-5th year
 - Annually – from 5th -10th year
- ✓ 1year after surgery best to undergo colonoscopy as a surveillance to look for tumour recurrence

- ✓ If clinical picture + CEA level is suspicious investigate
 - CXR
 - USS
 - Contrast CT abdomen
 - CT PET

Stoma

Types



Comparison between colostomy and ileostomy

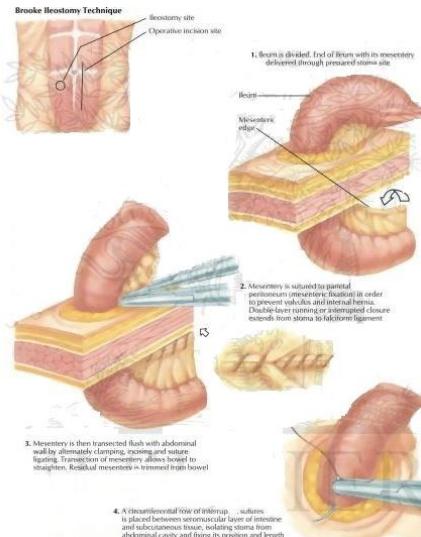
Colostomy	Ileostomy
Colon	Terminal part of the small intestine
Flushed with skin (leveled with the skin)	Spout out (since digestive enzymes → excoriation)
Generally in the left	In the right
Content - feculent , solid	watery
Closed appliance w/o fastening – aft. recovery	Cut the wafer smaller than the bowel loop – tight Fastening at the end – can be emptied at the bottom

Ileostomy

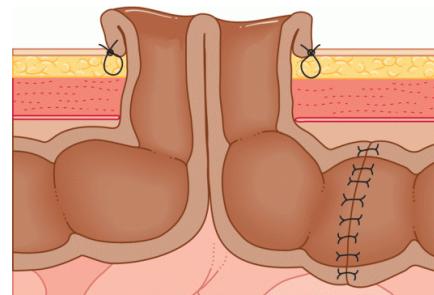
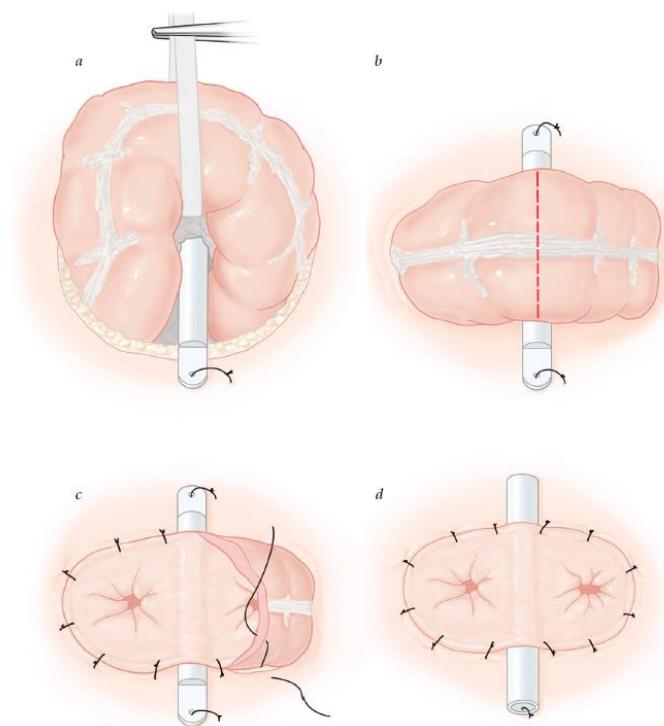
Procedure

- Loop – knuckle of terminal ileum brought out
Similar to loop colostomy
- End -

I° - divert faeces away from anastomotic site
Eg: low anterior resection



Loop colostomy



Procedure

- Colon brought to the surface
- Anti-mesenteric border opened
- Rod/similar device used to stop the open bowel falling back inside
- Rod removed after 7days

I° - temporary diversion of faeces

Adv. – simple to reverse

Replaced by loop ileostomy – since better blood supply facilitating closure, no odour,

Double barreled colostomy

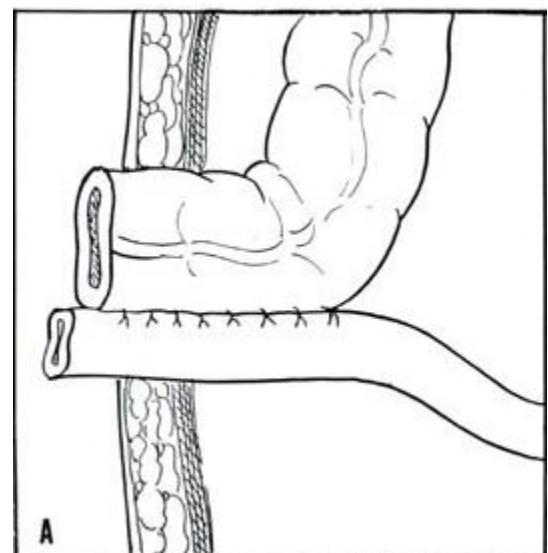
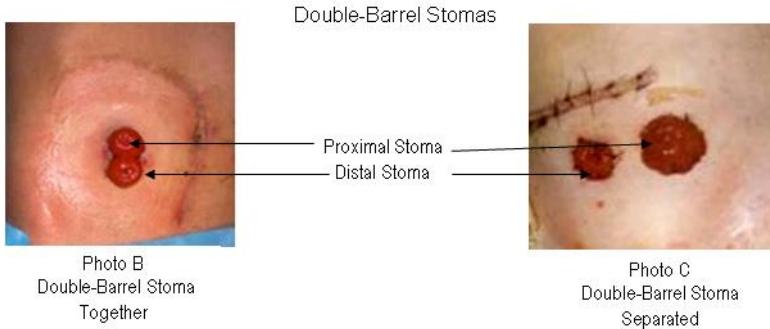
Procedure

- Proximal and distal resected colon brought out adjacent to each other
- Similar to loop colostomy but the intervening colon removed

I° - sigmoid volvulus (since sufficient distal colon present)

Proximal stoma – colostomy

distal - Mucus fistula



Permanent end colostomy

Procedure

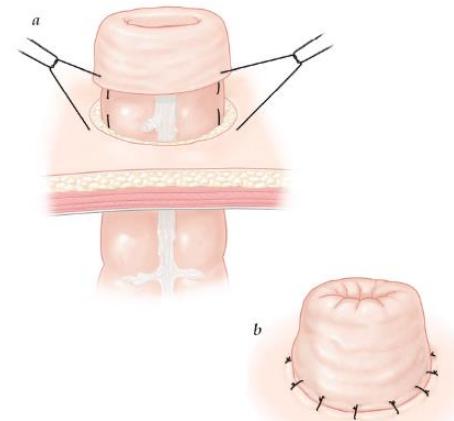
- Colon divided → proximal colon brought out

I° - APR

Primary anastomosis undesirable – gross faecal contamination
 Eg:- perforated diverticular disease (distal bowel maybe brought out as a mucous fistula)

Complications

Early	Late
Ischemia	Prolapse
Bleeding	Peristomal disease
Retraction	Bowel obstruction
Skin excoriation	

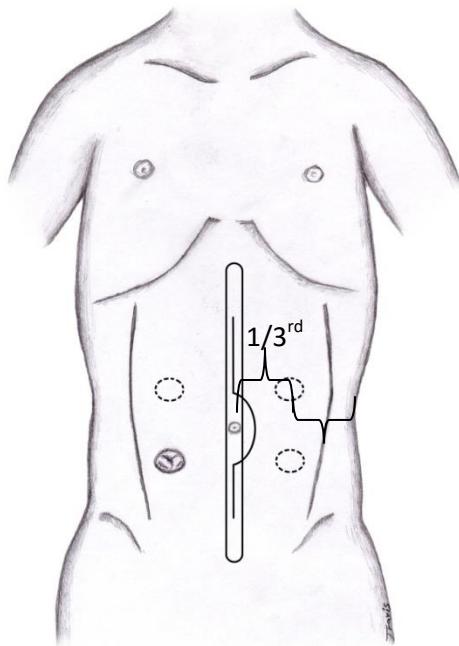


Colostomy care

- Psychological support
 - It's like wearing spectacles.
 - Will not leak, odour sealed
 - Introduce to other colostomy patients Eg: via stoma club
 - Is not a contraindication for sexual activity
- Changing the bag
 - Colostomy – every day, if using 2 piece system base plate can be changed less often
 Empty the bag once it is 1/3rd full. Wipe with tissue/gauze before clipping back.
 - Ileostomy – EOD for 1st 8 weeks
 >8 weeks → every 3-4 days provided no skin problems
 Never >5days
 - Measure the stoma weekly in the 1st 8 weeks – since change in size & shape with ↓ swelling
 - Refer ostomy booklet (removal and attachment)
- Stoma wash out enables complete emptying → can go without colostomy bag
- Diet
 - AVOID – **high/ moderate fibre diet** – esp. ileostomy, in colostomy irrigation
 Fibre → increase stool bulk → intestinal obstruction
 Odour producing foods – cabbage, radish, garlic, cucumber, knokol
 Tea and coffee
 High fat products
 Carbonated drinks, chewing gum, smoking → excess gas in stoma bag
 Large meals before sleep
 - Chew food well
 - Adequate amounts of water – 1 1/2 – 2L daily

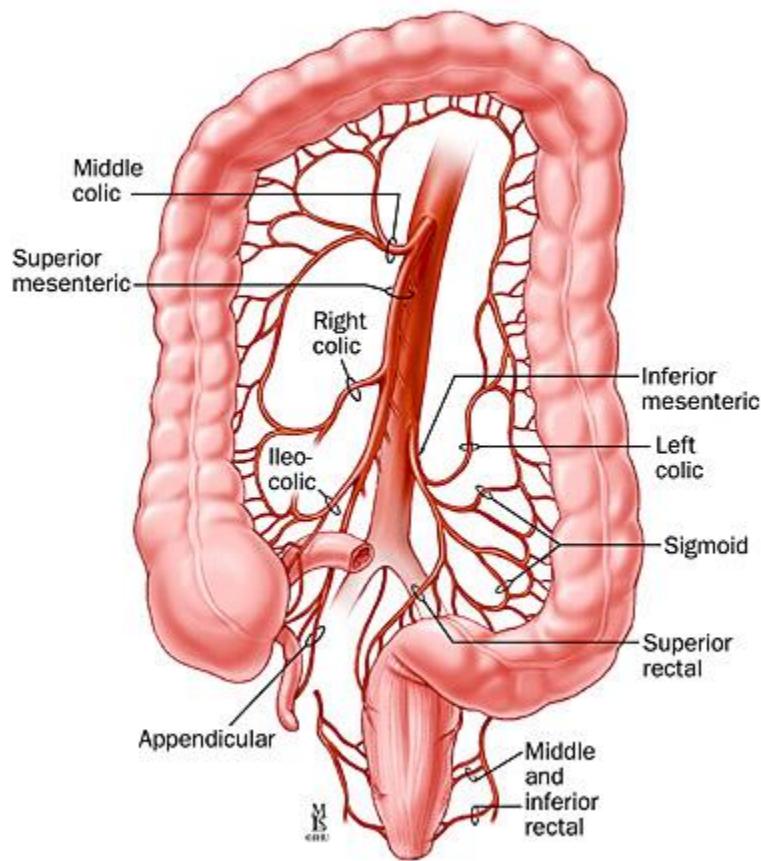
Marking the colostomy site

- ◎ Done by stoma therapist/surgeon
- ◎ Using indelible ink
- ◎ Stoma site marked within the rectus muscle – prevent stoma hernia and prolapse
- ◎ Pt. should be able to see the stoma and it should be easily accessible
- ◎ Away from bony points
- ◎ Fill 1/3rd of a colostomy bag with water and stick it at the proposed site → check the interference when walking, seated, and bending and in supine position
- ◎ Stoma site marked clearly when in the standing position



Site

1/3rd the distance from umbilicus to iliac crest



Arterial supply of the colon

Determines colorectal & lorengeal LN resection

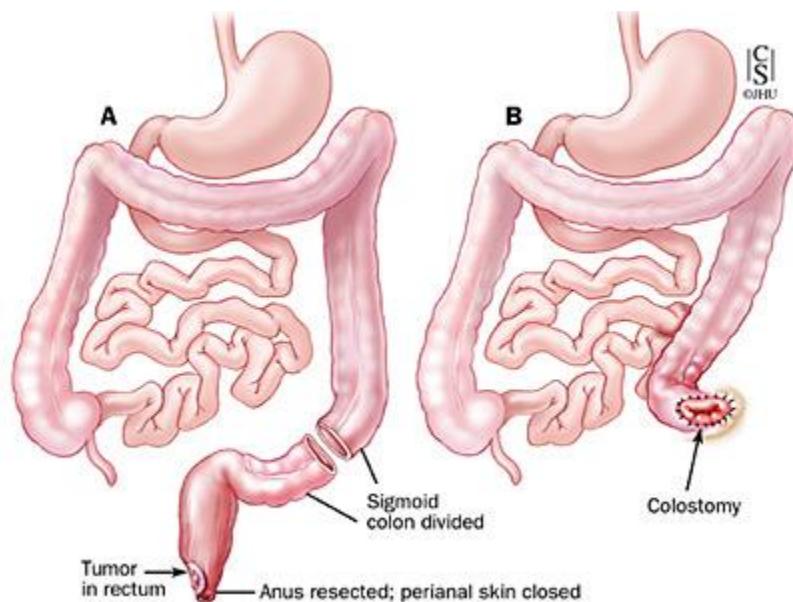
APR – Inferior mesenteric artery

Transverse colectomy – middle colic A.

Left hemicolectomy – left colic A

Right hemicolectomy – sup. mesenteric A.

Abdomino-perineal resection (APR) with permanent colostomy



I^o - lower rectal tumours (within 2cm of the anal verge)

- *Unable to spare anal sphincter → require permanent end colostomy*

Procedure –

- ❖ *Anus closed with a purse-string suture*
- ❖ *Rectum mobilized from above (via abdominal incision)*
- ❖ *Lower rectum and anus excised via perineal incision*
- ❖ *Rectum removed*
- ❖ *Proximal resection brought out as permanent end colostomy*
- ❖ *Subcutaneous fat and skin closed*
- ❖ *Drain placed in pelvis*

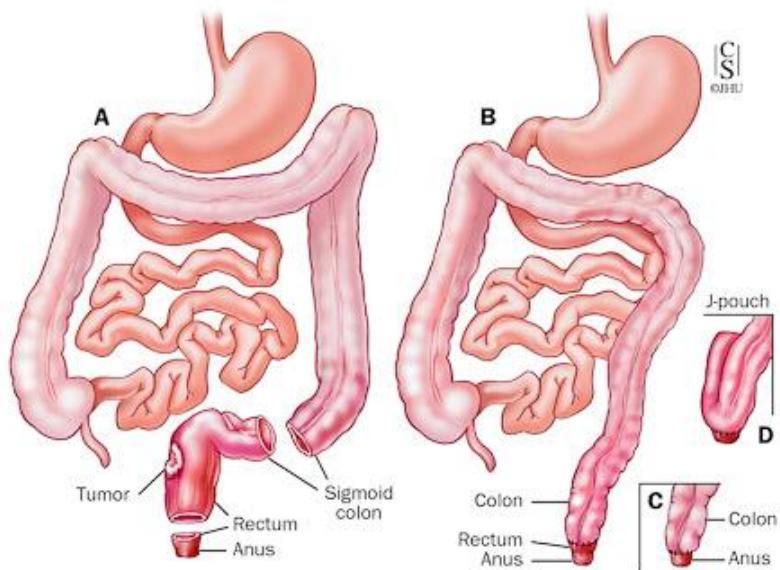
Duration – 3hrs

Disadv. – 1) require permanent colostomy
2) Duration of hospital stay – 14days
3) Long term follow up required

Post –op

- 1) High risk of haemorrhage in the first 24hrs → observe the drain

Anterior resection



I^o - upper rectal tumour (>2cm from anal verge)

Procedure

- Growth resected with a 2cm resection margin from anus – via the anterior abdominal wall
- Anastomosis between rectal stump and divided end of the mobilized left colon
- Anastomosis via mechanical stapling guns

In low anterior resections – colon pouch created by folding the distal left colon into a J configuration → create a common lumen (replacing the rectum's lost reservoir function)

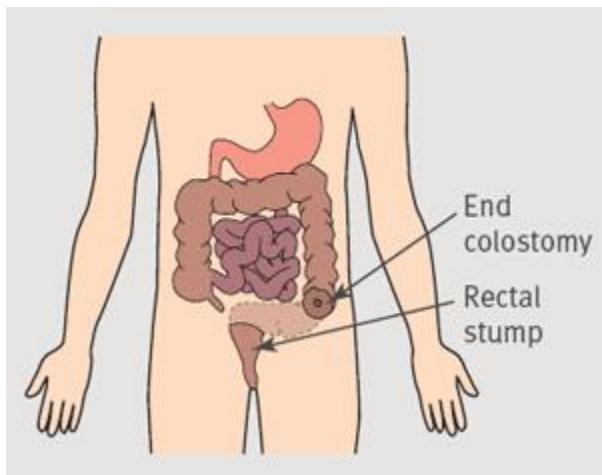
Complication – poor blood supply at lower rectum → delayed anastomotic healing → leakage

Therefore defunctioning stoma/ loop ileostomy created → remove effluent until anastomosis heals

Ileostomy reversal → done after 3/12

Contrast enema x-ray done to see anastomotic healing

Hartmann's procedure



I^o - palliative Sx – 1) unresectable rectal tumour
2) pt. unfit for a further major resection

Emergency Sx – for excision of obstructing/perforated colonic mass

Procedure

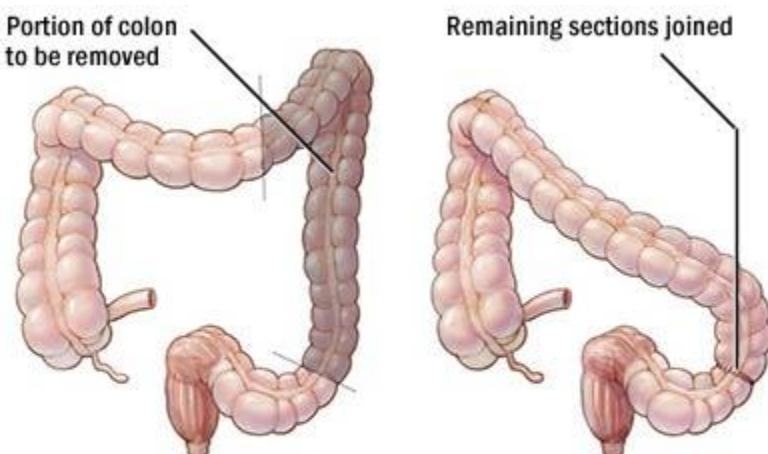
- ❖ Resection of the rectosigmoid colon
- ❖ Lower end of rectum closed and left In situ – rectal stump
- ❖ Formation of an end colostomy
- ❖ No anastomosis created

Duration – 2-3hrs

Colostomy maybe reversed in some following recovery – reversal of Hartmann Sx

Hospital stay – 10days

Left hemicolecotomy (anterior resection)

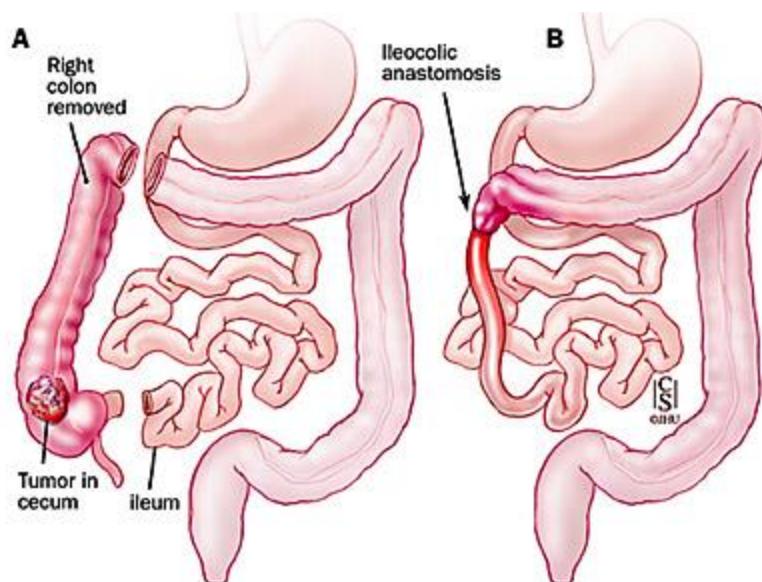


Procedure

- ❖ Descending colon +/- sigmoid colon removed abdominally
- ❖ End to end anastomosis

Duration – 1-2hrs

Right hemicolecotomy

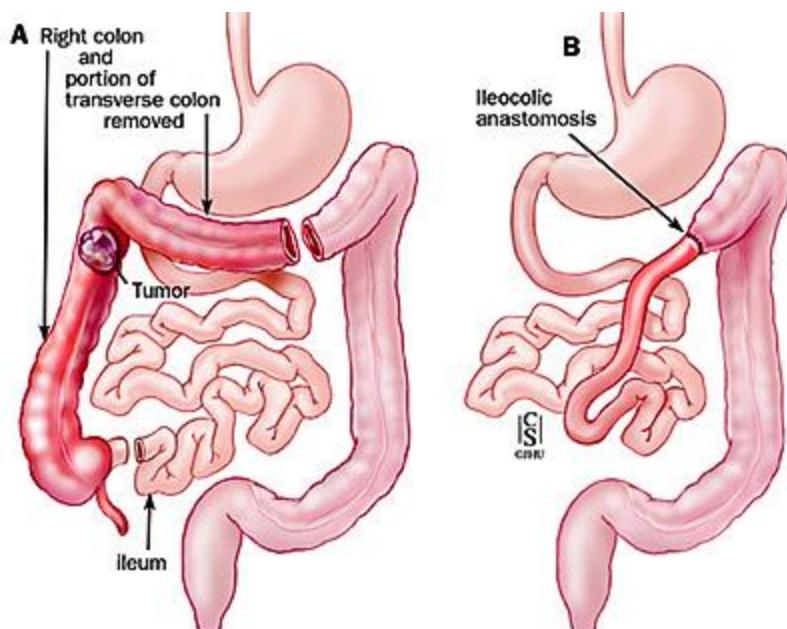


Procedure

- ❖ Terminal ileum, right colon and hepatic flexure mobilized abdominally
- ❖ Proximal resection margin is in the terminal ileum
 - Since the caecum is the most dilated part of the colon is not good for anastomosis
 - Small bowel-large bowel anastomosis is effective than large bowel- large bowel
- ❖ Ileocolic anastomosis

Duration – 1-2hrs

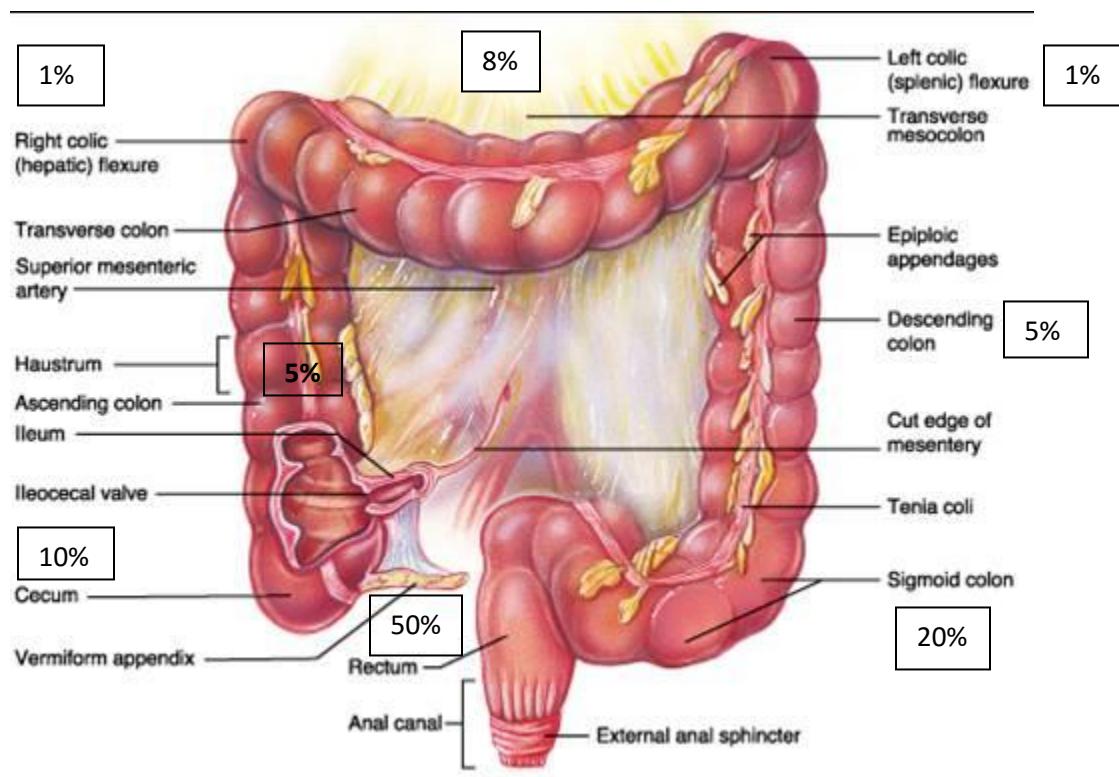
Extended right hemicolecotomy



Colorectal carcinoma

- ❖ Incidence – fairly common in SL; commoner in the west
However in Asia <40yr olds – 15% whilst in the west – 6%
Commoner in **females**

- ❖ Distribution



- ❖ Differences between right and left colonic CA?

Right colonic carcinoma	Left colonic carcinoma
Late presentation – symptomless	Early presentation – overt symptoms
Most involving the duodenum at time of Sx	
Presents with anaemia, LOW, asthenia, RIF mass	Bleeding PR
Histology – polypoidal/cauliflower like	Circumferential growths
Intestinal obstruction rare – polypoidal non obstructive growths + watery stools	Common – since circumferential constricting growths and solid stools*

- ❖ Why altered bowel habits in left colonic CA?
 - Constipation due to above*
 - Diarrhoea – stool putrefaction by abundant gut flora → watery stools

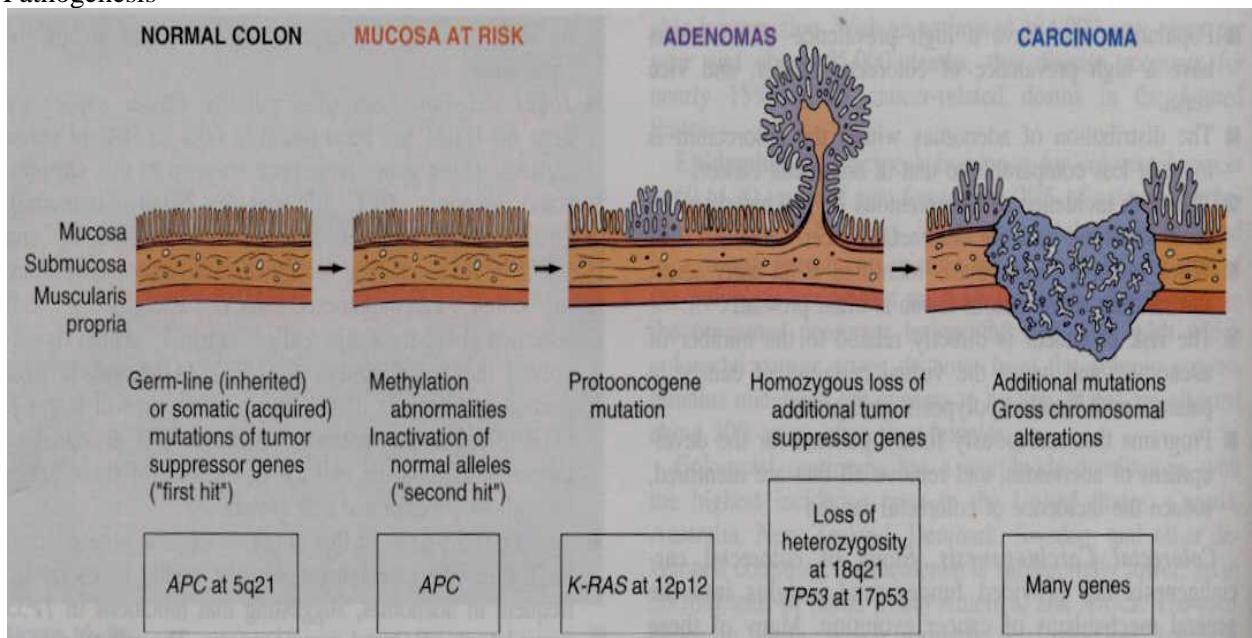
- ❖ Why spurious diarrhoea?
 - Adenocarcinoma → abundant mucin and K⁺ secretion – mucoid diarrhoea and ↓K⁺

❖ Aetiology

- Hereditary →

- 1) FAP (familial adenomatous polyposis) - <1% of colorectal CA
 - Autosomal dominant
 - Defect in APC gene (adenomatous polyposis coli) in the long arm of **chromosome 5**
 - 20% sporadic – new mutation
 - Clinical definition – presence of >100 colorectal adenomas
 - Polyps elsewhere → duodenal/ ampullary carcinoma
 - ♂=♀
 - Develop millions of polyps by 20yrs of age – 100% incidence
 - Therefore if suspecting FAP colonoscopy done at 20yrs
 - By 30yrs → 100% incidence of CA

Pathogenesis



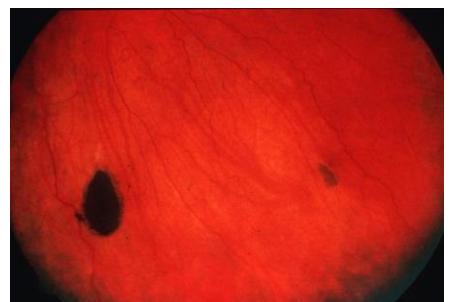
Mutation in APC gene (hit 1) → loss/deletion of the normal alleles (hit 2) → microadenoma → adenoma → mutations in KRAS & p53 genes → carcinoma

Variants of FAP – **Gardner's syndrome**

- Peripheral stigmata – multiple epidermoid cysts, Lipomata, osteoma of skull and mandible, desmoids tumours of the abdomen

Turcot's syndrome

- Increased risk of brain tumours
- Medulloblastoma – associated with FAP
- Glioblastoma – associated with HNPCC



Screening for FAP

1) Colonoscopy – starting at 20years (10-12 years) → annual
If no polyps at 20yrs → 5yearly until 50years of age

2) Slit lamp Ex/funduscopy
- CHRPE (congenital hypertrophy of retinal pigment epithelium)
- used for surveillance in children (non invasive)
- 70% of CHRPE → have polyps

Management

1) Prophylactic removal of colon + rectum

- ◆ Panproctocolectomy and formation of an ileoanal pouch
- Restorative proctocolectomy – sparing of anal canal and valve
- Complications – damage to pelvic n. → sexual dysfunction
Pouchitis
- ◆ If only few polyps in the lower rectum → colectomy + ileorectal anastomosis
- Adv. – no pelvic n. damage, no need of ileostomy
- Disadv. – 6 monthly flexi sigmoidoscopy and diathermy of rectal polyps
- ◆ Screen family members
- ◆ Complete polyposis register- identify and regular surveillance

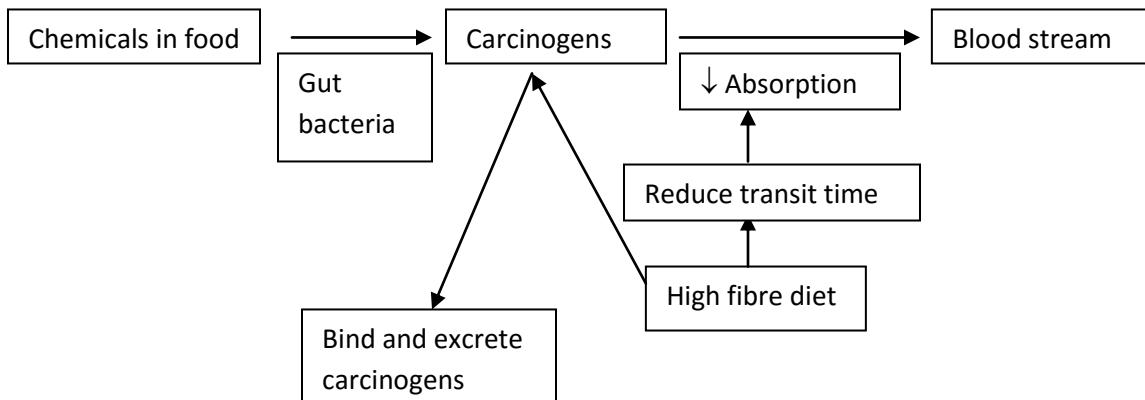
2) HNPCC (Lynch syndrome)

- 5% of colorectal Ca
- starts straight away as cancer
- Autosomal dominant
- Mutation in DNA mismatch repair gene
- increase risk of colorectal + endometrial, ovarian, stomach and SI CA
- affects mainly the right colon
- diagnosed by ‘Amsterdam criteria’
 1. ≥ 3 family members affected with one being a first degree relative of the other 2
 2. 2 successive generation affected
 3. Diagnosed before age 50
 4. FAP excluded
- Environmental

Lynch type I – gastric and colon CA

Lynch type II – reproductive CA

1) Diet →
◆ Low fibre diet



- ◆ High protein diet – meat → nitrosoamine (carcinogenic)
- ◆ High fat diet → long chain FA (carcinogenic)
- IBD
 - ◆ Ulcerative colitis

Cancer risk depends on –

 - 1) age of onset – younger
 - 2) Degree of disease – major attack
 - 3) Chronicity – frequent relapses
 - 4) Extent of disease – pancolitis

Crohn's disease also ↑ risk
- Other → ↑ alcohol intake, smoking, obesity

Pathology

- 95% of colorectal CA arise as a polyp
- Commonest type adenocarcinomas



Mucosa

- 1) adenocarcinoma – epith.
- 2) mucinous CA – goblet cells
- 3) carcinoid tumour – neuroendocrine cells

Submucosa : Fat - lipoma

Muscularis propria GIST – CT holding the muscle

Serosa : Illry deposits

Extra-intestinal - liposarcoma

Adenomatous polyps – 5-10yrs → adenoCA

Table 65.3 Classification of polyps of the large intestine

Class	Varieties
Inflammatory	Inflammatory polyps
Metaplastic	Metaplastic or hyperplastic polyps
Harmartomatous	Peutz-Jeghers polyp Juvenile polyp
Neoplastic	Adenoma <ul style="list-style-type: none"> – Tubular – Tubulovillous – Villous Adenocarcinoma Carcinoid tumour

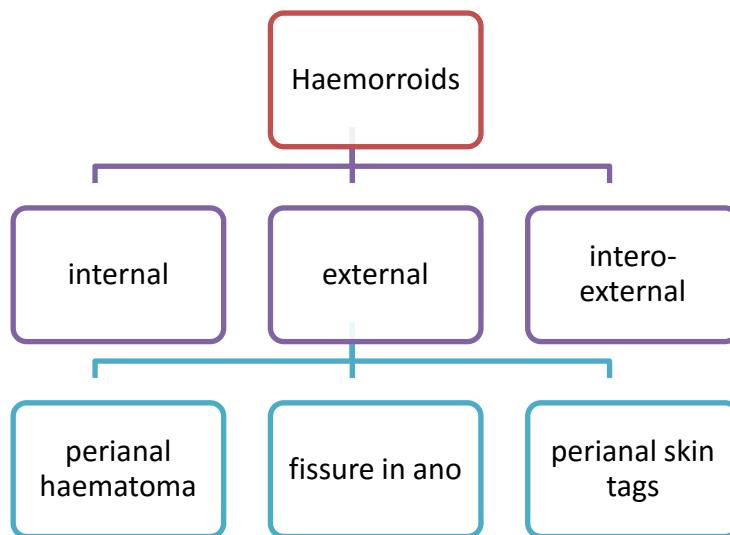
Colonic polyps
Surface polypoidal elevation

Features ↑ risk of malignancy

- 1) Sessile polyps (no stalk)
- 2) >2cm
- 3) Villous adenoma > tubular
- 4) no. of polyps

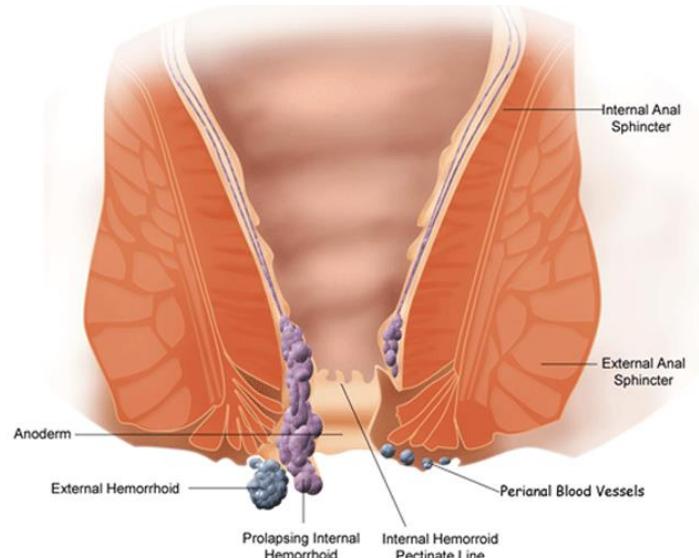
Haemorrhoids Mx

❖ Types



❖ Pathogenesis

- Straining at stools + traumatized by the passage of hard stools
 - ↓
 - Block VR → congestion of anal cushions
 - ↓
 - Dilatation of AV plexus
 - ↓
 - Stretching of suspensory muscles
 - ↓
 - Prolapse of rectal mucosa
 - Engorged anal mucosa – trauma → bleeding
 - Prolapse → soiling, mucus discharge, pruritus



❖ Grading of haemorrhoids

Stage I	Stage II	Stage III	Stage IV
No protrusion of haemorrhoids, yet.	Protruding haemorrhoids that spontaneously reduce!	Protruding haemorrhoids, possible to push back in manually!	Protruding haemorrhoids that can't be pushed back in manually anymore !

❖ Management

GENERAL – aim → avoid straining at stool, pass firm soft motion daily

- 1) High residue diet
- 2) Adequate fluid intake
- 3) Bulk laxative

SPECIFIC

Outpatient → first and second degree haemorrhoids

Sclerotherapy - I° (70% success rate): phenol 5% in arachis or almond oil (phenol sterilizes the oil. Oil is the main sclerosant)

Rubber band ligation - II° (90% success rate)

Rubber band ligation

- *Barrens band ligator*
- *Bands applied above the dentate line – otherwise painful*

Inpatient

Dilatation and banding

Haemorrhoidectomy - III°/IV° → closed/ stapled technique (less pain, op. time and hosp. stay)

Complications	Management
Post-op bleeding – reactionary → night of Sx Secondary → day 7/8 post op	Check haemodynamic stability Blood Tx If continuing → Sx 1) warm N/S rectal wash out 2) bleeding points – diathermy/ligate 3) if oozing – gauze packing around a wide bore rubber tube (removed after 48hrs)
Post –op pain → acute urine retention	Reduced – o. metronidazole, botulinum toxin inj. Analgesia
Perianal haematoma (thrombosed ext. pile)	Acute pain with straining + lump at anus Acute – evacuate haematoma under LA Discharging – hot baths Opioid analgesics Local cold compressors Reassure – spont. fibrosis
Anal stenosis	

IBD

	Ulcerative colitis	Crohn disease
Pathology	Only colon , limited ileal disease	Any part from mouth - anus
	Continuous involvement of the affected segment	Usually skip lesions
	Affects mucosa only	Transmural
	Widespread irregular superficial ulceration +/- pseudopolyps	Fissured ulceration Cobblestone appearance
	No granuloma formation	Granuloma characteristic
Complications	Rare	Stricture and fistula common
	Rare	Anal and perianal dis. common
	Toxic megacolon, perforation and haemorrhage can occur	Unusual

	High malignant potential	Low
	Intestinal obstruct. rare	Common (incomplete obstruct.)

Ulcerative colitis –Mx

Based on severity

Grade of severity	Criteria	Mx
Mild	Blood and mucus diarrhoea < 4/day No systemic signs	<ul style="list-style-type: none"> ❖ Setting – outpatient ❖ Stop acute attack – short course o. prednisolone steroids PR ❖ Assess compliance to sulfasalazine – adjust dose
Moderate	Blod and mucus diarrhoea >4/day No systemic signs	<ul style="list-style-type: none"> ❖ Setting – admit if dehydrated ❖ As above – o. prednisolone Steroid enemas 2x/day
Severe	Blood and mucus diarrhoea >4/day Systemic signs (≥ 1) <ul style="list-style-type: none"> ○ Fever $>37.5^{\circ}\text{C}$ ○ Tachycardia >90bpm ○ Hypoalbuminemia <30g/dL ○ Wt loss >3kg 	MEDICAL EMERGENCY <ul style="list-style-type: none"> ❖ Setting – admit ❖ Monitor vital signs – pulse, BP, temp ❖ Monitor weight – at admission 2/wk ❖ Monitor abdominal girth ❖ Daily – plain abd. X ray Stool chart IP/OP, SE ❖ Correct anaemia ❖ Nutrition – high calorie Severe - TPN ❖ IV hydrocortisone 100-200mg 4x/day ❖ Prednisolone rectal infusion
Toxic megacolon	AXR <ul style="list-style-type: none"> ○ Dilatation of transverse colon $>5.5\text{cm}$ ○ Presence of mucosal islands/intramural gas Sudden \uparrow in PR, temp – colonic perforation	Assessed by abd. Girth and AXR Sx – based on extent of involvement Rectum involved, <ul style="list-style-type: none"> ❖ Pt. unstable – total colectomy ❖ Pt. stable – total proctocolectomy (time consuming) + ileostomy Restorative proctocolectomy

Surveillance

UC – well controlled – surveillance colonoscopy every 5yrs

Poorly controlled – annual colonoscopy (risk of R/ colonic CA-poorly differentiated)

Sclerotherapy for haemorrhoids

Requirements

- *proctoscope,*
- *gloves,*
- *lignocaine gel,*
- *5% phenol in almond oil, syringe / Gabrielle needle, spinal*
(18G) or venflon needle.

Positioning

The patient is positioned as for a proctoscopic examination,

Technique

The haemorrhoids are visualized using the proctoscope, and about 2-3 ml of 5% phenol in almond oil is injected above each of the haemorrhoids.

May require a series of 2 to 3 injections at 6 weekly intervals.

The injection should be seen to raise a pale swelling which spreads immediately deep to the mucosa.

Complications

- **Too superficial** A white wheal indicates too superficial an injection and the risk of mucosal sloughing
- **Too deep** an injection may have serious consequences; in men, chemical prostatitis and impotence are well recognized rare.
- Complications and anovaginal fistulas have been reported in women.
- **Bradycardia.** vagally induced bradycardia. Therefore, leave the patient on the couch for sometime after injection and ensure that the pulse rate is within the acceptable physiological range.
Treatment for bradycardia may have to include intravenous atropine and / or oxygen therapy.
- **Allergic reaction** Rarely, phenol may induce an allergic reaction, which may include anaphylaxis. It is essential to have an emergency tray on-hand to deal with anaphylaxis.

Peripheral vascular disease

General

Age – majority >50, (young smokers – buerger's disease)

Sex – male > female

P/C

Pain over the calf while walking

DD

- Intermittent claudication
- Neurogenic „
- Venous „
- Musculoskeletal pain

H/P/C

Describe the pain in detail

Onset & progression

- When did the pt 1st begin to experience pain
- Describe the onset of pain
- Pain starts with the 1st step?
- How far the pt has to walk before he experience the pain → Claudication distance

Site of the pain (localize the anatomical site of the lesion)

- B/L buttocks & thighs – Aorto- iliac disease (ask for associated impotence)
- Thigh & calf – iliac disease
- Calf – femoro- popliteal disease
- Calf & foot – distal obstruction

Character of the pain & reproducibility

Cramp like pain felt in the muscles when walking make the patient stop.

Pts response to pain

- What patient does when he gets pain
- Does the patient rest & for how long
- Describe what happens if the pt continues to walk regardless of the pain

Aggravating & relieving factors

- Distance that pt able to walk depend on
- Walking uphill
- Speed of walking
- Changes in general health – anaemia, heart failure

Progression of symptoms over the time – collateral formation

Complications of the disease

Rest pain

- Severe pain, lasting for more than 2wks
- Develop in the fore foot, ↑ at night
- Relieved by keeping the leg down
- Pain doesn't respond to simple analgesics

PHx of ulcers, digital necrosis, amputations

Ulceration & gangrene → severe arterial insufficiency

- Painful erosions b/w toes
- Non healing ulcers over the dorsum of foot, on the shin, specially around the malleoli

Acute ischemic limb → paralyzed & insensate (not seen in chronic limb ischemia)

Compartment syndrome

- Difficulty in walking
- In limbs that have been subject to sudden ischaemia followed by revascularisation, oedema is likely.
- Muscles swell within fixed fascial compartments and this can itself be a cause of ischaemia
 - Local muscle necrosis and nerve damage due to pressure,
 - Distal effects such as renal failure secondary to the liberation of myocyte breakdown products.

The treatment is

- Urgent fasciotomy to release the compression

The usual site at which such surgery is necessary is the calf (especially the anterior tibial compartment), but compartment syndrome may occasionally affect the thigh and the arm.

- amputation

Rest pain
Ulcer }
Gangrene } chronic limb ischaemia

Aetiology

- Atherosclerosis
 - Risk factors- DM, HT, hyperlipidaemia, cigarette smoking, FHx
- Buerger's disease – young male smokers

Other manifestations of atherosclerosis

- Cerebrovascular
 - PHx of stroke, TIA, amaurosis fugax (transient blindness)
- Cardiac
 - PHx of MI, angina, Fx of heart failure
- Mesenteric
 - Abdominal pain 30-60 min after a meal which last for about 2hrs
- Renal
 - Uncontrolled 2ndry HT
- GUT
 - Impotence

Exclude DD

DD	Hx	Ex
Intermittent claudication	<p>Occurs after walking a distance</p> <p>Reproducibility</p> <p>Cramping aching pain</p> <p>Occurs in muscle groups (buttock, thigh, calf)</p> <p>Relieved quickly by standing still</p> <p>Develops quickly when walking uphill</p> <p>Not developed on 1st step</p> <p>Other evidence of atherosclerosis</p>	Arterial ulcers Gangrene Loss of LL arterial pulse Bruit
Neurogenic claudication	Sharp pain Relieved only when the pt sits /bends forward Takes long time to get relieved Finds easier when walking uphill	Evidence of nerve root compression
Venous claudication	Pain develops when prolong standing Ass. with swelling of the LL Relieved by elevation of the limbs PHx of DVT	Varicose veins Venous ulcers
Musculoskeletal pain	Pain present from the 1 st step Ass. joint pain	

What happened up to now – Ix, Rx, Sx

Effects of the symptoms on the pts life

- Day to day activities
- Impact on pts occupation
- Describe home environment & the occupational environment, & disabilities of the pt
- Psychological state of the pt

Ex

- ❖ Examine the peripheral vascular system 1st & do a quick Ex of the other systems

Inspection

- Proper exposure from the groin to toes
- Inspect the feet & toes
- Colour
- Loss of hair over the LL
- Tense tender calf with impaired dorsi-flexion (compartment compression)
- Venous guttering
- Buerger's test (Is the angle to which the leg has to be raised before it becomes pale)
- Ulcers
- Gangrene
- Amputation
- Flat foot

Palpation

- Temperature
- CRFT
- Pulses – carotid, radial, brachial, femoral, popliteal, dorsalis pedis
- Offer to measure the ABPI. (proper technique)
 - Measure the systolic BP at the level of the brachial artery in both arms & take the higher value
 - Apply the cuff a hands breath above the ankle joint
 - Locate the post. Tibial & dorsalis pedis using a hand held Doppler device
 - Start with one artery
 - Inflate the cuff until the Doppler signals are no longer heard.
 - Inflate 20mmHg above this point
 - Slowly deflate & take the reading of pressure at which the Doppler signals reappear
 - Repeat with the other artery
 - Take the higher value
 - Calculate ABPI
 - The ankle–brachial pressure index (ABPI) is the ratio of systolic pressure at the ankle to that in the arm.
 - Resting ABPI is normally about 1.0; values below 0.9 indicate some degree of arterial obstruction and less than 0.3 suggests imminent necrosis

Auscultate for bruits

Atrial fibrillation/ other cardiac arrhythmias → with a Hx of acute limb ischemia

Apex beat → ventricular aneurisms

Auscultate for evidence of valvular diseases (MS) → AF → systemic embolisation

What is your diagnosis?

- a. Peripheral vascular disease
 - b. Anatomical segment involved
 - c. Severity of the condition
 - d. Associated complications
- a. **1st discuss how you exclude other causes of claudication as mentioned in the Hx & Ex**

b. Anatomical segment

Segment involved	Hx	Ex
Aorto-iliac	B/L claudication in buttocks, thighs, calves	B/L femoral & distal pulses absent
	Impotence common	Bruit over the aorto iliac region
Iliac	U/L claudication of the thigh & calf	U/L absence of femoral & distal pulses Bruit over the iliac region
Femoro- popliteal	U/L claudication of the calf	Femoral pulse palpable U/L distal pulse absent
Distal obstruction	Claudication of the foot	Femoral & popliteal pulses + U/L ankle pulse absent

c. Clinical classification of the severity of the disease

stage	Clinical
I	Asymptomatic
IIa	Intermittent claudication with claudication distance >200m
IIb	" " " " <200m
III	Rest pain
IV	Ulceration / gangrene

Management

Investigation

- a) Confirm the diagnosis
- b) Assess the severity of the condition
- c) Assess the fitness for Sx

- Duplex scanning
- Angiography
- Digital subtraction angiography
- CT angiogram
- MR angiogram

Ix	Advantages	Disadvantages
Duplex scanning	1 st line Ix Non invasive Provides anatomical & functional Information	Operator dependent
Angiography <small>Classical angiography involves the injection of a radio-opaque solution into the arterial tree, generally by a retrograde percutaneous catheter method (Seldinger technique) usually involving the femoral artery (</small>	Performed if there is an indication for intervention	Hazards include thrombosis, arterial dissection, haematoma, renal dysfunction and allergic reaction.
DSA	Arterial system can be visualized in great detail	Injection of contrast via an arterial puncture Complications Puncture site haematoma A-V fistulas False aneurysms anaphylaxis
CT angiogram	Inject contrast medium via vein (avoid arterial puncture)	Large amount of contrast use Complications due to contrast
MRA	Can visualize the vessels without contrast	Expensive Not freely available

Treadmill

- For Patients themselves are poor at assessing claudicating distance
 - A useful objective measurement of distance to onset of pain.
 - However, a simple measured walk along a hospital corridors even more reliable.
- **Blood sugar** – DM (common risk factor for PVD)
 - **Hb** – exclude polycythaemia, anaemia → precipitate angina or claudication
 - **ESR & CRP** – inflammation & mycotic aneurysms
 - **Lipid profile (S. Cholesterol)** – atherosclerosis
 - **ECG** – exclude ass. coronary disease
 - **ECHO** – valvular lesions, mural thrombus on an akinetic ventricular wall
 - **CXR** – bronchial CA → common finding in end stage vascular dis. Both caused by smoking

Treatment

Conservative Rx

- Stop smoking
- Exercise to ↑ collateral circulation
- Learn to live within a claudication distance, involving a change in life & employment
- Weight loss – less effort to muscles
- Avoid prolong standing (always change the posture)
- Raising the heel of the shoe – less effort for the calf muscles
- Foot care to prevent minor trauma (may lead to gangrene)
- Treat co existing conditions – DM, HT, hyperlipidaemia

Drug

- Medication may be required for diseases associated with arterial disorders, such as hypertension and diabetes;
- Some anti-hypertensives (particularly **β-blockers**) may **exacerbate claudication**
- Raised blood lipids require active drug treatment but even when the lipid profile is normal a statin should be prescribed (e.g. 40 mg/day of pravastatin)
- An antiplatelet agent is also necessary, usually 75 mg day⁻¹ of aspirin,
- With 75 mg day⁻¹ of clopidogrel as an alternative for those who are aspirin intolerant
- Other agents, such as vasodilators, are unlikely to prove beneficial.
- Thrombolysis
- Fibrinolytic enzymes – streptokinase, TPA
- Infused into the clot which it dissolves takes time so not appropriate where limb viability is acutely threatened

Interventional

- Angioplasty
- Endoluminal stenting
- Bypass Sx (only severely handicapped)

What is angioplasty

- Blowing up a balloon within the vessel
 - ↓
- To stretch & fracture the blockage
 - ↓
- Allow more blood to pass through
- Most successful with concentric stenosis or blocks in the iliac system
- Unsuccessful with long blocks over 10cm, in the distal femoral & popliteal arteries
- An endovascular stent may maintain the patency
- Risk – distal embolization, vessel perforation

Preparation of pt for angioplasty

- Informed written consent
- Pre procedural Ix – most important renal function
- Preparation
- Proper hydration of the pt
- Administration of NAC in pts with impaired renal function
- Administration of steroids in pts with a high risk of developing contrast allergies

GANGRENE

- Gangrene implies death of macroscopic portions of tissue;
- It often affects the distal part of a limb because of arterial obstruction (from thrombosis, embolus or arteritis)
- This type of gangrene, which affects tissues that were initially sterile
- Must be distinguished from those forms of gangrene that derive mainly from infection (classically gas gangrene), although both may coexist.

Clinical features

- A gangrenous part lacks arterial pulsation, venous return, capillary response to pressure, sensation, warmth and function.
- The colour of the part changes through a variety of shades according to circumstances (pallor, dusky grey, mottled, purple) until finally
 - ↓
- taking on the characteristic dark-brown, greenish-black or black appearance
 - ↓
- which is caused by the disintegration of haemoglobin and the formation of iron sulphide.
- Dry gangrene occurs when the tissues are desiccated by
 - ↓
- gradual slowing of the bloodstream;
 - ↓
- it is typically the result of atheromatous occlusion of arteries.
 - ↓
- The affected part becomes dry and wrinkled, discoloured from disintegration of haemoglobin, and greasy to the touch.
- Moist gangrene occurs when infection and putrefaction are present;
- the affected part becomes swollen and discoloured and the epidermis may be raised in blebs.
- Crepitus may be palpated as a result of infection by gas-forming organisms.
- This situation is quite common in the feet of diabetics.
- **Separation of gangrene**
- A zone of demarcation between the truly viable and the dead

AMPUTATION

General

- Amputation should be considered when part of a limb is dead, deadly or a dead loss.
- A limb is dead when arterial occlusive disease is severe enough to cause infarction of macroscopic portions of tissue, i.e. gangrene.
- The occlusion may be in major vessels (atherosclerotic or embolic occlusions) or in small peripheral vessels (diabetes, Buerger's disease, Raynaud's disease, inadvertent intra-arterial injection).
- If the obstruction cannot be reversed and the symptoms are severe, amputation is required.

Summary box 53.2

Indications for amputation

Dead limb

- Gangrene

Deadly limb

- Wet gangrene
- Spreading cellulitis
- Arteriovenous fistula
- Other (e.g. malignancy)

'Dead loss' limb

- Severe rest pain
- Paralysis
- Other (e.g. contracture, trauma)

Neurogenic claudication

- is a syndrome, or collection of symptoms,
- associated with [degenerative spinal stenosis](#) in the [lumbar spine](#).
- It is often related to posture.
- The combination of the stenosis with certain back postures such as arching
- puts pressure on lumbosacral [nerve roots](#) and/or the cauda equina.
- Lumbar spinal stenosis is common in middle aged and elderly people;
- it generally takes a long time for the stenosis to develop and the symptoms of neurogenic claudication to appear.

Symptoms of Neurogenic Claudication

- Neurogenic claudication shows up as pain in the low back and leg.
- The pain may feel like muscle cramps.
- It is brought on during walking and/or extending the spine backwards (arching your back),
- and it is relieved by stopping, sitting, and/or bending forward at the waist. Other symptoms include pins and needles going down your leg, and weakness.
- Bowel or bladder problems may occur if the neurogenic claudication is severe.

[Neurogenic Claudication Treatment](#)

Treatment for neurogenic claudication begins conservatively.

Prescribe [pain medications](#) and/or physical therapy.

Exercises that emphasize flexing the spine forward.

Other conservative treatments for lumbar spinal stenosis include modifying your activity, wearing a back brace or belt and/or having a [spinal epidural injection](#) to calm the pain.

If continued pain from neurogenic claudication(greatly interferes with your lifestyle, it may be a sign that you need [back surgery](#). Usually, a [laminectomy](#) (also known as decompression surgery) is the [procedure given for spinal stenosis](#).

Venous claudication

- Rare Xn
- Occur in young
- Physically active pts after extensive DVT with persistent occlusion of pelvic veins
- Intensive physical activity leads to decompensation of the impaired venous outflow from the leg
- **Plethysmography** during walking documents a continuous ↑ volume of the affecting leg up to the onset of pain
The increasing painful tension of legs forces the pt to stop walking & best **relieved by elevation** of the leg

Neuropathic joints (Charcot's joints)

Joints damaged by trauma as a result of the loss of the protective pain sensation.

They were first described by Charcot in relation to tabes dorsalis.

They are also seen in

- Syringomyelia
- Diabetes mellitus
- Leprosy

The site of the neuropathic joint depends



On the localization of the pain loss:

- In tabes dorsalis, the knees and ankles are most often affected.
- In diabetes mellitus, the joints of the tarsus are involved.
- In syringomyelia, the shoulder is involved

Neuropathic joints are not painful, although there may be



Painful episodes associated with crystal deposition.



Presentation is usually with swelling and instability. Eventually severe deformities develop.

The characteristic finding is a swollen joint with abnormal but painless movement.

- This is associated with neurological findings that depend upon the underlying disease (e.g. dissociated sensory loss in syringomyelia or polyneuropathy in diabetes).
- X-ray changes are characteristic,
 - With gross joint disorganization and bony distortion.

Treatment is symptomatic. Surgery may be required in advanced cases.

RIF Mass & RIF Pain

DDs for RIF Mass –

- Appendicular mass
- Appendix abscess
- CA Caecum & ascending colon
- Ileal TB
- Crohn's disease
- Psoas abscess
- Ovarian mass / fibroid uterus
- Undescended testis with malignant changes
- Ileac lymphadenopathy
- Pelvic kidney / renal transplant
- Distended gall bladder
- Ileac artery aneurysm
- Ruptured epigastric artery
- Spigelion hernia
- Chondrosarcoma /osteosarcoma of the ileum

DDs for RIF Pain –

- Appendicitis
- Crohn's disease
- CA of caecum & R/colon
- PID

Table 67.1 Differential diagnosis of acute appendicitis

Children	Adult	Adult female	Elderly
Gastroenteritis	Regional enteritis	Mittelschmerz	Diverticulitis
Mesenteric adenitis	Ureteric colic	Pelvic inflammatory disease	Intestinal obstruction
Meckel's diverticulitis	Perforated peptic ulcer	Pyelonephritis	Colonic carcinoma
Intussusception	Torsion of testis	Ectopic pregnancy	Torsion appendix epiploicae
Henoch–Schönlein purpura	Pancreatitis	Torsion/rupture of ovarian cyst	Mesenteric infarction
Lobar pneumonia	Rectus sheath haematoma	Endometriosis	Leaking aortic aneurysm

Demographic data –

- Age - appendix mass- in young adults (10- 20 yrs), rare in extremes of age
 - Infants/children –wide lumen → less chance of obstruction
 - Elderly → appendix fibrosed & contracted→ less chance of FB impaction
 - CA caecum – older pts, usually > 50 yrs
 - CD – young (20-40 yrs)
- Sex – Female – ovarian / tubal masses
Male – malignant changes of undescended testis

PC – Abdominal Pain (associations varies depending on the cause)

- Eg: diarrhea – in CD
- Fever – in psoas abscess

HPC- Describe the pain,

- Time & nature of onset
- Site
- Character
- Severity
- Progression
- Duration
- Radiation
- Relieving / exacerbating factors
- Associated symptoms

Appendix mass –

- Initially – symptoms of acute appendicitis
 - N, V, LOA, constipation, rarely diarrhea – 12-24 hrs prior to onset of abd pain
 - (Profuse vomiting is not a feature – max 2 times)
 - Low grade fever
 - Typical presentation (50%)-
 - Vague peri umbilical pain – referred pain in T 10
 - After few hrs – pain shifts to RIF, become more severe
 - Colicky initially → continuous
 - Atypical presentation (50%)-
 - Pain may begin & remain in RIF
 - Pain only felt in center
 - Pain at both sites simultaneously
 - Pain may worse with coughing & moving – pt prefers to lie still with the hips & knees flexed.
 - Pelvic appendix – suprapubic pain with urinary frequency (due to bladder irritation)
 - Retrocaecal appendix – loin pain
 - Pelvic appendix irritating rectal wall
 - Retro ileal appendix irritating terminal SI
- } Diarrhea
- Appendix close to terminal ileum – Fx of intestinal obstruction (colics & abd distention)

➤ Ruptured appendix-

- There may be temporary remission / cessation of pain –
As tension in the distended organ is relieved → Fx of general peritonitis
generalized abd pain
N, V, sweating
Fever with rigors
- Hx of previous milder attacks of similar pain
- Fetor oris

*Appendix mass – Hx of 4-5 days of abd pain with a localized mass in RIF
No evidence of general peritonitis.

Appendix abscess –

- Hx of appendix mass + symptoms of an abscess
- ↓
- High fever with rigors
Sweating
Increased severe local pain

CA caecum / CA ascending colon –

- Pain – dull discomfort in the RIF
- Some pts presents with – diarrhea or features of intestinal obstruction- Vomiting, constipation
- **Features of anaemia** – lethargy, body weakness, SOB on exertion, palpitations, fainting
- Features of malignancy - LOW, LOA, malaise

Ileo-caecal TB – (due to swallowing of infected sputum or infected milk)

- Vague central pain for months → moves to RIF → become intense & settles in RIF
- General ill health – chronic cough, malaise
- Alternating constipation & diarrhoea
- LOW
- Contact Hx of TB / past Hx of pulmonary TB

Crohn's disease –

- Ad pt with crohn's disease or
- Fx of crohn's – Recurrent episodes of pain in RIF
 - General malaise, Wt loss
 - Episodes of diarrhea – intermittent with mild bleeding
 - Extra intestinal manifestations-
 - ✓ Uveitis, conjunctivitis
 - ✓ Oral ulcers
 - ✓ Arthritis / arthralgia
 - ✓ Skin rashes
 - ✓ Gall stones/ renal stones

- Complications –
 - Peritonitis - Intestinal perforations
 - Perianal , ischiorectal abscesses
 - Anorectal fistula
 - Enteric fistula (faecaluria, faecal vaginal discharge)

Psoas abscess –

- Pt felt ill for some months
- **High swinging fever**, night sweats, malaise
- Abd pain & back pain
- **Pain with movements of hip (pt tend to hold their hip in flexion)**

Ovarian / tubal mass –

- Dysmenorrhoea, Dyspareunia
- Dysuria
- Hx of ectopic pregnancy
- Irregular menstruation

Undescended testis with malignant changes –

- LOW, LOA
- Gynaecomastia
- Cough (due to lung mets)

Ileal lymphadenopathy –

- Symptoms depend on the cause of lymphadenopathy
- Generalized or local disease in limb, perineum or genitalia.

PMHx – co morbidities- DM, HT, BA, IHD (to assess fitness for surgery)

PSHx – renal transplant

Appendectomy

Surveillance Colonoscopy/ colonoscopy done previously & Bx which confirmed a certain Δ

Drug Hx – on NSAIDs (may exacerbate Crohn's)

Family Hx – HNPCC, FAP

Crohn's

Social Hx- contact Hx of TB

Smoking

- Ex-**
- III looking
 - Febrile (in appendix mass/ abscess, psoas abscess)
 - Pallor (in CA caecum)
 - Red eye, jaundice
 - Oral ulcers
 - Skin rashes – erythema nodosum
 - SuprACLavicular LNs
- } (in CD)

Abdominal Ex –

Appendicitis –

- Abdomen looks normal.
- RIF tenderness – maximum over Mc Burney's point
- overlying muscle guarding & rigidity – due to parietal peritoneal involvement
- Rebound tenderness – Elicited by stretching the parietal peritoneum
 - Ask the pt to cough → pain ↑
 - Percuss over RIF → pain ↑
 - Deep palpation & sudden release – do not practice (too painful)
- DRE –tenderness on R/S (pelvic appendicitis)
- Other signs –(not elicited now, unless in doubt)
 - Rovsing's sign – pressing on LIF causing pain in RIF
due to gases forced in to caecum from L/colon
 - Blumberg sign - Release of pr on LIF cause pain in RIF
 - Psoas sign – pelvic / retrocaecal appendix
Flexion of R/hip → ↓ pain – as appendix sitting on psoas muscle
 - Obturator sign – flexion & internal rotation of R/hip → pain
- Percussion- dullness (+ if peritonitis present)

Appendix mass – (associated with persistent low fever & tachycardia)

A tender distinct mass in RIF

Can not feel below it – bcoz it is fixed to RIF posteriorly.

Dull to percussion

Appendix abscess – (associated with swinging intermittent high fever & ↑ tachycardia)

Very tender mass

CA Caecum / ascending colon –

Non tender distinct hard mass

Usually fixed to posterior abd wall – maybe mobile

Temp: & pulse is normal – unless there is an associated pericolic abscess

Ileocaecal TB –

Firm, tender mass

Very indistinct – surface & edges are difficult to define

TB peritonitis – swollen & less flexible abdomen (doughy abd)

⊕ Crohn's disease –

Elongated sausage shaped mass-usually lies transversely in RIF(swollen terminal ileum)
Rubbery & tender

⊕ Psoas abscess –

RIF filled with a soft, tender, dull, compressible mass
Fullness in lumbar region when pressing the mass in RIF
Swelling may extend below the groin
Can empty the swelling below the groin in to the swelling above & vice versa
Back movements – painful & limited

⊕ Ileal lymphadenopathy –

Indistinct mass with no clear contours.
Mass follows the line of ileac vessels –may bulge forward just above the inguinal ligament
Must EX other lymph nodes & LLs –to try to find the cause

DRE

Ix -

Appendix mass / abscess –

- FBC –↑ WBC with neutrophil leukocytosis
- UFR – RBC & WBC/pyuria present if inflamed appendix sitting on ureter /bladder
(pyuria commoner in children-since shallow pelvis, appendix easily abuts on bladder)
- USS Abd –
 - Appendix – thickened wall & dilated lumen
 - Detect appendix mass & abscess
 - Rule out gynaecological pathology
- In all females of child bearing age – urine hCG
- Laparoscopy – Gold standard
 - Both Δtic & therapeutic
- Fine catheter aspiration of abd cavity –(rare) presence of bld /WBC in large amounts in peritoneal fluid→ need Sx

CA Caecum / ascending colon-

- Stool occult blood
- Double contrast barium enema (-ve test doesn't exclude small tumors)
- Colonoscopy
- USS abdomen : To visualize the synchronous lesions , liver deposits
- CT scan – to assess local invasion

Ileal TB –

- CXR , mantoux test, sputum culture
- TB PCR
- Barium meal and follow through –Strictures terminal ileum
- USS Abdomen – may show mass
- Δ may only be made by laparotomy – histological Ex or culture

Crohn's disease –

- AXR – obstruction, perforation, toxic dilatation
- Small bowel enema – skip lesions, strictures, mucosal ulceration (rose thorn ulcers), cobblestone mucosa, thick wall & a narrow lumen(string sign)
- Colonoscopy & Bx- skip lesions , Bx- non caseating granulomas
- CT scan – in complex presentations (abscess)

Psoas abscess
Ileal lymphadenopathy } USS Pelvis

Mx-

Appendix mass/ abscess –

Acute appendicitis → appendicectomy

Acute appendicitis + generalized peritonitis → Sx → (intraperitoneal lavage with N/S + AB)
Appendicectomy + drain

Appendicular mass - inflammatory mass → planned appendicectomy

Appendicectomy with next episode
Interval appendicectomy

- appendicular abscess → exploration + evacuation of pus +/- appendicectomy

Appendicular mass Mx-

In most cases it resolves spontaneously (80%)

Several schools of thoughts

1) **Perform appendicectomy in next episode** → indicating abscess formation

2) **Planned appendicectomy –**

Appendicectomy at next available list

However not beyond 7th day → peak period of adhesions

3) **Interval appendicectomy – (Not practiced)**

Appendicectomy performed 3 months later (6-8 wks – Ariyaratne sir)

I^o – in those whose symptoms resolve

Simple drainage of appendicular abscess done

(appendicectomy is delayed – to avoid the hazards of operating in an infected field)

➤ Disadvantages of interval appendicectomy –

- Should not use in young / elderly – poor ability to wall off inflammation
- Diagnosis uncertain
- Need 2nd admission

➤ Disadvantages of operating first hand –

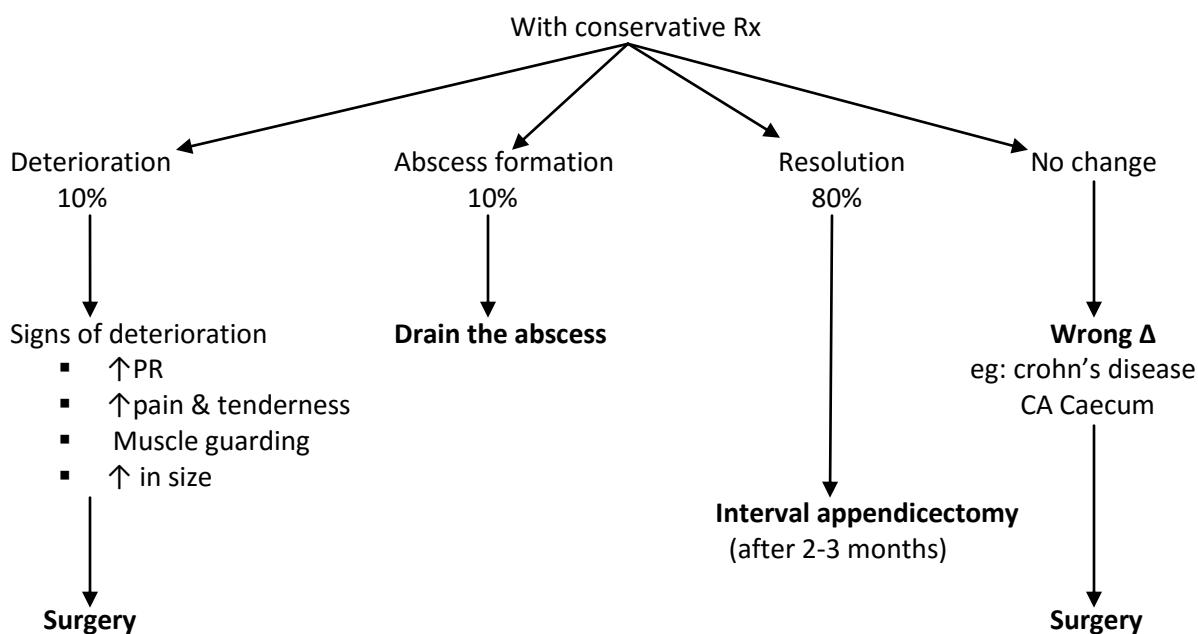
- Dissemination of infected material
- inflammation → vasodilatation → Haemorrhage ↑↑
- Faecal fistula

Post-op → wound infection

Residual abscess

Ochsner Sherren regime (delayed Rx of acute appendicitis)

- Not practiced now (never for acute appendicitis) – for those presenting > 48 -72 hrs since onset
- ✓ Pt kept in semi upright position in bed (Fowler's position)
- ✓ Keep under observation
- ✓ Monitor
 - PR – 4hrly
 - Temperature – twice daily (but in our wd QHT)
 - fluid balance chart
 - Palpate mass regularly & mark its limits on the skin(USS assessment is better if available)
- ✓ Liquid diet/ IV fluids
- ✓ Broad spectrum antibiotics & metronidazole (IV) } 48- 72hrs
- ✓ Purgatives are forbidden



➤ *Advantages of conservative Rx –*

Sx is difficult & dangerous – maybe difficult to find the appendix
Inflamed appendix may get ruptured

➤ *Disadvantages –*

Should not be done in very young & old pts – inability to localize the inflammation
Not done in young females due to risk of pelvic infections → subfertility
Risk of generalized peritonitis

Operative Rx –

Preferred by most surgeons

Methods –

- 1) Open appendicectomy
- 2) Laparoscopic appendicectomy

Pre-op preparation –

Confirm Δ

Analgesics – Diclofenac Na 100mg suppositories

Usually pt is young – thus no need of pre-op Ix

NBM from 10pm the night before Sx

Hydrate well – IV fluids (deficit + maintainance) since NBM

Monitor – PR, BP, respiration

*if emergency – NG decompression

IV Ranitidine + Na citrate

Send to theatre with IV AB to be given at induction

Cefuroxime 750mg 6 hrly

Flagyl 500mg tds

(Clean contaminated → risk of appendix rupture)

Incisions –

1. Lanz incision
2. Rutherford incision
3. Gridiron incision

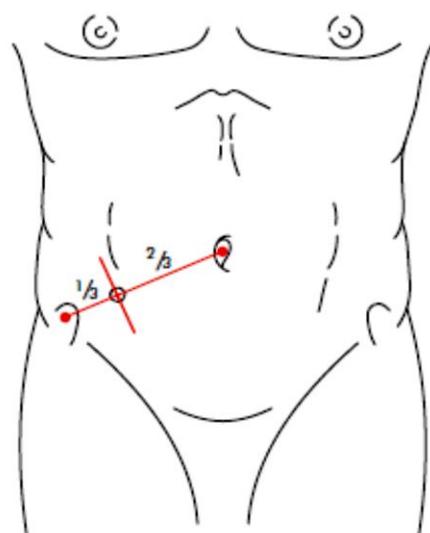


Figure 67.12 Gridiron incision for appendicitis, at right angles to a line joining the anterior superior iliac spine and umbilicus, centred on McBurney's point (courtesy of Mr M. Earley, FRSCI, Dublin, Ireland).

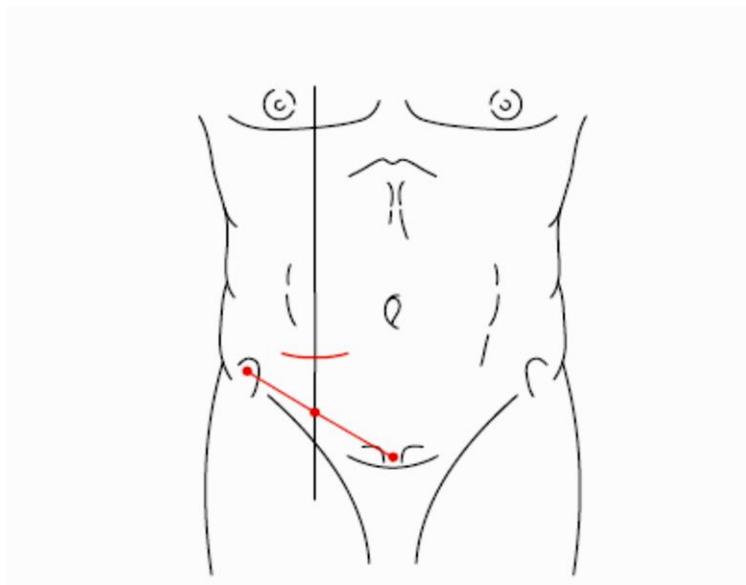


Figure 67.13 Transverse or skin crease (Lanz) incision for appendicitis, 2 cm below the umbilicus, centred on the mid-clavicular-midinguinal line (courtesy of Mr M. Earley, FRCSI, Dublin, Ireland).

Surgical procedure –

- Lanz incision – external oblique aponeurosis divided
- Appendix identified
- Mesoappendix clamped & appendicular artery ligated
- Appendix separated from appendicular bed
- Appendix ligated & clamped at the base
- Appendix removed → sent for histology
- Stump of appendix invaginated in to caecal wall via purse string sutures
- A drain is inserted to peritoneum

Open appendicectomy – (Dunn's)

- Amount of blood need to be preserved – 0
- GA
- Open time- 15-20 –mins
- Hospital stay – 4-5 days in the absence of residual sepsis
- Drains out – 3-5 days
- Sutures out – 5-7 days
- Off work – 1 month

Laparoscopic appendicectomy – (Dunn's)

- Amount of bld need to be preserved – 0
- GA
- Open time – 30-60 mins
- Hospital stay – 1-3 days in the absence of residual sepsis
- Drains out – 2-3 days
- Sutures out – absorbable
- Off work- 2 wks

Post- op Mx-

Keep NBM until anaesthetic effect wears off & bowel sounds appear

SA – 4 hrs

GA – 6 hrs

Monitor – PR, BP, RR → ¼ hrly x 2 hrs

½ hrly x 2hrs

Hrly x rest of 24 hrs

Look for developing sepsis in the wound or pelvis (QHT)

Hydrate

IV AB – since clean contaminated –continue 3 doses

If perforated – continue for 7 days

Can discharge on post op D1

Wound inspection & DRE – before pt discharge

Advice pt

- Clean & dressing after 5 days
- Suture removal – 10 days
- R/V at clinic with histology report
- come back if develop ↑ pain or a fever

➤ Advantages of operative Rx –

Diagnosis is certain

Short hospital stay

➤ Disadvantages –

Dissemination of infection

Wound infections

Haemorrhage

Faecal fistula

CA Caecum / ascending colon –

Pre-op bowel preparation & R/hemicolecotomy

Ileocaecal TB –

Nutrition & hygienic living conditions to prevent spread of infection

Anti TB drugs for 6 months – 2 yrs

Sx –

- I^o – when Δ is unclear, for perforations, abscess, bleeding, or obstructions
- Obtain tissue for Δ, either by histology or culture
- Evacuate pus & necrotic tissue
- Deal with complications

Summary box 5.18

Tuberculosis – pathology

- Increasingly being seen in the UK, mostly among immigrants
- Two types are recognised – ulcerative and hyperplastic
- The ulcerative type occurs when the virulence of the organism is greater than the host defence
- The opposite occurs in the hyperplastic type
- Small bowel strictures are common in the hyperplastic type, mainly affecting the ileocaecal area
- In the ulcerative type, the bowel serosa is studded with tubercles
- Localised areas of ascites occur in the form of cocoons
- The lungs and other organs may also be involved simultaneously

Summary box 5.19

Tuberculosis – clinical features

- Intestinal tuberculosis should be suspected in any patient from an endemic area who presents with weight loss, malaise, evening fever, cough, alternating constipation and diarrhoea and intermittent abdominal pain with distension
- The abdomen has a doughy feel; a mass may be found in the right iliac fossa
- The emergency patient presents with features of distal small bowel obstruction – abdominal pain, distension, bilious and faeculent vomiting
- Peritonitis from a perforated tuberculous ulcer in the small bowel can be another emergency presentation

Crohn's disease –

Medical Mx -

- Induction of remission
 - IV corticosteroids or oral prednisolone**
 - Cessation of smoking
 - Low fat, low linoleic acid containing diet
 - Oral steroids + azathioprine or mercaptopurine
- Maintenance of remission
 - Aminosalicylates
 - Azathioprine, 6 Mercaptopurine, Mycophenolate mofetil
- For glucocorticosteroid resistance
 - Methotrexate
 - IV cyclosporine
 - Infliximab
- Perianal disease
 - Ciprofloxacin + Metronidazole
- Control diarrhoea
 - Loperamide, Codeine

Surgical Mx

Best avoided bcoz of risk of malabsorption

Will not cure crohn's disease

Indications –

- Failure of medical therapy
- Complications –
 - Toxic dilatation, obstruction, abscesses, enterocutaneous fistula, Bleeding & Perforations
 - Ileocaecal disease
 - Perianal disease
 - Fulminant colitis or malignant changes
 - Failure to thrive in children

Sx -

- In ileal crohn's - Surgical resection of affected segment with end to end anastomosis / end ileostomy
 - In crohn's colitis –segmental colectomy
 - Strictureplasty – where several affected areas of bowel is present (widening of a strictured area)
 - panproctocolectomy – in severe cases
- pre-op preparation –
 - Check serum protein – if not satisfactory – IV feeding
 - Bowel preparation – in large bowel crohn's
 - Prophylactic antibiotics
 - If already on steroids - ↑the dose pre operatively

- ✓ Amount of bld need to be preserved – 2 units
- ✓ GA
- ✓ Open time- 90-150 –mins
- ✓ Hospital stay – 7-14 days in the absence of residual sepsis
- ✓ Drains out –0
- ✓ Sutures out – 7 days unless absorbable
- ✓ Off work – 6-8 wks

✚ Post-op Mx-

NG suction & IV fluids – until pt pass flatus
IV feeding – for those debilitated with severe crohn's
7-14 days hospital stay – to make sure the danger of fistula formation is passed
Long term follow up- due to tendency to recur

Psoas abscess-

- Drain the abscess
- Antibiotics
- Analgesics
- Find out the cause of the abscess & Rx it

Discussion

➤ What is Alvarado score?

A number of clinical & laboratory based scoring systems have been devised to assist Δ.
The most widely used is Alvarado score.

Symptoms	
Migratory RIF pain	1 point
Anorexia	1 point
Nausea & vomiting	1 point
Signs	
Tenderness in RIF	1 point
Rebound tenderness	2 point
Elevated temperature	1 point
Laboratory	
Leucocytosis	2 points
Shift to left	1 point
(neutrophil preponderance)	
Total score	10 points

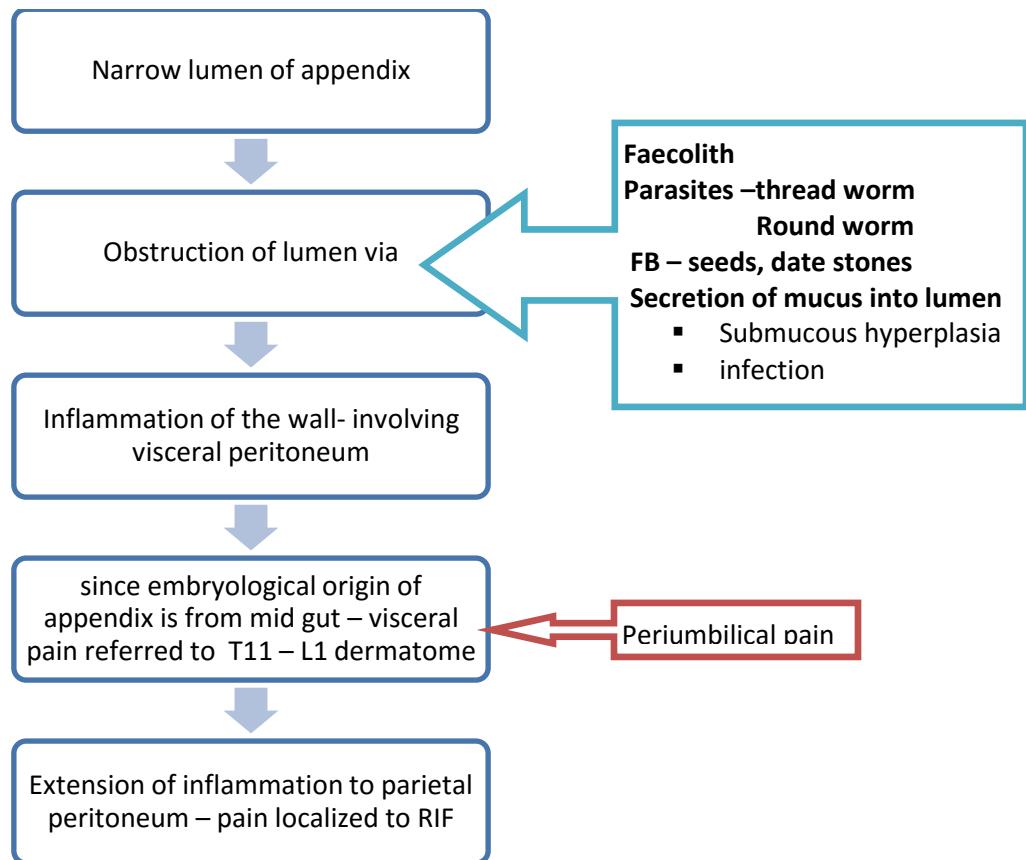
Score < 4 –strongly against a Δ of appendicitis.

No need to admit, but observe for 24 hrs → why?

bcoz in those with perforation of appendix, there is settling of symptoms prior to peritonitis

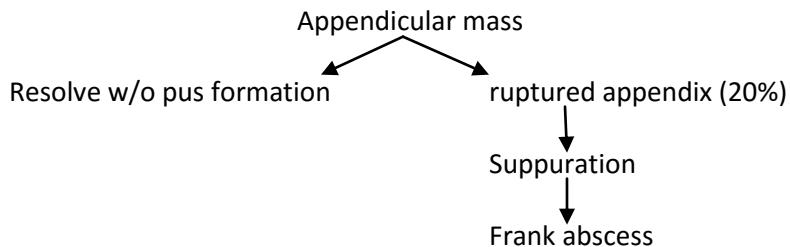
Score ≥7 – predictive of acute appendicitis → appendicectomy

➤ Pathophysiology of acute appendicitis –



➤ Appendicular mass formation –

Inflamed appendix surrounded by coils of bowel & greater omentum localizing the inflammation.



➤ Acute appendicitis + generalized peritonitis

Perforation of the appendix → settling of severe colicky pain and vomiting → peritonitis

3 stages

- 1) Shock stage – pale, sweating and anxious
 - ↑ PR, ↓ BP, resp. – rapid, shallow
 - Temp. subnormal
 - ↑↑ local tenderness in RIF
- 2) peritoneal reaction stg – severe RIF tenderness
 - rebound “
 - board like rigidity
 - marked rectal tenderness
- 3) Frank peritonitis – abd. Distension + absent BS

Pathological types of appendicitis

- 1) Catarrhal
- 2) Obliterative
- 3) Ulcerative
- 4) Gangrenous
- 5) Perforative

Patient with right hypochondrial pain

History

Introduction

Mrs., 40 yr old housewife, who is a mother of 3, from presented with....

Presenting complaint

Acute onset right hypochondrial pain
+/- jaundice

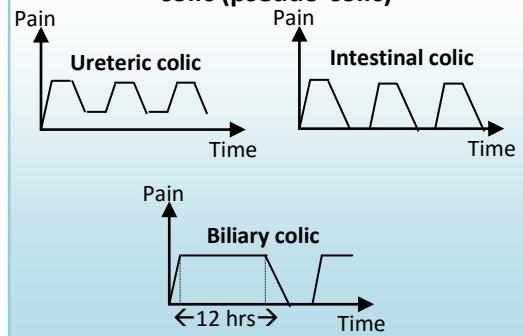
History of presenting complaint

1. Enumerate the presenting complaint
 - Is this the 1st episode?
 - Describe the pain
 - Site of the pain
 - Onset
 - Sudden
 - Gradual
 - Progression
 - How it progress with time?
 - How long it took to reach the peak of the pain?
 - Character
 - Colicky – Associated with obstruction of a muscular tube or by muscular dysfunction.
 - Constant
 - Radiation
 - Associated features
 - Nausea
 - Vomiting
 - Faintishness
 - Jaundice
 - Timing – How long the pain last and pain free interval
 - Severity – How bad the pain is....
 - Exacerbating & relieving factors

Differential Diagnoses

- *Biliary colic*
- *Acute cholecystitis*
- *Chronic cholecystitis*
- *Cholangitis*
- Ureteric colic
- R/ Lobe pneumonia
- Myocardial infarction
- Perforated peptic ulcer
- Acute pancreatitis
- Hepatitis

Difference between ureteric and biliary colic (pseudo-colic)



2. Identify the cause among the main differential diagnoses

	Cholelithiasis	Choledocholithiasis	Acute Cholecystitis	Chronic Cholecystitis	CA Gall Bladder	Pancreatitis
Patient factors	* Fat * Fair * Fertile * Females * Forty	* Fat * Fair * Fertile * Females * Forty	* 95 % of cases associated with gall stones * Acalculous cholecystitis associated in <i>Typhoid fever & Gas gangrene infection</i>	* Almost invariably associated with gall stones	* Relatively uncommon. * 85% cases associated with gall stones	
Site of the pain	* Right subcostal region, but may be epigastric	* Right subcostal region, but may be epigastric	* Right costal margin			* Epigastric pain
Onset	* Acute	* Acute	* Sudden onset			* Acute or chronic
Severity	* Severe pain * Restless * Rolls about in agony	* Severe pain * Restless * Rolls about in agony	* Severe pain			* Severe episodes
Progression	* Rising to a plateau	* Rising to a plateau				
Character	* Colicky exacerbations	* Colicky exacerbations	* The pain may 1st be colicky, indicating the stage of obstruction of cystic duct or neck of GB			
Radiation	* Inferior angle of right scapula	* Inferior angle of right scapula	* Inferior angle of right scapula	* Recurrent bouts of abdominal pain due to mild cholecystitis. * +/- Fever		* Radiate through the back
Timing	* Rising to a plateau. Persist for some time & then come to the baseline and again starts after some time	* Rising to a plateau. Persist for some time & then come to the baseline and again starts after some time		* Discomfort after fatty meals (Fatty meals --> Secretes cholecystokinin --> Gall bladder contracts onto the stones) * Often flatulence * Episodes of acute cholecystitis or symptoms produced by stone passing into CBD		* Closely resembling chronic cholecystitis * Right hypochondrial pain * Nausea, Vomiting * Weight loss * Later progression to obstructive jaundice
Exacerbating & reliving factors	* Is exacerbated with fatty meals	* Is exacerbated with fatty meals				* Related to episodes of alcohol intake * Relieved by beding forward
Associated features	* Vomiting * Sweating * Dyspepsia * Flatulence * Food intolerance, particularly fat * Alteration of bowel habits * +/- Jaundice Clay colored stools Dark urine (These symptoms lasts till the small stone pass through the sphincter of Oddi or it disimpacts and falls back to the dilated CBD)	* Vomiting * Sweating * Dyspepsia * Flatulence * Food intolerance, particularly fat * Alteration of bowel habits * +/- Jaundice Clay colored stools Dark urine (These symptoms lasts till the small stone pass through the sphincter of Oddi or it disimpacts and falls back to the dilated CBD)	* Fever * Vomiting * Sweating			
Aetiology	<p>Cholesterol stones</p> <ul style="list-style-type: none"> * Associated with elevated cholesterol * OCP, Pregnancy & increasing age * Family history, Obesity, low dietary fibre intake * Deficiency of bile salts due to interruption of entero-hepatic circulation caused by <i>removal of terminal ileum</i>. (Eg : in Crohn's disease) <p>Pigment stones(Ca bilirubinate with some CaCO₃)</p> <ul style="list-style-type: none"> * Haemolytic anaemias (Spherocytosis & sickle cell) --> Ask about family history of calculi <p>Mixed stones</p> <ul style="list-style-type: none"> * Has same metabolic origin as cholesterol stones 	<p>* 95 % of cases associated with gall stones * Acalculous cholecystitis associated in <i>Typhoid fever & Gas gangrene infection</i></p>	* Almost invariably associated with gall stones	* Relatively uncommon. * 85% cases associated with gall stones		* Alcohol * Gall stone disease

	Cholelithiasis	Choledocholithiasis	Acute Cholecystitis	Chronic Cholecystitis	CA Gall Bladder	Pancreatitis
Pathological effects	<ul style="list-style-type: none"> * Silent : gall stone lying free in the lumen of GB * Impaction in hartmann's pouch or in cystic duct --> Water absorbs --> Concentrated bile --> Chemical cholecystitis --> 2ry infection(Fever) (If impaction happens when the GB is empty --> Walls continue to secrete mucous --> Mucocoele) * Gallstone ileus GS ulcerate through the GB wall into duodenum --> impaction at distal ileum --> Intestinal obstruction 	<ul style="list-style-type: none"> * Gallstone migration into common bile duct --> Intermittant or complete obstruction of CBD --> Pain & jaundice * If the obstruction not relieved --> Chronic back pressure in the biliary system --> 2ry biliary cirrhosis and liver failure * Ascending cholangitis --> pain & jaundice complicated by intermittent fever with chills & rigors. Pt is toxic 	<ul style="list-style-type: none"> * Swollen GB --> press on the CBD --> Tinge of jaundice * It the obstruction not relieved --> Chronic back pressure in the biliary system --> 2ry biliary cirrhosis and liver failure * Ascending cholangitis --> pain & jaundice complicated by intermittent fever with chills & rigors. Pt is toxic 	<ul style="list-style-type: none"> * Repeated episodes of inflammation result in chronic fibrosis & thickening of the entire gall bladder wall, which may contain thick, sometimes infected bile. 		<ul style="list-style-type: none"> * Obstructive Jaundice * Steatorrhoea * Recently diagnosed DM
	Associated clinical syndromes					
	<ul style="list-style-type: none"> * Biliary colic * Acute Cholecystitis * Chronic Cholecystitis * Obstruction and/or infection of the common bile duct 					
	<ul style="list-style-type: none"> * Presence of gallstones in biliary tree is associated with <ol style="list-style-type: none"> 1. Acute or chronic pancreatitis 2. CA Gallbladder 					

3. Exclude other differential diagnoses

- R/ Lobe pneumonia
 - Fever with chills & rigors
 - Productive Cough
 - Dyspnoea
- Ureteric colic
 - Radiate from loin to groin
 - Colicky pain (Pain comes and goes, but it doesn't reach the base line.)
 - Associated UTI features – Dysuria, Frequency
 - Haematuria
- Myocardial infarction
 - Chest pain
 - Sweating
 - Nausea, Vomiting
 - Past history of angina
 - DM/Smoking/Sedentary life style/Alcohol
- Perforated peptic ulcer
 - Previous episodes of GORD
 - Dyspeptic symptoms
- Acute Pancreatitis
 - Epigastric pain radiate to the back
 - Relieved by leaning forward
 - Exacerbated by bouts of alcohol
- Hepatitis
 - Fever with jaundice

- Blood transfusions, IV drug use, Sexual promiscuous behavior
 - Unhygienic food outside
4. Ask the question to identify possible aetiology – Mentioned above
 5. Ask the questions to identify complications – Mentioned above
 6. Management done up to now

PMHx

- Previous similar episodes
- DM
- Hereditary spherocytosis
- Sickel cell disease (Rare in Sri Lanka)
- Typhoid fever
- Hypercholesterolaemia
- IHD/HT (Exclude MI)

PSHx

- Removal of ileum due to Crohn's disease

Drug Hx

- Octreotide
- Ceftriaxone
- OCP

Allergies

Family Hx :

- Recurrent gall stones
- Haemolytic anaemias

Social Hx :

Income
Occupation
Diet – low fibre diet predispose to gall stones
Alcohol and smoking
Life style - Sedentary

Examination

General Examination

- Weight/Height/BMI
 - Obese → gall stone disease
 - Weight loss → CA Gall bladder
- Dyspnoea (Pneumonia, Acute pancreatitis)
- Febrile (Ascending cholangitis, Acute cholecystitis)
- Icterus
 - Lemon tinge jaundice – haemolytic anaemia
 - Obstructive jaundice

- Intermittant – Chledocholithiasis (obstructive jaundice lasts till the small stone pass through the sphincter of oddi or it disimpacts and falls back to the dilated CBD)
- Cholidocholithiasis → Ascending infection → Inflamed duct system → Obstructive jaundice)
- CA Gall bladder – At the late stage
- Cholidocholithiasis → Chronic back pressure → 2ry biliary cirrhosis or liver failure
- In acute cholecystitis → swollen GB → Press on CBD → Tinge of jaundice
- Pallor – (Peptic ulcer disease, CA gall bladder)
- Look for signs of chronic liver disease (Jaundice, clubbing, leukonychia, ascites, B/L ankle oedema)

Abdominal Examination

Inspection

- Previous surgical scars – Removal of ileum due to IBD (Can result in cholesterol stones)
- Dilated veins
- Ascites
- Visible mass over right hypochondrium – Dilated gall bladder

Palpation

- Acute cholecystitis
 - Tenderness & guarding in the right hypochondrium (Distended, inflamed GB wrapped in inflammatory adhesions to adjacent organs, especially the omentum)
 - Murphy's sign – palpate the abdomen just below the tip of the 9th costal cartilage & ask the pt to take a deep breath. When the liver & the attached gallbladder descend & strike the palpating hand, the pt will experience a sharp pain which prevents further inspiration
 - Palpable enlarged GB, beneath the right costal margin
- Tender abdomen also seen in perforated GB (Chemical peritonitis, Perforated peptic ulcer and acute pancreatitis)
- Hepatitis – Enlarged Liver +/- tenderness

Percussion

- Free fluid – ascites (Peritonitis, Chronic liver disease)

Auscultation

CVS Examination

- Pulse rate/ BP – Indicate haemodynamic stability
- Murmurs

Respiratory system

- Look for reduced chest movements, impaired dullness, reduced air entry, bronchial breathing and crepts to exclude R/S lobar pneumonia.
- Acute pancreatitis → ARDS → Non cardiogenic pulmonary oedema (Fine end inspiratory crepitations)

CNS Examination

- Consciousness
- GCS

Summary

Problem list

Investigations

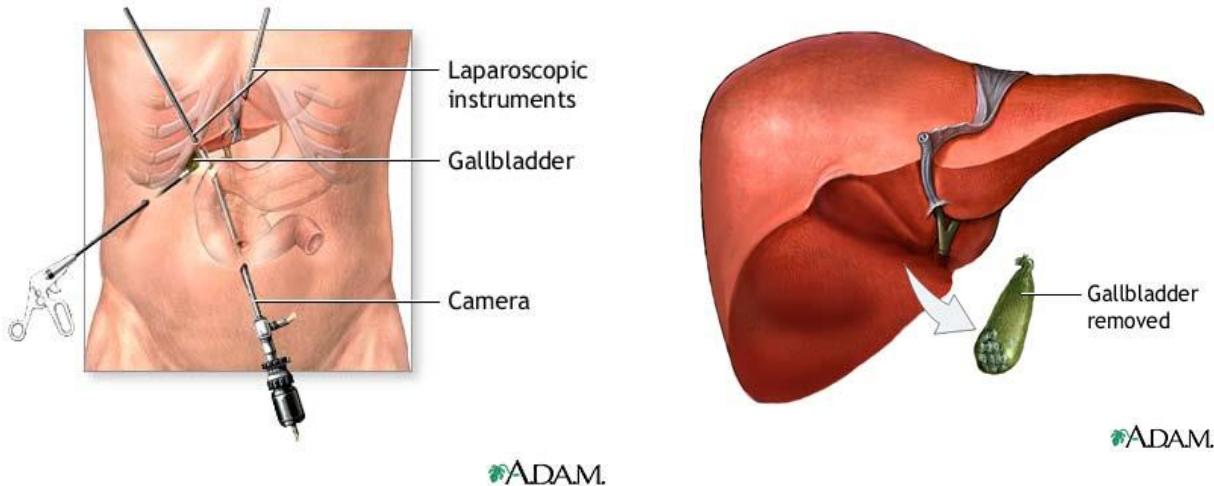
- Basic investigations
 - FBC – Leucocytosis (Acute cholecystitis)
 - CRP – Acute cholecystitis/ Ascending cholangitis
 - Serum bilirubin – Direct & Total (Conjugated bilirubinaemia)
 - SGPT/SGOT – hepatitis
 - PT/INR – If an invasive procedure required
 - BU/SE
 - Grouping & DT
 - RBS
 - UFR
 - ↓ Urobilinogen
 - ↑ Bile salts
 - Red cells – Ureteric colic
- Imaging
 - USS – abdomen
 - Presence of gall stones – Intensely echo-genic foci, which cast a clear acoustic shadow beyond them
 - Thickened wall of GB – Acute or chronic inflammation
 - Dilated CBD (if >7 mm → suggestive of CBD stones)
 - CT is unreliable in detecting stones in bile ducts, especially at the lower end
 - Plain abdominal X-ray
 - Radio-opaque gall stones in 10% cases
 - Occasionally calcified gall bladder – Porcelain gallbladder
 - Erect chest X-ray PA & lateral to exclude
 - Right lower lobe pneumonia
 - Perforated peptic ulcer – Air under the diaphragm
 - ARDS (In acute pancreatitis) – White lung fields
- Specific investigations
 - Blood culture & ABST – When cholangitis present
 - Serum amylase - ↑ in acute pancreatitis
 - Urgent ECG – exclude MI
 - Serum ALP – persistently ↑ in cholidocholithiasis
 - Viral Hepatitis
 - Hep A – IgM antibody
 - Hep B – HBs and HBe antigens

- MRCP (*Magnetic resonance cholangiopancreatography*) – Non invasive procedure which permits visualization of the biliary tree and contained calculi can be detected.
- ERCP (*Endoscopic retrograde cholangiopancreatography*)
 - Endoscopic intubation of the bile ducts through ampulla of vater. (More invasive than MRCP)
 - In addition to visualization can extract the stones. (often after diathermy sphincterotomy opening up sphincter of oddi)
 - Complications – Perforation, bleeding, pancreatitis
- To find out the aetiology
 - Lipid profile
 - Blood picture – To exclude haemolytic anaemia

Treatment

Management of Acute cholecystitis

- Insert IV cannula (16G)
 - Take blood for grouping and cross matching, FBC, RBS, BU/SE
 - IV fluid
 - 0.9% N. Saline 500ml rapid infusion
 - Nil by mouth (or fluids only)
 - Pain relief – opiates (Pethidine 75mg IM)
 - Antibiotics for 3-5 days (Gram –ve cover)
 - Cefuroxime IV 750mg 8/H
 - Bed rest
 - Anti-emetics – Promethazine 25mg IM
 - Monitor pulse, BP, temperature, WBC count
 - Treatment for acute cholecystitis is cholecystectomy
 - Elective cholecystectomy
 - Usually done early.
 - ✓ Urgent cholecystectomy during 1st 72 hrs of admission offers excellent alternative to the patient optimizing recovery and minimizing the disruption of their normal life style
 - ✓ Elective cholecystectomy is performed after 6 weeks, because of undoubtedly danger of further attacks.
 - Routinely performed laparoscopically
 - ✓ Minimal scarring
 - ✓ Rapid recovery
 - ✓ Open surgery may be needed in operative difficulties, anatomical aberrations, and equipment failures
 - Empyema of GB – Emergency cholecystectomy
 - If diagnosis is in doubt → Laparotomy done
 - Cholecystectomy is comparatively easy in 1st 24-48 hours. Dissection is facilitated by oedema of adjacent tissues
 - If > 48 hrs → Difficult surgery due to inflammatory adhesions
- 90% resolve on bed rest with antibiotics and pain relief



Management of chronic cholecystitis

- Cholectomy performed by either laparoscopy or laparotomy.
- Laparotomy approach if laparoscopic approach is not possible
 - Cystic duct is intubated and intra-operative cholangiogram performed by injecting contrast into CBD.
 - If stones present → Common bile duct explored and stones removed. Then latex T-tube is inserted into CBD and check X-Ray performed.
 - T-tube is removed on the 10th day post-operatively after check cholangiogram taken through the tube to confirm
 - The tubes are clear
 - And there's a free flow into the duodenum
- At laparoscopic cholecystectomy
 - Surgeon may or may not perform laparoscopic exploration of CBD or
 - More commonly may elect to wait for a postoperative endoscopic sphincterotomy and extraction of the stones using a Domia basket or balloon technique.
 - In our wards....
 - **Laparoscopic cholecystectomy performed.**
 - **Then intra-operative cholangiogram done. (This is done following ligation of the cystic duct and through the sphincter of oddi)**
 - **If stones present endoscopic sphincterotomy and extraction of the stones using a Domia basket or balloon technique is done.**

Surgical procedures to remove CBD stones

1. ERCP + Sphincterotomy + Basket extraction of CBD stone
2. ERCP + Sphincterotomy + Balloon sweeping
3. ERCP + Sphincterotomy + External Lithotripsy + Balloon sweeping or basket extraction

Indications

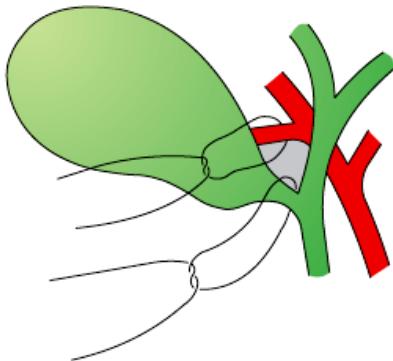
- *Symptomatic gallbladder stones*
- *Acute cholecystitis (calculous & acalculous)*
- *Polyps or tumours of the gallbladder*

Pre-operative preparation

- Informed written consent
- Investigations
 - 2D Echo (CVS)
 - CXR (Res)
 - FBC (Exclude Anaemia)
 - SGPT/SGOT (Exclude abnormal liver function)
 - PT/INR
 - S.creatinine (renal function)
 - Grouping & DT – 2 units of blood
- Prophylactic antibiotics – 2G cephalosporin is appropriate (Cefuroxime 1500mg IV), Give with pre-medication or at the time of induction)
- DVT prophylaxis
 - SC heparin or Graduated compression stockings
- Dental care
- Overnight Fasting
- Steam inhalation
- Pre-anaesthetic medication

Laparoscopic cholecystectomy – Procedure

- The patient is placed supine on the operating table.
- Following induction and maintenance of a general anaesthetic, the abdomen is prepared in a standard fashion.
- Pneumoperitoneum is established.
- A number of techniques are described. The author's preference is to use an open subumbilical cut-down with direct visualisation of the peritoneum to place the initial port. This port will function as the camera port. Many surgeons use a 'closed' technique using a Verres needle to establish pneumoperitoneum prior to placing the initial trocar.
- Additional operating ports are inserted in the sub-xiphoid area and in the right subcostal area. The patient is placed in a reverse Trendelenburg position, slightly rotated to the left. This exposes the fundus of the gall bladder, which is retracted towards the diaphragm.
- The neck of the **gall bladder is then retracted towards the right iliac fossa exposing Calot's triangle**. This area is laid wide open by dividing the peritoneum on the posterior and anterior aspects. The **cystic duct is carefully defined, as is the cystic artery**. The gall bladder is separated from the liver bed for about 2 cm to allow for confirmation of the anatomy. Unless there are specific indications.
- **A routine cholangiogram is not performed. (If Dr. Chandika comes → this is routinely done to exclude distal CBD stones)** However, if doubt exists regarding the anatomy, a cholangiogram is warranted.
- Once the anatomy is clearly defined and the triangle of Calot has been laid wide open, **the cystic duct and artery are clipped and divided**. The gall bladder is then removed from the gall bladder bed by sharp dissection and, once free, removed via the umbilicus. An endobag may be used for extraction of the gall bladder to prevent contamination of the umbilical wound.
- Recovery after laparoscopic cholecystectomy is rapid – 80% of patients are discharged within 24 hours and the remainder by day 2. Any untoward symptoms require immediate investigation



Ligatures passed and tied around the cystic artery and cystic duct.
The grey shaded area represents Calot's triangle.

Post-operative care

- Monitoring pulse, respiration, drains, bleeding & jaundice
- Pethidine 75 mg IM SOS
- IV fluids 0.9% NaCl 500ml, 5% dextrose 2500ml
- Sips on demand, light diet vespa
- Prop-up
- Steam inhalation
- Chest physiotherapy
- A shower next day
- Remove the drains except the T-tube which is kept for at least 10 days in cases of CBD exploration.
Following CBD exploration → Sphincter goes into spasms → Increase back pressure in biliary tree → To drain that T-tube is inserted → When the spasms resolves around after 10 days, drainage of bile through the T-tube is reduced)
- Discharge after 24-48 hours
- Review at clinic with histology report

Complications of Cholecystectomy

2 special dangers after cholecystectomy, whether performed by laparotomy or laparoscopy

1. Leakage of bile result from

- a. Injury to bile canaliculi in the GB bed or liver
- b. Injury to common hepatic or common bile duct
- c. Slipping of ligature or clip from the cystic duct
- d. Leakage from the CBD after exploration

(ERCP may identify the site of the leak, and the temporary stenting will ensure the adequate biliary drainage thus allowing the bile fistula to close spontaneously. If this doesn't occur, further exploration may be required)

2. Jaundice. This may be due to the following

- a. Missed stones in the CBD
- b. Inadvertent injury to the CBD
- c. Cholangitis or associated pancreatitis

(Residual stones in the CBD may require operative removal. Majority can be removed by ENDOSCOPIC SPHINCTEROTOMY or, if a T-tube still present in the CBD, by means of a Burhenne basket passed along the track formed by the tube under X-ray control)

Other complications

- Bile duct stricture
- Wound haematoma & sepsis

Carcinoma of the gallbladder

Pathology

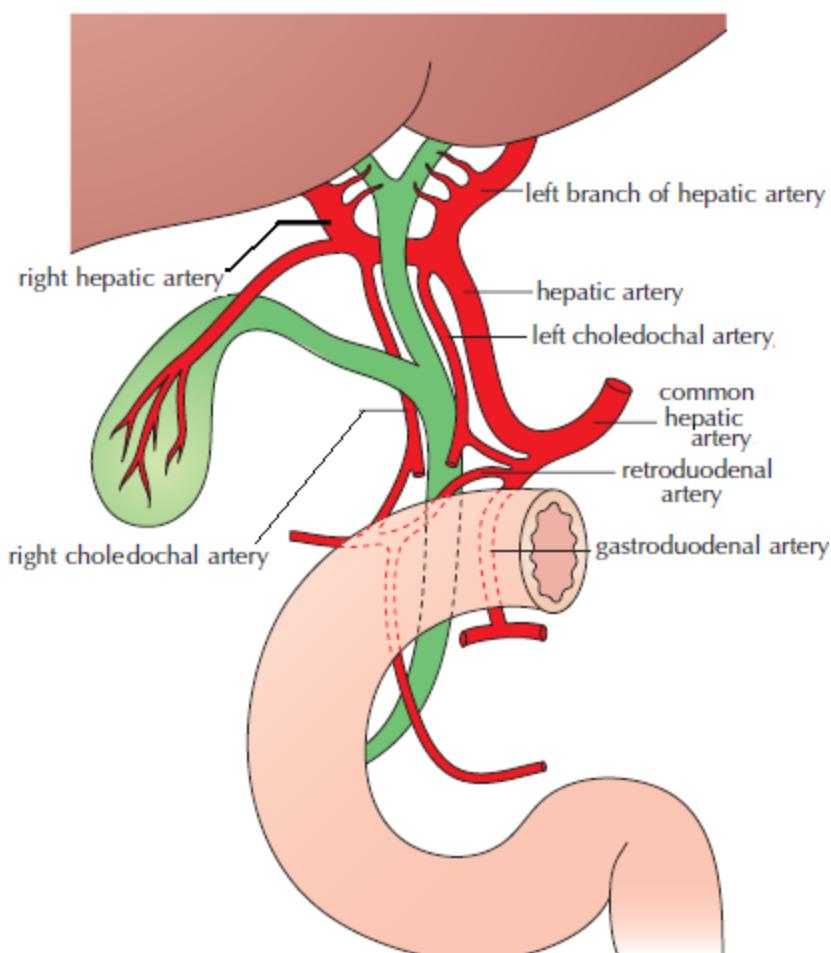
- Relatively uncommon tumour.
- Associated in about 85% of cases with the presence of gallstones.
- 50% of porcelain gallbladder are associated with this
- This is 4 times commoner in women than men
- 90% are adenocarcinoma
- 10% squamous carcinoma
- Spread
 - Local invasion – Liver & its ducts
 - Lymphatic spread – To the nodes in porta hepatis
 - Portal vein dissemination to the liver may occur

Clinical Features

- Picture resembling chronic cholecystitis with RHC pain, nausea, vomiting.
- Weight loss.
- Later progressing to obstructive jaundice. At this stage palpable mass may be present in gallbladder region.

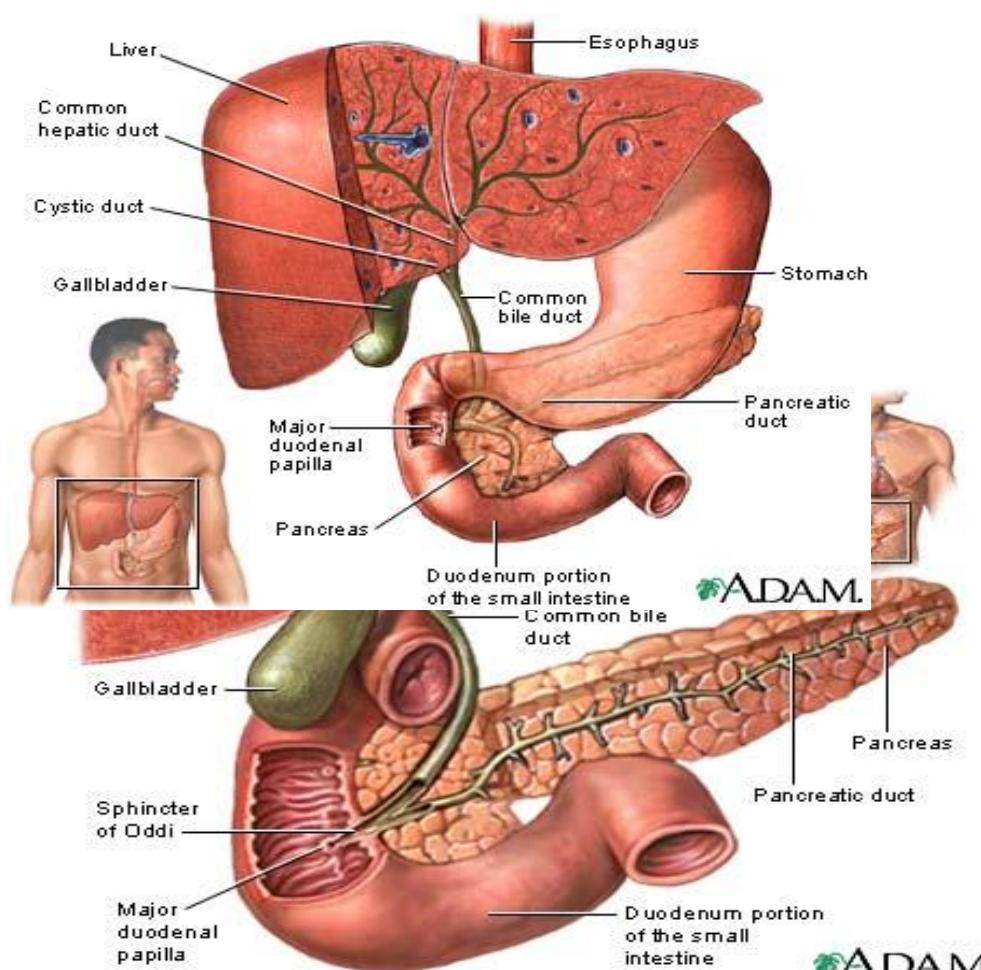
Management

- Occasionally, cholecystectomy performed for stones reveals the presence of an unexpected tumour. Under this long term survival may follow.
- But most are present late with liver metastases and nodal spread.
 - Radical liver resection or local excision only rarely possible.
 - Therefore usually poor prognosis.
 - Death within months.

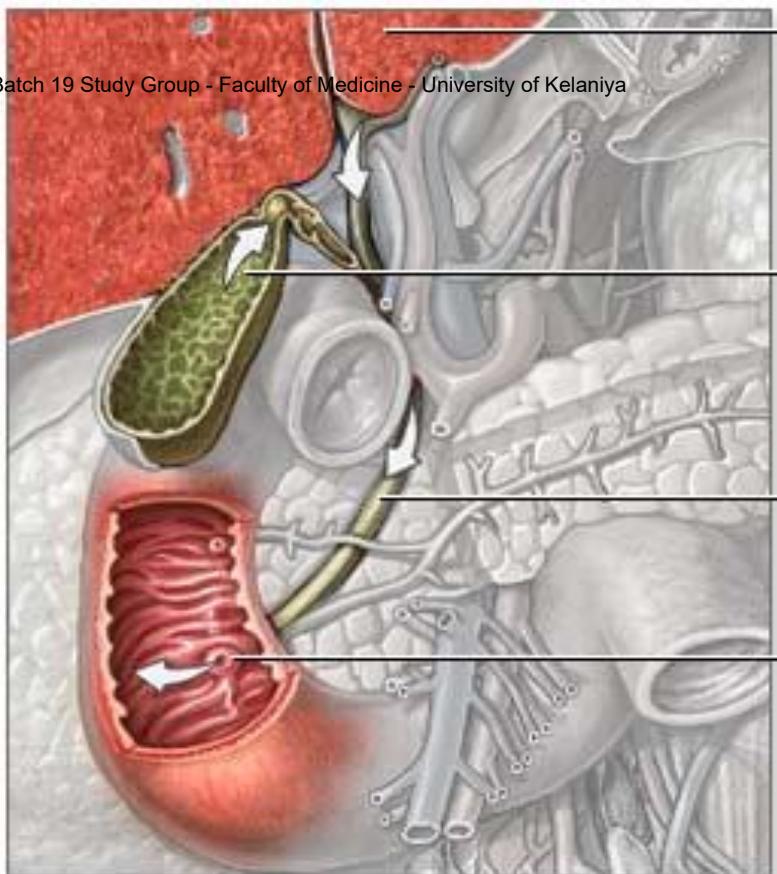


Discussion

- Anatomy of the gall bladder



Liver

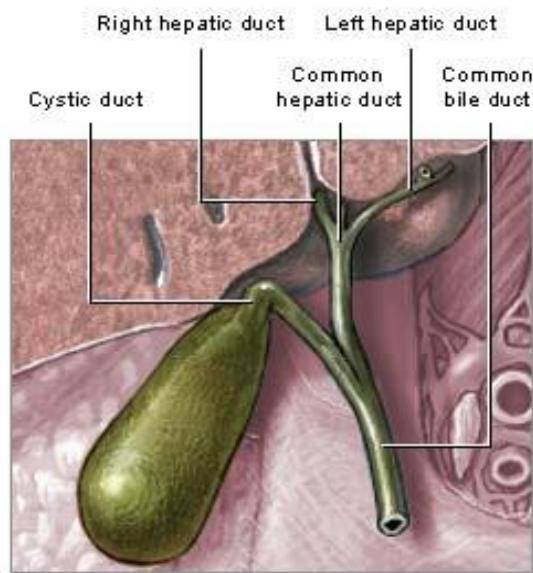
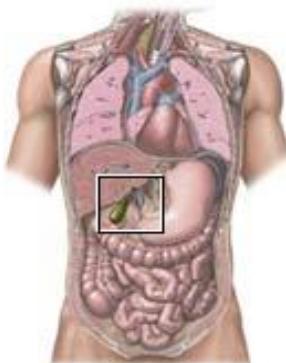


Gallbladder

Common
bile duct

Major duodenal
papilla

ADAM.

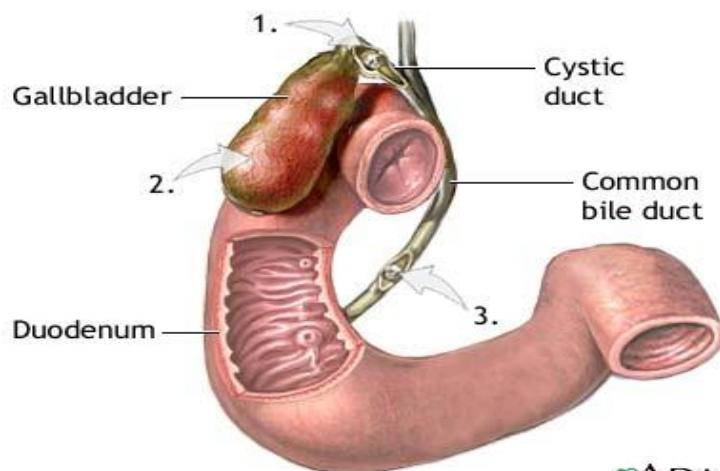


ADAM.

- Formation of gallstones depend on
 - Secretion of lithogenic bile
 - Bile which is supersaturated with cholesterol
 - Bile – cholesterol + phospholipids (principally lecithin)+ bile salts + water + conjugated bilirubin
 - Cholesterol is solubilized in bile as a micelle with lecithin & bile salts
 - Only when the appropriate ratios of lecithin & bile salts are present will cholesterol remain in solution & be prevented from forming crystals

- So low bile salts leads to supersaturation
- Metabolic abnormalities in the liver predispose to secretion of lithogenic bile
- High protein, high fat diet, Contraceptive pills, Pregnancy, obesity, low residue diet with inadequate fibre, loss of terminal ileum (interrupt enterohepatic circulation – results low bile salts)
- **Sepsis in the biliary tree**
 - The liver is thought to be consistently trapping small numbers of organisms from the portal or systemic blood
 - Some of these organisms may be excreted in the bile, & if they find an appropriate nidus (cholesterol crystals, mucus, epithelial debris) they will persist & proliferate.
 - They deconjugate & precipitate bile salts so that a mixed stone results
 - Sepsis in biliary tree that contain stones may cause bouts of
 - Acute cholecystitis (gallbladder infection)
 - Acute (ascending) cholangitis (bile duct infection)
- **Anatomical abnormalities that predispose to stasis**
 - Cul-de-sac
 - Incompletely drained branch of the tree
- **Abnormalities of the gallbladder epithelium**
- Types of gall-stones
 - **Cholesterol (20%)**
 - Occur due to lithogenic bile
 - Occur either as
 - A solitary, oval stone
 - Two stones (one indenting other)
 - Multiple mulberry stones associated with a strawberry gallbladder
 - Radio-lucent
 - **Bile pigment (5%)**
 - Small, black, irregular, multiple, gritty & fragile
 - Occur in haemolytic anaemias where excess of circulating bile pigment is deposited in the biliary tract
 - Radio-opaque
 - **Mixed (75%)**
 - Multiple, faceted one against the other & can often be grouped into two or more series, all of the same size, suggesting 'generations' of stones

- Where



the gall stones can act

1. Obstruction of the cystic duct leading to severe abdominal pain (biliary colic).
2. Infection or inflammation of the gallbladder (cholecystitis).
3. Blockage of the biliary ducts leading to the duodenum (biliary obstruction).

Choledocholithiasis & cholangitis

- Most stones in the common bile duct have originated in the GB, less commonly they arise in the intrahepatic ducts or common bile duct
- May be symptomless
- More often there are attacks of biliary colic accompanied by
 - Obstructive jaundice
 - Nearly always preceded by biliary colic
 - Jaundice will tend to fluctuate from day to day when ball-valve type of obstruction exist
 - Clay coloured stools & dark urine
- If the obstruction is not relieved, the back pressure in the biliary system results in secondary biliary cirrhosis & liver failure

Ascending cholangitis

- If infection of common bile duct supervenes, the jaundice & pain are complicated by rigors, a high intermittent fever & severe toxæmia (intermittent hepatic fever of Charcot)
- Tenderness in the right hypochondrium
- Organisms usually E.coli

Investigations

- Serum bilirubin ↑
 - Serum alkaline phosphatase ↑
 - LFT - May indicate associated liver cell damage
 - Blood culture & ABST
 - WBC
 - USS - May show dilatation of the bile duct & stones
 - PTC & ERCP - Show degree of dilatation & site of stones
-] When cholangitis suspected

Management

- Supportive
 - Fluid & electrolytes replacement
 - Antibiotics if ascending cholangitis present
 - Monitoring
- Urgent removal of stones done in unabating cholangitis, with high fever, positive blood culture & systemic disturbances
- Less urgency in pts with obstructive jaundice

Methods of removing stones

- Endoscopic removal (ERCP with sphincterotomy)

- Considered in pts who present a very poor operative risk & cannot be improved in short term
- Popular in pts over 70 yrs
- Overall success rate is over 90%
- **Open operation**
 - Appropriate in younger & fitter pts
 - It must consist of
 - Removal of the GB & its contained stones
 - Adequate duct exploration
 - Drainage of the biliary tree

Courvoisier's Law'

'If in the presence of jaundice the gallbladder is palpable, then the jaundice is **unlikely** to be due to stone'

- ✚ If the obstruction is due to a stone
 - The bladder is usually thickened and fibrotic → So it doesn't distend
 - Calculus obstruction is not usually complete → This allows some bile flow in to duodenum with decompression of the gallbladder
- ✚ If the obstruction due to other causes (Eg : CA head of the pancreas)
 - Usually gall bladder is normal
 - Therefore it can dilate in the presence of biliary obstruction

EXCLUSIONS TO ABOVE THEORY

Only rarely is the gallbladder dilated when the jaundice is due to stone

- Stone impacts on Hartmann's pouch → Mucocoele → At the same time a 2nd stone in CBD may produce Jaundice

Or when a stone forms in CBD in-situ

- Gallbladder is normal and it can get dilated in the presence of the obstruction by the CBD stone

However, in CA of the bile ducts arising above the origin of the cystic duct, the gallbladder, distal to the obstruction, will be collapsed and empty

THYROID ENLARGEMENT

FROM YOUR HISTORY AND EXAMINATION TRY TO ANSWER THESE QUESTIONS

1. Is it a swelling of the gland
2. If yes, what type of a goiter
3. What is the functional state
4. Any evidence of malignancy
5. Any other complications
6. Fitness for surgery

General information;	
Name	P/C ;
Age	goiter; cosmesis, compression
Sex	functional problems(control)
Residence	fear of malignancy(CA)
	recurrence(come back)

Analyze the P/C;

- Onset (event)
- Progression (rapid growth)
- Duration
- What happen in between(investigations, treatments)

(1) & (2) mainly by examination

3. What is the functional state

	Hyperthyroid	Hypothyroid
General	Irritability/behaviour change Restlessness Goitre Heat intolerance Insomnia Excessive sweating	Tiredness/malaise Hoarseness of voice Cold intolerance Change in appearance Goitre Constipation
GIT	Weight loss Increased appetite Vomiting Diarrhoea	Weight gain Anorexia
CVS	Breathlessness Palpitation	

CNS	Tremor Eye complaints(Diplopia)	Poor memory Depression Psychosis Coma Deafness
MUCULO SKETETAL	Muscle weakness Proximal myopathy	Arthralgia Myalgia Muscle weakness/Proximal myopathy
REPRODUCTIVE	Oligomenorrhoea Loss of libido	Menorrhagia or oligomenorrhoea in women Poor libido
OTHERS	Itching Thirst Onycholysis Tall stature (in children) Sweating	Dry, brittle unmanageable hair Puffy eyes Dry, coarse skin

4. ANY EVIDENCE OF MALIGNANCY

LOCAL SPREAD	LN	SYSTEMIC
Hoarseness - RLN Stridor, haemoptysis-trachea, Dysphagia-oesophagus (rare)	Any palpable LN	Lungs-haemoptysis, cough Bones-bone pain, scalp lump backache, hypercalcaemic features Liver Brain

- Recent rapid enlargement of a goiter

5. Any other complications

- Dysphagia(rare) - Compression, local infiltration of a CA
- Difficulty in breathing - Tracheal compression, kinking, infiltration by local spread

AETEOLOGY

	HYPERTHYROID	EUTHYROID	HYPOTHYROID
MNG	2 nd ry thyrotoxicosis (Plummer's)	Goitrogens I deficiency Physiological Defective synthesis	
DIFFUSE	1 ^{ry} thyrotoxicosis (Grave's)		Post thyroiditis
SOLITARY	Toxic adenoma	Malignant potential	Post thyroiditis

Grave's

- Other associated AI conditions:
Vitiligo, Type 1 DM
- Ophthalmopathy-common
- Dermopathy
- Pre-tibial myxoedema,
acropachy-rare
- F>M

Sporadic

- Biosynthetic defects
- F>M
- There may be a family Hx
- Peak at puberty and adult life

Endemic

- I₂ deficiency
- Goitrogens
- >10% of the area population is affected

Plummer's

- F>M
- CVS features predominate(AF, HF)

What have done up to now?

- Investigations- why, what, results and actions
- Treatments –when, what, response

PMHx

- Other AI diseases (DM, Vitiligo)
- Features of phaeochromocytoma – Episodic headache, sweating, palpitation
- Features of hyperparathyroidism

PSHx

- Any surgical procedure on gland

Dietary Hx

- Goitrogens (cabbage, manioc)
- I₂ intake

FMHx

- Thyroid CA
- Enlargement

SHx

- Functional problems due to condition
- Evidence of endemic goiter –peoples with goiters in the area

EXAMINATION

2. What is the type of goiter-MNG, Diffuse, Solitary and other relevant?

Examination of the gland

- Procedure –see the short case leaflet

Comment on;

- MNG, diffuse or solitary
 - U/L → tracheal deviation
 - B/L → compression
- Extent of the gland
- Evidence of retrosternal or retrotracheal extention
 - Retrosternal extension → pemberton's sign
- Features of thyroiditis - Inflammatory features
- Features of malignancy-
 - ✓ hard, skin attachment
 - ✓ Local invasion-carotids (berry's sign: infiltration of the carotid sheath → impalpable carotid pulse)
 - ✓ Muscle-reduced mobility on swallowing
 - ✓ LN spread-
 - central group
 - Upper anterior deep cervical + posterior deep cervical
 - Supraclavicular
 - Posterior triangle
 - ✓ Bone tenderness
 - ✓ Scalp lumps



Eye signs

- Lid retraction – can see the upper nictitating membrane
- Exophthalmos
- Proptosis
- Ophthalmoplegia
- Cheimosis

Hand

- Sweating
- Palmer erythema
- Tremors
- Clubbing
- Pulse : rate, bounding

WHO grading of goiter

Grade 1;

- (a) palpable goiter
- (b) visible with neck extended

Grade 2 : visible from near

Grade 3 : visible from afar

Grade 4 : giant goiter

- To find out the functional state- see the medicine note

Investigations

Investigation of any goitre

- Hormone assay
- USS of neck
- FNAC of thyroid

1. Function of the gland

- TSH(3rd generation-ultrasensitive)-also can diagnose subclinical disease,
- Free T₃/T₄

	<u>Low TSH</u>	<u>Normal TSH</u>	<u>High TSH</u>
<u>High FT4</u>	Hyper thyroid	TSH-secret. tumour (rare)	TSH-secret. tumour
<u>Norm FT4</u>	Sub-clinical hyper or T3-toxic.	Euthyroidism	Sub-clinical hypothyroidism
<u>Low FT4</u>	Hypopituitarism (rare)	Hypopituitarism	Primary hypothyroidism

2. Size and morphology of the gland

- USS
 - ✓ Find out cystic or solid
 - ✓ Can refine clinical findings
 - ✓ Help to follow up suspicious nodules
 - ✓ Locate intra-cystic growth

3. Nature and pathology of the swelling (to exclude malignancy)

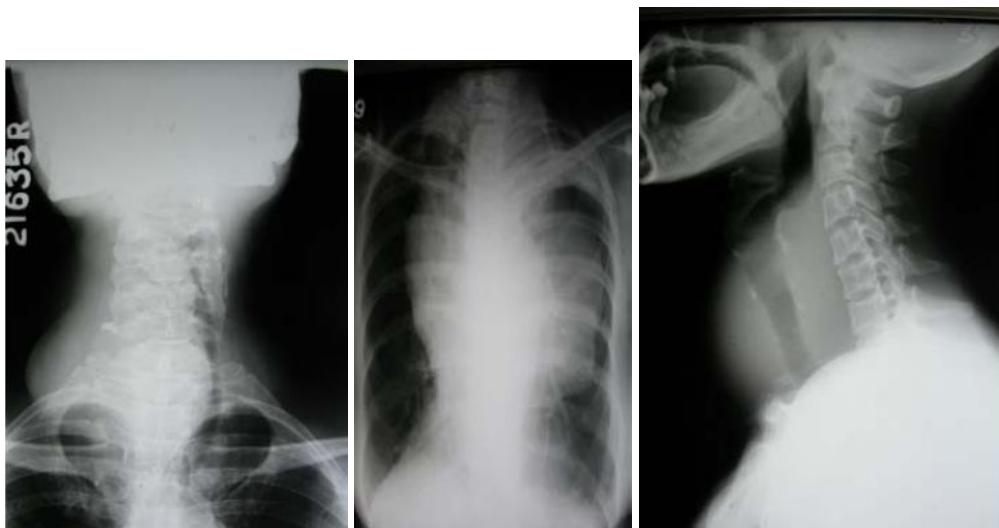
- FNAC

4. Special investigations

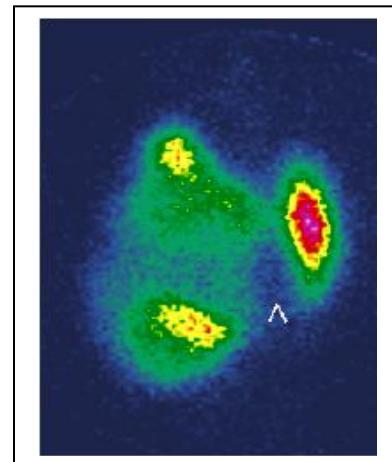
- Thyroid auto anti bodies
 - ✓ Microsomal thyroid antibodies/Thyroid peroxidase antibodies (TPO)
 - ✓ Anti-thyroglobulin AB
 - ✓ Anti TSH receptor AB

5. Imaging

- X-ray neck-
 - ✓ Tracheal deviation(AP)
 - ✓ Tracheal compression and retro tracheal extension- lateral
- X-ray thoracic inlet -retro sternal extension
- CT/MRI
 - Extent and ramification of the gland in the neck and chest
 - Retro-sternal extension
 - Infiltration of carotid



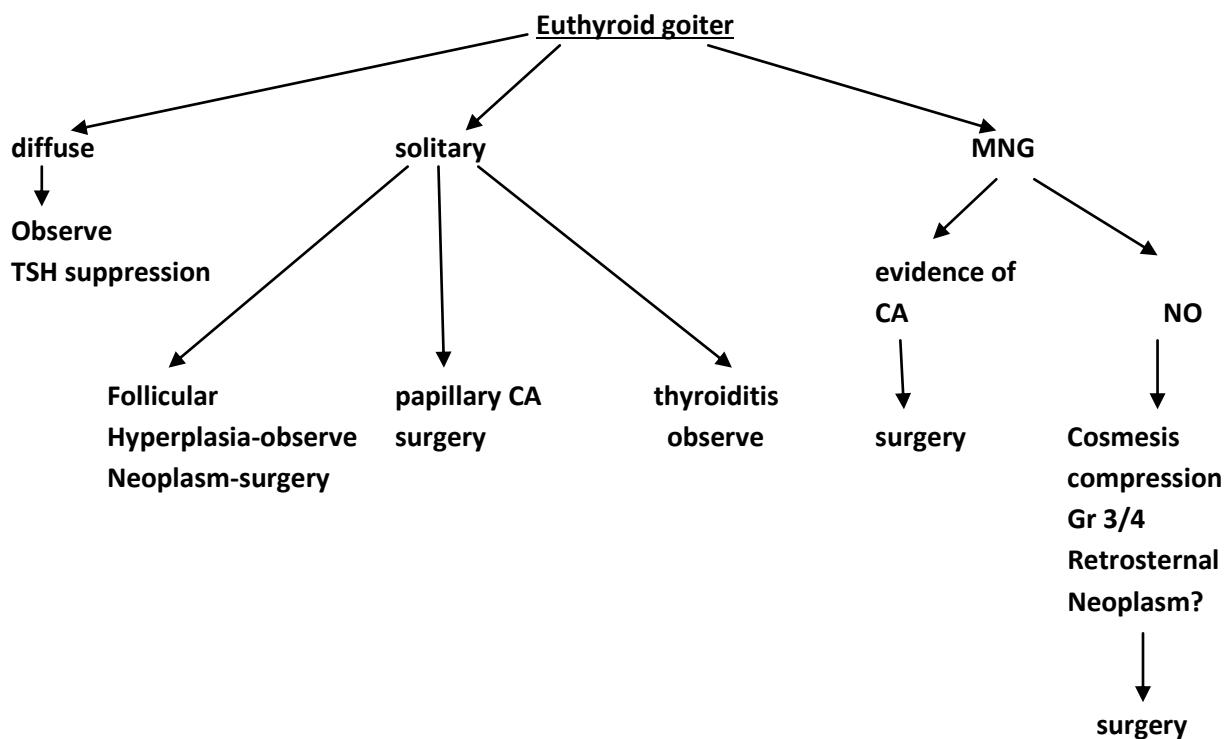
- Isotope scan
 - ✓ No use in differentiating malignant from benign (5% warm nodules are malignant & 80% of cold nodules are benign)
 - ✓ Detect activity level (focal or diffuse)
 - ✓ Active metastatic deposits
 - ✓ Follow up after surgery (especially CA)
 - ✓ Retrosternal extension



Management

Indications for surgery are

1. **Obstruction of airway**
2. **Retrosternal extension**
3. **Suspicion of malignancy**
4. **Secondary toxicosis**
5. **Cosmetic**
6. **Oesophageal compression (very rare with benign disease)**



Hyperthyroid goiters

MNG with toxicity	Grave's	Thyroiditis	Toxic nodule
Make the patient euthyroid medically	Refer the medicine note Mainly manage medically	Manage with propranolol	Hemi-thyroidectomy
Once euthyroid	Sx-total thyroidectomy Eye signs get worse with subtotal thyroidectomy		
✓ Surgery Near/total thyroidectomy (operation of choice) ✓ I^{131} ✓ Continue medical treatment(age, co-morbidities)			

Thyroid carcinoma

Evidence to suggest CA

- Hard goitres
- Solitary nodules in the extremes of age
- Dominant and Solitary nodules.
- Recent, rapid enlargement of a goitre
- A family history of thyroid cancer
- Goitre in a non endemic environment
- Onset of a goitre after menopause or in the elderly
- Evidence of local spread, fixity to adjacent structures
 - Diminished mobility on swallowing
 - Hoarseness of voice
 - Impalpable carotid pulse (Berry's Sign)
 - Airway (stridor) and swallowing difficulty
- Enlargement of the draining lymph nodes
- Goitre with a metastatic deposits
- Goitres in Males
- Combination of above

TNM Classification for thyroid cancer

- | | |
|----|---|
| T1 | 1 cm or less |
| T2 | >1-4cms |
| T3 | > 4cms |
| T4 | extension through the capsule of the thyroid. |
| | |
| N0 | nodal spread not detected |
| N1 | nodal spread present |
| | |
| M0 | blood stream metastases absent |
| M1 | blood stream metastases present. |

Type	Treatment
Papillary CA	Near/total thyroidectomy + central node dissection
Follicular CA	Near total/total thyroidectomy
Medullary CA	Total thyroidectomy + central & bilateral cervical node dissection
Anaplastic CA	Generally not attained surgically Radiation remains the mainstay

Pre operative preparation

1. Inform written consent
2. Make the patient clinically and bio chemically euthyroid
 - Carbimazole-anti thyroid
 - Propanolol-symptomatic control
 - Lugol's iodine- reduce vascularity
3. Optimize other co-morbidities
 - BA, DM, HT
4. ENT referral
 - To detect any RLN palsy, vocal cord problems prior to surgery and for legal protection
 - Done by ENT surgeon
 - With a indirect laryngoscope
5. Investigations
 - ✓ Thoracic inlet X'ray
 - To recognize tracheal deviation, kinking and tracheal compression
 - ✓ Hb- at least 10g/dl
 - ✓ Blood grouping and saving
 - ✓ Pre op serum Ca⁺²
 - Due to risk of accidental parathyroidectomy causing hypocalcaemia
 - Pre requisites
 - Calm the pt
 - Collect to a acid wash tube
 - Albumin correction has to be done

Group A investigation (well being)

Hb, grouping and saving, Platelet, PT/INR, ECG, CXR, FBS

Group B investigations (for the surgery)

X-ray Thoracic inlet, TSH, levels FNAC of thyroid, S. Ca

6. 6 hours fasting

Principles of thyroidectomy

1. GA with endotracheal intubation.
2. Sand bag under the shoulder and head positioned on a ring, neck extended, table foot down.
3. Skin crease incision about two finger breaths above the sternal notch, it should reach the sternocleidomastoid on each side.
4. Raised skin flaps (including subcutaneous fat and platysma) to the thyroid notch superiorly and sternal notch inferiorly, flaps may then be held by a self retaining retractor (joll's retractor).
5. Make a vertical incision through the deep fascia between the strap muscle and retract the strap muscle. If the goiter is large strap muscles are divided at their upper extremity because there nerve supply enters the lower part.
6. Divide the middle thyroid vein.
7. Draw down the upper pole and identify the superior thyroid vessel.
8. Ligate the superior thyroid vessel close to upper pole to avoid the external branch of superior laryngeal nerve.
9. Draw the gland medially and dissect the lateral connective tissue and identify the inferior thyroid artery.
10. Identify the RLN
11. Ligate the inferior thyroid artery as its' branches enters to the gland avoiding the RLN (lateral to RLN)
12. Ligate the inferior thyroid vein
13. The lobe is now free and may be removed by dividing the isthmus.
14. Achieve the haemostasis.
15. Suture the deep fascia between the strap muscle.
16. Close the wound in layers with suction drainage.

Thyroid operations

All thyroid operations can be assembled from three basic elements:

- Total lobectomy
- Isthmusectomy
- Subtotal lobectomy

**Total thyroidectomy = $2 \times$ total lobectomy +
isthmusectomy**

**Subtotal thyroidectomy = $2 \times$ subtotal lobectomy +
isthmusectomy**

**Near-total thyroidectomy = total lobectomy +
isthmusectomy + subtotal
lobectomy**

Lobectomy = total lobectomy + isthmusectomy

Post-operative management

- QHT
- Monitor PR, RR, and BP
- Observe for any difficulty in breathing or stridor
- Monitor drains- (Not for Prof. RF)
- Pain relief
- Symptomatic management

Postoperative complications

1. Haemorrhage

- A tension haematoma deep to the cervical fascia

2. Respiratory obstruction

- Most cases are due to laryngeal oedema
 - ✓ Important cause is a tension haematoma
- Very rarely due to collapse or kinking of the trachea
- Trauma to the larynx by anaesthetic intubation and surgical manipulation
 - ✓ especially vascular goiters, may cause laryngeal oedema without haematoma
- U/L or B/L RLN paralysis will not cause immediate respiratory obstruction
 - ✓ Unless laryngeal oedema is also present, but they will aggravate the obstruction.
- If releasing the tension haematoma does not immediately relieve airway obstruction, the trachea should be intubated at once.
- An endotracheal tube can be left in place for several days; steroids are given to reduce oedema and a tracheostomy is rarely necessary

3. Recurrent laryngeal nerve paralysis

- Unilateral or bilateral, transient or permanent

4. Thyroid insufficiency

5. Parathyroid insufficiency

- Removal of parathyroid glands
- infarction through damage to the parathyroid end-artery(more important)
- often both

6. Thyrotoxic crisis (storm)

- An acute exacerbation of hyperthyroidism
- Occurs if a thyrotoxic patient has been inadequately prepared for thyroidectomy

7. Wound infection - A subcutaneous or deep cervical abscess should be drained

8. Hypertrophic or keloid scar

- Is more likely to form if the incision overlies the sternum.
- Intradermal injections (If corticosteroid should be given at once and repeated monthly if necessary)

9. Stitch granuloma.

- Occur with or without sinus formation and after the use of non absorbable suture material.
- Absorbable ligatures and sutures must be used throughout thyroid surgery.
- Some surgeons use a subcuticular absorbable skin suture rather than the traditional skin clips or staples.
- Skin staples should be removed in less than 48 hours.

Immediate

- Primary haemorrhage
- Damage to surrounding structures

Early

- Reactional bleeding
- Laryngeal oedema
- Thyrotoxic crisis
- Tracheomalacia

Intermediate

- Hypothyroidism
- Recurrent laryngeal N palsy
- External laryngeal N palsy

Late

- Hypothyroidism
- Recurrence

Follow up in thyroid CA

Differentiated CA

- Obtain histology report-assess the risk(high or low)
- Obtain S. thyroglobulin level 6 weeks after the surgery
- Discuss with oncologist
- Map out residual disease with I¹³¹ scan
- Decide on I¹³¹ or other therapy

Medullary CA

- Most will require further(adjuvant) therapy
- Monitor with calcitonin levels

DISCUSSION

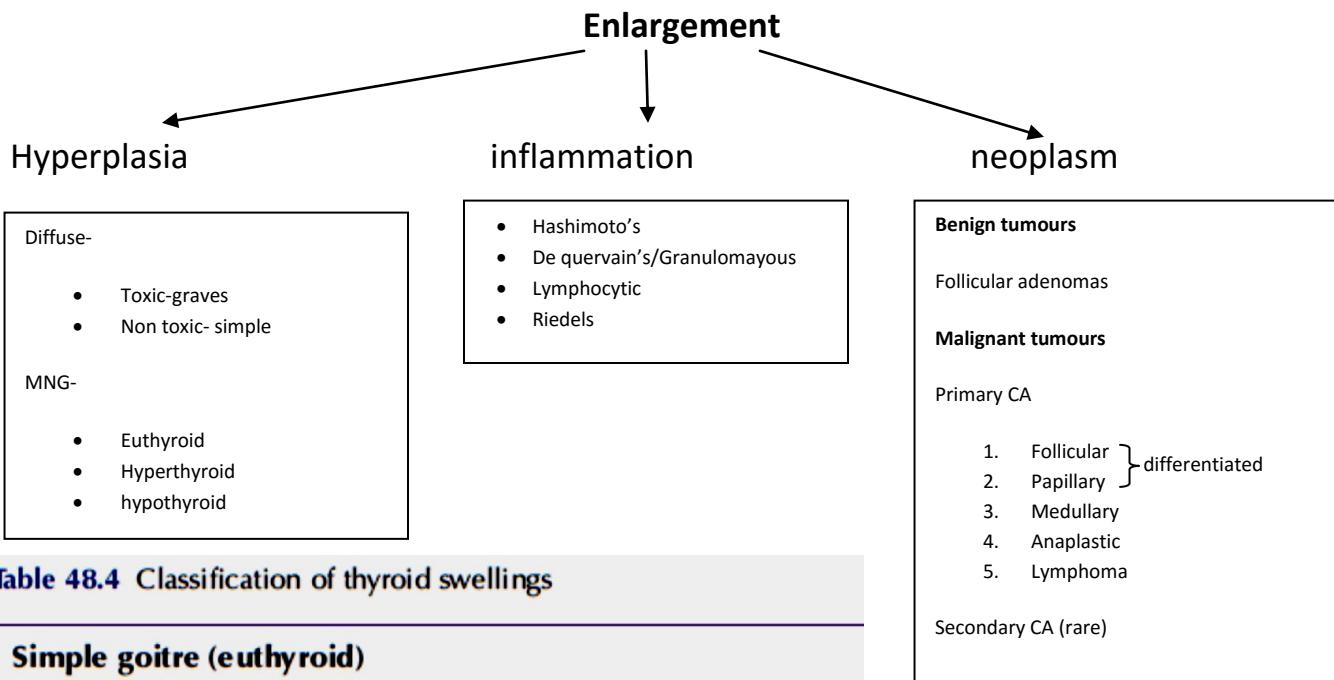


Table 48.4 Classification of thyroid swellings

Simple goitre (euthyroid)

Diffuse hyperplastic

 Physiological

 Pubertal

 Pregnancy

Multinodular goitre

Toxic

Diffuse

 Graves' disease

Multinodular

Toxic adenoma

Neoplastic

Benign

Malignant

Inflammatory

Autoimmune

 Chronic lymphocytic thyroiditis

 Hashimoto's disease

Granulomatous

 De Quervain's thyroiditis

Fibrosing

 Riedel's thyroiditis

Infective

 Acute (bacterial thyroiditis, viral thyroiditis, 'subacute thyroiditis')

 Chronic (tuberculous, syphilitic)

Other

 Amyloid

Simple goiter

Aetiology

- Stimulation of the thyroid gland by TSH (microadenomas in pituitary-rare)
- Low level of circulating thyroid hormones
 - ✓ Dietary deficiency of iodine-endemic
 - Daily requirement of iodine is about 0.1—0.15 mg
 - ✓ Defective hormone synthesis-sporadic
 - There is often a family history suggesting a genetic defect
 - Environmental factors may compensate (high I intake areas-no goiter)
 - ✓ Goitrogens-
 - TSH is not the only stimulus to thyroid follicular cell proliferation
 - Other growth factors including immunoglobulins exert an influence
 - ✓ Vegetables of the brassica family (cabbage)-thiocyanate
 - ✓ Drugs such as para-aminosalicylic acid (PAS)
 - ✓ Antithyroid drugs
 - ✓ Thiocyanates and perchlorates interfere with iodide trapping
 - ✓ Carbimazole and thiouracil - interfere with the oxidation of iodide and the binding of iodine to tyrosine
 - ✓ Iodides in large quantities are goitrogenic because they inhibit the organic binding of iodine and produce an iodide goiter

Stages in goitre formation are:

- Persistent growth stimulation causes diffuse hyperplasia;
 - ✓ Which may persist for a long time but is reversible if stimulation ceases;
- Later, as a result of fluctuating stimulation, a mixed pattern develops with areas of active lobules and areas of inactive lobules;
- Active lobules become more vascular and hyperplastic until haemorrhage occurs, causing central necrosis and leaving only a surrounding ring of active follicles;
- Necrotic lobules coalesce to form nodules filled with either iodine-free colloid or a mass of new but inactive follicles;
- Continual repetition of this process results in a nodular goitre. Most nodules are inactive and active follicles are present only in the internodular tissue.

Diffuse hyperplastic goitre

- ✓ First stages of the natural history.
- ✓ The goitre appears in childhood in endemic areas but,
- ✓ In sporadic cases, it usually occurs at puberty when metabolic demands are high — puberty goitre
- ✓ If TSH stimulation ceases, the goitre may regress, but tends to recur later at times of stress such as pregnancy.
- ✓ The goitre is soft, diffuse and may become large enough to cause discomfort.
- ✓ A colloid goitre is a late stage of diffuse hyperplasia when TSH stimulation has fallen off and when many follicles are inactive and full of colloid

Nodular goitre

- ✓ Nodules are usually multiple, forming a multinodular goitre.
- ✓ Occasionally, only one macroscopic nodule is found, but microscopic changes will be present throughout the gland: this is one form of a clinically solitary nodule.
- ✓ Nodules may be colloid or cellular and cystic degeneration and haemorrhage are common, as is subsequent calcification.
- ✓ Nodules appear early in endemic goitre and later (between 20 and 30 years) in sporadic goitre,
- ✓ Although the patient may be unaware of the goitre until the late 40s or 50s.
- ✓ All types of simple goitre are far more common in the female than in the male and the presence of oestrogen receptors in normal thyroid tissue and in nodular goitre is relevant.

Neoplasms of the thyroid

- **Benign tumours**
 - ✓ Follicular adenomas-present as clinically solitary nodules
- **Malignant tumours** (majority of primary growths is carcinomas)
 - ✓ Primary CA
 1. Follicular
 2. Papillary
 3. Medullary
 4. Anaplastic
 5. Lymphoma
 - ✓ Secondary CA(rare)
 1. Direct invasion from adjacent structures-oesophagus
 2. Blood born-breast, colon and kidney and from melanomas

Aetiology of malignant thyroid tumours

- ✓ **Irradiation** of the thyroid in childhood – Differentiated (papillary)
- ✓ Follicular carcinoma is high in endemic goitrous areas - **TSH stimulation**
- ✓ Malignant lymphomas can occur in autoimmune thyroiditis-**lymphatic infiltration**
- ✓ It is likely that all lymphomas of the thyroid arise in glands affected by such **thyroiditis**

Clinical features of thyroid neoplasms

- ✓ Thyroid swelling-commonest
 - Anaplastic - hard, irregular & infiltrating
 - Differentiated - firm and irregular-difficult to differ from benign swelling
- ✓ Enlarged cervical LN - may in papillary carcinoma
- ✓ RLN paralysis – may in locally advanced disease
- ✓ Small papillary tumours may be impalpable (occult carcinoma) even when lymphatic metastases are present (so-called lateral aberrant thyroid).
- ✓ Pain often referred to the ear- infiltrating

Diagnosis of CA

- **Clinical examination**
 - ✓ Most cases of anaplastic carcinoma can differentiate from Riedel's only by biopsy.
 - ✓ Multinodular goitre, and solitary nodules, particularly in the young male - not easy to exclude
 - ✓ localised forms of granulomatous thyroiditis and lymphadenoid goitre may simulate carcinoma
- **Isotope scanning**
 - ✓ Failure to take up radio-iodine is characteristic of almost all thyroid carcinomas
 - ✓ But occurs also in degenerating nodules and all forms of thyroiditis
- **Thyroid antibody**
 - ✓ Titres are often raised in carcinoma

Incisional biopsy may cause seeding of cells and local recurrence

Papillary carcinoma

- Most have a mixture of papillary and colloid-filled follicles (follicular predominance-some)
- If any papillary structure is present, the tumour will behave as a papillary carcinoma
- Histologically
 1. Papillary projections
 2. Characteristic pale, empty nuclei (Orphan Annie-eyed⁸ nuclei)
- Papillary carcinomas are very seldom encapsulated
- Multiple foci may occur
 1. Same lobe as the primary tumour
 2. Less commonly, in both lobes
- ✓ They may be due to
 - a) Lymphatic spread in the rich intra-thyroidal lymph plexus,
 - b) Multicentric growth
- Spread to the lymph nodes is common

- Blood-borne metastases are unusual unless the tumour is extra-thyroidal
- Extra-thyroidal -primary tumour has infiltrated through the capsule of the thyroid gland

Occult carcinoma

- Primary tumour may be no more than a few millimetres in size and is termed occult
- May present as LN in jugular chain with no palpable abnormality of the thyroid
- Term occult is now applied to all papillary carcinomas less than 1.5 cm in diameter.
- Excellent prognosis and are regarded as of little clinical significance.

Follicular carcinoma

- Macroscopically encapsulated
- Microscopically there is invasion of the capsule and of the vascular spaces in the capsular region
- Multiple foci are seldom seen
- Cannot identify as a follicular carcinoma by FNAC. Have to do lobectomy & histology to look for vascular & capsular invasion.
- Lymph node involvement is much less common than in papillary carcinoma
- Blood-borne metastases are almost twice as common and the eventual mortality rate is twice as high as papillary CA.

Hurthle cell tumours are a variant of follicular neoplasm in which oxyphil (Hurthle, Askanazy) cells predominate histologically. It is doubtful whether Hurthle cell neoplasms are ever benign and they may be associated with a poorer prognosis.

Prognosis in differentiated thyroid carcinoma

- Influenced by
 - a) Histological type
 - b) Age(much more dependant)
 - c) The presence of extra-thyroidal spread or major capsular transgression (in follicular CA)
 - d) Size of the tumour
- These allow separation of patients into
 1. Low-risk-25-year mortality rates of 2%
 2. High-risk -46%
- Much worse in males >40 years and in females >50 years
- Distant metastatic disease-adverse prognostic factor
- Lymph node metastases are not associated with worse prognosis
- Low-risk group account for 90% of cases of differentiated thyroid carcinoma

Surgical operations

Isthmusectomy

- In swellings confined to the thyroid isthmus, including small differentiated carcinomas
- Effective method of relieving tracheal obstruction and obtaining tissue for diagnosis in anaplastic carcinoma and lymphoma

Thyroid lobectomy

- Together with isthmusectomy is the appropriate operation for removal of a discrete thyroid swelling and for most patients with differentiated carcinoma

Near-total thyroidectomy.

- This consists of total thyroid lobectomy on the affected side and conservation of 1-2 g of thyroid tissue on the contralateral side
- Which preserves the blood supply to one or both parathyroids

Total thyroidectomy

The technique is essentially that of bilateral lobectomy and, if meticulous, the risk of complications is very low except for permanent hypoparathyroidism. The risk of hypoparathyroidism is variable but may be appreciable even in experienced hands

Additional measures

- Thyroxine
 - ✓ Suppress endogenous TSH production- basis that some tumours are TSH dependent
 - ✓ For all patients after operation for differentiated thyroid carcinoma
 - ✓ Not of value in follicular carcinoma
 - ✓ Unlikely to be of benefit in low-risk patients treated by lobectomy
 - ✓ Is obviously necessary after total thyroidectomy and in the majority of patients after near-total thyroidectomy

Patients with potential or actual distant metastases who may require repeated radioiodine administration for scanning and therapy should be given tri-iodothyronine (60—80 mg/day) because it is much shorter acting, and on stopping it, increased TSH secretion and thyroid avidity for iodine recover quickly so that radioiodine may be given after several days.

The patient is thereby spared weeks of developing thyroid insufficiency after stopping thyroxine before radioiodine may be given.

Radioiodine

- If metastases take up radioiodine they may be detected by scanning and may be treated with large doses of radioiodine
- For effective scanning
 - ✓ All thyroid tissue must have been ablated by either surgery or preliminary radioiodine
 - ✓ Patient must be hypothyroid to improve uptake
- Probably only indicated in patients with
 - ✓ Unresectable local recurrence or metastatic disease
 - ✓ High-risk patients
 - ✓ Those with a rising serum thyroglobulin level

In addition, if metastases take up radioiodine they are likely to be suppressed as effectively by treatment with thyroxine as by radioiodine. Cases in which suppression has failed and radioiodine has given permanent control appear to be uncommon.

- If metastases have been treated, the scan should be repeated at annual intervals and further therapeutic doses of radioiodine given as necessary.
- Solitary distant metastases may be treated by external radiotherapy

Thyroglobulin

- Value in the follow-up and in the detection of metastatic disease in patients who have undergone surgery for differentiated CA
- Thyroglobulin levels are, however, only an adjunct to careful clinical palpation of the neck
 - ✓ Local recurrence detectable clinically may be present with a low thyroglobulin.

Undifferentiated (anaplastic) carcinoma

- Mainly in elderly women
- Local infiltration is an early feature of these tumours with spread by lymphatics and by the bloodstream
- Extremely lethal tumours and survival is taken in months
- An attempt at curative resection
 - ✓ Only if there is no infiltration through the thyroid capsule and no evidence of metastases.
- Many of these aggressive lesions present in an advanced stage with tracheal obstruction and require urgent tracheal decompression.
- The trachea may be decompressed and tissue obtained for histology by isthmusectomy.
- Tracheostomy is best avoided.
- Radiotherapy should be given in all cases and may provide a worthwhile period of palliation as may combination chemotherapy [including doxorubicin (Adriamycin)].

Medullary carcinoma

- Tumours of the **parafollicular (C)**-cells derived from the neural crest
- Not unlike those of a carcinoid tumour and there is a characteristic **amyloid stroma**
- **Mainly arises from upper pole** as c-cells aggregate there
- High levels of serum **calcitonin** ($>0.08 \text{ ng/ml}$) are produced by many medullary tumours
- These levels fall after resection of a tumour and will rise again if the tumour recurs.
- This is a valuable tumour marker in the follow-up of patients with this disease
- Diarrhoea is a feature in 30% of cases and this may be due to 5-hydroxytryptamine or prostaglandins produced by the tumour cells.
- Some tumours are **familial** and may account for 10-20% of all cases
 - ✓ Frequently affects children and young adults
- **sporadic** cases occur at any age with no sex predominance(middle aged female- Dr.RF)
- Medullary carcinoma may occur in combination - syndrome **MEN IIa**
 - ✓ Adrenal phaeochromocytoma
 - ✓ Hyperparathyroidism (usually due to hyperplasia)
- When the familial form is associated with prominent mucosal neuromas involving the lips, tongue and inner aspect of the eyelids, with occasionally a Marfanoid habitus, the syndrome is referred to as **MEN type IIb**.
- **Family members of an index patient can be screened and pre-emptive surgery offered**
 - ✓ II B- has to be done by the age of 2 yrs
 - ✓ II A- by 5yrs
- LN spread occurs in 50-60% and blood-borne metastases are common
- Tumours are not hormone dependent and do not take up radioactive iodine
- Course of the tumour is unpredictable

Life expectancy is

1. **Excellent** if the tumour is confined to the thyroid gland,
 2. **Good** as long as metastases are confined to the cervical lymph nodes
 3. **Poor** once blood-borne metastases are present.
- Treated with total thyroidectomy and resection of involved lymph nodes with either a radical or modified radical neck dissection

Phaeochromocytoma must be **excluded** by measurement of urinary catecholamine levels **in all** cases before embarking upon thyroid surgery to avoid the potential hazards associated with this condition

Malignant lymphoma

- Uncommon tumour, most are B cell non Hodgkin lymphomas
- 70% are MALT(mucosa asso:) lymphomas
- Associated with hashimoto's
- Clinically may simulate anaplastic CA
- Common in female in their 70s and male under 40 also affected-causes compression
- True cut for diagnosis, staging must precede treatment
- Chemo with CHOP(cyclo, doxorubicin, vincristine & pred) & local irradiation are the main stay
- **Response to irradiation is good** and radical surgery is unnecessary once the diagnosis is established by biopsy.
- Although the diagnosis may be made or suspected on FNAC, sufficient material is seldom available for immune-cytochemical classification, and large-needle (Trucut) or open biopsy is usually necessary.
- In patients with tracheal compression, isthmusectomy is the most appropriate form of biopsy.
- **The prognosis is good** if there is **no** involvement of **cervical lymph nodes**.
- Rarely the tumour is part of widespread malignant lymphoma disease, and the prognosis in these cases is worse.