

For quick revision only

2012/2019

DEBOL

Bedside oriented surgery

COVID 19 (4th) Edition

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Preface

The book is made on common surgical cases & approaches by Ethiopian medical students to be used as a quick reference & guide. The aim of Debol is to enable & equip medical students with the basic & necessary surgical knowledge, skills & approaches in a very short period.

It was started in our clinical year attachment with the intension of collecting & comprising common surgical cases in one place for easy access by undergraduates with the ultimate goal of inspiring Ethiopian medical students to see beyond the wall.

The first edition was released during our C-I Surgery attachment (2008/2016). The 2nd & 3rd editions were released during 2009/2017 & 2010/2018 respectively & now this edition, COVID 19 or 4th edition, is special by being the first to be made after graduation. This edition is orchestrated in a briefly manner to hasten the maximum amount of knowledge to be acquired by undergraduates. Long cases beyond the sample histories; also contain 'raw materials' like risk factors, causes, clinical presentation, differential diagnosis, complications, investigations & principle of management. The short case parts mainly contain the indications, contraindications & complications of a procedure. Besides YouTube links are also provided for those who have access to the internet.

The last portion of this book presents basic surgical instruments to make undergraduate familiar to the operation theater & easily understand the language & skill of surgery.

Finally, I strictly advise you to use Debol as a quick recap & take off point for further reading from our standard text books of surgery.

For scholars of Ethiopia, the figure discrepancy we got from china about COVID19 during this pandemic is a lesson to get focused on building our own literatures since our standard texts books are also filled with such inapplicable informations.

Acknowledgment

For everything that had been and that ever will be, it is all by the will of God. So I thank God first & most.

I would like to express my deepest gratitude to Dr. Biruk Birhanu (Co-Author of Debol during our C-I attachment) & Dr. Dan Alemayehu (Manager of contributors' team during our C-I & C-II attachments) who have dedicated their valuable time in making this book come to reality. If it was not for you, I know this will never happen.

I would also like to thank participants from janhoy batch (names listed on the next page) for their adamantine dedication & contribution in the preparation of DEBOL BEDSIDED ORIENTED SURGERY. Besides for the many countless medical websites from which I took pictures & used their video link.

Last but not least, I would like to thank Dagmawi Mulugeta (Founder of Medicos Art Club, CII-JU), Dr. Eneyew Mebratu (Assistant professor of General surgery, UoG), Dr. Cheru Lilay (General surgery resident, UoG) & Rahel Nega (C-I, UoG) for giving me a constant support, collecting suggestions & opinions in the editing process of this book.

Knowing all of you is a benison.

Dr. Daniel Fentaneh Solomon (GP)

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Head injury sample history



[@talentofmedstu](https://t.me/talentofmedstu) (telegram)

In memory of those who lost their precious life in COVID19. May their soul rest in peace.

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This book is dedicated to Janhoy batch
GC of 2010/2018
Gondar



Comments on the previous editions of Debol

During our stay in medical school we had the chance to read several books authored in the west or east that detail experiences of other nations. We as a society had been prisoners to those literatures which do not consider our local context. Debol in its essence has opened a new chapter of surgical practice in Ethiopia, where we will break free from the trend of dependency and put forward our own standards of surgical care.

*Dr. Abraham Ariaya
General surgery resident, SPHMMC
Founder of Hakim page*

Debol, this book indeed helped numerous medical students to understand surgery better. As a new clinical trainee the science & skill can be truly overwhelming. Yet a book like Debol can make it a lot easier. Pointing out the most relevant & basic entities from the vast ocean of knowledge. It shows students the way of good history taking, physical examination & differential diagnosis with elaboration. Plus sample histories for common surgical cases. Improving the overall ability of the students in approaching a patient! The additional pros of the book is paying special attention to Ethiopia & considering the context of diseases in our country. So thank you Janhoys for this marvelous, exemplary job & I believe the new edition has a lot to offer.

*Dr.Elias Gebru Aimero
Psychiatry resident, JU
Author of “ኤቶፖላ” & “ፑቶረ”*

I believe it is a good reference book for undergraduate students especially for those who are new to the ward environment & clerking. I got the chance to read Debol on my NRMP exam, I hadn't known it before but it was a great help on that time...But details must be read from standard books till Debol replace Schwartz (which I believe in the near future).

*Dr.Mezgebu Alemneh
General surgery resident, UoG*

Debol is a simple & facile way to grasp the basics of surgery. It is baldly written. It has helped a lot of students in their clinical attachments & exam preparations.

*Dr.Yonas A.Tiruneh
General practitioner
Author of “ስለ ትናንሽ አለላዎች”*

I had my first run in with Debol when I was finishing up C-1, at which time I was fed up going through 3 to 4 surgical books just to get ready for my exams. But in Debol I found a contextualized guide that was easy to get around, it is a reading material based on the everyday of the ward. It was basically like borrowing a note from that really smart kid who takes a note during class & rounds. I truly believe Debol can be the footmark to have our own standards of care based on our context and not only in surgery aspect.

*Dr.Natanel Asres
MD, MPH candidate*

Ministry of health, Advisor to the state minister of Health

Debol is a precise but clear & understandable hand book. It is not only helpful for clinical year students but also for GPs to recap previous teachings in a short time. Honestly I am grateful for the authors & editors who spent their time & energy to bring forward such an amazing contribution. I am certain that the new edition will bring more to the table.

*Dr.Abigya Aschalew
General practitioner, Gondar university hospital*

My surgical qualification exam was like football field... I missed a lot of questions on my long exam but on the course of the exam penalty was founded & DEBOL made the score...

*Dr.Geleta Petros
Hawassa university, Intern*

Debol is a book that melted down the hellacious ocean of surgery in to a weeny pond so that every medical student can enjoy & we did!!! Many thanks to #Janhoy_batch and the special person Dr.Daniel Fentaneh.

*Dagmawi Mulugeta #DMF
Jimma university, C-II
Founder of MAC/Medicos Art Club*

I am very delighted to express my hearty gratefulness for the authors of the book for the incredible deed. Before writing my own, I tried to ask my friends for their comments on the book & almost all of them dared to say "it was our savior" It made our life easier. And I believe these are the most appropriate words to describe the book.

*Rediet Ararsa
Bahirdar university, C-II*

Debol helped us a great deal when we were attaching surgery. It is to the point and helps to guide what to look for when one wants detailed explanation. It is also written in Ethiopian context which makes it easily usable for Ethiopian students.

*Daniel Habtamu
Addis Ababa university/TASH C-I*

I failed C-I short exam. I have no clue about what to be asked and to answer. It was very challenging for me but after that, I repeat the whole year & read Debol. It is short, precise & time saving book...Thank you Dr.Daniel. Looking for the better edition!

*Natnael Seyoum
Wolkite university, C-I*

Surgery was my first attachment in C-I. As a first attachment and being new for the hospital environment, Debol has helped me much. I think the book gives direction for those who lost the track and minimizes the time loss in deciding what to study. But it should add more cases with a brief explanation including the management. I hope it won't be just a book but a great as bailey and Schwartz. Thanks for the help.

*Esuendale Anteneh
Wachamo university, C-I*

Debol is very helpful, it makes your way of learning smoother & be familiar for surgery. I am thankful for the person who has prepared it by scarifying his time.

*Bereket Alemayehu
Mekelle University, C-I*

Debol has immensely helped me throughout my surgical rotation both as a C-I & C-II student. It has tried to familiarize us with the approaches required for selected patient cases. I can say it has addressed the major points students have found challenging by presenting it in an easy way to understand. For these I would like to thank the contributors for their dedication to help medical students throughout the country. You are honestly an exemplary model to most of us. Thank you.

*Selamawit Tefera
Myungsung Medical College/korea hospital, C-II*

"If I had eight hours to chop down a tree, I'd spent six hours sharpening my axe." Abraham Linclon. That's what you did. You spent a lot to write such meticulously organized book & made our lives easier.

*"Nothing is particularly hard if you divided it into small jobs." Henry Ford
You divided surgical bulk into pieces that we can shoulder. You did great, but we think it's your beginning, make use of your gift to go further. We are short of words to express our gratitude.*

*"Online batch" students(C-I)
Gondar/GCMHS*

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CASE REPORT FORMAT

Short cases

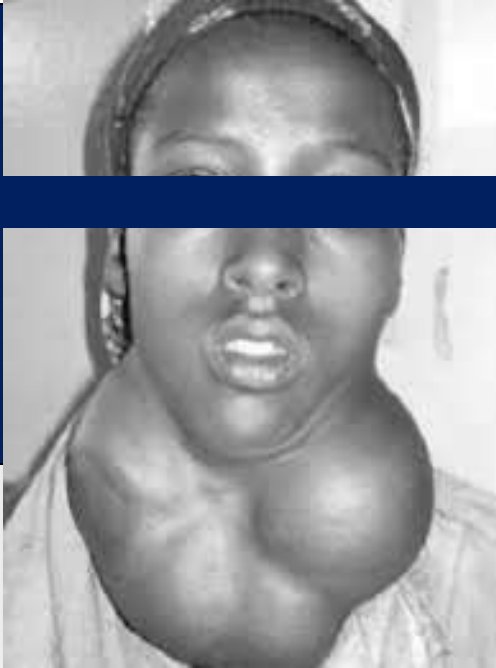
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Long cases

Chapter 1

Goiter



Brain storming

1. List down possible DDx for anterior neck swelling?
2. List down possible causes of goiter (enlarged thyroid gland)?
3. A 25years old female patient presented to your OPD with anterior neck swelling of 3years duration. How you approach the patient?

Approach to patients with anterior neck swelling

- A. Anticipate the possible causes(DDx) of anterior neck swelling
- B. Take history & do physical examination to narrow your differential diagnosis.
- C. Put your impression (Assessment) & proceed to investigate the patient. If your impression is Goiter, make it specific. Is it simple(non-toxic) or toxic? Is it multinodular, solitary nodule or diffuse? Inflammatory ,neoplastic...?
- D. Know the management principles

Possible DDx for anterior neck swelling

- Goiter
- Thyroglossal cyst
- Lipoma
- Lymphadenopathy
- Brachial cleft abnormality
- Cystic hygroma
- Subhyoid bursitis...



TMNG



Thyroglossal duct cyst



Lipoma

History taking

In order to write a good history on thyroid enlargement, follow the lists below

About the swelling;

Duration, how the patient noticed the swelling, initial site & progression...

Pressure symptoms;

Stridor, dyspnea, dysphagia...

Abnormal function manifestations;

Toxic/hyperthyroidism

Hot intolerance, irritability, emotional lability, palpitation, sleep disturbance, weight loss despite good appetite, diarrhea, tremor, menstrual abnormality (oligo/amenorrhea)...

Hypothyroidism

Cold intolerance, weight gain, fatigue, slow intellectual & motor activity, menorrhagia, constipation...

If toxic symptoms exist;

What is the timing between the swelling & thyrotoxic symptoms? This may give you clue in favor or against Graves' disease.

Ask similar illness in the vicinity

Favors endemic goiter

Living place/Highland area

Favors endemic goiter since highland water sources have low iodine due to erosion

Take medication history

Medications with goitrogen potential like Iodide, amiodarone, lithium...

Dietary history

Goitrogen intake like Cabbage, Cassava...

Such diets contain heavy metals which compete with iodine to be taken by thyroid tissue.

Ask family history of similar illness

Inborn errors of metabolism/dyshormogenesis

Fever, Pain in the neck

Favors inflammatory goiter

For thyroid cancer ask;

Constitutional symptoms of malignancy, family history of thyroid cancer, history of radiation therapy to the head & neck

For suspected metastatic thyroid cancer, be familiar with the commonest sites of metastasis then dig out the symptoms for each site.

History of hoarseness of voice in case of recurrent laryngeal nerve involvement, swellings in the neck for lymphnode metastasis, History of hemoptysis for lung & bone pain for skeletal metastasis...

Physical examination

General Appearance

Watch carefully your patient's dressing style due to the hot intolerance or cold intolerance.

Vital Signs**Blood Pressure**

Thyrotoxic patients may have wide pulse pressure due to systolic hypertension

Pulse rate

Tachycardia in thyrotoxic patients(>85bpm)

Bradycardia in hypothyroid patients

Temperature

In cases of inflammatory goiter fever may be present.

Lympho-glandular examination**Thyroid examination**

- A. Inspection
- B. Palpation
- C. Percussion
- D. Auscultation

Inspection

Size (estimate)

Shape

Site

Overlying skin color change

Visible Pulsation

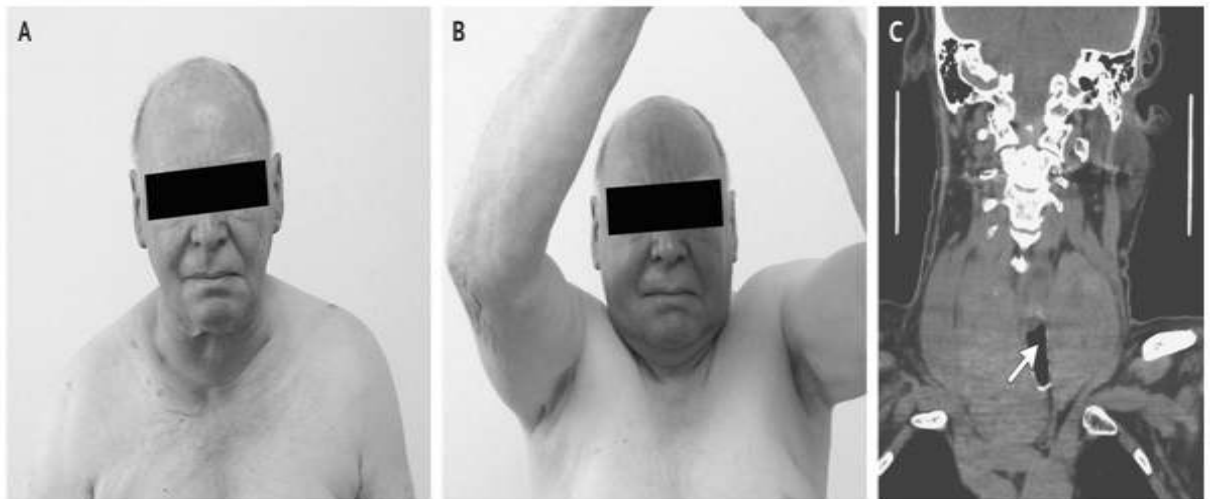
Movement with deglutition & protrusion of tongue

Because thyroid gland is enclosed by pretracheal fascia, it moves with swallowing.

In solitary thyroid swelling, look for upward movement of the swelling on protrusion of the tongue. This will enable you to differentiate a thyroid nodule from thyroglossal duct cyst.

Pemberton's sign

Ask the patient to raise both upper limbs above the head and keep it for at least for 1 minute. If there is retrosternal extension, the patient will have congestion and puffiness in the face with respiratory distress.



Retrosternal extension

Pemberton's sign is done in patients with compliant of aero-digestive tract obstruction & on those the lower border of thyroid gland is not visible during deglutition.

Palpation

- Hotness
- Tenderness
- Size (Measure)
- Surface (Smooth Vs Nodular)
- Border (Regular Vs Irregular)
- Consistency (Soft Vs Firm Vs Hard)
- Retrosternal extension (Try to palpate the lowest tracheal ring above the sternal notch)
- Fixity to overlying structure
- Thrill (Check on upper pole)
- Position of trachea (Central Vs Deviated)

- Kocher's test

Kocher's test is a test for presence of tracheal compression. The gland is pressed slightly on either side of trachea. If the trachea is already compressed, the patient will have stridor.



Kocher's test

- Berry's sign

It is an examination for carotid pulse. Berry's sign is positive, means carotid pulse is not palpable on that side.

Methods of palpation

1. Standing in front of the patient
2. Standing behind the patient

Percussion

If you suspect retrosternal extension, percuss over the manubrium & appreciate resonant or dull note.

Auscultation

Auscultate over the upper poles of the swelling for bruit.

Reporting format

Inspection

There is about 8X6cm butterfly shaped anterior neck swelling which moves with deglutition. The lower border is visible on swallowing. It is slightly deviated to the right side. There is no visible pulsation or overlying skin color change. Pemberton's sign is negative.

Palpation

There is 9X7cm non-tender, nodular, firm anterior neck mass with regular border. There is no fixity to the overlying skin. Its temperature is comparable to other parts of the body. The lower tracheal ring is palpable above sternal notch. There is no thrill. Kocher's test is negative. Carotid arteries are palpable bilaterally (Berry sign is negative). The trachea is central.

Percussion

Auscultation

No bruit over the swelling

Questions

1. What you want to look after taking vital signs & doing thyroid gland examination?
2. What are the systemic signs of thyrotoxicosis, hypothyroidism, retrosternal extension & metastasis?

Systemic signs of thyrotoxicosis

HEENT = Eye signs

Exophthalmos

Exophthalmos is an abnormal protrusion of the eye ball. It is said to be present when the eyeball is seen beyond the superior orbital margin during top view or when both the upper & lower sclera are visible when looking forward.



Exophthalmos

Lid lag

Steady the patient's head with one hand & ask the patient to look at your finger. Ask him/her to look up and down following your finger.

In case of thyrotoxicosis, the lid may lag while the eyeballs move downward. This will make the upper sclera to become visible.



Lid lag

Lid retraction

Visibility of upper sclera due to spasm of upper eyelid.

Absence of wrinkling

Steady the patient's head with one hand. Ask the patient to look up at the ceiling. There may be absence in wrinkling of forehead in cases of thyrotoxic patients.

Failure of convergence

Ask the patient to look at your finger from a distance. Then bring it suddenly in front of the patient's eye.

Failure of convergence may be present in thyrotoxic patients.

Integumentary system

Warm moist skin

Musculo-skeletal system

Pretibial myxedema

CNS

Tremor

A. Finger

Ask the patient to stretch out both the upper limbs and spread out the fingers.



Finger tremor

B. Tongue

Ask the patient to protrude the tongue resting on the lower lip.

Signs of hypothyroidism

- Edema of face & legs (HEENT & MSS)
- Delayed relaxation of deep reflexes (CNS)
- Pendred's sign (on CNS): Goiter with severe sensory neural hearing impairment.

Signs of retrosternal extension

- Increased JVP (CVS)
- Horner syndrome--Caused by lesion along the sympathetic pathway that supplies the head, eye, and neck.
 1. ptosis
 2. Anhidrosis
 3. Miosis...

Signs of metastasis

- Hard cervical lymphnodes (LGS)
- Nodules on skull (HEENT)--Rapidly growing, pulsatile & warm swelling. Erosion of the skull may be present.
- Long bone metastasis (MSS)
- Chest effusion & consolidation (RS)
- Nodular liver & ascites (Abdominal Examination)

<https://www.youtube.com/watch?v=ta-s-ZWRk6g>

Click &
watch!!!
Make your life
easy!

Sample History

Chief complaint

Anterior neck swelling of 2 years duration

HPI

This patient was last relatively healthy 2 years back at which time she noticed a swelling on her left lower neck. The swelling was initially pea sized but it later progressed to grow upward & medially to attain its current size, shape & location.

06 months prior to her admission, she started to experience harsh noise when breathing which get worsens during supine position. But no difficulty of swallowing. Associated with this she started to experience palpitation, heat intolerance, profuse sweating & unquantified weight loss to the extent her skirts become loose. She had also irregular menstrual cycle for the past 07months which come every 2 to 3months.

03 months prior to admission she visited our hospital where blood sample & sample from the swelling was taken. Then she was given whitish scored oval tablet to be taken three times daily & reddish scored circular tablet to be taken twice daily. She was taking her medications adherently. Currently she has no palpitation or heat intolerance.

- *Many peoples in her vicinity have similar illness*
- *Her regular dietary habit is 'injera' made of 'teff' & 'shirowot' made of 'atter.' She occasionally eats cabbage.*
- *No history of drug intake except the medication explained above.*
- *No history of fever, chills or neck pain*
- *No family history of similar illness*
- *No history of head & neck radiation therapy*
- *No history of swelling in the neck or axilla)*
- *No history of bone pain, hemoptysis or yellowish discoloration of the eyes*
- *No history of dyspnea, orthopnea, PND or lower leg swelling*
- *No history of chronic cough, contact with a chronic cougher or previous TB treatment*
- *No self/family history of DM, HTN or asthma*
- *She was screened for RVI 7months back & found to be non-reactive*

Finally she was admitted to our hospital.....

Investigation

Laboratory

- TFT /TSH, T3, T4/
- Anti-thyroid antibody assessment

Pathology

FNAC

- Reliable in Papillary, medullary, anaplastic thyroid cancer diagnosis.
- Not reliable in differentiating...
 1. Follicular adenoma from follicular carcinoma
 2. hurthle cell cancer
 3. hashimoto's thyroiditis from thyroid lymphoma

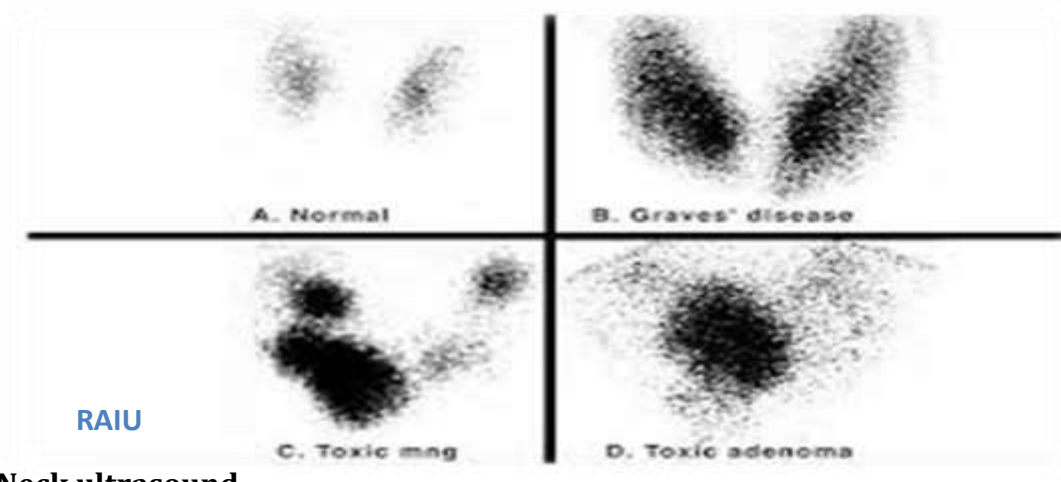


Ultrasound guided FNAC

Imaging

Thyroid RAIU (radio-active iodine uptake) scanning

- Activity of the gland--Hot, Warm or Cold



Neck ultrasound

- Solid Vs cystic
- Risk of malignancy--By looking at its echo texture, shape, border, calcifications, vascularity ...
- Targeted aspiration (USG-FNAC)

Chest & Thoracic inlet x-ray

- Retrosternal goiter
- Tracheal deviation & compression
- Pulmonary metastasis



CT, MRI & PET

Goiter

Any enlargement of the thyroid gland is referred to as a goiter

It could be toxic or non-toxic. Uninodular, multinodular or diffuse ...

Causes

- i. Simple goiter
- ii. TMNG
- iii. Graves's disease
- iv. Toxic adenoma
- v. Inflammatory goiter
- vi. Neoplastic goiter



Simple goiter/non-toxic

Common in females due to the estrogen receptors in the thyroid tissue. Endemic goiter (iodine deficiency), goitrogen intake (dietary, drugs), thyroiditis (sub-acute & chronic), familial goiter & neoplastic goiter are among the causes of simple goiter. Endemic goiter is the commonest cause of simple goiter.

Clinical presentation

- Most asymptomatic
- If symptoms occur, patients often complain of;
 - ✓ Pressure sensation in the neck
 - ✓ Compressive symptoms; Dyspnea, Dysphagia
 - ✓ Acute pain may occur in case of hemorrhage into the gland.
 - ✓ Substernal goiters--Positive Pemberton's sign
- Physical examination of multinodular non-toxic goiter may reveal nodules of various size & consistency

TFT--Normal/euthyroid

Complications

- Tracheal obstruction
- In 30% of patients secondary thyrotoxicosis may occur
- Calcification of the gland
- May have premalignancy potential. Follicular thyroid cancer is the commonest type of cancer which can arise from simple goiter.

Management

- Large goiters
Give exogenous thyroid hormone to reduce TSH stimulation & gland growth (due to negative feedback mechanism).
- Endemic goiters
Supplement with iodine.
- Surgical resection
Indications
 - Toxic features
 - Goiters causing obstructive symptoms
 - Goiters suspected for being malignant or proven by FNA
 - Goiters cosmetically unacceptablePreferred methods of resection
 - Near-total or
 - Total thyroidectomy with lifelong T4 therapy

TMNG/ Toxic multinodular goiter

Clinical Presentation

- Cardiovascular symptoms are common
- Eye signs are infrequent unlike graves' disease
- Occurrence of neck swelling & thyrotoxic symptoms aren't simultaneous

Investigation

TSH level -- Suppressed

Free T3 & T4 -- both elevated

RAI scan -- shows multiple nodules with increased uptake

Management

Control hyperthyroidism

- Anti-thyroid drugs

The aim is to render the patient euthyroid. Usually administered in preparation for thyroidectomy or ablation with radioactive iodine. If the patient is not rendered euthyroid before the procedures, thyroid storm will happen.

The medications dose should be titrated depending on TSH & T4 level on follow-ups. It should continue until the patient is euthyroid (using clinical & laboratory evidences)

PTU (propylthiouracil) is the drug of choice (100-300 mg PO TID).

Alternative → Methimazole . Mechanism of action;

- ✓ Both reduce thyroid hormone production by inhibiting the organic binding of iodine & the coupling of iodotyrosines .
- ✓ In addition PTU inhibits peripheral conversion of T4 to T3. Making it drug of choice for treatment of thyroid storm.
- ✓ PTU also has less risk of transplacental transfer compared to methimazole. Preferred in pregnant & breast feeding women.

- B-blockers

B-blockers are useful to alleviate catecholamine response of thyrotoxicosis. It should be considered in all symptomatic (thyrotoxic) patients & elderly patients with cardiac disease.

Treatment should continue 1 week after surgery. Because the half life of T4 reaches up to 7 days.

Propranolol & Atenolol (Long acting) are drugs among this group.

Radioactive Iodine (RAI) thyroid ablation

Indications

- ✓ Elderly patients with small or moderate sized goiters
- ✓ Those who relapsed after medical or surgical therapy
- ✓ When anti-thyroid drugs or surgery is contraindicated

Absolute contraindications

- ✓ Pregnant or planning to conceive soon (<6 months) after the treatment
- ✓ Breast feeding mother

Relative contraindications

- ✓ Young patients with thyroid nodules
- ✓ Young patients with ophthalmopathy

Surgical treatment

- ✓ Pre-op preparation with anti-thyroid drugs
- ✓ 7-10 days prior to surgery administer Lugol's iodine solution or Saturated potassium iodide. They reduce vascularity of the gland & decrease the risk of precipitating thyroid storm.
- ✓ Near total or total thyroidectomy is recommended to avoid recurrence & risk of repeating the surgery

Graves' disease



- An autoimmune disease with a strong familial predisposition
- Common in females (5:1)

Clinical presentation

Thyroidal manifestations

- Thyrotoxicosis
- Soft & diffuse goiter

Extra-thyroidal manifestations

- Eye signs and CNS symptoms are common

Specific to graves' disease

- Ophthalmopathy
 - ✓ Lid lag (von graefe's sign)
 - ✓ Spasm of upper eye lid (Dalrymple's sign)
 - ✓ Prominent staring
 - ✓ Exophthalmos, conjunctival swelling & congestion
- Dermopathy
 - ✓ Pretibial myxedema--Due to deposition of glycosaminoglycans

Physical examination findings in thyroid gland of graves' patient

- The thyroid gland is usually soft, diffusely & symmetrically enlarged
- There may be overlying bruit & or thrill
- There may be loud venous hum in supraclavicular space

Investigations

TSH level -- Suppressed

Free T3 or T4 level--May or may not be elevated

RAIU scan--increased uptake & diffusely enlarged gland

Management

- Medical/anti-thyroid drugs
- Thyroid ablation
- Surgical

Toxic Adenoma

Autonomous, solitary overactive nodule with inactive surrounding tissue. Typically occur in young patients with recent growth of long standing nodule along with symptoms of hyperthyroidism.

Thyroid gland on physical examination usually reveals a solitary nodule without palpable thyroid tissue on the contralateral side.

RAI scan shows hot nodule with suppression of the rest of the gland.

Antithyroid drugs, thyroid ablation & surgery (lobectomy/isthmusectomy) are the principles in toxic adenoma management.

Inflammatory Goiter

Thyroid gland is inherently resistant to infection due to

- Its extensive blood & lymphatic supply, high iodide content & fibrous capsule

Inflammatory goiter may be toxic or non-toxic

A) Acute (suppurative) thyroiditis

More common in children. Often preceded by upper respiratory tract infection or otitis media.

Clinical presentation;

- Severe neck pain radiating to the jaws/ear
- Fever, chills
- Odynophagia
- Dysphonia

Investigations

- CBC may reveal leukocytosis
- FNA for gram stain, culture & cytology

Complications

- Systemic sepsis
- Tracheal /esophageal rupture
- Jugular vein thrombosis
- Laryngeal chondritis & perichondritis
- Sympathetic trunk paralysis

Management

- Parenteral antibiotics
- Drainage of abscess
- Thyroidectomy for persistent abscess & failure of open drainage

B) Sub acute thyroiditis (granulomatous thyroiditis)

It may be painful or painless.

Painful

It is thought to be viral in origin or post-viral inflammatory response. It has four stages (Hyperthyroidism→euthyroid→hypothyroidism→ resolution & return to euthyroid state)

The patient may present with sudden or gradual onset neck pain which may radiate to the mandible or ear. History of preceding upper respiratory tract infection often presents. Physical examination may reveal enlarged, tender & firm gland.

Investigations

Early stage

- TSH decreased, T4 & T3 elevated
- ESR >100mm/h
- RAI uptake=decreased

Management

Since it is self limiting disease, the treatment is primarily symptomatic relief.

- Pain relief
 - NSAIDS
 - Steroids→in severe cases
- Short term thyroid replacement may be necessary to shorten duration of symptoms

Painless

It is considered to be an autoimmune disease. The physical examination result may be normal sized or minimally enlarged, firm, non-tender gland.

Investigation results are similar to the painful one except normal ESR.

Patients with symptoms may need B-blockers & thyroid hormone replacement.

C) Chronic lymphocytic thyroiditis (Hashimoto's)

It is a transformation of thyroid tissue to lymphoid tissue. Leading cause of hypothyroidism & Common in females (10-20:1)

Clinical presentation

- ✓ Minimally or moderately enlarged firm & nodular gland
- ✓ 20% present with hypothyroidism while 5% present experience hyperthyroidism

Investigation

- ✓ Elevated TSH
- ✓ Thyroid auto-antibodies present

Management

- ✓ Overtly hypothyroid patients need thyroid hormone replacement therapy. (Levothyroxine)

D) Reide's thyroiditis

It is the replacement of all or part of the thyroid parenchyma by fibrous tissue.

Clinical presentation

- ✓ The pt may present with symptoms of hypothyroidism & hypoparathyroidism → since the gland is replaced by fibrous tissue
- ✓ Typically presents as painless, hard ("woody") anterior neck mass, with fixation to the surrounding tissue

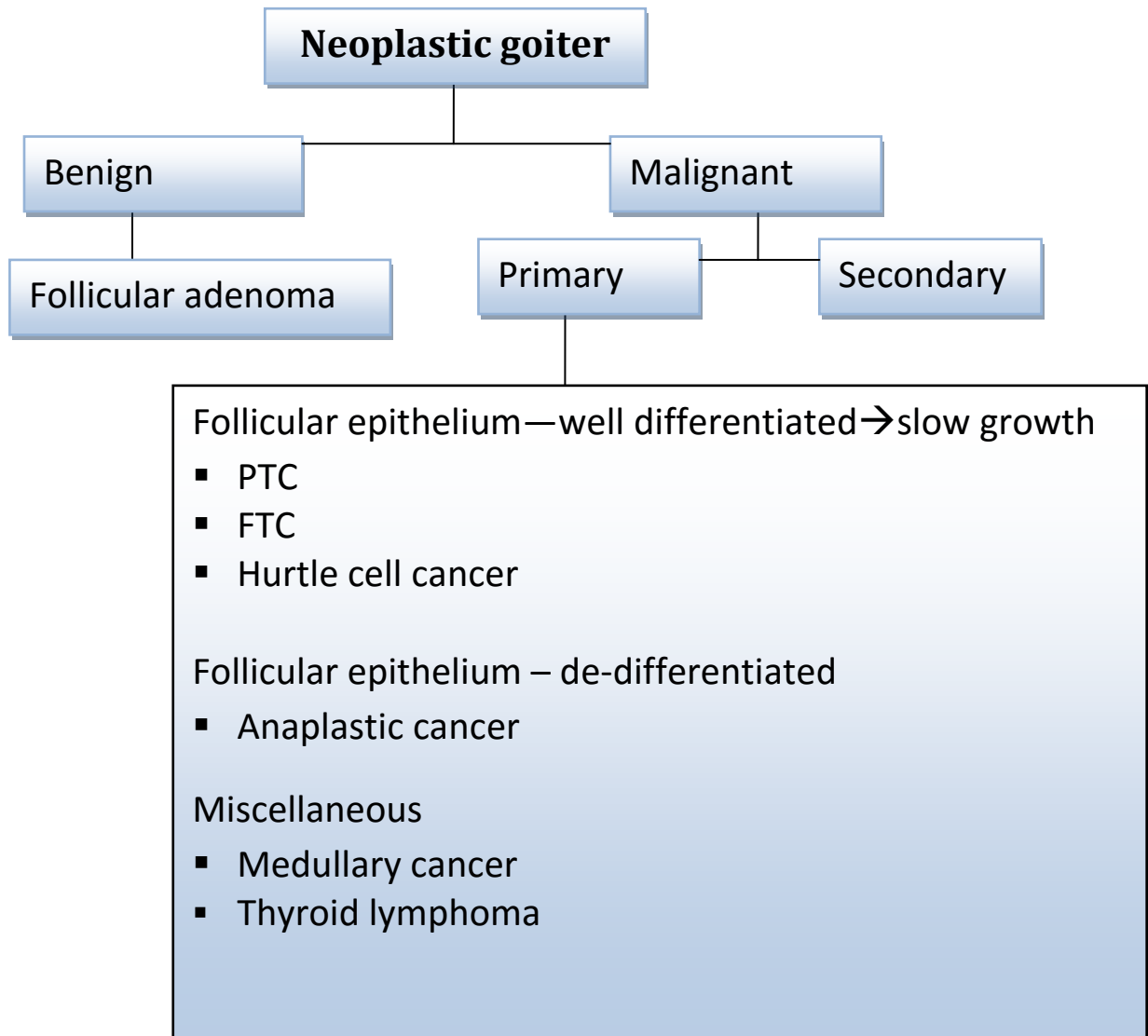
Diagnosis

- ✓ Open thyroid biopsy

Management

- ✓ Surgery
- ✓ Hypothyroid patients need thyroid hormone replacement therapy

Neoplastic Goiter



Primary

A. Papillary thyroid cancer (PTC)

- Cover 80% of all thyroid malignancies
- Predominant in children & individuals exposed to radiation
- Lymphoid metastasis is the commonest route
- Distant metastasis to→Lungs, bone, liver & brain
- Diagnosis is made by FNAC
- Management
 - Surgery--Total /near total thyroidectomy
 - Post-op
 - Radioiodine therapy
 - Radiotherapy & chemotherapy
 - Thyroid hormone

B. Follicular thyroid cancer (FTC)

- Covers 10% of thyroid cancer
- Occur more commonly in iodine deficient areas
- Often present as solitary thyroid nodule
- Hematogenous metastasis is the commonest route
- Diagnosis
 - FNAC is unable to distinguish benign from malignant disease (follicular adenoma from follicular thyroid cancer)
 - Difficult to diagnosis in pre-op patients unless there is distal metastasis
- Management
 - Surgery—Lobectomy/ Total thyroidectomy
 - Post-op
 - Radioiodine therapy
 - Radiotherapy & chemotherapy
 - Thyroid hormone

C. Hurtle cell cancer (subtype of follicular cancer)

- Covers 3% of thyroid cancer
 - It can't be diagnosed with FNA Since it's characterized by vascular & capsular invasion

D. Anaplastic cancer

- Early local infiltration→aggressive
- Typically patients present with long standing neck mass, which rapidly enlarged & become painful with associated dysphagia, dyspnea, dysphonia. The patient may also complain of bone pain, weakness, cough...
- Poor prognosis

E. Medullary thyroid cancer

Arise from Para-follicular/C cells & may occur in combination with adrenal pheochromocytoma and hyperparathyroidism.

The lump usually is found at supero-lateral neck.

Management

1. Total thyroidectomy
2. External beam radiation

F. Thyroid Lymphoma

- Non Hodgkin's B-cell type

Secondary (Metastasis to thyroid gland)

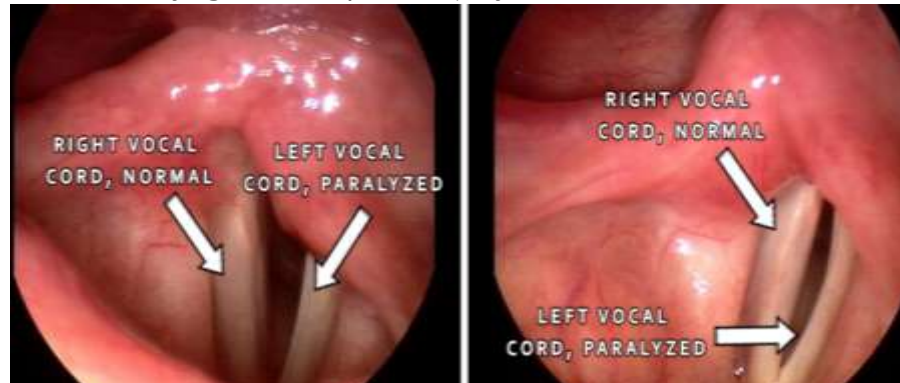
- Thyroid is rare site of metastasis
- Sites→kidney, breast, lung, melanoma...

Complications of thyroid surgery (specific to thyroid surgery)

- Hemorrhage
- Airway obstruction

Causes of airway obstruction in thyroid surgery;

- Laryngeal edema
Management -- Intubate then give Steroids to reduce the edema
- Recurrent laryngeal nerve/RLN injury



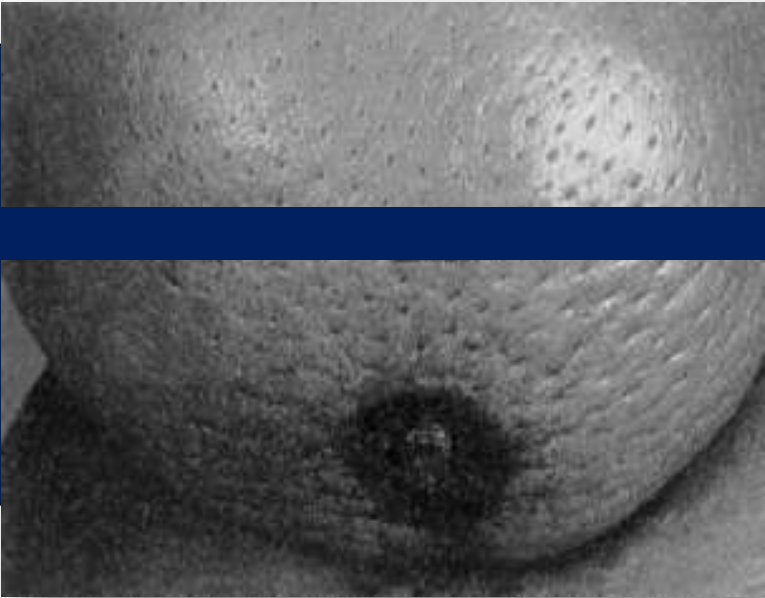
- ✓ Bilateral RLN injury--Patients present with respiratory distress
Management
 - Tracheostomy/Chordotomy
- ✓ Unilateral RLN injury-- patients manifest with hoarseness of voice
Management
 - Re-innervation
 - Medialization→injection therapy by forming edema
- Haematoma



- ✓ Patients experience respiratory symptoms due to the compression effect of the hematoma collection.
Management
 - Remove all the stitches & decompress it urgently(release the blood collection)

- ✓ Hypocalcemic tetany due to involvement in parathyroid gland may result in laryngeal spasm which can lead to airway obstruction
Management
 - Intubate the patient then supplement with calcium
- **Tracheomalacia**
- **Wound infection**
- **Thyroid storm**
 - ✓ It is a condition of hyperthyroidism accompanied by fever, CNS agitation or depression, cardio-vascular & GI dysfunction including hepatic failure
 - ✓ Due to Poor pre-op preparation
 - ✓ Management of thyroid storm
 - B-blockers
 - Oxygen supplementation, Hemodynamic support
 - Pyrexia → give non-aspirin compounds
 - Lugol's iodine or sodium ipodate (IV route)
 - PTU
 - Corticosteroids -- To prevent adrenal exhaustion & block hepatic thyroid hormone conversion
- **Hypothyroidism**
 - ✓ Management -- levothyroxine
- **Hypoparathyroidism**
 - ✓ May be transient due to ischemia/manipulation or permanent incase of removal of the gland.
 - ✓ May be subclinical/asymptomatic or symptomatic
 - ✓ May be symptomatic
 - Perioral parasthesia, carpopedal spasm, laryngospasm, seizure, tetany...
 - Elicit signs of hypocalcemia
 - ✓ Chovestik sign
 - ✓ Trousseau sign
 - Management -- In symptomatic patients supplement with calcium
- **Superior laryngeal nerve injury**

They can't produce high pitch sound & the management is speech therapy.
- Other complications
 - ✓ Keloids, Stitch granuloma...



Chapter 2

Breast

Brain storming

1. A 36 years old nulligravida lady presented to your OPD with a complaint of left breast swelling of 1 year duration. How to approach this patient?
2. List the management principles for breast cancer?

How to approach patients with breast disorders

Common complaints

- Breast pain
- Breast lump
- Nipple discharge
- Nipple retraction
- Surface appearance change

DDx-1: Breast pain

- Cyclical breast pain
- Area of fibroadenosis
- Mastitis
- Breast abscess
- Inflammatory breast cancer....



Mastitis

DDx-2: Breast lump

- Breast cyst
- Fibroadenoma
- Breast abscess
- Fat necrosis
- Hematoma
- Breast ca....

DDx-3: Nipple discharge/pathologic

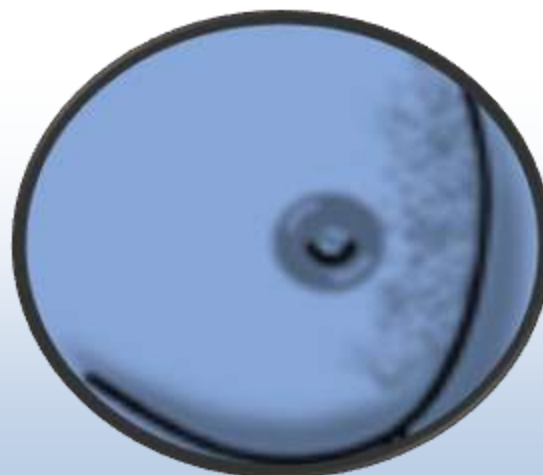
- Duct ectasia
- Intraductal papilloma
- Ductal carcinoma insitu...

DDX-4: Nipple retraction/Recent

- Slit like nipple retraction
 - Duct ectasia
 - Chronic periductal mastitis
- Circumferential nipple retraction
 - Carcinoma

Ddx-5: Surface appearance change

- Paget's disease
- Eczema
- Breast cancer



Breast Cancer

Risk factors

Hormonal

Increased (unopposed) estrogen exposure due to

- Early menarche (age <12)
- Late menopause (age >55)
- Nulliparity
- Never breastfed (Breast feeding is thought to be protective from breast cancer)
- First full term pregnancy >30years of age (First child at early age is also protective)
- Exogenous hormones
Did your patient have Hormone Replacement Therapy (HRT) in the past 5 years?
- Obesity
In postmenopausal women the adipose tissue acts as major source of estrogen.

Non hormonal

- History of high dose radiation therapy. E.g., mantle radiotherapy for Hodgkin's lymphoma
- Alcohol abuse--known to increase serum level of estradiol

Genetics

- Familial breast cancer. Is there a first degree relative with breast cancer?
- History of endometrial, ovarian or colonic cancer

Miscellaneous

- Female sex
- Increasing age (65^{plus})

Clinical presentation

History

Breast lump is the commonest presenting symptom in breast cancer patients.

Describe in your HPI;

- When & how the patient noticed the swelling?
- Site & progression?
- Is there associated nipple discharge? If yes, what is the color of discharge? Is the discharge unilateral or bilateral?
- Is there any nipple retraction. If yes, is it recent retraction or not?
- Is there associated ulceration or erythema of overlying skin?
- Is there associated axillary mass?

- If you suspect metastatic spread in advanced cases, ask;
 - Bone pain, fracture history (pathological bone fracture)--Bone metastasis
 - Breathing difficulties -- Malignant pleural effusion
 - Yellowish discoloration of eyes & skin--Liver metastasis
 - Look for symptoms of raised ICP in case of Cerebral metastasis



Breast cancer

NB

Common sites of breast cancer metastasis

1. Local spread

Skin, muscles, chest wall...

2. Lymphatic metastasis

Axillary, internal mammary, supraclavicular lymphnodes ...

3. Hematogenous metastasis

- Skeletal metastasis (lumbar vertebrae, femur, thoracic vertebrae, rib & skull...)
- Liver, lungs & brain

Physical examination

Lymphoglandular Examination

Breast examination

Inspection

First: Position the patient

- Arms by side
- Arms straight up in the air
- Hands on hips
- Bending forward



Then comment on;

- Symmetry of breasts
Use nipple line for comparison
- Breast size & shape
Compare both sides
- Look for peau d'orange appearance



Peau d'orange appearance

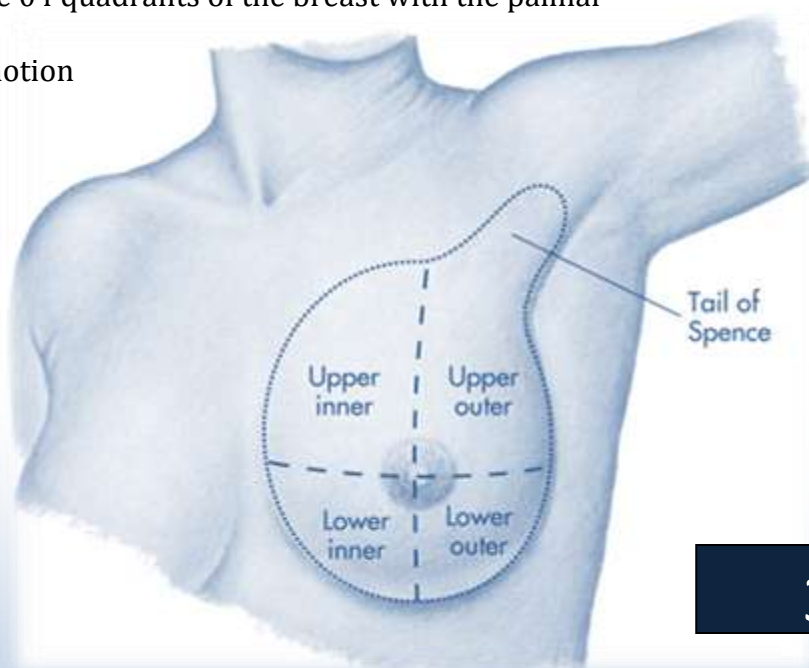
This appearance is due to cutaneous lymphatic obstruction & edema. It is more prominent on elevation of the hands.

- Inspect for skin or nipple retraction. Skin retraction is accentuated by extending patient's arms forward while sitting down & leaning forward.
- Inspect for nipple discharge. You can also elicit the discharge (if any) by squeezing the nipple.
- Look for ulceration & characterize it

Palpation

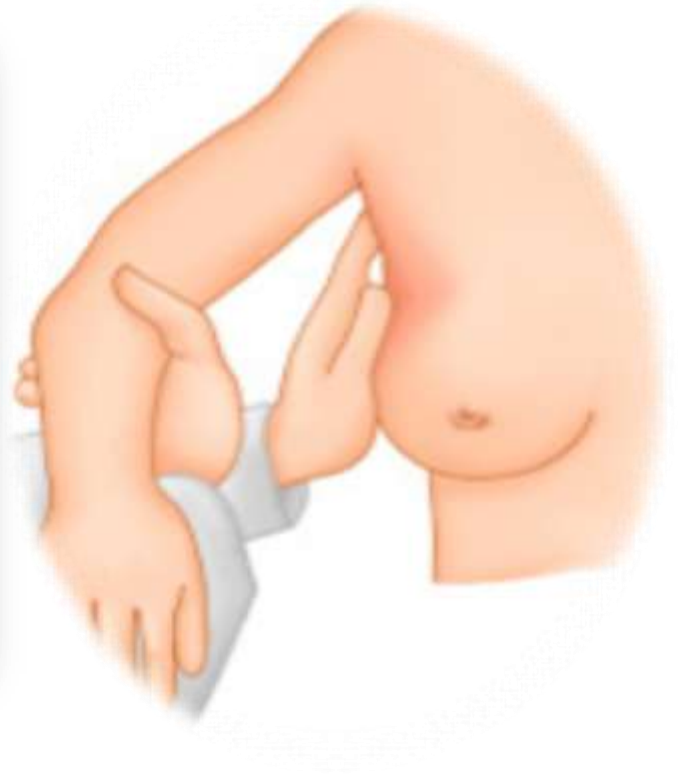
Technique

- Supine position, examine all the 04 quadrants of the breast with the palmar aspect of your fingers
- Avoid a grasping or pinching motion

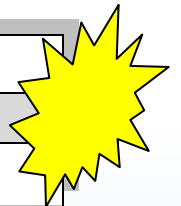


Appreciate & report your findings;

- Site of the swelling
Upper outer quadrant is the commonest site for breast cancer swelling
- Consistency of the swelling
Hard in breast cancer
- Border of the swelling
Irregular in breast cancer
- Surface of the swelling
Nodular in breast cancer
- Fixation
May be fixed to overlying or underlying structures
- Report if any tenderness exists
- Never forget to look for Axillary and supraclavicular lymphadenopathy!
- Also do upper extremity neurologic (motor & sensory) examination in case infiltration of brachial plexus occurred.



Watch video @ [https://www.youtube.com/watch?v= p8PobUp2Yo](https://www.youtube.com/watch?v=p8PobUp2Yo)



Sample history

Chief complaint

Breast swelling of 6 months duration

HPI

This is a 38 years old nulligravida lady who was last relatively healthy 6 months back at which time she noticed small swelling on her left breast while she was taking shower. Initially the swelling was pea sized but later it progressed to attain its current size & shape. 01 month prior to admission she started to experience bright red bleeding from her left nipple but no history of breast pain. Associated with this she noticed change in nipple position & orange peel like skin appearance change over her the left breast.

- *Her menarche was at the age of 12. It was regular, comes every 28 days, stays for 3-4 days, moderate in amount & associated with mild abdominal discomfort*
- *The patient doesn't notice any cyclical changes of the swelling with her menses.*
- *No family history of similar illnesses.*
- *No history of HRT or OCP use.*
- *No history of radiation therapy.*
- *No history of chronic alcohol consumption.*
- *No history of breast trauma*
- *No history of breast or abdominal surgery.*
- *No history of swelling in the neck or axilla.*
- *No history of bone pain, breathing difficulty or yellowish discoloration of the eyes.*
- *No history of cough, contact with chronic cougher or previous TB treatment.*
- *No self or family history of DM, HTN or asthma*
- *She has been screened for RVI 01 month back & found to be Non-reactive.*

Finally she was admitted to our hospital walking by herself.

Investigation

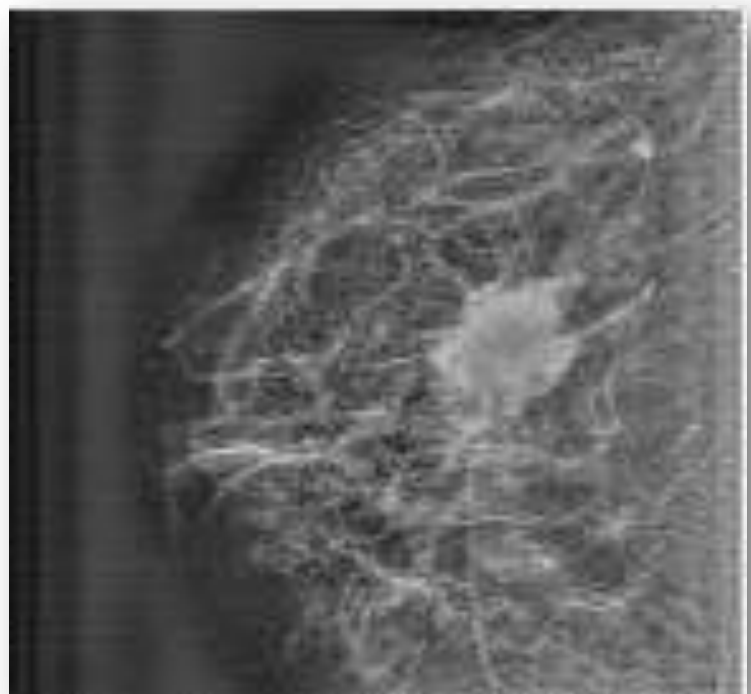
1. Imaging studies

Mammography

Imaging of breasts either in medio-lateral or cranio-caudal view by a selenium coated x-ray plate which will come in direct contact with the breast.

Sensitivity of this investigation will increase with age as the breast becomes less dense.

- What to look for breast cancer in mammography?
 - A solid mass with or without stellate features
 - Asymmetric thickening of breast tissue
 - Clustered micro-calcifications

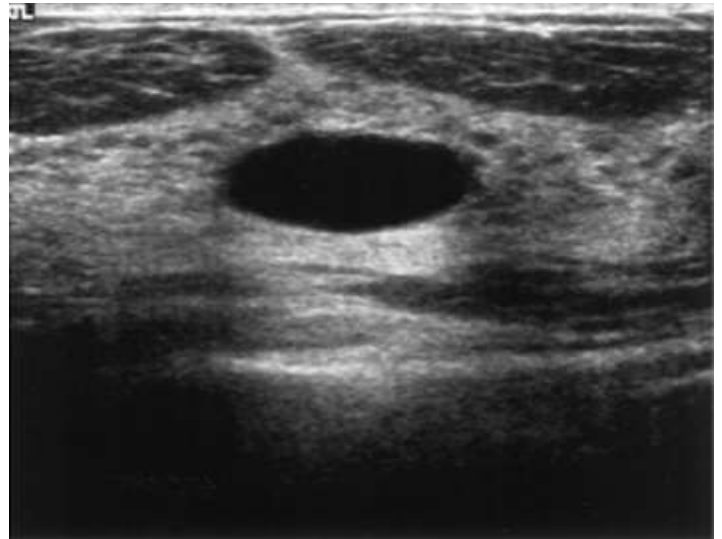


Breast Ultrasound

- Breast ultrasound can be used in young women with dense breasts in whom mammograms are difficult to interpret.
- It can distinguish cysts from solid lesions
- It can localize impalpable areas of breast pathology
- It can guide FNAC, core biopsy...

Drawback/ breast ultrasound

- It is not ideal for lesions $\leq 1\text{cm}$ in diameter



Breast Cyst

What to look for a cyst on ultrasound?

- Well circumscribed wall
- Smooth margins
- Echo-free center

What to look for breast cancer on ultrasound?

- Irregular walls
- Acoustic enhancement

What to look on ultrasound for benign breast mass generally?

- Well defined margins
- Round or oval shape
- Smooth contour
- Weak internal echoes

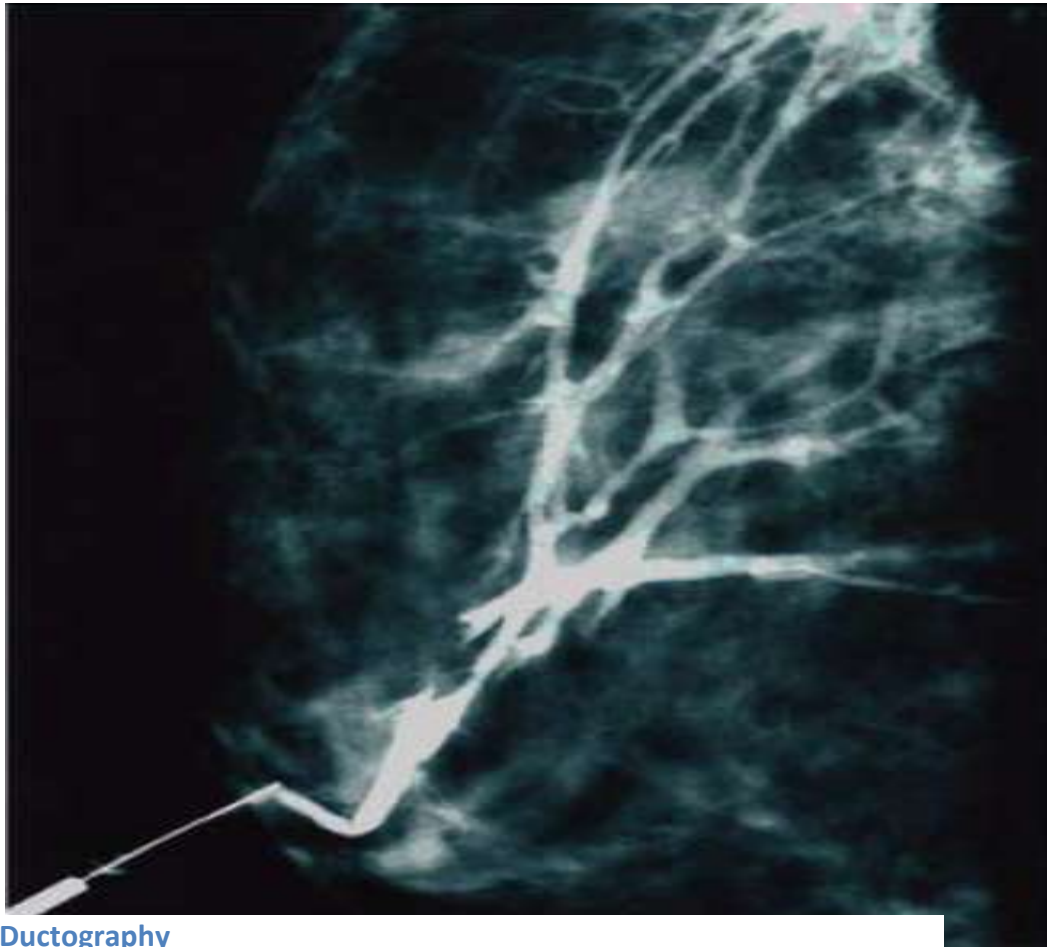
Ductography

In ductography radio-opaque contrast media will be injected via the nipple. Then mammography will be done.

This work-up is primarily indicated for blood stained nipple discharge.

What to look on ductography?

- Intraductal papilloma present as a small filling defect surrounded by the contrast media
- Cancerous breast lesions may appear as irregular masses or as multiple intraluminal filling defects



Ductography

MRI

2. Pathology

FNAC

- ✓ The least invasive technique for obtaining a cytological diagnosis.
- ✓ Drawback—It can't distinguish invasive cancer from in-situ disease

Core biopsy

- ✓ Differentiates invasive cancer from in-situ cancer
- ✓ Pre-operative assessment of hormone receptors can be done

3. Routine workup

- ✓ CBC-- May show Anemia, leukocytosis...

4. Metastatic workup

- Laboratory studies
 - ALP Level
 - If there is an increase in ALP, it may suggest bone or liver metastasis
- Radiological – chest x-ray
- Abdominal Ultrasound
- Bone scan

Breast cancer staging

Questions

- For up to which TNM stage of Breast cancer is breast surgery is a treatment option?
- If there is distal metastasis, what is the TNM staging?

Management principles

Surgery

- Mastectomy /simple, modified radical or radical mastectomy/
- Lumpectomy
- Breast conservative surgery
- Sentinel lymph node biopsy

Radiation therapy

Chemotherapy

Hormonal therapy

Post mastectomy complications

- Seroma
- Wound infection
- Skin flap necrosis
- Pain
- Phantom breast disorder
- Arm morbidity
- Pneumothorax
- Brachial plexopathy

Bacterial mastitis

Bacterial mastitis can be;

1. Lactational
2. Non-lactational

Clinical Presentation

- Cardinal signs of inflammation
 - ✓ severe pain
 - ✓ swollen breast
 - ✓ Erythema
 - ✓ warm to touch
- When the cellulitic stage progress to breast abscess, there will be high grade fever & fluctuant swelling. You will be able to appreciate the fluctuant swelling unless it is deep seated.

Management

- Cellulitic stage
 - ✓ Proper antibiotics -- Penicillins or Cephalosporins
 - ✓ Analgesics
 - ✓ Appropriately fitting supportive bra
 - ✓ Warm compress
 - ✓ Emptying the breast with breast suction pump
 - ✓ If it is not resolving within 48hrs or tense indurations occur after being emptied or underlying abscess→consider repeated aspiration
- Stage of abscess
 - ✓ Consider repeated aspiration with or without ultrasound guidance. Proper antibiotic coverage needed. Staphylococcus aureus is the commonest micro-organism that causes breast abscesses.
 - ✓ Incision & drainage(I&D)—consider it for large abscesses with purulent discharge



Breast abscess

NB

Antibioma

Antibioma is a large, sterile, brawny edematous swelling that will form if antibiotic is used in the presence of undrained pus. Simply it is an antibiotic induced swelling.

Fibroadenoma

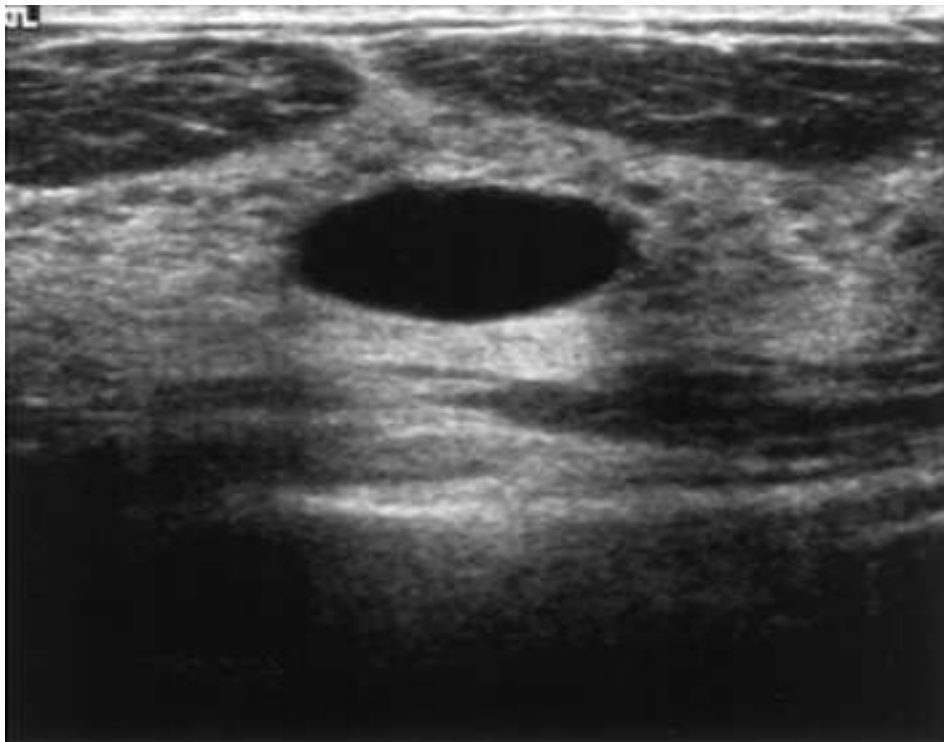
Fibro-adenomas are benign solid tumors with no malignancy potential. They are common in younger women aged 15 to 25 years.

On physical examination they are firm in consistency & slip easily under the examining fingers, also called “Breast mouse of the breast.”

On excision, they are well-encapsulated masses that may detach easily from surrounding breast tissue.

Breast cyst

Cysts within the breast are fluid-filled, epithelium-lined cavities. A palpable mass can be confirmed to be a cyst by aspiration or ultrasound. Cyst fluid can be straw colored, opaque, or dark green and may contain flecks of debris.



Breast cyst

Chapter 3

GOO



Brain storming

1. List causes of GOO?
2. List the complications of PUD?
3. List the complications of Gastric cancer?

DDx for GOO 2^o ?

Benign causes

- **PUD***
Decreasing in incidence due to triple therapy.
- Gastric polyp
- Caustic ingestion
- Gastric TB
- Pancreatic pseudocyst
- Bezoars
- Post surgical complication
- **Infantile Hypertrophic Pyloric stenosis***
(IHPS)-- Pediatric group

Malignant causes

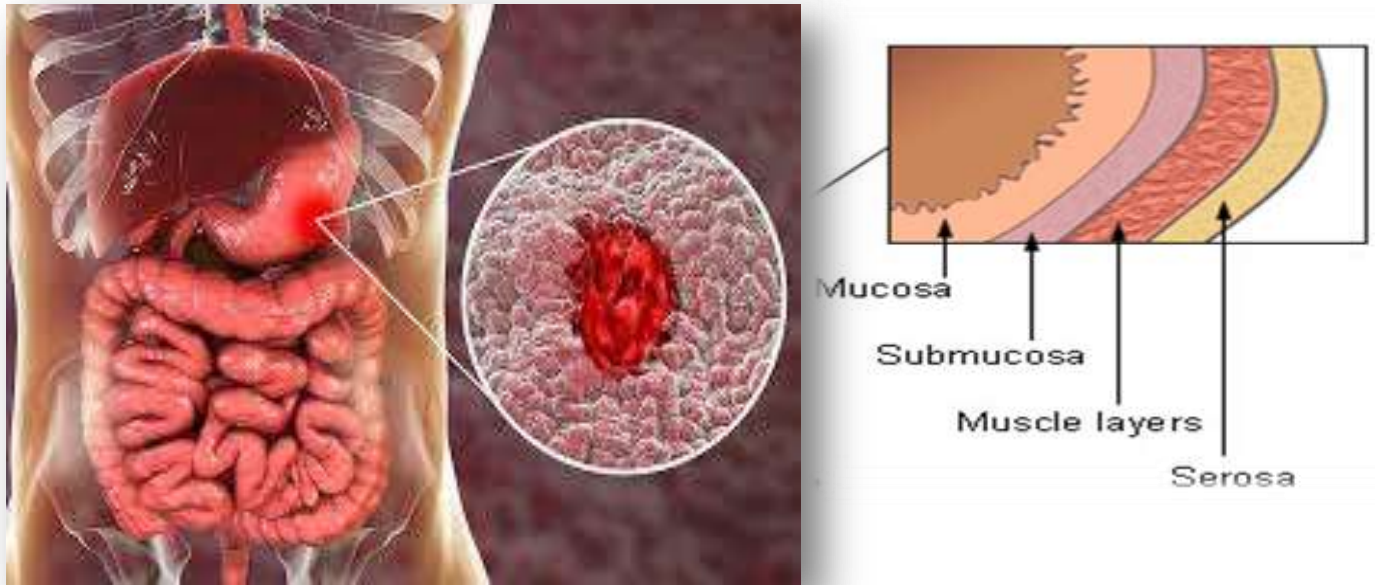
- **Gastric cancer***
- Pancreatic cancer
- Less frequent...
 - Gastric lymphoma
 - Duodenal cancer
 - Ampullary cancer
 - Cholangiocarcinoma

(*) Selected for discussion...

PUD & its complications

Introduction

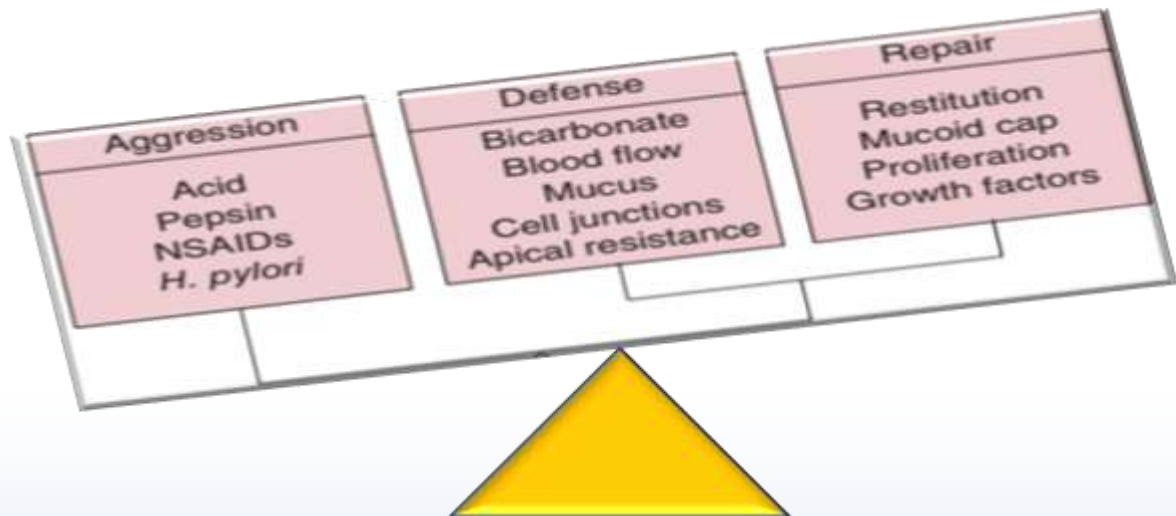
Peptic ulcers are focal defects in the gastric or duodenal mucosa that extend into the submucosa or deeper.



The natural history of PUD ranges from resolution without intervention to the development of complications like bleeding, perforation & gastric outlet obstruction.

Pathophysiology

PUD occurs due to imbalance between acid pepsin and mucosal defense mechanisms.



Risk factors

- **H.pylori infection**

Spiral or helical gram-negative rod bacteria with 4 to 6 flagella that causes 90% of duodenal ulcers and roughly 75% of gastric ulcers.

In general H. pylori predisposes to ulceration, both by acid hyper secretion and by compromising the mucosal defense mechanisms.



- **NSAIDs including Aspirin**

NSAID use causes ulcers predominantly by compromise of mucosal defenses.

Complications of PUD (specifically hemorrhage and perforation) are much more common in patients taking NSAIDs.

Patients taking NSAIDs or aspirin need concomitant acid suppressing medication if any of the following risk factors is present.

- Age over 60
- History of PUD
- Concurrent steroid intake
- Concurrent anticoagulant intake
- High-dose NSAID or acetylsalicylic acid

Aspirin*
Celecoxib
Diclofenac
Ibuprofen*
Indomethacin
Meloxicam
Naproxen*
Oxaprozin
Piroxicam



- **Smoking**

Smoking increases gastric acid secretion and duodenogastric reflux. It also decreases both gastroduodenal prostaglandin production and pancreaticoduodenal bicarbonate production.



- **Alcohol**

- **ZES/Gastrinoma**

- **Psychological stress**

- **Physiological stress/PUD in Trauma & Burn**

Curling described duodenal ulcer in burn patients. Decades later, Cushing described the appearance of acute peptic ulceration in patients with head trauma. Then the name coined as curling & Cushing ulcer.

What is the mechanism behind the formation of ulcer in such patients?

Clinical presentation

History

Abdominal pain

More than 90% of patients with PUD complain of abdominal pain. The pain is typically non-radiating, burning in quality, and located in the epigastrium.

Patients with duodenal ulcer often experience pain 2 to 3 hours after a meal and at night.

Two thirds of patients with duodenal ulcers will complain of pain that awakens them from sleep.

While the pain of gastric ulcer more commonly occurs during eating.

Associated symptoms

- Nausea, bloating
- Hematemesis/Melena



Physical examination

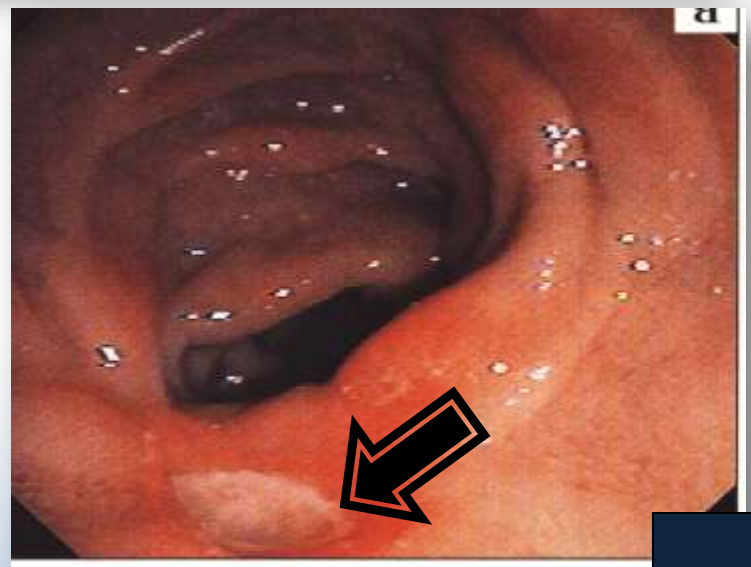
- General appearance
- Vital signs
- Epigastric tenderness ...

Investigation

UGI Endoscopy

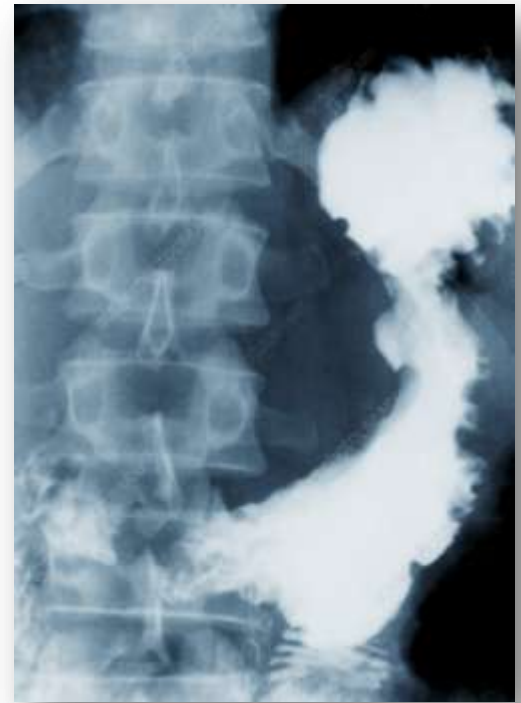
Upper GI endoscopy has both diagnostic & therapeutic importance.

- What to look?
For ulcers, protruding mass or any active bleeding



Barium meal

It demonstrates barium within the ulcer crater.



H.pylori tests (Stool antigen, Serum antibody)



H.Pylori stool Antigen Kit

ECG /electrocardiogram

ECG should be done in elderly patients & patients with co-morbid illness like DM, Hypertension & dyslipidemia who present with dyspeptic symptoms. This will help you to rule out the life threatening condition, acute coronary syndrome. The rationale behind this workup is the consideration of the dyspepsia symptom in such patients could be an angina equivalent.

Serum gastrin level

Management of PUD

Aim

- ☐ Symptomatic relief
- ☐ Healing the ulcer
- ☐ Preventing recurrence

Non pharmacologic treatment

Life style modifications

- Stop smoking
- Avoid alcohol & NSAIDS



Pharmacologic

Antacids

Mechanism of action

- Antacids react with hydrochloric acid in the stomach to form salt and water which inhibits peptic activity by raising the pH
- Magnesium antacids tend to be the best buffer



H₂-receptor antagonists

- structurally similar to histamine
- E.g., Cimetidine ,Famotidine...



Protein pump inhibitors (PPIs)

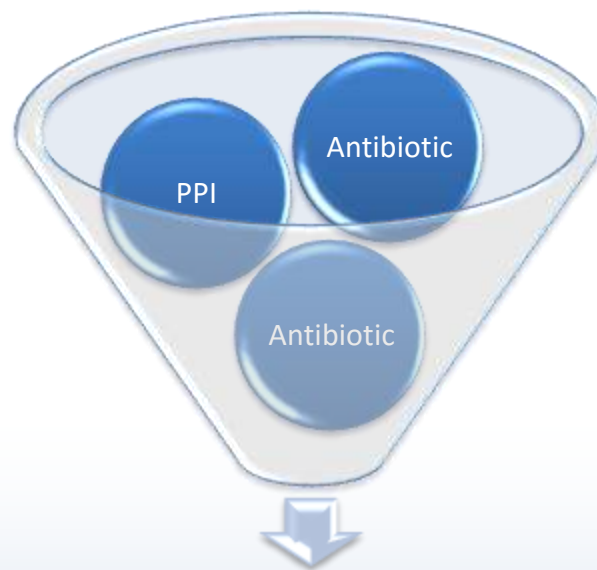
- Most potent anti-secretory agents
- E.g., Omeprazole, pantoprazole...

Sucralfate

- Dissociates under the acidic conditions of the stomach & produce a kind of protective coating that can last for up to 6 hours.

Triple (eradication) therapy

In case of confirmed H.pylori infection...



Tripple therapy

One Protein Pump Inhibitor/PPI and 2 antibiotics

✓ Antibiotics

Clarithromycin, amoxicillin or metronidazole

Duration = 2 weeks

For failure of triple therapy, quadruple therapy with bismuth added to the triple regimen is recommended.

NB

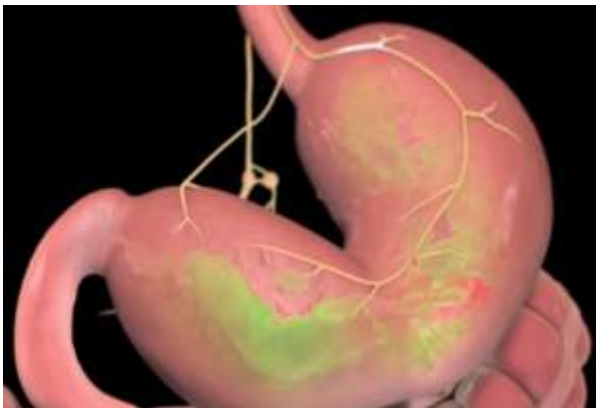
Serology tests for H.pylori shouldn't be used as a test of cure or eradication since they could remain positive for long period of time.

Surgical treatment for PUD is indicated for:

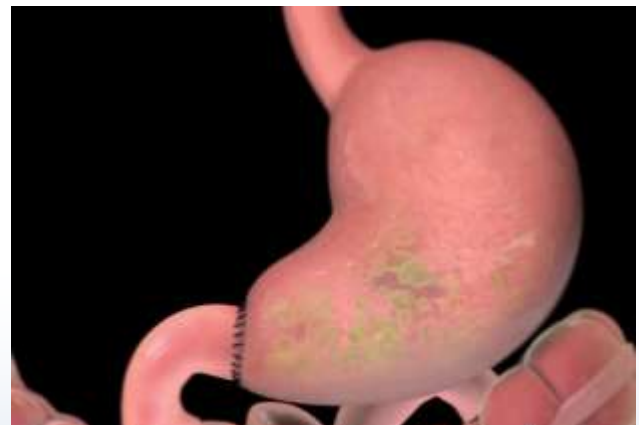
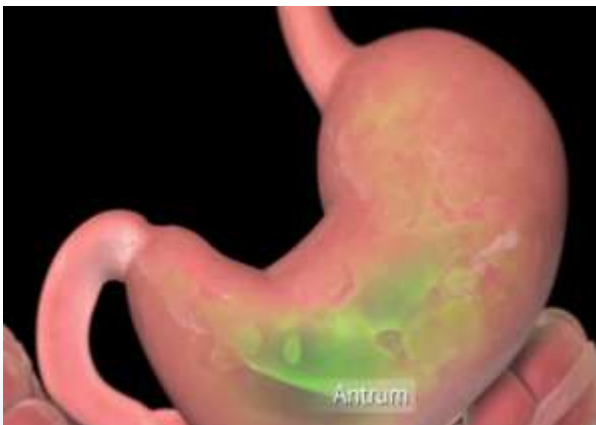
- Perforation
- Hemorrhage
- Obstruction
- Intractable ulcer

Surgery options

1-Vagotomy



2-Antrectomy



3-Partial gastrectomy

Discussion on complications of PUD

The three most common complications of PUD, in decreasing order of frequency, are

- Bleeding —The commonest complication
- Perforation —The fatal complication
- Obstruction

1. UGI bleeding 2^o PUD



Upper GI bleeding refers to bleeding that arises from the GI tract proximal to the ligament of Treitz.



History

Patients with a bleeding peptic ulcer typically present with melena and/or hematemesis.

Hematemesis

- Hematemesis is the vomiting of red blood or coffee-ground material from bleeding in the GIT above the ligament of Treiz(duodenojejunal flexure)
- On your HPI describe the mode of onset, duration, frequency and amount



NB

Hematochezia is the passage of bright red or maroon blood from the lower GIT bleeding. The usual source of bleeding is from the sigmoid colon, rectum or anal canal of various causes .

Melena

Melena is the passage of tarry, foul smelling stool which indicates bleeding above ileo-cecal valve.



Melena

Physical examination

- General Appearance
- Vital signs
 - Shock may be present, necessitating aggressive resuscitation and blood transfusion
- Look for systemic signs of Anemia

Procedures

Nasogastric aspiration is usually confirmatory of the upper GI bleeding & reduces patients' need for vomiting.

DDx of UGIB

Mallory Weiss syndrome

- It is a mucosal and submucosal tear that occur near the gastroesophageal junction.
- Classically, these lesions develop in alcoholic patients after a period of intense retching and vomiting after binge drinking, but they can occur in any patient who has a history of repeated emesis.

Esophageal varices

- Esophageal varices are dilated veins in distal esophagus or proximal stomach due to elevated portal venous system pressure.
- 90% of cirrhotic patients develop esophageal varices & 25-30% of develops hemorrhage.
- Present with sudden, painless UGIB which is often massive.

Erosive esophagitis

- Exposure of the esophageal mucosa to the acidic gastric secretions in GERD/Gastro-esophageal reflux disease leads to an inflammatory response, which can result in blood loss.

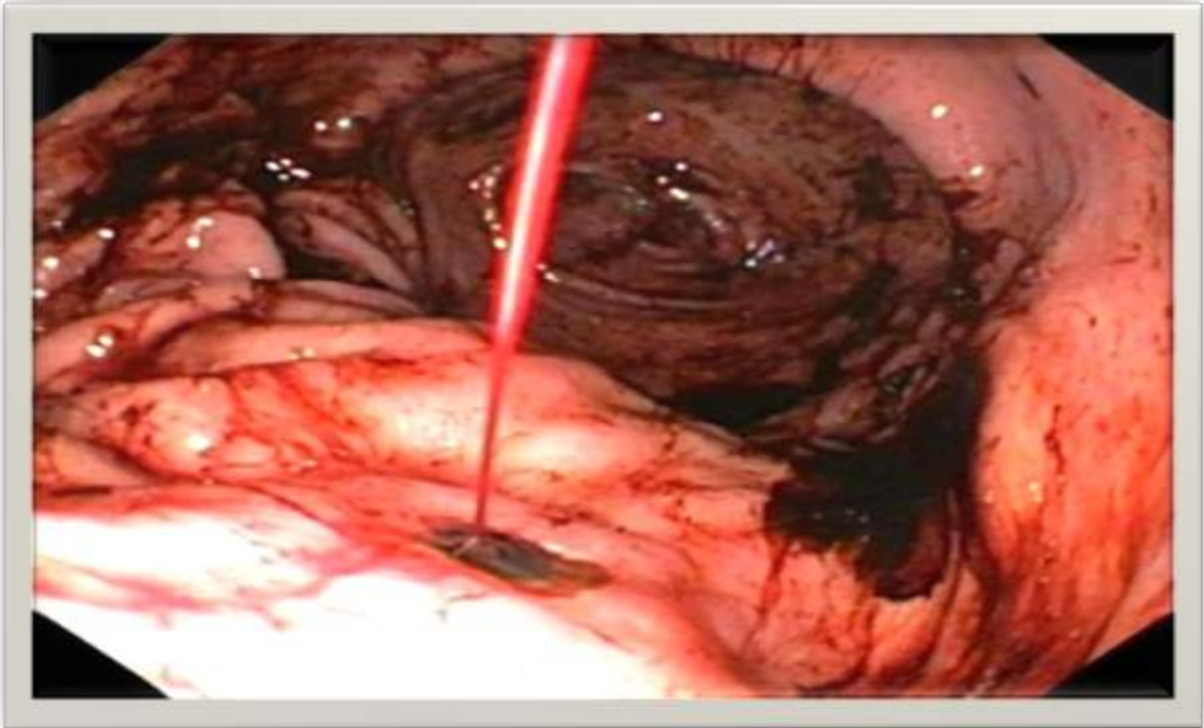
Gastric cancer

- Discussed in detail on the next section

Investigation

- **Hematocrit**
Severe anemia may be masked by the hemoconcentration early in the course
- **Blood group & Rh**
- **Cross match**

- **Endoscopy** should be done early to diagnose the cause of the bleeding and to assess the need for hemostatic therapy



UGIE of actively bleeding ulcer

Management

Whatever the cause of UGIB, the principles of management are identical;

- ABCD of life, The patient should be resuscitated (Fluid, blood)
- Investigate urgently to determine the cause of the bleeding
- Definitive treatment then follows

Indications for Surgery in Gastrointestinal Hemorrhage

- ✓ Hemodynamic instability despite vigorous resuscitation (>6 units transfusion)
- ✓ Failure of endoscopic techniques to arrest hemorrhage
- ✓ Recurrent hemorrhage after initial stabilization (with upto two attempts at obtaining endoscopic hemostasis)
- ✓ Shock associated with recurrent hemorrhage
- ✓ Continued slow bleeding with a transfusion requirement exceeding 3 units/day

2. Perforated PUD



History

Perforated peptic ulcer usually presents as an acute abdomen. The patient can often give the exact time of onset of the excruciating abdominal pain.

Physical examination

General appearance

Acutely sick looking

Vital signs

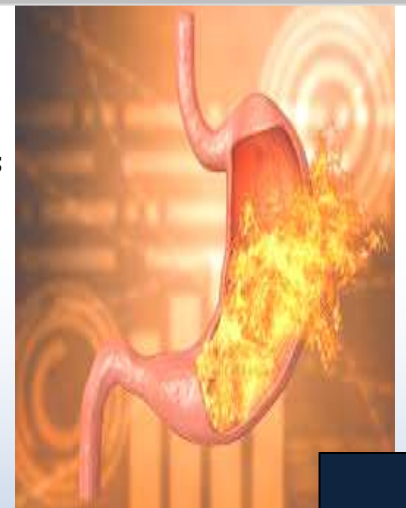
Frequently accompanied by fever, tachycardia & respiratory distress

Look for systemic signs of dehydration

Look for Signs of ileus

Look for Peritoneal signs

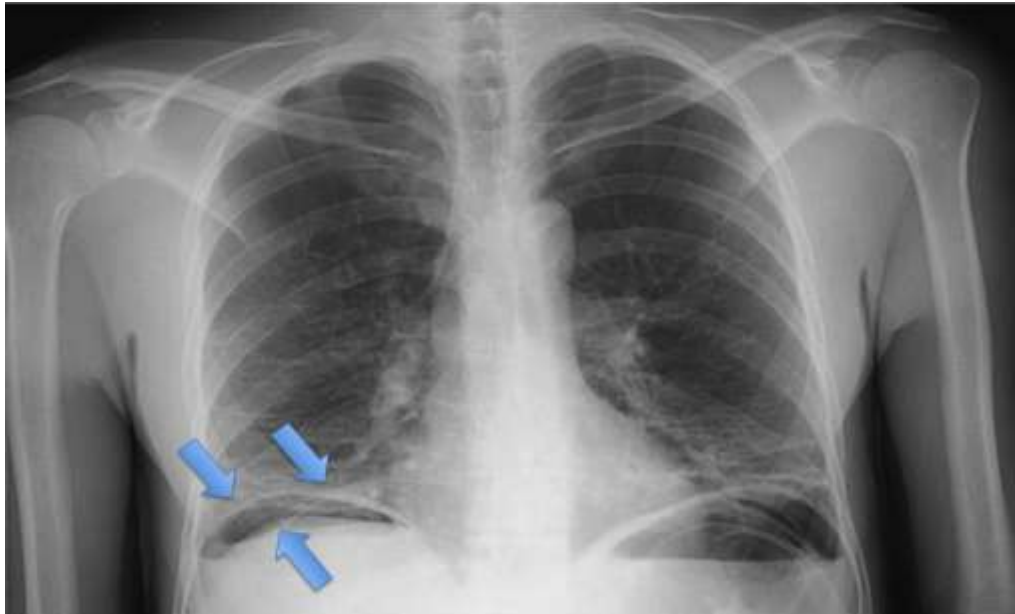
Usually, marked involuntary guarding and rebound tenderness is evoked by a gentle abdominal examination in peritonitis patients.



Investigation

Upright chest X-ray

Free air is found in about 80% of patients under the right hemi-diaphragm.



Management

Surgical emergency

After the diagnosis is made, operation is performed in an expeditious fashion following appropriate fluid resuscitation



3. Gastric Outlet Obstruction/GOO 2^o PUD

Introduction

Gastric outlet obstruction is usually due to duodenal or pre-pyloric ulcer disease. The obstruction may be an acute one from inflammatory **swelling** and peristaltic dysfunction or chronic from **fibrosis**.

Clinical Presentation

Patients typically present with non-bilious vomiting. Weight loss may be prominent depending on the duration of symptoms.

On physical examination succussion splash may be audible with stethoscope placed in the epigastrium.

Investigation

- The diagnosis is confirmed by endoscopy.



- Barium Meal
- Serum electrolytes may show profound hypokalemic hypochloremic metabolic alkalosis secondary to loss of gastric juice rich in hydrogen, chloride, and potassium ions.
- Renal function test— Pre-renal azothemia, acute kidney injury

Management

- Insert Naso-Gastric tube/NGT for suctioning & relief of the obstructed stomach
- Rehydrate the patient with IV fluid
- Correct electrolyte disturbance
- Medical management (gastric acid suppressants)
- Definitive management

Surgery

Type of surgery depends upon the cause of obstruction

✓ In GOO 2^o PUD, Vagotomy & antrectomy → standard

Gastric surgery complications

Early Complications

- Bleeding
- Anastomotic leak
- Stomal obstruction
- Duodenal blow out (Billroth-II)
- Afferent Loop Syndrome
- Efferent loop Syndrome

Late Complications

- Ulcer recurrence
- Alkaline gastritis
- Dumping Syndromes
- Post Vagotomy diarrhea
- Malabsorption Syndromes

Gastric cancer

Introduction

Gastric cancer remains one of the most common forms of cancer worldwide. It was the leading cause of cancer deaths in the world until 1980s when it was overtaken by lung cancer.

Its incidence & death rate in western countries has declined over the recent few decades.

Types

- Adenocarcinoma ~ > 95 %
- Lymphoma ~ 4 %...

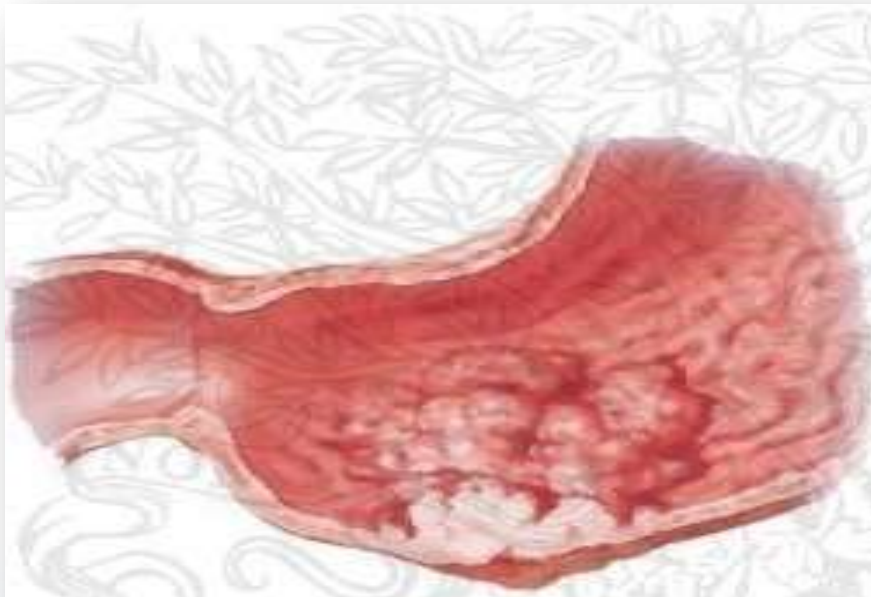
Metastasis sites

Common sites of metastasis

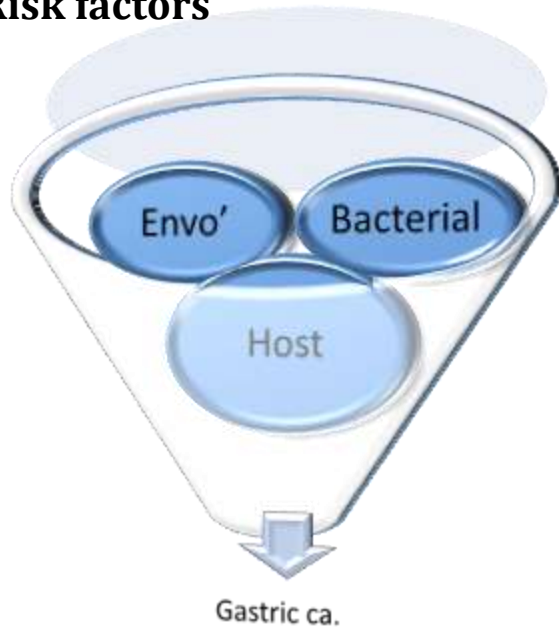
- Liver
- Peritoneal surfaces
- Lymphnodes

Less common sites of metastasis

- Ovaries
- CNS
- Bone
- Lungs



Risk factors



Bacterial

- H.Pylori remains an important risk factor for Gastric cancer. Patients have 3 times increased risk.
- Atrophic gastritis → intestinal metaplasia → dysplasia...

Environmental factors

- Dietary
 - Use of salted & smoked foods as preservative
 - ✓ Dietary nitrates have been impugned as a possible cause of gastric cancer. Gastric bacteria convert nitrate into nitrite, a proven carcinogen.
- Life style
 - Smoking & alcohol consumption
- Radiation exposure

Host related

- Obesity
- Familial predisposition
- Pernicious anemia
- Gastric polyps
- FAP, HNPCC
- Previous gastric surgery
- Blood Group A

Clinical presentation

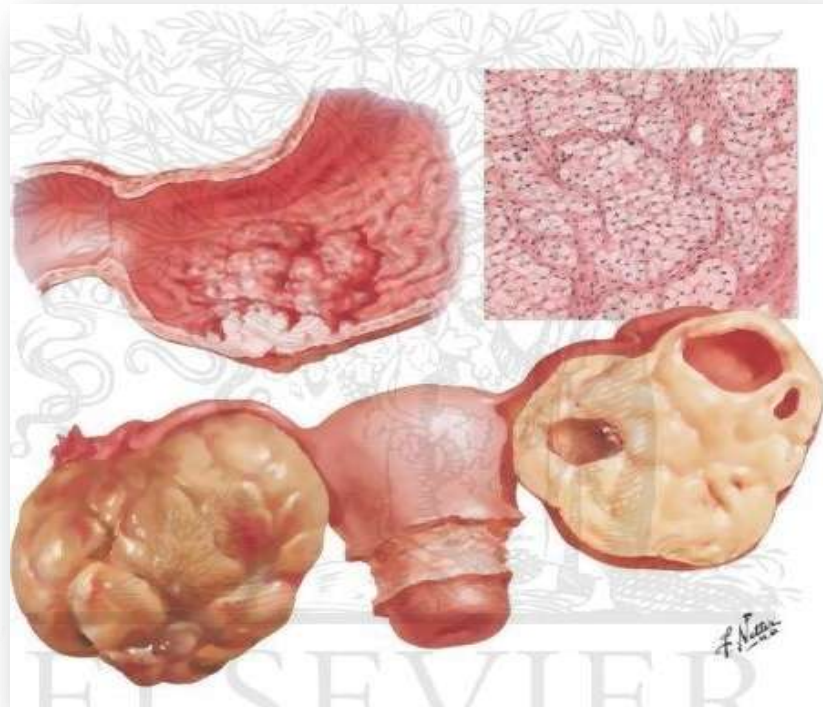
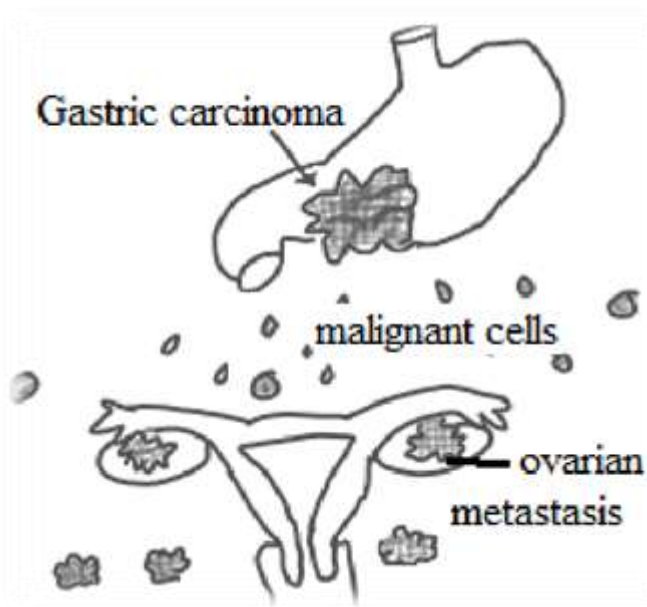
Early stages of gastric cancer are asymptomatic. Most patients have advanced incurable disease at the time of presentation.

Symptomatic in advanced stage;

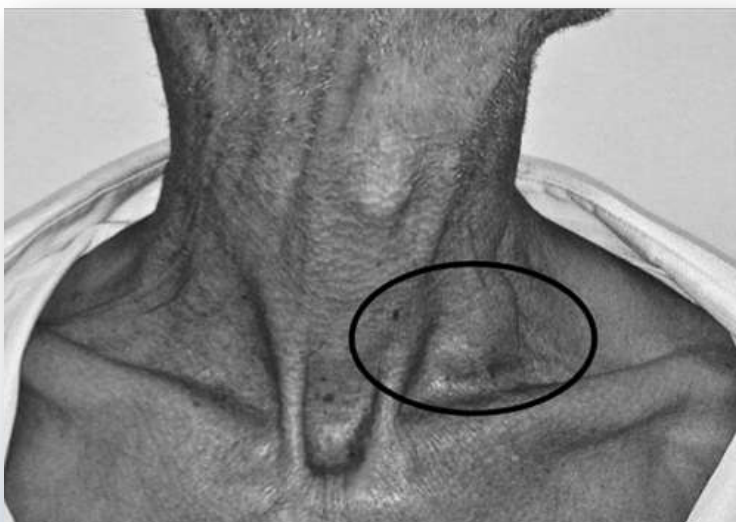
- Constitutional symptoms of malignancy
 - Weight loss, Anorexia & Easily fatigability
- GI symptoms
 - Early satiety (due to mass effect or poor distensibility)
 - Nausea, vomiting...
- Symptoms & signs of anemia
 - Overt bleeding in <20% of patient-- melena/hematemesis
 - Chronic occult blood loss is common and manifests as iron deficiency anemia and heme-positive stool
- Dysphagia
 - Common if the cancer is in the proximal stomach
- Advanced distal tumors
 - symptoms & signs of GOO
- Metastasis
 - Liver--Icteric sclera, Hepatomegally



- Metastasis to peritoneal surfaces
 - Blumer's/rectal shelf on digital rectal examination
 - Ascites
 - Krukenberg's tumor—drop metastasis to ovary



- Lymphnode metastasis
 - Virchow's node
 - Sr. Marry Joseph's nodule
 - Irish node



Virchow's node

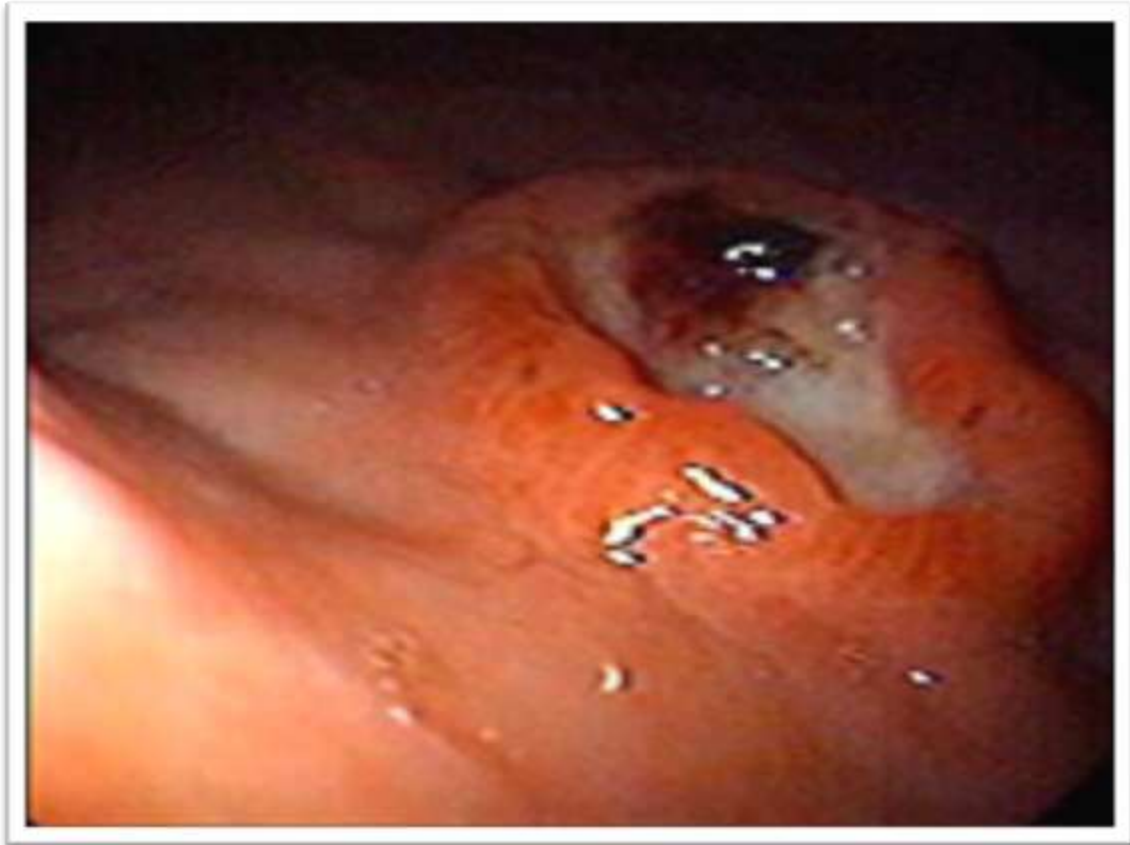


Sr. Marry Joseph's nodule

Investigation

Diagnostic

- UGI Endoscopy



Imaging

- Double contrast barium meal

Labratory

- CBC
- Liver and Renal function tests
- Serum electrolytes
- Coagulation profiles
- Tumor markers: CEA , CA 19-9, CA724

Staging investigations

- EUS (endoscopic ultrasonography)
- CT of abdomen/pelvis, MRI
- CXR...

Staging

Read on TNM staging of gastric cancer

Management

Multidisciplinary approach

- Surgery
- Chemotherapy
- Radiation
- Combination of the above

IHPS/Infantile hypertrophic pyloric stenosis

Common in 3-6weeks of age

Risk factors

- Male sex
5 times more common in males
- Family history
- Drugs
Erythromycin in early infancy
- B & O blood group

Clinical presentation

History

- Non-bilious vomiting
 - Progressively become projectile
 - Occurs immediately after feeding
- After vomiting the patient becomes very hungry & wants to feed again
- The patient becomes increasingly dehydrated. Wet dippers become less frequent
- Yellowish discoloration of the body (Jaundice)→due to indirect hyperbilirubinemia

Physical examination

- Palpation of “olive” shaped, firm, movable mass in the RUQ of the abdomen, a pyloric mass. Best palpated after vomiting.
- Presence of visible gastric peristalsis from left to right best seen after eating
- Look for signs of dehydration
The 4 important DHN signs & symptoms in well-nourished child are:
 - Mental status
 - Eye ball sunkenness
 - Drinking
 - Skin turgor

Investigation

Abdominal Ultrasound

What to look for IHPS on ultrasound?

- Channel length
In IHPS >16mm
- Pyloric thickness
In IHPS >4mm
- Pyloric diameter
In IHPS <12mm

Contrast studies

What to look?

- String sign
Due to elongated pyloric channel
- Shoulder sign
Due to bulging of pyloric muscle into the antrum
- Double tract sign
Parallel streaks of barium in the narrowed channel

Management

- **Medical emergency not surgical**

Fluid resuscitation with correction of electrolyte abnormalities

NB***IHPS is associated with Hypochloric, hypokalemic metabolic alkalosis

- **Surgical management**

Pyloromyotomy

Sample history

Chief complaint

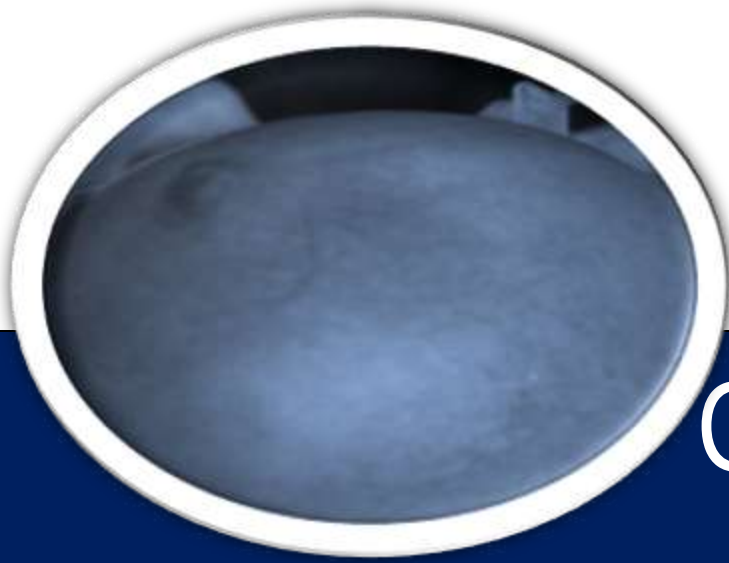
Vomiting of 1 month duration

HPI

This patient was last relatively healthy 1 month back at which time he started to experience non-projectile, non-blood tingled, non-bilious vomiting of ingested matter 2-3 times/day. The vomiting always starts about 2 hours after taking meal and it is aggravated by hunger. Associated with this he has loss of appetite, feeling of early satiety & significant weight loss of 4kg for the past 06 months (from 70 to 66kg → >5% in 06 month). 04 days prior to admission he started to experience projectile, blood tingled, non-bilious vomiting of ingested matter 5-times per day. In addition he has tinnitus, blurring of vision & light headedness. For the past 02 years he was having intermittent burning type of epigastric pain with no radiation which was aggravated by taking spicy foods like "key wot." The pain usually awakens him at night. For the above complaint he visited a nearby health center where he was given Omeprazole to be taken 2 times/day for 14 days.

- ✓ *No history NSAID use*
- ✓ *No history of cigarette smoking or chronic alcohol consumption*
- ✓ *His regular dietary habit is 'injera' made of 'teff' & 'shiro' made of 'atter.'*
- ✓ *He has no history of previous abdominal surgery.*
- ✓ *No history of burn or trauma to the head*
- ✓ *No history of similar illness in the family*
- ✓ *No history of radiation therapy*
- ✓ *No history of swelling in the neck or axilla*
- ✓ *No history of yellowish discoloration of the eye or itching sensation*
- ✓ *No history of chronic cough, contact with chronic cougher or previous TB treatment*
- ✓ *No self/family history of DM, Hypertension or asthma*
- ✓ *Has been screened for RVI 08 months back & found to be NR*

Finally he was admitted to our hospital supported by his families.

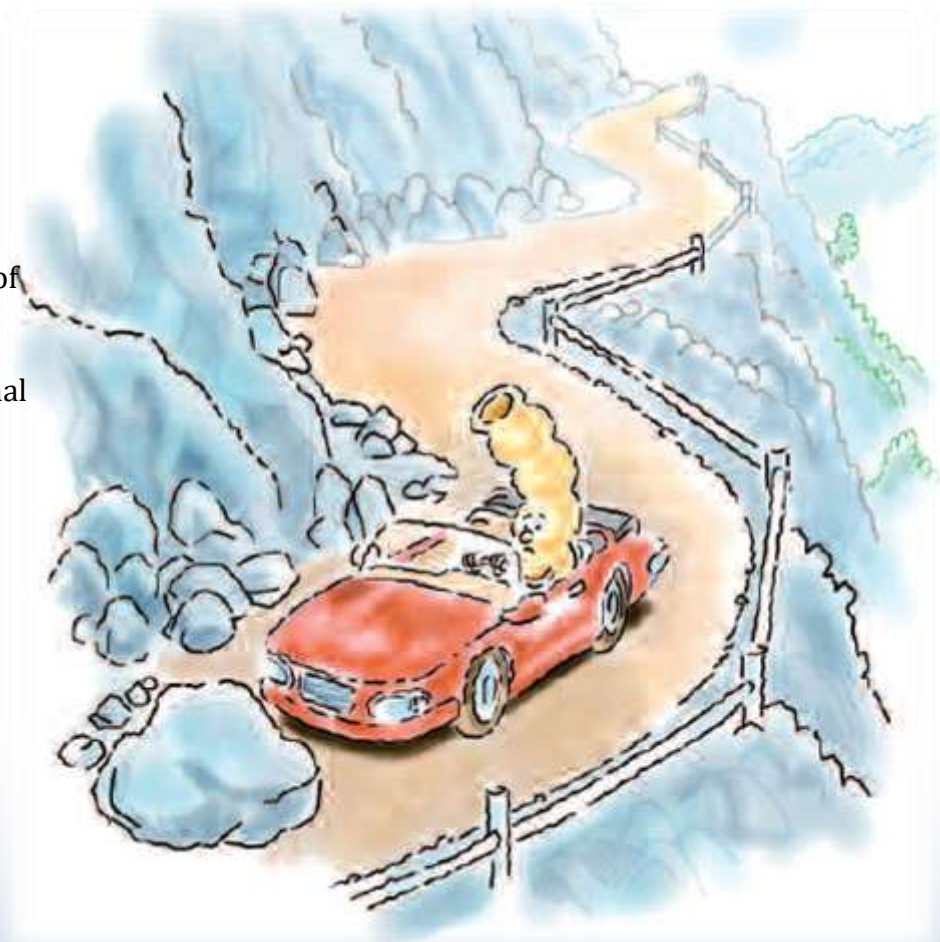


Chapter 4

Intestinal obstruction

Brain storming

1. By listing the cardinal features of obstruction ,try to differentiate SBO from LBO?
2. List the triads on plain abdominal x-ray for SBO & LBO?
3. How you manage gangrenous sigmoid volvulus?



Introduction

Intestinal obstruction occurs when the luminal content of the GIT is prevented from passing distally.

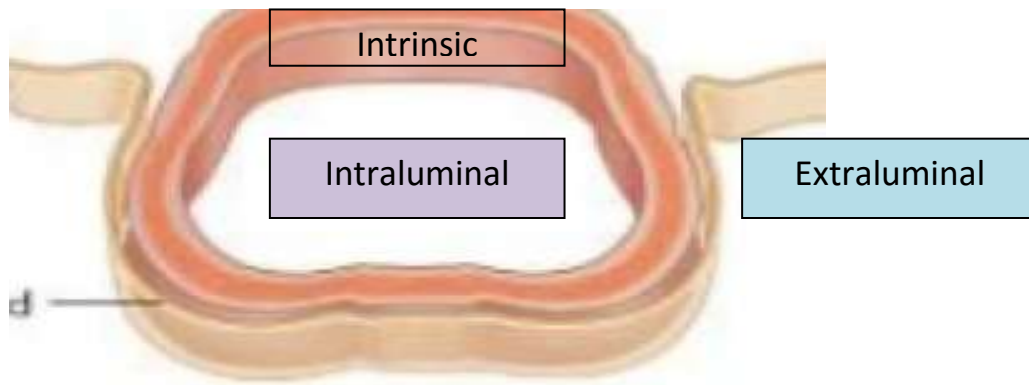
Classification

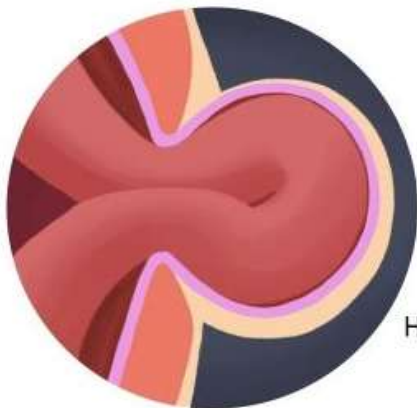
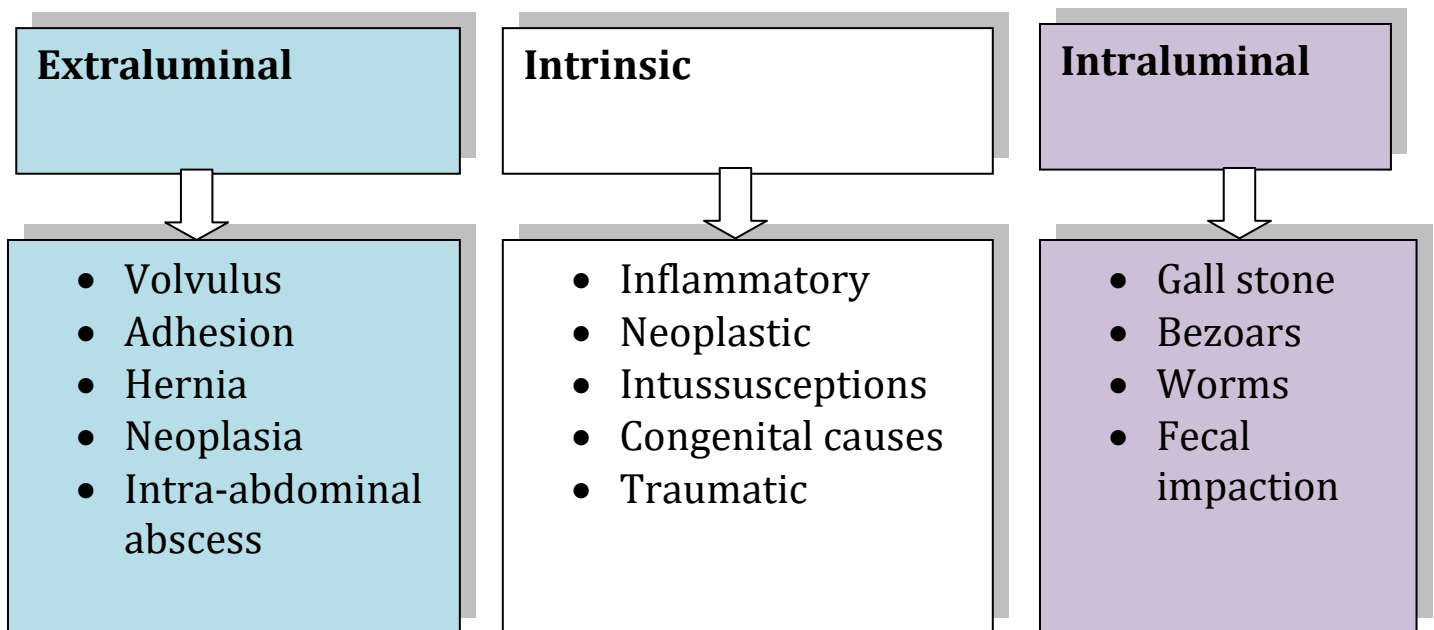
1. Based on type, it could be;
 - ✓ Dynamic (Mechanical) Obstruction
 - ✓ Adynamic Obstruction (Paralytic Ileus)
2. Based on lumen, it could be;
 - ✓ Complete obstruction
 - ✓ Incomplete obstruction
3. Based on presence of complications, it could be;
 - ✓ Simple/viable
 - ✓ Strangulated
4. Based on the intestine involved, it could be;
 - ✓ Small bowel obstruction
 - ✓ Large bowel obstruction

Causes

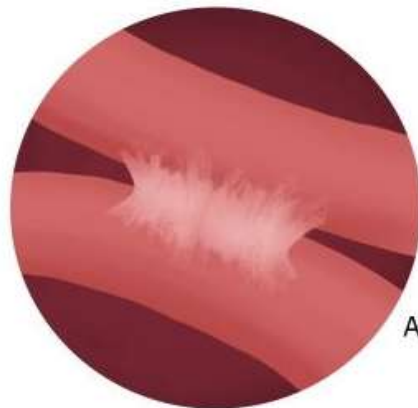
Causes of intestinal obstruction can be classified in to;

1. Extraluminal causes of intestinal obstruction
2. Intrinsic causes of intestinal obstruction
3. Intraluminal causes of intestinal obstruction

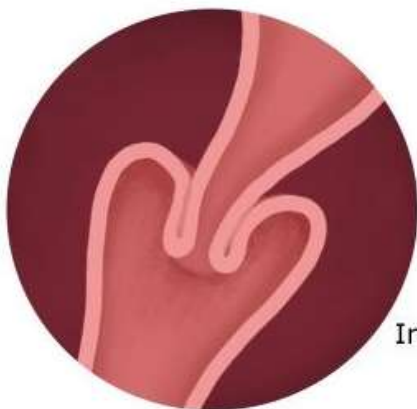




Herniation



Adhesions



Intussusception



Volvulus

Volvulus

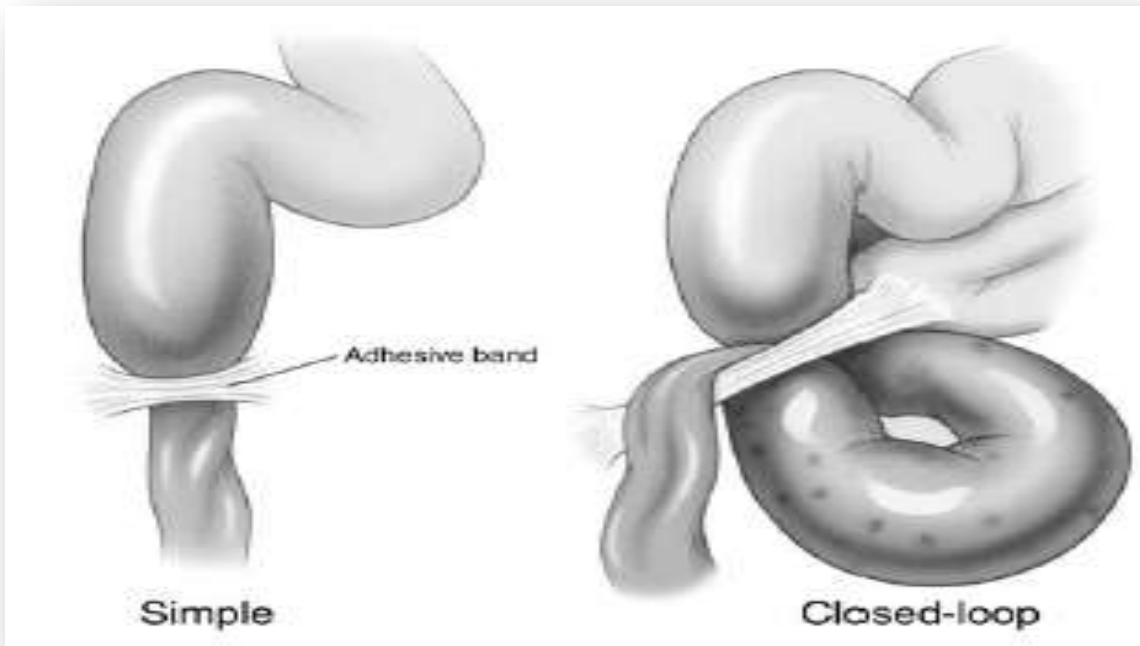
Volvulus is a twisting of portion of bowel about its mesentery. It is common in rural parts of Ethiopia because of;

- Redundant small bowel (vegetarians like dietary habit)
- Heavy meal (1-2times/day) &
- Strong abdominal muscle



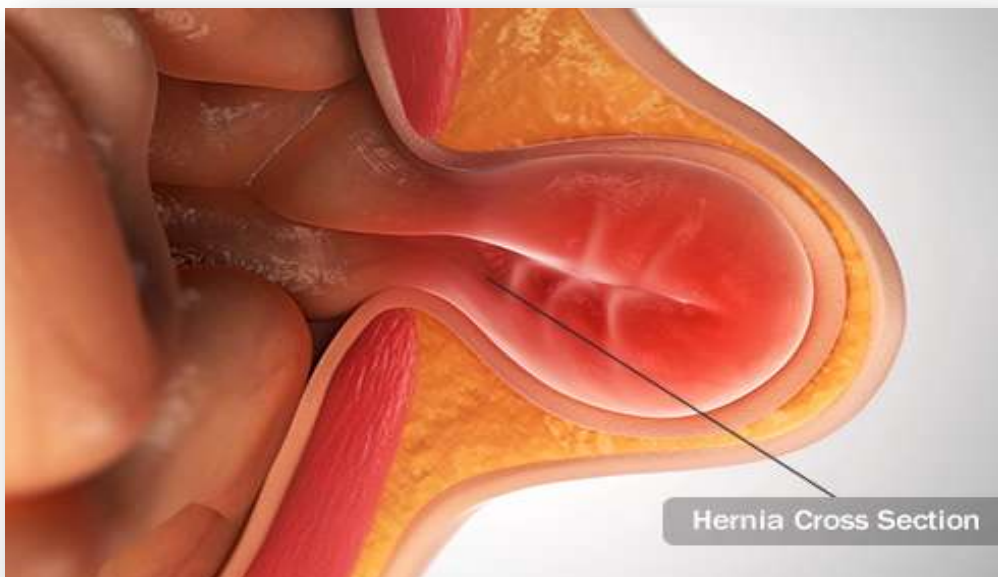
Post-op adhesion

- In patients with previous history of abdominal or pelvic surgery
- Common in developed countries & major cities of Ethiopia



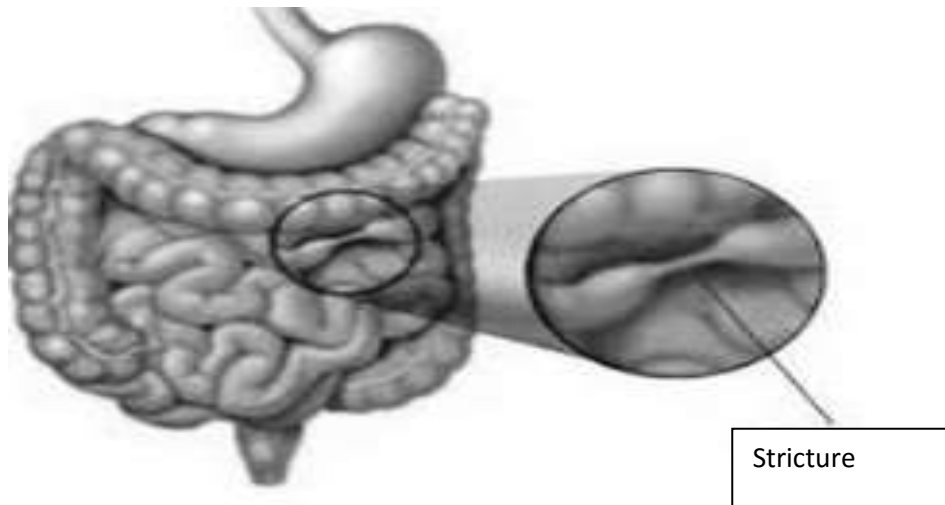
Incarcerated hernia

- Incarceration means permanent trapping. The portion of the intestine may become imprisoned in the defect which allowed the hernia.
- Strangulated hernia
Arterial & venous occlusion of an incarcerated hernia.



Stricture

- Inflammatory bowel disease --Crohn Disease
- Diverticular disease



Infection

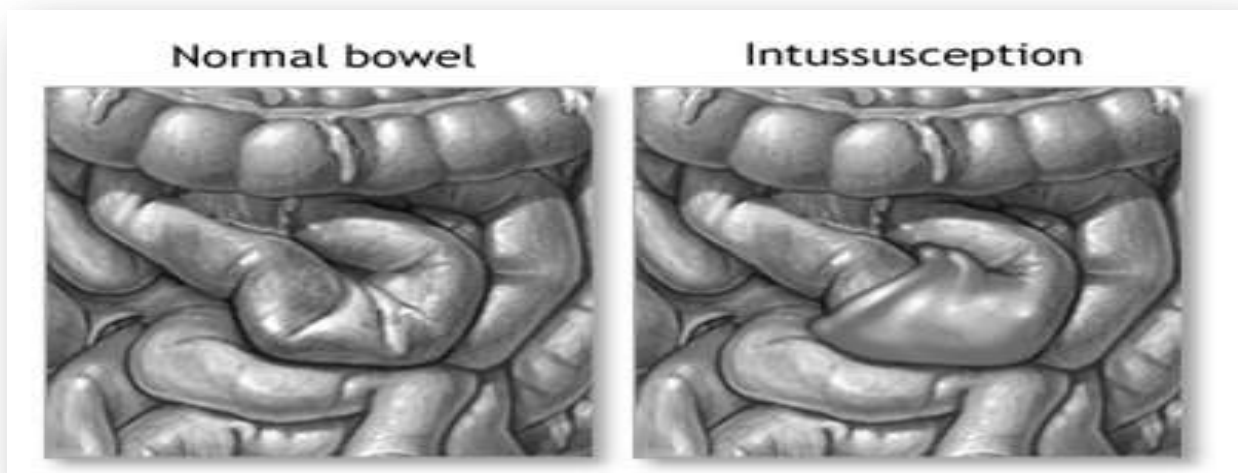
- Intestinal TB...

Malignancy

- Colonic cancer, lymphoma...

Intussusception

- Pediatric group



Fecal impaction

Imperforate anus—In Neonate

History

The four cardinal symptoms

i. Nausea & Vomiting

- ✓ More frequent in proximal obstruction
- ✓ If the timing between the onset of the abdominal pain & the vomiting is early suspect proximal obstruction

ii. Crampy abdominal pain

- ✓ Simple obstruction--intermittent pain
- ✓ Strangulated—steady/constant pain
- ✓ Obstruction located distally is associated with less emesis. The initial and most prominent symptom is the abdominal pain.

iii. Constipation

- ✓ Absolute constipation/obstipation is the absence of feces & flatus
- ✓ Patients with relative constipation can pass flatus

iv. Distension

- ✓ In distal SBO the abdominal distension is central
- ✓ In proximal SBO the distension is minimal
- ✓ In LBO patients experience pronounced distension

Physical examination

General Appearance

- Acutely sick looking (in pain, vomiting...)
- Quietly lying down or not?

Vital signs

- tachycardia and hypotension, due to the severe dehydration
- Fever suggests the possibility of strangulation

Look for systemic signs of dehydration

- HEENT, Skin turgor, capillary refill

Abdominal examination

- Inspection
 - Abdominal distension, visible peristalsis, Look for surgical scars, Look hernia sites carefully...
- Auscultation--Bowel sounds
 - Early--Hyperactive because of the effort to propel luminal contents past the obstructing point
 - Later--Hypoactive because the intestine will become fatigued and dilates, with contractions becoming less frequent and less intense
- Palpation--Tenderness, guarding and rigidity suggest peritonitis
- Percussion
 - Tympanicity /hepertympanic
 - Look also for signs of fluid collection
- *Digital Rectal Examination (DRE)**
 - Presence or absence of fecal matter
 - Blood on examining finger may suggest malignancy or strangulation

Sample history

Chief complaint

Abdominal pain of 3 days duration

HPI

This patient was last relatively healthy 3 days back at which time he started to experience sudden onset severe intermittent crampy periumbilical abdominal pain without known aggravating or relieving factor. There was no radiation of the abdominal pain noticed by the patient. 3 hours after the onset of the pain he started to experience bilious, non-projectile, non-blood tingled, non-foul smelling vomiting about 5-7times/day. Associated with this he started to experience failure to pass flatus of 03 days duration associated with abdominal distention. 01 day prior to presentation he totally failed to pass feces.

For the above compliant he visited a local health center where he was given 01 bag of Normal saline and referred to our hospital for better investigation and management.

- *No history of previous abdominal surgery*
- *No history of fever, weight loss or swelling in the neck, axilla & groin*
- *No history of chronic cough, contact with chronic cougher or previous TB treatment*
- *No history of yellowish discoloration of the eyes or itching sensation*
- *His regular diet is “shiro” made of “atter” & “injera” made of “teff”, 3times/day.*
- *No self/ family history of DM,HTN or asthma*
- *Not screened for RVI but he has no history of multiple sexual partner, chronic diarrhea or HZV attack.*

Finally he was admitted to our hospital.....

Investigation

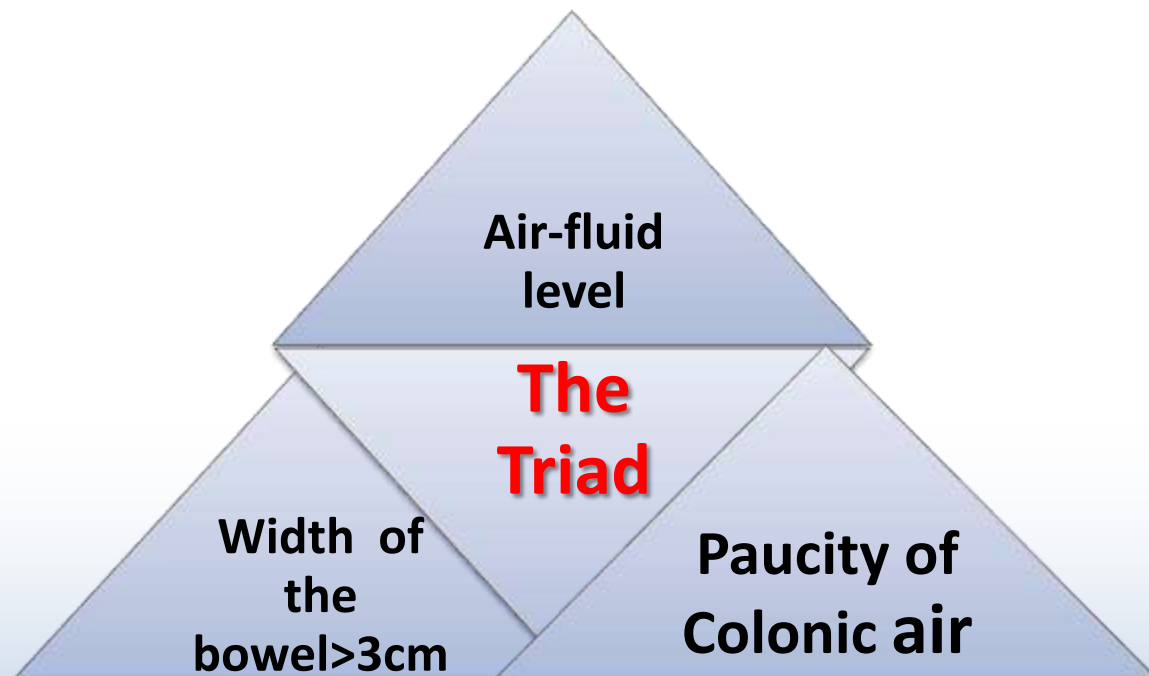
Imaging

Plain abdominal x-ray





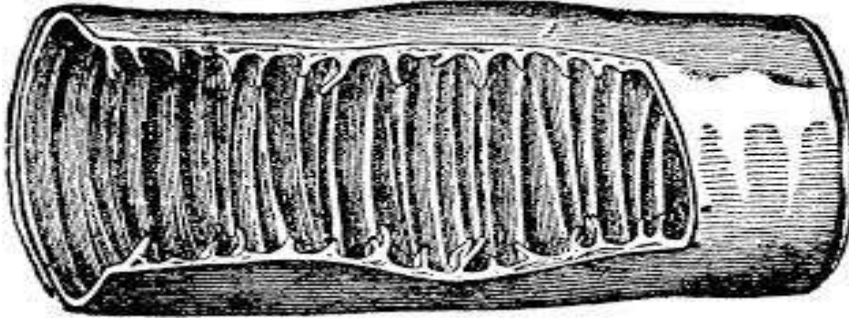
What to look on plain abdominal x-ray for SBO? The triad!



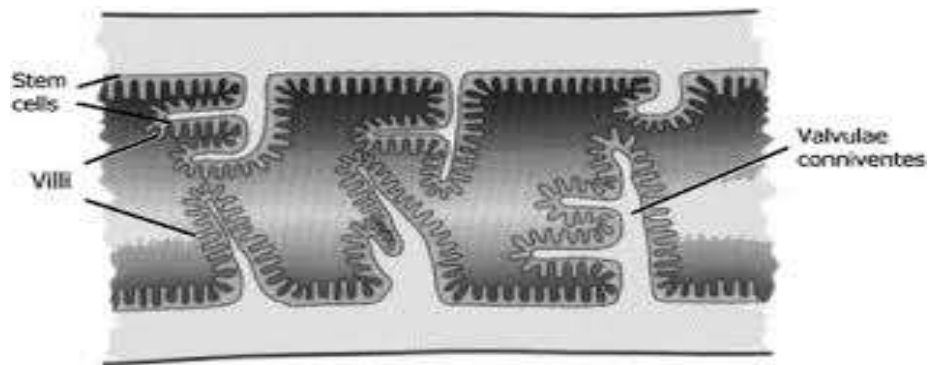
Additional evidences for small bowel involvement on x-ray;

- Site
The obstructed bowel is central & lies transversely
- Anatomical landmarks

Valvulae conniventes of small bowel completely pass across the width of the bowel & are regularly spaced “**coiled spring appearance**”



Portion of small bowel



Coiled spring

Laboratory

- Complete blood count(CBC)
 - ✓ Leukocytosis → ischemic bowel
 - ✓ Hemoconcentration
- Serum electrolytes
- RFT

Management

- Supportive management
 - Definitive management
- i. Supportive management
- ✓ Keep NPO/nothing per Os
 - ✓ Secure Intravenous/IV line
 - ✓ Resuscitation, electrolyte replacement
 - ✓ NGT/Nasogastric tube decompression



- ✓ Catheterize
- ✓ Antibiotics
- ✓ Monitor input, output, vital signs and abdominal condition

ii. Definitive management

Surgical treatment

- ✓ Operative decompression
 - The type of surgical procedure required will depend on the cause of obstruction

NB*

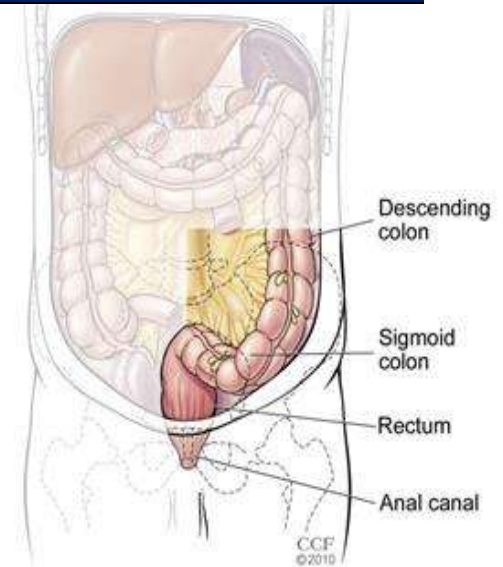
What are the causes of dehydration & electrolyte loss in such patients?

- 1) Reduced oral intake
- 2) Defective intestinal absorption
- 3) Loss as a result of vomiting
- 4) Sequestration in the bowel lumen
- 5) Transudation of fluid in to the peritoneal cavity

Large Bowel Obstruction/LBO

Risk factors

- Age
Elderly
- Anatomical predisposing factors
For e.g., in Sigmoid volvulus;
 - Long mesentery
 - Narrow base
 - Elongated colon (redundant)
- Diet
High residue diet
- Chronic constipation...



Clinical presentation

History

The four cardinal symptoms

- i. Abdominal distension
- ii. Failure to pass feces & or flatus
- iii. Crampy abdominal pain
- iv. Nausea & Vomiting

Onset of symptoms

In abrupt onset consider acute obstructive events like Cecal or sigmoid volvulus.

In gradual /Chronic process consider diverticular disease, malignancy...

Physical examination

- ☐ General appearance
- ☐ Vital signs
- ☐ Signs of dehydration
- ☐ Abdominal examination, Digital rectal examination

Abdominal examination

- Inspect
Distension, hernia sites...
- Auscultation
- Palpation
Look for tenderness...
- Percussion
Hyper-tympanicity...

DRE

- Hard stool—fecal impaction
- Empty vault--obstruction proximal to the level your finger can reach

Sample History

Chief complaint

Failure to pass feces of 3 days

HPI

This patient was last relatively healthy 03 days back at which time he started to experience severe intermittent crampy lower abdominal pain without known aggravating or relieving factor. Associated with this he has failure to pass feces of 03 days & flatus of 02 days duration with progressive distension of the abdomen. He has nausea but no vomiting.

He had history of similar episode #03 months back for which he was deflated with rectal tube in our hospital.

- ✓ *No history of rectal bleeding, tenesmus or weight loss*
- ✓ *No history of tinnitus, vertigo or blurring of vision*
- ✓ *No history of previous abdominal surgery*
- ✓ *No history of chronic cough, contact with chronic cougher or previous TB treatment*
- ✓ *No self/family history of DM, HTN or asthma*
- ✓ *Screened for RVI 2 months back & found to Non reactive*

Finally he was admitted to our hospital supported by his families

Investigation

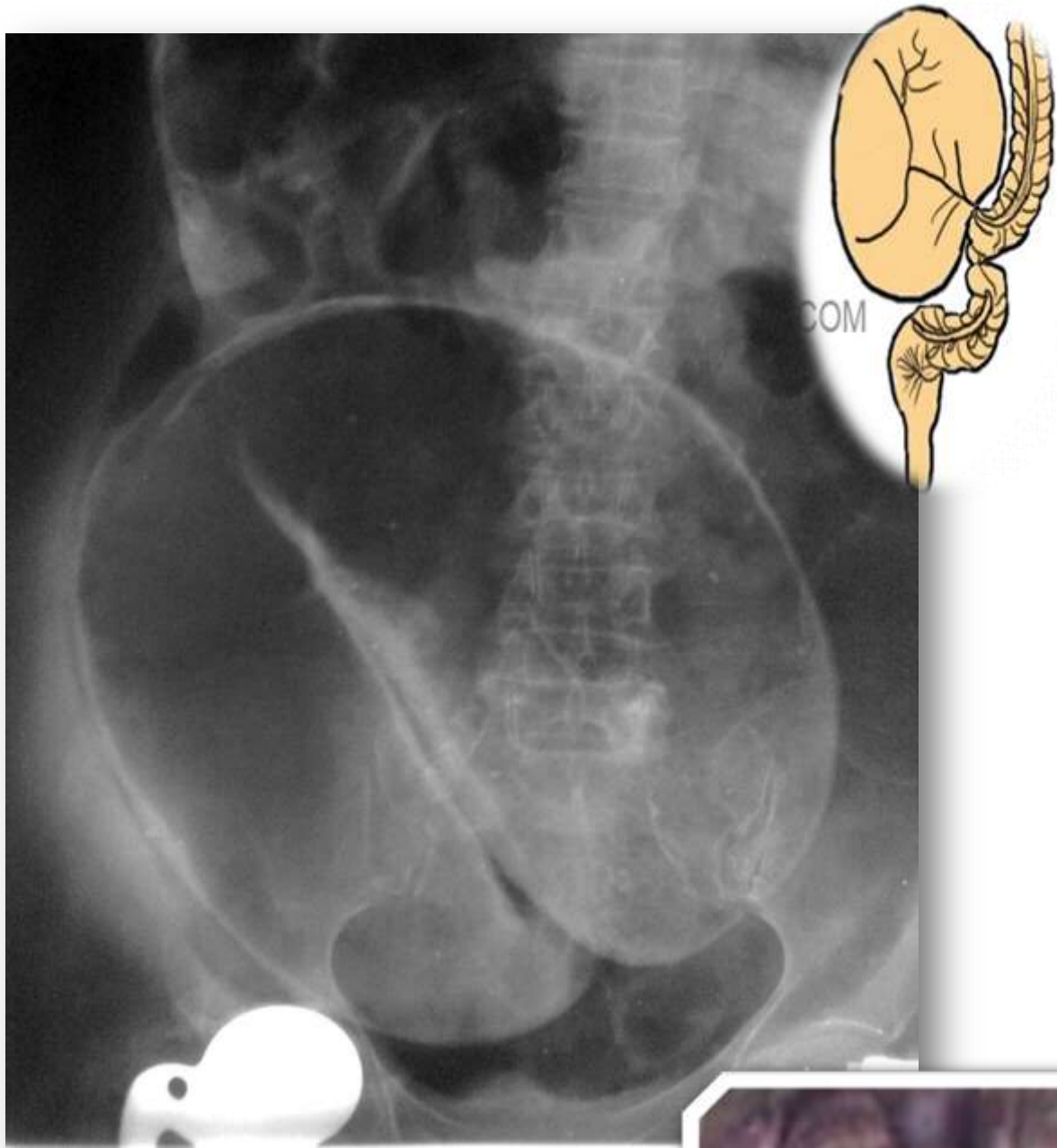
Imaging

Plain abdominal x-ray

What to look on the x-ray for LBO?

1. Dilated bowel loop
 - >06 cms for large bowel
 - >09 cms for cecum
2. Paucity of air
 - Absent or reduced air in the rectum
 - Important to differentiate complete from partial Obstruction
3. Air fluid level



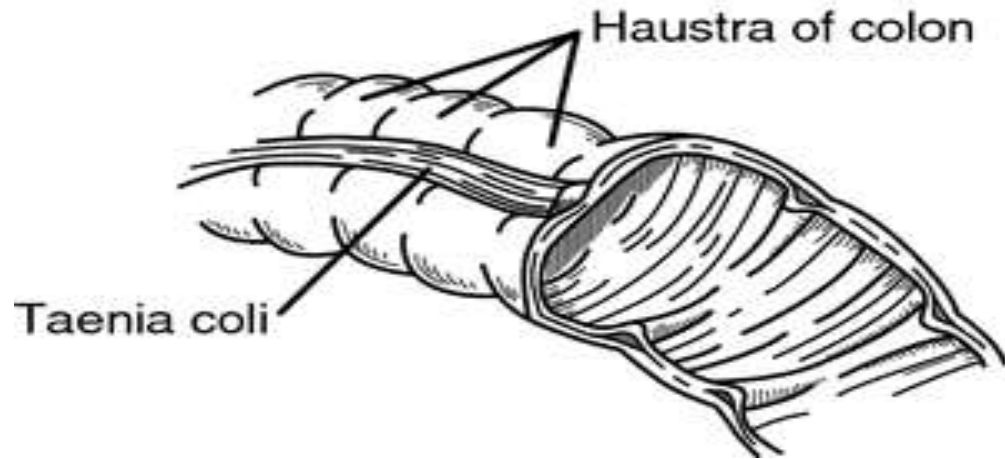


“Coffee bean” appearance

Sigmoid volvulus

How to differentiate LBO from SBO on x-ray?

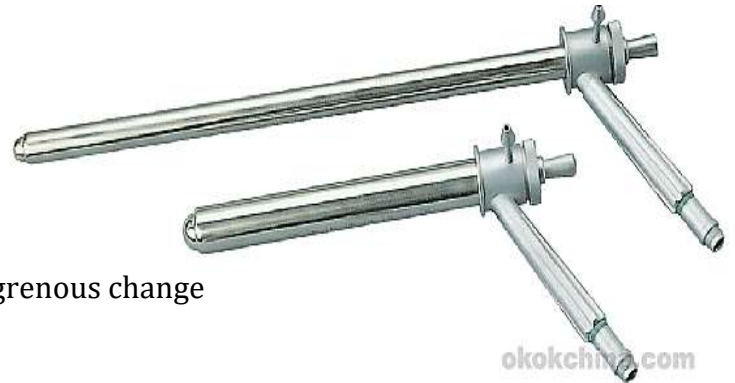
- Is the distension central or peripheral
- Which anatomic markers exist on the x-ray?
 - Haustral markings—LBO



- Valvulae conniventes--SBO
- Is the colonic air absent in the rectum or in the whole length?
 - Absent in the whole length in case of SBO
 - Rectal air absent in case of LBO

Lower GI Endoscope

- E.g., sigmoidoscopy has diagnostic & therapeutic importance



Contrast studies with enema

- Contraindicated in case of perforation or gangrenous change

CT-scan

Laboratory

- ☐ CBC
- ☐ Serum electrolyte
- ☐ RFT

Summary on acute obstruction

		SBO		LBO
		Proximal	Distal	
	Vomiting	Early & profuse	delayed	Mayn't have vomiting
	Abdominal Pain		Predominant (periumblical)	lower abdominal
	Constipation	It may take 1 or 2 days to empty the bowel distal to the obstruction. Because it was already there!!!		Early
	Distension	Minimal	Central distension	Pronounced distension
Radiograph		Little evidence of dilated loops	Multiple dilated small bowel loops	The colon proximal to the obstruction is dilated *Small bowel will be dilated only if incompetent ileo-caecal valve

Management

Management depends on the underlying cause

1. Fecal impaction → cleansing enema
2. Colorectal cancer → Discussed on next chapter
3. Cecal volvulus → Surgical approaches like cecopexy, cecostomy, and cecal resection
4. Sigmoid volvulus →

Goal

- A. To prevent the development of gangrene
- B. To address the anatomic abnormality that led to the obstruction

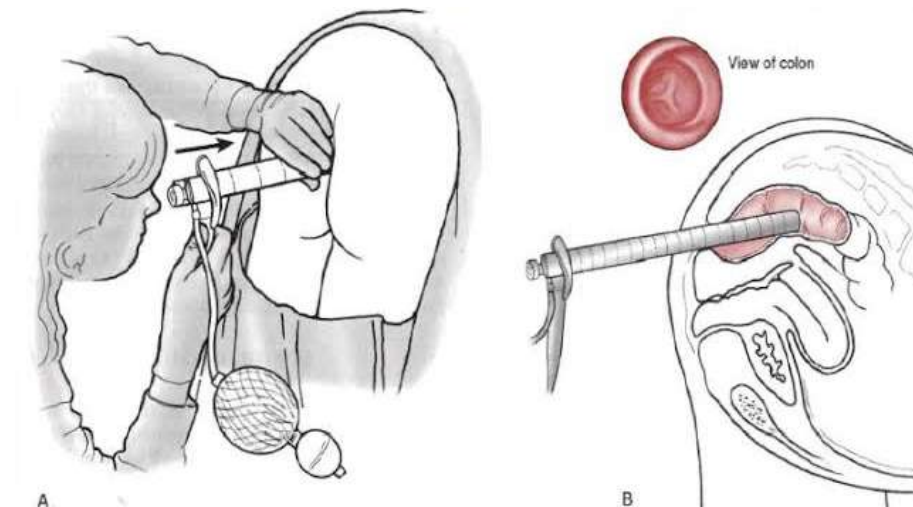
❑ Questions to be answered include;

- Is it simple obstruction (viable)?
- Is it strangulated?

NB* strangulation is a contraindication for deflation

Simple/viable sigmoid volvulus

➤ Deflation--With sigmoidoscope & a flatus tube



- The tube should be secured for 24 hours
- There is 50% risk of recurrence
- The definitive management is surgery
- Surgery should be done 01-02 weeks after the deflation. Why the gap 1-2 weeks?
To buy time for the inflammation & edema to subside.

- **Pre-op preparation**
 - Bowel preparation –refer under short cases of Debol
- **Definitive management→surgery**
 - Surgical options
 - ✓ Resection & anastomosis
 - ✓ Paul-mikulicz procedure
 - ✓ Sigmoid colectomy

Strangulated sigmoid volvulus

- **Secure Iv line**
 - Resuscitate
 - Antibiotics-broad spectrum
 - **Catheterize**
 - Follow urine output-adequacy of resuscitation
 - **Exploratory Laparotomy**
 - Gangrenous sigmoid resection
 - Hartman's procedure→Sigmoidectomy, Proximal colostomy, Closed distal end
- NB* intestinal strangulation is a surgical emergency. Evidences to consider strangulation;

On History ▪ Abdominal pain become steady ▪ Fever	Physical Examination ▪ General appearance Quietly lying down ▪ Vital signs ○ Febrile ○ Tachycardic ▪ Abdominal examination ○ Peritoneal signs positive (Tenderness, Rigidity)	Investigation ▪ CBC=leukoctosis
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Intussusception*

- Peak incidence between 5 & 10 months of age
- 90% idiopathic, but upper respiratory tract infection (URTI) or acute gastroenteritis (AGE) may precede the condition
- ileo-colic intussusception is common in most children (77%)

Clinical presentation

History

- bloody diarrhea (currant jelly stool)
- intermittent crying with the laps towards the abdomen

Physical examination

- Dance's sign--elongated mass in RUQ with absence of bowel in the RLQ
- DRE → blood stained mucus on examining finger (use little finger)

Investigation

Barium enema--in ileocolic intussusception → Claw sign



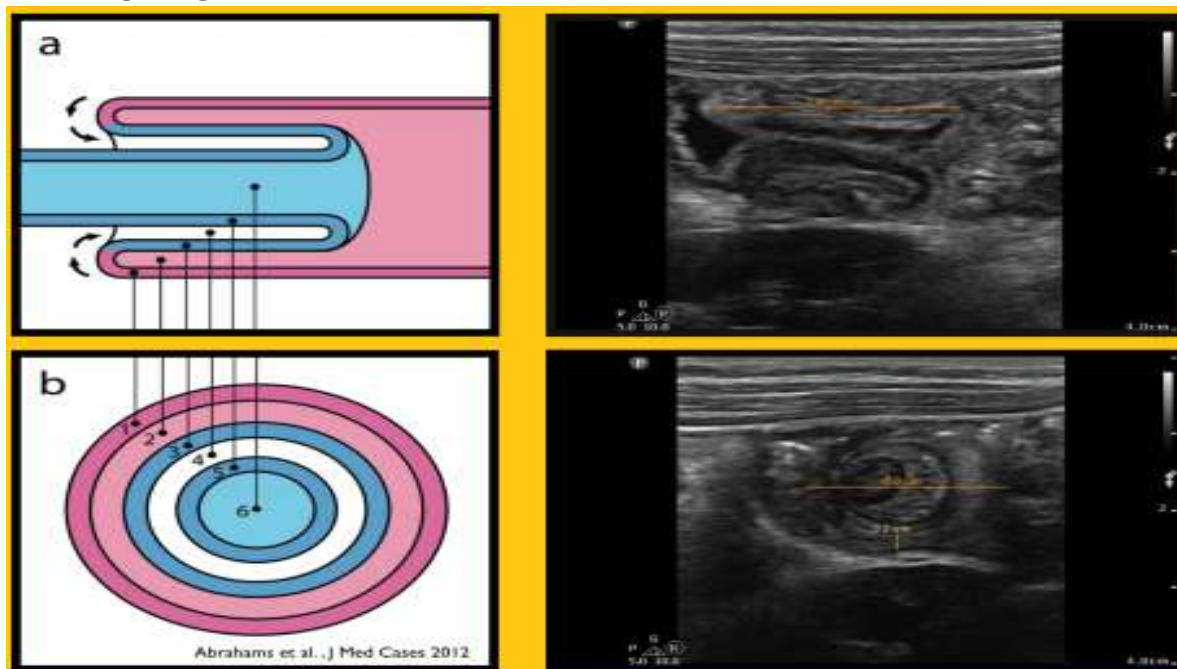
Abdominal ultrasound → high diagnostic sensitivity

1. Doughnut sign

Appearance of concentric rings in transverse section



2. Target sign



Treatment of intussusception

- Resuscitation
- IV antibiotics
- Radiographic (pneumatic or hydrostatic) or surgical reduction



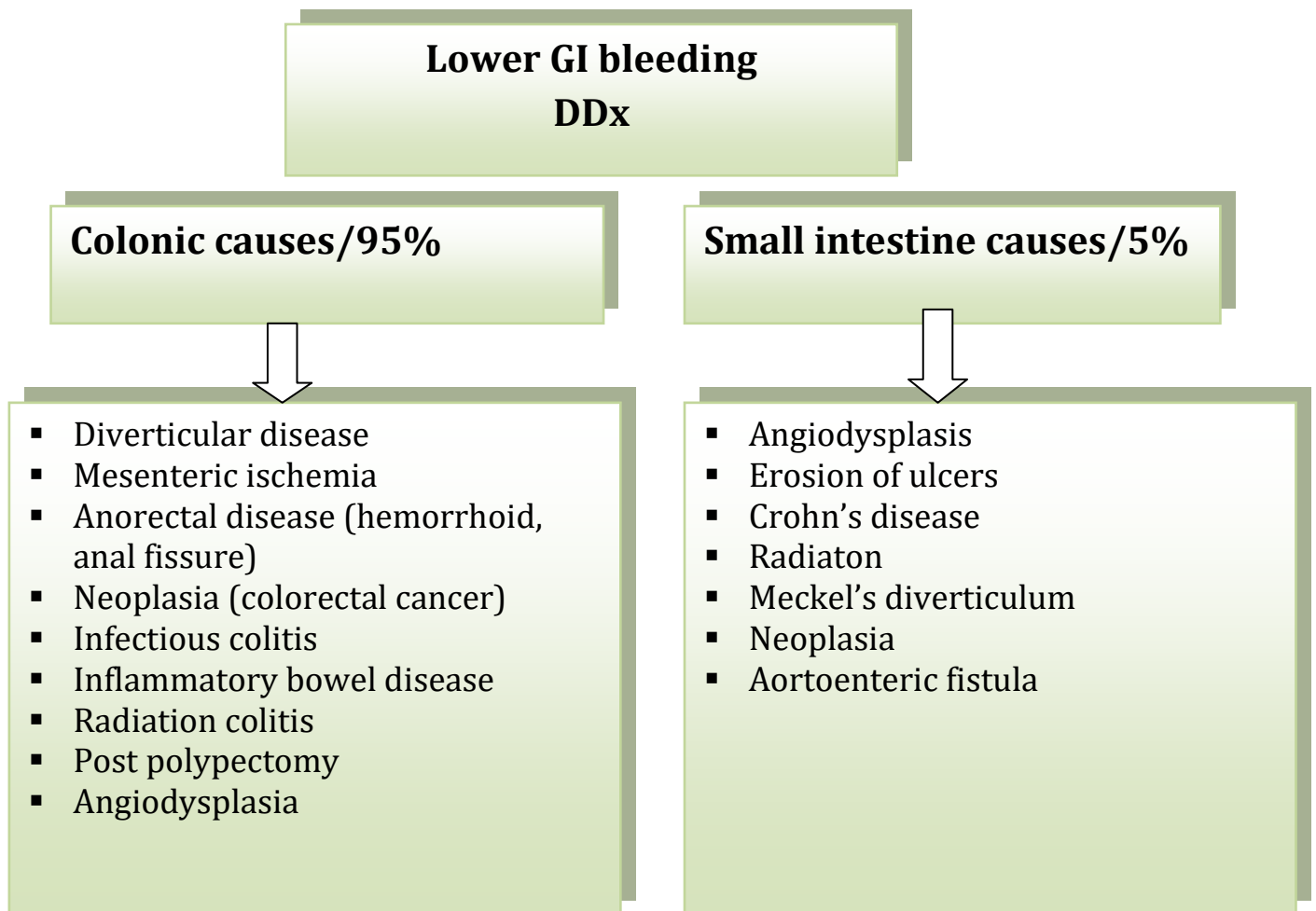
Chapter 5

Colorectal cancer

Brain storming

1. List DDx for lower GI bleeding?
2. List the advantages of colonoscopy over sigmoidoscopy?
3. Write components of bowel preparation?

Lower GI bleeding



Colorectal cancer

Risk Factors

- Aging, >50yrs → increased risk
- Genetic factors
 - Progression from premalignant to invasive cancer
 - FAP (Familial adenomatous polyposis)
 - Mutation in APC gene
 - More than 100 colonic adenomas are diagnostic
 - Rare but in known FAP patients life time risk of developing colorectal cancer is 100% by age of 50
 - Prophylactic surgery is indicated to prevent colorectal cancer
 - HNPCC (hereditary non-polyposis colon cancer) or lynch syndrome
 - Error in mismatch repair

- More common than FAP
 - In known HNPCC patients, there is 70-80% life time risk of developing colorectal cancer
 - Patients with HNPCC should be subjected to regular colonoscopic surveillance
- Familial colorectal cancer (Hereditary)
 - Accounts 10-15% of colorectal cancer
 - Risk increases with number of first degree relatives affected
- History of breast cancer/BRCA 2
- History of prostate or lung cancer in men
- Environmental & host factors
 - obesity & sedentary life style
 - “**SAD**” factors
 - **S**moking
 - **A**lcohol abuse
 - **D**ietary factors

Dietary

- High intake of red meat

Red meat components (haem & N-nitroso compounds) have shown effect in the DNA of colorectal mucosa
- High intake of animal fat

Direct toxic effect to the colonic mucosa → early malignant change
- Low fiber diet

Low fiber diet increases exposure to dietary carcinogens. Increased roughage is associated with reduced bowel transit time & reduced exposure.
- Inflammatory
 - Inflammatory bowel disease/IBD, Particularly chronic ulcerative colitis/UC → chronic inflammation predisposes the mucosa to malignant changes
- Miscellaneous
 - Previous history of surgery for colorectal cancer → Recurrence risk 20-40%
 - Pelvic irradiation
 - Ureterosigmoidostomy

Clinical presentation

Clinical presentation of colorectal cancer depends on tumor size, type & location.

History

- Rectal bleeding, overt or occult
 - ✓ For suspected occult bleeding→work up your patient with fecal occult blood test(FOBT)
- Change in bowel habit--Chronic constipation or diarrhea
- May complain of abdominal pain
- Feeling of incomplete voiding
- Symptoms of Intestinal obstruction→in left sided colonic cancer
- Tenesmus→common in rectal cancer
- Anemia symptoms (tinnitus, blurring of vision & light headedness)→Common as initial presentation in Right sided colonic cancer
- Constitutional symptoms of malignancy
 - ✓ Unexplained weight loss
 - ✓ easy fatigability
 - ✓ anorexia

Physical examination

General Appearance

- Chronically sick looking
- Nutritional status (looks malnourished)

Vital Signs

HEENT

- Signs of anemia→pale/paper white conjunctiva
- Signs of liver metastasis→icteric sclera

LGS

Chest examination

- If metastasis to the lungs, signs of pleural effusion may be appreciated

Abdominal examination

- In advanced cases there may be palpable abdominal mass, hepatomegally & signs of ascites
- DRE/digital rectal examination
 - In case of rectal cancer-- Characterize tumor size, location, surface, consistency, fixation to the underlying or overlying structure

NB*

Common sites of distal metastasis

- Liver
- Lung
- Carcinomatosis =Diffuse peritoneal metastasis

Modes of spread (colorectal ca)

- 1) Local
- 2) Lymphatic
- 3) Hematogenous
- 4) Transcoelomic

NB*

- Tumors that arise from distal rectum may metastasis initially to the lungs
- 75-80% patients present with localized disease
- Adenocarcinoma >95%
- When you write your assessment try to be specific→Colonic or rectal cancer

Discussion on selected DDx

1. Diverticular disease

- A diverticulum is a sac-like protrusion of the colonic wall
- Diverticulosis merely describes the presence of diverticula
- Diverticulitis refers to inflammation of diverticula

Diverticular disease is a term encompassing diverticulosis and diverticulitis.

Symptomatic diverticular disease includes hemorrhage, inflammation (diverticulitis), or complications of diverticulitis (such as abscess, fistula, obstruction, or free perforation).

Diverticular bleeding is thought to result from progressive injury to the artery supplying that segment. The segmental weakness of the artery will predispose for the rupture into the lumen.

2. Mesenteric ischemia

- Mesenteric ischemia can be secondary to either acute or chronic arterial or venous insufficiency.
- Predisposing factors include preexisting cardiovascular disease (AF, CHF, and acute myocardial infarction), recent abdominal vascular surgery, hypercoagulable states, medications (vasopressors and digoxin), and vasculitis.
- Acute colonic ischemia is the most common form of mesenteric ischemia. It tends to occur in the watershed areas of the splenic flexure and the rectosigmoid colon, but can be right-sided in up to 40% of patients. Patients present with abdominal pain and bloody diarrhea.

3. Anorectal disease

- The major causes of anorectal bleeding are;
 - Hemorrhoids
 - Anal fissures and
 - Rectal cancer

Hemorrhoids & anal fissure → discussed under short cases of Debol.

4. Inflammatory bowel disease

Ulcerative Colitis

- Ulcerative colitis is much more likely than Crohn's disease to present with GI bleeding.
- UC is a mucosal disease that starts distally in the rectum and progresses proximally to occasionally involve the entire colon.

- Patients can present with up to 20 bloody bowel movements per day. These episodes are accompanied by abdominal cramping, tenesmus, and occasionally abdominal pain.

Crohn's disease

- Crohn's disease typically is associated with guaiac-positive diarrhea and mucus-filled bowel movements but not with bright-red blood.
- Crohn's disease can affect the entire GI tract. It is characterized by skip lesions, transmural thickening of the bowel wall, and granuloma formation.

Sample history

Chief complaint

Bleeding per rectum of 06 months duration

HPI

This patient was last relatively healthy 06 months back at which time she started to experience dark red bleeding per rectum with associated tinnitus, blurring of vision & light headedness. In addition she has loss of appetite, easy fatigability & significant weight loss of 8% for the past 06 months (59 to 54kg). She also complains of dull aching left lower abdominal pain & mucoid, foul smelling diarrhea 4-5times/day. But she has no tenesmus, feeling of incomplete defecation, abdominal distension or failure to pass feces.

For the above complaints she visited a local health center in Dabat 02 weeks prior to admission where stool examination was done & given yellowish circular tablet to be taken four tabs per day for three days. But there was no improvement in her symptoms & she was finally referred to our hospital for better investigation & management.

- *Her father died 20yrs back @ the age of 73 by similar illness*
- *No history of breast, endometrial or ovarian cancer*
- *She has no history of previous abdominal surgery*
- *Her regular dietary habit is "injera" made of "teff" & shirowot made of "atter."*
- *She has no history of chronic alcohol consumption or cigarette smoking*
- *She has no history of radiation therapy*
- *No history of yellowish discoloration of the eyes, bone pain or hemoptysis*
- *No history of chronic cough, contact with chronic cougher or Previous TB treatment*
- *She has no self or family history of DM, Hypertension or asthma*
- *She was screened for RVI 4 months back & found to be non-reactive*

Finally she was admitted to our hospital.....

Investigation

Diagnostic

Colonoscopy

Advantages

- ✓ You can see the entire colon
- ✓ Has advantage on detecting synchronous cancer
 - Synchronous colorectal carcinoma refers to more than one primary colorectal carcinoma detected in a single patient at initial presentation. Or patient presented with colorectal cancer within 06 months after surgery was done for colorectal cancer.
- NB*Metachronous
Defined as a secondary colorectal cancer occurring more than 6 months after the index cancer
- ✓ You can take biopsy

Disadvantages

- ✓ Most invasive
- ✓ Needs bowel preparation & IV sedation
- ✓ Risk of perforation, bleeding
- ✓ Costly

Sigmoidoscopy

Advantages

- ✓ Enemal bowel preparation only but sedation isn't necessary
- ✓ Slight risk of perforation or bleeding

Disadvantages

- ✓ You will see only up to splenic flexure (60cms) & you may miss detecting synchronous polyps
- ✓ Colonoscopy is required if polyp is found

Imaging

✓ **Double contrast barium enema (DCBE)**

A double-contrast barium enema is a form of contrast radiography in which x-rays of the colon and rectum are taken using **two forms** of contrast to make the structures easier to see.

(1) Liquid containing barium (that is, a radio-contrast agent) is put into the rectum

(2) Air is also put into the rectum and colon to further enhance the x-ray

What to look on DCBE?

- Constant irregular filling defects (**'Apple core' sign**)

Advantages of DCBE

- Examines entire colon
- Good sensitivity for polyps >1cm

Disadvantages

- Require bowel preparation
- Less sensitivity for polyps <1cm
- May miss lesions in sigmoid colon
- Colonoscopy required if positive result

✓ **Abdomino-pelvic CT-Scan--**For diagnosis & staging

Laboratory

- **FOBT(fecal occult blood test)**—in suspected occult bleeding
- **CBC**
- **OFT**
- **Tumor markers**
 - ✓ Serum CEA level → More sensitive indicator of recurrence (important for post-op follow-up), but no role in screening or diagnosis

Metastatic workup

- **Abdominal Ultrasound**
- **Chest x-ray**
- **Abdominal/ pelvic/chest CT-Scan**
- **Abdominal/ pelvic MRI**

Management

Pre-op preparation

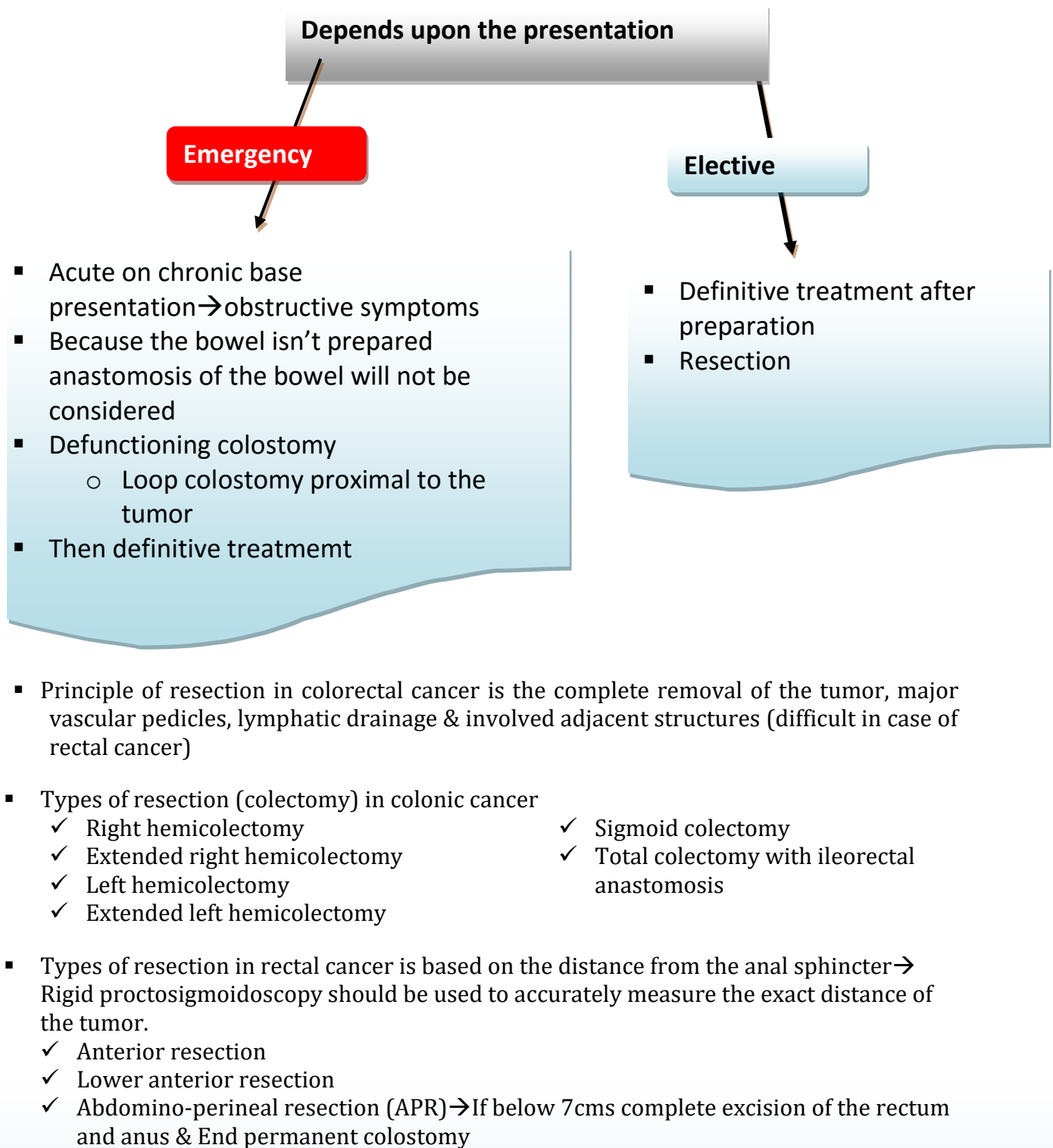
- ✓ Correct Anemia-- Hematocrit should be >30 (Pre-op). If emergency surgery is needed, consider blood transfusion
- ✓ Bowel preparation -- For elective patients. What are the steps to prepare bowel?

Principles of colorectal cancer management

- ✓ Surgery
- ✓ Radiation therapy
- ✓ Chemotherapy
- ✓ Combination of the above



Surgical treatment of colorectal cancer



Post-op complications

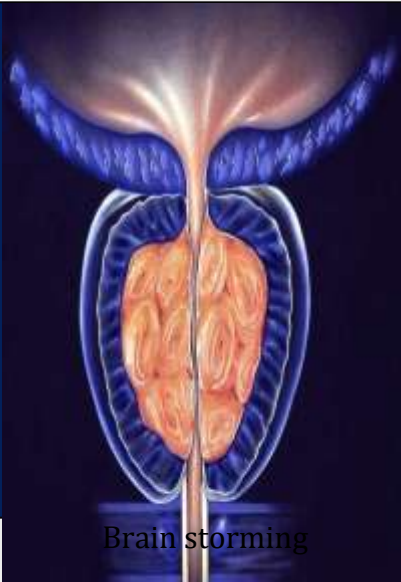
- Bleeding
- Anastomosis leak
- Colostomy related complications
- Intra-op injury to the adjacent structures
- DVT & embolism
- Wound related complications
- Anesthesia related complications
- Recurrence

Post-op follow-up

- Nutritional rehabilitation
- Serum CEA level
- Metastatic work up
- Psychological support

Chapter 6

Bladder Outlet obstruction BOO



Brainstorming

1. List causes of BOO?
2. Explain the possible digital rectal examination results of prostatic cancer & BPH?

Bladder outlet obstruction 2^o to? _____

- Benign prostatic hypertrophy/ BPH
- Prostatic cancer
- Bladder cancer
- Urethral stricture
- Bladder neck contracture
- Bladder stone
- Neurogenic bladder...



Benign prostatic hypertrophy

- Benign Prostatic Hypertrophy is the hyperplasia of the prostate gland on peri-urethral & transitional zone
- Common cause of BOO in elderly

Clinical presentation

History

- Lower Urinary Tract Symptoms/LUTS → The voiding dysfunction that results from prostatic enlargement & Bladder Outflow Obstruction (BOO)
- LUTs
 1. Voiding/obstructive symptoms
 2. Storage/irritative symptoms

Obstructive /voiding symptoms

- Hesitancy
 - Difficulty to initiate urine
- Poor flow
 - Is it improved by straining or not?
- Intermittent stream
 - Stops & starts
- Dribbling
- Sensation of poor bladder emptying



Irritative /storage symptoms

- Frequency (put it in day to night ratio)
- Urgency
- Nocturia
- Urge incontinence



Not all men with BPH have LUTs & the vice versa

Things to consider on history after LUTs

- Severity of symptoms & how they are affecting the patients' quality of life
- Precipitating factors
 - Postponement of micturation
Common after heavy drinking of alcohol in social gathering
 - Medications
 - Perianal pain
 - Urinary tract infections
- General health issues including sexual history (Erectile & ejaculatory dysfunction)
- Medication intake which can induce retention
 - E.g., antihistamines, antihypertensives, anticholinergics, tricyclic antidepressants ...
- Previously attempted treatments

Severity score

International prostate symptom score (IPSS)

- scored out of 35 & has 7 components--it tells us how severe the patients' symptoms are
- 0-7=Mild
- 8-19=Moderate
- 20-35=Severe

Physical examination

Suprapubic area—check for distended bladder

Digital rectal examination

Prostate

1. Size--if the upper border is reachable, estimate the size (finger→Centimeter)
2. surface
3. Consistency
4. Contour
5. Fixity
6. Medial sulcus

Possible digital rectal examination results

BPH

Size=Enlarged
Surface=Smooth
Consistency=Firm
Contour=well defined
Fixity=Not fixed to rectal mucosa
Medial sulcus= palpable

Urethral stricture

- Palpable beadings on urethral examination
- Normal prostate findings on DRE
- you can't advance a catheter

Prostatic ca

Size=Enlarged
Surface=May be nodular
Consistency=hard
Contour=ill defined/irregular
Fixity= fixed to rectal mucosa
Medial sulcus =obliterated

On DRE, also assess...

- Tenderness → Prostatitis
- Absence or presence of fluctuation → Prostatic abscess
- Anal sphincter tone & bulbocavernosus muscle reflex → neurological disorder

DRE reporting format

- **Inspection**
 - No ulceration or visible protruding mass
- **Palpation**
 - Normotonic anal sphincter
 - There is palpable mass anteriorly which is non-tender with smooth surface & regular border. Firm in consistency, no fixity to the rectal mucosa. It has palpable medial sulcus but the upper border isn't reachable.
 - No blood on the examining finger

Investigation

- **PSA/prostatic surface antigen**
- **Urine analysis**
- **RFT**
- **Serum electrolyte**
- **Urine culture...**

Management

1. Emergency→acute urinary retention (AUR)
 - Catheterize the patient. See Debol short cases for catheterization.
 - Arrange uro-surgical follow-up
2. Non-emergency case
 - Link to uro-surgical clinic

Uro –surgical management options

I. Watchful waiting

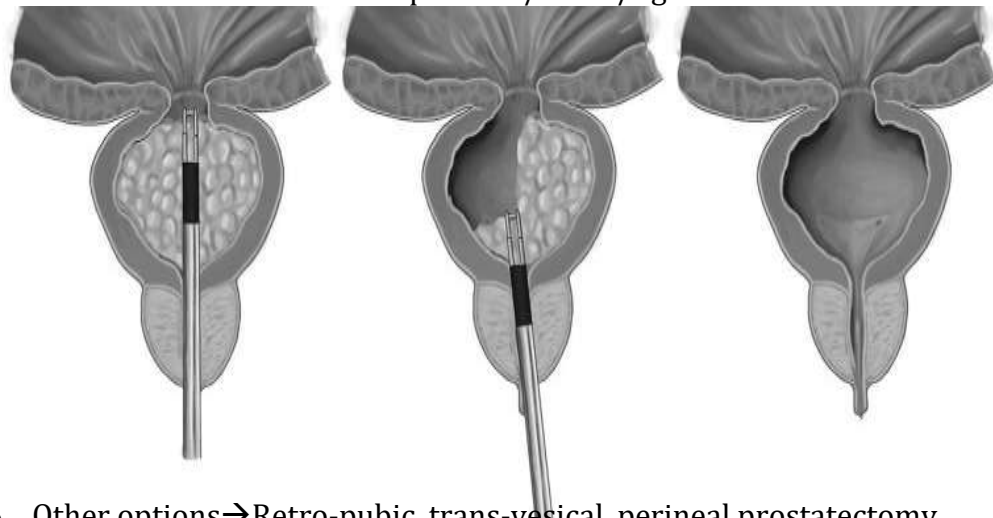
- a. For mild symptoms, follow 1 to 2 times yearly
- b. Offer suggestions that help to reduce symptoms
 - i. Avoid caffeine and alcohol
 - ii. Avoid decongestants and antihistamines

II. Medical management

- a. Alpha adnergic blockers--act on α receptors of the smooth muscle in the prostate gland and decrease its tone
- b. 5-alpha reductase inhibitors -- block the conversion of testosterone to the more potent form DHT(Dihydrotestosterone) → this will shrink the prostate gland over several months

III. Surgical management

- Trans-urethral resection of prostate/TURP/- gold standard



- Other options → Retro-pubic, trans-vesical, perineal prostatectomy
 - Pernineal— obsolete now a days
 - Retro-pubic-- commonly used in Ethiopia currently

Indications for surgery

- AUR
- Chronic urinary retention & renal impairment
 - Hydroureter or hydronephrosis
 - Residual urine ≥ 200 ml
 - Uremic manifestations
- Complications of BOO
 - E.g., infection, stone, diverticulum formation...
- Severe symptoms bothering the patient and affect his quality of life
- Failed medical treatments

Specific complications to BPH Surgery

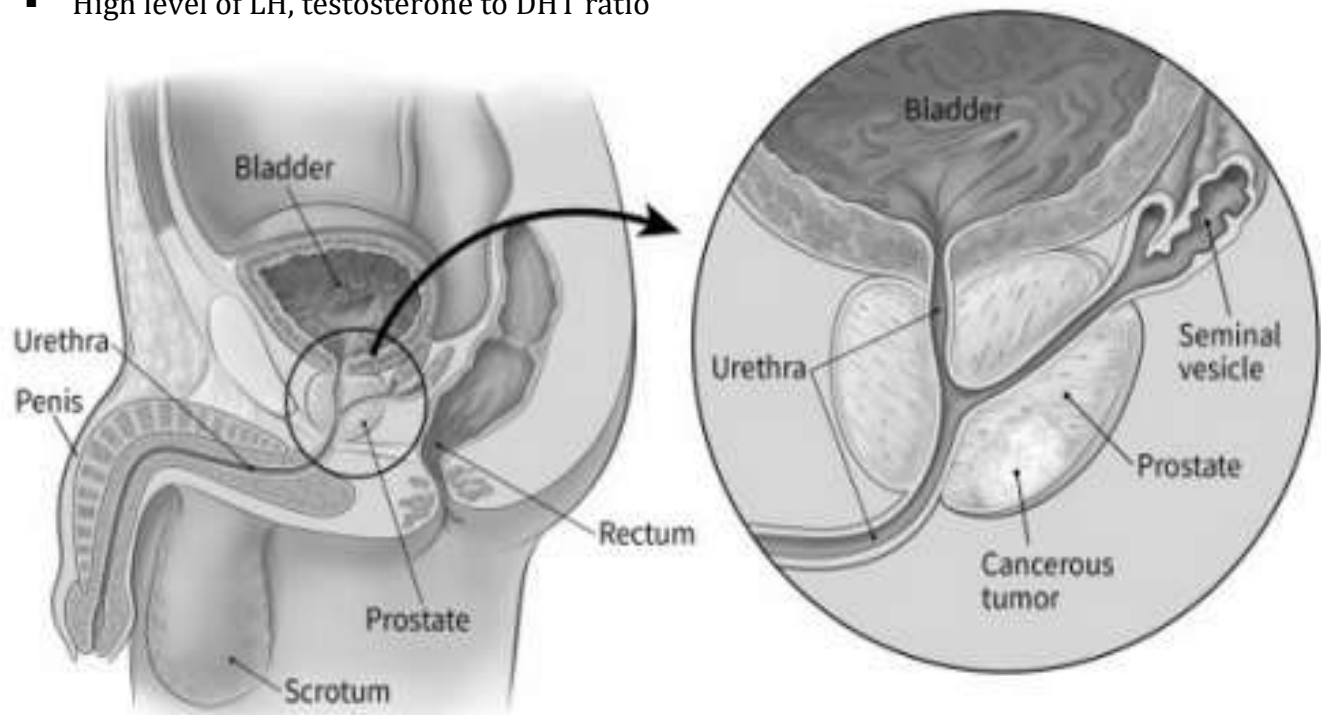
- 1) Bleeding
- 2) Retrograde ejaculation
- 3) Incontinence
- 4) Impotence
- 5) Urethral stricture
- 6) Bladder neck contracture...

Prostatic cancer

- 70% arise from peripheral zone → when compared to BPH, prostatic cancer cause LUTs later
- 95% are adenocarcinoma

Risk factors

- Genetics
- Dietary fat
- Obesity
- High level of LH, testosterone to DHT ratio



Clinical presentation

- Urinary complaints/retention
- Hematuria
- Advanced prostatic cancer
 - ✓ Weight loss, anorexia
 - ✓ Anemia
 - ✓ Bone pain with /without pathologic fracture
 - ✓ Neurologic deficit from spinal cord compression
 - ✓ Lower extremity pain & edema due to obstruction of venous & lymphatic tributaries by nodal metastasis

NB

The most common site for prostate cancer to spread is to the pelvic lymph nodes and bone

Investigation

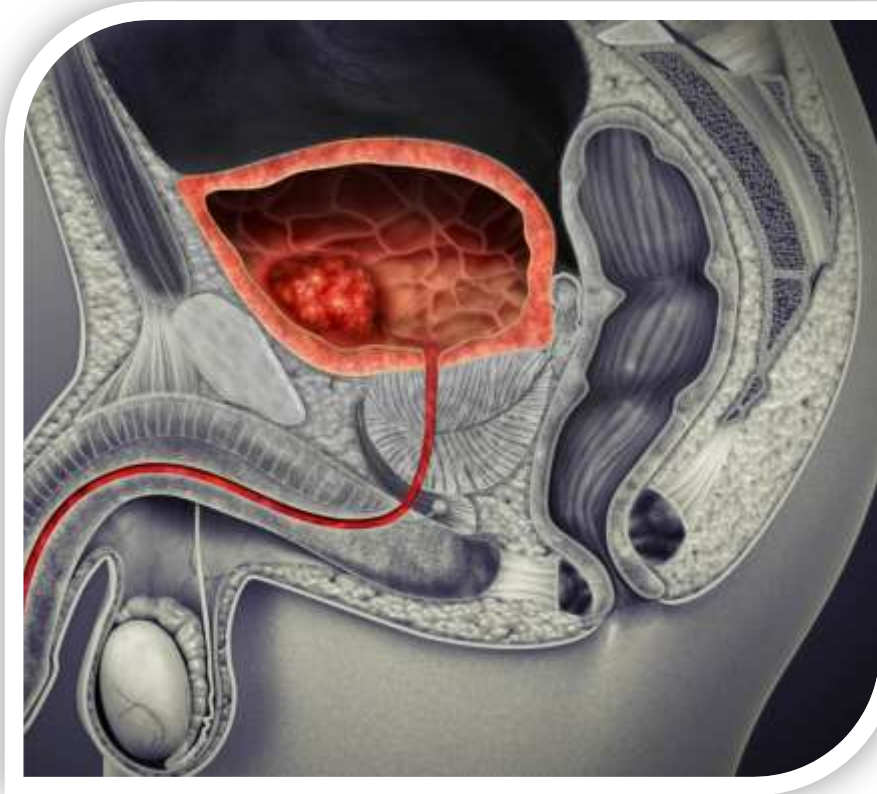
- PSA
 - ✓ Screening for prostate cancer → 4ng/ml
 - ❑ PSA velocity
 - ❑ PSA density [percentage of free PSA]--The lower the percentage of free PSA the higher the likelihood of cancer
- Biopsy (prostatic)
 - ✓ Done when PSA level is elevated or abnormal DRE findings
- Ultrasound
 - ✓ Prostatic cancer may have the following features
 - Enlarged prostate
 - Nodular surface
 - calcifications
 - Capsular invasion
- CT/MRI/Bone scan → For staging

Management

- Localized
 - Surgery
 - Radiation
 - ADT/androgen deprivation therapy
 - Active surveillance
- Metastatic
 - Palliative management
 - Attempt to slow the progress

Bladder cancer

Transitional/urothelial cell cancer covers 90% of bladder cancer



Risk factors

- Environmental factors/80%
 - ✓ Cigarette smoke
 - ✓ Occupational smoke exposures
- Medical risk factors
 - ✓ Radiation therapy to the pelvis
 - ✓ Chemotherapy with cyclophosphamide
 - ✓ Prolonged indwelling catheter
- Schistosoma species → *S. hematobium*

Clinical presentation

- Around 20% -- irritative lower urinary tract symptoms/ LUTs
- Around 80% -- painless gross hematuria

NB*

Source of hematuria--based on the timing during micturition

- ✓ At the beginning of urine stream → urethra
- ✓ At the end → bladder neck + prostate
- ✓ Throughout → high up in the kidneys & ureter

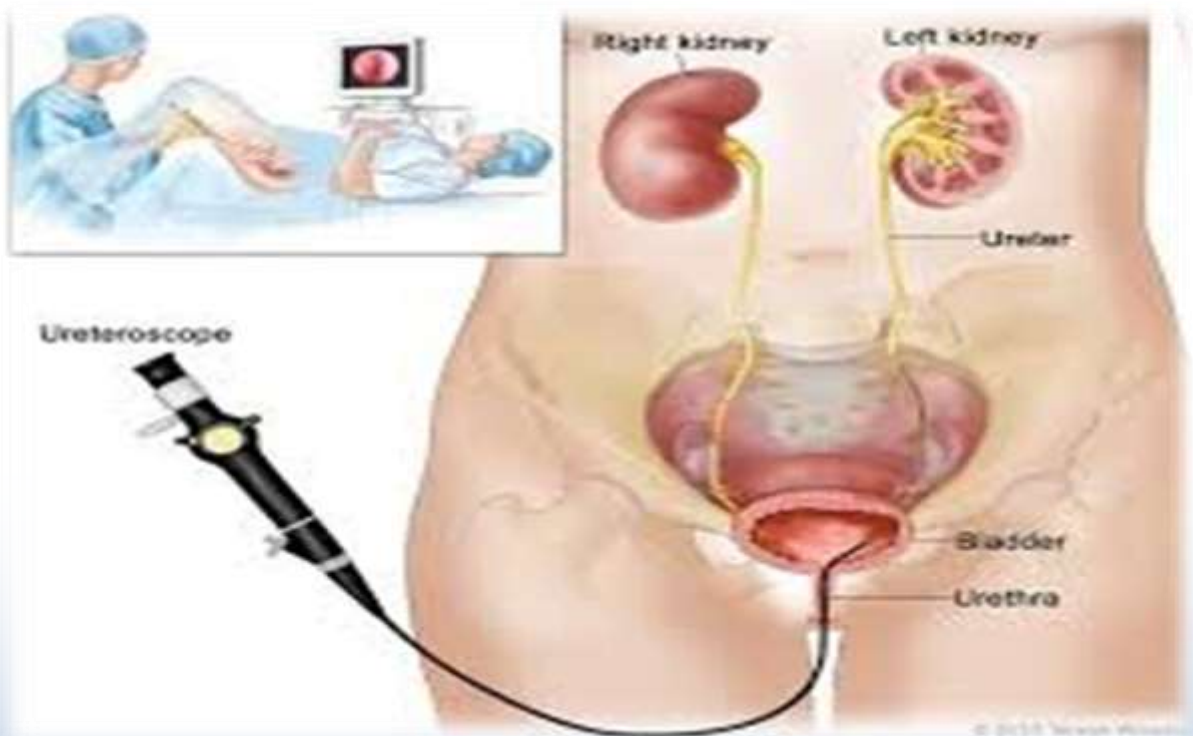
- Metastatic site signs & symptoms in case of advanced bladder cancer
 - ✓ Pelvic/ bone pain
 - ✓ Lower extremity edema --Due to compression of iliac vessels
 - ✓ Flank pain--Due to ureteral obstruction

Investigation

- **Abdomino-pelvic ultrasound**



- **Cystoscopy**



- Urine cytology
- Urine analysis
- Urine culture
- LFT

Management

Non muscle invasive

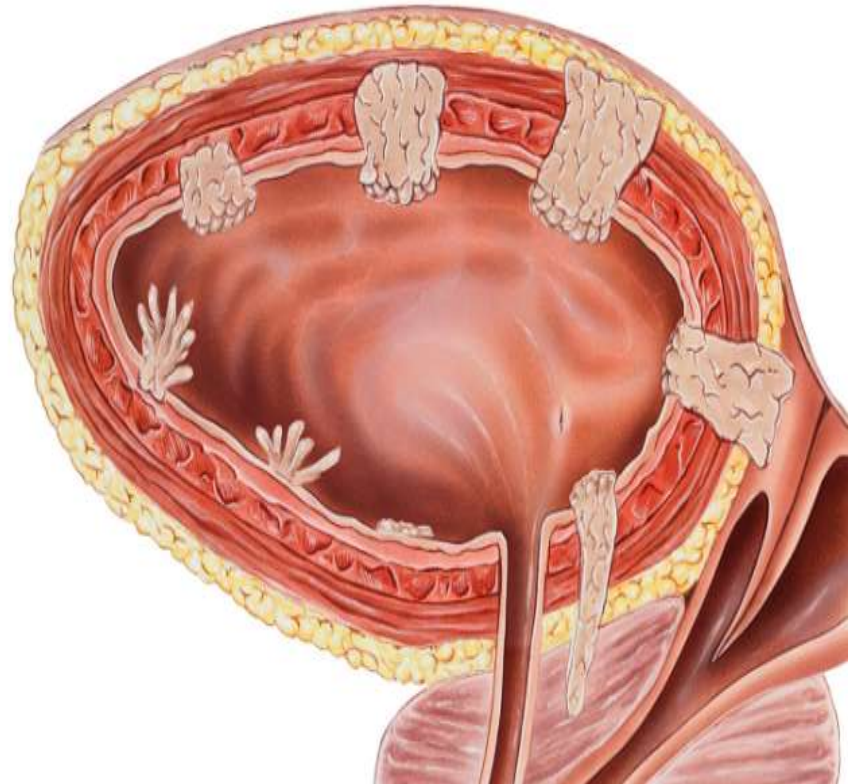
- Immunotherapy & chemotherapy
Intravesical BCG
- TURBT
Electrocautery
- Radical cystectomy
High risk

Muscle invasive

- Radical cystectomy
- Pelvic lymphadenectomy
- Urinary diversion
- Neoadjuvant chemotherapy

Metastatic

- Chemotherapy
- Radiation therapy



Urethral stricture

Causes

- Infectious
 - Gonococcal infection
History of penile discharge, dysuria, repeated treatments for sexually transmitted infections/STI
 - TB
Prostate is the commonest site—multiple stricture
- Traumatic
 - Instrumentation
E.g., Repeated transurethral catheterization
 - Accidents
 - Post-operative

Clinical presentation

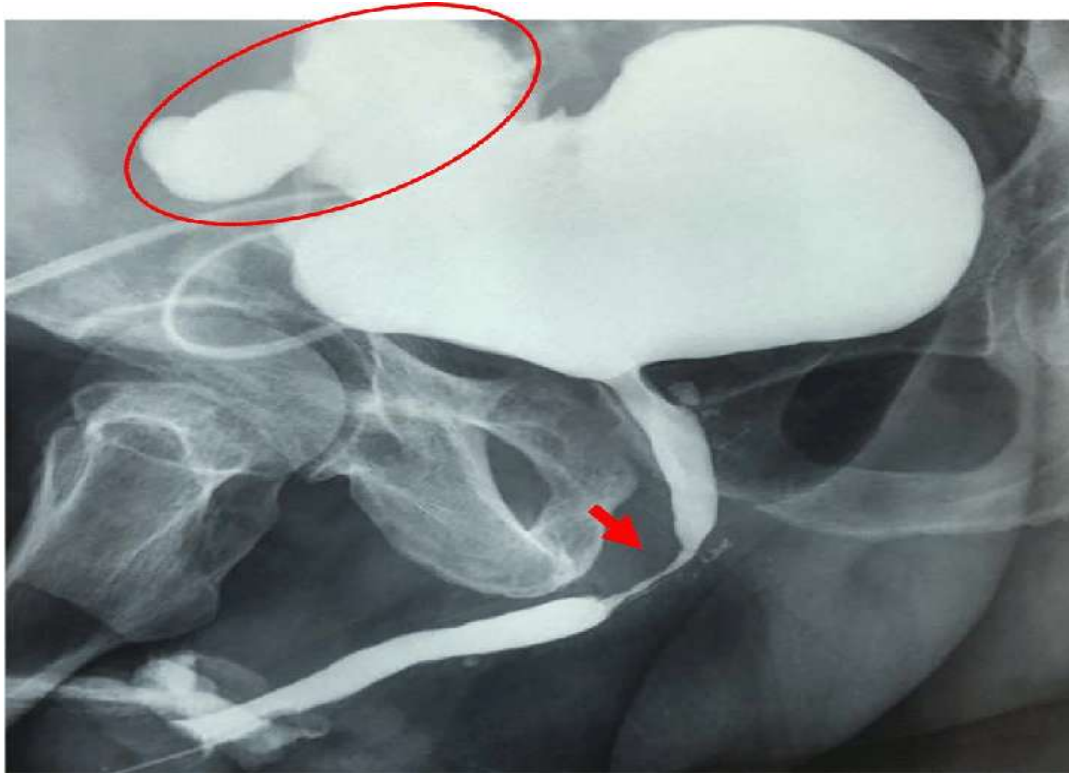
- The voiding symptoms of urethral stricture are very similar to BPH
- Suspect the diagnosis in a young man with poor urinary stream

Investigation

- Urethroscopy



- Urethrography



Urethral stricture with bladder diverticula

Management

- Urethral dilation
- Endoscopic (internal) urethrotomy
- Urethroplasty

NB

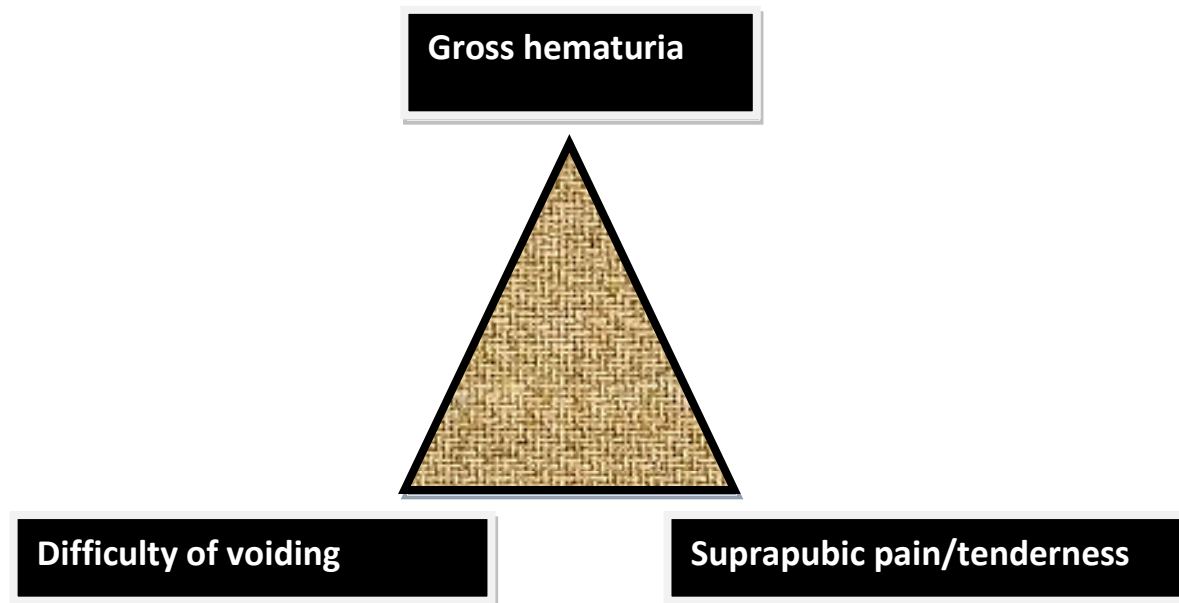
In case of AUR 2^o urethral stricture → suprapubic cystostomy

Bladder, prostatic, urethral stone

- Common in male children
- Is the difficulty of urination position dependent or not? Position dependent in bladder stone
- Pain on the tip of the penis & history of stone passage suggests → urethral

Bladder trauma

Triads



Complications of BOO (Generally)

- Urinary retention
 - Acute Urinary Retention (AUR)
 - Chronic urinary retention--The vesiculoureteral junction incompetence may lead to bilateral hydronephrosis...
- Renal insufficiency
- Recurrent UTI
- Gross hematuria
- Bladder calculi
- Diverticulum formation
- Renal Failure-rare

Sample history

Chief complaint

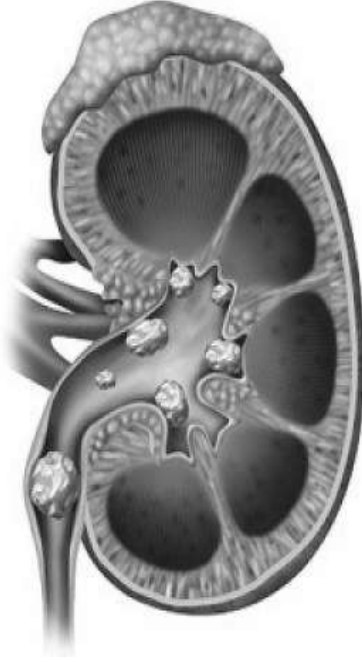
Difficulty of urination of 06 months duration

HPI

This patient was last relatively healthy 06 months back at which time he started to experience difficulty of urination which made him strain to initiate & maintain his urination. Associated with this he started to experience urgency & frequency of urination with day to night ratio of 10:6, which make his sleeping time difficult. He has also post-micturation dribbling & a feeling of incomplete voiding. 1 month back he was totally unable to pass urine after drinking about 2 Liters of local “tella” on a social gathering for which he visited our hospital where he was catheterized. The catheterization was successful.

- *No history of penile discharge, genital ulcer or multiple sexual partner*
- *No history of chronic cough, contact with a chronic cougher or previous TB treatment*
- *No history of trauma to the pelvis*
- *No history of anorexia, easily fatigability or significant weight loss*
- *No history of reddish discoloration of urine, cigarette smoking, radiation therapy to the pelvis or river water contact*
- *No history of medication intake like anti-histamines & anti-cholinergics*
- *No history of position dependent sudden cessation of urine*
- *No history of fever, chills or rigor*
- *No history of tinnitus, blurring of vision or light headedness*
- *No history of bone pain, hemoptysis or yellowish discoloration of the eyes*
- *No self /family history of DM, hypertension or Asthma*
- *Screened for RVI & found to be non-reactive 06 months back.*

Finally he was admitted to our hospital.....



Chapter 7

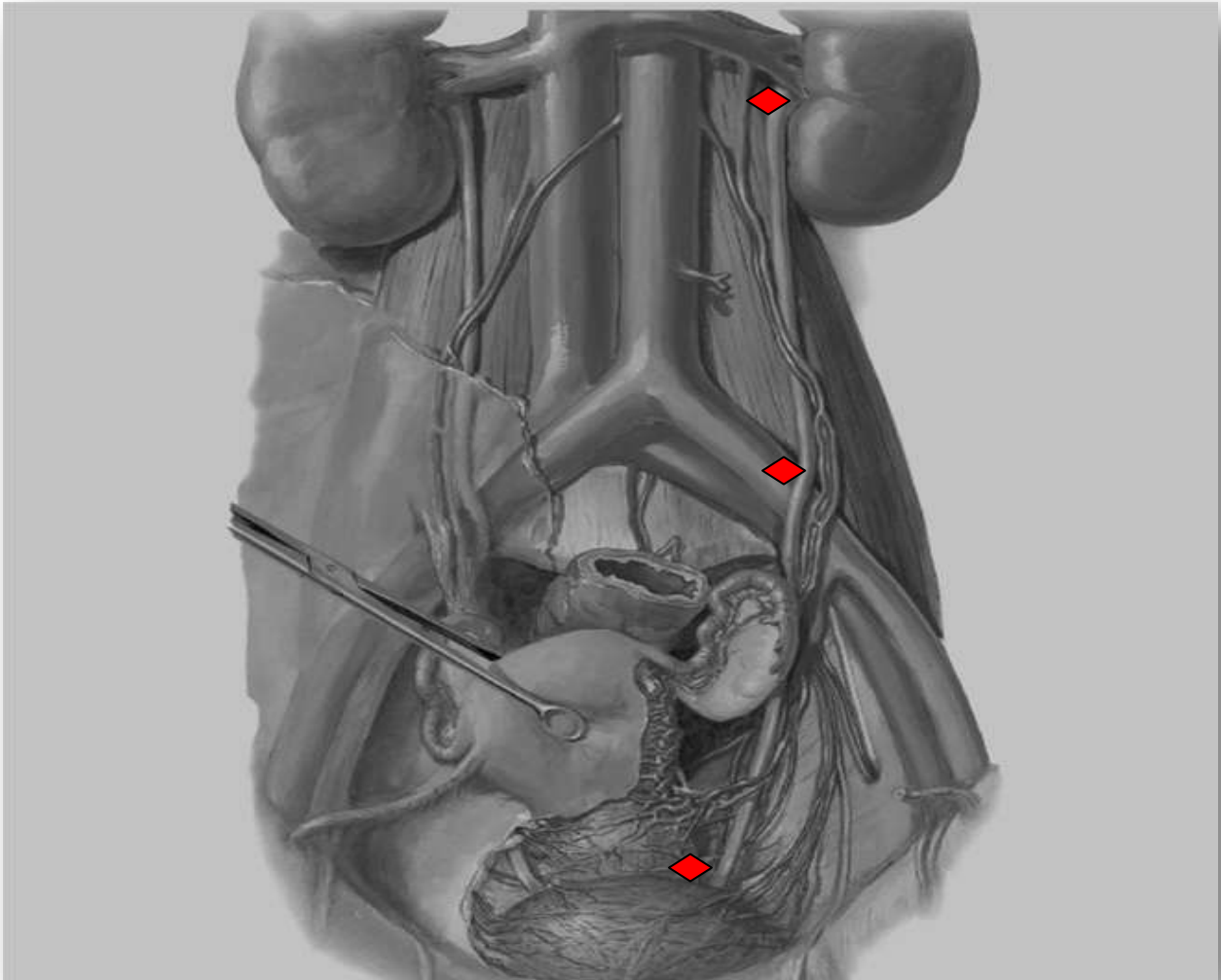
Nephrolithiasis

Brain storming

1. List the chemical type of kidney stones?
2. How you would like to work up a case suspected to have nephrolithiasis?
3. What are the conservative managements of kidney stone?



- Nephrolithiasis refers to calculi in the kidney. In the ureter it is called ureterolithiasis, almost always it originate from the kidney.
- Common sites of obstruction
 - ✓ Uretro-pelvic junction
 - ✓ Over the iliac artery/pelvic brim
 - ✓ Uretero-vesical junction



- 80-85% of stones pass spontaneously
- Recurrence rate after 5 years is 50%

Chemical types

- Calcium stones cover around 75% of renal stones. They primarily contain calcium oxalate & less often calcium phosphate.
- Struvite [Magnesium ammonium phosphate] stones -15%



- Uric acid stones – 6%
- Cystine stones – 2%



Risk factors

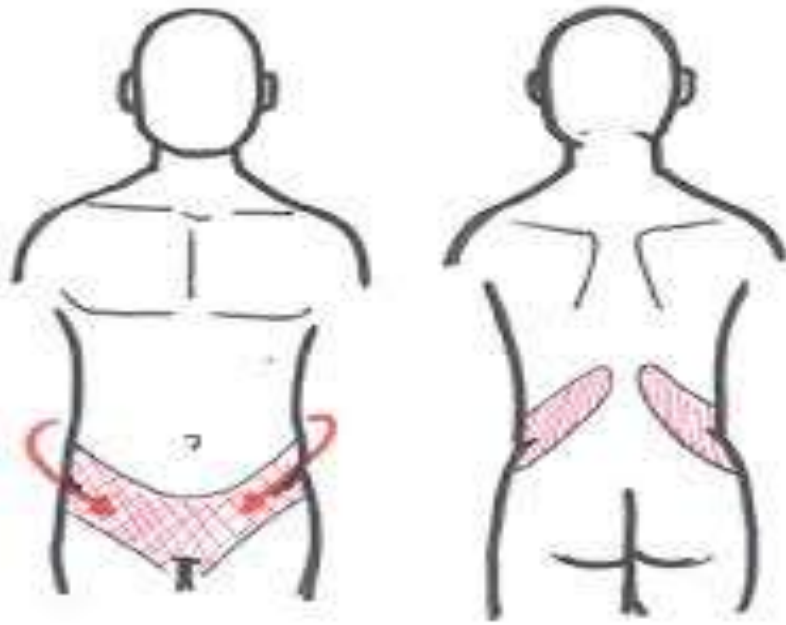
- Dietary
 - ✓ Low fluid intake
Taking low fluid will predispose to increased concentration of urinary solutes which result in stone formation.
 - ✓ Protein rich diets/meat ,fish eggs
This diets lead to hyperuricemia.
 - ✓ Vitamin A deficiency
Vit.A deficiency will predispose to desquamation of renal epithelium, this in turn acts as a nidus for stone formation.
- Environmental factors
 - ✓ Living in hot climate area
- Anatomical abnormalities which result in inadequate drainage
 - ✓ Horse shoe kidney, un-ascended kidney
- Medical conditions
 - ✓ Urinary tract infections (e.g., due to prolonged catheterization)
 - ✓ Hyperparathyroidism
Hyperparathyroid patients literally pass their skeleton in their urine. Resulting a great increase in elimination of calcium through kidney, this in turn predisposes to stone formation.
 - ✓ Gout → uric acid stones
 - ✓ Crohn disease → hyper oxaluria & magnesium malabsorption
 - ✓ Hypertension
 - ✓ Obesity
 - ✓ Immobilization
 - ✓ Some medications...
 - Acyclovir,sulfadiazine
 - Loop diuretics → increase calcium renal excretion
 - Gluco-corticosteroids → increase bone resorption
 - ✓ Previous nephrolithiasis –recurrence
- Surgery related
 - ✓ Gastric bypass procedures, bariatric surgery, short bowel syndrome → predispose to enhanced enteric oxalate absorption, this in turn increases risk of stone formation
- Family history of nephrolithiasis

Clinical Presentation



May be clinically silent/asymptomatic

- Symptomatic stones are commonly associated with pain in the flank area
- Symptoms are usually produced when stones pass from the renal pelvis into the ureter
- Pain typically waxes and wanes in severity, and develops in waves or paroxysms that are related to movement of the stone in the ureter and associated ureteral spasm
- Site of obstruction determine the location of the pain
 - Upper ureteral or renal pelvic obstructions lead to flank pain which is dull aching to pricking type
 - Lower ureteral obstructions lead to colicky type of flank pain with radiation from the loin to the groin area. The radiation is due to irritation of genitofemoral nerve



- Patients are usually quite agitated and have difficulty in getting a comfortable position (writhe) unlike peritonitis patients who quietly stay on their bed.
- The severity of the pain isn't associated with the size of the stone
- In majority of symptomatic patients there is microscopic or gross hematuria
- Nausea and vomiting commonly accompany ureteral colic, as a result of pressure on the renal capsule. This is due to common innervation pathway of the renal pelvis, stomach & intestine → the celiac axis and vagal nerve afferents
- At risk of recurrent UTI
The patient may present with dysuria, frequency, urgency, pyuria (U/A), fever in case of upper UTI

NB*

Urinary urgency or frequency may also be present if the obstruction is in the distal ureter

Acute renal colic – the most excruciatingly painful event a person can endure

✓ Pain > child birth, broken bones, gunshot wounds, burns, surgery...

Complications of nephrolithiasis

- Recurrent UTI
- Calculus hydronephrosis
- Pyonephrosis
- Squamous cell cancer
- Renal failure/in bilateral struvite stones
 - bilateral staghorn/struvite calculi can lead to renal failure over years because these stones typically are not symptomatic unless they result in urinary tract obstruction or infection

- **Pyelonephritis**
 - Flank pain, fever, chills, Nausea & Vomiting
 - Costo-vertebral angle tenderness/CVAT is positive
 - Leukocytosis on CBC, nitrite & leukocyte esterase positive on urine analysis
 - Manage with proper antibiotic coverage
- **Renal Cell Carcinoma (RCC)**
 - Risk factors for RCC include cigarette smoking, Obesity, hypertension, Long term dialysis...
 - The patient may present with flank pain, Hematuria, Palpable abdominal/flank mass which is firm, homogenous, non-tender & moves with respiration...
 - Management principles for RCC include surgery, radiation therapy, chemotherapy, hormonal therapy, immunotherapy or combinations of the above.
- **Perirenal abscess**
- **Bladder cancer**
- **Renal trauma**
- **Polycystic kidney disease**
 - PCKD patients may present with hematuria, hypertension, bilateral renal mass with nodular, firm to hard (sometimes cystic) findings
- **Renal TB**
 - Frequency is the earliest symptom, sterile pyuria, Hematuria...
- **In females consider gynecologic cases**
 - Ovarian torsion
 - Ectopic pregnancy...

Sample history

Chief complaint

Left flank pain of 1 day duration

HPI

This patient was last relatively healthy 1 day back at which time she started to experience sudden onset severe intermittent colicky left flank pain with radiation to the left groin area which was aggravated by going up stairs but no relieving factor noticed by the patient. The pain started while she was trying to urinate after drinking about 1.5 liters of tella on social gathering. Associated with this she experienced nausea & 4 episodes of non-bilious, non-blood tingled vomiting of ingested matter. She also has dysuria, urgency & frequency of 5:3 ratio but no fever, chills or rigor. In addition she noticed decreased amount of urine when compared to the previous times. This patient had history of deep dull aching right flank pain 01 year back for which she visited a private clinic in Gondar town where abdominal ultrasound was done and told to have small renal stones. Then she was given p.o antipain & advised to drinking plenty of water. She was also told to have check-up ultrasound every 3 month but she didn't have follow-up since then.

- ✓ Her last normal menstrual period/LNMP was 03 wks back, no abnormal vaginal bleeding noticed by the patient
- ✓ No history of foul smelling vaginal discharge
- ✓ She usually drinks up to 02 glass of water/day
- ✓ Her regular dietary habit is "shiro" made of "atter" & "enjera" made of "teff."
- ✓ No history of urethral catheterization
- ✓ No history of prolonged immobilization
- ✓ No history of abdominal surgery
- ✓ No history of medication intake except the antipain
- ✓ No self/family history of DM, Hypertension or gout
- ✓ No history of tinnitus, blurring of vision or light headedness.
- ✓ No history of chronic cough, contact with chronic cougher or previous TB treatment
- ✓ No history of anorexia, easily fatigability or significant weight loss
- ✓ No history of bone pain or hemoptysis
- ✓ she has been screened for RVI 1yr back & found to be non-reactive

Finally she was admitted.....

Investigation

Laboratory

- **Urinalysis**
 - Hematuria
 - Leukocytes , bacteria
 - Crystals of calcium oxalate , uric acid , cystine
 - PH (> 7 or < 5). Uric acid stones have $\text{PH} < 5$. $\text{PH} > 7$ may suggest urea splitting organisms like proteus, pseudomonas, klebsiella or struvite stones
- **CBC -Anemia(CKD) , leukocytosis (infection)**
- **Serum electrolytes**
- **RFT**
- **24hrs urine profile**

Imaging

Ultrasonography

- Advantage
 - No radiation
 - Sensitive for the diagnosis of urinary tract obstruction
 - Can detect radiolucent stones missed on KUB
- Drawback
 - May miss small stones & ureteral stones

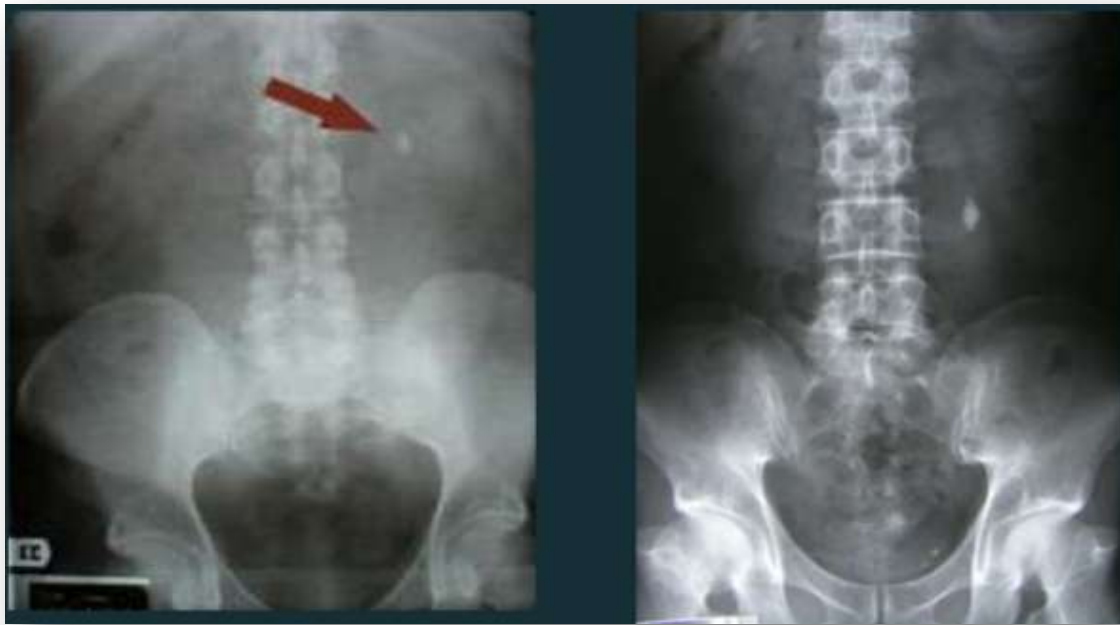




KUB/kidney- ureter- bladder/ x-ray

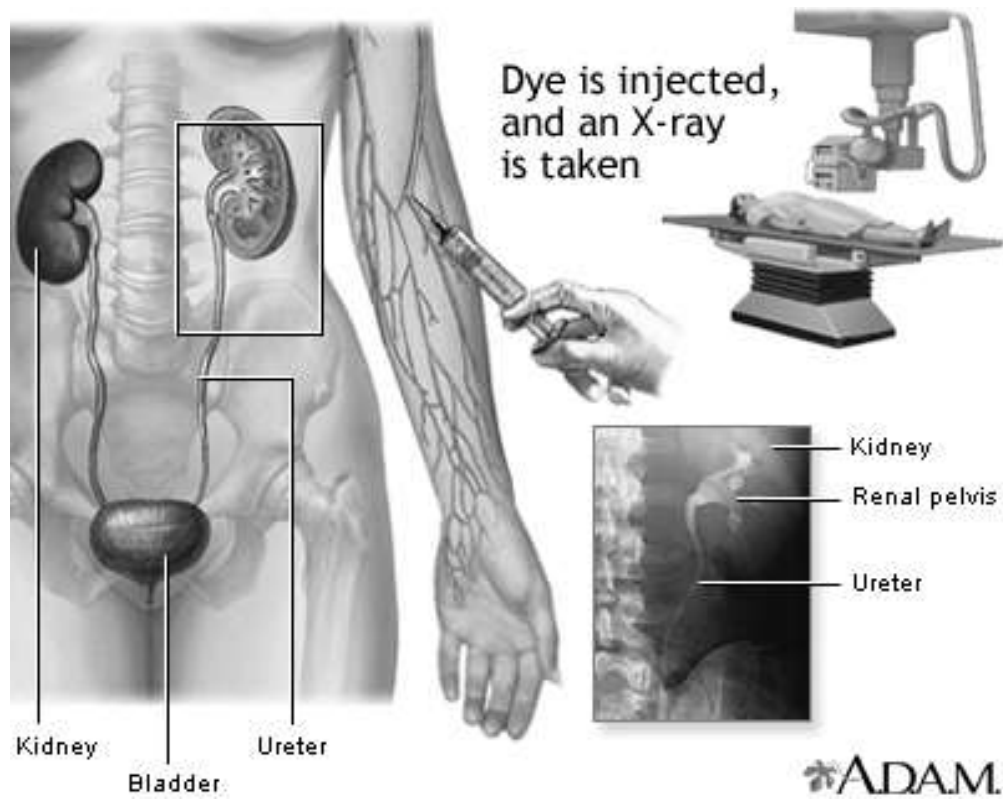


- KUB Will sufficiently identify large radio-opaque stones
- Drawback
 - It will miss radiolucent uric acid stones or stones over bony structures
 - It will not detect obstruction



IVP/Intravenous pyelography

- It tells us the presence & anatomical position of a calculus
- Also gives information on the function of the kidneys
- Hydronephrosis, Hydrouretronephrosis can also be seen
NB
Ureter >6mm – hydroureter
- Drawback
 - Potential contrast reaction
 - Higher radiation exposure



IVP

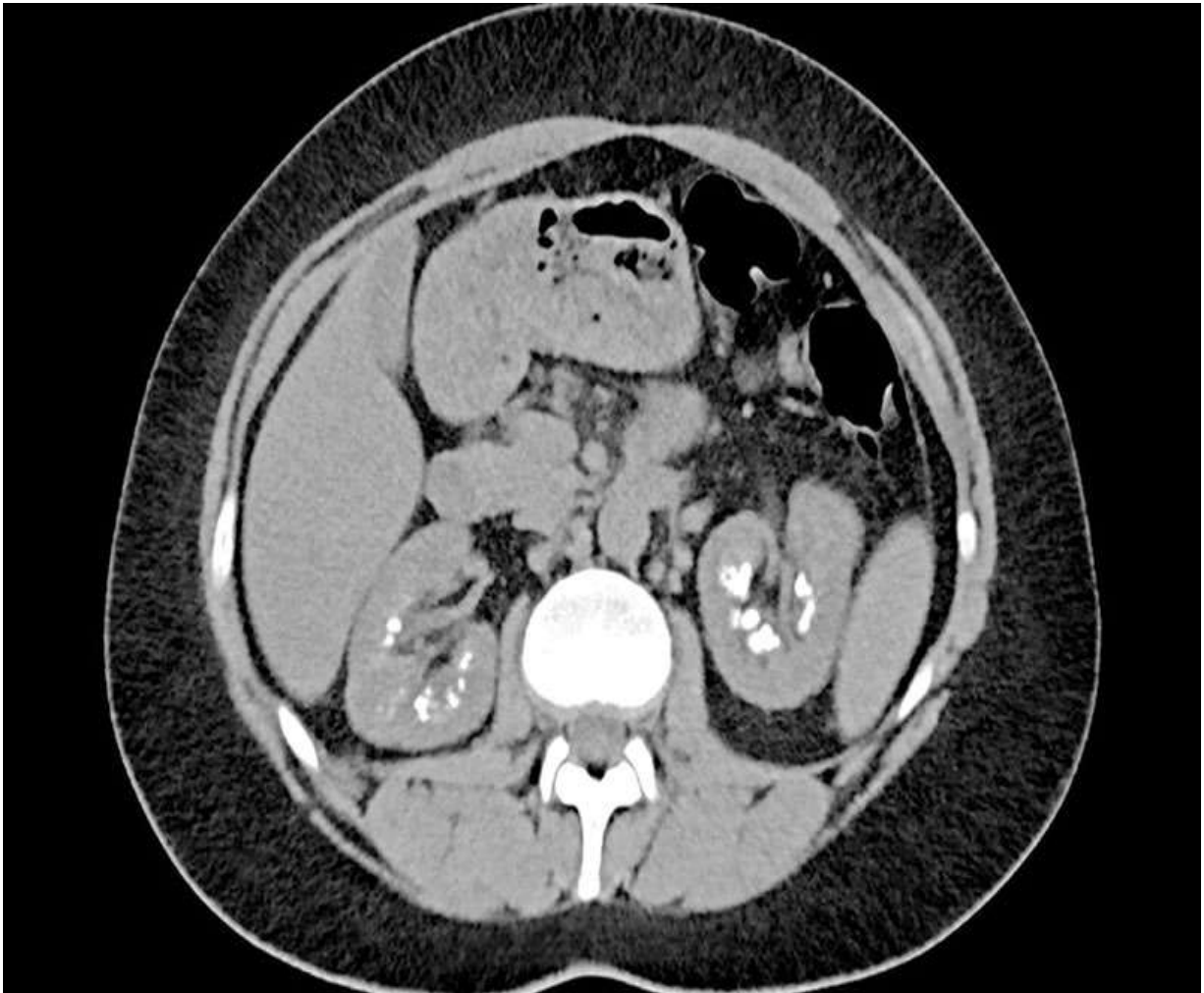
▪ **Retrograde pyelography**

- Anatomy of ureter & renal pelvis ; definitive diagnosis of calculus can be made
- Stones [including radiolucent ones] , ureteral kinks , strictures , tortuositities will be seen



Retrograde pyelography

- **CT-Scan**
95-100% sensitivity

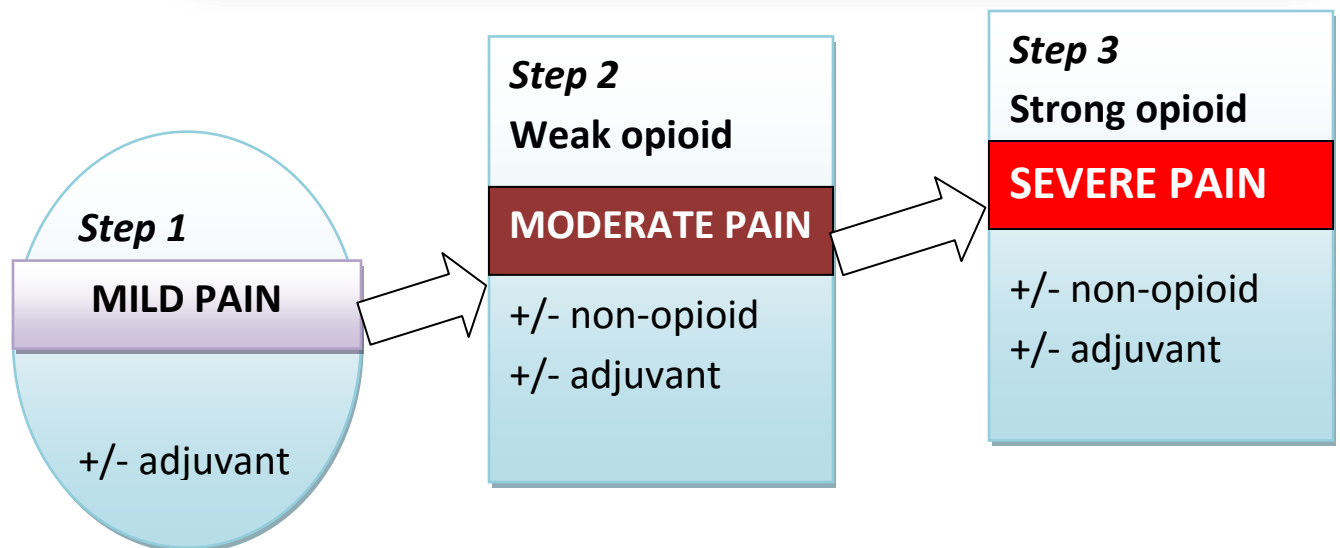
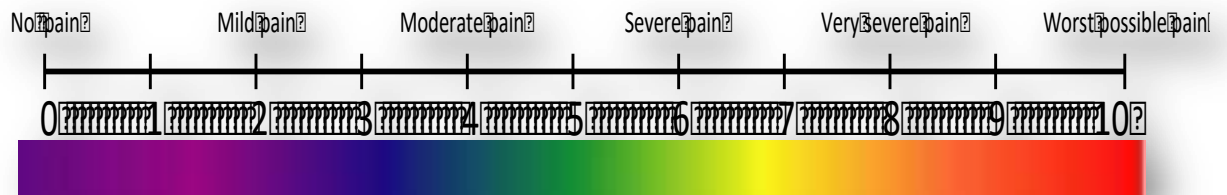


Management

Medical therapy

❖ Pain management

- Manage according to WHO pain management ladder



<i>Non-opioids</i>	ibuprofen or other NSAID, paracetamol (acetaminophen), or aspirin
<i>Weak opioids</i>	codeine, tramadol, or low-dose morphine
<i>Strong opioids</i>	morphine, fentanyl, oxycodone, hydromorphone, buprenorphine
<i>Adjuvants</i>	antidepressant, anticonvulsant, antispasmodic, muscle relaxant, bisphosphonate, or corticosteroid

Combining an opioid and non-opioid is effective, but do not combine drugs of the same class.

Time doses based on drug half-life (“dose by the clock”); do not wait for pain to recur

Consider prophylactic laxatives to avoid constipation

❖ **Dietary measures**

- Increase fluid intake/hydration
- Avoid excess salt , protein intake

❖ **alpha-adrenergic blockers**

Has ureteral smooth muscle relaxing effect

❖ **Dissolution of calculi using sodium bicarbonate or potassium citrate**

- Uric acid and cystine stones

❖ **Chemoprophylaxis**

- Limitation of dietary components
- Addition of stone formation inhibitors/magnesium & citrate/ or intestinal Calcium binders
- Allopurinol

❖ **Surgical care**

- ✓ <4mm -- can easily pass , >8mm -- unlikely to pass
- ✓ Obstructed , infected system

Surgical options

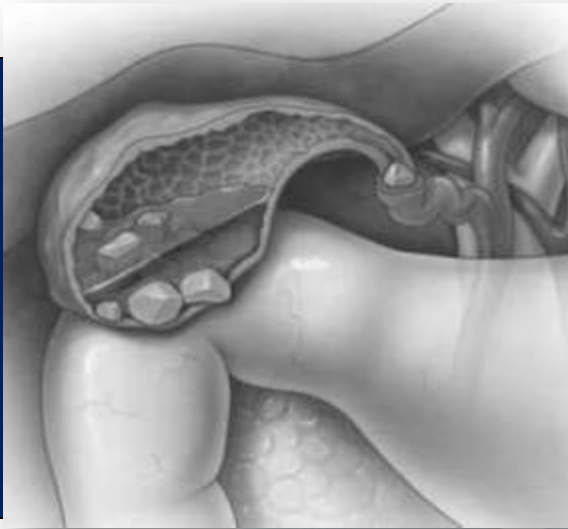
- a. Non-invasive procedures
 - PNL (percutaneous nephrolithotomy)
 - ESWL (extracorporeal shock wave lithotripsy)
- a. Open surgery
 - Pyelolithotomy--Indicated for stones in the renal pelvis
 - Nephrolithotomy

Emergency department management of renal colic

- **Secure IV line & give;**
 - ✓ Analgesics
 - ✓ Fluid
 - ✓ Antiemetic
- **Obstructed , infected system→ needs emergent surgical**

Practical tip on conservative management

- ☐ Advise them to drink plenty of water (UoP=2L/day)
- ☐ Prescribe P.O analgesics to be taken during episodes of pain
- ☐ Dietary modification
- ☐ Check-up ultrasound every 3-6 month to follow stone size

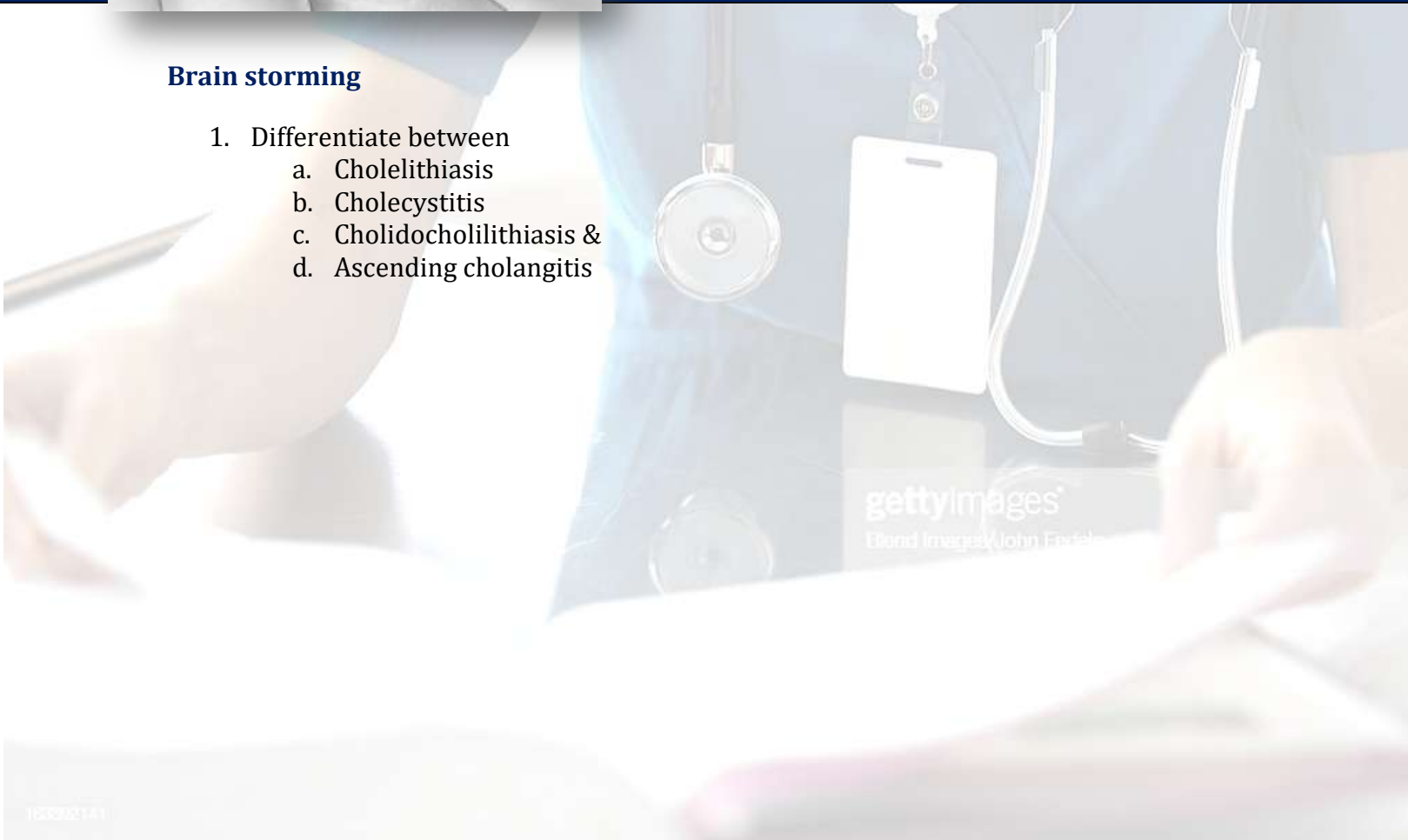


Chapter 8

Cholelithiasis

Brain storming

1. Differentiate between
 - a. Cholelithiasis
 - b. Cholecystitis
 - c. Cholidocholilithiasis &
 - d. Ascending cholangitis



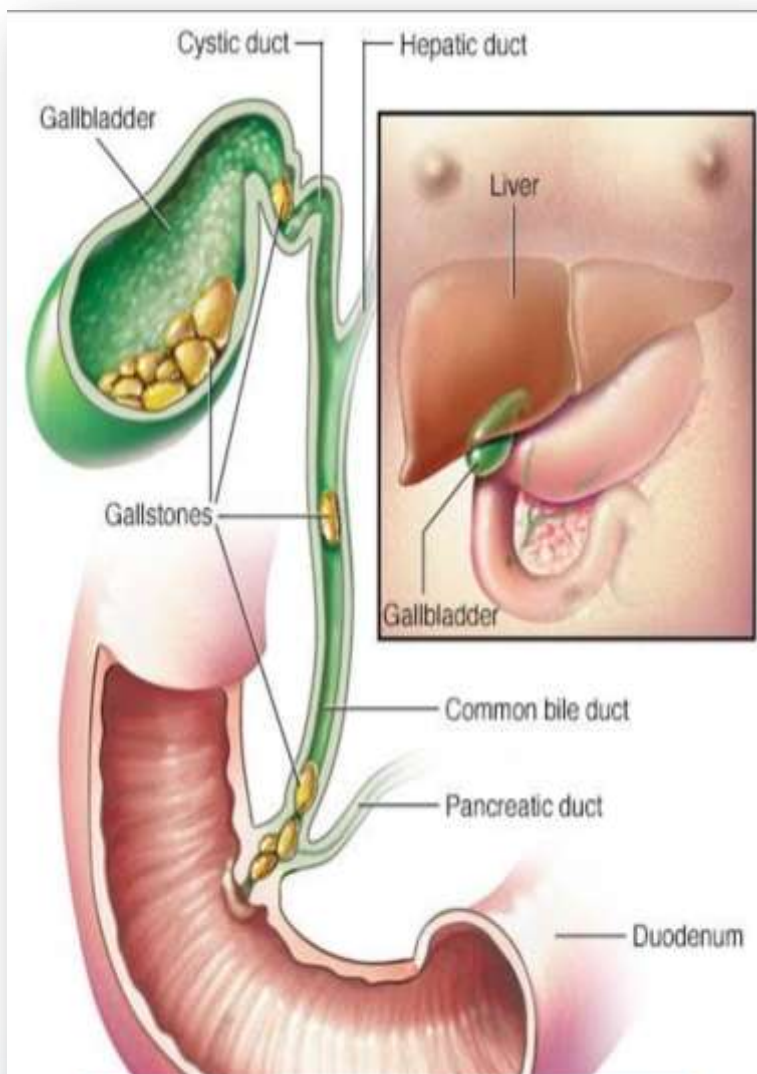
Introduction

Gallbladder

- Concentrates and stores hepatic bile during the fasting state and delivers it into the duodenum in response to a meal.
- Vagally mediated cephalic phase activity and the release of CCK regulate contraction of gallbladder

Bile

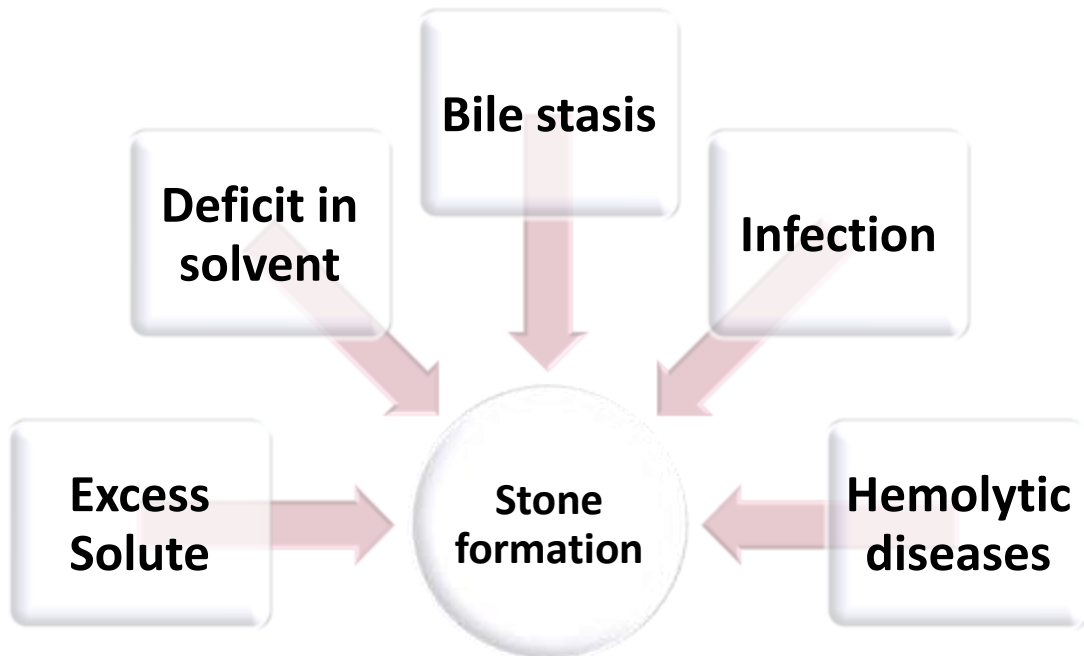
- Composed of water, electrolytes, bile salts, proteins, lipids, and bile pigments
- Aid in the digestion and absorption of fats in the intestines
- 95% will be reabsorbed through entero-hepatic circulation



Terminologies

- **Cholelithiasis**
Stone in the gall bladder
- **Choledocholithiasis**
Stone in the common bile duct
- **Cholecystitis**
Inflammation & infection of gall bladder
- **Cholangitis**
Inflammation & infection of common bile duct

Cholelithiasis



Gallstones are classified by their cholesterol content as either

1. Cholesterol or
2. Pigment stones

Cholesterol stones

- 70% to 80% gall bladder stones are cholesterol stones containing calcium salts in their center, or nidus. Pure cholesterol gallstones are uncommon (10%)
- <10% are radiopaque



Pigmented stones

A. Black

- ✓ Small & tarry
- ✓ Frequently associated with hemolytic conditions such as hereditary spherocytosis and sickle cell disease or cirrhosis
- ✓ Account for 20% to 30% of gall stone disease



B. Brown

- a. Soft & earthy
- b. Usually secondary to bacterial infection caused by bile stasis

Stages of the disease process

1. Lithogenic stage
2. Asymptomatic stage
3. Symptomatic stage
4. Complicated stage

Risk factors

- Female sex
 - The higher rates in young women is almost certainly a result of pregnancy and sex steroids
 - 03 times increased risk

- Increasing age

- Obesity

Due to enhanced cholesterol synthesis and secretion(cholesterol as excess solute)

- Terminal ileal resection

Enterohepatic circulation will be affected →95% bile salt absorption (bile salt--deficit in solvent)

- Gastric surgery

Vagotomy(e.g., for surgical management of PUD) will cause denervation & stasis of gall bladder. During stasis, water will be absorbed in the gall bladder due to the concentrating function of gallbladder which will leave bile & cholesterol. This will predispose to stone formation.

- Total parenteral nutrition/TPN—predispose to gall bladder stasis

- Pregnancy

- Qualitative change in bile acid synthesis which reduces the ability of bile to solubilize cholesterol
- Progesterone induced gallbladder stasis

- Estrogen replacement therapy

- Estrogen →cholesterol synthesis & secretion (excess solute)

- Dietary factors

- fatty food

- Cirrhosis

- reduced hepatic synthesis and transport of bile salts and nonconjugated bilirubin
- high estrogen levels
- Impaired gallbladder contraction in response to a meal...

- Crohn's disease—due to entero-hepatic circulation impairment

- Hereditary spherocytosis, sickle cell disease, and thalassemia

- Due to hemolysis

- Drugs like estrogen formulation, somatostatine analogues
 - Estrogen formulations
 - E.g. OCP, Medications for Prostatic cancer
 - Mechanism→by increasing biliary cholesterol secretion
 - Somatostatine analogues
 - Mechanism→CCK inhibition which in turn decreases gallbladder emptying
 - Colibrate (hypolipidemic drug)
 - Mechanism→by increasing hepatic elimination of cholesterol via biliary system
- Increasing age
- Family history & genetics

Clinical Presentation

Cholelithiasis

- Most patients remain asymptomatic from their gallstones
 - Some patients develop symptomatic gallstones, with biliary colic caused by a stone obstructing the cystic duct
 - Pain -- character
 - **Site**
Localized to the RUQ or epigastrium
 - **Quality**
Colicky
 - **Pattern**
Episodic
 - **Radiation**
May radiate to the right upper back or between the scapulae
 - **Aggravating & relieving factors**
Usually begins postprandial after fatty meal
 - ➔ **Associated symptoms**
 - ✓ Nausea & vomiting
 - ✓ Fever if complications occur
- List down the complications of having stone in the gallbladder?



Pain characterization

→OPQR²S²

- Onset(1)
- Pattern(3)
- Quality(4)
- Radiation(6)
- Relieving & Aggravate factors(7)
- Severity(2)
- Site(5)

E.g.... sudden onset severe intermittent colicky RUQ pain with radiation to the right upper back which was aggravated by taking fatty meals & relieved by....

Complications of cholelithiasis

1. **Cholecystitis** →Acute gangrenous cholecystitis →Gall bladder empyema
→Emphysematous cholecystitis
2. **Choledocholithiasis** with or without ascending cholangitis
Discussed in detail on the next chapter
3. **Gallstone pancreatitis**
Blockage of the pancreatic duct by an impacted stone or temporary obstruction by a stone passing through the ampulla may lead to pancreatitis
 - Pain--severe steady sharp epigastric/mid-abdominal
 - Radiation to the back
 - Relieving factor--leaning forward
 - Associated symptoms--anorexia, nausea, vomiting , diarrhea may be present
4. **Gallstone ileus**
5. **Mirizzi's syndrome**; Distended gallbladder causing common bile duct obstruction.
It is one of the causes for elevated LFT
6. **Gallbladder carcinoma** ...

Cholecystitis

i. Acute cholecystitis

Acute cholecystitis is secondary to gallstones in 90-95% of the cases (calculus cholecystitis)

Clinical presentation

History

- Abdominal pain
 - The pain characterization for acute cholecystitis is similar to that of cholelithiasis (described previously) except on the pattern. The pattern of the pain in acute cholecystitis is **constant (steady)**.
- Associated symptoms
 - **Fever**, nausea, and vomiting
 - Jaundice may be there if & only iff Mirrizi's syndrome is present

Physical examination

- RUQ tenderness and guarding are usually present inferior to the right costal margin, distinguishing the episode from simple biliary colic
- **Murphy's sign**
 - inspiratory arrest with deep palpation in the right upper quadrant
- When inflammation spreads to the peritoneum, patients develop more diffuse tenderness, guarding and rigidity.
Guarding=contraction of abdominal muscles on palpation

DDx

- PUD (Perforated/ without perforation)
- Acute pancreatitis
- Acute hepatitis
- Acute pyelonephritis
- Liver abscess
- Renal calculi
- Diverticulitis
- Pneumonia
- Appendicitis
Before it shifts to RLQ
- Myocardial ischemia/MI
Elderly patients & patients with co morbid illness like DM, Hypertenison, dyslipidemia may not present with typical MI symptoms. So it is better first to rule out life threatening conditions by doing ECG, troponin...
- Herpes zoster attack/HZA involving Intercostal nerves
- Gallbladder cancer--Pain is late manifestation in malignancy

Sample history

Chief complaint

Right upper abdominal pain of 03 days duration

HPI

This patient was last relatively healthy 03 days back at which time she started to experience sudden onset severe intermittent colicky right upper quadrant abdominal pain with radiation to the right upper back which was aggravated by taking fatty meal but no relieving factor noticed by the patient. Associated with this she has also nausea & bilious, non-projectile, non-blood tingled, non-foul smelling vomiting about 4-5times/day immediately after taking a meal. In addition to this she started to experience low grade intermittent fever but no chills or rigor. For the above complaint she visited local health center at Dabat town, 01 day prior to admission, where she was given #01 bag of IV fluid & referred to our hospital for better investigation & management.

- ✓ *She was on oral contraceptive pill for the last 3 years but no history of other medication intake*
- ✓ *She is a grand multipara mother*
- ✓ *She has no history of abdominal surgery*
- ✓ *She has no history of similar illness in the family*
- ✓ *She has history of burning epigastric pain for the past 02 years but no history of NSAID use or chronic alcohol consumption*
- ✓ *She has no history of yellowish discoloration of eye, itching sensation or color change in the urine & stool*
- ✓ *No history of multiple sexual partner, contact with jaundiced person or blood transfusion*
- ✓ *She has loss of appetite but no significant wt loss or easily fatigability*
- ✓ *She has no history of chronic cough but has history of contact with known TB patient who was on Anti-TB medication after diagnosed by sputum & CXR examination.*
- ✓ *She has no self or family history of DM, hypertension or Asthma.*
- ✓ *She was screened for RVI 9 month back and found to be NR.*

Finally she was admitted to our hospital by.....

Investigation

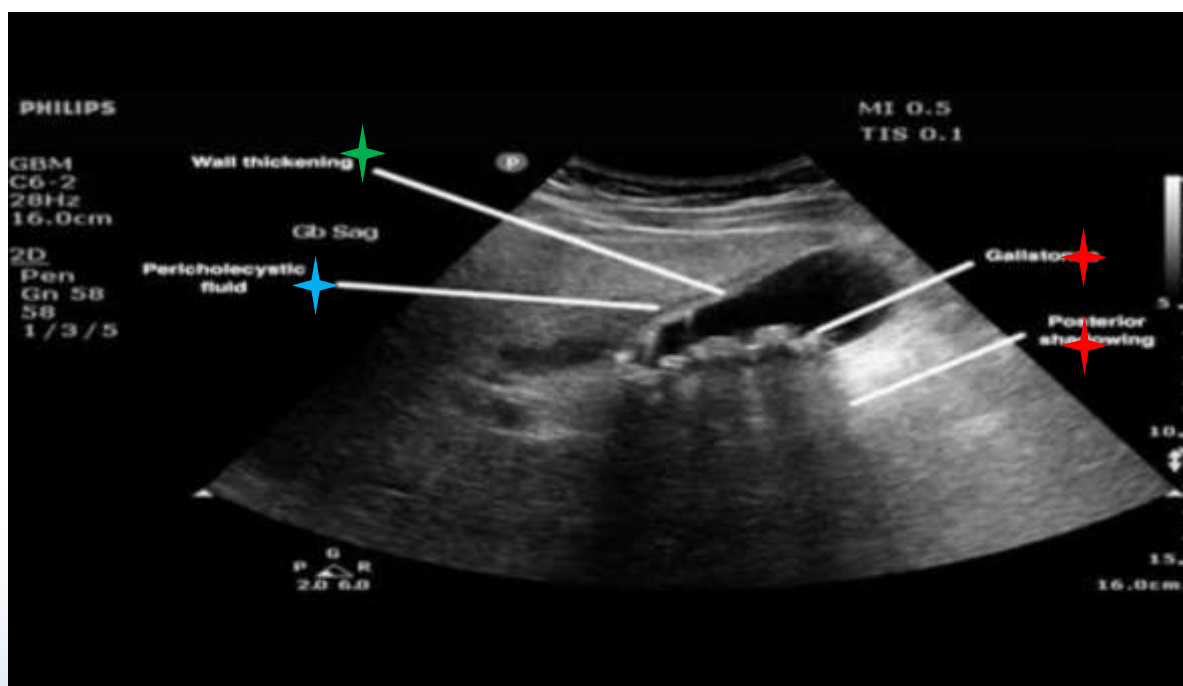
Laboratory

- **CBC**
Leukocytosis
- **Urinalysis**
Pyelonephritis
- **Amylase /Lipase**
Pancreatitis
- **LFT**
Sr bilirubin (direct & total), Albumin, PT & PTT—obstructive jaundice

Mirrizi syndrome→also causes obstructive jaundice→LFT elevated
- **RBS –DM**
Acalculous cholecystitis

Imaging

- **Abdominal Ultrasound**
 1. Thickening of the gallbladder wall ($>4\text{ mm}$)
 2. Pericholecystic fluid
 3. Gallbladder Stone /acoustic shadow
 4. Gallbladder distention
 5. Sonographic Murphy's sign





Ultrasound features of cholecystitis

➤ **HIDA scan**

Positive if there is lack of filling of the gallbladder in 4 hours

Management

➤ **Supportive**

- IV fluids
- NPO/nothing per os
- Analgesics
- IV Antibiotics--with coverage of gram-negative aerobes as well as anaerobes

➤ **Surgical treatment**

- Cholecystectomy is the definitive treatment for patients with acute cholecystitis
- Early cholecystectomy should be performed if the patient present within 72hours
- Delayed/interval cholecystectomy is performed 6 to 10 weeks after initial medical therapy if the patient presented after 72hours
- Cholecystectomy could be open or laparoscopic



ii. **Chronic cholecystitis**

- Ongoing inflammation with recurrent episodes of biliary colic from cystic duct obstruction is referred to as chronic cholecystitis.
- About 2/3 of patients with gallstone present with chronic cholecystitis characterized by recurrent attacks of pain.
- Scarring, and a nonfunctioning gallbladder are characteristics for chronic cholecystitis
- Management
 - Elective cholecystectomy
 - While waiting for surgery the patient should
 - avoid dietary fats and large meals
 - Control comorbidities

What is acalculus cholecystitis? In what kind of patients it may occur? What clinical feature/s may be helpful in differentiating it from calculus cholecystitis patients?

TOKYO GUIDELINE (TG18)

Severity assessment—acute cholecystitis

Grade I (Mild)	Acute cholecystitis in healthy pt with no organ dysfunction. Mild inflammatory change in the gall bladder making cholecystectomy a safe & low-risk operative procedure ➤ Immediately initiate antibiotics & general supportive care (IV fluid, electrolyte compensation, NPO, IV analgesics) ➤ Early Laparoscopic cholecystectomy (LC) within 7days (within 72hrs is better) of onset of symptoms ➤ Conservative Rx but worsening in conditions or no improvement observed within 24 hrs→reconsider early LC if <7days since symptoms onset or biliary drainage (cholecystostomy)
Grade II (Moderate)	Associated with any one of the following conditions 1. Elevated WBC ($>18,000/\text{mm}^3$) 2. Palpable tender mass in RUQ of abdomen 3. >72 hrs of compliant duration 4. Marked local inflammation (gangrenous cholecystitis, pericholecystic abscess, hepatic abscess, biliary peritonitis, emphysematous cholecystitis) ➤ General support + antibiotics ➤ Perform urgent/early LC if the pt is in good performance status & advanced LC technique is available ➤ If not—urgent /early biliary drainage or delayed /elective LC ➤ Culture from blood, bile or both
Grade III (Severe)	Associated with dysfunction of any one of the following organs/systems 1. Cardiovascular dysfunction 2. Neurological dysfunction 3. Respiratory dysfunction 4. Renal dysfunction 5. Hepatic dysfunction 6. Hematological dysfunction ➤ General support + antibiotics ➤ Urgent /early biliary drainage in pts with high surgical risk ➤ Early LC at advanced center if neither negative predictive factor (i.e. jaundice ($\text{Bil-T} \geq 2$), neurological dysfunction, respiratory dysfunction) nor favorable organ system failure & if the pt has good performance status ➤ Culture from blood, bile or both

Chapter 9

Obstructive (surgical) jaundice

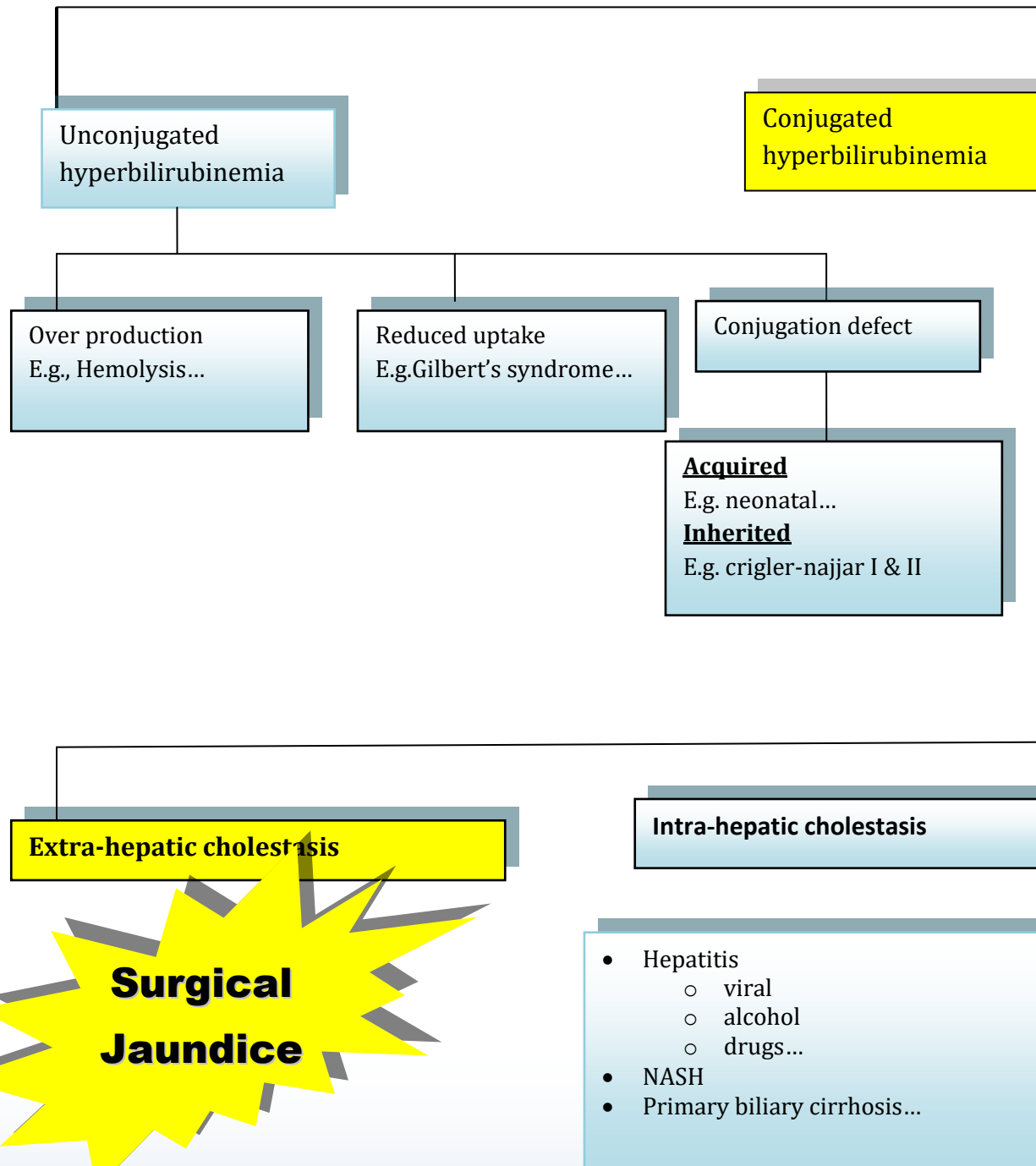


Brain storming

1. List causes of surgical jaundice?
2. List symptoms of surgical jaundice?
3. Mention components of Reynold's pentad?



Causes of Jaundice-Recap



Obstructive jaundice 2^o to? _____



Intraductal causes

Extraductal causes

Intraductal causes

- Cholelithiasis
- Neoplasms
 - Cholangiocarcinoma
 - Gall bladder cancer
- Biliary stricture
 - Surgical trauma
 - Erosion by gallstone
- Parasitic infection
 - Ascaris lumbricoides
- Primary sclerosing cholangitis/PSC/
- AIDS cholangitis
- Choledochal cyst
- Biliary TB--rare

Extraductal causes

- Pancreatitis
- Neoplasms
 - ✓ Periapillary cancer (Pancreatic head cancer, Cholangiocarcinoma & Duodenal cancer)
- Portal adenopathy
 - Metastasis from GIT, breast...
 - TB
- Cystic duct stones
 - Mirizzi's syndrome

History

Obstructive jaundice

1. Pale stool

Bilirubin is reduced by bacterial enzymes in the colon to a series of molecules, termed urobilinogens. In complete biliary obstruction (surgical jaundice) or severe intrahepatic cholestasis, feces may take the appearance of clay color due to the absence of urobilinogen.

2. Dark urine

In conditions associated with elevated conjugated plasma bilirubin concentrations (eg, intrahepatic or extrahepatic cholestasis), the kidney is responsible for 50 to 90 percent of conjugated bilirubin excretion

3. Pruritus

Related to the circulating bile acid & our body response...

4. Jaundice

NB

Urine darkening, stool changes & pruritus are often noticed by the patient before clinical jaundice. Usually clinical jaundice is noticed by the patient & the family when it reaches 6-8mg/dl. Experienced physicians can usually detect it at 2.5-3mg/dl.

Additional history to dig in obstructive jaundice patients, which may give clue of the underlying cause

- Known gallstone disease
- Presence/absence of pain, location & character
 - Stone in Common bile duct → severe colicky pain
 - Periapillary cancer → mild discomfort
- Acuteness of symptoms
 - Stone in common bile duct → long duration
 - Periapillary cancer → short duration (1-3 months)
- Presence of systemic symptoms
 - Fever → cholangitis
 - Weight loss → Significant in periapillary cancer
- Symptoms of gastric stasis → GOO
 - Early satiety, belching, vomiting
- Anemia symptoms -- Usually present in periapillary cancer
- Hepatitis risk factors
- Previous biliary surgery
- Previous malignancy
- GI bleeding
- Patients age & associated conditions
- Diabetes or diarrhea of recent onset
- alcohol, drugs & medications

Physical Examination

- General appearance
 - Chronically sick looking
 - The patient may be jaundiced, with intense itching at presentation or visible scratch marks
- Vital signs are always vital as the name implies
- Signs of jaundice
 - Sclera
 - Skin
- Look for lymphadenopathy
- Gall bladder may be palpable on abdominal examination → **Courvoisier sign**
 - In such case suspect underlying peri-ampullary cancer
- Signs of cirrhosis
 - Ascites, GI bleeding due to portal hypertension
- Xanthomas—suggestive of primary biliary cirrhosis/PBC
- Excoriation marks on integumentary system examination
 - Suggestive of prolonged cholestasis /high grade biliary obstruction

NB

- Malignancy is more commonly associated with the absence of pain & tenderness

1) Cholidocholilithiasis

- Refers to gallstone in CBD (common bile duct)
- Commonly occur secondary to passage of stone from gall bladder to CBD. Primary formation of stone in CBD is less common.

Clinical Presentation

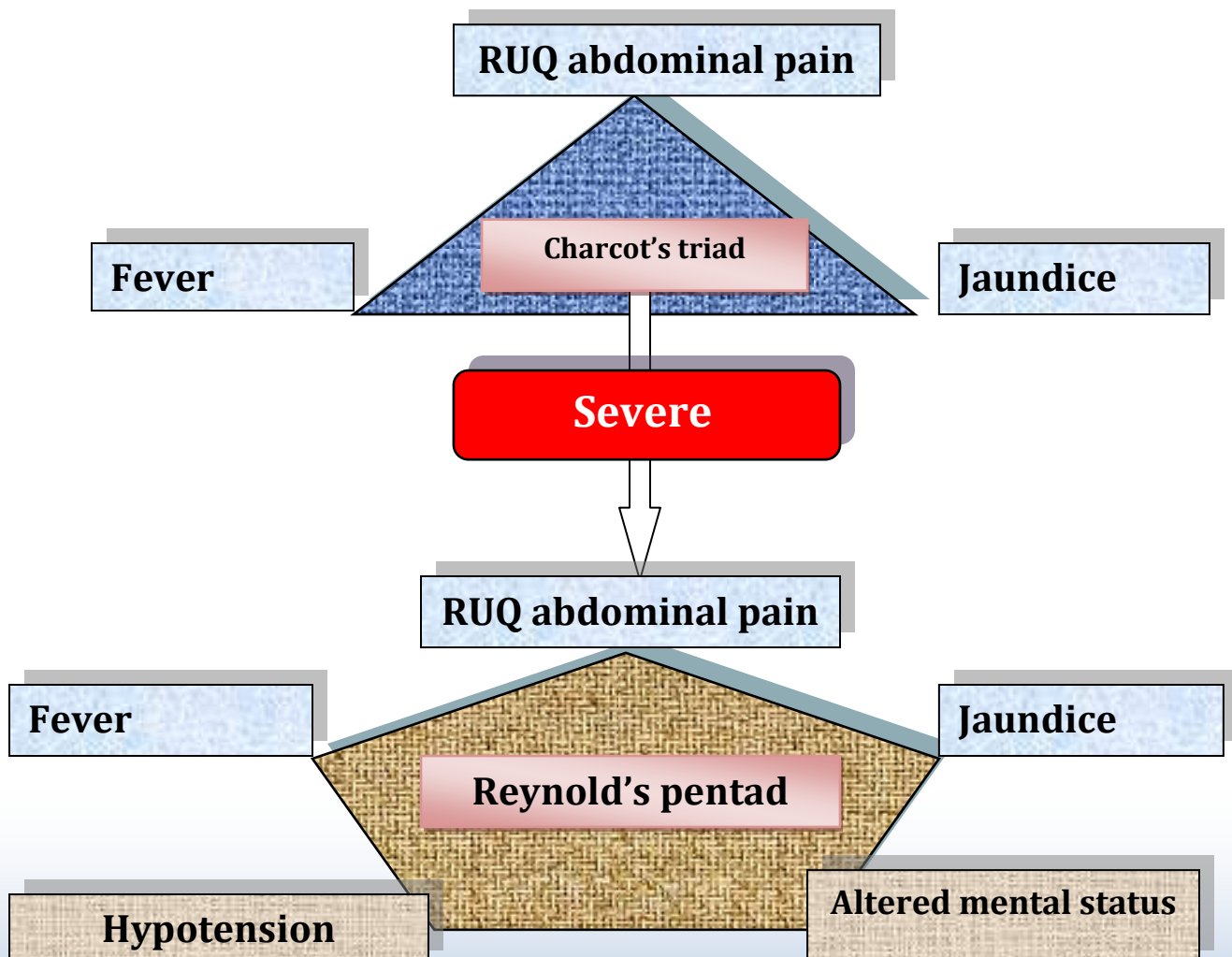
- Asymptomatic in 85% of patients
- Symptomatic → Pain is similar to biliary colic, usually intermittent & associated with nausea and vomiting

Complications

A. Acute cholangitis

Acute cholangitis is a clinical syndrome that develops as a result of stasis & infection in the biliary tract. It is caused primarily by bacterial infection. The organisms typically ascend from the duodenum. cholangitis was first described by charcot as a serious & life threatening condition.

The clinical manifestation can range from charcot's triad to Reynold's pentad when severe.



B. Acute pancreatitis

Acute pancreatitis is an acute inflammatory process of the pancreas. It is usually associated with severe acute upper abdominal pain and elevated blood levels of pancreatic enzymes

Pain character

- ✓ Pattern- steady
- ✓ Quality-sharp
- ✓ Radiation-- to the back
- ✓ Relieving factor--leaning forward
- ✓ Site - epigastric/mid-abdominal
- ✓ Severity--Severe
- ✓ Associated symptoms
 - Nausea, vomiting, Anorexia, diarrhea...may be present

2) Periampullary carcinoma

1) Pancreatic cancer

- Highly lethal malignancy
- 95% of pancreatic cancer arise from the exocrine portion
- 75% arise at the pancreatic head & neck

Risk Factors

- ✓ Sporadic→40%
- ✓ Smoking→30%
- ✓ Dietary factors→5-10% --Especially processed red meat
- ✓ Hereditary→5-10%
- ✓ DM→2times increased risk
- ✓ Underlying chronic pancreatitis→ <5%
- ✓ Alcohol→not independent risk factor unless there is underlying chronic pancreatitis
- ✓ Industrial carcinogen exposure

Clinical Presentation

History

- ✓ Painless obstructive jaundice
- ✓ If abdominal pain present;
 - Mid epigastric pain
 - May radiate to mid/lower back
 - Unrelenting in nature--night time
- ✓ Significant weight loss due to the carcinoma or malabsorption from exocrine pancreas insufficiency
- ✓ Onset of DM within the previous years
- ✓ Migratory thrombophlebitis (Trousseau sign) + venous thrombosis

Physical Examination

- ✓ Palpable, non tender gall bladder/ **Courvoisier sign**
- ✓ Skin excoriations
- ✓ Advanced
 - Ascites
 - Palpable abd.mass
 - Hepatomegally--metastasis
 - Splenomegally--portal venous obstruction
 - Sister mary joseph's nodule
 - Virchow's node
 - Blumer's shelf also possible→palpable rectal mass in rectal pouch

II) Cholangiocarcinoma

Clinical Presentation

History

- Obstructive jaundice symptoms(jaundice,pruritis, clay colored stool & dark urine)
- Weight loss...(variable)
- Abdominal pain--Dull ache in RUQ of the abdomen

Physical Examination

- ✓ palpable gall bladder may be present
 - Courvoisier sign

Sample history 1

Chief complaint

Failure to communicate of 1 hour duration

HPI

This is a known DM patient for the past 10 years on NPH 20/10. Who had overall poor diabetic control (chart revision of the chronic follow-up). But his families claimed he was adherent to his medication.

He was last relatively healthy 04 weeks back at which time he started to experience dark colored urine & clay colored stool associated with itching sensation which began from his hands & later progressed to include all of his body. The itching worsens during night time. 2 weeks prior to admission his families began to notice persistent yellowish discoloration of his eyes. In addition to this he started to experience steady epigastric pain without radiation or known aggravating or relieving factor. He has also low grade intermittent fever but no chills or rigor. 03 days prior to admission he started to experience nausea & non blood tinged, non bilious vomiting of ingested matter about 6 times /day. For the above complaints he visited a traditional healer where he was cauterized on his back. One hour prior to admission he experienced failure to communicate.

- ✓ *Has anorexia, easily fatigability & unquantified weight loss for the past 1 month to the extent his trousers become loose*
- ✓ *Has history of tinnitus, blurring of vision & light headedness*
- ✓ *No history of cigarette smoking or chronic alcohol consumption*
- ✓ *His regular dietary habit is "shiro" made of "atter" & "injera" made of "teff"*
- ✓ *No history of similar illness in the family*
- ✓ *No history of previous abdominal surgery*
- ✓ *No history of multiple sexual partner, contact with jaundiced patient or blood transfusion*
- ✓ *No history of medication except the insulin injection explained above*
- ✓ *No history of chronic cough, contact with chronic cougher or previous TB treatment*
- ✓ *No self/family history of HTN or asthma. No family history of DM*
- ✓ *He has been screened for RVI 1 year back & found to be NR*

Finally, he was admitted_____

Sample history 2

Chief complaint

Yellowish discoloration of the eyes of 01 month duration

HPI

This patient was last relatively health 01year back at which time he began to experience severe intermittent colicky right upper abdominal pain with radiation to the right shoulder. The pain was aggravated while taking fatty meals but no relieving factor noticed by the patient. 03 months prior to admission he started to experience low grade intermittent fever but no chills or rigor. 01 month prior to admission his families started to notice intermittent deep yellowish discoloration of the eye. In addition he started to experience mild itching sensation all over his body but no change in urine or stool color. For the above complaints he visited a traditional healer 3 times, where he was given herbal medication to be drunk & to be used as skin ointment. He was also cauterized on his back. But he didn't get any relief from his symptoms for which he came to our hospital.

- ✓ *He has history of nausea but no vomiting*
 - ✓ *No history of failure to communicate/altered mentation*
 - ✓ *No history of easily fatigability or significant weight loss*
 - ✓ *No history of smoking or alcohol abuse*
 - ✓ *No history of multiple sexual partner, contact with jaundice person or blood transfusion*
 - ✓ *No history of abdominal surgery*
 - ✓ *No family history of similar illness*
 - ✓ *No history of chronic cough or previous TB treatment but he has history of contact with known TB patient who was on Anti-TB medication after sputum examination & CXR was done.*
 - ✓ *No self/family history of DM, hypertension or asthma.*
 - ✓ *He was screened for RVI 9 month back and found to be NR*
- Finally he was admitted to our hospital.....*

Investigation

Laboratory

- **Liver function test/LFT**
 - Serum bilirubin (Total & Direct), ALP, SGOT, SGPT, GGT
Predominant rise in ALP in relation to SGOT & SGPT reflects cholestasis(intra/extra-hepatic) .
 - PT→ PT prolongation with Vitamin K administration is compatible with obstructive jaundice
- **Viral markers**
Hepatitis B surface antigen/HBsAg & Hepatitis C virus antibody/HCVAb
- **CBC**—acute cholangitis
- **Urine bilirubin**

Imaging

- **Abdominal Ultrasound**
Important for documenting stones in the gallbladder (if still present) & determines size of CBD (Common bile duct is said to be dilated when the diameter is >8mm)
- **Contrast CT-scan (abdominal)**
- **MRI**

Diagnostic

- **Cholangiography**
 - ERCP (Endoscopic retrograde cholangiopancreatography)
 - Gold standard for Diagnosing CBD stones
 - Has a therapeutic advantage at the time of diagnosis
 - PTC (Percutaneous transhepatic cholangiography)
 - Examination of liver and bile ducts by x-ray. This is accomplished by the insertion of a thin needle into the liver carrying a contrast medium to help to see blockage in liver and bile ducts.

NB

In both cases fluorescent fluids are used to create contrasts & make the diagnosis possible

Management

1) Choledocholithiasis management in the face of complications

Acute cholangitis

TOKYO GUIDELINE (TG18)

Severity assessment--acute cholangitis

Grade I (Mild)	➤ Doesn't meet the criteria for G III & GII ➤ After Dx immediately start antibiotics & general supportive care (IV fluid resuscitation, Correct electrolyte derangement, IV analgesics) ➤ Perform biliary drainage when no symptom improvement is observed within 24hr
Grade II (Moderate)	Associated with any two of the following conditions 1. Abnormal WBC ($>12,000/\text{mm}^3$ or $<4,000$) 2. High fever ($>39^\circ\text{C}$) 3. Age ≥ 75 yrs 4. Hyperbilirubinemia (Bil-T $\geq 5\text{mg/dl}$) 5. Hypoalbuminemia ($>$upper limit of normal value $\times 0.7$) ➤ Antibiotics + general supportive care ➤ Perform biliary drainage immediately ➤ Culture from blood/ bile or both
Grade III (Severe)	Associated with dysfunction of any one of the following organs/systems 1. Cardiovascular dysfunction 2. Neurological dysfunction 3. Respiratory dysfunction 4. Renal dysfunction 5. Hepatic dysfunction 6. Hematological dysfunction ➤ Apply organ support & emergency biliary drainage ➤ Culture from blood/ bile or both

- ❑ Consider surgical procedures (endoscopic, percutaneous or operative intervention) to remove the cause of acute cholangitis after biliary drainage & amelioration of organ failure (i.e. once the acute illness has been resolved)

Acute pancreatitis → Billiary pancreatitis

- Stabilize the patient
- ERCP
- Cholecystectomy +/- CBD exploration

2) Periampullary cancer

I) Pancreatic cancer

- Palliative
 - ✓ Pain → Narcotics, Celiac plexus block
 - ✓ Jaundice and pruritis
 - Stenting
 - Cholidochoduodenostomy
 - Cholidochojejunostomy
 - Cholecystojejunostomy
 - ✓ Duodenal obstruction (20%) → Prophylactic bypass in unresectable disease /controversial
 - ✓ Adjuvant therapy → Chemotherapy, Radiotherapy
- Curative
 - ✓ Surgical resection--whipple's (with or without pylorus sparing)
 - ✓ Adjuvant therapy
 - Chemotherapy
 - Radiotherapy

II) Cholangiocarcinoma

- Surgery is the only curative treatment
 - But greater than half of the patients have unresectable tumor on intraoperative finding
 - biliary decompression + cholecystectomy (to prevent cholecystitis)
 - Unresectable perihilar tumor → Roux-en-Y cholangiojejunostomy
- Distal bile duct tumors often resectable → Pylorus preserving pancreaticoduodenostomy
- No proven benefit of adjuvant chemotherapy
- intraoperative radiation and external beam radiation for unresectable tumor

Chapter 10

Liver abscess

Brain storming

1. Classify liver abscess based on the etiology & master the patient approach?
2. List down DDx for RUQ abdominal pain?



Etiologic wise Liver abscess classification with patient approach

1) Pyogenic liver abscess

Risk Factors

- Impaired bile drainage
 - Biliary tract diseases → causing ascending cholangitis
- Systemic bacteremia → Hematogenous dissemination
 - E.g., subacute endocarditis, Infected indwelling catheter, Pyelonephritis...
- Local spread of infection
 - Like gangrenous cholecystitis, perforated ulcers, sub-phrenic abscess...
 - Diverticulitis, Crohn's disease → direct extension to liver
- Hepatic trauma
 - Penetrating trauma → Direct inoculation of microorganisms
 - Blunt trauma → By causing localized hepatic necrosis, hemorrhage, bile leakage...
- Pyogenic liver abscess may occur as a complication of...
 - Cholangiography, percutaneous transhepatic stents, endoscopic stent placement, biliary-enteric anastomosis...

NB

E.coli is the commonest causative agent (2/3 of cases)

Clinical Presentation

History

Usually sub-acute and nonspecific, leading to a delay in presentation, diagnosis & treatment

- RUQ abdominal pain
 - Referred pain to the right shoulder may be present
- Fever, chills
- Anorexia, malaise
- Yellowish discoloration of eyes & skin may present in 1/3rd of patients
- Anemia of chronic illness (look for anemia symptoms)
- Cough may be present due to diaphragmatic irritation

NB

When abscesses are seen in children & adolescents, you should have high index of suspicion for underlying immunodeficiency, severe malnutrition or trauma

Physical examination

Abdomen

- Tender hepatomegally
- Intercostal tenderness → differentiate it from acute cholecystitis
- Palpable mass may not be present

Chest

- Decreased breath sound may be present on the right basal lung zone
- Pleural or hepatic friction rub may be present due to diaphragmatic irritation or inflammation of glisson capsule respectively

HEENT & Integumentary system

- Jaundice may be present (1/3rd of pts) usually when associated with biliary tract disease

Complications of pyogenic liver abscess

- Generalized sepsis
- Pleural effusions, Empyema, pneumonia...
- Intraperitoneal rupture which is frequently fatal
NB*
Usually the abscess does not rupture, but develops a controlled leak resulting in a perihepatic abscess.
- Hemobilia (Hemorrhage in bile)
- Hepatic vein thrombosis

Investigation

- **CBC**
Leukocytosis
- **ESR**
Raised ESR
- **Elevated ALP level**
Significant abnormality in the remaining LFTs is unusual
- **Blood culture**
Only in 50% of patients show the causative organism

Imaging

▪ Abdominal ultrasound

What to look for abscess on ultrasound?

- Round/oval hypoechoic lesions
- Well defined border
- Variable number of internal echoes

▪ CT-scan

- Hypodense with peripheral enhancement
- May contain air fluid level

Management

Antibiotics

- ❑ Aminoglycoside + clindamycin + ampicillin/vancomycin.
 - ✓ Fluoroquinolones can replace aminoglycosides, and metronidazole can be used instead of clindamycin
- ❑ Duration
 - ✓ 02 weeks

Aspiration and Percutaneous Catheter Drainage

- ❑ Aspiration → useful in guiding subsequent antibiotic therapy
- ❑ Percutaneous catheter drainage is only beneficial for a minority of patients because most pyogenic abscesses are quite viscous
- ❑ Patients in whom percutaneous drainage is not appropriate include...
 - ✓ multiple large abscesses
 - ✓ known intra-abdominal source that requires surgery
 - ✓ an abscess of unknown etiology
 - ✓ Ascites
 - ✓ abscesses that require transpleural drainage

Surgical Drainage

- ❑ Indications
 - ✓ patients that have failed non-operative therapy
 - ✓ those who need surgical Rx of the underlying source
 - ✓ those with multiple macroscopic abscesses
 - ✓ those on steroids
 - ✓ those patients with concomitant ascites

2) Amebic liver abscess

Risk Factors

- ✓ Low socioeconomic status and unsanitary conditions are significant independent risk factors
 - Contaminated food & water
- ✓ living or visiting an endemic area
- ✓ Presence of immunosuppression

NB

E. histolytica is the causative organism

Clinical presentation

- May be acute
With fever and RUQ abdominal pain, or
- Sub-acute
With weight loss, less frequent fever and abdominal pain
- The usual amebic liver abscess case doesn't present with concurrent colitis, but patients may have had dysentery within the last year

Complications of Amebic liver abscess

Peritonitis

- Due to rupture in the majority and secondary to necrotizing or perforated amebic colitis

Thoracic amebiasis

- ✓ Empyema, broncho-hepatic fistulas and pleuropulmonary abscess
- ✓ If the abscess ruptures into the pleural cavity → it usually occurs suddenly, collapsing the lung & filling up the pleural space
- ✓ If the abscess ruptures into the bronchi, this complication causes sudden onset of cough with expectoration of copious brown sputum

Pericardial amebiasis

- ✓ Acute pericarditis with tamponade

Hematogenous spread

Lung, brain, skin and genitourinary tract...

Investigation

Laboratory studies

- ✓ **CBC**
- ✓ Mildly elevated **ALP**
- ✓ Serologic tests (**fluorescent antibody test**) → may remain positive after clinical cure
- ✓ **Stool examination**

Imaging

- ✓ **Ultrasound**
- ✓ **CT-scan**
- ✓ **MRI**

Diagnostic Aspiration

- ✓ Usually done when amebic serologies are negative and a pyogenic cause needs to be ruled out
- ✓ The fluid of an amebic abscess is odorless, Gram stain and cultures are negative

NB*

Aspiration should not be done if an echinococcal cyst or a cancer is suspected

Management

Antibiotics

Drug of choice → Metronidazole

Drainage

- ✓ Indications
 - Patients that have no clinical response to drug therapy within 5–7 days
 - Those with a high risk of abscess rupture (cavity >5 cm in diameter)
 - The presence of lesions in the left lobe → because they have risk of rupture into the pericardium
- ✓ Drainage may be by;
 - Percutaneous drainage/Image-guided
Procedure of choice for decreasing the size of an abscess.
 - Surgical drainage → Indications
 - Abscesses that have failed to respond to more conservative therapy
 - Life-threatening hemorrhage
 - When the amebic abscess erodes into a neighboring viscus
 - Sepsis due to a secondarily infected amebic abscess if percutaneous treatment fails

DDx & Discussion

❑ Biliary diseases

Refer the chapter on obstructive jaundice

❑ Hepatocellular cancer (HCC)

Risk factors

- Hepatitis B & C infection
- Alcoholic cirrhosis
- Hemochromatosis...

Clinical Presentation

History

- ✓ Dull, vague RUQ pain, sometimes referred to shoulder
- ✓ Constitutional symptoms of malignancy
- ✓ Yellow discoloration (50%)
- ✓ Gastrointestinal bleeding (10%)
 - Esophageal varices (50%) → This patients have an extraordinarily poor prognosis, with a median survival measurable in weeks.
 - Intraportal thrombus
This further increases the portal pressure and makes bleeding varices more difficult to control

NB

Slowly growing tumor but majority of patients present at an advanced stage and most are beyond curative treatments

Physical examination

- General appearance
Chronically sick looking, looks severely malnourished
- HEENT
Pale conjunctive, icteric sclera
- LGS
Check Virchow's node
- Abdominal examination
 - ✓ Hepatomegaly
 - Character--Nodular, irregular & hard
 - Vascular bruit may be heard in 25% of the cases
 - ✓ Signs of abdominal fluid collection(fluid shift, fluid thrill)

NB

Typical signs of decompensated liver disease include ascites, jaundice or encephalopathy

Investigation

- **Imaging**
 - ✓ Abdominal ultrasound
 - ✓ CT/MRI
- **Laboratory**
 - AFP>500ng/dl
- **Metastatic work-up**

Management

- ✓ Surgical resection
 - But majority of patients are not eligible because of the tumor extent or underlying liver dysfunction
- ✓ Other treatment options
 - Radiation therapy
 - chemotherapy
 - Liver transplantation

❑ Hydatid cyst

- ✓ Parasitic infestation/ E. granulosus

Risk factors

- History of living or visiting an endemic area
- Ingestion of food or water contaminated by the definitive host

Clinical Presentation

- Patient presentation depend on
 - ✓ The site, size, stage of development of the parasite
 - Site
 - Liver (63%) & lung (25%)
 - ✓ Whether the cyst is alive or dead
 - ✓ Whether the cyst is infected or not
- Initially
 - ✓ Non-specific
 - ✓ RUQ/ epigastric pain, cough, low-grade fever & sensation of abdominal fullness
- Mass/pressure effect→after long duration
 - ✓ liver
 - Symptoms of obstructive jaundice & abdominal pain
 - Biliary rupture→Biliary colic, jaundice & urticaria
 - ✓ Lung involvement
 - Chronic cough, dyspnea, pleuritic chest pain & hemoptysis
 - ✓ Cerebral involvement
 - Headache, dizziness & decreased level of consciousness

Investigation

- Serology
Determination of specific antigens and immune complexes of the cyst with ELISA
- Imaging
 - ✓ Ultrasound, CT-scan
 - Classic findings include calcified thick walls, often with daughter cysts
 - Ultrasound defines the internal structure, number, and location of the cysts and the presence of complications
 - Computed tomography/CT gives more specific information about the location and depth of the cyst within the liver
- ERCP
 - ✓ show communication between the cysts and bile ducts
 - ✓ Can be used to drain the biliary tree before surgery

Management

- Most are asymptomatic on presentation, but complications such as pulmonary infection, cholangitis, rupture, and anaphylaxis give good reason to consider treatment for all
- Basic principles of the management;
 - ✓ Eradication of the parasite within the cyst
 - ✓ Protection of the host against spillage of scoleces
 - ✓ Management of complications
- The treatment options;
 - ✓ Medical
Antihelminthics
 - ✓ Percutaneous aspiration & drainage
 - ✓ Surgical

DDx...continued

- ☐ Pneumonia
- ☐ Empyema (pleuropulmonary)

Sample History

Chief complaint

Abdominal pain of 04 months duration

HPI

This patient was last relatively healthy 04 months back at which time he started to experience gradual onset constant dull aching type right upper abdominal pain with radiation to the right upper back & inter scapular region without aggravating or relieving factor noticed by the patient. Associated with this he has low grade intermittent fever, loss of appetite & nausea but no vomiting, diarrhea or constipation. In addition he has easily fatigability & significant weight loss of 8kg for the past 04months (70kg to 62kg). 03 months prior to the admission he started to experience tinnitus & light headedness but no blurring of vision. 01 month prior to the re-admission his wife noticed yellowish discoloration of his eyes but no itching sensation, urine or stool color change. For the above complaints he visited a nearby health center in 'Chilga' where he was given #04 drugs; red & yellow circular, white oval & omeprazole, each to be taken 2times/day for 10days but he didn't get relief from his abdominal pain. Then the patient was referred to our hospital for better investigation & management. After the 1st admission to our hospital, aspiration was taken from his right upper abdomen. Then he was discharged with metronidazole to be taken 3times/day, 3capsules at once for 10 days & appointed for follow-up. But 20 days prior to the re-admission the patient discontinued his medication complaining of 'no improvement' by medical treatment & went to a traditional healer. He got cauterized on both of his arms & at the back of his neck.

- ✓ *He has no history of contact with jaundiced person, blood transfusion or multiple sexual partner*
- ✓ *He used to drink local 'areke' up to 50gm/day for about 20 years*
- ✓ *He has no history of cough, hemoptysis or bone pain*
- ✓ *He has history of palpitation but no dyspnea, orthopnea, PND or lower leg swelling*
- ✓ *He has no history of abdominal trauma or surgery*
- ✓ *He has no history of contact with chronic cough or previous TB treatment*
- ✓ *No self or family history of DM, hypertension or asthma*
- ✓ *He has been Screened for RVI 3years back & found to be non reactive*

Finally he was admitted to our hospital supported by.....

Chapter 11

Fracture

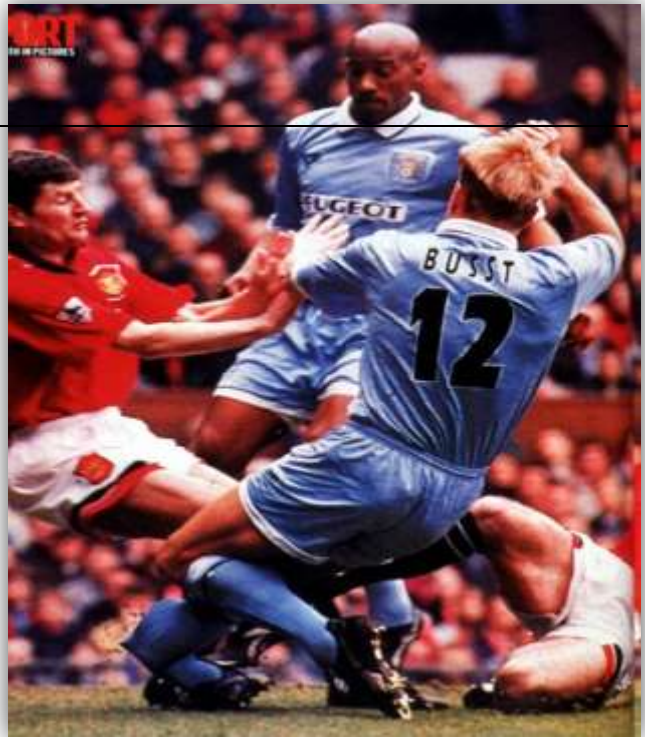


Brain storming

1. List the components of ATLS primary & secondary survey?
2. List down the definitive management options for fracture?
3. List complications of fracture?
4. What is the importance of having both AP & lateral x-ray in fractured patients?

Possible causes of fracture

- Traumatic fracture
 - Motor vehicle accidents
 - Gunshot injuries
 - Blast & bomb injuries
 - Assaults
 - Stone, knife, sickle, axe, stick
 - Fall down injuries
 - Burn
- Stress fractures
- Pathologic fracture



NB

- You must apply ATLS primary survey /ABC of life / in all trauma patients before taking detailed history & doing physical examination,
 - **A**irway
 - **B**reathing
 - **C**irculation
 - **D**isability/Neurologic
 - **E**xposure
- ATLS secondary survey
 - Detailed history
 - Physical examination
 - Investigation
 - Definitive management

The history note for fracture should contain

- Mechanism of injury
- Duration
- Degree of violence
 - ✓ Motor vehicle accident
 - Speed of the vehicle?
 - Crush or not?
 - Was there external damage to the vehicle? External damages like deformation to the front from Head-on collision. This raises suspicion of hip dislocation...
 - Pedestrian or passenger?
If passenger;
 - **Pre-crash**
 - What was the pre-crash location of the patient? It may suggest the type of fracture.
E.g. lateral compression fracture of pelvic bones from a side impact in vehicle collision
 - Was the patient wearing seat belt/not?
 - **Post-crash**
 - What was the post-crash location? Inside or ejected
 - If ejected, what was the distance thrown? & the landing condition/stony ground???
 - Was the patient crushed by an object? If so, explain the site of crush, weight of the object & the duration
- ✓ Gun shot
 - Distance of shooting?
 - Type of gun?
 - Entry & exit wound of the bullet?
- ✓ Fall down
 - Height or depth of fall
 - Landing condition—stony ground or not



✓ Blast & bomb injury

- What was the patient distance from the blast?
- Primary blast injury or secondary
 - secondary → from the blast effect due to accelerated debris or objects
- Specify the injury Site
- Estimate the extent of bleeding if there is any overt bleeding
- Change in limb function

Is the patient able to use the injured limb/not?
- Concomitant injury
 - Head injury
 - History of loss of consciousness, abnormal body movement...?
 - Spinal cord injury
 - Is there any urine/ fecal incontinence or inability to void?
 - Bladder injury
 - Hematuria, suprapubic pain & difficulty of voiding?
- As with all trauma patients, documenting any available history regarding pre-existing medical conditions, medication and drug allergies will be useful...

Physical examination

Signs of fracture

Definitive

- Deformity
- Abnormal movement
- Crepitus
- Visible fractured bone



Colles fracture/"dinner fork deformity"

Unspecific

- swelling
- tenderness



Musculoskeletal system (MSS) examination

General approach

- ❖ Inspection [look]
- ❖ Palpation [feel]
- ❖ Range of Motion [move]
- ❖ Measurement [measure]

Look

- Expose both sides
- Adequate light

Specific to the site of interest

- Look for asymmetry
- Deformity
- Visible fractured bone
- Swelling
- Laceration
- Bruising /discoloration
- Abnormal movement
- Atrophy...



Compound fracture



Feel

- Temperature
 - Compare both sides of extremities with back of your hand
- Tenderness
- Check pulses
 - ✓ If lower extremity
 - ⊕ Dorsalis pedis artery
 - ⊕ Posterior tibialis artery
 - ⊕ Popliteal artery...
 - ✓ If upper extremity
 - ⊕ Radial artery
 - ⊕ Brachial artery
- Compare muscle bulk by measuring with tape meter
- Assess capillary refill
 - If >2sec → abnormal
- Swelling → characterize...

Move

Range of motion (Active)

- Ask the patient to move the affected site
- Watch for decreased or increased movement of the joint compared to the other side as well as the normal
- Watch for pain with movement
- Listen for crepitus or “popping”
- Watch for abnormal movements

ROM (passive)

- Move the joints passively, comparing the end points to the active
- Again note any decreased or increased movement
- Pain with the movement
- Crepitus or “popping”

Measure

- Apparent length
 - From xiphisternum or umbilicus to medial malleolus
- Real length
 - From greater trochanter of the femur up to the medial malleolus
- True length
 - Between two bony prominences



Complications of fracture

- ❑ Delayed union
 - If > 4month
- ❑ Non-union
 - If > 6month

NB

Causes of delayed union & non-union

- Inappropriate immobilization
- Infection
- Bone loss
- Soft tissue interposition...

Tests of union

Clinical

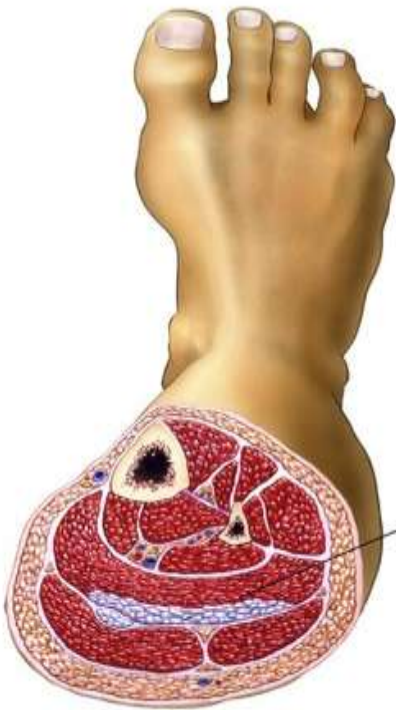
- Absence of mobility between fragments
- Absence of tenderness on firm palpation
- Absence of pain when angulation stress is applied

Radiological

- Visible callus bridging the fracture & blending with both fragments → early sign & reliable
- Continuity of the bone trabeculae across the fracture

- ❑ Mal-union
 - Overlapped
 - Angulated
- ❑ Shortening
 - Significant when >2cms
 - Causes
 - i. Malunion
 - ii. Actual bone loss or crushing
 - iii. Interference with growth plate (in children)
- ❑ Avascular necrosis
 - Death of bone due to deficient blood supply
 - Common sites
 - i. Head of the femur after femoral neck fracture/hip dislocation
 - ii. Proximal half of Scaphoid bone
 - iii. Body of talus
- ❑ Infection
 - Virtually confined to open fractures
 - Read more about bone infection under short cases of Debol
- ❑ Neuro-vascular injuries
 - Nerves
 - E.g.
 - i. Humerus fracture may lead to radial nerve injury resulting in wrist drop
 - ii. Knee—injury to common peroneal nerve→foot drop
 - Vascular
 - E.g.
 - i. Shoulder dislocation—axillary artery
 - ii. Supracondylar fracture of humerus/elbow dislocation--Brachial artery
 - iii. Knee dislocation/proximal tibial fracture--Popliteal artery
- ❑ Compartment syndrome—Signs/the 6Ps

1. Pain
 2. Pallor
 3. Pulselessness
 4. Paresthesia
 5. Paralysis
 6. Perishingly (extremely) cold



Compartments of lower leg



Compartment syndrome

NB

Common sites of compartment syndrome

- Lower leg
- Forearm
- Foot
- Hand
- Gluteal region
- Thigh...

- ❑ Pulmonary complications
E.g. Fat embolism

NB

Generally fracture is classified as;

1. Closed fracture or
2. Open/compound fracture based on its communication to the external environment.

Normal repair of tubular bone has five stages

1. Stage of hematoma
2. Stage of cellular perforation
3. Stage of callus
4. Stage of consolidation
5. Remodeling

Sample history

Chief complaint

Bullet injury of 02 hours duration

HPI

This patient was apparently healthy 02 hours back at which time he sustained a bullet injury to his left leg from about 2 meters distance by a known offender (his neighbour) on land dispute. The bullet entered around his left knee anteriorly & exited on lateral side of his left thigh. He had moderate bleeding from the site of injury but no loss of consciousness or trauma to other sites.

- ✓ *He has tinnitus, vertigo & blurring of vision*
- ✓ *No history of chest pain or breathing difficulty*
- ✓ *No history of urine or fecal incontinence*
- ✓ *No history of lower abdominal pain or reddish discoloration of urine*
- ✓ *No self/family history of DM, hypertension or Asthma*
- ✓ *Not screened for RVI but has no history of chronic diarrhea, herpes zoster attack or multiple sexual partner*

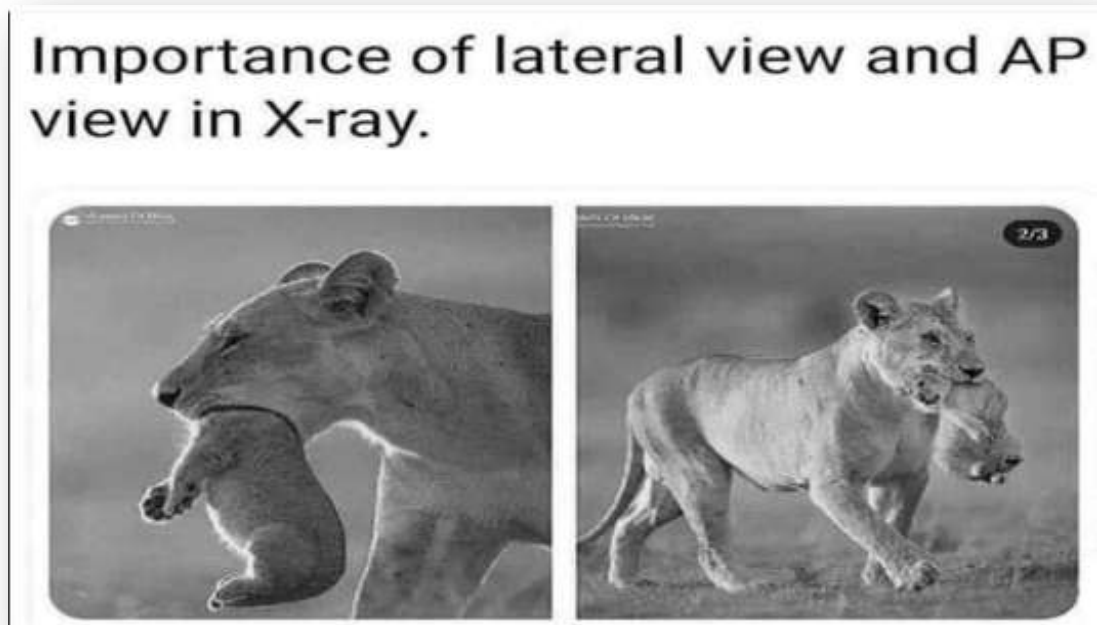
Finally he was admitted supported by.....

Investigation

Imaging

➤ X-ray

Antero-posterior (AP) & Lateral view, Why?



From your x-ray you should answer....

- ✓ Traumatic, Fatigue or pathologic fracture?
- ✓ Displaced or not? If displaced, in which direction?
- ✓ Pattern of the fracture?
- ✓ Alignment of the fragments, satisfactory or not?
- ✓ Recent fracture or not? If not, evidences of union may be there
- ✓ Associated injury? To adjacent joints or bones.

NB

Patterns of fracture

1. Transverse
2. Oblique
3. Spiral
4. Segmental
5. Comminuted(>2 fragments)
6. Compression (crush)
7. Green stick (peculiar to children) → incomplete fracture

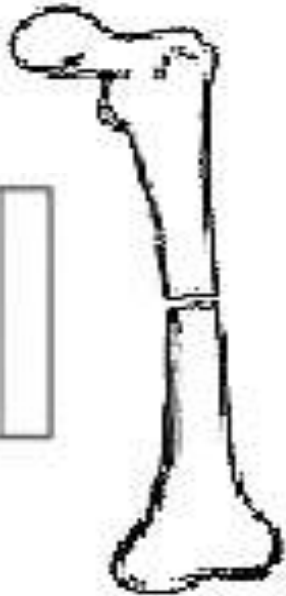
These patterns may indicate

- Nature of causative violence
Important for choosing easiest method of reduction
- The likely stability of fragments after reduction

Transverse



Transverse fracture.



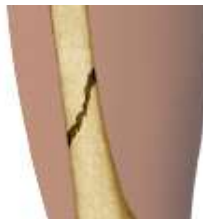
Transverse



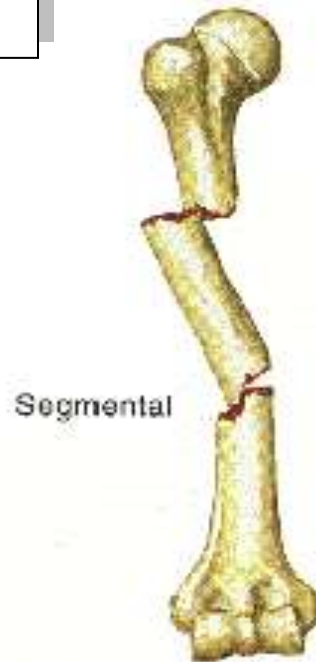
Oblique



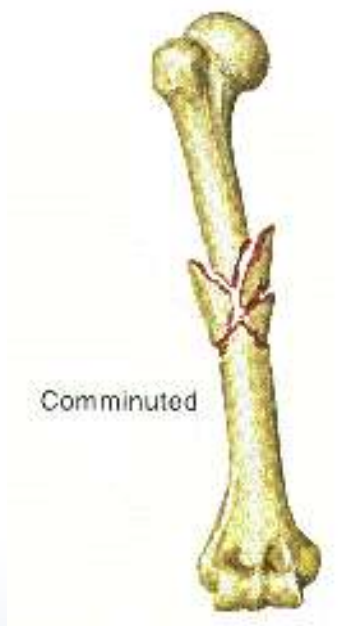
Oblique



Segmental



Comminuted



Compression



Green stick fracture



Investigations...continued

- CT-scan
- Angiography
- Arthroscopy

Laboratory

- Hct
- BG & Rh, x-match

Management

ATLS

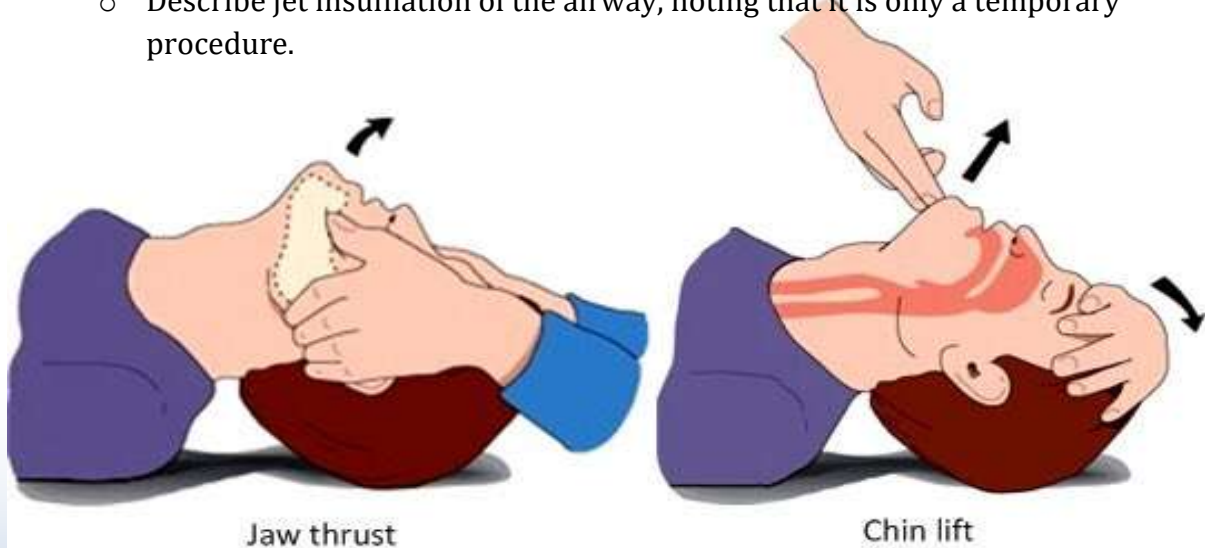
ATLS primary survey

1. AIRWAY MAINTENANCE WITH CERVICAL SPINE PROTECTION

- Assessment
 - Ascertain patency
 - Rapidly assess for airway obstruction
- Management

Aim→To establish a patent airway

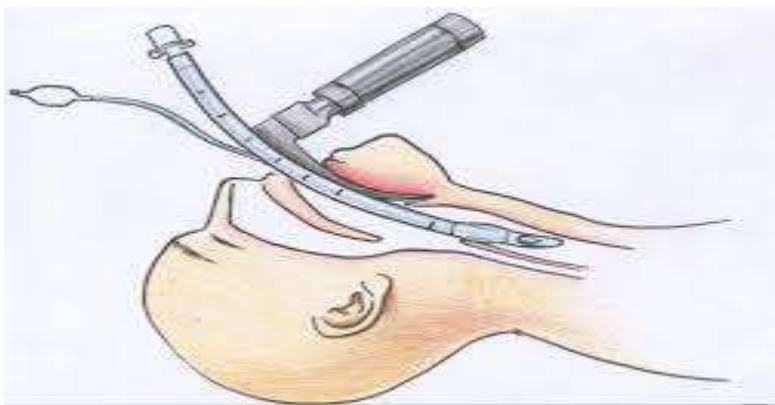
 - Perform a chin-lift or jaw-thrust maneuver
 - Clear the airway of foreign bodies
 - Insert an oropharyngeal airway
 - Establish a definitive airway
 - ✓ Intubation
 - ✓ Surgical cricothyroidotomy
 - Describe jet insufflation of the airway, noting that it is only a temporary procedure.



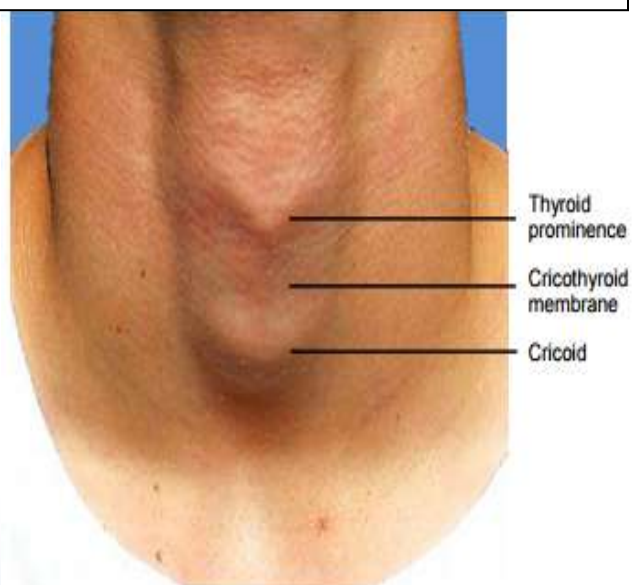
Oropharyngeal/ gudel airway



Endo-tracheal intubation



Cricothyroidotomy



- Maintain the cervical spine in a neutral position with manual immobilization as necessary when establishing an airway
- Reinstatement of immobilization of the cervical spine with appropriate devices after establishing an airway

2. **B**REATHING: VENTILATION AND OXYGENATION

a. Assessment

- i. Expose the neck and chest, and ensure immobilization of the head and neck
- ii. Determine the rate and depth of respirations
- iii. Inspect and palpate the neck and chest for tracheal deviation, unilateral and bilateral chest movement, use of accessory muscles, and any signs of injury
- iv. Percuss the chest for presence of dullness or hyperresonance
- v. Auscultate the chest bilaterally

b. Management

- i. Administer high-concentration oxygen
- ii. Ventilate with a bag-mask device
- iii. Alleviate tension pneumothorax
- iv. Seal open pneumothorax
- v. Attach a CO₂ monitoring device to the endotracheal tube
- vi. Attach a pulse oximeter to the patient



3. CIRCULATION WITH HEMORRHAGE CONTROL

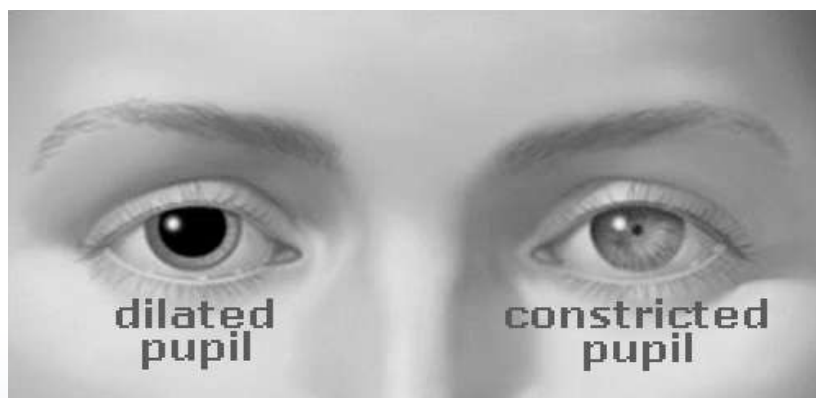
- a. Assessment
 - i. Identify source of external, exsanguinating hemorrhage
 - ii. Identify potential source(s) of internal hemorrhage
 - iii. Assess pulse: Quality, rate, regularity, and paradox
 - iv. Evaluate skin color
 - v. Measure blood pressure, if time permits
- b. Management
 - i. Apply direct pressure to external bleeding site(s)
 - ii. Insert two large-caliber IV catheters
 - iii. Simultaneously obtain blood for hematologic and chemical analyses; BG & Rh, cross-match...
 - iv. Initiate IV fluid therapy with warmed crystalloid solution and blood replacement
 - v. Prevent hypothermia
 - vi. Consider presence of internal hemorrhage and potential need for operative intervention, and obtain surgical consult

4. **DISABILITY: BRIEF NEUROLOGIC EXAMINATION**

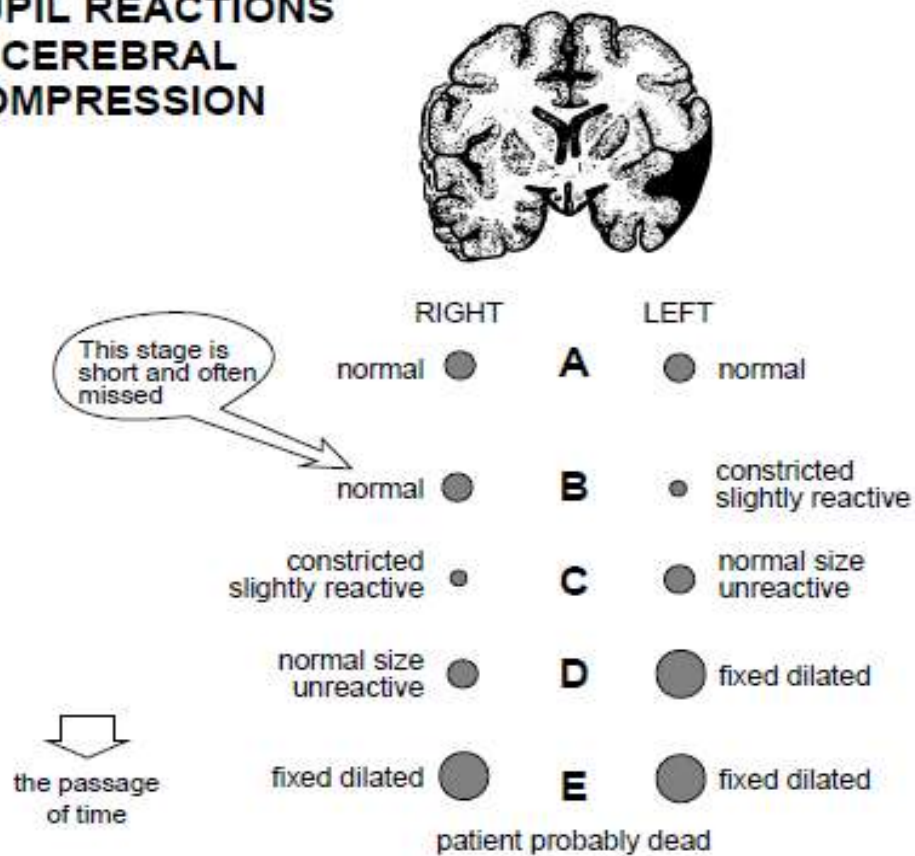
A) Determine the level of consciousness using the GCS

Score	Eyes opening (E)	Verbal (V)	Motor (M)
6			Obeys commands
5		Normal oriented conversation	Localizes to pain
4	Spontaneously	Confused	Withdrawal/flexion
3	To verbal command	Inappropriate/words only	Abnormal flexion (decorticate)
2	To painful stimulus	Sounds only	Extension (decerebrate)
1	No response	No sounds	No motor response
T		Intubated patient	

B) Check pupils for size and reaction



PUPIL REACTIONS IN CEREBRAL COMPRESSION



C) Assess for lateralizing signs and spinal cord injury

5. EXPOSURE/ENVIRONMENTAL CONTROL

Completely undress the patient, but prevent hypothermia & inspect for concomitant injuries

ATLS Secondary survey

After stabilization of the patient

- Take detailed history & do physical examination
- Work up/investigate the patient
- **Definitive management ***
 - A. Reduction
 - B. Immobilization
 - C. Rehabilitation

A. Reduction

1. Closed manipulation
 - Using general anesthesia but sometimes use of local/regional anesthesia may be possible
 - Grasp the fragments through the soft tissue then adjust them as nearly as possible to their correct position
2. Mechanical traction (With or without manipulation)
 - ✓ Used when the contraction of large muscles exert a strong displacing force
 - ✓ The traction may be weight or screw device
3. Operative reduction
 - Reduction under direct vision



B. Immobilization

- Why we immobilize the affected site?
 - To prevent angulation/displacement
 - To prevent movement that may interfere with union
 - To relieve pain

NB

Immobilization must include the joint above & below the fracture

How to immobilize???

1. POP (plaster of Paris)

- Indications
 - Un-displaced fractures
 - Tolerable displacement
 - Closed reduction of displacement possible
 - Fracture in children (Upper and lower extremities)



➤ Types

- Circular
- Slab

➤ Applications of POP

- Long arm
- Short arm
- Long leg

- Padded
- Unpadded

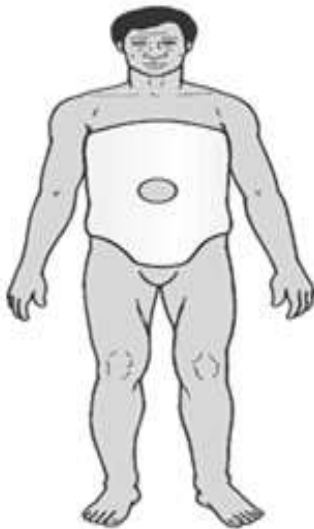
- Short leg
- Hip spica



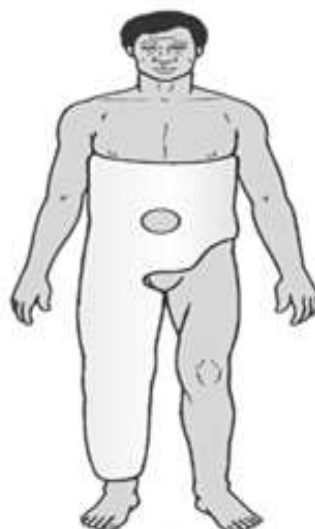
Short arm cast



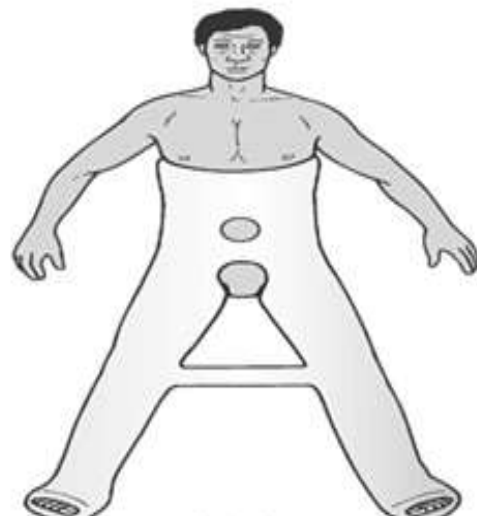
Long arm cast



Body jacket cast



Single hip spica



Double hip spica

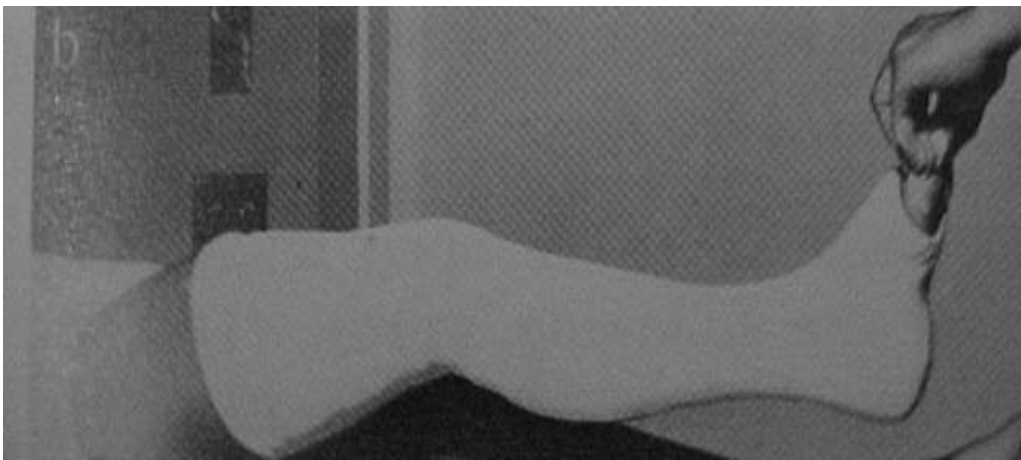


Long leg cast



Short leg cast

- Advantages of POP
 - Easy to do
 - No risk of infection
- Disadvantages of POP
 - Damage to vessels & nerves by compression
 - Compartment syndrome
 - Management
 - All constrictive dressings must be released
 - If no significant change after 30-60 min of monitoring → fasciotomy should be done
 - Difficult for wound care
 - Needs strict control



2. Splinting

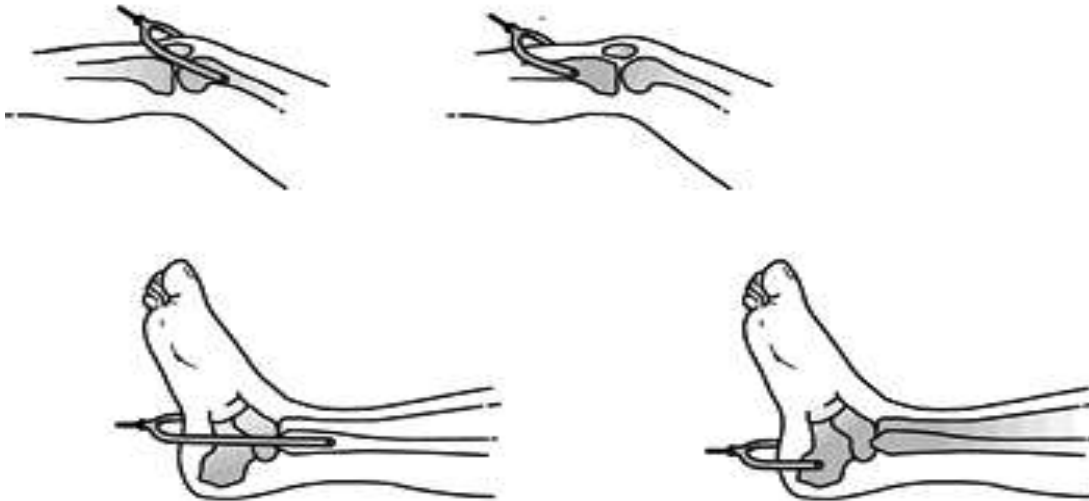
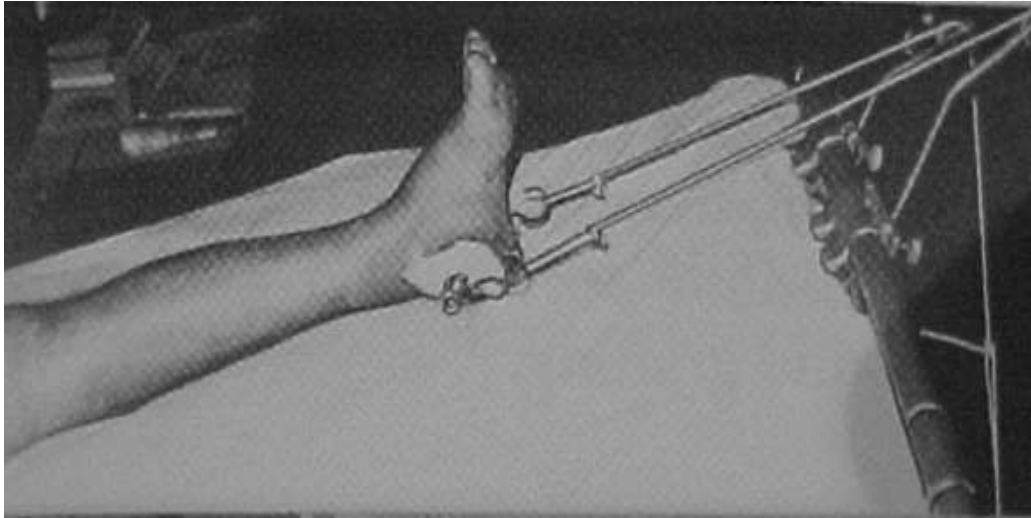
Traction

A. Skin traction



- Commonly used in children

B. Skeletal traction

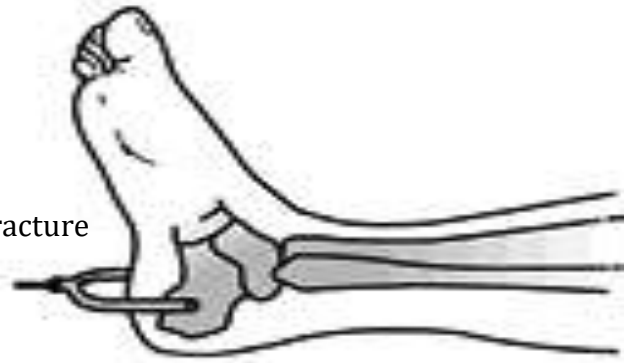


Pin insertion sites

- Condyle of the femur (distal femur)
- proximal tibia
 - Pin
 - Insert from lateral to medial
 - Where?
 - 1 finger below then 1 finger lateral to tibial tuberosity
 - Why?
 - To prevent neurovascular damage
 - Common peroneal nerve is damaged may result in foot drop
 - Weight to be applied
 - $1/10^{\text{th}}$ to $1/6^{\text{th}}$ of patient's weight

- Calcaneus

- Pin
 - Insert from medial to lateral
- Weight applied
 - Usually up to 3kg
- Commonly used for femoral neck fracture

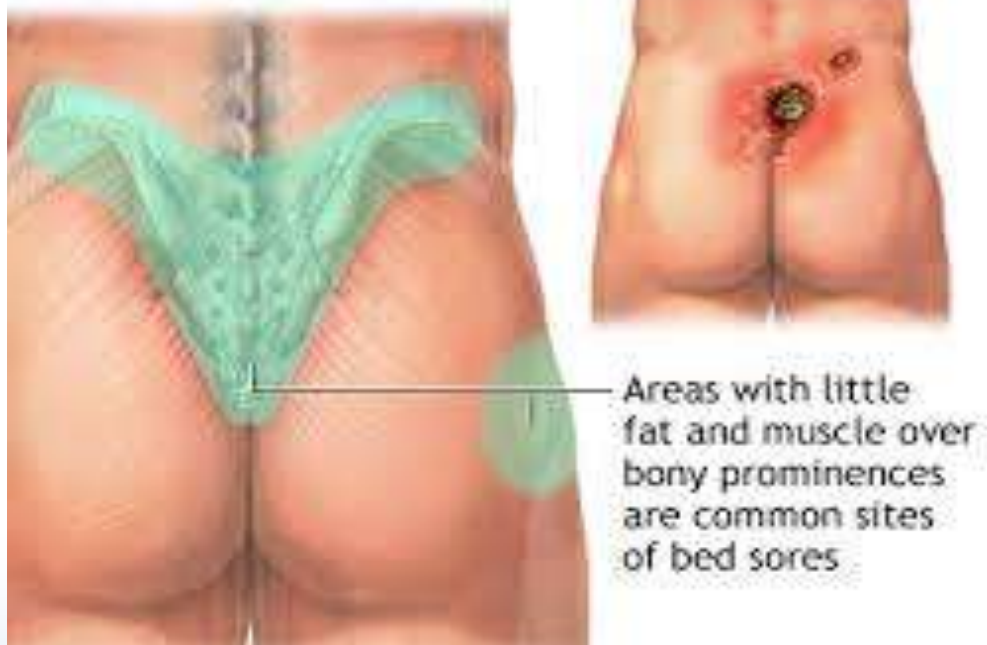


Disadvantages of traction

- Long immobilization time (usually up to 06 wks)

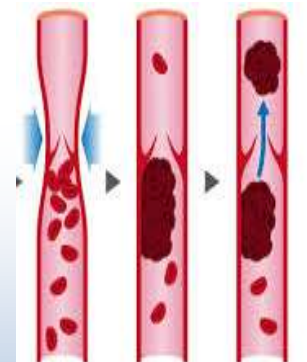
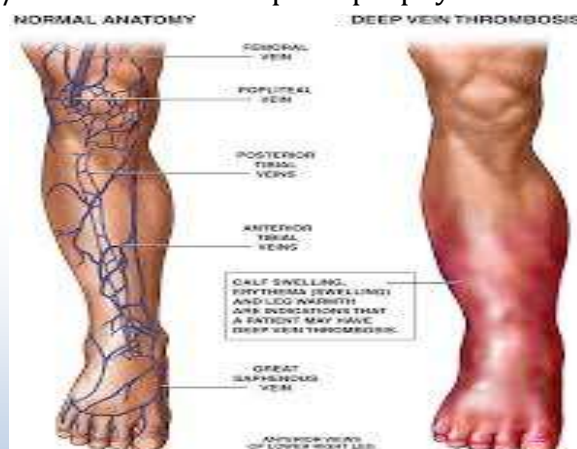


- bed sore → on bony prominences





- Pin site infection--Grade
 - Grade I
Pain (tenderness)
 - Grade II
All cardinal signs of inflammation present
 - Grade III
Serous discharge
 - Grade IV
Purulent discharge
 - Grade V
Bone involvement (osteomyelitis)
- When to act
 - Grade V
Remove
 - Grade IV + loose pin
Remove
 - Grade IV
Antibiotics
- Joint and muscle contracture
- Deep vein thrombosis, pulmonary embolism
 - UFH/un-fractionated heparin prophylaxis should be given



Patient follow-up after traction

- Follow by measuring the true length
→ Shortening up to 2cms may be acceptable
- For a significant shortening, add weight & for lengthening decrease the weight

3. External & internal fixation /operative treatment/

➤ Internal fixation

- Methods
 - Metal Plate held by screws/pins
 - Intramedullary nailing with or without cross screw fixation
 - Sites where it is commonly used
→ Femur, Tibia, Humerus
- Tension band wiring
 - wire tied over 2 kirschner wires or pins
 - Site
→ Patella, olecranon, Ankle

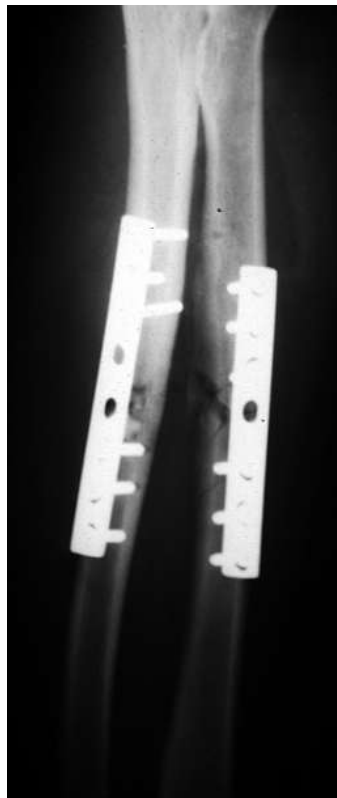


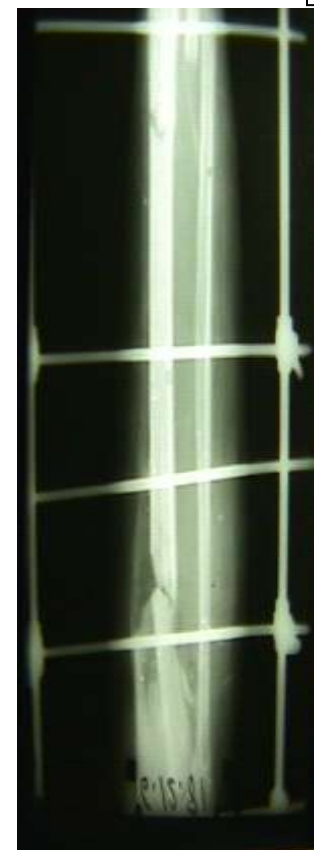
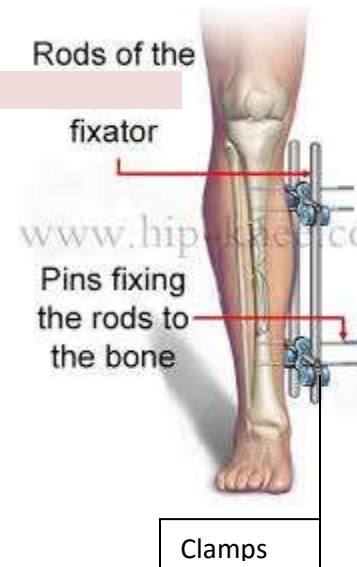
Plate & Screw



Intramedullary nailing

➤ External fixation

- For grade III open fractures (according to Gustilo and Anderson grading)
- External fixator should be applied 1.5-2cms away from the wound site
- Advantage
 - ✓ wound accessible for wound care
 - ✓ skin graft or flap rotations possible after wound healing
- Disadvantage
 - ✓ pin tract infection



Operative treatment /internal & external fixation

- ✓ Advantages
 - Anatomical reduction
 - Early immobilization & return of function
- ✓ Disadvantages
 - Expensive
 - Risk of infection

C. Rehabilitation

- Purpose
 - To preserve & restore function to normal as far as possible
- Should begin as soon as fracture comes under definitive treatment
- Physiotherapist supervision is necessary



Gustilo and Anderson Open Fractures classification

➤ Criterias for the classification

1. Wound size
2. Level of contamination
3. Soft tissue injury
4. Bone injury

Type	Wound size	Level of contamination	Soft tissue injury	Bone injury
I	<1cm long	Clean	Minimal	Simple, minimal comminution
II	>1cm long	Moderate	Moderate, some muscle damage	Moderate comminution
III				
A	Usually >10cm long	High	Severe with crushing	Usually comminuted; soft tissue coverage of bone possible
B	Usually >10cm long	High	Very severe loss of coverage; usually requires soft tissue reconstructive surgery	Bone coverage poor; variable, may be moderate to severe comminution
C	Usually >10cm long	High	Very severe loss of coverage plus vascular injury requiring repair; may require soft tissue reconstructive surgery	Bone coverage poor; variable, may be moderate to severe comminution

NB

Soft tissue management

- Local Management
 - ✓ Compound fracture
 - Treat the wound
 - Irrigation and Debridement
 - Soft tissue coverage
 - ✓ Elevate the injured limb
 - ✓ Rest the injured ligaments
- General Management
 - ✓ TAT
 - 3000IM stat—Adults
 - 1500IM stat—children
 - ✓ Analgesics
 - Based on WHO pain management ladder
 - ✓ Antibiotics



Chapter 12

Head Injury

Brain storming

1. Classify head injury based on GCs?
2. List the signs of basal skull fracture?
3. Describe the CT-scan findings for epidural hematoma, acute & chronic subdural hematoma?
4. List down the Cushing triads?
5. How you approach head injury patients?

Introduction

Monro-kellie doctrine states that the total volume of the intracranial contents must remain constant because the cranium is a rigid & non-expansile container.

Total volume=Brain tissue (80%) + CSF fluid (10%) + intravascular blood (10%)

Venous blood & CSF fluid may be displaced out of the container to provide pressure buffering (keeping the total volume constant). So early after injury patients may have normal intracranial pressure/ ICP, but once the limit of displacement reached ICP rapidly increases.

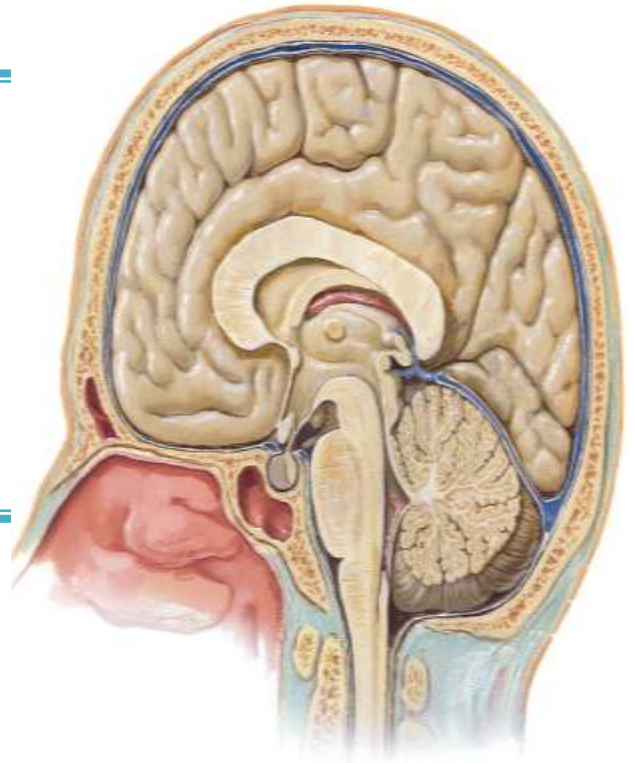
CPP/cerebral perfusion pressure is the pressure gradient required to perfuse the cerebral tissue.

CPP= Mean arterial pressure (MAP) – ICP

MAP = $\frac{1}{3}$ (Systolic BP) + $\frac{2}{3}$ (Diastolic BP)

Normal CPP range = 50 – 70 mmHg

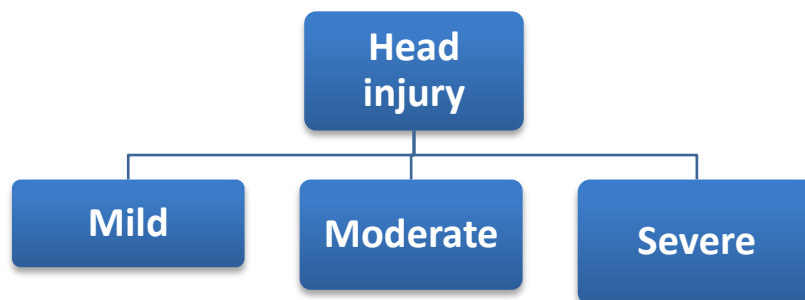
Normal ICP in adults' < 10-15mmHg



Classification of head injury

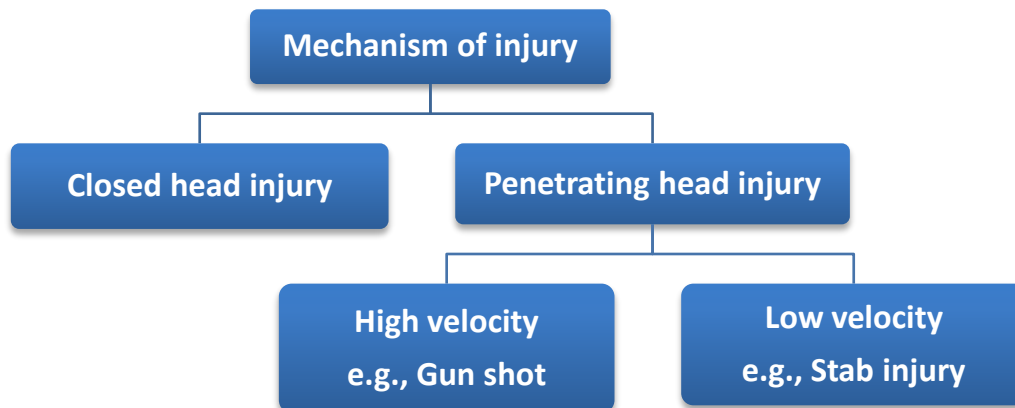


1. Based on GCS



- ☐ **GCS = 3-8**
Severe head injury
- ☐ **GCS = 9-12**
Moderate head injury
- ☐ **GCS = 13-15**
Mild head injury

2. Mechanism of injury

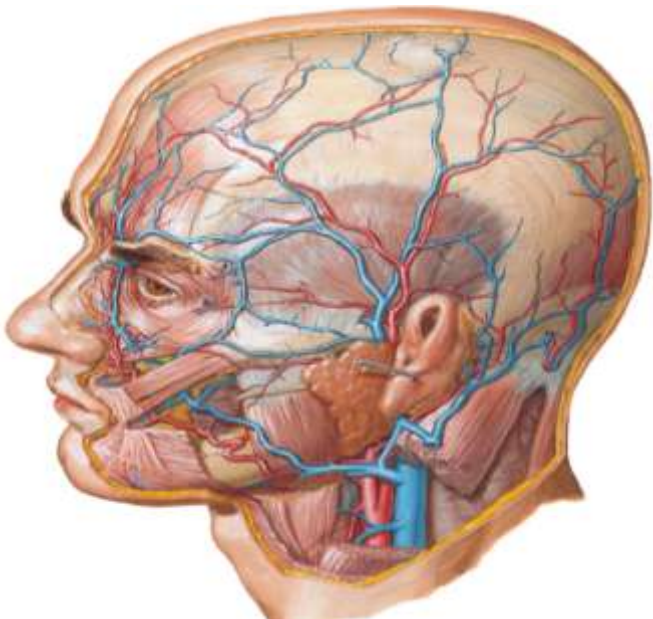


Head injury components

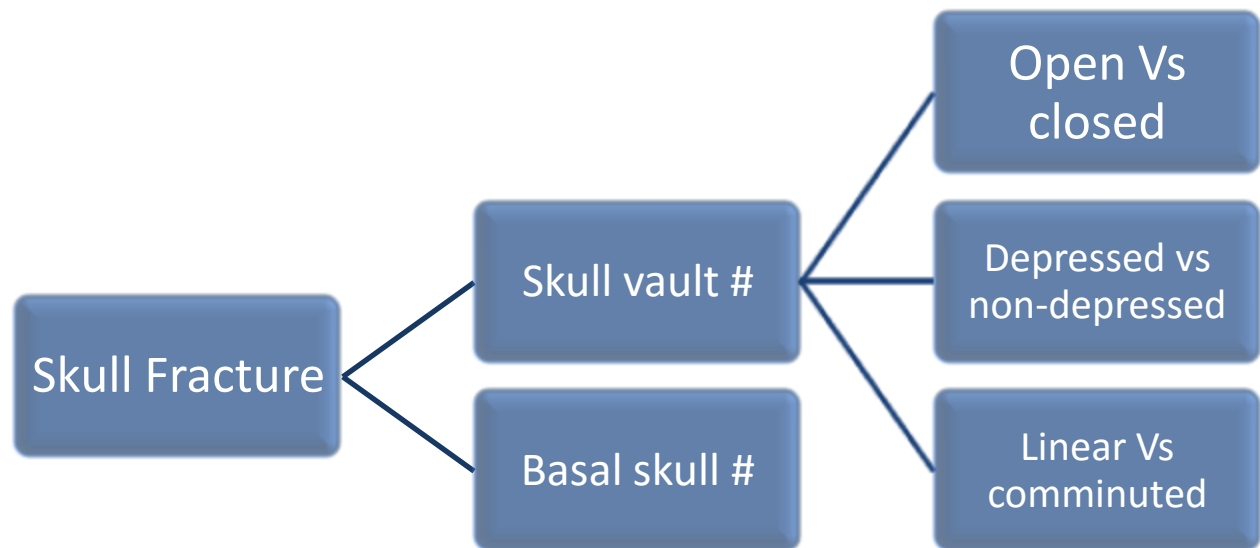
1. Scalp laceration
2. Bone fracture
3. Traumatic brain injury

1. Scalp laceration

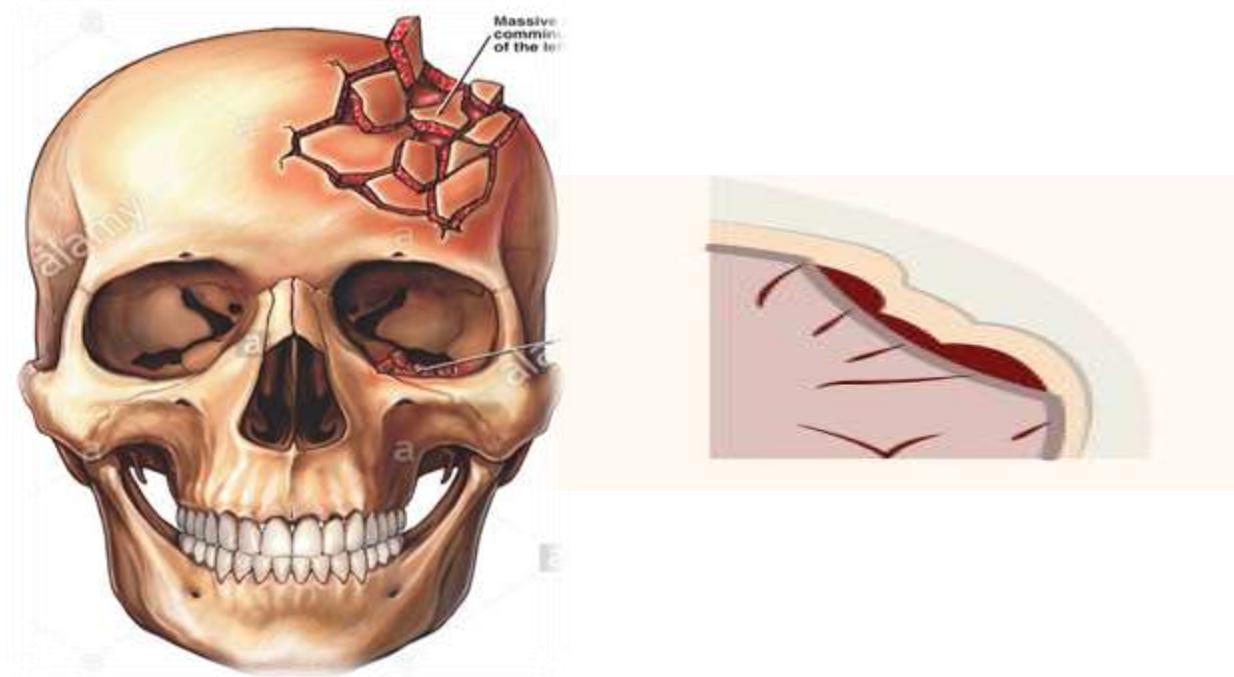
- ☐ Significant bleeding may occur since the scalp is highly vascularized
- ☐ Management
 - Apply direct pressure
 - Closely inspect the injury if laceration only
 - ✓ Laceration repair
 - ✓ Debridement & closure



2. Bone fracture/#



Skull vault fracture



- A closed fracture is covered by intact skin. An open or compound fracture is associated with disrupted overlying skin
- Simple or linear fractures do not need any treatment. But patients should be observed for 24 hours for possible intracranial bleeding
- Depressed skull fracture can cause brain or dural laceration and brain compression

- Depressed skull fracture is managed by elevation of bigger fragments and removal of smaller fragments
 - Indication for elevation
 - Neurologic deficit
 - Loss of consciousness
 - Seizure
 - Cosmetic areas



Basal skull fracture

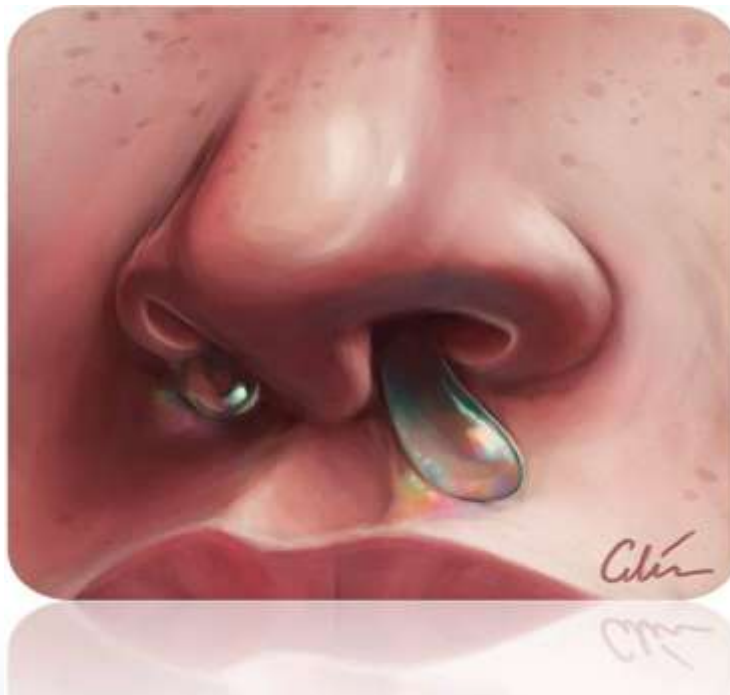
- **Otorrhea**

- CSF drainage from the ears
 - Copious clear drainage from the ears make the diagnosis obvious



- **Rhinorrhea**

- CSF drainage from the nostrils
 - Copious clear drainage from the nostrils make the diagnosis obvious



- **Battle sign**
 - Post-auricular/mastoid ecchymoses



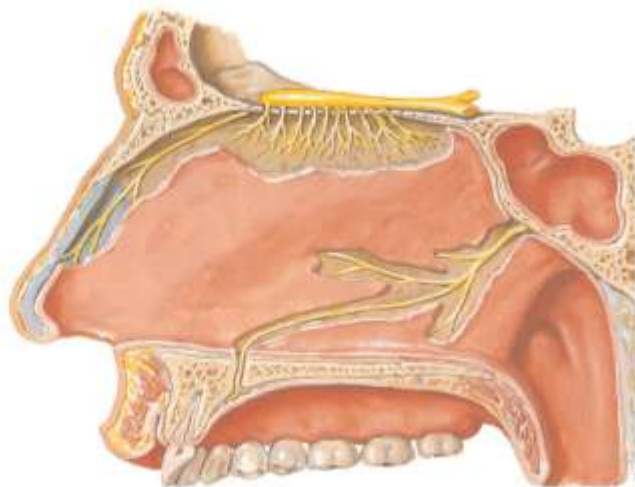
- **Raccoons eye**
 - Periorbital ecchymoses



- **Cranial nerve palsy**



- **Anosmia**



Basal skull fracture management

- CSF leak
 - ✓ Elevation of head off the bed for several days may heal it. In addition lumbar drain can augment this method. Lumbar drain allows the defect to heal by eliminating normal hydrostatic pressure.
- Traumatic cranial neuropathy
 - ✓ Facial nerve palsy
Give steroids. If no response after 48-72hours surgical decompression of the petrous portion of CN-VII may be considered

3. Traumatic brain injury/TBI

TBI

Primary

Secondary

- Primary traumatic brain injury occurs at the time of impact and includes injuries such as brainstem and hemispheric contusions, diffuse axonal injury & cortical lacerations
- Secondary brain injury occurs at some time after the moment of impact and is often preventable. The principle causes of secondary brain injury can be classified as extra-cranial & intracranial causes
 - Extra cranial
 - Hypoxia
 - Hypotension
 - Intracranial
 - Hematoma
 - Brain swelling(edema)
 - Infection

Closed head injury

Types

- Concussion
- Contusion
- Diffuse axonal injury

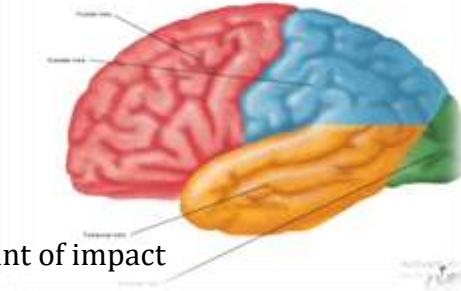
Concussion

- ☐ Defined as temporary neuronal dysfunction following non-penetrating head trauma.
- ☐ The head CT is normal, and deficits resolve over minutes to hours.

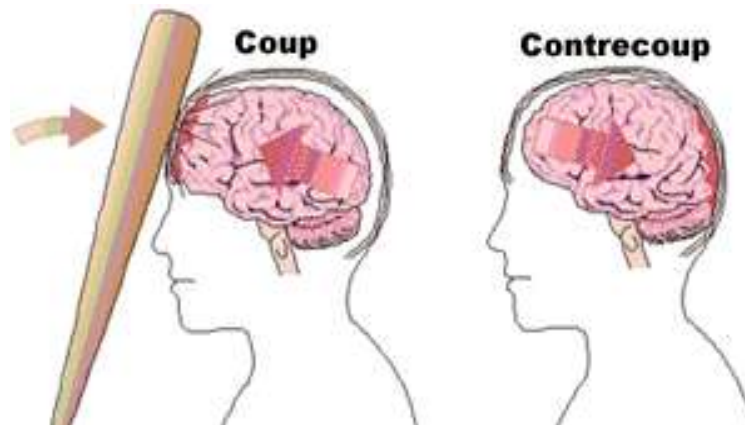


Contusion

- ☐ A bruise of the brain
- ☐ The contused areas appear bright on CT scan
- ☐ Frontal, occipital, temporal are common sites for contusion



- ☐ Coup
Contusion at the point of impact
- ☐ Counter coup
Contusions also may occur in brain tissue opposite the site of impact. This is known as a conter coup injury. These contusions result from deceleration of the brain against the skull.
- ☐ Contusion also occurs as the brain slides forwards and backwards over the ridged cranial fossa floor



Diffuse axonal injury

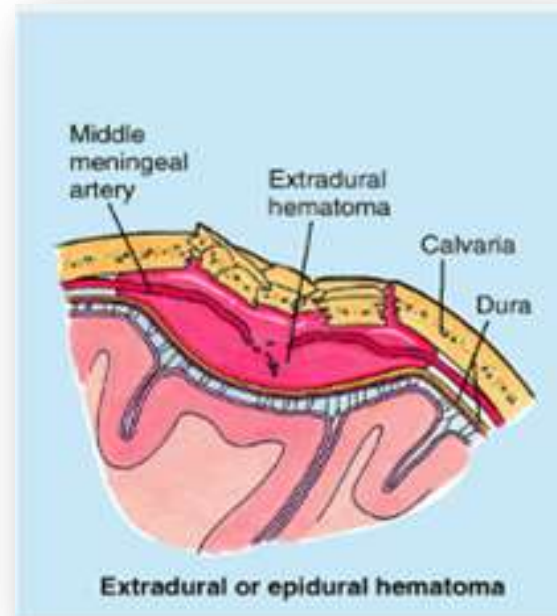
Diffuse axonal injury is caused by damage to axons throughout the brain, due to rotational acceleration and then deceleration.



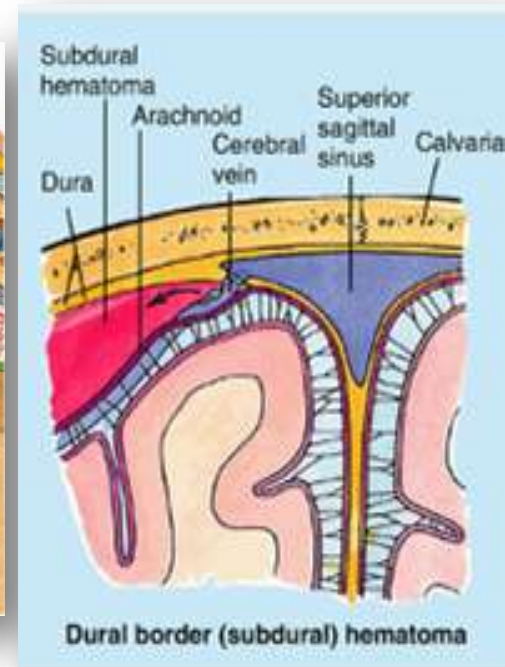
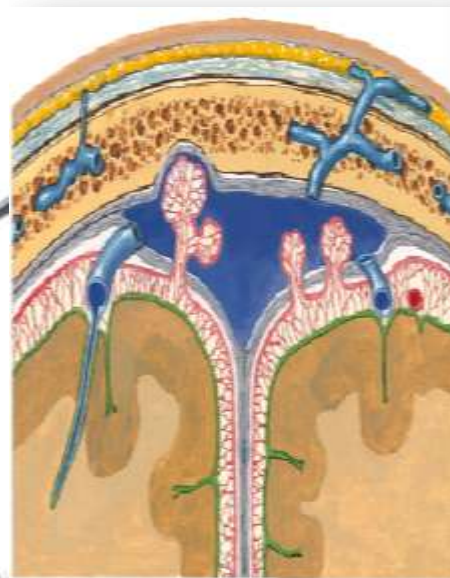
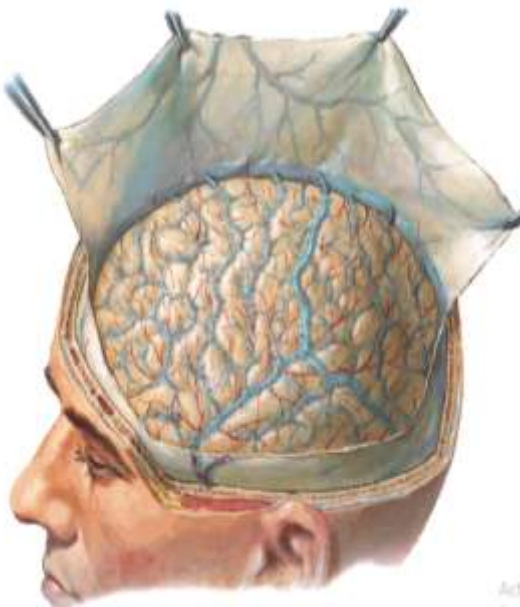
Traumatic intracranial hematoma

Intracranial hemorrhage can be classified as

1. Epidural hematoma



2. Subdural hemorrhage

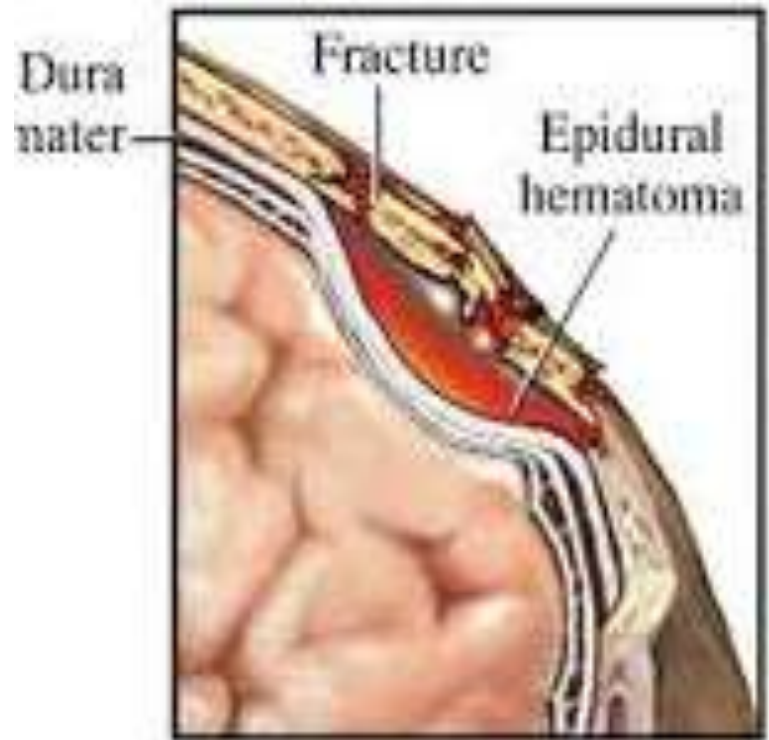


3. Subarachnoid hemorrhage

4. Intra-parenchymal/ intra-cerebral hemorrhage

Epidural hematoma/extradural hematoma

- Accumulation of blood between the skull and the dura
- EDH is common at temporal bone due to the pterion, the thinnest part of the skull, that overlies the largest meningeal artery.



Clinical presentation

Classic presentation

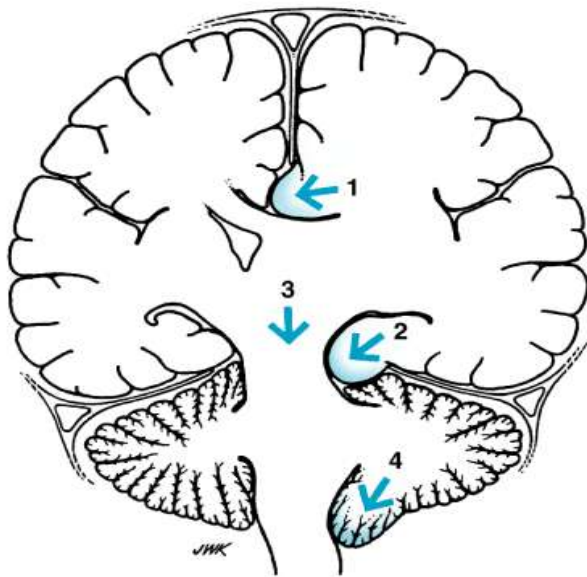
1. Loss of consciousness
Due to concussive effect of head trauma
2. The patient awakens & will have "lucid interval"
Due to subclinical hematoma expansion
3. Deterioration
As a result of brain compression & herniation
 - ✓ Contralateral hemiparesis
 - ✓ Reduced consciousness level and
 - ✓ Ipsilateral pupillary dilatation

NB

Only around 20% of patients present with the classic presentation

NB

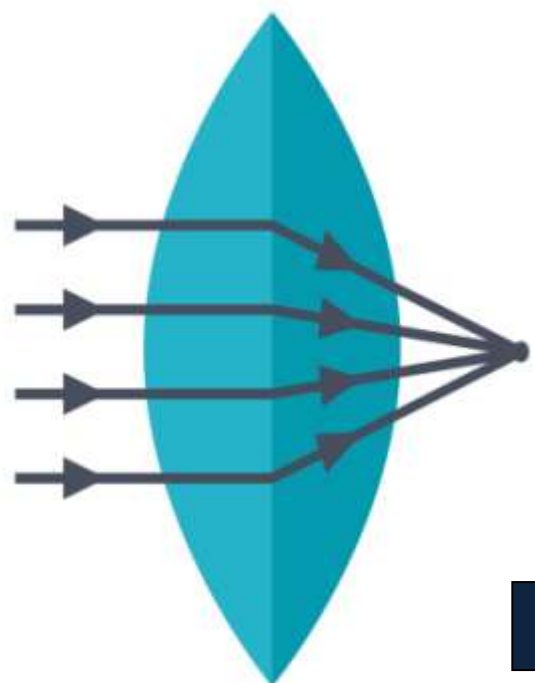
Brain herniation types



1. Subfalcine herniation
2. Uncal herniation
3. Transtentorial herniation
4. Tonsillar herniation

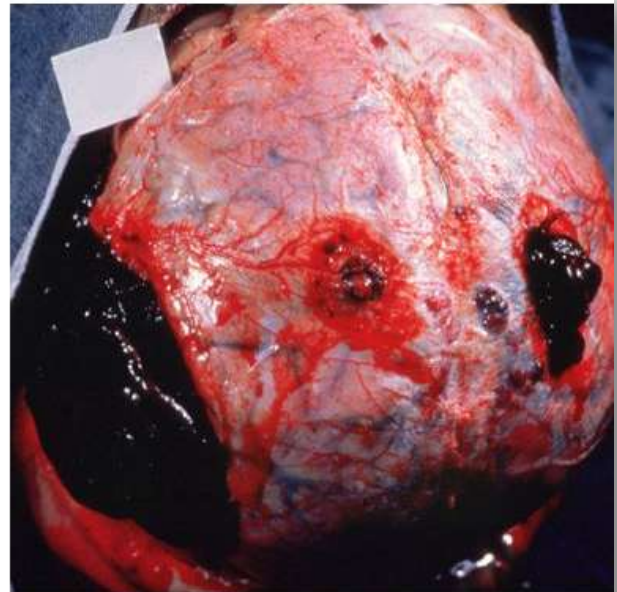
CT-scan of EDH

- Bright/hyperdense
- Biconvex in shape (lenticular), and has a well-defined border that usually respects cranial suture lines



Management of EDH

- Craniotomy--for evacuation of the congealed clot and hemostasis



Subdural hemorrhage

SDH is the accumulation of blood between the arachnoid membrane and the dura. It can be classified as;

- Acute SDH
- Chronic SDH

Acute SDH

- Usually results from venous bleeding, typically from tearing of a bridging vein running from the cerebral cortex to the dural sinuses



- Elderly and alcoholic patients are at higher risk for acute SDH formation after head trauma due to brain atrophy.
- Present with an impaired consciousness level from the time of injury, but further deterioration can occur as the hematoma expands

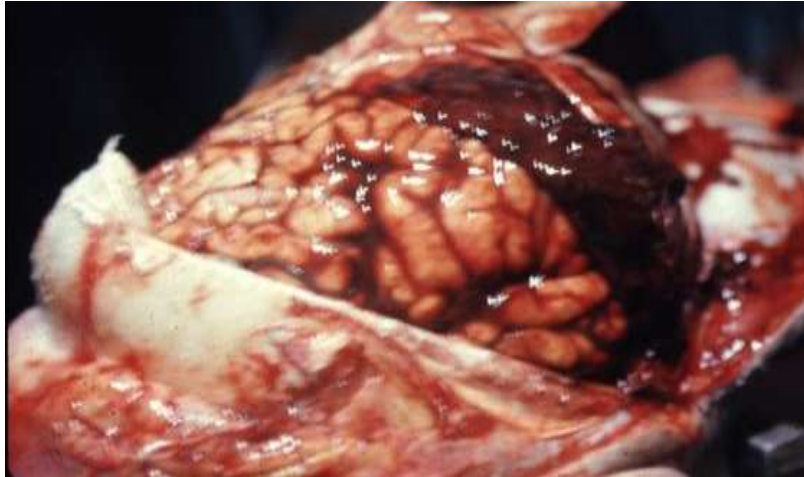
CT-Scan of acute SDH

- The clot is bright or mixed-density
- Crescent-shaped (lunate), may have a less distinct border, and does not cross the midline due to the presence of the falx



Management

- Open craniotomy for evacuation of acute SDH is indicated for any of the following:
→ Thickness >1 cm, midline shift >5 mm, or GCS drop by two or more points from the time of injury to hospitalization

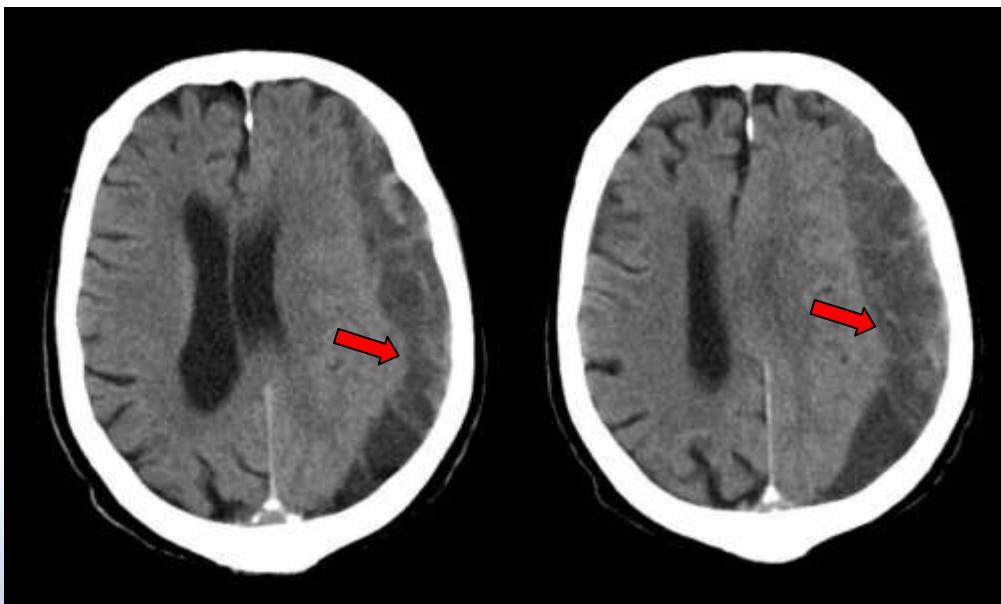


Chronic SDH

- Chronic SDH is a collection of blood breakdown products that is at least 2 to 3 weeks old.
- Alcoholics, elderly and patients on anticoagulation are at higher risk for developing chronic SDH
- Patients may present with headache, seizure, confusion, contralateral hemiparesis or coma
- Chronic SDHs often occur in patients without a clear history of head trauma, as they may arise from minor head injury

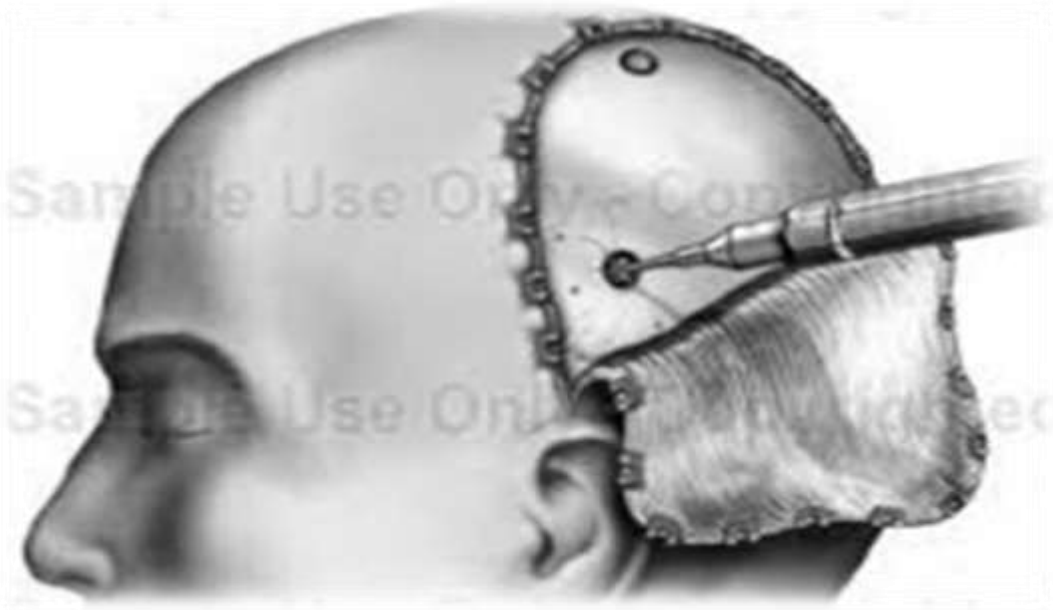
CT-Scan of chronic SDH

- Hypodense



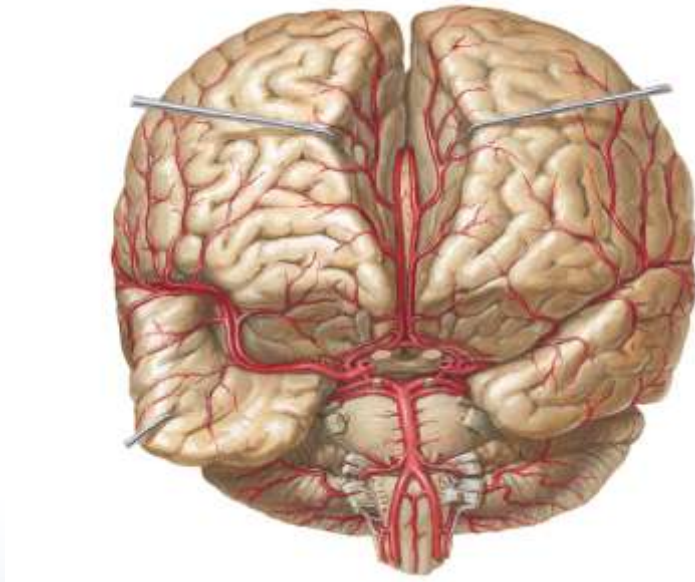
Management

- A chronic SDH >1 cm or any symptomatic SDH should be surgically drained
- Procedure--Burr hole



Intraparenchymal hemorrhage

Isolated hematomas within the brain parenchyma are most often associated with hypertensive hemorrhage or arteriovenous malformations. But traumatic intraparenchymal bleeding may occur in a contused area of brain.



- Delayed traumatic intracerebral hemorrhage is most likely to occur within the first 24 hours.
- Patients with contusion on the initial head CT scan should be reimaged after 24 hours.
- Indication for craniotomy
 - Any clot volume $>50\text{cm}^3$
 - Clot volume $>20\text{cm}^3$ with neurologic deterioration (GCS=6-8) & midline shift $>5\text{mm}$ or basal cistern compression

Head injury patient approach

❑ Primary survey

- Airway
- Breathing
- Circulation
- Disability
- Exposure

NB

Read in detail about primary survey on the previous chapter

❑ Secondary survey

- History
- Physical examination
- Investigation
- Definitive management

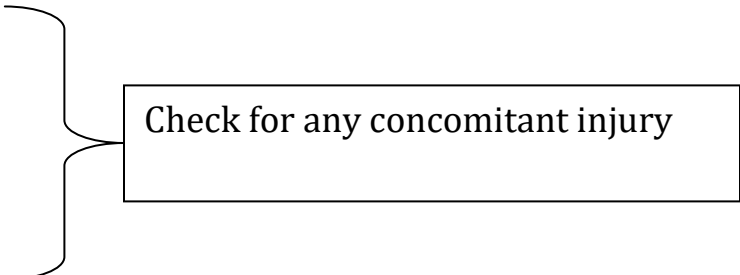


History of HI patients should include;

- Mechanism of injury
- History of loss of consciousness, abnormal body movement...
- Medication history
- Alcohol /illicit drug use
- Psychiatric problem

Physical examination should focus on;

- General appearance
- Vital signs
 - Pick any derangement
 - Have high index of suspicion for Cushing triad

- HEENT
 - Head
 - On inspection there may be evidence of external head injury such as subgaleal haematoma or scalp laceration which may be a cause of significant external blood loss.
 - Palpation of a scalp laceration may reveal an underlying skull fracture & depression
 - Look for clinical evidence of basal skull fracture
 - Head= Battle's sign
 - Ear= CSF otorrhea, haemotympanum, active bleeding
 - Eye= Raccoon eyes
 - Nose=CSF rhinorrhea
 - Chest examination
 - CVS examination
 - Abdominal examination
 - GUS examination
 - IGS examination
 - MSS examination
 - Nervous system examination
- Focus on
- GCS
 - Mini-mental status examination
 - Cranial nerve examination
 - Motor examination
- 
- Check for any concomitant injury

Sample history

Chief complaint

Axe injury of 5 hours duration

HPI

This Patient was last relatively healthy 5 hours back at which time she sustained axe attack to the head & distal forearm by a known offender, her psychiatric brother in law. She was attacked 02 times; one on the left side of the head and the other on the right distal part of the forearm. After the injury she lost her consciousness for 04hours. She was found falling on a cement floor by her mother. She had massive bleeding from the site of injury. They took her to local health center where they tried to stop the bleeding by dressing. She was given diclofenac 75 mg IM stat & TAT 3000 IU IM stat. Then she was referred to our hospital for better management. 01 hour prior to admission she gained her consciousness, while she was on the way to our hospital. She was totally unable to use her right upper extremity. In addition she experienced 02 episodes of projectile, bilious vomiting on her arrival to our hospital, associated with throbbing type of generalized headache which worsen during bending. The only thing she remembered about the accident was running & trying to escape from the attacker but no history of forgetfulness for events that happened before the accident or those events after she re-gained her consciousness. She has tinnitus, blurring of vision & light headedness.

- *No history of abnormal body movement, failure to control urine or feces*
- *No history of failure to use other extremities except the injured one*
- *No history of watery or bloody discharge from the ears or nostrils*
- *She has no history of breathing difficulty or chest pain*
- *No history of illicit drug use or alcohol abuse*
- *No self and family history of DM, Hypertension or asthma*
- *Not screened for RVI but has no history of chronic diarrhea, multiple sexual partner or herpes zoster attack.*

Finally she was admitted by an ambulance.

Investigation

Imaging

Skull x-ray (AP & lateral)

- Is there fracture?
 - If yes, is it depressed or not? If depressed, is it significant?
 - ✓ It is said to be significant when the depression is greater than the thickness of the skull bone.
- Pattern of fracture
 - Linear or comminuted?



CT-scan

-Summarized

Epidural hematoma	Acute SDH	Chronic SDH
Hyperdense (bright) lesion	Hyperdense (acute blood)	Hypodense
Lentiform (lens-shaped or biconvex)	Crescent shaped (lunate)	Crescent shaped (lunate)
Well defined border (between the skull and brain)	May have less distinct border	May have less distinct border
May or may not cross the midline	Doesn't cross midline	Doesn't cross midline



Laboratory

- Hct
- Blood group & Rh
- X-match

Definitive management

It may be observation, craniotomy or burr hole based on the underlying disease condition & indication.

Mild head injury

- ☐ Observation for 24 hours
- ☐ Criteria for discharge
 - ✓ Patient must have GCS=15/15 with no focal neurological deficit
 - ✓ verbal and written head injury advice must be given to the patient and patient's attendant
 - advice to return if the patient experiences persistent or worsening of headache despite analgesia, persistent vomiting, drowsiness, visual disturbance such as double or blurred vision, and development of weakness or numbness in the limbs.

Moderate to severe head injury

Aim

To prevent secondary brain injury

NB

The cervical spine must be immobilized!

Cerebral contusion

- ✓ Admit for observation since these lesions tend to mature and expand 48–72 hours following injury
- ✓ Rarely requires emergent evacuation
- ✓ Some patients may require delayed evacuation to reduce mass effect

Extradural (epidural) hematoma

- ✓ Immediate surgical evacuation via craniotomy & hemostasis

Subdural hematoma

- ✓ Acute SDH → Evacuation via craniotomy based on indications
- ✓ Chronic SDH → Evacuation via burr hole(s) based on indications

Intraparenchymal hemorrhage

- ✓ Reimage after 24hrs & craniotomy if indication met

Medical management

- Head elevation 30°
- Seizure control
- Normothermia
- Sedation +/- muscle relaxant
- Normocapnia
- Avoid obstruction of venous drainage from head--check the cervical collar
- Mannitol
- Serum electrolyte balance
- GI prophylaxis

Long term complications of head injury

- Post-concussional symptoms
 - headache, dizziness
 - impaired short-term memory and concentration
 - easy fatigability, emotional disinhibition and depression
- Seizures
- Delayed CSF leak

NB

Symptoms of raised ICP

- Projectile Vomiting
- Headache
 - Classically, morning headache which may awaken the patient from sleep, worsen by coughing/sneezing, bending. It progressively worsens over time.
- Seizure
- Dropping consciousness...

Signs of raised ICP

- Cushing's triad
 - BP
 - Increased systolic blood pressure, widened pulse pressure
 - PR
 - Bradycardia
 - RR
 - Abnormal respiratory pattern
- Lateralizing signs
- Papilledema (swelling of optic disc)
- CN VI palsy

Chapter 13

Esophageal cancer

Brain storming

1. List risk factors for esophageal cancer?
2. List DDx for dysphagia?



Introduction

- Squamous cell carcinoma/SCC accounts for the majority of esophageal cancer. Adenocarcinoma follows
- SCC is common in middle 1/3 of esophagus & adenocarcinoma is common in the lower 1/3.
- More common in men than women (3-4:1)
- Occurs at the age of 6th-7th decade

Risk factors

- Hereditary factors
- Cigarette smoking
- Chronic alcohol consumption
 - ≥ 30 gm/day for non-smokers and ≥ 15 gm/day for smokers
- High BMI, especially in those with central obesity(> 25 kg/m²)
- GERD and associated Barrett esophagus
- Diet
 - Drinking scalding hot liquids
 - Vitamin deficiency (e.g. Riboflavin)
 - Certain foods
E.g. betel quid and areca nuts
 - Zinc deficiency & low selenium level
- Underlying esophageal diseases like achalasia, caustic stricture
- Infectious conditions
 - HPV infection(serotype 16 & 18)
- Prior gastrectomy
- Atrophic gastritis
- Tylosis

Clinical presentation

History

- Dysphagia is the most common presentation
 - Initially for solid foods eventually progressing to include liquid food
- Weight loss (2nd most common symptom)
- Bleeding (from the tumor)
- Epigastric or retrosternal pain
- Bone pain(with metastasis)
- Hoarseness of voice(with recurrent laryngeal nerve infiltration)
- Respiratory symptoms(due to invasion or aspiration)
 - persistent cough ...

Physical examination

Typically esophageal cancer patients won't have findings unless it has metastasized

- General appearance
Chronically sick looking (cachexic)
- HEENT
Look for signs of anemia and liver involvement/ metastasis
- LGs
Lymphadenopathy in the lateral cervical or supraclavicular areas (especially look for Virchow's node)
- Chest
Malignant pleural effusion in case of metastasis
- CVS
 - Try to appreciate ejection systolic murmur & gallop in case of high output heart failure due to severe anemia
- Abdominal examination
 - Try to appreciate hepatomegaly (in case liver metastasis). If any characterize the mass
 - Elicit signs of abdominal fluid collection /dullness, fluid shift & fluid thrill

DDx

- **Esophageal Stricture**
 - majority of the strictures result from long-standing gastroesophageal reflux
 - treatment usually involves dilation combined with acid-suppressive therapy
- **Benign esophageal tumors**
 - Esophageal leiomyoma (accounts for $\geq 50\%$)
- **Achalasia**
 - Characterized by
 - Aperistalsis
 - Partial or incomplete relaxation of the lower esophageal sphincter/LES
 - Increased resting tone of the LES
 - Pathogenesis
 - 1^o achalasia--poorly understood

- 2^o achalasia-- chagas disease → trypanosome cruzi by distracting of myoenteric plexuses
 - Other causes
 - DM, polio, sarcoidosis, amyloidosis, malignancy and caustic stricture
- Clinical feature of achalasia
 - Similar to that of SCC of the esophagus
 - Sensation of food sticking in the lower esophagus
 - Nocturnal cough
 - Retrosternal Chest pain induced by eating
- **Gastric cancer**
 - Read more on gastric cancer chapter 3/ GOO causes
- **Esophageal perforation**
 - Most esophageal perforations are iatrogenic (59 %)
 - Causes of non-iatrogenic esophageal perforation is
 - ✓ spontaneous rupture 15 % (Most common cause)
 - ✓ foreign body ingestion 12 %
 - ✓ trauma 9% and
 - ✓ malignancy

Sample history

Chief complaint

Difficulty of swallowing of 6 months duration

HPI

This patient was last relatively healthy six months back at which time he began to experience difficulty of swallowing initially for solid foods like 'injera' and bread but latter progressed to liquid foods with dull aching type of retrosternal pain. He has unquantified weight loss to the extent his trousers became loose & easy fatigability at minimal exertion. Associated with this he has blurring of vision, tinnitus and light headedness. Since 6 years back he had the retrosternal pain and burning epigastric pain for which he repeatedly took omeprazole p.o. at local health center & private clinic. But he got no improvement in his symptoms. He has history of alcohol intake about 05 bottles of beer per day for the past 25 years but no history of smoking.

- ❖ He was screened for RVI and found to be NR*
- ❖ He has usually eats "Injera" made of "Teff" with "Shiro wot."*
- ❖ He has no history of radiation therapy to the neck or chest.*
- ❖ He has no history of multiple sexual partners*
- ❖ He has no history of drug use other than the medication mentioned above*
- ❖ He has no history of previous surgery*
- ❖ He has no history of similar illness in the family*
- ❖ He has no history of change in voice*
- ❖ He has no history of cough, dyspnea or hemoptysis.*
- ❖ He has no self or family history of DM, Hypertension or Asthma*

Finally he was admitted.....

Investigation

- **UGI endoscopy**
To visualize growth and take **biopsy** *
- **Ultrasound**
Endoscopic ultrasound is the most sensitive for determining
 - Depth of penetration (for T staging/TNM)
 - Presence of enlarged peri-esophageal lymphnodes (for N staging/TNM)
- **Barium swallow**
 - Very sensitive for detecting strictures and intraluminal mass
 - Detect mucosal irregularities and filling defects
 - Helps to study the distal anatomy in obstructing tumors that are not accessible for endoscopy
 - Draw backs
Does not allow staging or biopsy
- **Bronchoscopy**
 - To exclude invasion of trachea or bronchus (for cancer of the upper & middle 1/3 of esophagus)
- **PET scan**
 - Detects occult distant lymph node metastasis & bone spread
- **Esophageal Monometry**
 - Measures the motility and function of the esophagus and esophageal sphincter
- **Chest x-ray**
 - To look for mediastinal widening or posterior tracheal indentation
 - To rule out aspiration pneumonia
- **Laboratory studies**
 - CBC, Serum electrolyte, OFT
- **Metastasis workup**

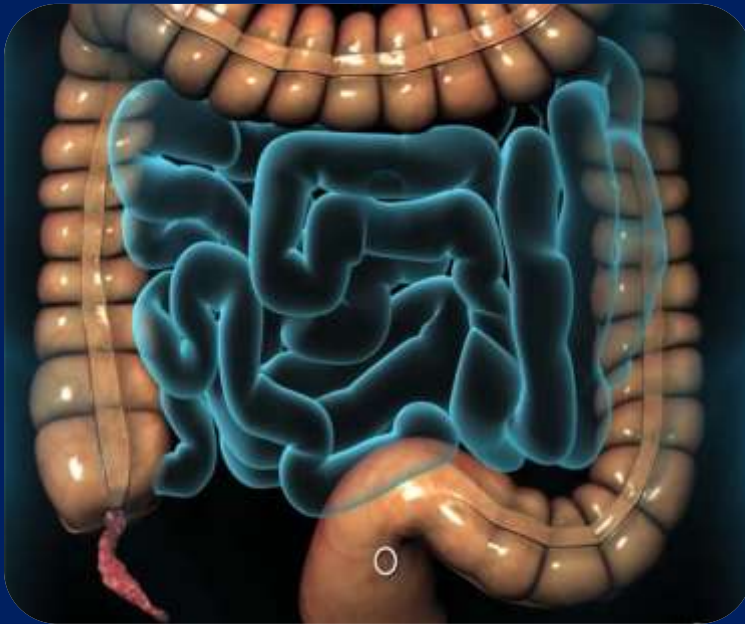
Management

Treatment plan depends on the following

- Staging of the cancer
- Location of the cancer in the esophagus
- General health of the patient

Treatment options

- Endoscopic mucosal resection
- Surgery
 - Transhiatal, Right thoracotomy (Ivor-Lewis), left thoracotomy, Radical-En-Block
- Chemotherapy
- Radiotherapy
- Combination of the above
- Palliative therapy
- Laser phototherapy
- Stent



Chapter 14

Appendicitis & Appendiceal mass

Brain storming

A) A 23years old female patient presented with RLQ abdominal pain of 4hours duration

1. What additional information you want to ask the patient?
2. What physical examination findings you expect?
3. How you would like to investigate the patient?

B) What are the components of alvarado score?



Approach to patients with acute abdomen (General)

Introduction

- ❑ The term acute abdomen refers to signs and symptoms of abdominal pain and tenderness, a clinical presentation that often requires emergency surgical therapy.
- ❑ Characterized by severe, sudden onset, acute, short duration usually within 24hrs
- ❑ Many diseases, some of which are not surgical, can produce acute abdominal pain and tenderness. Therefore, every attempt is made to make a correct diagnosis so that the chosen therapy, Surgical or medical, is appropriate.
- ❑ Most surgical disease associated with an acute abdomen result from infection, obstruction, ischemia or perforation.
- ❑ Visceral abdominal pain
Vague and poorly localized pain due to visceral afferent nerves to the underlying viscera, usually the pain is due to distension of a hollow viscus.
- ❑ Parietal abdominal pain
Sharper and better localized pain due to somatic nerve supply to the parietal peritoneum
- ❑ Referred pain
It is pain perceived at a site distant from the source of stimulus. For example, irritation of the diaphragm may produce pain in the shoulder

History taking in acute abdomen

Questions must be open-ended whenever possible and structured to disclose the onset, character, location, duration, radiation, and chronology of the pain experienced

Pain characterization/OPQR²S²

- ❑ Onset(1)
- ❑ Pattern(3)
- ❑ Quality(4)
- ❑ Radiation(6)
- ❑ Relieving & Aggravate factors(7)
- ❑ Severity(2)
- ❑ Site(5)

E.g. Sudden onset(1) severe(2) intermittent(3) colicky(4) RUQ(5) pain of 4 hours duration with radiation to the right upper back(6) which is aggravated by taking fatty meals & relieved by....(7)

Onset & duration

- Sudden onset Vs gradual onset
- Duration → lapsed time before presentation

Pattern

- Constant Vs intermittent

Quality

- Crampy, Colicky, Gnawing...

Radiation

- Is the pain in one place? Or does it move around your body?

DDx -- Radiation
Right Shoulder
Liver Gallbladder Right hemi-diaphragm
Left Shoulder
Heart Tail of pancreas Spleen Left hemi-diaphragm
Scrotum and Testicles
Ureter

Relieving & aggravating factors

- What makes the pain worse? What makes the pain better?

For e.g. ,	Pancreatitis	PUD	Acute cholecystitis	Appendicitis	Ureteral colic
Aggravating factors	-Lying supine	-Hunger-DU -Food-GU	-Deep breathing -Fatty meal	-Movement -Cough	!
Relieving factors	-Leaning forward	-Food-DU -Vomiting-GU -Antacid		-Lying quietly	!

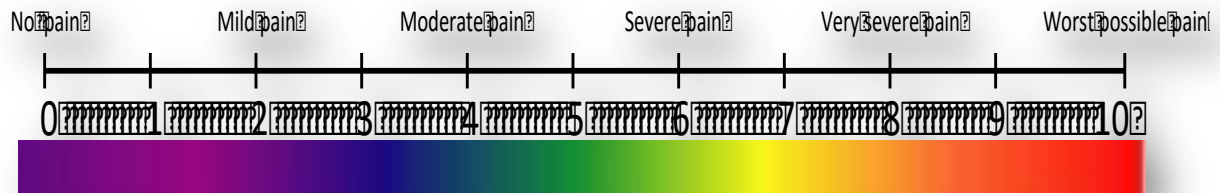
Site

→Where is your pain?

Severity

Mild/moderate/severe/very severe/worst possible pain...

- On a scale of 0-10, how bad is your pain?



After characterizing of the chief complaint ...

- List the associated symptoms, with appropriate characterization of each
 - E.g., Vomiting -- Describe about mode of onset, timing, content of vomitus, projectile or not, amount, frequency...
- Dig for risk factors, DDx & complications through your HPI
- Past medical history
- Gynecologic history

Physical examination in acute abdomen

General Appearance

- Acutely sick looking or not?
- Quietly lying down or Agitated?
 - ✓ If there is peritoneal irritation the patient will experience worsening in pain with any activity that moves or stretches the peritoneum. These patients typically lie in the bed quietly and often maintain flexion of their knees and hips to reduce tension on the anterior abdominal wall.
 - ✓ In case of ureteral colic, biliary colic & ischemic bowel disease the patients continually shift/writhe and fidget in bed while trying to find a position that lessens their discomfort

Vital signs

Look for any derangement—vital signs are vital all the time as the name implies!

- BP, PR, RR, T⁰
- PSO₂, UoP

Abdominal examination

Inspection

- Proper positioning of the patient
Supine position of the patient on a couch with proper exposure of the abdomen,
- Look for symmetry of the abdomen and flank fullness by standing in front of the patient
- Look for shape of the abdomen. Is it flat, scaphoid or distended?

- Look for contour of the umbilicus
Normally the umbilicus is slightly retracted, inverted with horizontal slit
- Look for abdominal movements with respiration
Normally there is gentle rise in the abdominal wall during inspiration and fall during expiration.
Markedly diminished or absent abdominal movement (silent abdomen) signify generalized peritonitis
- Look for visible peristalsis
Vigorous peristalsis of the abdomen signify obstruction of the pylorous (GOO) and intestinal obstruction
- Look for scars and striae
- Look for hernia sites
Support and lift the patient's head with the shoulder with your left arm and let him cough repeatedly while observing sites of hernia
- Look for prominent superficial veins

Auscultation

Place the stethoscope just to the right of the umbilicus, and listen to low-medium pitched gurgles interposed by high pitched noise or tinkle

- Normal sounds consist of clicks and gurgles with a frequency of 5-30 per minute
- Increased frequency of bowel sounds occur in diarrhea and mechanical intestinal obstruction
- Reduced or absent bowel sounds occur in paralytic ileus and generalized peritonitis

Palpation

Superficial palpation

- Ask the patient if there is any abdominal area which is tender? If there is tender abdominal area, start superficial palpation far away from the tender site and palpate the tender site at last. If there is no tenderness at any site, start at LLQ and move quadrant-by-quadrant in clockwise or anti-clockwise rotation

- Mould your relaxed and warm hand to the abdominal wall, not to hold it rigid and avoid sudden poking with the finger tips
- Gently palpate the abdomen quadrant by quadrant for abdominal tenderness, superficially palpable abdominal masses/organs and abdominal resistance while looking at the patient's face

Deep palpation

- To delineate abdominal mass or presence of organomegaly (enlarged liver or spleen)
- ***To check for guarding, rigidity and rebound tenderness
 - Presence of guarding, rigidity and rebound tenderness indicate generalized peritonitis
 - **Guarding:** involuntary reflex contraction of abdominal wall muscles overlying an inflamed viscus; classically seen in uncomplicated acute appendicitis
 - **Rigidity:** involuntary reflex rigidity of abdominal wall muscles; board-like rigidity is seen in diffuse peritonitis
 - **Rebound tenderness:** Deeply and slowly palpate the abdomen, and check for the presence of sudden pain while the examiner releases his hand from the abdomen

Percussion

- ✓ Dullness
- ✓ Tympanic
 - *The normal abdomen is tympanic
- ✓ Hypertympanic

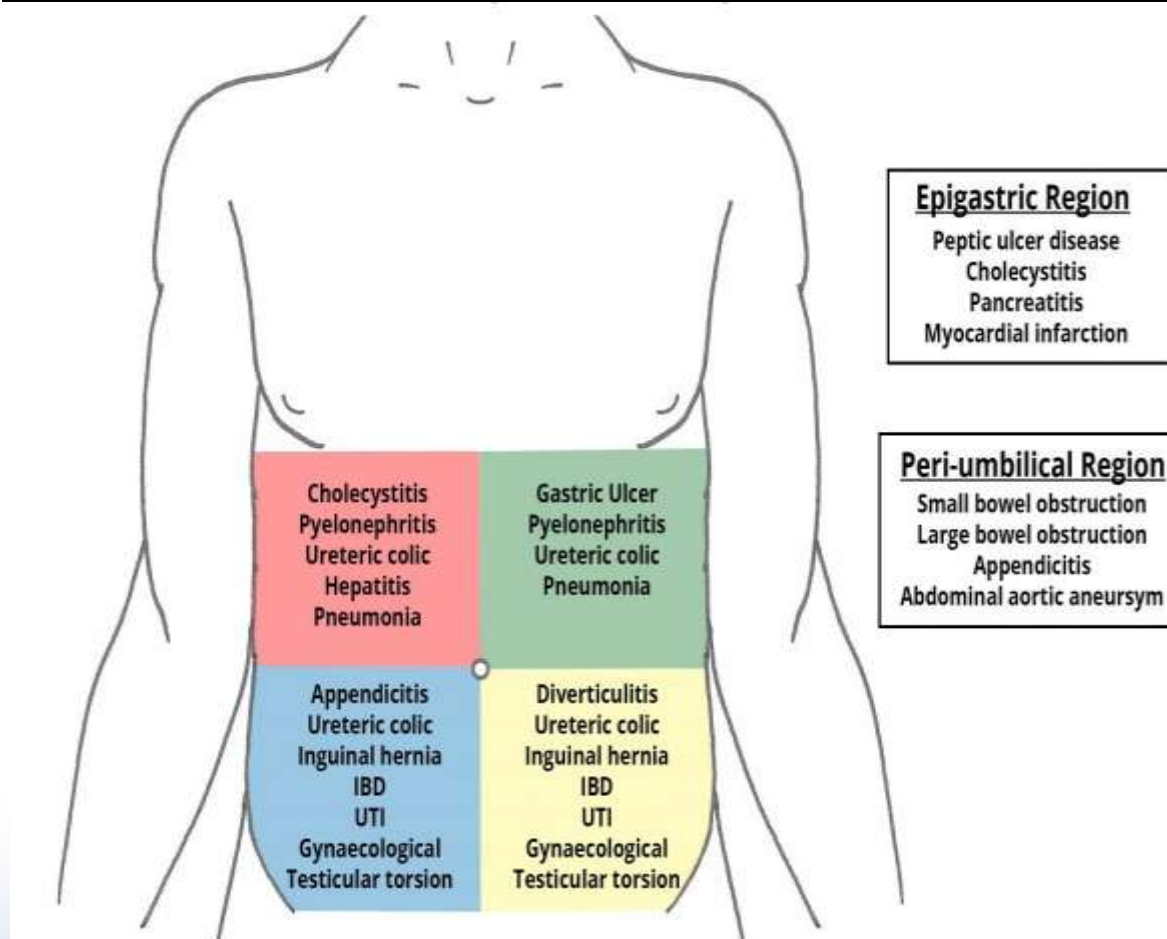
5Fs* for abdominal distension

- ✓ Fluid, Feces, Flatus, Fetus, Fat

Table showing some abdominal findings based on the cause of distension

	Intestinal obstruction	Gross ascites	Large ovarian cyst
Inspection	-Distended	-Distended -Flank fullness -Umbilicus everted	-Distended -Umbilicus vertical and drawn up
Auscultation	Increased and noisy bowel sound initially		
Palpation			Swelling arising out of the pelvis. You can't get below
Percussion	Hypertympanic to percussion	-Dullness in flanks -Shifting dullness and fluid thrill present	-Resonant in flanks -Dullness centrally

DDx for abdominal pain



Investigation for acute abdomen

- ☐ Laboratory studies
- ☐ Imaging studies
- ☐ Diagnostic laparoscopy

Laboratory

- CBC
- Urine HCG
- Urinalysis
- Stool examination for ova and parasites
- Serum electrolytes
- RFT
- Amylase, lipase
- Total and direct bilirubin
- Alkaline phosphatase

Imaging

- Abdominal ultrasonography
- Plain radiographs
- CT-scan

Diagnostic laparoscopy

Discussion on appendicitis (one of the cause of acute abdomen)

Appendicitis

Introduction

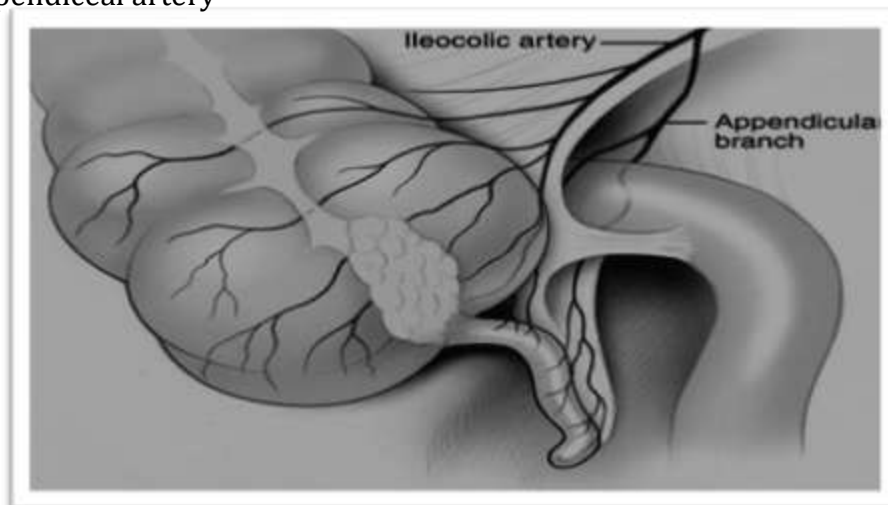
- ❑ Appendicitis is an inflammation of the vestigial vermiform appendix
- ❑ It is one of the most common causes of the acute abdomen and one of the most frequent indications for an emergent abdominal surgical procedure worldwide

Anatomy of appendix

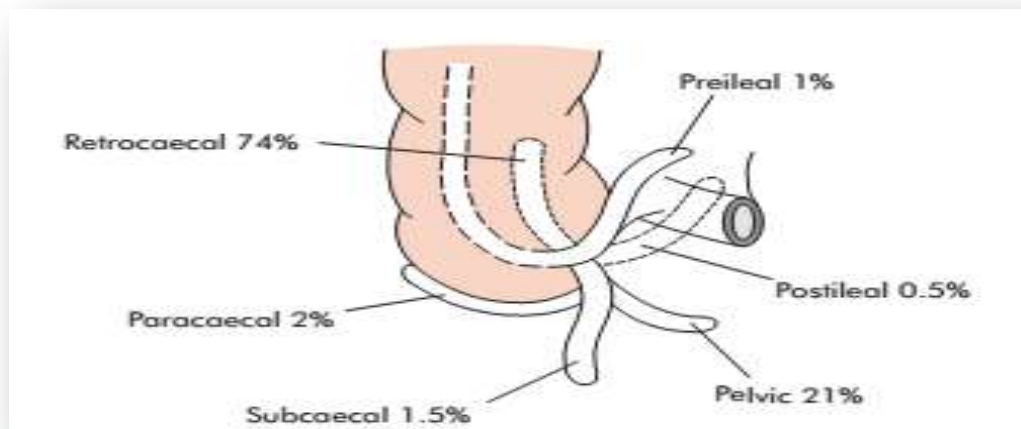
In the adult, the average length of the appendix is 6-9 cm; however, it can vary in length from <1 to >30 cm.

The outer diameter varies between 3 and 8 mm, whereas the luminal diameter varies between 1 and 3 mm.

Appendix gets its blood supply from superior mesenteric artery → Ileocolic artery → Appendiceal artery



Different position of appendix



The most common location is retrocaecal. The varying location of the tip of the appendix likely explains the myriad of symptoms that are attributable to the inflamed appendix.

Pathogenesis

- Appendiceal obstruction has been proposed as the primary cause of appendicitis
- Appendiceal obstruction may be caused by fecaliths (hard fecal masses(common)), calculi, lymphoid hyperplasia, infectious processes, and benign or malignant tumors
- Obstruction → Increase in luminal and intramural pressure → Distension → the distension will stimulate the visceral afferent nerve fibers entering the spinal cord at T8-T10, leading to vague central or periumbilical abdominal pain → Well-localized pain occurs later in the course when inflammation involves the adjacent parietal peritoneum.
- The distension also results in thrombosis and occlusion of the small vessels in the appendiceal wall and stasis of lymphatic flow. Bacterial overgrowth also occurs within the diseased appendix.

Clinical presentation

History

- Abdominal pain
 - ✓ Starts with periumbilical and diffuse pain that eventually localizes (shifts) to the right lower quadrant
 - ✓ Use OPQR²S² to characterize the pain
- Associated symptoms
 - ✓ Anorexia, nausea, vomiting, fever

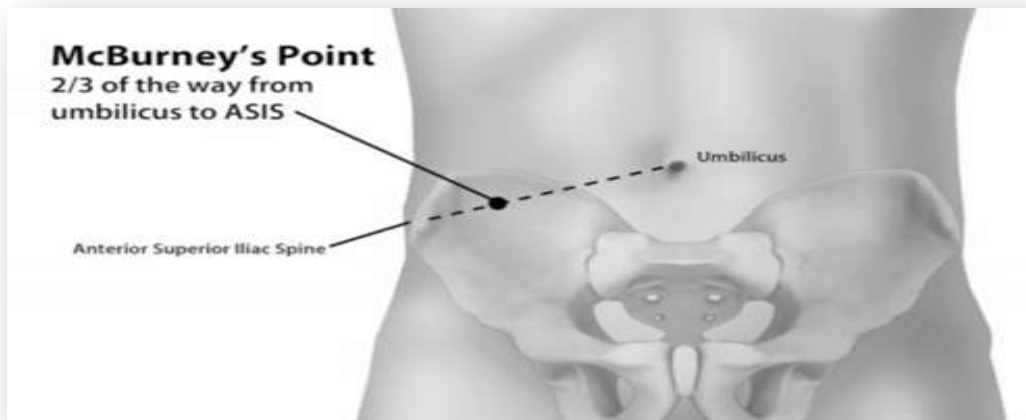


Physical examination

- General appearance
 - ✓ The patients move slowly and prefer to lie supine due to the peritoneal irritation
- Vital sign
 - ✓ Fever, ↑PR...

- Abdominal examination
 1. Tenderness (direct & rebound) at McBurney's point
 2. Rovsing's sign/indirect tenderness
 3. Psoas sign
 4. Obturator sign

1. Tenderness (direct & rebound) at McBurney's point



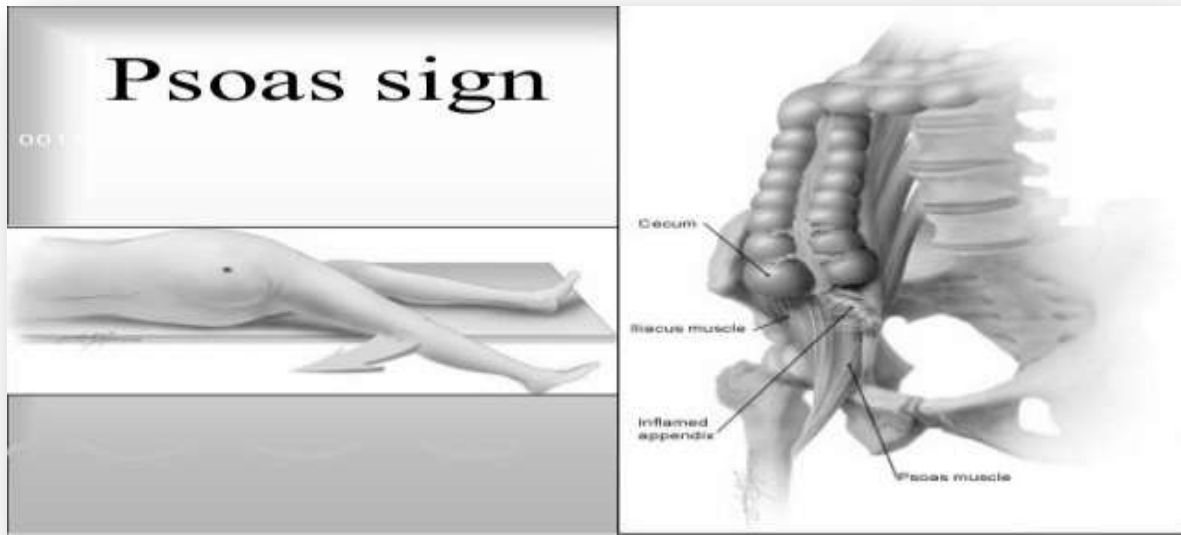
- McBurney's point tenderness is described as maximal tenderness at 1.5 to 2 inches from the anterior superior iliac spine (ASIS) on a straight line from the ASIS to the umbilicus (or around 1/3 from ASIS & 2/3 from umbilicus)

2. Rovsing's sign/indirect tenderness



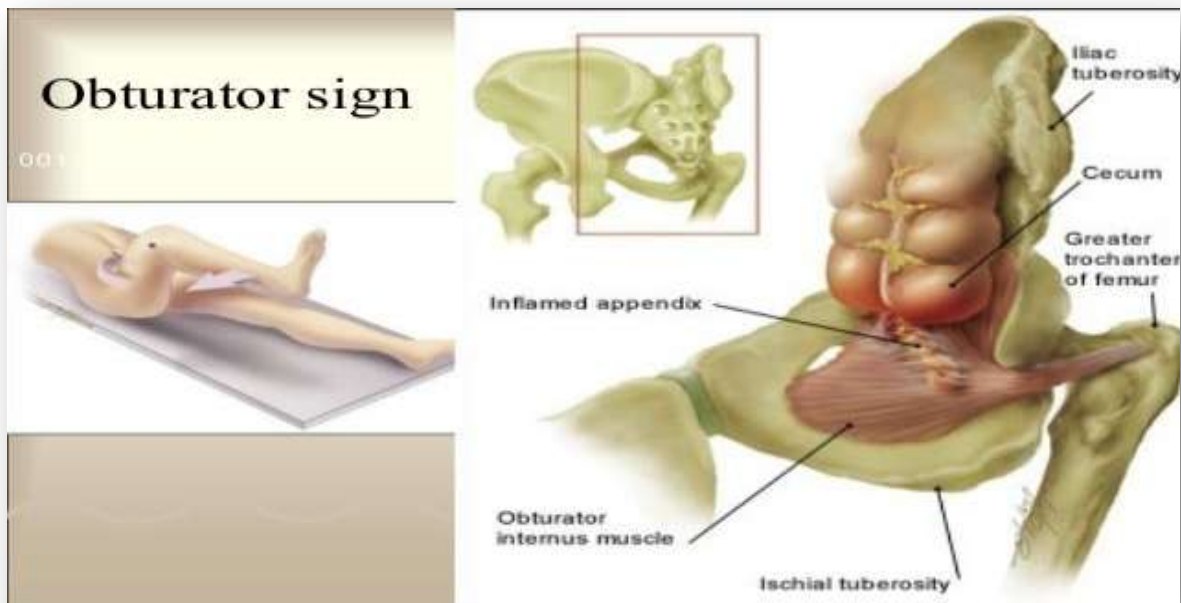
Rovsing's sign refers to pain in the right lower quadrant with palpation of the left lower quadrant. This sign is also called indirect tenderness and is indicative of right-sided local peritoneal irritation

3. Psoas sign



The psoas sign is associated with a retrocecal appendix. This is manifested by right lower quadrant pain with passive right hip extension. The inflamed appendix may lie against the right psoas muscle, causing the patient to shorten the muscle by drawing up the right knee. Passive extension of the iliopsoas muscle with hip extension causes right lower quadrant pain.

4. Obturator sign



The obturator sign is associated with a pelvic appendix. This test is based on the principle that the inflamed appendix may lay against the right obturator internus muscle. When the clinician flexes the patient's right hip and knee followed by internal rotation of the right hip, this elicits right lower quadrant pain

Complication of acute appendicitis

- Perforation (perforated appendicitis)
- Appendiceal abscess– due to late presentation

Investigations

Laboratory

- CBC
 - ✓ ↑WBC with left side shift
- Urine HCG
 - ✓ To rule out gynecologic causes of acute abdomen like ectopic pregnancy
- Urine analysis
 - ✓ Pylonephritis, DKA...

Imaging

- Abdominal ultrasound
 - ✓ Sonographic findings consistent with acute appendicitis include an appendix of 7 mm or more in anteroposterior diameter, a thick-walled, noncompressible luminal structure seen in cross section referred to as a target lesion, or the presence of an appendicolith.
 - ✓ Reliable in confirmation but not in excluding the diagnosis.



- CT-Scan

Clinical scoring --The modified alvarado score

"MANTREL"

Symptoms	Score
Migratory abdominal pain	1
Anorexia	1
Nausea and vomiting	1
Signs	
Tenderness (Direct)	2
Rebound tenderness	1
Elevated temperature	1
Laboratory	
Leucocytosis	2
Total	9

A management guide based upon the total point includes:

- 0 - 3
 - Considered to have a low risk of appendicitis
 - Should be discharged with advice to return if there was no improvement in symptoms
- 4 - 6
 - Should be admitted for observation and re-examination
 - If the score remains the same after 12 hours, operative intervention is recommended.
- 7 - 9
 - ✓ A male patient should proceed to appendectomy.
 - ✓ A female patient who is not pregnant should undergo diagnostic laparoscopy, then appendectomy if indicated by the intraoperative findings.

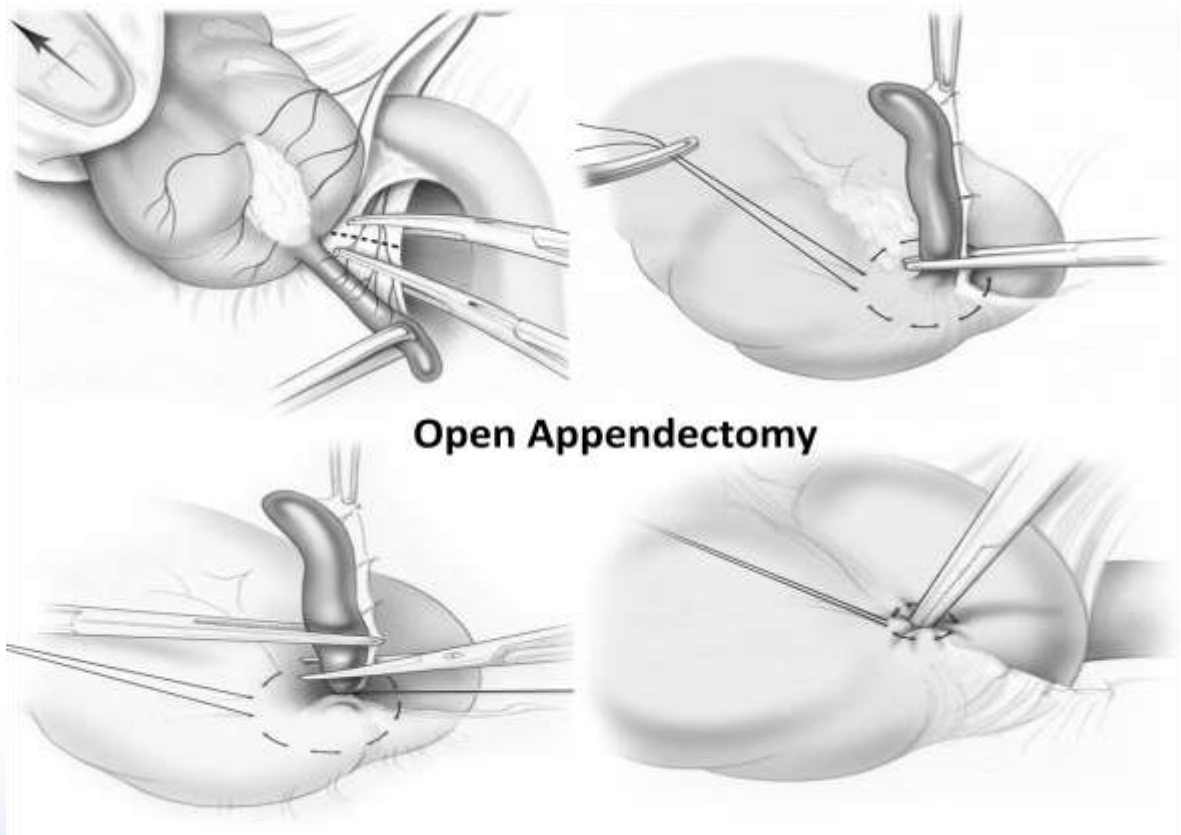
Management

❑ Pre-op

- Adequate hydration with IV fluids, NPO
- Prophylactic Antibiotics
 - ✓ Aim
To preventing wound infection and intra-abdominal abscess following appendectomy
 - ✓ Time
It should be administered within a 60-minute "window" before the initial incision
 - ❑ Non-perforated appendicitis
 - a single preoperative antibiotic dose → adequate
 - ❑ Perforated appendicitis
 - Regimen → start empiric broad-spectrum antibiotic therapy with activity against gram-negative rods and anaerobic organisms pending culture results
- Prepare OR material
- Take informed consent

❑ Operative

- Appendectomy (Open Vs Laparoscopy)



❑ Post op

- Post op follow-up (vital signs including UoP, abdominal condition...)
- NPO till bowel is active then start on a clear liquid diet (sips) then advance to regular diet as tolerated
- Early ambulation
- Pain management according to WHO pain management scale
- Antibiotics are not required postoperatively in non-perforated appendicitis

Discharge

Patients with non-perforated appendicitis can be discharged within 24hours

NB

Patients with Perforated appendicitis, post-operatively often have an ileus, and diet should only be advanced as the clinical situation warrants. Patients may be discharged once they tolerate a regular diet, usually in five to seven days

Follow-up clinic (give appointment)

Post op complications

- ❖ Surgical site infection
- ❖ Stump appendicitis
 - Incomplete appendectomy represents a failure of removing the entire appendix on the initial procedure

Differential diagnosis for right iliac fossa mass

1. Parietal swellings

Become more prominent on head/ leg raising test

- **Parietal wall abscess**

- ✓ Pyogenic that occurs in hematoma or pyaemic abscess in DM patients
- ✓ Very tender, warm surface, fever, chills & rigor

- **Desmoids tumor**

- ✓ Unencapsulated fibroma
- ✓ Occurs in multiparous(repeated stretching of abdominal wall), abdominal wall injury (e.g. laparotomy)...
- ✓ Firm to hard in consistency
- ✓ Treatment
No capsule, so wide excision followed by reconstruction by using mesh

2. Intra-abdominal swellings

a) From normal structures

i) From intestine

- **Appendicular mass***

- ✓ Tender, soft to firm in consistency
- ✓ Consists of omentum, terminal ileum, caecum, with pericaecal fat & inflammatory edema
- ✓ Management
Conservative management by IV antibiotics, fluids & as well as bowel rest. Patients should be closely monitored.
If fever, tenderness, and leukocytosis improve, diet can be slowly advanced, usually within three to five days. Patients are discharged home when clinical parameters have normalized.

An attempt to remove the appendix in appendicular mass may result in fecal fistula. It is recommended interval appendectomy to be done 6-8 weeks later. This will prevent recurrence of appendicitis & excludes neoplasms. But conservative treatment failure, as evidenced by bowel obstruction, sepsis, persistent pain, fever or leukocytosis requires immediate appendectomy.

- **Appendicial abscess***

- ✓ Very tender, firm & fixed mass...
- ✓ Relatively patients will have delayed presentation
- ✓ High grade fever, tachycardia
- ✓ Management
 - Abscess drainage
 - Proper antibiotic coverage

Q? What is phlegmonous appendix? Pus in the lumen of appendix.

- **Ileocaecal TB**
 - ✓ Chronic, non-tender, firm, nodular mass which may have mobility
 - ✓ Features of TB are present
 - ✓ Management
Limited resection followed by ileocolic anastomosis
- **Carcinoma caecum**
 - ✓ More in females, 40-50years
 - ✓ Bleeding per rectum, severe anemia
 - ✓ Hard, irregular, fixed mass
 - ✓ Psoas spasm--indicate infiltration
 - ✓ Management
Right radical hemicolectomy
- **Ameboma**
 - ✓ Tender, soft to firm mass...
- **Intussusceptions**
 - ✓ For detail info read under SBO
- **Actinomycosis**
 - ✓ Rare, 2-3months after appendicectomy
 - ✓ Hard, indurated, tender mass with multiple sinuses
 - ✓ Unlike TB→no narrowing of gut lumen & lymphnode enlargement

ii) From Lymph nodes

- **Acute lymphadenitis**
- **Lymphoma**
- **Secondaries (metastasis)**

iii) Retroperitoneal structures

- **Sarcoma**
 - ✓ Common in young
 - ✓ Huge, nodular, fixed to the posterior abdominal wall, edema of legs (IVC obstruction), hydronephrosis (pressure on ureter)
 - ✓ Management
Wide excision then radiation therapy or chemotherapy
- **Aneurysm**
 - ✓ Ileac artery→rare, elderly patients
 - ✓ Soft, pulsatile, bruit & thrill
- **Iliopsoas abscess**

In females

- **Ovarian cyst**
 - ✓ Smooth surface, round border, cystic, freely mobile & can be pushed back to pelvis
- **Fibroid of uterus/ myoma**
 - ✓ Firm to hard, nodular
- **Tobo-ovarian mass**
 - ✓ Tender, soft to firm, can be bilateral, pelvic infection present/PID

b) From abnormal structures

- **Undescended kidney**
- **Normal mobile kidney**
- **Undescended testis/ seminoma**

Chapter 15

Splenic abscess



SPLEENIC ABSCESS

Mechanisms of splenic abscess

- Hematogenous infection
- Contiguous infection
- Hemoglobinopathy
- Immunosuppression, including HIV infection and chemotherapy
- Trauma

Clinical presentation

- Fever
- Left upper quadrant pain
- Splenomegaly

NB

Presentation frequently is delayed

Complication

- The abscess may rupture & cause peritonitis

Investigation

- CBC
Leukocytosis
- The diagnosis is confirmed by abdominal ultrasound or CT scan

Management

- Broad-spectrum antibiotics should be started
 - Revise the antibiotics based on culture results
 - Duration=02 weeks
- Drainage of the splenic abscess by percutaneous means under radiological guidance
- Splenectomy
- Treatment of the underlying cause

Post-splenectomy possible complications

- Hemorrhage resulting from a slipped ligature
- Left basal atelectasis
- Pleural effusion
- Damage to adjacent structures
 - A fistula may form from damage to the greater curvature of the stomach during ligation of the short gastric vessels
 - Damage to the tail of the pancreas may result in pancreatitis, a localized abscess or a pancreatic fistula
- Axillary or other venous thrombosis
 - Because of rise in blood platelet count
 - If platelet count exceeds $1 \times 10^6 \text{ml}^{-1}$, prophylactic aspirin is recommended

- Septicaemia
 - Etiologic agents
Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenzae and Escherichia coli
- Opportunist post-splenectomy infection (OPSI)
OPSI is a major concern in such patients
 - Prevention methods
 - appropriate and timely immunization
 - antibiotic prophylaxis
 - Health education
 - Prompt treatment of infection

NB

Risk of overwhelming sepsis is greatest within the first 2–3 years after splenectomy

Case reporting format for bedside, round & exam



History

ID

Here is Ato Mamo Lakew a 55 years old male married orthodox Christian farmer from koladba admitted to Gondar university hospital, department of surgery ,surgical ward, bed no# 20 on Miazia 20/2012 e.c

Previous Admission

None

Chief complaint

Difficulty of urination of 06 months duration

HPI

This patient was last relatively healthy 06 months back at which time he started to experience difficulty of urination which made him to strain to initiate & maintain his urination. Associated with this he started to experience urgency & frequency of urination with day to night ratio of 10 to 6 which made his bed time difficult. He also has post-micturition dribbling & a feeling of incomplete voiding. 1 month back he was totally unable to pass urine after drinking about 2 Liters of local “tella” on a social gathering for which he visited our hospital where he was successfully catheterized.

- No history of penile discharge, genital ulcer or MSP
- No history chronic cough, contact with a chronic cougher or previous TB treatment.
- No history trauma to the pelvis.
- No history of anorexia, easily fatigability or significant weight loss
- No history of reddish discoloration of urine, cigarette smoking, radiation therapy to the pelvis or river water contact.
- No history medication intake like anti-histamines & anti-cholinergics
- No history of fever, chills or rigor.
- No history of tinnitus, blurring of vision or light headedness.
- No history of bone pain, hemoptysis or yellowish discoloration of the eyes.
- No self /family history of DM, Hypertension or Asthma
- Screened for RVI & found to be NR 06 months back.

Finally he was admitted to our hospital walking by himself.

Past illnesses

- No history of childhood illnesses like chicken pox, mumps or small pox. Not vaccinated.
- No history of previous surgery, trauma, psychiatry problems or drug allergy.

Review of system /ROS

H.E.E.N.T

- Head:
No history of headache or head injury

- Eyes:
No history of blurring of vision, pain in the eyes, eye itching, or spontaneous lacrimation
- Ears:
No history of Earache, difficulty of hearing, ear discharge, vertigo or tinnitus
- Nose:
No history of nasal bleeding or discharge
- Mouth and throat:
No history of gum bleeding, tooth extraction

Lymphoglandular system (LGS):

- No history of swelling in the neck, axilla or groin.
- No history of heat or cold intolerance.

Respiratory System (RS):

No history of cough, chest pain or fast breathing

Cardiovascular system (cvs):

No history of dyspnea, orthopnea or PND

Gastrointestinal system (GIS):

No history of diarrhea or constipation

Genitourinary system (GUS):

No history of flank pain, see HPI

Integumentary system (IGS):

No history of skin rash or ulcers

Muskuloskeletal system (MSS):

No history of loss of limb function or joint pain.

Central nervous system (CNS):

No forgetfulness, abnormal body movement or insomnia.

Personal History

- He was born and raised in Koladba, where he lived all his life. He had a healthy childhood and was an active boy who liked helping his father around the farm. There is no school near his village and like his parents, he never went to school. But he is able to read and write numbers.
- He is a farmer and also raises cattle, sheep and goat.
- He usually eats 'injera' made of 'teff' & 'shiro' made of 'atter'
- He is currently married and has ten children. All are alive & healthy.
- He occasionally drinks alcohol but no smoking

Family History

Father and mother:

- Both his father and mother are dead. His father died around 30 years ago at unknown age by unknown cause while his mother died two years ago at age 86 by natural cause.

Siblings:

- He has two sisters and four brothers. All are alive & well.

Family Diseases:

- No family history of DM, hypertension, Asthma, tuberculosis, allergy or sudden deaths.

Physical Examination

General Appearance

Acutely sick looking, not in cardiopulmonary stress, looks well nourished, conscious & alert

Vital signs

BP:

110/70mmHg, right arm, sitting position

PR:

95beat per minute/bpm, Right radial artery, regular & full volume

RR:

18 breath/min, shallow & regular

T⁰:

36.2⁰c, axillary, at10 AM

Weight: 65kg

Height: 160cms

BMI: 25.4

H.E.E.N.T

Head:

Normal hair distribution, No scar or tenderness

Ears:

Normal contour of pinna, Clear external ear canal.

Eyes:

- No periorbital edema, ptosis, exophthalmoses
- Pink conjunctivae, Non icteric sclera

Nose:

The nasal septum is central. There is no polyp or active discharge

Mouth and throat:

- No fissure or ulceration on the lip
- The gums are intact, no active bleeding. There are no carious teeth, extraction, dentures or filling.
- Tongue – no atrophied papillae, the buccal mucosa is pink & wet.

Lymphoglandular system

- There is no significantly enlarged lymphadenopathy in peripherally accessible lymph node areas
- The thyroid gland is not enlarged
- Descended testicles bilaterally

Respiratory System

Inspection:

- No peripheral or central cyanosis or clubbing of the finger nails
- Breathing is shallow and regular
- No Subcostal or Intercostal retraction
- No chest wall deformity or surgical scar
- No chest lag

Palpation:

- Trachea is central
- No subcutaneous emphysema
- No chest wall tenderness
- Tactile fremitus is comparable on both sides
- Chest expansion is symmetrical

Percussion:

- Resonant all over lung fields

Auscultation:

- Vesicular breath sound all over the lung field
- No added breath sound (wheezing, crepitations, stridor or pleural friction rub)

Cardiovascular system

Arterial:

- All peripherally accessible arteries are palpable
- No thickening of vessels wall
- No radio-femoral delay

Venous:

- JVP is not raised
- No distended neck vein
- Negative Hepato-jugular reflex

Precordial examination**Inspection:**

- No precordial bulging
- Quite precordium
- The apical impulse is not visible

Palpation:

- No palpable heart sound
- The PMI is at the 5th ICS lateral to MCL, localized & tapping.
- There is no parasternal or apical heave. There is no thrill.

Auscultation:

- S1 & S2 well heard
- No murmur or gallop

Abdominal examination**Inspection:**

- The abdomen is flat & symmetrical
- It moves with respiration.
- The flanks are not full
- No distended abdominal vein
- No surgical scar
- The umbilicus is inverted with circular slit
- Hernia sites are free
- No visible pulsation or peristalsis

Auscultation:

- Normo-active bowel sound (8/min)
- No bruit over renal artery, abdominal aorta or liver area

Palpation:

- Superficial palpation:
 - There is no tenderness
 - No superficially palpable mass
- Deep palpation:
 - No organomegally

Percussion:

- No sign of fluid collection (shifting dullness/fluid thrill)
- Total vertical liver span (TVLS) along the right mid-clavicular line = 6cms

Digital rectal examination/ DRE

- Inspection
 - No ulceration or visible mass
- Palpation
 - Normal anal sphincter tone
 - There is a mass which has smooth surface, regular border, firm consistency, not fixed to rectal mucosa & palpable medial sulcus but the upper border isn't reachable
 - No blood on the examining finger

Genitourinary System

- No costo-vertebral angle tenderness (CVAT)
- Kidneys are not bimanually palpable

Integumentary System

- No palmar or plantar pallor
- No rash, scar or ulcer

Musculoskeletal System

- No muscle tenderness or spasm.
- No bone deformity or tenderness.
- No stiffness of joints
- No edema.

Nervous system

Mental Status:

- GCS=15/15→E4V5M6
 - conscious & alert
- MMSE
 - He knows what day it is, where he is and what his name is. →Orientation
 - He remembers what he ate for breakfast. He also remembers where he used to live→Memory
 - He speaks in moderate voice with no hesitancy or gaps in the flow and rhythm of his words. →Speech

Cranial Nerve examination:

CN-I:

He can smell soap via each nostril.

CN-II:

- He can differentiate 2 fingers at about 6 meters. (Visual Acuity)
- He sees wagging of finger approximately 100° from axis of eye. (Visual Fields)
- He differentiates green and red colors. (Color Appreciation)

CN-III, IV & VI:

- The eyes can move in all directions. There is no nystagmus or diplopia. The pupils are round, regular in outline and equal in size. They react to light directly and consensually.

CN-V:

- He identifies light touch and pin prick over the mandibular, maxillary and ophthalmic areas of the face. He closes his eyes at the touch of the cornea with a cotton swab. Contraction of the temporal and masseter muscles is symmetrical and strong.

CN-VII:

- The face is symmetrical at rest and during voluntary movements (smiling, frowning).
He can close both eyes equally and forcefully.

CN-VIII:

- He can hear rubbing of the fingers on both ears.

CN-IX & X:

- The soft palate rises in the midline when saying 'ah!'
- He can swallow his saliva

CN-XI:

- The Sternocleidomastoid and trapezius muscles contract on turning the head and on shrugging the shoulder against resistance, respectively.

CN-XII:

- The tongue protrudes in the midline and shows no fasciculation or atrophy.

Motor examination:

- Muscle bulk:
 - Comparable muscle bulk between the left and the right extremities. There is no spontaneous as well as induced fasciculation.
- Muscle tone and power;

	TONE		POWER	
	Upper	Lower	Upper	Lower
Right	Normo-tonic	Normo-tonic	5	5
Left	Normo-tonic	Normo-tonic	5	5

Reflexes:

- **Superficial reflexes:**
 - Abdominal reflex is absent both in upper and lower quadrants.
 - Corneal reflex is intact in both eyes .
 - Plantar reflex is down going on both sides.

- **Deep tendon reflexes:**

	Biceps	Triceps	Supinator	Patellar	Ankle
Right	++	++	+	++	+
Left	++	++	+	++	+

- **Clonus:** No clonus

Coordination:

Finger to nose, heel to shin and rapid alternating movement of the arm were done without any abnormalities.

Sensory:

- He identifies light touch and pin prick over the extremities and trunk.
- He appreciates the form of a key by means of only touch (Stereognosis)
- He recognizes writings of different numbers on his palm (Graphesthesia)
- He is able to differentiate 2 pin pricks up to 4 mm apart over the finger tips (2 pt discrimination).
- He is able to recognize different movements of the toes with his eyes closed.(Position sense)
- Vibration sense was not assessed due to lack of Tuning Fork.

Meningeal Sign:

- No neck stiffness.
- Kernig's Sign is negative.
- Brudzinski's Sign is negative



SHORT CASES



R

Chapter 16

RESPIRATORY RELATED

CHEST INJURY

- 25% of all injuries
- As in all trauma patients you should apply ATLS 1^o survey followed by 2^o survey
- Investigations
 - X-ray is the first choice
 - Chest tube (Diagnostic & therapeutic)
 - Chest Ultrasound
 - CT scan

Immediate life threatening conditions after chest injury

1. Airway obstruction

- Dentures, teeth, secretion & blood
- Bilateral mandibular fracture, expanding neck hematomas, laryngeal trauma (thyroid & cricoids fracture) or tracheal injury
- Management
 - ✓ Removal of foreign bodies
 - ✓ Intubation with cervical spine protection

2. Tension pneumothorax

- It is a one way valve air leak from the lung or chest wall. Air is forced into the thoracic cavity without any means to escape. This will cause lung collapse → mediastinal displacement → decreased venous return → compromising opposite lung
- Clinical presentation
 - Tachypnea, dyspnea, distended neck veins, tracheal deviation, hyper-resonance & absent breath sound
- Management
 - Immediate decompression
 - Large bore needle in to the 2nd ICS at MCL/mid clvicular line
 - Chest tube insertion

3. Pericardial Tamponade

- Similar clinical presentation as tension pneumothorax
- In all patients with penetrating chest injury near the heart with shock, cardiac injury should be entertained unless proven otherwise
- Classically there will be increased venous pressure, decreased arterial pressure, tachycardia & muffled heart sound
- Diagnosis
 - CXR -- enlarged heart shadow
 - Echocardiography -- fluid in pericardial sac
- Management
 - Needle pericardiocentesis → for stabilization until definitive management, but there may be high risk of iatrogenic injury to the heart
 - Definitive management
 - Operative (sternotomy or left thoracotomy & repair of the heart)

4. Open pneumothorax (sucking chest wound)

- Large open defect in the chest (>3cm)→equilibrium between intrathoracic & atmospheric pressure→air in hemothorax not lung→hypoventilation
- Management
Closing defect with sterile occlusive plastic dressing taped on 3 sides to form a 1 way valve (prevents tension pneumothorax) & chest tube inserted in a site remote from injury site.
 - Definitive management
Formal debridement & closure

5. Massive hemothorax

- Usually from intercostals/IC vessels or internal mammary artery
- Clinical presentation
 - Hemorrhagic shock (flat neck veins), absent breath sounds & dull on percussion
- Management
 - correct the shock (read about shock management under short cases of Debol/miscellaneous)
 - Insert IC drain
 - Intubation (in some cases)
 - Thoracotomy
Indications
 - Drainage of 1500ml of blood or ongoing hemorrhage of >200ml/hr over 3-4hrs

6. Flail chest

- 3 or more contiguous ribs are fractured in atleast 02 locations
- There will be paradoxical movement of free floating segment of chest wall
- Management
Mechanical ventilation until fibrous union of broken ribs occur
 - O₂ administration, adequate analgesia & physiotherapy

Potentially life threatening injuries after chest injury

1. Thoracic aortic disruption

- Clinical presentation
 - Asymmetry of upper or upper & lower extremity BP
 - Widened pulse pressure
 - Chest wall contusion
- CXR
 - widened mediastinum

2. Tracheobronchial injuries

- Subcutaneous emphysema with respiratory distress
- Diagnosis
Bronchoscopy
- Management
Intubation of unaffected bronchus followed by operative repair

3. Blunt myocardial injury

- Should be suspected in patients with blunt trauma & ECG abnormality
- Echocardiography
 - Wall motion abnormalities
- At risk of developing sudden onset dysarrhythmia

4. Diaphragmatic injuries

- Should be suspected in any injuries to or below 5th intercostals space
- Herniation of abdominal contents to chest & strangulations may occur
- Most are silent
- Diagnosis
 - CXR after NG-Tube insertion—herniation of stomach
 - Contrast studies of the upper & lower GI tract
 - CT-scan

5. Esophageal injury

- Usually from penetrating trauma
- Clinical presentation
 - Odynophagia/ pain on swallowing foods & fluids
 - Subcutaneous/mediastinal emphysema
 - Pleural effusion
 - Unexplained fever within 24hr of injury
- Management
 - Operative repair & drainage

6. Pulmonary contusion

- Major cause of hypoxemia after blunt trauma
- Clinical presentation
 - Natural progression of worsening of hypoxemia for 1st 24-48 hrs
- Diagnosis
 - contrast CT-scan—confirmatory
 - Hemoptysis or blood in endotracheal tube
- Management
 - Mild→O₂ administration ,aggressive pulmonary toilet & adequate analgesia
 - Severe→mechanical ventilation

CHEST TUBE INSERTION(THORACOSTOMY)

Indications

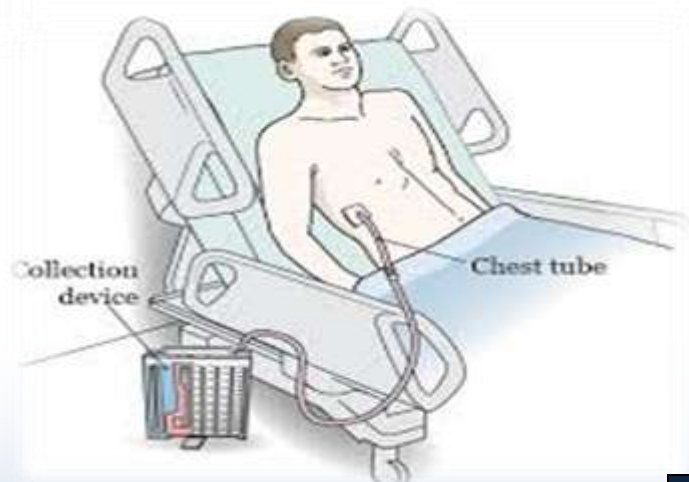
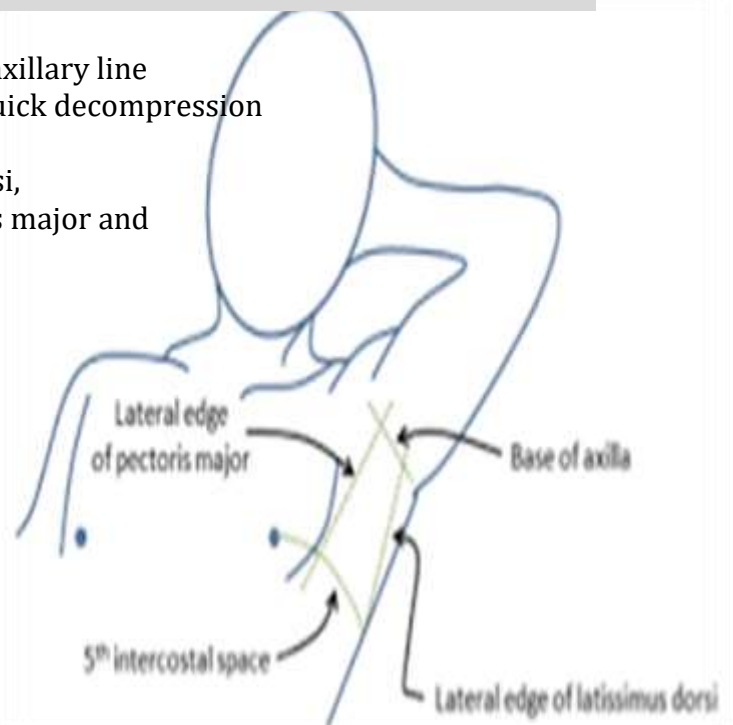
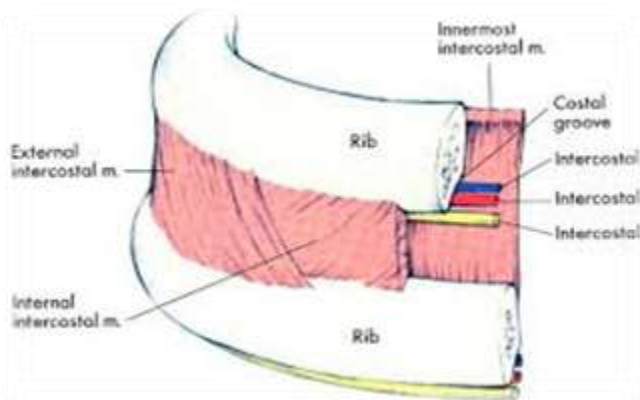
1. Empyema
2. Pneumothorax
3. Hemothorax
4. Chylothorax
5. For pleurodesis...

Relative Contraindications

1. Bleeding diathesis
2. Skin infection over the chest tube insertion site
3. Transudative pleural effusion

Site of insertion

- Generally
 - At 5th Intercostal Space (ICS) in mid axillary line
 - At 2nd ICS in mid clavicular line for quick decompression
- **Safe triangle***
 1. Anterior border of the latissimus dorsi,
 2. The posterior border of the pectoralis major and
 3. The superior border of the fifth rib



When to remove?

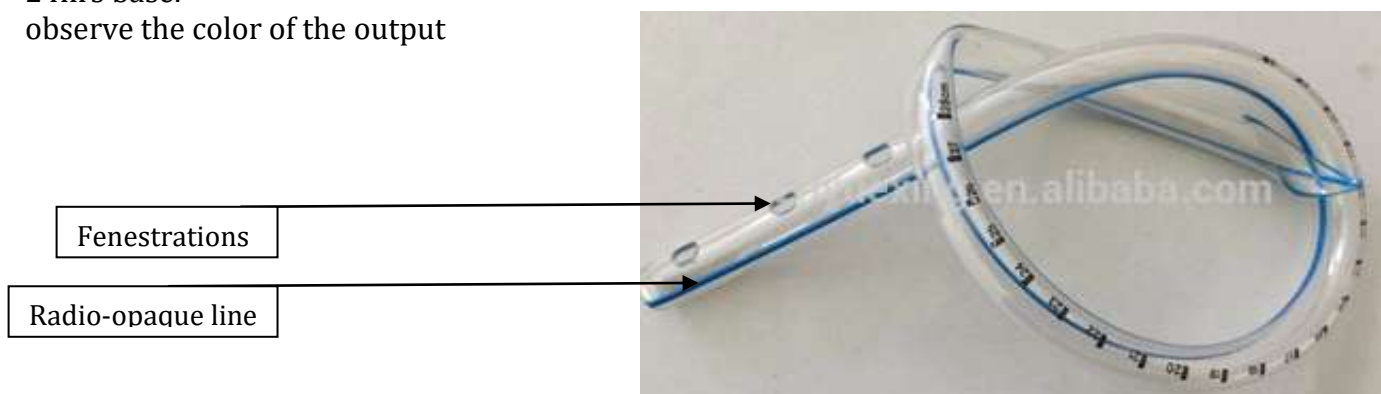
- To minimize the risk of infectious complications, the tube should be removed as soon as it is safe to do so.
 - Removal Criteria: Pneumothorax
 - When the lung is fully expanded (on CXR)
 - No visible air leak is present and air does not accumulate when suction is removed.
 - Removal Criteria: pleural fluid
 - When the lung is fully expanded
 - Fluid output is less than ~ 200 mL/day.

Complications

1. Hemorrhage
2. Organ injury (lung, mediastinum...)
3. Damage to intercostals nerves & vessels
4. Introduction of pleural infection
5. Subcutaneous emphysema (usually at the site of insertion)
6. Pulmonary edema
 - Why?
Rapid drainage of large volumes of pleural fluid lead to rapid pulmonary expansion (RPE)→pulmonary edema
To minimize risk of pulmonary edema, it's better to limit initial drainage to 1-1.5 liters.

NB

- To check the functionality of chest tube, look;
 - Continuous bubbling in the bottle (air)
 - Oscillations (fluid)—e.g. by asking the patient to cough
- In order to decide on the removal of the chest tube, follow the chest drain output on 24hrs base.
- observe the color of the output



THORACOSTOMY—Click on the link below & watch the video

<https://www.youtube.com/watch?v=kEc8fn6ownc>

Chest tube insertion procedure

Materials

- Sterile sheets, gloves and gown
- Antiseptic solution, swabs
- Local anesthesia(lidocaine)
- Syringe, scalpel blade and suture
- Needle holder, dissecting forceps, scissors
- Chest drainage tube
- underwater seal drainage bag containing about 200 mL sterile water
- one straight clamp, one curved clamp,
- Dressing and adhesive tape

Procedure

- Position the patient
- Follow possible aseptic techniques to make everything sterile
- Mark the site of insertion
- Infiltrate the skin, subcutaneous tissue and the parietal pleura with 3-5mg/kg injection lidocaine without epinephrine.
- Make a short skin incision with a scalpel blade at the level of the upper border of the rib →at the selected site.
- Separate intercostal muscles using curved clamp and reach up to the parietal pleura.
- Complete the blunt dissection with the index finger
- Thrash the chest tube held by a haemostatic forceps through the pleura, and the tube is inserted into the pleural cavity.
- Aim the tube apically for evacuation of a pneumothorax and basally for evacuation of any fluid
- The tube is clamped with a haemostatic forceps and the closed end is cutoff...the tube is connected to a water seal drainage bag so that the tube inside the bag remains under the level of the water.
- The drain is fixed to the skin by inserting a stitch through the skin but not piercing the drain.
 - Insert a stitch in circular fashion through the skin around the tube and kept untied (Which will be tied after removal of the tube).
- A sterile dressing is applied at the exit of the tube.
- Control chest x-ray should be done after 08hrs of the procedure to check proper insertion



ENDOTRACHEAL INTUBATION

Indications

- **Airway problems**--inability to maintain a patent airway with potential airway compromise due to;
 - Inhalational injury
 - Retropharyngeal hematoma
 - Facial fractures
- **Breathing problems**
 - inability to maintain adequate oxygenation by face-mask O₂ supplementation
 - presence of apnea
- **Disability problems**
 - Inability to protect airway due to altered mental status(GCS≤8)
 - Risk of aspiration of blood or vomitus
 - Sustained seizure activity

Contraindication (relative)

Unstable cervical spine injury—it needs care to insert ET tube

How to know the correct placement of the tube?

1. Audible breath sound (auscultate bilateral chest)
2. Hearing no borborygmi in the epigastrium
 - If you hear it during inspiration → it suggests esophageal intubation
3. CXR
4. CO₂ detector (capnograph or calorimetric CO₂ monitoring device)

Complications

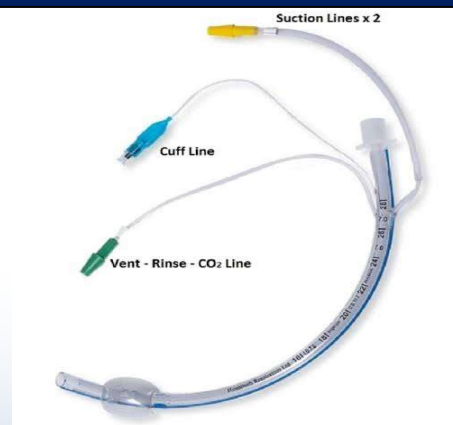
- Malposition → esophagus intubation
- Vomiting
- Bronchospasm
- Exacerbation of spinal cord injury

Click the link below & watch the video

https://www.youtube.com/watch?v=10enx5T-2_8



Laryngoscope

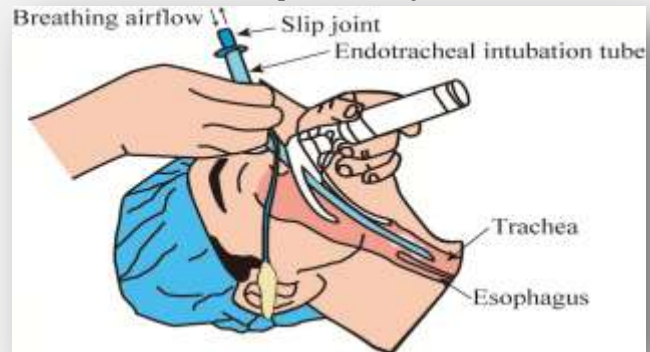


Endotracheal tube

Procedural note on ET insertion

Materials

- Laryngoscope
 - check size – the blade should reach between the lips and larynx – size 3 for most patients
 - turn on light
- Cuffed endotracheal tube
- Syringe for cuff inflation
- Face mask
- Ventilation bag
- Oxygen supply
- Suction
- Adhesive tape
- Monitors
 - Like: end-tidal CO₂ monitor, pulse oximeter, cardiac monitor, blood pressure
- Medications in awake patient: hypnotic, analgesia, short-acting muscle relaxant (to aid intubation)

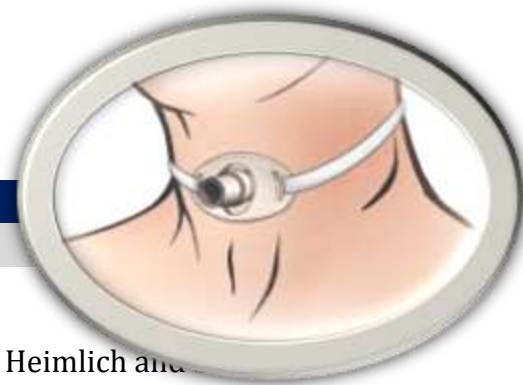


Laryngoscope insertion technique

- Give medications if required
- Pre-oxygenate patient with high concentration oxygen for 3-5mins
- Position patient
 - Neck flexed to 15°, head extended on neck (i.e. chin anteriorly), no lateral deviation
- Stand behind the head of the patient
- Open mouth and inspect: remove any dentures/debris, suction any secretions
- Holding laryngoscope in left hand, insert it looking down its length
- Passing the tongue
 - Slide down right side of mouth until the tonsils are seen
 - Now move it to the left to push the tongue centrally until the uvula is seen
- Advance over the base of the tongue until the epiglottis is seen

Tube insertion technique

- Apply traction to the long axis of the laryngoscope handle (this lifts the epiglottis so that the V-shaped glottis can be seen)
- Insert the tube in the groove of the laryngoscope so that the cuff passes the vocal cords
- Remove laryngoscope and inflate the cuff of the tube with ~ 15ml air from a 20ml syringe
- Attach ventilation bag/machine and ventilate (~10 breaths/min) with high concentration oxygen and observe chest expansion and auscultate to confirm correct positioning
- Consider applying CO₂ detector or end-tidal CO₂ monitor to confirm placement
- Secure the endotracheal tube with tape
- If it takes more than 30 seconds, remove all equipment and ventilate patient with a bag and mask until ready to retry intubation



TRACHEOSTOMY

Indications

- Acute upper airway obstruction. E.g.
 - Severe croup in children
 - Upper airway foreign body that can't be dislodged with Heimlich and cardiac life support maneuvers
 - a large pharyngo-laryngeal tumor
- For management of patients who require prolonged artificial ventilation
 - E.g. Severe tetanus
- Bilateral vocal cord paralysis
 - E.g. if recurrent laryngeal nerve/RLN is injured during thyroidectomy
- Laryngeal fracture or laryngo-tracheal separation in whom cricothyroidectomy may cause further damage
- After major surgery involving the oral cavity, pharynx, larynx or neck
 - Since there is a potential risk for upper airway obstruction due edema
- For patients with neurologic deficits that impair protective airway reflex

Site

- 2nd & 3rd tracheal rings

Complications

- Intra-op
 - ✓ Hemorrhage, Injury to paratracheal structures like carotid artery, recurrent laryngeal nerve (RLN)...
- Early
 - ✓ Hemorrhage
 - ✓ Pneumothorax
 - ✓ Pneumo-mediastinum
 - ✓ Tube blockage
- Late
 - ✓ Local infection, Tracheal stenosis, Difficulty in decannulation, Persistent tracheo-cutaneous fistula...

TRACHEOSTOMY—click the link below & watch video

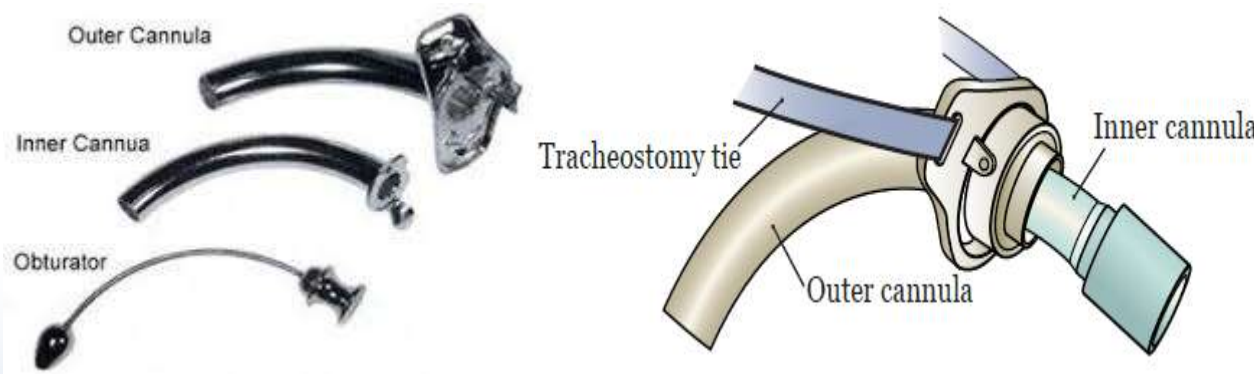
<https://www.youtube.com/watch?v=osfsrvvoHGU>



	Tracheostomy	Intubation (ET)
Advantage	• Ease of replacement (once tract has formed) • Speech, mobility, and swallowing enhanced • Patient can be nursed outside of ICU • Ease of suctioning • Patient comfort	• Rapid insertion by skilled consultant in most settings • Lack of need for surgical procedure (risk, expense) • Lack of stomal complications
Disadvantage	• Complications at cuff site • Stomal complications • Possible contribution to ultimate laryngeal injury, Tracheo-innominate artery fistula formation • Possible increase in pulmonary infections • Access to mediastinum by infectious agents after local surgery • High mortality for inadvertent decannulation before tract formation	• Complications at cuff site • Laryngeal complications • Replacement requires skill at all times • Generally requires ICU level supervision • Injuries at nose or mouth

Tracheostomy techniques

1. Percutaneous tracheostomy
2. Operative tracheostomy



Operative tracheostomy procedure

1. Incision

- Supine position, extend the neck using shoulder roll & stabilize it by doughnut cushion
- Under possible aseptic technique
 - ✓ Inject lidocaine (with epinephrine)
 - ✓ 2 cm horizontal skin incision—2 finger breadth above sterna notch

2. Dissection

- Retract superior & inferior skin flaps
- Make vertical incision in the midline fascia b/n the strap muscles—usually bloodless plane
- Laterally retract the strap muscles & the anterior jugular veins
- Proceed the dissection vertically in the midline through the pretracheal tissue & fat. Place the lateral retractors deeper in the wound as the dissection proceed to a deeper level
- The cricoids cartilage & thyroid isthmus are encountered
- Retract the isthmus by Allis clamp superiorly
 - ✓ If difficult to retract, transect it. A horizontal incision is made in the anterior suspensory ligament of the thyroid, which is between the inferior edge of the cricoid cartilage & the isthmus. A curved hemostat is used to dissect the thyroid isthmus from the anterior surface of the trachea & the thyroid isthmus is transected using electrocautery. Care is taken to not violate the anterior surface of trachea or to pass hemostat deep to the cricoid cartilage
 - ✓ The pretracheal tissue is palpated in this area for a “high riding” innominate artery before the tracheostomy incision is made.
 - ✓ The anterior surface of trachea is further cleaned ; the tracheal fascia is incised vertically in the midline & bluntly dissected laterally
 - ✓ A 2ml injection of 4% lidocaine plane is given intraluminally. This is especially important in awake pts to prevent coughing & anxiety while placing the tracheostomy tube
 - ✓ Before entering the airway all necessary instruments & previously tested tracheostomy ube should be readily available & placed in order of need on mayo stand
 - ✓ The anesthetist untapes the ETT, holds it in place & waits for instruction
 - ✓ A horizontal incision (5-8mm length) is made directly above the tracheal ring of choice (2nd, 3rd or 4th) using scalpel, taking care not to puncture the cuff or ETT
 - ✓ The incision continues in a manner necessary to remove an anterior portion of the ring
 - ✓ The trachea is delivered into the wound & stabilized using retractors already in place or a cricoids hook

- ✓ A 2.0 silk suture is placed through the inferior & superior tracheal rings, from outside to inside the lumen. The needles are removed & the sutures are not cut :the ends are brought out through the wound & can be used for retraction
- ✓ The ETT is now withdrawn under direct visualization .The cuff is deflated & ventilation is stopped
- ✓ The tracheostomy tube will be placed (with obturator in place) into the airway under direct visualization. The tube is introduced at a right angle & then turned inferiorly
- ✓ Once the tube is in place the obturator is removed, the inner cannula is placed the cuff is inflated & the anesthesia circuit is hooked to the tracheostomy tube. Confirm return of CO₂ . Auscultate the chest bilaterally
- ✓ Place tracheostomy ties around the neck, Sew the flanges of the tube—to prevent accidental decannulation
 - Remove the stay sutures @ 5th post op day

Chapter 17

GASTRO-INTESTINAL RELATED

ABDOMINAL INJURY

- One of the commonest surgical emergency
- Based on post resuscitation physiologic condition pts with Abdominal injury can be;
 - a. Hemodynamically normal
 - b. Hemodynamically stable
 - c. Hemodynamically unstable

Types

- I. Blunt abdominal injury(BAT)
- II. Penetrating abdominal injury

BAT

- BAT with poly trauma is the commonest cause of death in young population.
- Mechanism of injury/MOI in BAT may be Road traffic accident (commonest), Fall down injury, Seat belt syndrome, Assault...
- Mechanism of BAT injury
 1. Rapid deceleration
 2. Crushing(compression)effect

Approach for BAT patient

- Primary survey and act accordingly
- Secondary survey
 - History
 - Pain, abdominal distention, fever
 - Physical examination
 - Tachycardia, tachypnea
 - Abrasion, contusion, ecchymosis over the abdomen
 - Abdominal tenderness
 - Febrile, guarding, decrease bowel sound—suggestive of peritonitis
 - Follow abdominal girth by measuring with tape meter
 - Investigation
 - Follow with serial Hct
 - CBC, Blood group & Rh, Cross match, Urine analysis
 - OFT

- Imaging
 - Contrast CT--gold standard for intra-abdominal diagnosis
 - ✓ Demonstrate vascular & visceral perfusion of solid organ
 - ✓ Severity grading
 - ✓ Sensitive for blood, so very important in dx of retroperitoneal injury.
 - ✓ Difficult in dx of hollow viscous injury (sensitive only 29%-35%)
 - ✓ Used for stable patient but for unstable patient laparotomy is indicated
 - Focused abdominal sonar for trauma (FAST)—Ultrasound evaluation of abdomen to look for intrabdominal fluid
 - Accurate to detect >10ml of free blood (positive) but operator & experience dependent
- Diagnostic laparoscopy
- Diagnostic peritoneal lavage (DPL); used to assess presence of intrabdominal blood. Steps;
 - I. Empty stomach and bladder by gastric tube & catheter respectively
 - II. Insert cannula below the umbilicus & push posterior-caudally
 - III. Aspirate blood, if >10ml --insert 1000ml RL solution to the abdomen & aspirate again
- Interpretation; If RBC >100,000/ul & WBC>500/ul -- it is positive which is equivalent to 20ml of blood. i.e laparotomy is needed

Liver injury

- The mortality associated with these blunt hepatic injuries is 14.9%.
- Mechanisms of blunt hepatic injury
 1. Rapid deceleration--Tear hepatic tissue, vascular and ligament attachment
 2. Crushing effect--Compression b/n of rib cage and vertebral column

➤ Grading

Grade	hematoma(s.a)	laceration(cm)	vascular injury
1	Subcapsular tear (<10% of surface area)	<1cm parenchymal depth	-
2	Sub capsular tear 10-50%	1-3cm depth <10cm length	-
3	Sub capsular tear >50%	>3cm depth	
4		Parenchymal disruption(25-75% of H.lobe)	
5		Parenchymal disruption(>75% of H.lobe)	Juxtahepatic vein injury (Vena cava, Major hepatic vein)
6	-		Hepatic avulsion

Management

- Depends upon
 - The hemodynamic status of the patient
 - Grade of liver injury, and
 - Presence of other injuries and medical comorbidities
- The unstable patient with a positive FAST scan or DPA/DPL requires emergent abdominal exploration and control of bleeding may be through a damage-control approach or by using specific techniques for liver hemostasis.
- Stable patient who do not have other indications for abdominal exploration can be observed.
- Stable patients with right-sided penetrating thoracoabdominal injuries that lacerate the liver can also be observed, provided there are no associated intraabdominal injuries.

SPLenic INJURY

- Splenic injuries appear as disruptions in the normal splenic parenchyma, frequently with surrounding hematoma and free intra-abdominal blood.
- Spleen is commonly the bleeding intra-abdominal organ in unstable patient undergoing laparotomy.

Mechanism of blunt splenic injury can include;

- Direct compression
- Deceleration mechanism-- that tears the splenic capsule or parenchyma, mainly at areas fixed or tethered to the retro peritoneum

SPLENIC INJURY GRADING

Grading	
I	✓ Hematoma: 10% of surface area ✓ Laceration: <1 cm in depth into the parenchyma
II	✓ Hematoma: 10 to 50% of surface area ✓ Laceration: 1 to 3 cm in depth
III	✓ Hematoma: >50% of surface area ✓ Laceration: >3 cm in depth or involving a trabecular vessel
IV	✓ Segmental or hilar vessels laceration & ✓ >25 % of spleen is devascularizes
V	✓ Hematoma: shattered spleen ✓ Laceration: hilar vascular injury which devascularizes spleen.

Approach

- Primary survey “ABC of life”
- Secondary survey—History, Physical examination, Investigation & management

Investigation

- FAST—hypo echoic (i.e, black) rim of sub-capsular or intraperitoneal fluid
- On Contrast CT
 - ✓ Hemi peritoneum
 - ✓ Hypo density
 - ✓ Contrast blush-hyper dense areas within the splenic parenchyma
 - ✓ Active extravasation of contrast -ongoing bleeding
- ✓ organ-based ultrasound imaging
- ✓ MRI

Management

- ✓ Unstable & positive FAST scan or DPA/DPL requires emergent exploratory laparotomy
- ✓ Stable & low-grade (I to III) blunt or penetrating splenic injuries can be observed safely by contrast CT
- ✓ Splenectomy may be a safer option, especially in the unstable patient with multiple potential sites of bleeding

Pancreatic injury

- Rare but remain a serious problem. Because the pancreatic enzymes result in massive systemic inflammation, with subsequent poor outcomes.
- Mechanism of injury
 - Direct penetration
 - Crushing
- It is difficult to diagnose, because;
 - Physical examination is less helpful
 - It is a retroperitoneal organ, so DPL, FAST, etc are non-specific

- CT remains the mainstay of accurate diagnosis, but limited sensitivity
 - CT finding of pancreatic injury include;
 - Disruption of parenchyma
 - surrounding fluid, or
 - Hematoma and stranding in the adjacent soft tissue.
- Elevated serum amylase levels 3 hours after admission shows pancreatic injury. It may be sensitive But unknown specificity.
- Imaging of pancreatic ducts with ERCP and MRCP may be helpful, especially for those who lack supporting studies. They may assist in planning therapy and determining an operative approach
- The mainstay of therapy for pancreatic injuries is surgical, exposure of the entire gland to evaluate the pancreas comprehensively.

Small Bowel Injuries

- ✓ The small intestine is one of the most frequently injured organs due to large percentage of the abdomen it occupies.
- ✓ Penetrating injuries can vary from tiny perforations to large destructive injuries that destroy circumferential segments of small bowel.
- ✓ The repair of small bowel injuries depends on the extent of intestinal wall destruction in relation to the luminal circumference.
 - Small perforations that can be closed without compromising the intestinal lumen can be debrided and repaired with one or two layers.
 - Injuries occupying over 50% of the intestinal wall circumference should be addressed with resection and anastomosis either by stapled or hand sewn anastomoses for intestinal resections. Hand-sewn anastomoses are frequently constructed in two layers
 - The damage control approach is important for rapid closure of perforations to control contamination and to control bleeding.

Damage control surgery

- ✓ Is operation done until the physiological derangement can be corrected
- ✓ It has two goals:
 1. stopping any active surgical bleeding
 2. Controlling any contamination
- ✓ The key philosophy is
 - to keep the patient alive at any cost
 - Be part of the resuscitation process, in which there is initial surgical control of hemorrhage and contamination followed by rapid closure
- ✓ The stages of damage control surgery
 - I. Patient selection
 - II. Control of hemorrhage and control of contamination
 - III. Resuscitation continued in the intensive care unit
 - IV. Definitive surgery
 - V. Abdominal closure

Post operative complications in abdominal surgery

Enteric Fistula

- The communication occurs between two parts of the GI tract or adjacent organs.
 - ✓ Internal fistula (e.g., enterocolonic fistula or colovesicular fistula).
 - ✓ An external fistula (e.g., enterocutaneous fistula or rectovaginal fistula) involves the skin or another external surface epithelium
- Over 80% of enterocutaneous fistulas represent pathogenic complications that occur as the result of enterotomies or intestinal anastomotic dehiscences. Fistulas that arise spontaneously without antecedent iatrogenic injury are usually manifestations of progression of underlying Crohn's disease or cancer.
- Fistulas have the potential to close spontaneously. Factors inhibiting spontaneous closure, however, include;
 - ✓ Malnutrition
 - ✓ Sepsis
 - ✓ inflammatory bowel disease
 - ✓ cancer
 - ✓ radiation
 - ✓ obstruction of the intestine distal to the origin of the fistula
 - ✓ foreign bodies
 - ✓ high output
 - ✓ short fistulous tract (<2 cm) and epithelialization of the fistula tract
- The most common presentation of an enteric fistula is in a postoperative patient with abdominal distension and tenderness, a low grade fever, leukocytosis and failure to recover normally from abdominal surgery.
- Iatrogenic enterocutaneous fistulas usually become clinically evident between the fifth and tenth postoperative days. Fever, leukocytosis, prolonged ileus, abdominal tenderness, and wound infection are the initial signs. The diagnosis becomes obvious when drainage of enteric material through the abdominal wound or through existing drains occurs. These fistulas are often associated with intra-abdominal abscesses. Peritonitis can be developed within 24 to 48 hour

1. Enterocutaneous fistulas

- ✓ The anatomic classification of enteric fistulas is based upon the segment of bowel from which it originates (ie, gastrocutaneous, colocutaneous)
- ✓ The etiologic classification is based upon the underlying disease process (ie, postoperative, trauma, foreign body, Crohn's disease, diverticulitis, tuberculosis, malignancy) . Most fistulas are the result of bowel injury during surgery, a leak from a bowel anastomosis, or erosion of mesh into adjacent bowel

- ✓ The physiologic classification is based on the volume of fistula output.
 - A low output fistula drains less than 200 mL/day.
 - A moderate output fistula drains between 200 and 500 mL/day.
 - A high output fistula drains more than 500 mL/day

Enteroatmospheric (exposed) fistulas- occur in the midst of an open abdomen with no overlying soft tissue.

MANAGEMENT OF ENTEROCUTANEOUS FISTULAS

- The treatment of enterocutaneous fistulas should proceed through an orderly sequence of steps.
 1. Stabilization. Fluid and electrolyte resuscitation is begun. Nutrition is provided, usually through the parenteral route initially. Sepsis is controlled with antibiotics and drainage of abscesses. The skin is protected from the fistula effluent with ostomy appliances or fistula drains.
 2. Investigation. The anatomy of the fistula is defined using the studies described earlier.
 3. Decision. The available treatment options are considered, and a timeline for conservative measures is determined.
 4. Definitive management. This entails the surgical procedure and requires appropriate preoperative planning and surgical experience.
 5. Rehabilitation.

The overall objective is to increase the probability of spontaneous closure. Nutrition and time are the key components of this approach. Most patients will require TPN; however, a trial of oral or enteral nutrition should be attempted in patients with low-output fistulas originating from the distal intestine.

The somatostatin analogue octreotide is a useful adjunct, particularly in patients with high-output fistulas; its administration reduces the volume of fistula output, thereby facilitating fluid and electrolyte management. Further, octreotide may accelerate the rate at which fistulas close; however, its administration has not clearly been demonstrated to increase the probability of spontaneous closure.

Stoma complications

Based on time	Medical (metabolic)→treated medically	Surgical→treated surgically
Early	▪ Skin irritation ▪ High output(>1.5L) ▪ Leakage ▪ Ischemia	• Necrosis • Retraction • SBO • Wound infection • Sepsis
Late	▪ Dehydration ▪ Nephrolithiasis ▪ Cholelithiasis	• Prolapse • Peristomal hernia • Dehiscence

Postgastrectomy problems

- ✓ Up to 30% of patients who have had operations on the stomach are afflicted with chronic symptoms that have been relegated to the category of postgastrectomy syndromes
- ✓ But it is a misnomer since some of these patients have not had a gastrectomy (e.g., dumping after a pyloroplasty or fundoplication)
- ✓ The incidence is substantially higher in the first postoperative year, but most patients report improvement within 1 year after surgery
- ✓ However, a small percentage (<5%) of patients after a variety of gastric operations have persistent debilitating symptoms

A) Dumping Syndrome

- ✓ DS is a constellation of GI and vasomotor symptoms, which present postprandially due to rapid gastric emptying caused by ...
 - Procedures that alter the normal intragastric pressure/volume relationship
 - proximal gastric vagotomy
 - sleeve gastrectomy
 - fundoplication

- Procedures that alter Out flow resistance
 - Pyloroplasty
 - Gastrojejunostomy
 - Gastroduodenostomy
- Procedures that alter both have the highest incidence of dumping
 - Gastrectomy=67% of patients present with early dumping symptoms and 38% with late dumping
 - Roux-en-Y gastric bypass=75%
 - Billroth II = 70%

i) Early dumping

- Postprandial gastrointestinal discomfort, which may include nausea, vomiting, diarrhea, and cramps, with vasomotor symptoms such as diaphoresis, palpitations, and flushing
- Systemic manifestations include palpitations, tachycardia, fatigue, a need to lie down following meals, flushing or pallor, diaphoresis, lightheadedness, hypotension, headache, and possibly syncope, diarrhea, nausea, cramps, bloating, and borborygmi
- Why...?? Attributable to bowel distention, relative hypovolemia, GI hormone hypersecretion, and autonomic dysregulation. These mean Dumping symptoms are triggered by rapid gastric emptying of hyperosmolar voluminous chyme that causes bowel distention, hypermotility, and splanchnic blood pooling. This leads to both the GI and vasomotor symptoms that characterize early DS. Hormones play an important role as mediators of this pathophysiologic response
 - ✓ vasoactive intestinal peptide [VIP]
 - ✓ serotonin,
 - ✓ bradykinin
 - ✓ norepinephrine)
- Abdominal symptoms include early satiety, epigastric fullness or pain,
- Clinically significant dumping occurs in 5% to 10% of patients after pyloroplasty, pyloromyotomy, or distal gastrectomy
- The symptoms are thought to be the result of the abrupt delivery of a hyperosmolar load into the small bowel due to ablation of the pylorus or decreased gastric compliance (attributed to the rapid emptying of hyperosmolar chyme (particularly carbohydrates) into the small bowel. This leads to net fluid retention due to the osmotic gradient and probably releases one or more vasoactive hormones, such as serotonin and vasoactive intestinal polypeptide.)
- 15 to 30 minutes after a meal

ii) Late dumping

- patients complain of the same constellation of symptoms hours after eating(2-3hours)
- Symptoms like perspiration, faintness, decreased concentration, and altered levels of consciousness, among others.
- This phenomenon is thought to be secondary to hypoglycemia from a postprandial insulin peak. this mean...Late dumping involves a reactive hypoglycemia brought on by the rapid and high initial glucose load presented to and absorbed by the small intestine, leading to a GLP-1-mediated inappropriately high insulin response and hypoglycemia. This overshoot insulin cause the adrenal gland to produce catecholamines, which cause a constellation of symptoms, including tachycardia, tachypnea, diaphoresis, and lightheadedness. Late DS is more frequent in patients who had early DS.
- Early dumping tends to improve with time, whereas late dumping tends to persist or exacerbate.

▪ Diagnosis of DS

- An oral glucose challenge will confirm the diagnosis of DS ...the principle is DS cause tachycardia, immediate in the early and low RBS 1 to 3 hours after meal and increased hct due to hyperosmolarity of the GI content...so the patient told to fasting for at least 10 hours then give him 50g sugar and follow the PR, Hct, RBS every 30 minute then the tset is positive if ...
 - your heart rate increases by 10 beats per minute 30 minutes after you drink the glucose solution
 - your blood test results show a 3 percent increase in your hematocrit 30 minutes after you drink the glucose solution
 - your blood test results show low blood glucose 1 to 3 hours after you drink the glucose solution
 - It is reported to have a sensitivity and specificity of up to 100% and 94%, respectively.
- Scintigraphic gastric emptying study
 - The diagnosis of early DS in a patient with clinical symptomatology may also be confirmed with a scintigraphic gastric emptying study, in which greater than 50% of an isotope-labeled solid meal has emptied within 1 hour.
 - The principle her is taking radioactive substance then taking series images or follow with camera to see how the gastric emptying is fast. normally the half-life of solid is 2 hours with total 4 hours but fluid is 12 minute half-life ...anything faster than this shows DS

▪ Differential diagnosis of DS includes

- ✓ Gastroparesis
- ✓ Partial small bowel obstruction
- ✓ Anastomotic stricture

- ✓ Postvagotomy diarrhea
- ✓ Inflammatory bowel disease
- ✓ Irritable bowel disease
- ✓ Bacterial overgrowth
- ✓ Islet cell tumor as the cause of a severe refractory hypoglycemia

Management of dumping syndrome

- ✓ Treated conservatively with dietary changes (frequent small meals that are high in protein and fat and low in carbohydrates for early dumping; increased carbohydrate consumption for late dumping)
- ✓ Daily intake should be divided into at least 6 meals.
- ✓ Symptoms tend to resolve in most patients as they learn to avoid foods that aggravate it
- ✓ If dietary manipulation fails, the patient is started on octreotide, 100 µg subcutaneously twice daily. This can be increased up to 500 µg twice daily if necessary. The long-acting depot octreotide preparation is useful. Octreotide not only ameliorates the abnormal hormonal pattern seen in patients with dumping symptoms, but also promotes restoration of a fasting motility pattern in the small intestine
- ✓ Only a very small percentage of patients with dumping symptoms ultimately require surgery. Most patients improve with time (months and even years), dietary management, and medication
 - Diet modification
 - Liquids and solids should be separated.
 - Diets should be high in protein and fat, and simple sugars should be avoided.
 - Vasomotor symptoms can often be ameliorated if the patient lies down for 30 minutes after meals.
 - Elimination of milk and dairy products has been successful in many patients.
 - Another simple therapy is adding dietary fiber.
 - Guar gum and pectin are useful in increasing the viscosity of the food, but their poor taste limits patient compliance.
 - Pharmacologic
 - Antidiarrheal
 - ✓ Tincture of opium is especially effective in relieving diarrhea associated with DS
 - ✓ Imodium
 - Nausea Rx
 - ✓ Promethazine
 - ✓ proton pump inhibitors
 - ant motility for pain and decrease diarrhea
 - antibiotic to decrease the bacteria overload that cause bloating and more gas

- Octreotide
 - ✓ For diate modification resistant
 - ✓ For both late and early through inhibition of
 - ✓ hormone mediators
 - ✓ delays gastric emptying time and inhibits splanchnic vasodilation
- Surgical management
 - ✓ most patients responsive for diate modification and medication with in months to years so don't rush to re operation
 - ✓ There are a variety of surgical approaches, none of which work consistently well.

B) Diarrhea

- It occurs soon after surgery and usually is not associated with other symptoms, a fact that helps to distinguish it from dumping.
- The cause of postvagotomy diarrhea is unclear. Possible mechanisms include intestinal dysmotility and accelerated transit, bile acid malabsorption, rapid gastric emptying, and bacterial overgrowth.
- Another theoretical cause of diarrhea following gastric surgery is fat malabsorption due to acid inactivation of pancreatic enzymes or poorly coordinated mixing of food and digestive juices
- Management of diarrhea
 - ✓ Some patients with postvagotomy diarrhea respond to cholestyramine, while in others codeine or loperamide may be useful. Octreotide should also be tried

C) Gastric Stasis

- Due to a problem with gastric motor function (retrograde peristalsis in a Roux limb) or caused by a mechanical obstruction (anastomotic stricture, efferent limb kink from adhesions or constricting mesocolon, or a proximal small-bowel obstruction).
- The gastric motility abnormality could have been preexisting and unrecognized by the operating surgeon. Alternatively, it may be secondary to deliberate or unintentional vagotomy, or resection of the dominant gastric pacemaker
- Gastric stasis presents with vomiting (often of undigested food), bloating, epigastric pain, and weight loss

D) Bile Reflux Gastritis and Esophagitis

- Reflux of bile into the stomach is common after operations that eliminate the pyloric sphincter. In most patients there are no serious clinical sequelae. However, approximately 2 percent of patients develop alkaline reflux gastritis, a syndrome of persistent burning epigastric pain and chronic nausea that is aggravated by meals. The diagnosis is made primarily by excluding other causes of symptoms.
- Therefore, attributing postoperative symptoms to bile reflux is problematic because most asymptomatic (around 2% are symptomatic) patients have bile reflux as well.

E) Weight Loss

- The causes of weight loss after gastric surgery generally fall into one of two categories:
 - altered dietary intake or
 - malabsorption
- If a stool stain for fecal fat is negative, it is likely that decreased caloric intake is the cause. This is the most common cause of weight loss after gastric surgery, and may be due to small stomach syndrome, postoperative gastroparesis, anorexia due to loss of ghrelin, or self-imposed dietary modification because of dumping and/or diarrhea

F) Anemia

Iron absorption takes place primarily in the proximal GI tract, and is facilitated by an acidic environment. Intrinsic factor, essential for the enteric absorption of vitamin B12, is made by the parietal cells of the stomach. Vitamin B12 bioavailability also is facilitated by an acidic environment.

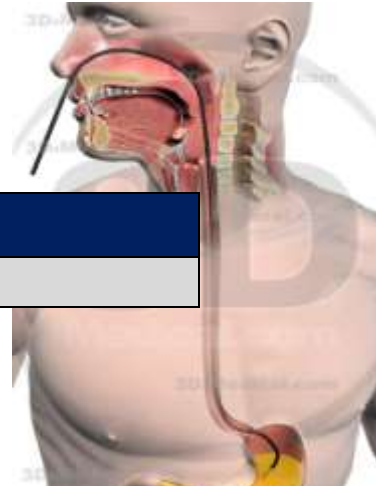
Anemia is the most common metabolic side effect in patients who have had a gastric bypass for morbid obesity. It also occurs in up to one third of patients who have had a vagotomy and/or gastric resection. Iron deficiency is the most common cause, but vitamin B12 or folate deficiency also occurs, even in patients who have not had total gastrectomy

Gastric bypass patients should be given oral iron supplements, and monitored for iron, B12, and folate deficiency. Patients who have had a vagotomy and/or gastrectomy should be similarly monitored with periodic determination of hematocrit, red blood cell indices, iron and transferrin levels, B12, and folate levels.

G) Bone Disease

Gastric surgery sometimes disturbs calcium and vitamin D metabolism. Calcium absorption occurs primarily in the duodenum, which is bypassed with gastrojejunostomy. Fat malabsorption may occur because of blind loop syndrome and bacterial overgrowth, or because of inefficient mixing of food and digestive enzymes. This can significantly affect the absorption of vitamin D, a fat-soluble vitamin. Both abnormalities of calcium and vitamin D metabolism can contribute to metabolic bone disease in patients following gastric surgery. The problems usually manifest as pain and/or fractures years after the index operation. Musculoskeletal symptoms should prompt a study of bone density. Dietary supplementation of calcium and vitamin D may be useful in preventing these complications

Routine skeletal monitoring of patients at high-risk (e.g., elderly males and females and postmenopausal females) may prove useful in identifying skeletal deterioration that may be stopped with appropriate treatment.



NASO-GASTRIC TUBE (NGT)

Indications

1. Gastric decompression
 - Removal of accumulated gastrointestinal air & fluid to relieve its pressure effect
2. Bowel irrigation
 - To lavage (wash) the stomach in case of poisoning or overdose of medications
3. For feeding--In patients with risk of aspiration
4. For administration of medication--To clients unable to swallow medications without aspirating it to the lungs
5. To obtain specimen from gastric content for analysis
6. After bowel surgery--To promote healing
7. To administer radiographic contrast media to GIT for imaging

Contraindications

- Absolute
 1. severe mid-face trauma
 2. Recent nasal surgery
- Relative
 3. coagulation abnormality
 4. Esophageal stricture → fear of perforation
 5. Esophageal varices → fear of bleeding

How to confirm that the tube is in stomach?

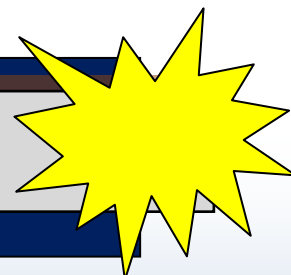
- Inject about 5mL of air through the tube. Audible gurgling sound in the epigastrium with a stethoscope indicate the tube is in the stomach
- Aspirate the tube with a syringe → if the aspiration is greenish gray colored fluid → the tube is in the stomach
- Listen to the end of the tube at the nose → the sound of moving air indicate that the tube is in trachea, not in the stomach.
- X-ray

Complications

- Some degree of patient discomfort--can induce gagging or vomiting
- GERD
- Esophageal perforation
- Epistaxis
- Respiratory tree intubation /malposition

Click the link below & watch the video

<https://www.youtube.com/watch?v=en5ctZlnOyA>



Procedural note on NGT insertion

Materials

- Nasogastric tube –Adult 16-18Fr
- KY gel
- drainage bag
- gloves
- Syringe
- stethoscope
- Glass of water with a straw
- adhesive tape



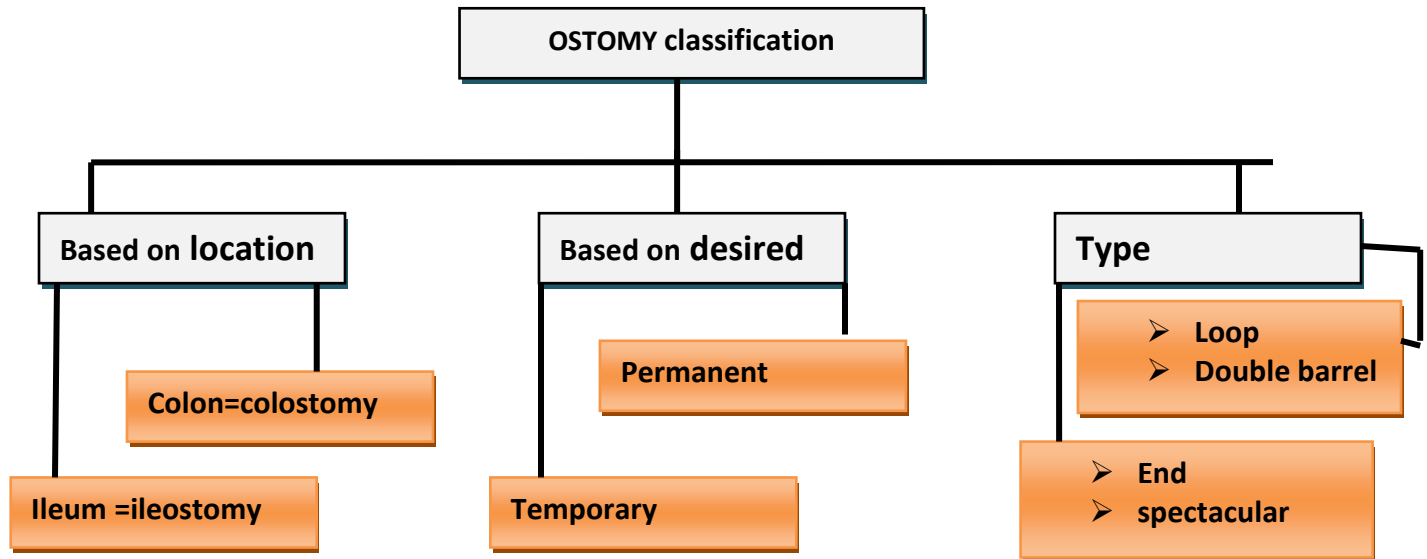
Procedure

- Explain the patient that a tube is going to be inserted through his nose & that...
 - ✓ He/she has to swallow the tube
 - ✓ There may be some cough during insertion of the tube
- Measure distance from the nostril to the tip of ear lobe then from the earlobe to the tip of xiphoid process → mark
- Lubricate the NG tube & choose the nostril which has the wider channel
- Pass the NG tube horizontally through the nose. As the tube touches the posterior pharyngeal wall the patient will gag
 - ✓ Patient may be given a little water to sip. The tube is then slowly advanced as the patient is asked to swallow the tube. During swallowing the cricopharyngeus muscle will relax and the tube will enter into the esophagus.
 - ✓ when the pt breathes → Stop inserting the tube for a moment
 - ✓ don't use force
- The tube is further advanced till the second ring in the tube lies at the level of nostril when the tip will lie in the stomach
- The tube is secured to the nose with an adhesive tape
- The tube is then connected to a plastic drainage bag to siphon out the stomach contents
- If required the stomach content may be sucked out

STOMA (COLOSTOMY , ILEOSTOMY)

General indication

- When restoration of intestinal continuity is contraindicated or not feasible



Indication for temporal “ostomy”

1. Gangrenous bowel obstruction
2. Penetrating colon injuries
3. High risk of anastomosis leak
 - ✓ In Immunocompromised patients, Severely malnourished patients...
4. Hemodynamic instability
 - ✓ In patients with trauma, Sepsis...
5. Complex perianal fistulas

NB

As a general rule, closure of a temporary colostomy is delayed for weeks to months to assure complete resolution of the underlying condition and to permit softening of the adhesions. However, closure of the colostomy may be performed within three to six weeks if studies indicate that the need for diversion no longer exists (eg, if the distal anastomosis is well healed)

Indication for permanent “ostomy”

1. After excision of the rectum for carcinoma
2. Total abdominal proctocolectomy for severe IBD

"Stoma" complications

- Early (<3months)
 - Leakage
 - Skin irritation--Common in ileostomy
 - Stomal necrosis
 - Stomal retraction
- Late
 - Parastomal hernia
 - Stomal prolapse
 - Stomal stenosis
- Psychosocial problem

Colostomy

- ✓ Colostomy refers to a surgical procedure where a portion of the large intestine is brought through the abdominal wall to carry stool out of the body.
- ✓ Colostomy bypasses distal colon, rectum or anus.
- ✓ May be
 - Temporary
 - ✓ To decompress an obstructed or perforated distal colon
 - ✓ To permit healing of fistulous tract or acute inflammatory process distally
 - ✓ For protection of distal anastomosis when delayed healing is anticipated
 - Permanent
 - ✓ In case if distal rectum & anorectal sphincter mechanisms are removed

NB

Colostomy diarrhea may be present

Ileostomy

- ✓ Ileostomy refers to a surgical procedure where a portion of the small intestine (the ileum) is brought through the abdominal wall to carry stool out of the body.
- ✓ Ileostomy bypasses the entire colon & rectum.
- ✓ May be temporary or permanent

NB

Ileostomy patients are more likely to develop fluid and electrolyte problems ++ skin irritation

How to differentiate stomas?

1. The content that is coming out
 - ✓ Mucus--in colostomy
 - ✓ Watery--in ileostomy
2. Skin around the stoma
 - ✓ More skin irritation in ileostomy
 - ✓ Pink in colostomy
3. Odor
 - ✓ Foul smelling in colostomy
4. Size of stoma
 - ✓ Wider in colostomy than ileostomy
5. If you insert your hand in stomas you will be able to differentiate loop from end ostomy
6. ? Site??--Difficult

STOMAL PHYSIOLOGY

- ✓ Physiologically left-sided colostomy output is very similar to that of normal bowel movements.
- ✓ There are essentially no significant physiologic abnormalities associated with left-sided colostomy; for that reason we will be focus to ileostomy output
- ✓ Ileostomies become functional post-operative within 48 hours but colostomies within 96 hours
- ✓ Ileostomy has 3 phase of adaptation post operatively

1st phase

- In the first three days, the output is bilious and liquid in nature and increases daily with the maximum output occurring on the third or fourth day
- So the critical period for acute dehydration was the third to eighth day and recommended aggressive fluid and electrolyte replacement.
- That why patients should not be discharged before 9 or 10 days after ileostomy construction
- Why this happen? Due to Small bowel adaptation following ileostomy creation results in increased reabsorption of water and electrolytes. In a normal individual, between 1500–2000 mL per day exit the terminal ileum into the colon. With ileostomy adaptation, 70% to 80% of this output is reabsorbed

2nd phase

- Which occurs from the fourth to the sixth day following operation, the output stabilizes, thickens, and even decreases slightly.

3rd phase

- Occurs from the first week to the eighth week following operation and is associated with a steady decrease in volume and thickening of the stoma output
- After complete adaptation, the output from an end ileostomy created without significant ileal resection stabilizes between 200 and 700 cc/day. What are the adaptation mechanisms? Within a few hours after bowel resection, the remnant small intestine displays evidence of epithelial cellular hyperplasia. With additional time, villi lengthen, intestinal absorptive surface area increases, and digestive and absorptive functions improve
- What nutrition increase ileostomy out put?
 - Fiber diet >16g/day
 - High fat
 - Obesity
- What nutrition decrease ileostomy out put?
 - Elemental diet
 - Fasting

✓ Nutrition

- What is the out point of ileum length below or above which risk of malabsorption is considered?→100cm...!!!
- Ileostomies associated with less than 100 cm of resected terminal ileum are able to maintain essentially normal nutrition.
- Resection of greater than 100 cm of terminal ileum or bacterial overgrowth may result in B12 malabsorption, and vitamin B supplementation may be necessary to prevent megaloblastic anemia.

✓ Metabolic Changes

Metabolites	Normal	With ileostomy
Sodium in stool	2 and 10 mEq/day	60 mEq/day
Water	100 to 150 mL	650 ml
Potassium	5 to 15 mmol	6 mmol
Chloride	1 to 2 mmol	34 mmol

- Symptomatic salt depletion, however, is rare due to renal compensation. Individuals with ileostomies have been shown to have chronically elevated mineralocorticoid levels that increase water and sodium reabsorption, compensating for the increased losses in the stool. Renal compensation combined with a normal diet make a chronic dehydration and salt depletion a rare circumstance in individuals with well-adapted ileostomy
- calcium, and magnesium levels are unaffected unless extensive terminal ileum has been resected
- Water intake plays virtually no role. The bacteriologic composition of the ileostomy fluid is different from that of normal small or large intestine. The total number of bacteria in ileostomy effluent is approximately 80 times greater than that in the normal ileum. The ileostomy effluent contains a 100-fold increase in the number of aerobes, a 2500-fold increase in coliform bacteria, and increases in total anaerobes compared to the normal ileum.
- The single most important factor influencing ileostomy output is the amount of uninjured intestine proximally
- Overproduction of gastric acid contributes to the volume of ileostomy output. Ileostomy output decreases when patients receive anti secretory agents such as omeprazole, but such benefits are reaped only with patients with large ileostomy output (2.61 L/day) or those who have had significant amounts of small bowel resected.

✓ Ileostomy vs urolithiasis or cholelithiasis

- Urolithiasis =Uric acid stones are rare in the normal population, but comprise 60% of stones in individuals with an ileostomy
 - Due to Low urinary volume and pH combined with increased concentrations of calcium and oxalate are thought to be responsible for this phenomenon.
 - In addition, decreased urinary volume and decreased urinary pH facilitate stone precipitation

- Rx for uric acid= Prophylaxis and treatment consist of increasing daily fluid to increase urinary volume and urinary pH
- Gall stone
 - Significant terminal ileal resection or extensive terminal ileal disease does affect the entero-hepatic circulation. Bile absorption and/or depletion may occur, resulting in increased saturation of bile that leads to bile salt precipitation and subsequent stone formation.

BOWEL PREPARATION

- Aim
To decrease the bacterial load, this in turn decreases risk of infection & anastomosis leak
- Total duration
For 03 days
- How
→02 methods

1. Mechanical bowel preparation

**Using cleansing enema
(Water & soap)**

2. Pharmacologic bowel preparation

- ✓ **Laxatives**
 - **Castor oil**
 - **Polyglycol solution**

→Additional measures to be taken

- Keep on fluid diet for 03 days
- Antibiotics
 - ✓ Which drug to choose?
 - Coverage--Broad spectrum
 - Route—P.O. than IV
 - Poorly absorbable from GI system
 - ✓ Drug of choice (DOC)
 - Metronidazole & Neomycin
 - If neomycin isn't available we can use others like Ampicillin, Amoxicillin, erythromycin...
 - ✓ When?
 - If the surgery is scheduled tomorrow morning, the antibiotic should be given today at 1:00 PM then 2:00PM & 11:00PM. (i.e. in local time-- 7, 8 (in the afternoon) & 5 O'clock in the night)
 - Antibiotics also should be given 30 minutes prior to surgery--
Ceftriaxone 1gm IV stat
- NPO (nothing per os)→08 hours prior to surgery

NB*

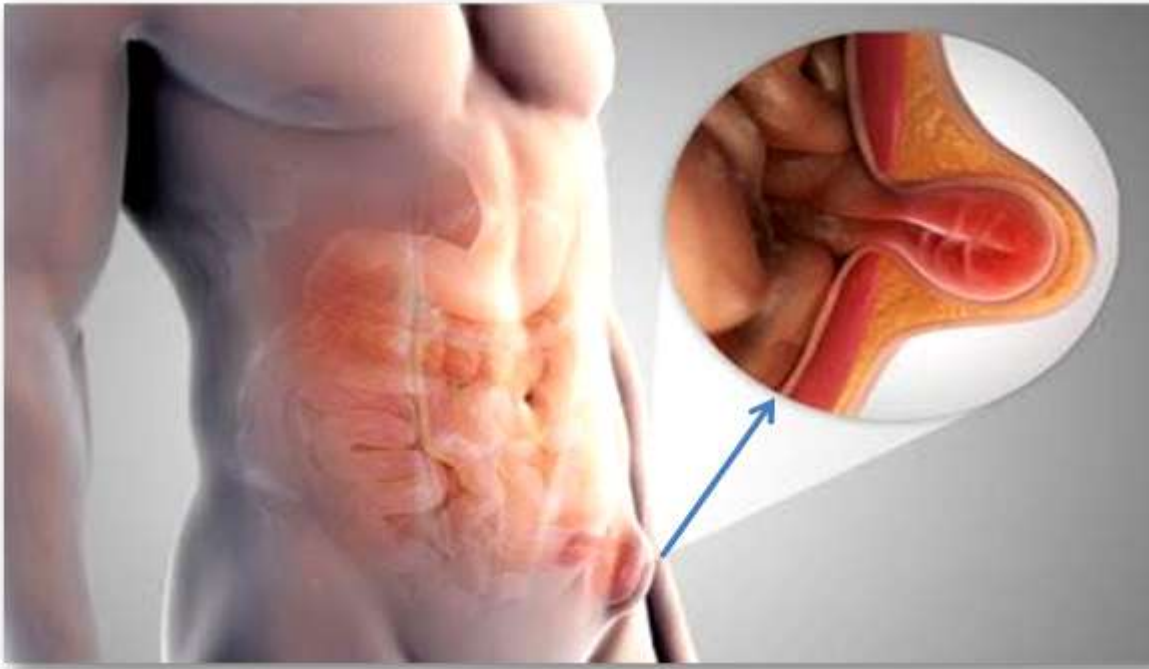
Complete bowel obstruction & perforation are contraindications for bowel preparation

		Treatment	
		<i>New order</i>	
<i>22/10/12</i>	<i>2:00 LT/D</i>	<i>Cleansing enema BID#3days</i>	
		<i>castor oil 15ml P.O BID#3days</i>	
		<i>Fluid diet for 3days</i>	
<i>24/10/12</i>	<i>2:00 LT/D</i>	<i>Added order</i>	
		<i>Amoxicillin 500mg p.o at</i>	
		<i>7:00LT/D, 8:00LT/D & at 5:00LT/N</i>	
		<i>Metronidazole 500mg p.o at 7:00LT/D</i>	
		<i>8:00LT/D & 5:00LT/N</i>	
		<i>MF & NPO after mid night</i>	
			<i>Dr.Daniel F</i>

HERNIA

Introduction

A hernia is defined as an abnormal protrusion of an organ or tissue through a defect in its surrounding walls



- ❑ Hernias by themselves usually are harmless, but nearly all have a potential risk of having their blood supply cut off (becoming strangulated). If the blood supply is cut off at the hernia opening in the abdominal wall, it becomes a medical and surgical emergency

Terminologies

- ❑ Reducible hernia is when hernial contents can be re-placed within the surrounding musculature.
- ❑ Irreducible or incarcerated hernia is when the hernia cannot be re-placed/reduced.
- ❑ A strangulated hernia has compromised blood supply to its contents, which is a serious and potentially fatal complication

Predisposing factors

Any condition that increases the pressure of the abdominal cavity may contribute to the formation or worsening of a hernia.

- Heavy lifting
- Coughing
- Straining during a bowel movement or urination
- Ascites
- Obesity



Anatomical weakness

- Where structures pass through the abdominal wall
- Where muscles fail to overlap
- Where there is no muscle

Congenital

Collagen fiber disorders

Composition

1. The Sac:

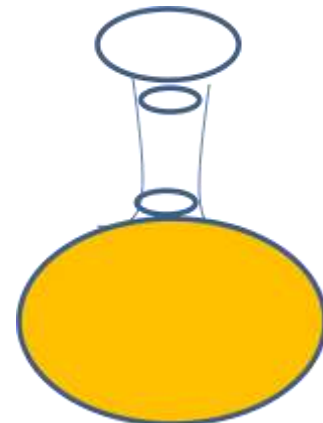
A folding of peritoneum consisting of a mouth, neck, body and fundus.

2. Coverings:

Derived from layers of the abdominal wall.

3. Contents:

Which could be anything from the omentum, intestines, ovary or urinary bladder.



"١٢٣" as analogy

Types of abdominal hernias

Groin

Inguinal*

- Indirect
- Direct

Femoral *

Anterior

- Umbilical*
- Epigastric*
- Spigelian

Pelvic

- Obturator
- Sciatic
- Perineal

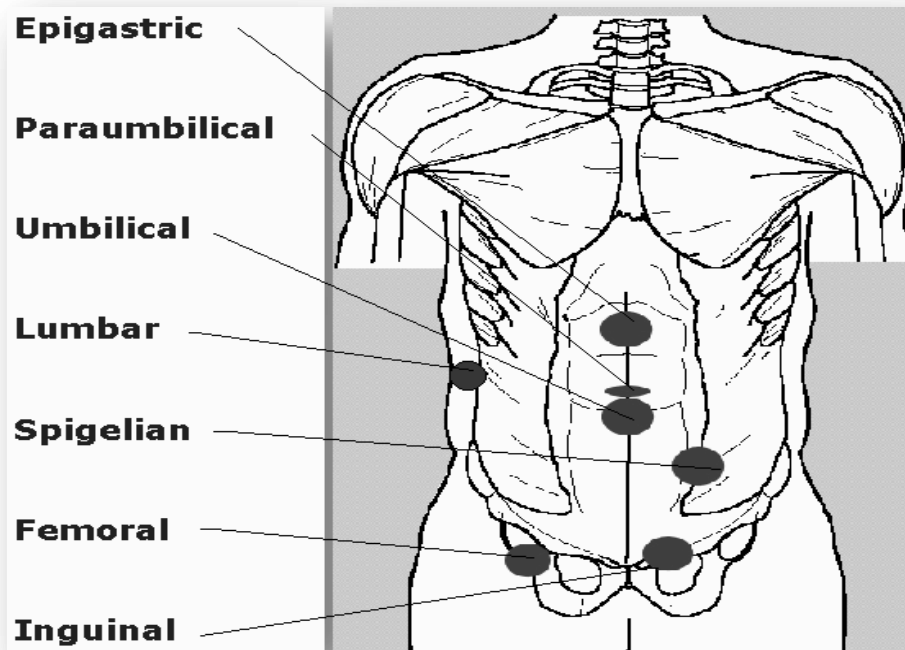
Posterior

➤ Lumbar

- Superior triangle
- Inferior triangle

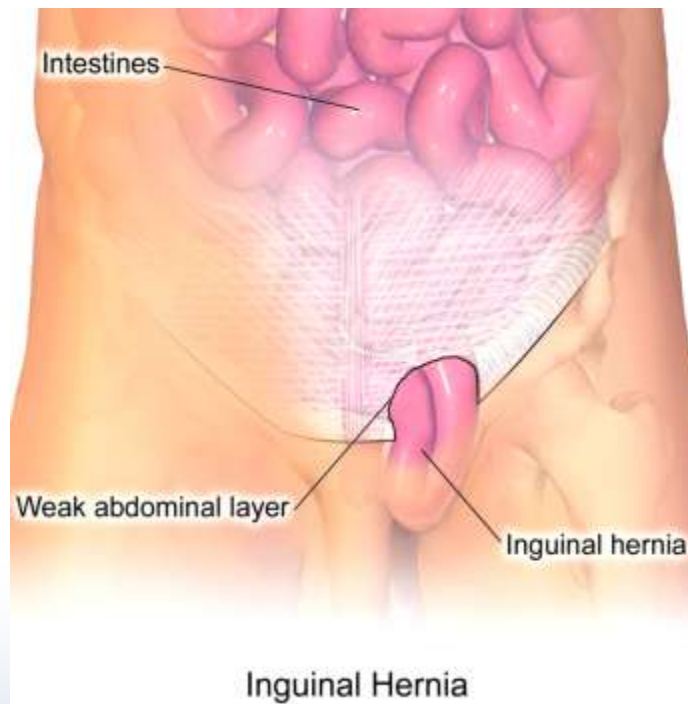
Miscellaneous

- Incisional *



1) Inguinal hernia

- Makes up 75% of all abdominal wall hernias
- Occurring up to 25 times more often in men than women

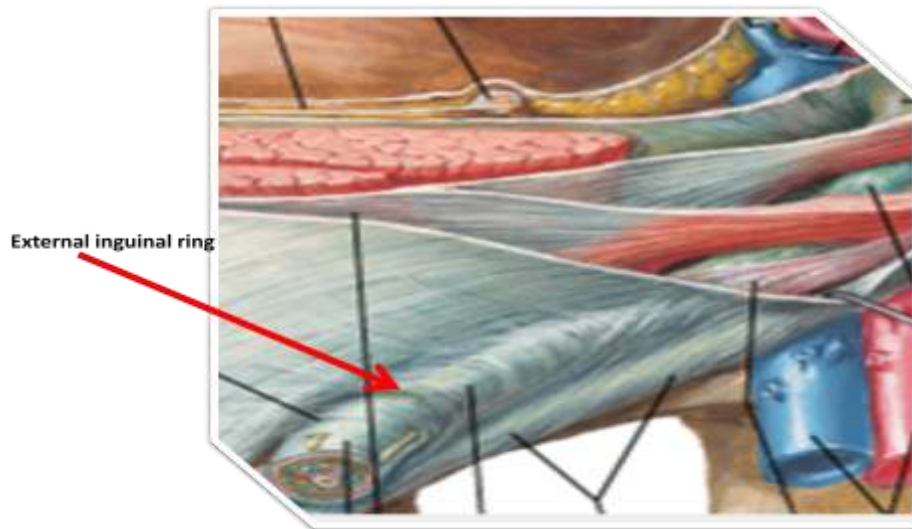


Anatomy of inguinal canal

The canal extends between the internal (deep) inguinal and external (superficial) inguinal rings.

Contents

The inguinal canal contains the spermatic cord, ilio inguinal nerve and the round ligament of the uterus in females.



Spermatic cord contains:

3 arteries:

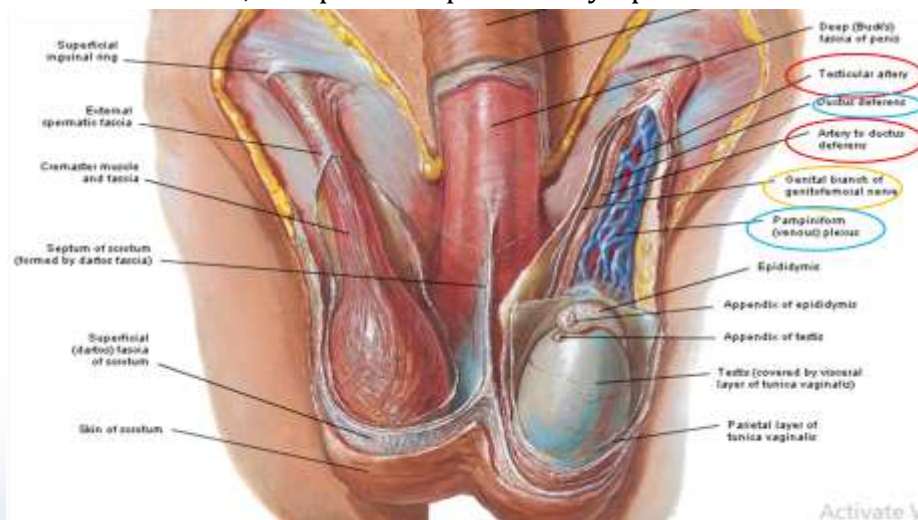
--Testicular, Ductus deferens, Cremasteric artery

3 nerves:

--Cremasteric, Genital branch of the genitofemoral nerve & Autonomics

3 other things:

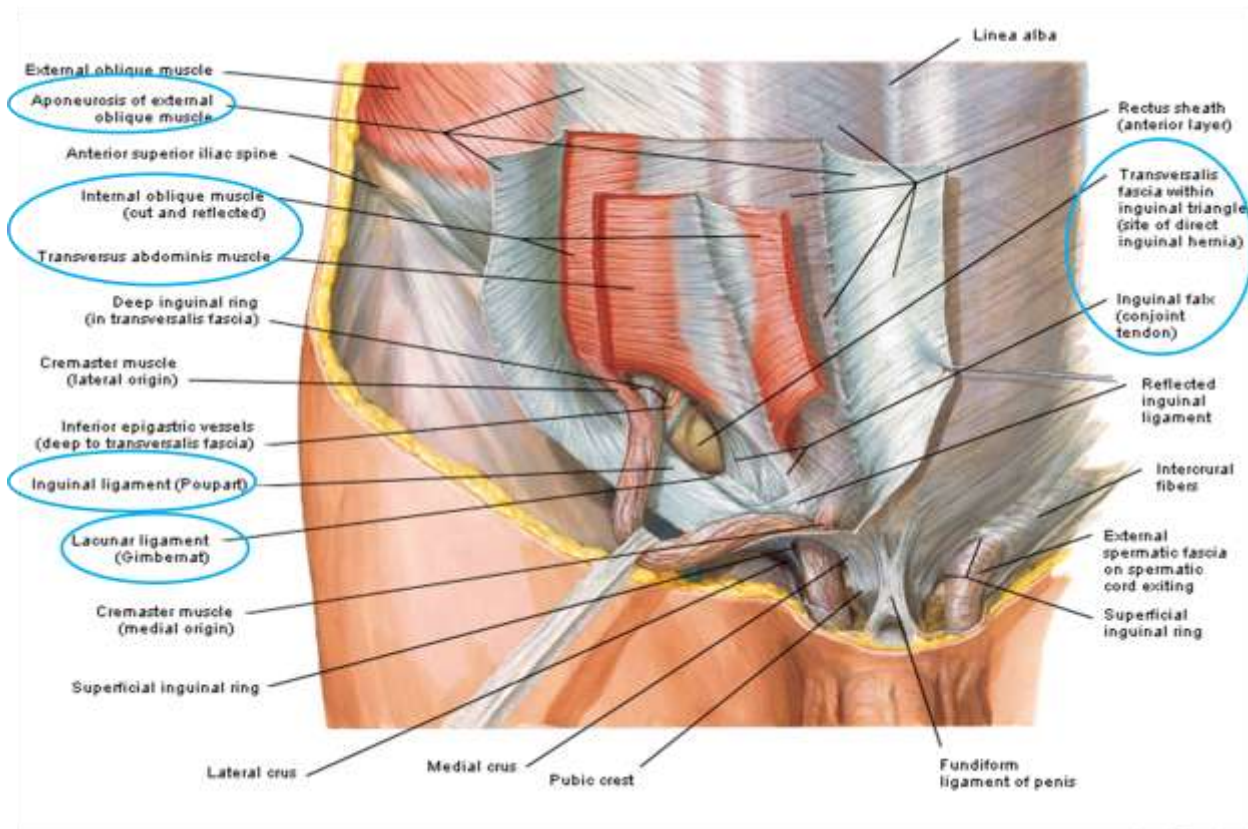
--Ductus deferens, Pampiniform plexus & Lymphatics



Boundaries of inguinal canal

(MALT) ²

- ❖ Superior wall [roof]: 2 Muscles:
 - Internal oblique Muscle
 - Transverse abdominus Muscle
- ❖ Anterior wall: 2 Aponeuroses:
 - Aponeurosis of external oblique
 - Aponeurosis of internal oblique
- ❖ Lower wall [floor]: 2 Ligaments:
 - Inguinal Ligament
 - Lacunar Ligament
- ❖ Posterior wall: 2Ts:
 - Transversalis fascia [laterally]
 - Conjoint Tendon [medially]



Hesselbach's triangle

- ✓ The inferior epigastric vessels serve as its superolateral border,
- ✓ The rectus sheath as medial border, and
- ✓ The inguinal ligament as the inferior border

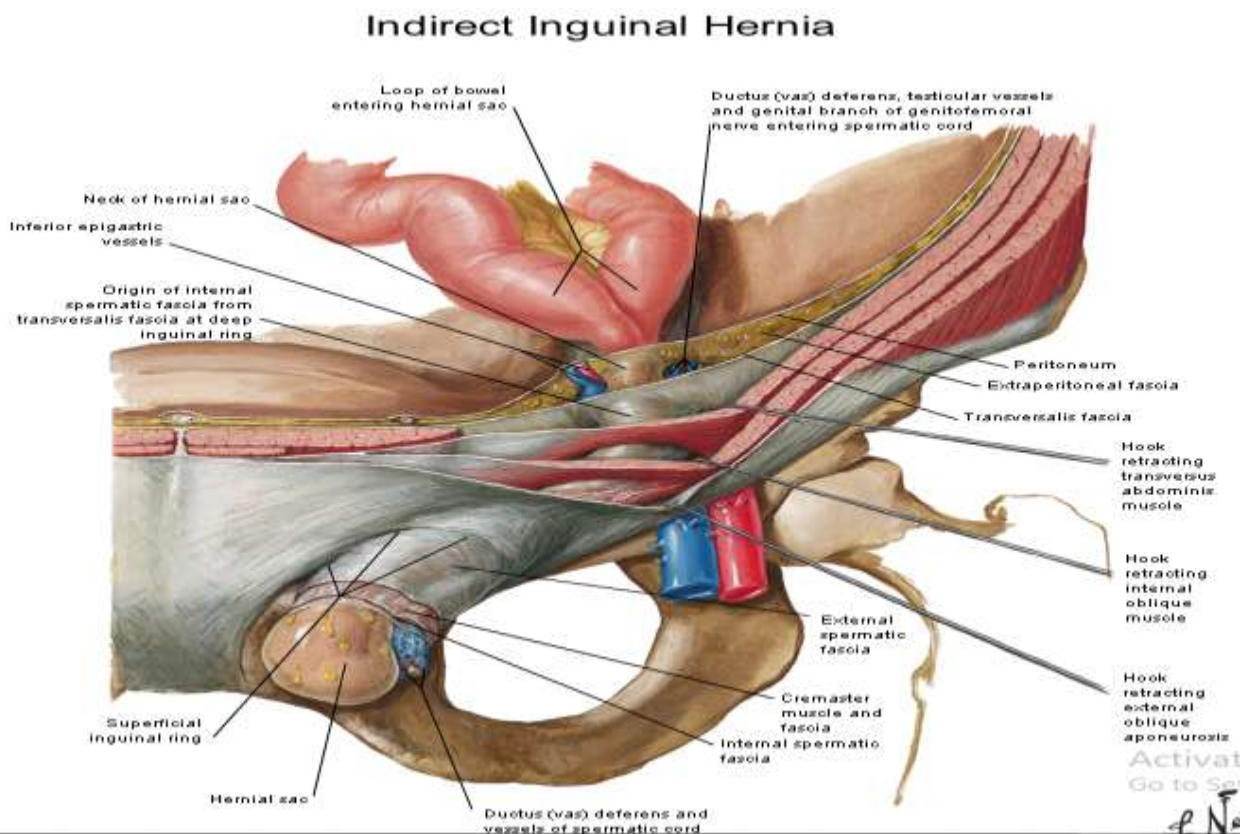
Types of inguinal hernia

- Indirect inguinal hernia (2/3rd)
- Direct inguinal hernia

Indirect inguinal hernia

The sac of an indirect inguinal hernia passes from the internal inguinal ring obliquely toward the external inguinal ring and ultimately into the scrotum

Follows pathway that testicles made during prebirth development. This pathway normally closes before birth but remains a possible place for a hernia.

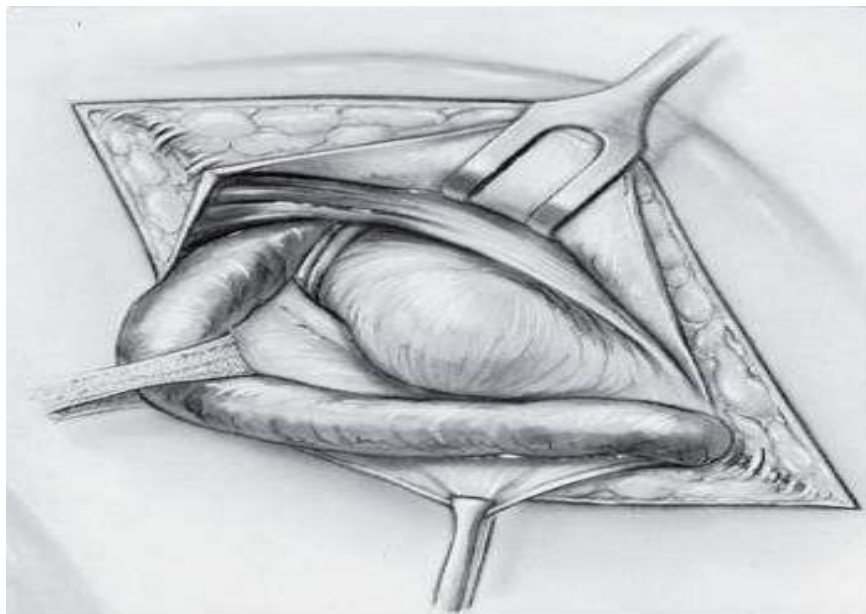




Indirect inguinal hernia

Direct inguinal hernia

This occurs slightly medial to the site for the indirect hernia, in a place where the abdominal wall is naturally slightly thinner. Hesselbach's triangle!



Source: Doherty GM, Way LW: *Current Surgical Diagnosis and Treatment*, 12th Edition: <http://www.accesssurgery.com>
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Diagnosis in a hernia

1. History

- ✓ Complain a bulge in the inguinal region
- ✓ There may be associated pain or vague discomfort in the region, but groin hernias are usually not extremely painful unless incarceration or strangulation has occurred.

2. Physical examination

- ✓ The inguinal region is examined with the patient in both supine and standing positions.

Inspection

- ✓ Standing position
 - It will enable us to see the full extent of the swelling
 - Direct hernias may not be seen on supine position
- ✓ Shape (pyriform, round...)
- ✓ Location (Rt/Lt), Asymmetry
- ✓ Any scars, overlying skin changes
- ✓ Cough impulse (+ve/-ve)
- ✓ Any visible peristalsis



Palpation

- ✓ Temperature
- ✓ Tenderness,
- ✓ Consistency
 - Soft & elastic in case of enterocele
 - Firm & doughy in omentocele
 - Bag of worms in varicocele
- ✓ Reducible or not? If reducible, by itself or manually?
- ✓ Measure(Cms)

Can you get above the swelling? Try to reach above the swelling

- Inguinal hernia--cannot get above
- Pure scrotal swelling--will get above (reading assignment)

Is the pubic tubercle medially or lateral to the swelling?

- Suggests either inguinal or femoral hernia. Lateral to the pubic tubercle in case of femoral hernia & medial in case of inguinal hernia.
- Make your self familiar with identifying the pubic tubercle. Self practice!

Is the inguinal ligament above or below the swelling?

- Above to inguinal ligament in inguinal hernia & below in femoral hernia.
- Self practice! Appreciate your inguinal ligament...ASIS...

Is the testis identifiable?

- Suggests either congenital or acquired hernia
- In case of congenital hernia the testis is full incorporated.

Maneuvers in hernia examination

Maneuver for small swelling

- Put your fingers half the inguinal canal (between deep & superficial inguinal ring)
- Then ask the patient to cough→ You may be able to appreciate the small hernia

Maneuver to identify Femoral, Direct or indirect

i)Zieman's technique

- ☐ Reduce the swelling
- ☐ Finger placement
 - Index finger—deep inguinal ring
 - Middle finger—superficial ring
 - Ring finger—saphenous opening

Click the link below & watch video on how to examine hernia

<https://www.youtube.com/watch?v=JR27rKrhvKE>

- ❑ Then ask the patient to cough
- ❑ Implication
 - Index finger—indirect hernia
 - Middle finger—direct hernia
 - Ring finger---femoral hernia



ii) Internal ring occlusion test

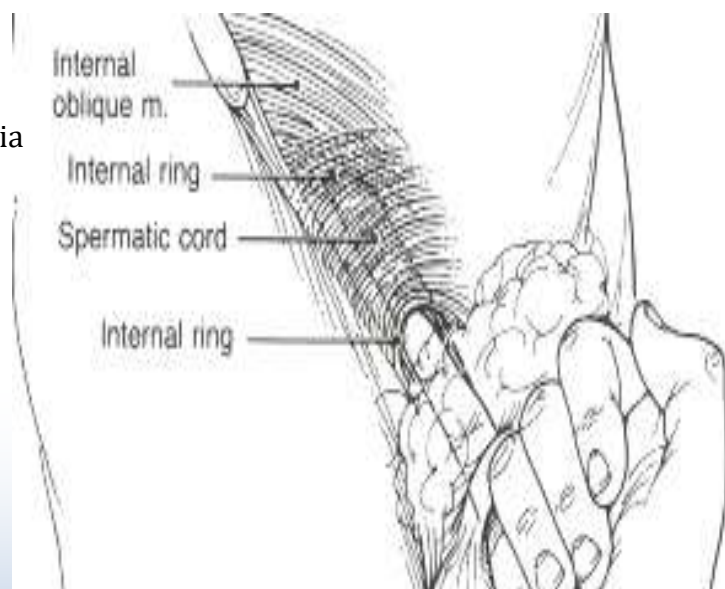
- Reduce the hernia
- Occlusion of the deep ring by thumb
- Then holding the thumb in position ask the pt to stand then cough
- Implication
 - ✓ If no bulging → indirect
 - ✓ If bulging → direct

iii) Invagination test

- A fingertip is placed into the inguinal canal by invaginating the scrotum to detect a small hernia.

-Implication

- ❖ Tip of finger--indirect
- ❖ Pulp of the finger—direct hernia
- ❖ Size of the external ring



Your final assesement should include;

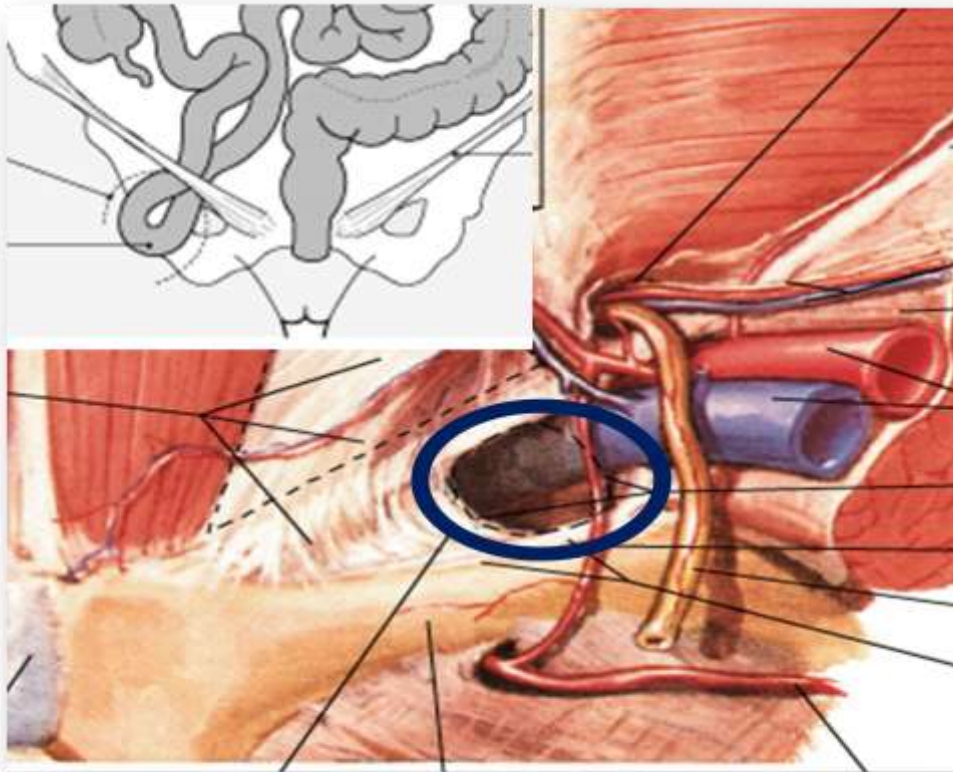
1. Site (inguinal)
2. Right/Left
3. Reducible/Irreducible
4. Complete/Incomplete
5. Direct/Indirect

E.g,

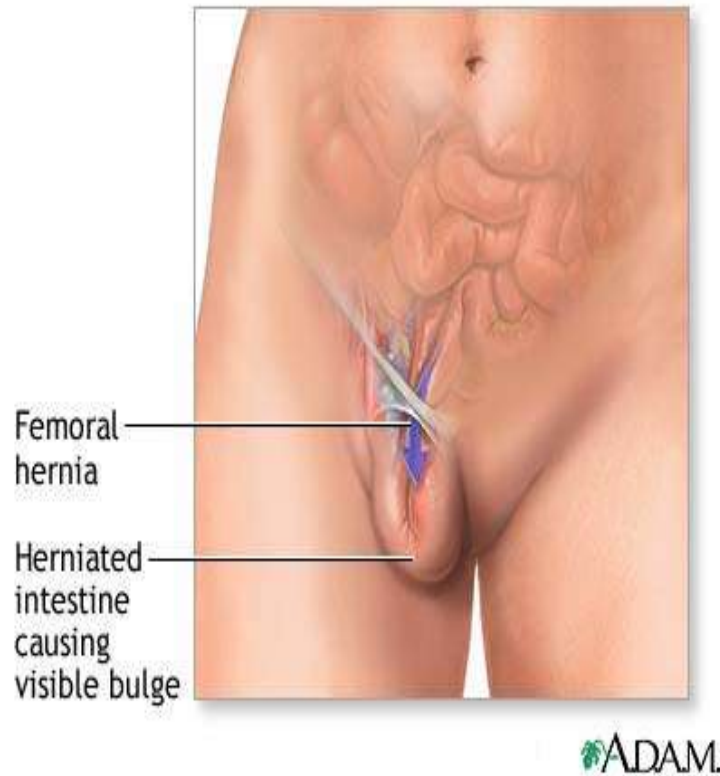
Rt. side incomplete indirect reducible inguinal hernia

2) Femoral hernia

- The femoral canal is the way that the femoral artery, vein, and nerve leave the abdominal cavity to enter the thigh.
- Although normally a tight space, sometimes it becomes large enough to allow abdominal contents (usually intestine) into the canal.
- This hernia causes a bulge below the inguinal



- Rare and usually occurring in women, these hernias are particularly at risk of becoming irreducible and strangulated.



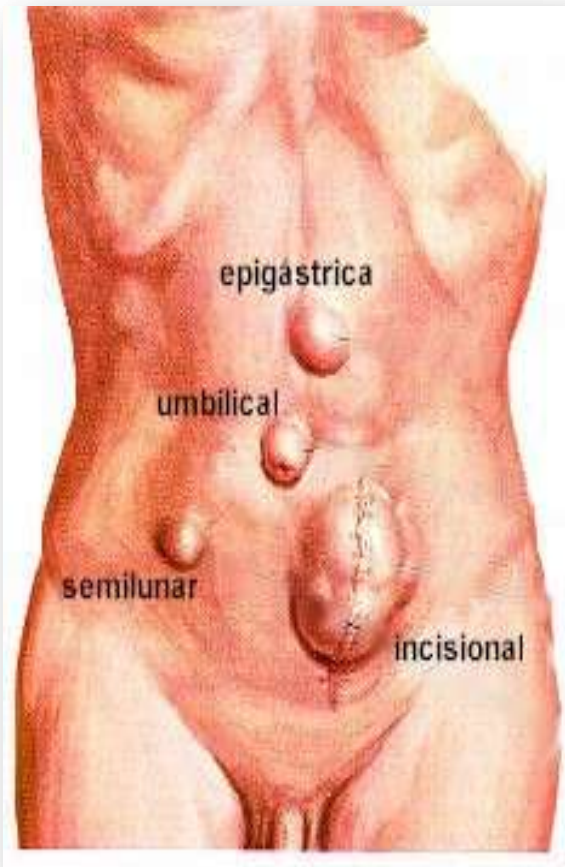
3) Umbilical hernia

- Occur in infants & children.
- Boys affected more than girls.
- Tend to resolve without any treatment by around the age of 5 years.
- Obstruction and strangulation is rare.



4) Incisional hernia

- Abdominal surgery causes a flaw in the abdominal wall that must heal on its own.
- This flaw can create an area of weakness where a hernia may develop.
- This occurs after 2-10% of all abdominal surgeries, although some people are more at risk.
- After surgical repair, these hernias have a high rate of returning (20-45%).



5) Epigastric hernia

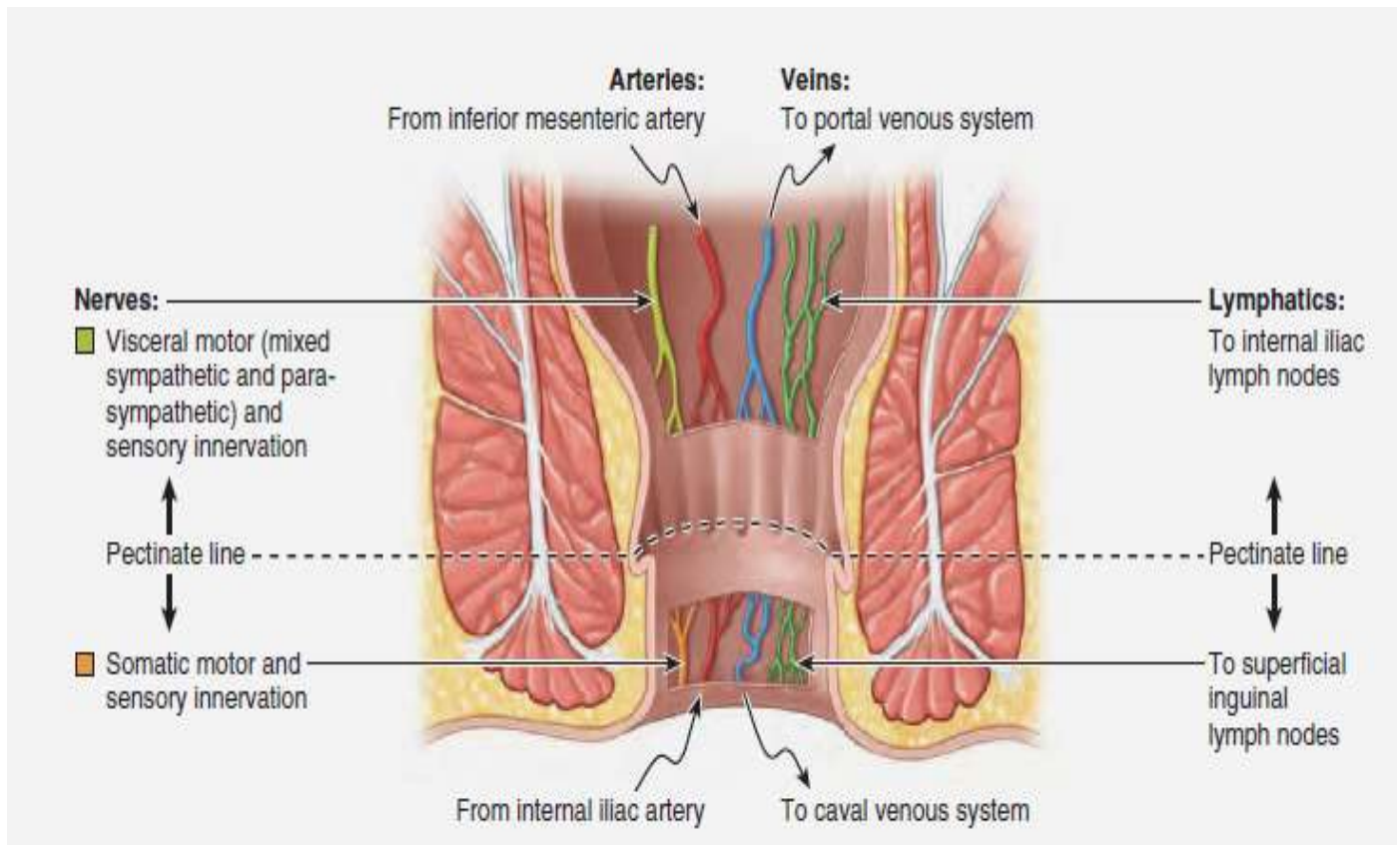
- Occurring between the navel and the lower part of the rib cage in the midline of the abdomen, these hernias are composed usually of fatty tissue and rarely contain intestine.
- Formed in an area of relative weakness of the abdominal wall, these hernias are often painless and unable to be pushed back into the abdomen when first discovered.



Epigastric hernia

Hemorrhoids, anal fissure, fistula in ano & perianal abscess

Anatomy of Anal canal/Recap

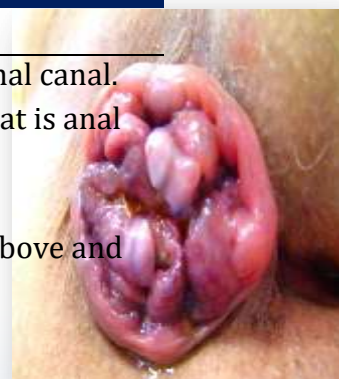


Hemorrhoids

Introduction

Hemorrhoids are normal, vascular tissue within the submucosa located in the anal canal. They are thought to aid in anal continence by providing bulk to the anal canal that is anal Cushing.

Internal hemorrhoids originate above the dentate/pectinate line, while external hemorrhoids occur below this level. Mixed component hemorrhoids can occur above and below the dentate line.



Internal hemorrhoids	External hemorrhoids
▪ Supplied inferior mesenteric ▪ Above the dentate line ▪ Venous to portal system ▪ Columnar epithelium ▪ Visceral innervation... painless	▪ Supplied from pudendal artery ▪ Below dentate line ▪ Venous to systemic ▪ Squamous epithelium ▪ Somatic innervation.... painful

Pathogenesis

- The pathogenesis of symptomatic hemorrhoids is most likely due to the weakening of the connective tissue within these vascular cushions which produces bleeding, with or without prolapsing of the hemorrhoidal tissue.

Predisposing factors

- Why weakness of the connective tissue happens??
 - ✓ low-fiber dietary intake
 - ✓ excessive straining
 - ✓ chronic constipation
 - ✓ prolonged sitting
 - ✓ pregnancy
 - ✓ advanced age
 - ✓ Patients on anticoagulation or antiplatelet therapy this cause seepage with or without protrusion
 - ✓ Internal anal sphincter dysfunction may also play a role in the development of hemorrhoidal disease

Clinical presentation

- Bleeding, protrusion, and pain are among the most common symptoms but Occult blood loss resulting in anemia is rare.
- Patients with grade III or IV internal hemorrhoids may also complain of a sensation of fullness, fecal incontinence, and constant mucous discharge.
- Acute thrombosed internal or external hemorrhoids are usually associated with severe pain and a palpable perianal mass or lump
- Examination of the patient with hematochezia, although tailored by the age of the patient, should include sufficient investigations to rule out a proximal source of bleeding such as inflammatory bowel disease and neoplasia.
- Internal hemorrhoids are generally not palpable on digital examination in the absence of thrombosis. Internal hemorrhoidal bundles appear as bulging purplish-blue veins, and prolapsed internal hemorrhoids appear as dark pink, glistening, and sometimes tender masses at the anal margin.
- Thrombosed external hemorrhoids are acutely tender and have a purplish hue.

- The physical examination includes inspection, DRE and anoscopy
 - ✓ DRE
 - Inspect for perianal areas for excoriations, inflammation, ulcers and lumps
 - Lubricate your gloved index finger, and gently insert your index finger tip into the anal canal in a direction pointing toward the umbilicus
 - Notice for sphincter tone of the anus, tenderness, indurations, irregularities or nodules
 - Insert your finger into the rectum as far as possible
 - Rotate your finger in clockwise and anti-clock wise directions to palpate the rectal surface for nodules, irregularities or indurations
 - Examine the posterior surface of the prostate gland in male patient, and identify its lateral lobes and the medial sulcus
 - Characterize the size, shape, tenderness and consistency of the prostate (normal prostate is rubbery and non tender)
 - Gently withdraw your finger
 - Note for blood staining of fecal matter on the glove
 - Anoscopy

Anoscopy has the advantage of being a quick, relatively painless, inexpensive procedure that can be performed in an unprepped patient to diagnose hemorrhoids and exclude other distal anorectal disorders.

 - If hemorrhoids or other pathology is not found, colonoscopy should be done regardless of age, to rule other pathologic sources for the rectal bleeding.

Based on the physical and history can be classified

- **Grade I**
Visualized on anoscopy and may bulge into the lumen but do not prolapse below the dentate line, bleed during defecation
- **Grade II**
Prolapse out of the anal canal with defecation or with straining but reduce spontaneously, bleed and secrete mucus
- **Grade III**
Prolapse requiring manual reduction
- **Grade IV**
Prolapse that cannot be manually reduced, generally mixed hemorrhoids that include internal and external components

Grade	Extent of prolapse
1 st degree	Hematochezia, No visible prolapse
2 nd degree	Spontaneously reducible prolapse
3 rd degree	Manually reducible prolapse
4 th degree	Irreducible prolapse, at risk of strangulation

Thrombosed external



Internal, first-degree internal



Internal second degree



Internal third degree



Internal fourth degree



Management of hemorrhoids

Nonsurgical treatment

- Life modification
 - The initial and most efficacious nonoperative treatment is fiber diet, fluid intake
 - avoiding prolonged sitting or straining on the toilet, perianal hygiene, and avoiding foods or other triggers for constipation
- Topical anti hemorrhoid creams for symptomatic relief only , never modify the pathophysiology of the diseases. These preparations include local anesthetics, vasoactive agents, and corticosteroids
- The main goal of nonoperative treatment isto reverse the pathology of hemorrhoidal disease and to provide symptomatic reliefbut it is time taking and needs patient compliance
- Indicated for low grade 1 & 2

Office-based surgical procedures

- Rubber Band Ligation
- Sclerotherapy
- Infrared and laser coagulation

Surgical management

- Excisional Hemorrhoidectomy
- Milligan-Morgan or open hemorrhoidectomy
- Closed Ferguson hemorrhoidectomy

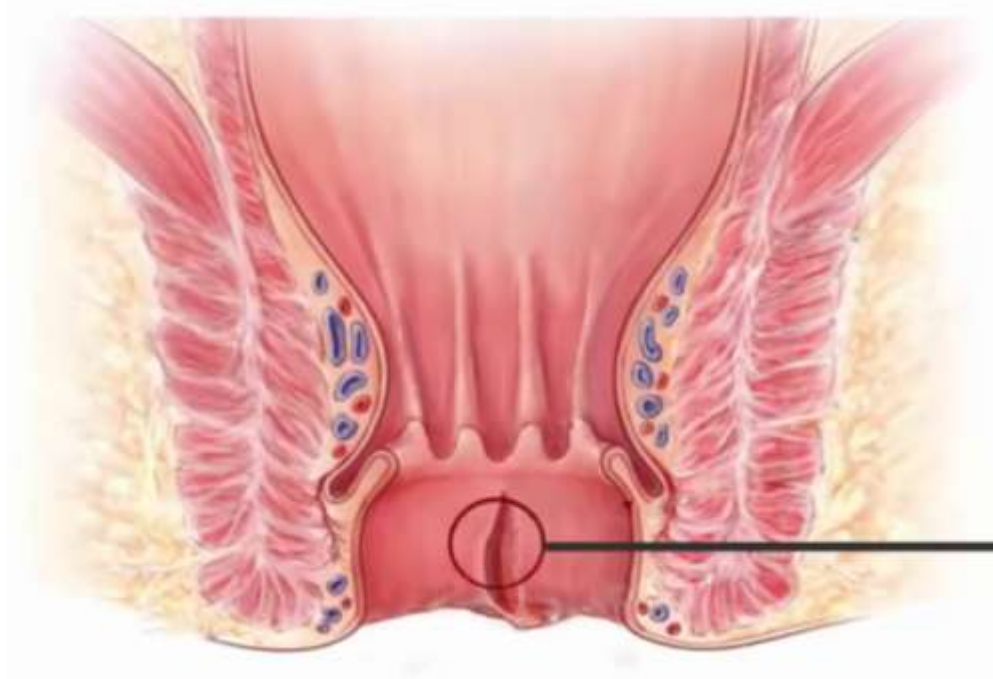
Complications of hemorrhoidectomy

- Post-op pain
- bleeding
- Urinary retention
- Infection
- Transient incontinence
- Anal stenosis

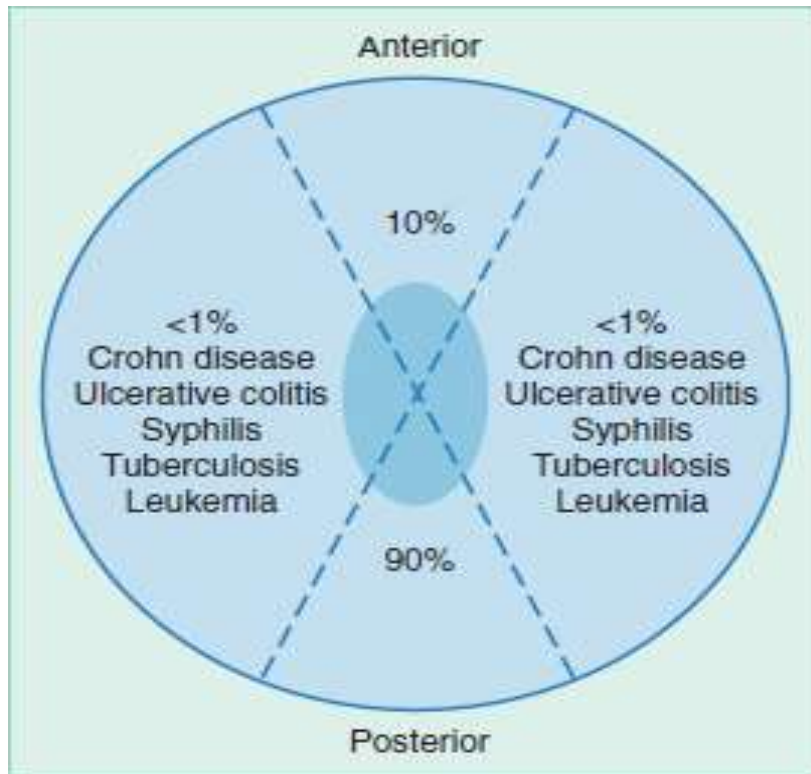
Anal fissure

Introduction

- Anal fissure is described as a linear defect, or laceration, in the anoderm, located between the dentate line and the anal verge



- It can be acute or chronic, An acute fissure is a simple laceration, whereas a chronic anal fissure is an ulceration with built-up scarred edges and exposed internal anal sphincter muscle fibers at its base
- Chronic fissure (>6week) is defined by these three findings;
 - Visible muscle,
 - A skin tag (sentinel tag), and
 - Hypertrophied papilla proximal to the fissure.
- Importantly, acute and chronic anal fissures are almost always located in the midline, with the posterior location predominating. Why?? Because low blood supply and more stress during sitting. However, in 10% of women and in 1% of men, anal fissure can be seen in the anterior midline.
- Fissures located off the midline are usually associated with more serious systemic diseases such as Crohn disease and immunodeficiency syndromes(AIDS, syphilis ,TB and malignancy) those generally causes atypical anal fistula characterized by...
 - ✓ Fissures off the true midline,
 - ✓ shaggy large defects with undermined edges and
 - ✓ granulation tissue in the base



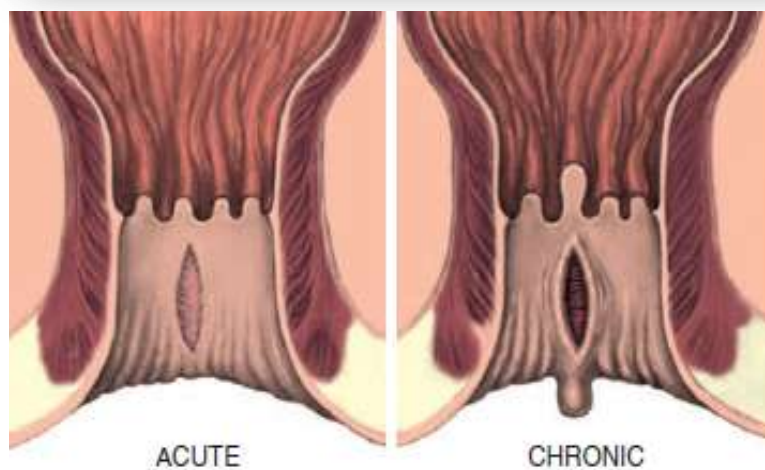
- Why these patients always experience excruciating anal pain? Because of its location extending onto the very sensitive anoderm somatic innervations that mean distal to dentate line with defecation and bleeding.
- In the immunocompromised patient, a fissure or an ulcer and a concomitant mass should raise the question of malignancy. Lymphoma, leukemic ulcer, and anal canal epithelial tumors are often associated with surface defects

Etiology

- Trauma to the anal canal, because of passing hard stools, is probably the most frequent cause of anal fissure
- Patients will often remember the exact time the fissure developed based on the symptoms. Classically, this will almost always be associated with preceded episodes of constipation.
- Infections also cause anal fissure
 - ❖ TB, syphilis
 - ❖ STI associated with immunosuppression (HSV, CMV, chancroid, chancer)....those causes atypical anal ulcers
 - ❖ The treatments for these disease processes are different, and therefore it is important to recognize the differences between anal canal fissures and atypical anal canal ulcers

Clinical presentation

- Tearing, burning type of pain that lasts minutes up to hours in acute case and described as a cutting or tearing sensation during the act of defecation.
- Blood on toilet soft tissue
- Chronic fissure → visible muscle, a skin tag (sentinel tag), and hypertrophied papilla proximal to the fissure
- May also present with lump, discharge from the open wound, pruritus...
- Examination must be carefully performed; the pain caused by an aggressive examination of an anal fissure is not easily forgotten by the patient
- Simple spreading of the buttocks to gently roll open the anal verge will usually demonstrate the fissure



Anoscopy and complete endoscopy should be postponed and done after the fissure is healed.

Investigations

- AFB
- VDRL or dark-field wet prep microscopy
- PICT
- Colonoscopy
- From ulcer→ biopsy, culture

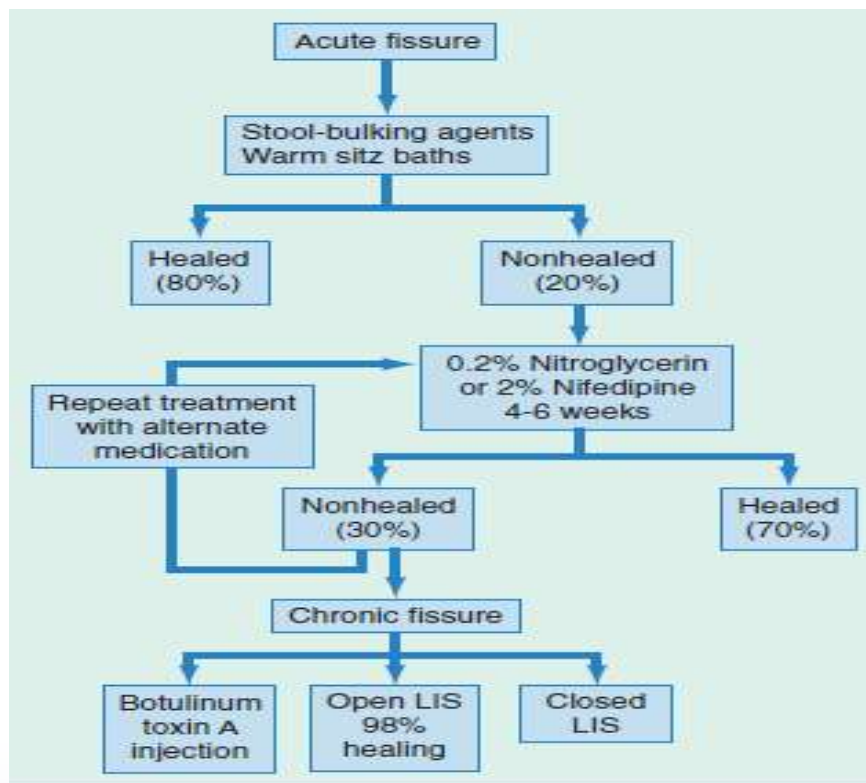
Management of anal fissure

First-line therapy to minimize anal trauma includes

- Bulk agents/high fiber food
- Warm sitz baths
- Stool softeners
- Analgesic creams
- Vasodilators /topical

Surgical therapy

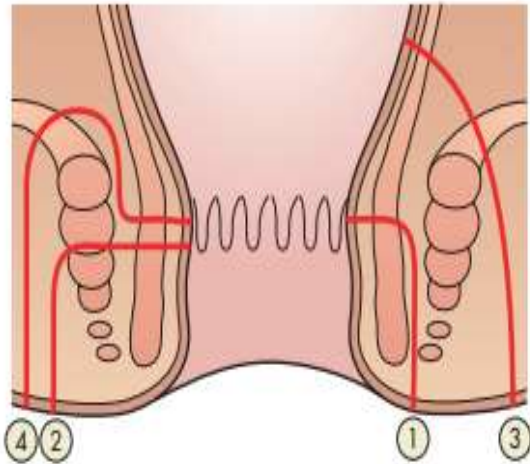
- Recommended for chronic fissures that have failed medical therapy
- Lateral internal sphincterotomy is the procedure of choice. The aim of this procedure is to decrease spasm of the internal sphincter by dividing a portion of the muscle



Fistula in ano/ anorectal fistula

Fistula in ano is an abnormal tract or cavity with an external opening in the perianal area by identifiable internal opening.

While the majority of fistulas are cryptoglandular in origin, trauma, Crohn's disease, malignancy, radiation, or unusual infections (tuberculosis, actinomycosis, and chlamydia) may also produce fistulas.



Risk factors

- ✓ Previous anorectal abscess
- ✓ Cryptoglandular infection (Infection → abscess → fistula)
- ✓ Trauma
- ✓ CD (Crohn's disease)
- ✓ Anal fissures
- ✓ Anal carcinoma
- ✓ Radiation therapy for prostatic ca.
- ✓ Actinomycosis
- ✓ TB
- ✓ LGV

Clinical presentation

History

- ✓ Perianal discharge, Pain, Swelling
- ✓ Bleeding, Diarrhea
- ✓ Skin excoriation

Physical examination

Look for

- ✓ External opening
- ✓ Spontaneous discharge

DRE

- ✓ Fibrous/chord like tract beneath the skin
- ✓ Abscess if not yet drained
- ✓ Indurations

The key points in anal fissure examination is to determine the site of the internal opening; the site of the external opening(s); the course of the primary track; the presence of secondary extensions; and the presence of other conditions complicating the fistula.

Proctosigmoidoscopy may aid the clinical examination.

Full examination under anaesthesia should be repeated before surgical intervention

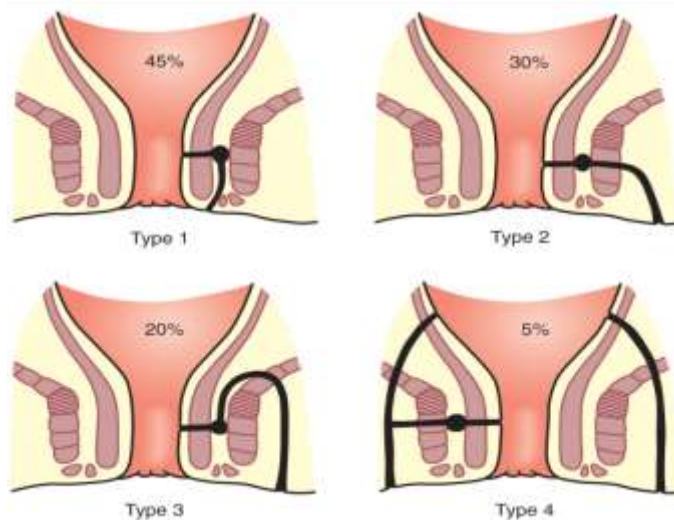
Work up

- Ultrasound is useful to determine whether a fistula is relatively straight forward or not
- Magnetic resonance imaging (MRI) is the 'gold standard' for fistula imaging but due to its limited availability and cost usually reserved for difficult recurrent cases

Classification

Classified according to their relationship to the anal sphincters

1. Inter-sphincteric/most common
2. Trans-sphincteric
3. Supra-sphincteric
4. Extra-sphincteric

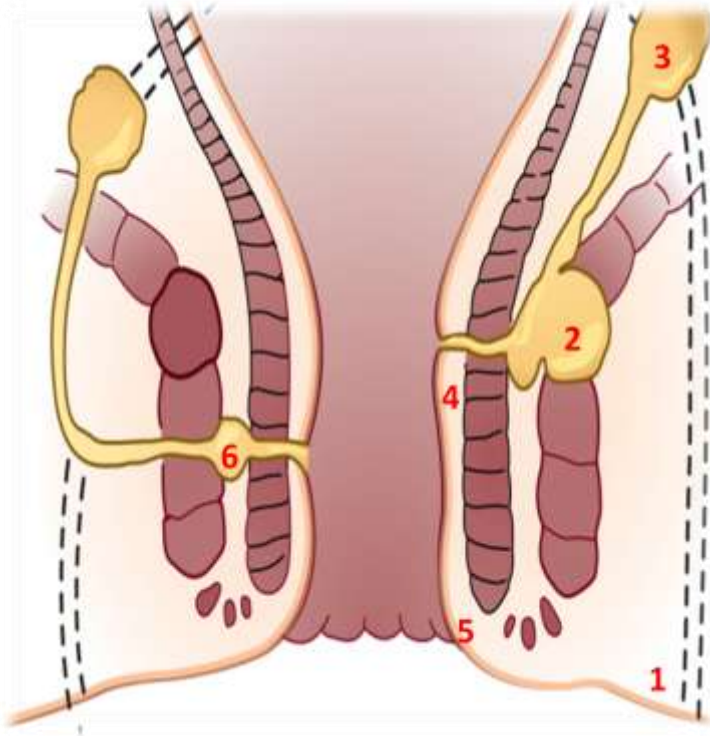


Management

- ✓ Drainage
- ✓ Eradicating the fistulous tract

Ano-rectal abscess

Infection of an anal gland (cryptoglandular infection) which are found in the intersphincteric plane results in the formation of an abscess that enlarges and spreads along one of several planes in the perianal and perirectal spaces.



Anatomical classification

1. Perianal abscess
2. Ischio-rectal abscess
3. Pelvi-rectal supralelevator abscess
4. Submucosal abscess
5. Marginal abscess
6. Inter-sphincteric abscess

Perianal abscess

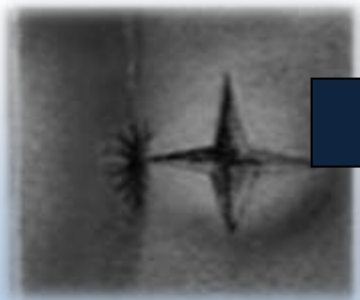
- ✓ It is the most common manifestation of ano-rectal abscess and appears as a painful swelling at the anal verge
- ✓ Often detected by inspection or by digital rectal examination. Examination reveals an indurated, hot, tender perianal swelling.
- ✓ Occasionally, patients will present with fever, urinary retention, or life threatening sepsis
- ✓ Underlying conditions include fistula-in-ano (most common), Crohn's disease, diabetes, immunosuppression...exist

Investigation

- Blood sugar level
- PICT
- CBC

Principles of mgt

- Anorectal abscesses should be treated by drainage as soon as the diagnosis is established with appropriate antibiotic coverage.
- Lack of fluctuance should not be a reason to delay treatment



Chapter 18

GENITO-URINARY
RELATED

URETHRAL CATHETERIZATION

Indications

- Therapeutic
 - ✓ Acute Urinary Retention(AUR)
 - Due to BPH or Blood clots
 - ✓ For continuous bladder irrigation
 - ✓ Chronic urinary retention that cause hydronephrosis
 - ✓ Hygienic care for bedridden pts with incontinence
 - ✓ To Instill medication
- Diagnostic
 - ✓ For collection of uncontaminated urine sample
 - ✓ To monitor urine output
 - ✓ Imaging of the urinary tract

Contraindications

- In presence of traumatic injury to lower urinary tract
E.g. Urethral tear→this condition must be suspected in patients with a pelvic or straddle-type injury.
- Relative contraindication
 - Urethral stricture
 - Recent urethral or bladder surgery

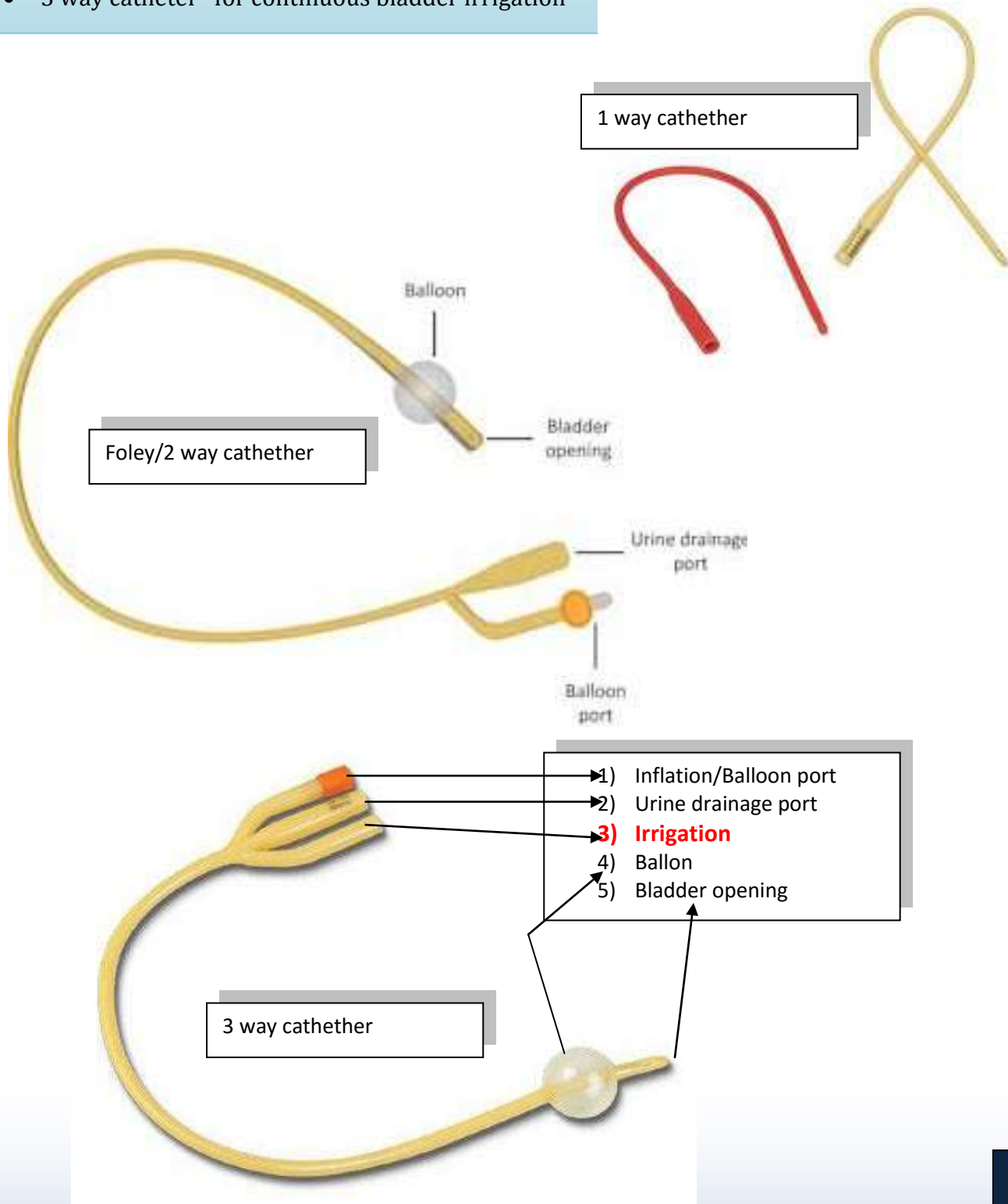
Complications

- Urinary tract infection/UTI
- Urethral stricture
- Urethral perforation
- Bleeding
- Paraphimosis
- Non deflating retention balloon
- Chronic catheterization may lead to...
 - Bladder calculi formation
 - Bladder cancer

Click the link below & watch video on urethral catheterization of
Male-<https://www.youtube.com/watch?v=hhGqHDHIVXo>
Female-<https://www.youtube.com/watch?v=JaT3ZWB2OHw>

Types of catheter

- condom catheter
- straight catheter(1 way)
- Foley catheter (2 way)
- 3 way catheter--for continuous bladder irrigation



Procedural note on urethral catheterization

Materials

- ✓ Sterile gloves, sterile dressing, sterile draping sheet
- ✓ Catheter, urine bag
- ✓ lubricant, antiseptic soln.
- ✓ syringe, distilled water
- ✓ lidocaine

Procedure

- ✓ Explain the procedure to the patient
- ✓ Position → supine with legs apart (frog leg position with knees flexed)
- ✓ Scrub and put on sterile gloves
- ✓ Clean the genitalia with antiseptic solution and drape the area
- ✓ Clean the glans penis with antiseptic solution
- ✓ Using a syringe with no needle, instill 5-10 mL of lidocaine 2% into the urethra. Place a finger on the meatus to help prevent spillage. Allow 2-3 minutes before proceeding with the urethral catheterization (not the usual experience in our setup)
- ✓ The penis is held vertically upwards to straighten the penile urethra
 - by encircling a sterile gauze around the penis
- ✓ The lubricated Foley's catheter is then pushed gently through the external urethral meatus and gradually advanced till it reaches the bladder
 - urine will be seen coming out through the catheter
- ✓ Advance the catheter a little further
- ✓ Inflate the balloon with 10 mL of sterile water
- ✓ After the balloon is inflated the catheter is gently pulled outward to confirm that the balloon is properly inflated
- ✓ Connect the catheter to the urine bag

What may be the cause of failure to insert a catheter?

- Size of the catheter
- Urethral stricture
- Bladder neck contracture
- Tight phimosis
- Meatal stenosis

SUPRAPUBIC CYSTOSTOMY

Indications

1. Acute urinary retention
 - When urethral catheter cannot be passed secondary to urethral stricture, bladder neck contractures...
2. Urethral trauma
 - Have high index of Suspicion of urethral trauma in patients with pelvic fracture/Saddle-type injuries
 - Triad (Clinical presentation)
 1. Blood at urethral meatus
 2. Inability to urinate &
 3. Palpably distended bladder
3. Management of complicated lower genito-urinary tract infection
 - E.g. acute bacterial prostatitis
4. Requirement for long term urinary diversion
 - E.g. neurogenic bladder

Contraindications

1. Absolute
 - a. If the bladder is not distended
 - b. History of bladder cancer
2. Relative
 - a. Coagulopathy
 - b. Previous lower abdominal or pelvic surgery
 - c. Pelvic cancer

Complications

- Extravasation (Intraperitoneal & or extraperitoneal)
- Obstruction (by blood, mucous, kinking...)
- Exit site infections or leakage
- Serious complications of the procedure include bowel perforation and other intra-abdominal visceral organ injuries
- Possible complications from long-term catheter use include; recurrent symptomatic UTI, bladder and renal calculi formation, deterioration of renal function, and increased risk of bladder cancer

Suprapubic cystostomy techniques

- 1) Open surgical technique
- 2) Percutaneous Suprapubic Cystostomy

Click below & watch → open surgical cystostomy video

https://www.youtube.com/watch?v=GoHLB3j_ZAA





SCROTAL SWELLING

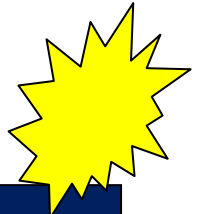
DDX for scrotal swelling

- ✓ Hernia (read under short cases of Debol about hernia)
- ✓ Hydrocele (read below...)
- ✓ Hematoma
- ✓ Epididymal cyst
 - Cystic degeneration of the epididymis filled with crystal clear fluid.
- ✓ Testicular torsion (read below...)
- ✓ Epididymo-orchitis (read below...)
- ✓ Testicular tumor
- ✓ Varicocele
 - It is a result of dilation of veins that drain into the internal spermatic veins
 - Left side → most commonly affected
 - Examination of a man with varicocele when he is upright reveals a mass of dilated, tortuous veins lying posterior to and above the testis. It may extend up to the external inguinal ring, and the Valsalva maneuver can increase the degree of dilation
- ✓ Syphilitic gumma...

How to differentiate?

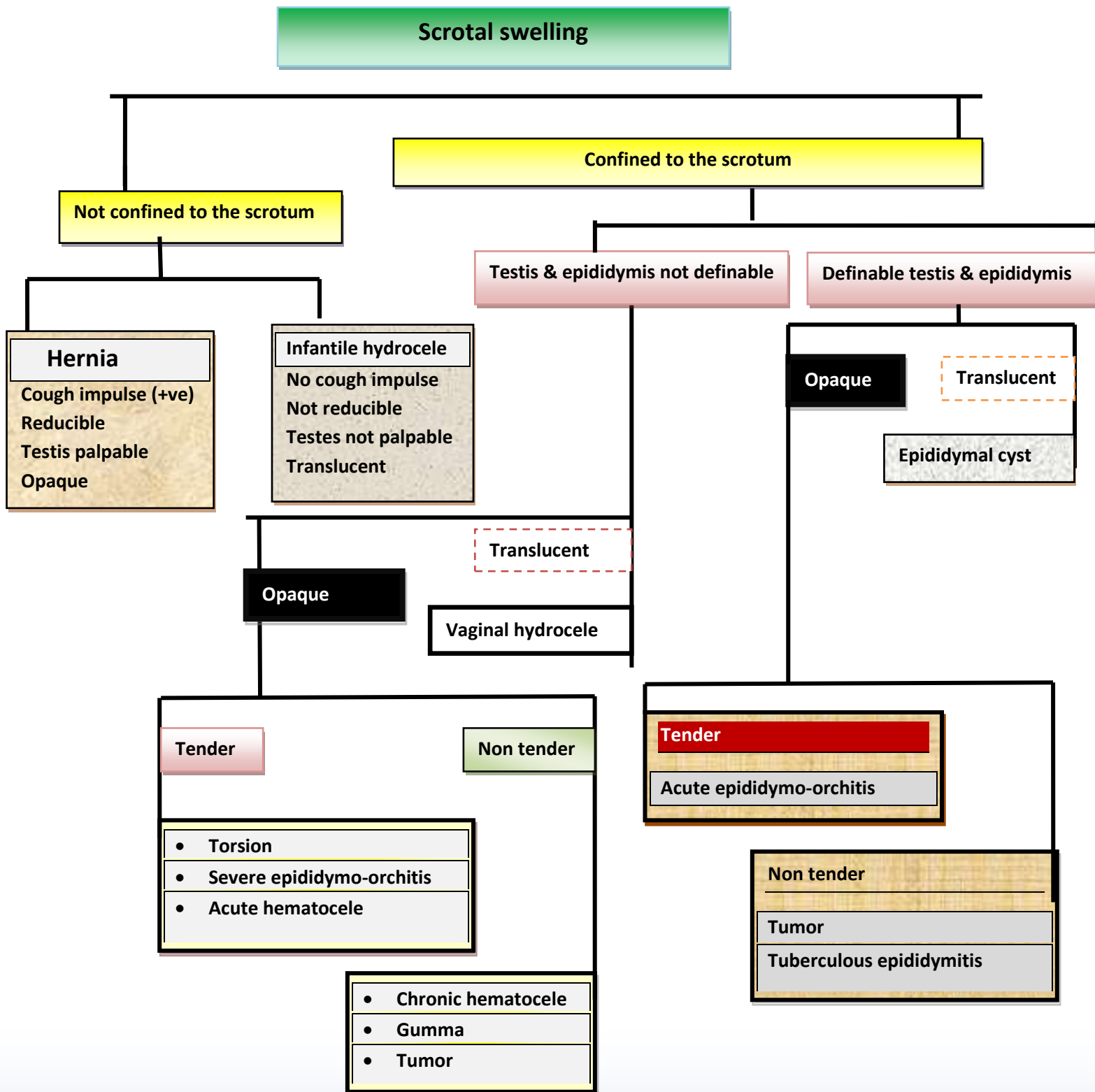
Answer the following during your Physical Examination...

1. Can you get above the swelling?
2. Can you identify the testis & epididymis?
3. Is the swelling translucent?
4. Is the swelling tender?



Click the link below & watch video on how to examine scrotal swelling @
<https://www.youtube.com/watch?v=CCpxEePuc24>

Then use the algorithm below...



Hydrocele

A hydrocele is an abnormal collection of serous fluid in a part of the processus vaginalis, usually the tunica.

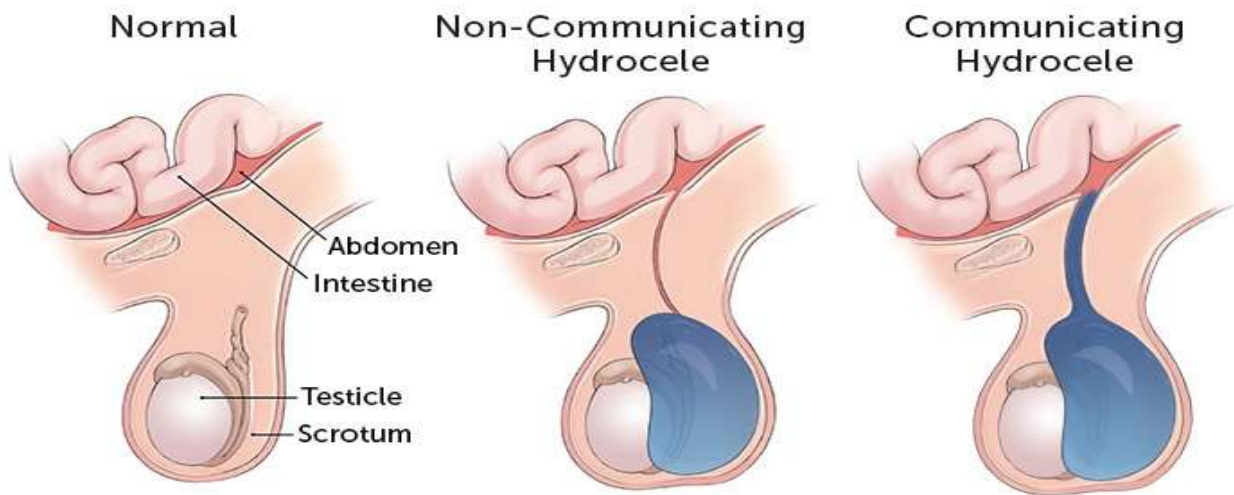


Produced in four different ways;

1. By excessive production of fluid within the sac
2. By defective absorption of fluid
3. By interference with lymphatic drainage of scrotal structures
4. By connection with the peritoneal cavity via a patent processus vaginalis (congenital).

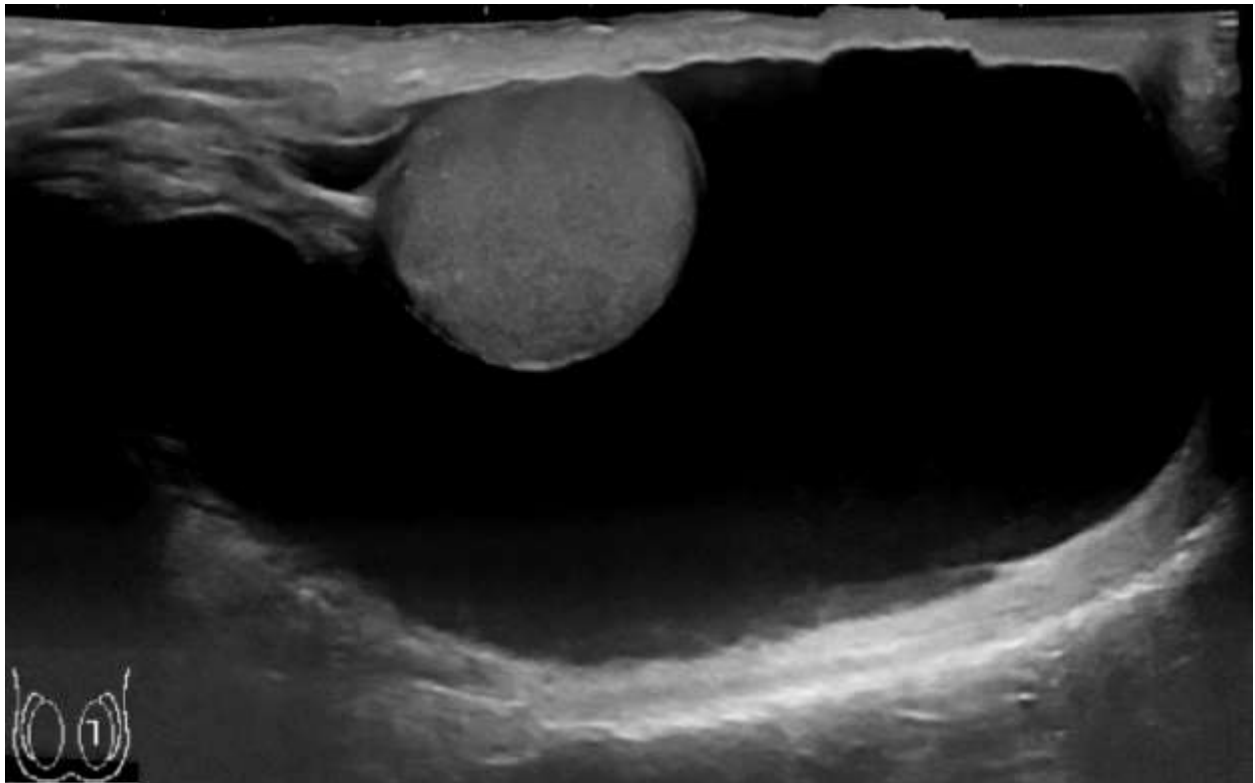
Transillumination test





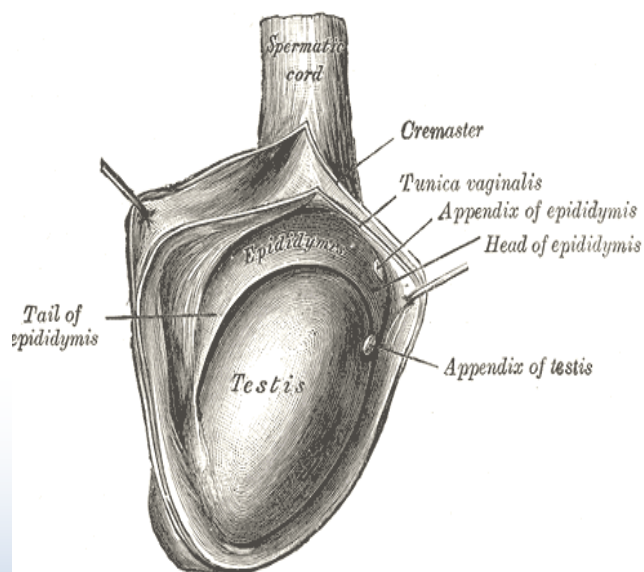
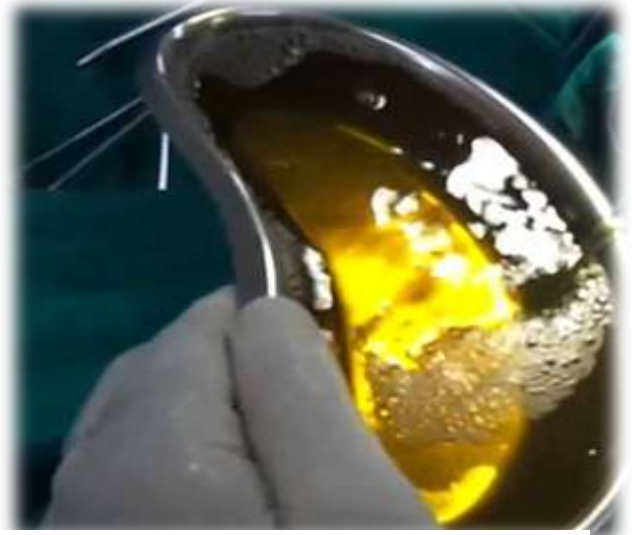
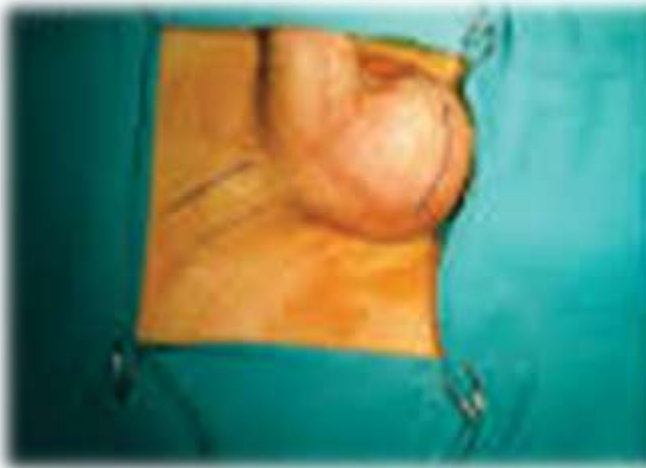
Most communicating hydroceles spontaneously close by 1 year of age. However, persistent communicating hydroceles and presence of bowel content within the hydrocele sac may require surgical correction.

Appearance on ultrasound



Management

Surgical correction is only required if the patient has symptoms secondary to the size of or discomfort associated with the hydrocele



Eversion of Sac

Testicular torsion



- Testicular torsion refers to the twisting of the spermatic cord structures resulting in subsequent loss of blood supply to the epididymis & the testis.
- a urologic emergency that requires rapid diagnosis and intervention in order to maintain viability of the testicle

Predisposing factors for torsion

- Undescended testis
- Testicular tumor
- “bell clapper deformity”--High investement of the tunica vaginalis causes the testis to hang within the tunica vaginalis like a clapper in a bell

Clinical presentation

History

- ✓ Sudden onset of testicular pain
- ✓ Subsequent swelling
- ✓ The patient feels nauseated & may vomit

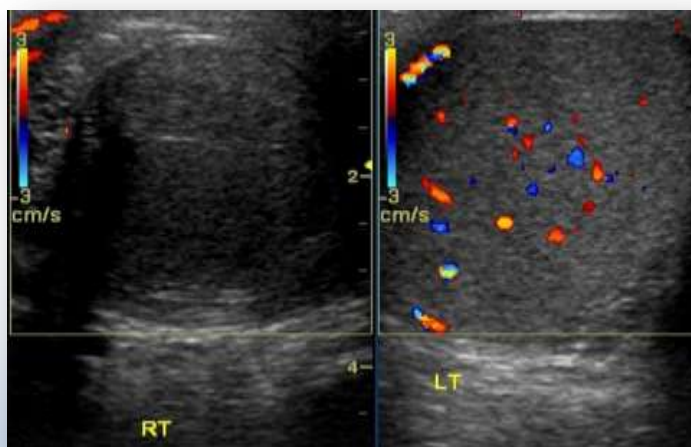
Physical examination

- ✓ Inspection--Swollen, asymmetric scrotum
- ✓ Palpation
 - Tender, firm testicle
 - high riding testicle
 - may have horizontal lie of testicle
 - The posteriorly positioned epididymis may be positioned differently
 - No pain relief with elevation of the testicle→makes it worse
 - The spermatic cord may appear thickened
 - cremasteric reflex→lost



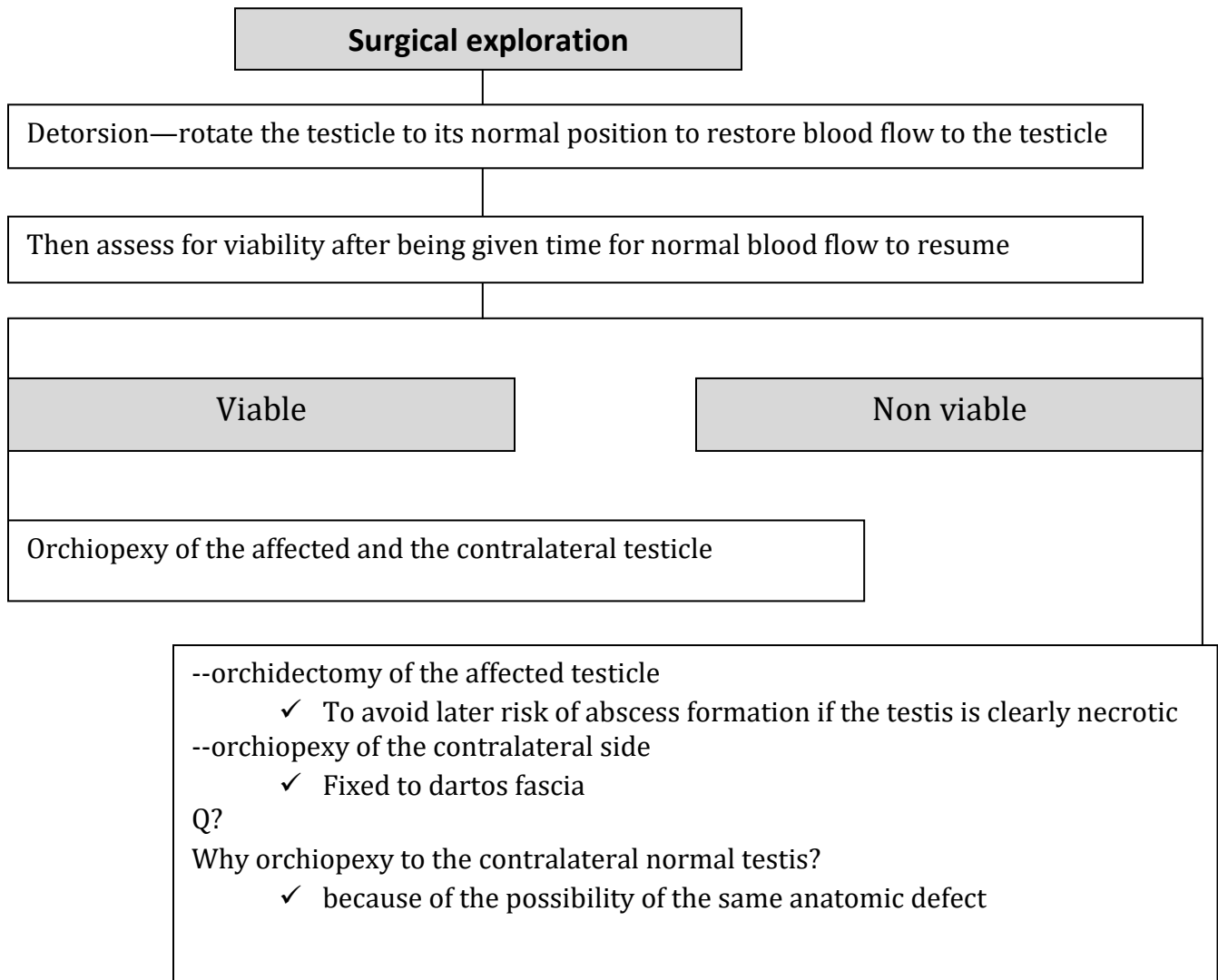
Investigation

- ✓ Doppler scrotal ultrasound
 - What to look? Decreased/absent intra-testicular blood flow on the affected testis
 - Other advantages--Can rule out an associated testicular tumor
- NB*Diagnostic testing should not delay treatment

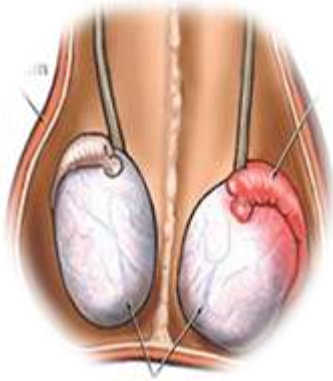


Management

- Immediate surgical exploration
 - Early diagnosis and treatment are vital to saving the testicle and preserving future fertility.
 - The rate of testicular viability decreases significantly after 6 hours from onset of symptoms
 - Immediate surgical exploration (<6hrs after the onset of symptoms) can salvage an ischemic testis in >80% of pts
 - if 12hrs lapsed the rate decreases to 20%



Epididymo-orchitis



Inflammation confined to the epididymis is epididymitis. when it spreads to the testis--called epididymo-orchitis

Causes

Epididymo-orchitis is typically a result of bacterial infection originating in the urinary tract.

Generally it arises from a genital infection in sexually active young men with the commonest causative microorganism being chlamydia trachomatis. But in older male patients it arises from urinary infection (as in case of BOO) or secondary to urethral catheterization

Clinical presentation

- History
 - ✓ Unilateral scrotal pain
 - ❖ The onset is fairly rapid, but not as sudden as testicular torsion
 - ✓ swelling of the epididymis & or testis
 - ✓ Often with fever
 - ✓ May have initial symptoms of urinary or genital infection
- Physical examination
 - ✓ The scrotum wall becomes red, edematous & shiny
 - ✓ May become adherent to the epididymis
 - ✓ Elevation of the testis reduces the pain

Investigation

- CBC
 - WBC count is often raised
- Ultrasound
 - ✓ May provide supporting evidence
 - ❖ Such as increased blood flow to the epididymis
 - ❖ A reactive hydrocele may be present
 - ❖ Reduced testicular blood flow→In case of ischemic orchitis 2^o to intratesticular infection
- Urine analysis(U/A) →pyuria, leukocytosis

Management

- Antibiotics
 - ✓ Route-peros/P.O.
 - ❖ If the patient is not markedly febrile & is otherwise stable
 - ✓ Route-Parenteral→hospitalization
 - ❖ If high fever or significantly elevated WBC or if hemodynamically unstable

- ✓ Initial Drug of choice/DOC in young patients
 - ❖ Doxycycline or
 - ❖ QuinoloneContact tracing → Rx if necessary
- ✓ Initial DOC in elderly patients
 - ❖ Quinolone
 - ❖ If evidence of systemic sepsis
 - ✓ IV antibiotics directed at urinary pathogens
- ✓ Duration
 - ❖ It should continue for at least 02 weeks or until the inflammation subside
- Other measures
 - ✓ All patients should drink plenty of fluid
 - ✓ Scrotal support
 - ✓ Analgesics
- Surgery
 - ✓ Drainage indicated if suppuration occurs

HYPOSPADIA, EPISPADIA & BLADDER EXSTROPHY

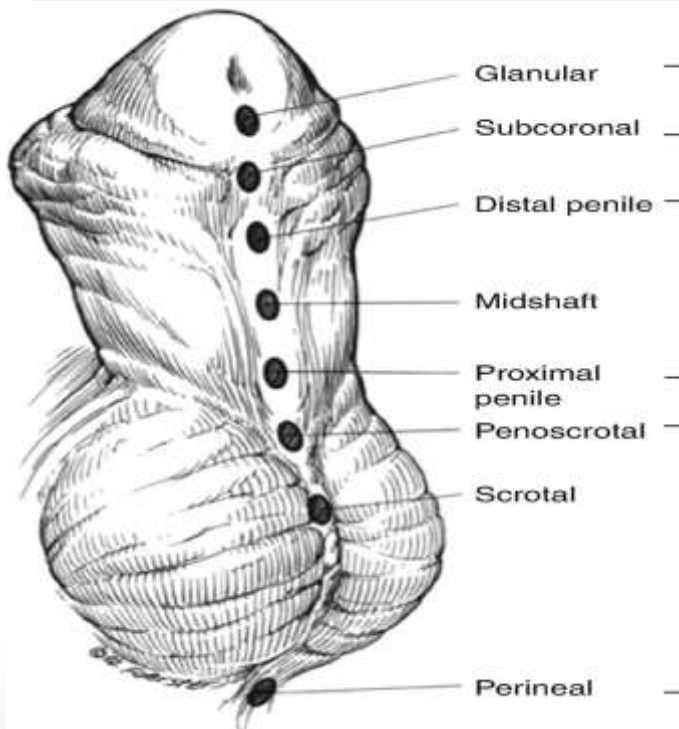
Hypospadias

- ✓ The most common congenital abnormality of the urethra in males in which it opens abnormally on the ventral surface of the penis (1 in 200–300 male live births).



- ✓ There are 3 characteristic features
 - external meatus opens on the underside of the penis or the perineum
 - ventral aspect of the prepuce is poorly developed ('hooded prepuce') and
 - ventral deformity of the erect penis (chordae)

Classification



- ☐ Distal-50%
 - Glanular
 - Coronal
 - Sub coronal
- ☐ Middle-30%
 - Penile shaft
- ☐ Proximal-20%
 - Penoscrotal
 - Scrotal
 - Perineal

Features

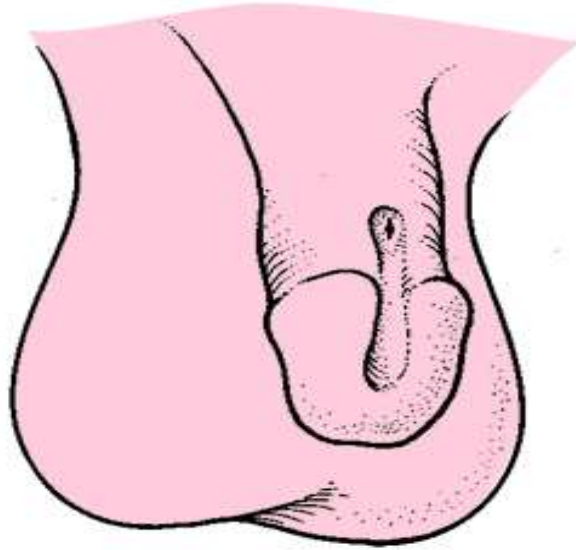
- ✓ Absence of urethra and corpus spongiosum distal to abnormal urethral orifice
- ✓ Bowing or bending of penis distal to abnormal urethral opening (chordee), with poorly developed prepuce over inferior aspect
- ✓ Urine soakage over the scrotum with dermatitis and infection
- ✓ Associated congenital anomalies

Treatment

- ✓ Surgery is the mode of treatment
- ✓ Timing
 - Early surgical correction (6-18months of age) to all boys with hypospadias regardless of its severity
- ✓ General principle of the surgery
 - Penile straightening
 - Placing the urethral meatus at tip of the glans
 - Constructing a neourethra
 - Creating a symmetrical, conical glans
 - Achieving a cosmetic skin coverage of the penis

Epispadias

- ✓ Urethral opening on the dorsum of the penis and is associated with an upward curvature of the penis. Epispadias are very rare but often coexists with bladder exstrophy and other severe developmental defects. Most common site is at the abdomino-penile junction.



Treatment

- Correction of incontinence of urethra
- Urinary diversion

Bladder exstrophy

- ✓ Incomplete development of the infraumbilical part of the anterior abdominal wall and anterior wall of the bladder. Associated with spina bifida and other anomalies. 1:50 000 births (m:f-4:1)



Features

- ✓ Penis is broad and short ± bilateral inguinal hernia
- ✓ Red mucus membrane of posterior bladder wall protrudes out
- ✓ With visible urine efflux from ureteric orifice
- ✓ Umbilicus absent with separation of the pubic bones
- ✓ females, defective external genitalia

Problems

- ✓ Repeated soakage
- ✓ Ulceration
- ✓ Pain
- ✓ Recurrent pyelonephritis
- ✓ Renal failure (50% of pt die)

Long-term complications

- ✓ stricture at the site of anastomosis
- ✓ hyperchloremic acidosis
- ✓ adenoma and adenocarcinoma at the site of a ureterocolic anastomosis(20-fold)

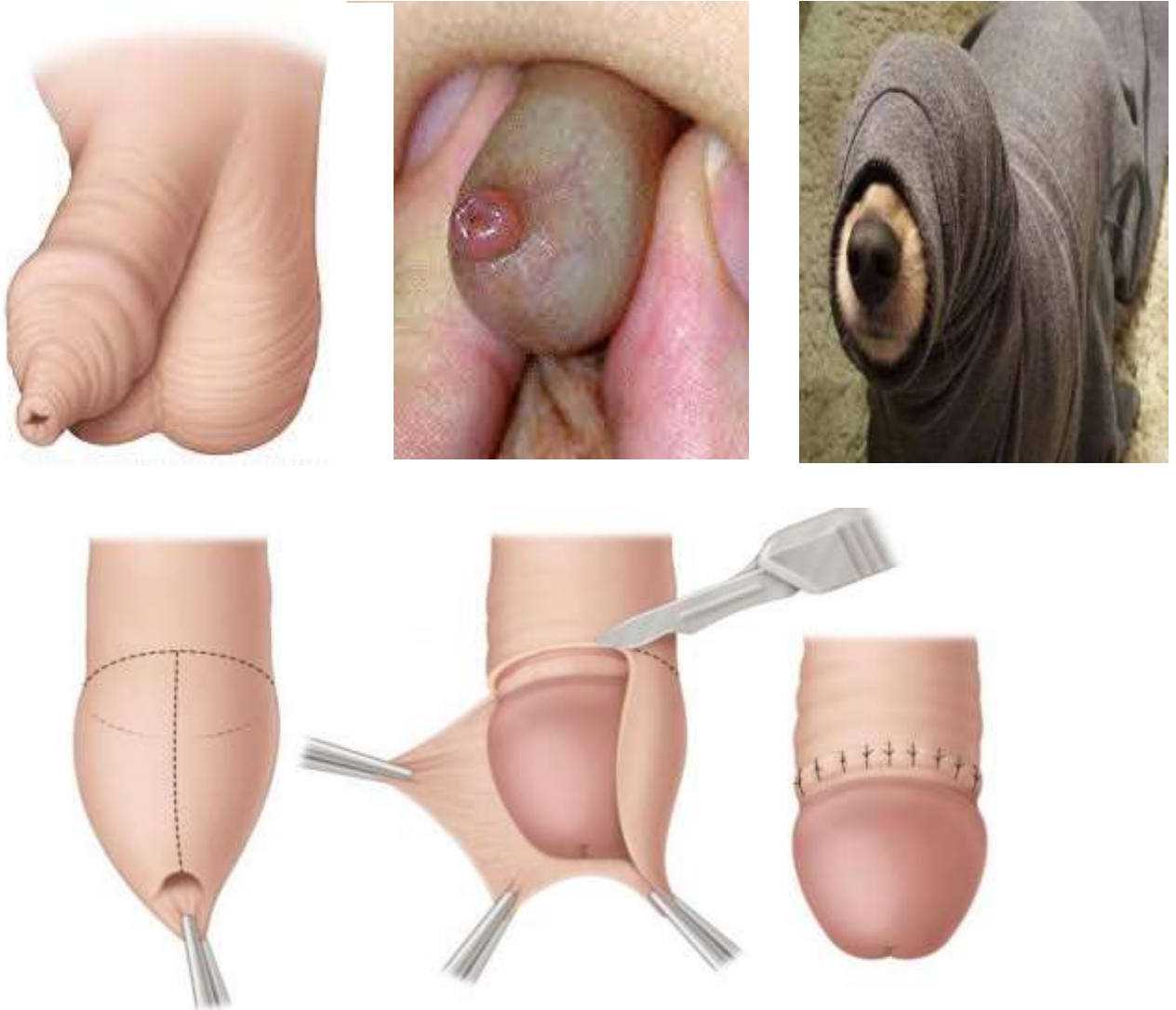
Treatment

- ✓ Bladder is closed in the 1st yr of life, osteotomy of both iliac bones, reconstruction of the bladder neck and sphincters.
- ✓ urinary diversion

Phimosis & paraphimosis

Phimosis

- ❑ Phimosis is an abnormal constriction of the opening in the foreskin that precludes retraction over the glans penis.
- ❑ Severe cases of phimosis can result in erectile dysfunction or impede the urinary stream



Read on paraphimosis

Chapter 19
MUSCULO-SKELETAL
RELATED

AMPUTATION

Indications

- ✓ PVD/peripheral vascular disease/--DM accounts around 50%
- ✓ Trauma
 - High grade open fractures with associated nerve injury, soft tissue loss & ischemia + un-reconstructable neurovascular injury
- ✓ Tumor
 - Malignant bone & soft tissue tumors
- ✓ Infections
 - fulminating gas gangrene
 - acute/ chronic bone infection that is unresponsive to antibiotic or surgical debridement
- ✓ Congenital anomalies/ deformities
- ✓ Burns & frostbite

Types of limb amputations

1. Leg amputations→ can be divided into 2 broad categories:

Major amputations:

- Below-knee amputation/BKA
- Knee disarticulation
- Above-knee amputation (transfemoral)
- Rotationplasty
 - ✓ Foot being turned around and reattached to allow the ankle joint to be used as a knee
- Hip disarticulation
- Hemi-pelvectomy

Minor amputations:

- Amputation of digits
- Partial foot amputation
- Ankle disarticulation

2. Arm amputation

- Amputation of digits
- Metacarpal amputation
- Wrist disarticulation
- Forearm amputation (transradial)
- Elbow disarticulation
- Above-elbow amputation (transhumeral)
- Shoulder disarticulation and forequarter amputation
- Krukenberg procedure

General principle of amputation

- Appropriate management of Skin, Bone, Nerves & Vessels
- Procedure
 - Remove the diseased tissue and any crushed bone
 - Smooth uneven areas of bone
 - Seal off blood vessels and nerves
 - Cut and shape muscles so that the stump, or end of the limb, will be able to have an artificial limb (prosthesis) attached to it

Complications of Amputation

- Phantom pain
- Hematoma
- Infection
- Failure of wound to heal
- edema
- Contracture
- Neuroma
- Psychological distress
- Complications due to immobility & pressure...

Types of prosthesis

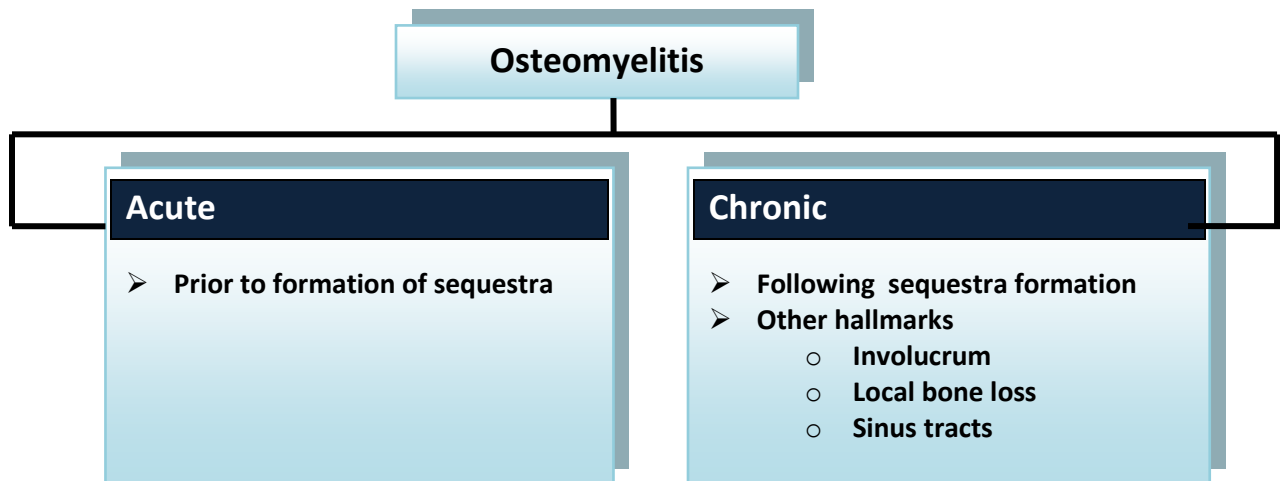
1. Endoprosthesis--implants used in orthopedics surgery
2. Exoprosthesis --external replacement for lost limb. May be temporary or permanent.

MANAGEMENT OF FRACTURE

Read under long cases of Debol (fracture on the management part)

BONE INFECTION & TUMOR

OSTEOMYELITIS



NB

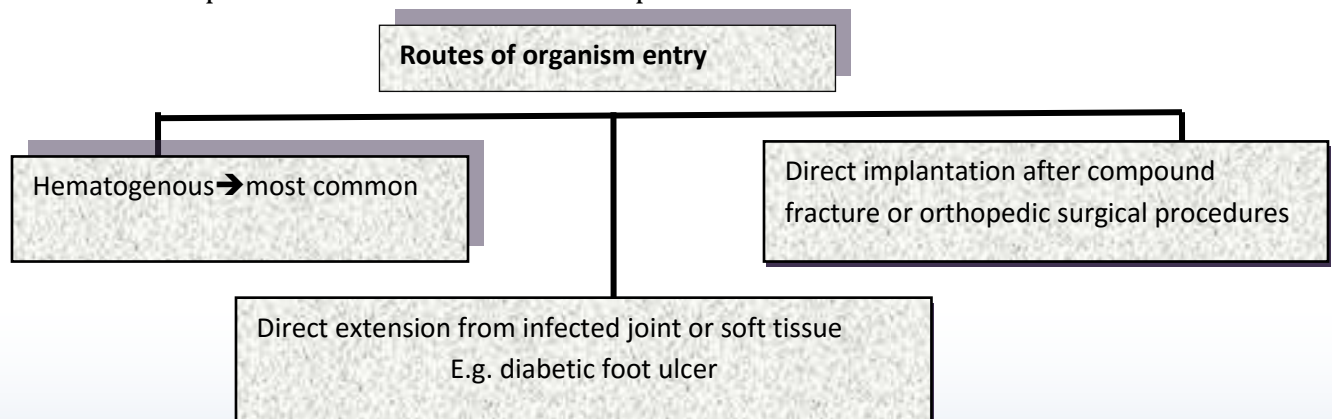
Sequestrum=necrotic bone

Involucrum=new bone formation around the dead bone

- Although the time limits delineating one category from the other are somewhat arbitrary, as a rule
 - ✓ infections up to 3 months→acute
 - ✓ Longer than 3 months→chronic
 - ✓ Some include a third category, "subacute," for patients who exhibit symptoms that endure longer than 3 months but who have not developed extensive necrotic bone

Etiology

- Pyogenic osteomyelitis (S.aureus)--common
 - Common Sites
 - ✓ In children→long bones--metaphysis
 - ✓ In adults→Vertebrae
- Tuberculous osteomyelitis
 - The spine is the most common site→pott's disease



Risk factors

- ✓ Immunodeficiency disorders
- ✓ Sepsis
- ✓ Minor trauma coincident with bacteremia
- ✓ Indwelling vascular catheters
- ✓ Chronic hemodialysis vascular access
- ✓ Sickle cell disease

Clinical presentation

- Classic signs of inflammation
 - Local bone pain, swelling, Redness...
- Fever
- Draining sinus

Investigation

- CBC--Leucocytosis may be observed in acute osteomyelitis but is unlikely in chronic osteomyelitis
- Increased ESR & CRP
- Blood culture--may be positive in about half of cases of acute osteomyelitis
- Discharge analysis--Culture and Gram staining
- Radiograph--may be normal up to 10days
 - Within 72 hr →deep-soft tissue edema (swelling) with displacement of the deep muscle planes
 - In 5-7 days→Periosteal reaction
 - After 7-14 days→Lytic bone changes, which are not visible until 30-50% of the bony matrix is destroyed
 - Infection in flat and irregular bones can take longer to appear
- Radionuclide scanning
- MRI—very sensitive for the detection of acute osteomyelitis and demonstrates abnormal marrow edema as early as three to five days following onset of infection

Complications of chronic osteomyelitis

- Pathologic fracture
- Sepsis, endocarditis
- Squamous cell carcinoma (Marjolin's ulcer) of sinus tract
- Osteosarcoma
- Growth plate involvement in children--impaired bone growth
- Septic arthritis

Management

Acute recent infection

1. Establish free drainage
2. Antibiotics--choice depends on
 - Age
 - Underlying medical condition
 - Suspected pathogens and their susceptibility pattern
 - Antibiotic safety and efficacy and availability
 - DOC
 - Cloxacillin /Nafcillin /clindamycin plus
 - Ceftriaxone /cefotaxime /cefuroxime or chloramphenicol
 - Duration # 3-6 weeks
 - IV to P.O.--When patients' condition has stabilized, usually 5 -10day of IV antibiotics
 - Afebrile for the past 48- 72 hours
 - Local signs and symptoms of infection are reduced considerably
 - Peripheral leukocyte count has normalized
 - ESR has decreased by at least 20 percent or there has been a 50 percent decrease in the concentration of CRP

➔ Indicators of quiescence are...

 - Pus discharge ceases
 - Wound become lined by healthy granulations
3. Indication & Principles of surgical Rx
 - Indications
 - Unresponsive to specific antibiotic Rx (Prolonged fever, Persistent pain and swelling)
 - Evidence of persistent soft tissue abscess/ subperiosteal collection
 - If concomitant joint infection is suspected
 - Chronic osteomyelitis
 - Principle of surgical mgt
 - Adequate drainage
 - Extensive debridement of necrotic tissue
 - Mgt of dead space
 - Adequate soft tissue coverage
 - Restoration of blood supply

Bone Tumors

Risk factors

- Paget's disease
- Radiation
- Fibrous dysplasia
- Hereditary...

Classification of bone tumors

Primary

Osteogenic tumors

1. Benign
 - ✓ Osteoid osteoma
 - ✓ Osteoblastoma
2. Malignant
 - ✓ Osteosarcoma

Chondrogenic tumors

1. Benign
 - ✓ Osteochondroma
2. Malignant
 - ✓ chondrosarcoma

Marrow derived

Malignant

- Myeloma
- lymphoma

Others

- Ewing's sarcoma

Secondary/metastasis

→ common

1. Breast
2. Prostate
3. Lung
4. Renal
5. Thyroid

Primary

Bone forming /osteogenic tumors

Benign

Osteoid osteoma	Osteoblastoma
➤ < 2cm in diameter	➤ Larger
➤ < 25yrs of age	➤ Relatively older
➤ Affect appendicular skeleton esp. femur & tibia	➤ Affect mostly the spine
➤ Very painful (nocturnal) which responds to aspirin	➤ Dull achy pain which does not respond to aspirin
➤ Elicits tremendous amount of reactive bone	➤ Less reactive bone

Malignant/ Osteosarcoma

- Most common primary bone malignancy
- Most common in distal femur, followed by proximal tibia
- Aggressive lesions that metastasize through the blood stream early in their course
 - The lungs are common sites of metastases

Cartilage tumors

- Osteochondroma
 - Benign cartilaginous capped bony projection → growing away from the joint towards the diaphyseal region
 - Can be pedunculated or sessile
- chondrosarcoma
 - 2nd most common malignant bone tumor
 - They arise in central portions of the skeleton
 - Patients present with painful progressively enlarging masses
 - Metastases show predilection for lungs and skeleton

Others

- Ewing's sarcoma
 - Tends to arise in the diaphyses of long bones/pelvis
 - Clinical presentation
 - painful mass + general symptoms with fever, anemia
 - Increased ESR
 - X-ray
 - 'moth-eaten' bone + 'onion skin' periosteal rxn

Metastatic (2^o) tumor

- Can be
 - Lytic → Kidney, Lung...
 - Sclerotic → Prostatic ca.
 - Mixed...

Common sites of metastasis

- Spine
- Proximal femur
- Proximal humerus

FOOT DEFORMITY

Can be congenital or acquired

- Examples of acquired causes
 - bunion/Hallux valgus
 - Hallux virus
 - hammer toe
 - foot drop
 - flat feet
- Examples of congenital causes
 - pigeon toe
 - club foot
 - pes caves
 - rocker bottom foot....etc

Club foot (congenital talipes equinovarus)

- Relatively common birth defect, occurring in about one in every 1,000 live births
- Approximately 1/2 of people with clubfoot have it affect both feet, which is called bilateral club foot. In most cases it is an isolated disorder of the limbs. It occurs in males twice as frequently as in females.
- The affected foot appears to have been rotated internally at the ankle
- Without treatment, people with club feet often appear to walk on their ankles or on the sides of their feet. However, with treatment, the vast majority of patients recover completely during early childhood and are able to walk and participate in athletics just as well as patients born without the deformity.

Diagnosis

- Clinical
- Foot x-ray→Dr's interest
- In-utero ultrasound

Treatment

- French method
- ponseti method
- surgery

Chapter 20

HEENT+LYMPHOGLANDULAR
RELATED

SALIVARY GLAND TUMORS ,INFECTIONS & CALCULI

SALIVARY GLANDS

Major

1. Parotid gland
2. Submandibular gland
3. Sublingual gland

Minor

Dispersed throughout the aerodigestive tract

Salivary gland tumors (SGTs)

Clinical presentation of benign SGTs

- Painless, slow growing mass
- Well circumscribed
- Almost always freely mobile
 - Face→parotid gland
 - unaffected facial nerve function in benign SGTs
 - Angle of jaw→DDx
 - Submandibular gland
 - Tail of parotid gland
 - Floor of mouth→Sublingual gland
- Parotid→80% of the SGTs
 - 80% benign
 - pleomorphic adenomas→most common, around 60%
 - warthin tumor→5-15%

Clinical presentation of malignant SGTs

- Rapid growth
- pain
- Bleeding
- Airway compromise
- Nerve dysfunction
 - E.g. paresthesia, facial palsy...
- Skin fixation

Read on how to examine mass under short case of Debol (swelling examination)

Salivary gland infection & calculi

80% in Submandibular gland

Submandibular calculi (Sialolithiasis)

List the factors which encourage stasis in the submandibular duct?

1. Anatomy of the duct → gravity
2. Quality of mucus

Clinical presentation

- The main symptoms are;
 1. swelling
 - ✓ start to worsen just before eating
 2. Pain
 - ✓ dull aching
 - ✓ worsen during eating
 - ✓ goes away before the swelling

***purulent saliva may be present

Submandibular sialadinitis

- It is secondary to the presence of a stone in its duct or the damage done by a stone which has passed through the duct.
- Staphylococcus aureus → commonest etiology

Clinical presentation

- Pain, which is;
 - severe, throbbing, continuous
 - Exacerbated by eating

CLEFT LIP & PALATE

Clefting of the lip and or palate occur around the 8th week of embryogenesis, either by;

- Failure of fusion of the medial nasal process and the maxillary prominence or
- Failure of mesodermal migration and penetration between the epithelial bilayer of the face

Etiology--Multifactorial

- Familial—more common in cleft lip or combined cleft lip and palate (Risk increased by 4%)
- increased parental age, drug use (steroids /diazepam) and infections during pregnancy
- Protein and vitamin deficiency
- Rubella infection
- Radiation
- Chromosomal abnormalities
- smoking during pregnancy

Classification

I. Cleft lip alone

- ✓ Central –Rare
 - In upper lip. Between two median nasal processes. (Hare lip)
- ✓ Lateral--commonest
 - maxillary and median nasal process
 - can be unilateral or bilateral
- ✓ Incomplete → cleft lip does not extend into nose
- ✓ Complete cleft lip → extends into nasal floor
- ✓ Simple cleft lip → only cleft in the lip
- ✓ Compound cleft lip → cleft lip with cleft of alveolus

II. Cleft of primary palate (in front of incisive foramen) only

- a. Complete—absence of pre-maxilla (anterior hard palate)
- b. Incomplete—rudimentary pre-maxilla
 - i. Unilateral
 - ii. Bilateral
 - iii. Median

III. Cleft of secondary palate (behind the incisive foramen) only

- a) Complete—nasal septum and vomer are separated from palatine process
- b) Incomplete
- c) Sub-mucous
 - Cleft with soft palate involvement
 - Cleft without soft palate involvement

IV. Cleft of both primary and secondary palates

V. Cleft lip and cleft palate together

- Often associated with other congenital anomalies of cardiac, GI, nervous system
 - E.g. Pierre-Robin syndrome, Klippel-Feil syndrome, Stickler's syndrome, Shprintzen's syndrome, Down's syndrome, Treacher-Collin's syndrome, Apert's syndrome and trisomy.

Problems in Cleft Disorders

- Difficulty in sucking and swallowing. This is commonly observed in cleft palate than in cleft lip.
- Speech is defective especially in cleft palate. Mainly to phonate B, D, K, P, T and G.
- Altered dentition or supernumerary teeth
- Recurrent upper respiratory tract infection
- Respiratory obstruction (in Pierre Robin syndrome)
- Chronic otitis media, middle ear problems
- Cosmetic problems
- Hypoplasia of the maxilla
- Problems due to other associated disorders

Treatment for Cleft Lip

- Principles of cleft lip repair
 - ❖ “Rule of 10’ should be fulfilled
 - 10 pound in weight
 - 10 weeks old
 - 10 gm % hemoglobin
 - ❖ Before 6 months--should be operated
 - ❖ Proper postop mgt like control of infection, training for sucking, swallowing and speech
 - ❖ Millard advancement flap is commonly used for unilateral cleft lip repair
 - ❖ Bilateral cleft lip repair can be done either in single or two stages (with 6 months gap between each stage)
 - ❖ Management of associated primary or secondary cleft palate deformity

Cleft Palate

- It is due to failure of fusion of the two palatine processes
- Defect in fusion of lines between premaxilla (developed from median nasal process) and palatine processes of maxilla one on each side
- When premaxilla and both palatine processes do not fuse, it leads into complete cleft palate (Type I cleft palate)
- Incomplete fusion of these three components can cause incomplete cleft palate beginning from uvula towards posteriorly at various lengths. So it could be Type II a–bifid uvula, Type II b–bifid soft palate (entire length) or Type II c –bifid soft palate and posterior part of hard palate (but anterior part of hard palate is normal).

Feature

- Small maxilla with crowded teeth, absent/poorly developed upper lateral incisors
- Bacterial contamination of upper respiratory tract with recurrent infection is common
- Chronic otitis media with deafness may occur
- Swallowing difficulties to certain extent and speech problems can occur
- Cosmetic problems
-

Treatment for Cleft Palate

- Cleft palate is usually repaired in 12-18 months. Early repair causes retarded maxillary growth (probably due to trauma to growth center and periosteum of the maxilla). Late repair causes speech defect.
- Both soft and hard palates are repaired--called palatoplasty
- Regular examination of ear, nose and throat during follow up period
- Post-op speech therapy
- Secondary management:
 - Hearing support--using hearing aids if defect is present, control of otitis media
 - Speech problems – speech therapy
 - Dental problems corrected by proper dentist opinion, and reconstructive surgery
 - Orthodontic management

Chapter 21

MISCELLANEOUS

Penetrating neck injury (PNI)

- ⇒ that penetrate the platysma (platysma violation)
- ⇒ Every penetrating neck injury (PNI) must be inspected (but not probed) to determine if the platysma muscle has been violated. If not, the wound is considered superficial and not a PNI
- ⇒ A more subtle injury that may not be identified is a fracture of the larynx due to blunt trauma
- ⇒ Signs and symptoms include hoarseness, subcutaneous emphysema or a palpable fracture
- ⇒ The management is based on the patient hemodynamic stability and zone of the neck but currently In part due to advances in diagnostic imaging; a "no-zone" management strategy has been developed. Using this approach, patients are categorized as unstable or stable, rather than according to the zone of the neck injury, with unstable patients transferred rapidly to the operating room.
- ⇒ Mortality rates for PNIs appear to be highest with Zone I injuries (below the cricoid cartilage), due to ...
 - The proximity of mediastinal structures
 - the severity of the vascular injuries sustained
 - Surgical challenges.
- ⇒ Exsanguination (excessive blood loss that causes death) is the most common cause of immediate death and the carotid artery is the structure most often involved. The incidence of carotid artery injury from a PNI ranges from 6 to 17 percent. Concomitant injury beyond the neck is common
- ⇒ The specific anatomic borders, which define the zones of penetrating neck injury, have varied using either the cricoid cartilage or level of clavicles to differentiate Zone I from Zone II injury, which is thought to represent a minor modification of no real clinical significance
 - ✓ Zone I
 - including the thoracic inlet, up to the level of the cricothyroid membrane
 - is treated as an upper thoracic injury.
 - require a median sternotomy with extension to an anterior sternocleidomastoid incision or supraclavicular incision with or without clavicular head resection
 - Mortality rates for PNIs appear to be highest with Zone I injuries (below the cricoid cartilage), due to ...
 - The proximity of mediastinal structures
 - the severity of the vascular injuries sustained
 - Surgical challenges
 - ✓ Zone II
 - between Zones I and III
 - is the area of controversy
 - a transverse

- cervical collar incision may provide access to both sides of the neck, with the potential to extend along the anterior sternocleidomastoid muscle
- ✓ Zone III
 - above the angle of the mandible
 - Is treated as a head injury.
 - A difficult anatomic zone of injury for distal vascular control. At times, subluxation, dislocation, or resection of the mandible may be necessary to gain operative vascular control.

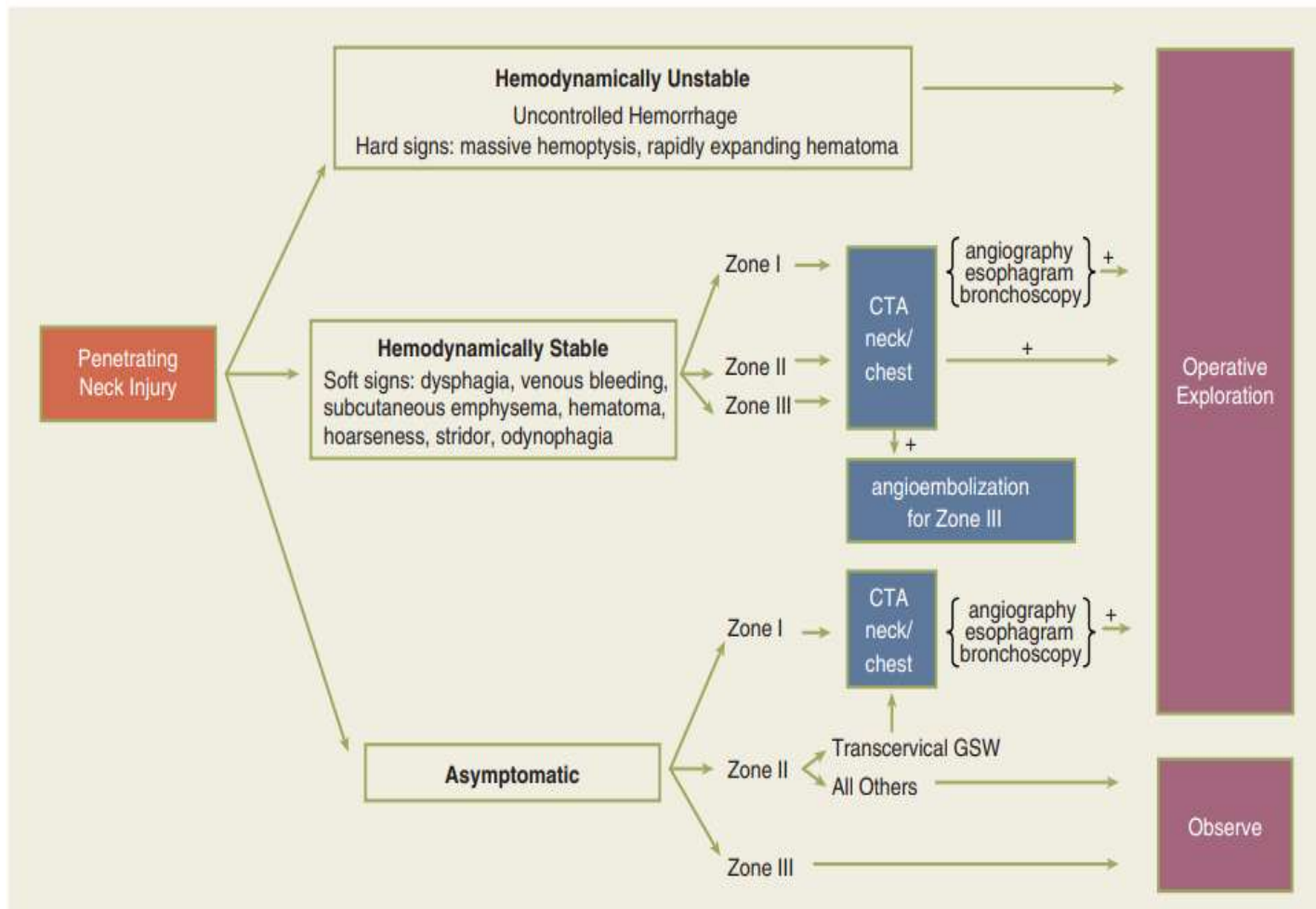
What are the hard and soft signs of PNI?

Hard signs	Soft signs
• Hard signs of vascular injury ✚ Severe or uncontrolled hemorrhage ✚ Rapidly expanding or pulsatile hematoma ✚ Thrills and bruits ✚ Hypotension unresponsive to aggressive fluid resuscitation ✚ Absent or diminished radial pulse ✚ Neurologic deficits consistent with cerebral ischemia (stroke). • Hard signs of aerodigestive injury ✚ air bubbling from a wound ✚ massive hemoptysis ✚ respiratory distress While there is disagreement about whether a large amount of subcutaneous air should be considered a hard or soft sign of aerodigestive injury, there is no doubt that it requires urgent evaluation	• proximity wounds (injury within 1 to 2 cm of a major vessel in the presence of other soft signs of vascular injury), • minor hemorrhage • mild hypotension that responds to fluid resuscitation • minor hemoptysis or hematemesis, • subcutaneous or mediastinal air • non-expanding hematoma, dysphonia, and dysphagia.

✓ Apply ATLS

- most experts advocate immediate intubation in patients with evidence of significant injury or respiratory difficulty (eg, subcutaneous air, airway compromise, significant bleeding or hematoma, voice alteration, stridor)
- In almost all cases, the wound should not be explored in the ED. For life-threatening bleeding from a neck wound that does not stop with direct pressure, the clinician can insert a Foley catheter into the wound and fill the balloon with 10 to 15 mL of saline or until resistance is felt. This may temporarily tamponade bleeding until definitive treatment is available. If bleeding is severe, neither direct pressure nor a Foley catheter effectively controls the hemorrhage, and no surgeon is available, the emergency clinician may have no choice but to attempt to explore the wound and control bleeding by other means (eg, placing a clamp)
- Place all PNI patients on continuous cardiac and pulse oximetry monitoring, with supplemental oxygen as needed to maintain the oxygen saturation above 92 percent. Two large bore (eg, 14 or 16 gauge) IV lines should be placed contralateral to the injury if possible, as ipsilateral venous injuries may exist.
- Airway management
 - ✚ Signs that strongly suggest the need to secure the airway immediately in patients with significant facial or neck trauma include
 - Significant bleeding or neck hematoma
 - Hemoptysis
 - Subcutaneous neck emphysema
 - Bruit or thrill
 - Distorted neck anatomy
 - Stridor
 - Difficulty or pain when swallowing secretions
 - Abnormal voice, especially hoarseness ("hot potato voice")
 - ✚ So what are the options of air way management in PNT....basically blinded intubation is contraindicated! the following are the best options ...
 - 'Awake look' intubation
 - Intubation with direct visualization of structures by direct laryngoscope, flexible endoscope, or rigid fiberoptic or video laryngoscope while the patient is breathing spontaneously with slight sedation or local anesthesia
 - Tracheostomy
 - If the above (awake intubation) unavailable and the patient is with significant head and neck trauma mean while can't rule out the laryngeal or tracheal injury.

Then classify ...



Algorithm for the management of penetrating neck injuries. CTA = computed tomographic angiography; GSW = gunshot wound.

EXAMINATION OF MSS, SWELLING & ULCER

Examination of musculoskeletal system (MSS)

General approach

- ❖ Inspection [look]
- ❖ Palpation [feel]
- ❖ Range of Motion (ROM) [move]
- ❖ Measurement [measure]

i) Look

- Expose both sides , adequate light

Specific to the site of interest

- Look for asymmetry
- Deformity
- Visible fractured bone
- Swelling
- Laceration
- Bruising /discoloration
- Abnormal movement
- Atrophy...

ii) Feel

- Temperature
 - Compare both sides of extremities with back of your hand
- Tenderness
- Check pulses
 - ✓ If lower extremity
 - ⊕ Dorsalis pedis artery
 - ⊕ Posterior tibialis artery
 - ⊕ Popliteal artery...
 - ✓ If upper extremity
 - ⊕ Radial artery
 - ⊕ Brachial artery
- Compare muscle bulk--By measuring with tape meter
- Asses capillary refill--If >2sec→abnormal
- Swelling →characterize...

iii) Move

Range of motion (Active)

- Ask the patient to move the affected site
- Watch for decreased or increased movement of the joint compared to the other side as well as the normal
- Watch for pain with movement

- Listen for crepitus or “popping”
- Watch for abnormal movements

ROM (passive)

- Move the joints passively, comparing the end points to the active
- Again note any decreased or increased movement
- Pain with the movement
- Crepitus or “popping”

iv) Measure

-  Apparent length
 - From xiphisternum or umbilicus to medial malleolus
-  Real length
 - From greater trochanter of the femur up to the medial malleolus
-  True length
 - Between two bony prominences

Examination of swelling/mass

Inspection

- Shape
- location
- Overlying skin color change
- Any ulceration or discharge

Palpation

- Temperature → compare
- Tenderness
- Size--Measure
- Number
- Surface (Smooth Vs Nodular)
- Border (Regular Vs irregular)
- Fixity--To the overlying or underlying tissue
- Consistency (Soft Vs firm Vs hard)

Specific

- Fluctuant--Indicate abscess collection
- Transillumination---E.g scrotal swelling (hydrocele)
- Reducibility—E.g. hernia , hemorrhoidal swelling
- Compressibility--Characteristic of vascular hemangioma
- Pulsatility

Click below & watch video on how to examine swelling

<https://www.youtube.com/watch?v=GUX6UpRiU9c>

NB

When you are asked to list DDx for a swelling...Think of the structures that are found underlying/overlying the swelling like fibrous connective tissue, adipose tissue, vessels, skeletal muscles, nerves & bone. Then list the benign & malignant causes of swelling of each structure. E.g. fibroma, fibrosarcoma (for fibrous Connective tissue)

Examination of ulcer

Inspection

- Location of ulcer. E.g.
 - ✓ Varicose ulcer--lower leg
 - ✓ Rodent ulcer— face, nose
 - ✓ Tuberculosis ulcer--over the neck
- Size
- Number --Single or multiple
 - ✓ Is there any similar ulcer in other part of body?
- Margin--Type
 1. Healing ulcer--Have 3 lines
 - White (outer), blue (center) & red (inner)
 2. Inflamed margin
 - Red, irregular with inflamed surrounding skin
 3. Fibrosed margin
 - Thicken white skin margin without the blue line of growing epithelium
- Floor of the ulcer (surface of ulcer)
 - ✓ Granulation, sloughing , discharge...
- Surrounding skin
 - ✓ Redness
 - ✓ Pigmentation
 - Dark pigmentation of skin →typical for varicose ulcer
 - Hypopigmentation of surrounding skin→in non-healing ulcer

Palpation

- ✓ Surrounding skin for hotness and tenderness
- ✓ Ulcer--Edge →confirm size
- ✓ Floor--see if it bleeds on touch
- ✓ check the involvement of underlying structures

Related examination

- ✓ Related lymph nodes
- ✓ Related arteries, veins and nerves
- ✓ Movement in neighboring joints
 - Restriction to movement indicates muscle involvement or painful inflammation...

Click below & Watch video on how to examine ulcer @

<https://www.youtube.com/watch?v=S1FpVuyUJ6Q>

WOUND

Definition

A wound is a break in the integrity of the skin or tissues, associated with disruption of the structure and function.

CLASSIFICATION OF WOUNDS

I. Surgical Wound classification

- a) Clean wound--Infective rate is less than 2%
 - Herniorrhaphy, excisions
 - Surgeries of the brain, joints, heart, transplant...
- b) Clean contaminated wound--Infective rate is 10%
 - Appendicectomy, Bowel surgeries
 - Gallbladder, biliary and pancreatic surgeries
- c) Contaminated wound--Infective rate is 15-30%
 - Acute abdominal condition, Open fresh accidental wounds
- d) Dirty infected wound--Infective rate is 40-70%
 - Abscess drainage, Pyocele, Empyema gallbladder, Fecal peritonitis.

II. Rank and Wakefield Classification

A. Tidy wounds

- Incised (sharp objects), clean, healthy wound without any tissue loss. Heal by primary intention. Primary suturing is done.

B. Untidy wounds

- Crushed, devitalized, unclean. Wound dehiscence, infection, delayed healing is common. Liberal excision of devitalized tissue and healing by secondary intention is the management. Secondary suturing, skin graft or flap may be needed.
- E.g--crushing, tearing, avulsion, devitalized, vascular injury, multiple irregular wounds and Burns

III. Classification based on Type of Wound

- Clean incised wound → clean-cut wound with linear edge
- Lacerated wounds--ragged edges with devitalization
- Bruising and contusion--soft tissue injury with discoloration and haematoma formation without skin break
- Hematoma—subcutaneous, intramuscular, subfascial or intra-articular blood collection
 - Small hematoma get absorbed. Large hematoma once get infected should be drained under general/regional anesthesia adequately.
- Closed blunt injury
- Puncture wounds and bites
- Abrasion--superficial shearing of skin (only epidermal injury). Heals by epithelialization.
- Traction and avulsion injury
- Crush injury--caused by war wounds, road traffic accidents, tourniquet.

- Leads to Compartment syndrome, Muscle ischemia, Gangrene and loss of tissue.
- War wounds and gunshot injuries
- Injuries to bones and joints → may be open or closed
- Injuries to nerves → either clean cut or crush
- Injuries to arteries and veins (major vessels)
- Injury to internal organs → may be of penetrating or nonpenetrating (blunt) types
- Penetrating wounds
 - Stab, bullet

III. Classification based on Thickness of the Wound

- Superficial wound → only epidermis and dermal papillae
- Partial thickness → deep dermis. With only deepest part of the dermis, hair follicle shafts and sweat glands are left behind.
- Full thickness → entire skin and subcutaneous tissue causing spacing out of the skin edges.
- Deep wounds → across deep fascia into muscles or deeper structures.
- Complicated wounds → associated with injury to vessels or nerves
- Penetrating wound → penetrates into either natural cavities or organs.

IV. Classification based on Involvement of Structures

- Simple wounds → only one organ or tissue
- Combined wounds → mixed tissues

V. Classification based on the Time Elapsed

- Acute wound → up to 8 hours of trauma
- Chronic wound → after 8 hours of trauma

WOUND HEALING

Wound healing is complex method to achieve anatomical and functional integrity of disrupted tissue

Types of Wound Healing

- Primary Healing (First Intention) → Clean incised wound or surgical wound. Wound edges can be approximated by sutures. More epithelial regeneration than fibrosis.
 - Heals rapidly with complete closure. Scar will be linear, smooth, and supple.
- Secondary Healing (Second Intention) → Extensive soft tissue loss like in major trauma burns and wound with sepsis. Heals slowly with fibrosis. A wide scar often hypertrophied and contracted. May lead to disability.
- Healing by Third Intention (Tertiary Wound Healing or Delayed Primary Closure) → after wound debridement and control of local infection, wound is closed with sutures or covered using skin graft. Primary contaminated or mixed tissue wounds heal by tertiary intention.

Phases of Wound Healing

Inflammatory Phase (Lag or Substrate or Exudative Phase)

- It begins immediately after wound healing. It lasts for 4-6 days.
- Features of inflammation are rubor, calor, tumour, dolor and loss of function.
- Macrophages secrete fibroblastic growth factor which enhances angiogenesis.
- PMN leukocytes appear after 48 hours which secrete inflammatory mediators and bactericidal oxygen derived free radicals.
- These cells also remove clots, foreign bodies and bacteria.
- Chemical factors involved in wound healing are: Growth factor—platelet derived, epidermal, transforming, Interleukin, Tumour necrosis factor, Prostaglandins, Collagenase, Elastase.

These actions are reduced in diabetes mellitus, Cushing's syndrome and immunosuppression increasing the rate of sepsis.

Proliferative Phase (Collagen/Fibroblastic Phase)

- Collagen and glycosamines are produced by fibroblasts.
- It begins in 7 days and lasts for 6 weeks.
- Hydroxyproline and hydroxylysine are synthesized by specific enzymes using iron, alpha ketoglutarate and vitamin C.
- Tropocollagen is produced which aggregates to form collagen fibrils.
- 80-90% of their final strength (in postoperative wounds) is achieved in 30 days.

Remodeling Phase (Maturation Phase)

- It begins at 6 weeks and lasts for 2 years.
- There is maturation of collagen by cross-linking which is responsible for tensile strength of the scar.
- Collagen production is not present after 42 days of wound healing.

Scar strength is 3% in 1 week; 20% in 3 weeks; 80% in 12 weeks. But it will never get its original strength.

Management of Wounds

- ABC of life
 - Incised wound----primary suturing after thorough cleaning.
 - lacerated wound ----wound is excised and primary suturing
 - Crushed or devitalized wound ---- there will be edema and tension in the wound. So, after wound debridement or wound excision edema is allowed to subside for 2-6 days. Then delayed primary suturing is done.
 - Deep devitalized wound ---- after wound debridement, it is allowed to granulate completely. Later if the wound is small, secondary suturing is done. If the wound is large, a split skin graft is used to cover the defect.
 - Wound with tension ---- fasciotomy is done so as to prevent the development of compartment syndrome.
 - Vascular or nerve injuries are dealt accordingly
 - Internal injuries has to be dealt accordingly. (intracranial by craniotomy, intrathoracic by intercostal tube drainage, intra-abdominal by laparotomy).
 - Antibiotics, fluid and electrolyte balance, blood transfusion, tetanus toxoid (0.5 ml intramuscular to deltoid muscle), or antitetanus globulin (ATG) injection.
-
- Wound debridement (wound toilet, or wound excision) →is liberal excision of all devitalized tissue at regular intervals (of 48-72 hours) until healthy, bleeding, vascular tidy wound is created.
 - Primary suturing →means suturing the wound immediately within 6 hours. It is done in clean incised wounds.
 - Delayed primary suturing →means suturing the wound in 48 hours to 10 days. It is done in lacerated wounds. This time is allowed for the edema to subside
 - Secondary suturing→means suturing the wound in 10-14 days or later. It is done in infected wounds. After the control of infection, once healthy granulation tissue appears, secondary suturing is done.

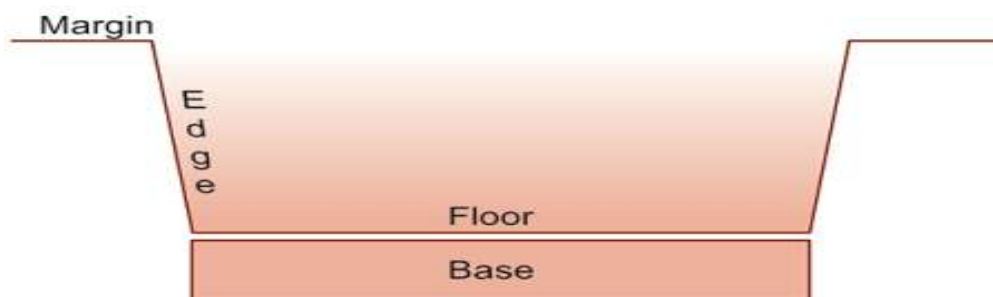
ULCER

Definition

An ulcer is a break in the continuity of the covering epithelium, either skin or mucous membrane.

Parts of an Ulcer

- Margin: It may be regular or irregular. It may be rounded or oval.
- Edge: Edge is the one which connects floor of the ulcer to the margin.
 - ❖ Sloping edge--It is seen in a healing ulcer. Its inner part is red because of red, healthy granulation tissue. Its outer part is white due to scar/fibrous tissue. Its middle part is blue due to epithelial proliferation.
 - ❖ Undermined edge-- is seen in a tuberculous ulcer. Disease process advances in deeper plane (in subcutaneous tissue), whereas (skin) epidermis proliferates inwards.
 - ❖ Punched out edge-- is seen in a gummatous (syphilitic) ulcer and trophic ulcer. It is due to endarteritis.
 - ❖ Raised and beaded edge (pearly white) -- is seen in a rodent ulcer (Basal Cell Carcinoma). Beads are due to proliferating active cells.
 - ❖ Everted edge (rolled out edge)-- It is seen in a carcinomatous ulcer(SCC) due to spill of the proliferating malignant tissues over the normal skin.
- Floor: It is the one which is seen. Floor may contain discharge, granulation tissue or slough
- Base: Base is the one on which ulcer rests. It may be bone or soft tissue.



Classification

Clinical

- a. Spreading ulcer:
 - ❖ Edge is inflamed and edematous
- b. Healing ulcer:
 - ❖ Edge is sloping with healthy pink/red granulation tissue with serous discharge.
- c. Callous ulcer:
 - ❖ Floor contains pale unhealthy granulation tissue with indurated edge/base. It lasts for many months to years. Ulcer doesn't show any tendency to heal. It is due to callous attitude of the patient.

Pathological

1. Specific ulcers:
 - ✓ Tuberculous ulcer
 - ✓ Syphilitic ulcer--It is punched out, deep, with "wash-leather" slough in the floor and with indurated base
 - ✓ Actinomycosis
2. Malignant ulcers:
 - ✓ Carcinomatous ulcer
 - ✓ Rodent ulcer
 - ✓ Melanotic ulcer

Nonspecific ulcers:

1. Traumatic
 - ✓ Physical→Electrical
 - ✓ Chemical→Caustics
 - ✓ Mechanical→Dental ulcer, pressure from splint, etc...
2. Arterial--Atherosclerosis, TAO, Raynaud's disease
3. Venous--Venous ulcer, Post-thrombotic ulcer, gravitational ulcer
4. Neuropathic (Neurotrophic or Perforating)
 - ✓ Diabetes, leprosy, tabes dorsalis, spina bifida, paraplegia, syringomyelia
5. Tropical ulcers--Tropical countries
6. Metabolic →Diabetes, Gout
7. Secondary to diseases like
 - Rheumatoid arthritis
 - Erythrocyanosis frigida
 - Osteitis Deformans (Paget's disease of the bone)
 - Avitaminosis
8. Ulcers complicating blood diseases
 - Sickle cell anemia
 - Mediterranean anemia
 - Felty's syndrome (look for spleen)
9. Ulcer occurring on paralysed leg
 - ✓ usually seen in anterior poliomyelitis
10. Factitious ulcer (Artefact ulcer)
11. Diphtheritic desert sore
12. Yaws

13. Decubitus ulcer
14. Iatrogenic ulcer → extravasation of IV fluid
15. Ulcers in congenital arteriovenous fistula
16. Miscellaneous → Martorell's ulcer

Classical sites for commonly seen ulcers

<i>Type of ulcer</i>	<i>Site</i>
Venous ulcer	Just above the medial malleolus (Gaiter area)
Arterial ulcer	Tips of toes and between the toes (where the pressure is lowest), over the malleoli and heel (pressure areas)
Neuropathic ulcer	Over the heads of the first and second metatarsal .
Traumatic ulcer (footballer's ulcer)	Shin (the tibia is subcutaneous and there is lack of underlying muscle with resultant reduced blood supply)
Rodent ulcer (BCC)	Above a line joining the angle of the mouth to the lobule of the ear
Tuberculous ulcer	Seen in neck, axilla and groin (where tuberculous lymph nodes are seen)
Gummatous ulcer	Over the subcutaneous bones such as sternum, skull, tibia, etc.
Trophic ulcers	On the heel and ball of the foot

Types of discharge & the probable cause

Discharge	Cause
Serous	In healing ulcer
Seroanguineous	Tuberculous ulcer
Greenish/ bluish	Ulcer infected with pseudomonas
Yellow discharge & creamy pus	Staphylococci
Watery & opalescent	Streptococcus
Yellow granules	Actinomycosis

GRANULATION TISSUE

It is proliferation of new capillaries and fibroblasts intermingled with red blood cells and white blood cells with thin fibrin cover over it. Granulation tissue signifies healing. It is usually pink with red dots. The red dots are seen at the sites of capillary loops.

Types

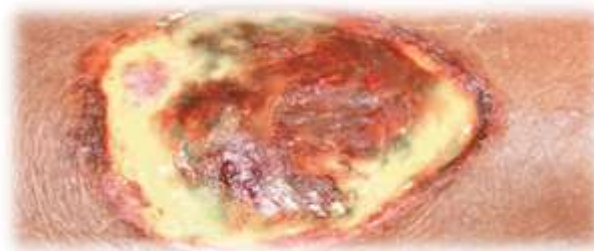
Healthy granulation tissue

- It occurs in a healing ulcer. It has got sloping edge. It bleeds on touch. It has got serous discharge. 5 Ps of granulation tissue → Pink, Punctate hemorrhages, Pulseful, Painless, Pin head granulation. Skin grafting takes up well with healthy granulation tissue.



Unhealthy granulation tissue

- Pale with purulent discharge. Its floor is covered with slough. Its edge is inflamed and edematous. It is a spreading ulcer



INVESTIGATIONS

- Discharge analysis → Gram stain, AFB, Culture and sensitivity, cytology
- Edge biopsy → Biopsy is taken from the edge because edge contains multiplying cells. Usually two biopsies are taken. Biopsy taken from the centre may be inadequate because of central necrosis
- X-ray of the part to look for periostitis/osteomyelitis
- FNAC of the lymph node
- Chest X-ray, Mantoux test in suspected case of tuberculous ulcer
- Blood sugar
- Doppler

Debridement of an ulcer

- It is removal of devitalized tissue. Small ulcers are debrided in ward. Large ulcers are debrided in operation theatre under general anaesthesia. All dead, devitalized, necrotic tissues are removed. Slough should be separated adequately before debridement. Often devitalized tissue separates on its own by autolysis if not enzymes like collagenase are used for debridement. Hydrotherapy and dressings are mechanical nonselective method of debridement.
- Ulcer cleaning
 - is done using dilute povidone iodine and normal saline (better and ideal). It should be done daily or two times a day depending on the severity.
- Dressing of an ulcer
 - is done to keep ulcer moist, keep surrounding skin dry, reduce pain, soothen the tissue, protect the wound and as an absorbent for the discharge

Discussion on some common ulcer types

TRAUMATIC ULCER

- ✓ Such ulcer occurs after trauma. It may be mechanical— dental ulcer along the margin of the tongue due to tooth injury; physical like by electrical burn; chemical like by alkali injury.
- ✓ Such ulcer is acute, superficial, painful and tender. Secondary infection or poor blood supply of the area make it chronic and deep.
- ✓ Footballer's ulcer is a traumatic ulcer occurring over the shin of males due to direct knocks on the shin. It is staphylococcal infection with a chronic and deep ulcer.
- ✓ Traumatic ulcers can occur anywhere in the body due to trauma.
- ✓ Trauma causes infection, necrosis, fascitis, crush injury, endarteritis of the skin leading into formation of large/deep nonhealing ulcer.
- ✓ Treatment depends on size and extent of ulcer. Regular dressing, later skin grafting is done.

TROPHIC ULCER (PRESSURE (BED) SORE/ DECUBITUS ULCER)

Pressure sore is tissue necrosis and ulceration due to prolonged pressure. Blood flow to the skin stops once external pressure becomes more than 30 mmHg (more than capillary occlusive pressure) and this causes tissue hypoxia, necrosis and ulceration. It is more prominent between bony prominence and an external surface.

Clinical presentation

- ◆ Occurs in 5% of all hospitalized patients
- ◆ Painless ulcer which is punched out
- ◆ Ulcer is non-mobile with base formed by bone

Management

- ◆ manage the underlying cause
- ◆ Nutritional supplementation
- ◆ Rest, antibiotics, slough excision, regular dressings
- ◆ Vacuum assisted closure (VAC): It is the creation of intermittent negative pressure of minus 125 mmHg to promote formation of healthy granulation tissue. Negative pressure reduces tissue edema, clears the interstitial fluid and improves the perfusion. Increases the cell proliferation and so promotes the healing. A perforated drain is kept over the foam dressing covered over the pressure sore. It is sealed with a transparent adhesive sheet. Drain is connected to required vacuum apparatus.
- ◆ Once ulcer granulates well, flap cover or skin grafting is done
- ◆ Excision of the ulcer and skin grafting
- ◆ Flaps—local rotation or other flaps (transposition flaps)
- ◆ Cultured muscle interposition

PREVENTION

◆ Proper care

- ✓ Change in position once in 2 hours
- ✓ Lifting the limb upwards for 10 seconds once in 10 minutes
- ✓ Nutrition
- ✓ Use of water bed/air bed/air-fluid floatation bed and pressure dispersion cushions to the affected area
- ✓ urinary and fecal care
- ✓ hygiene
- ✓ psychological counseling
- ✓ Regular skin observation
- ✓ keeping skin clean and dry (using regular use of talcum powder); oil massaging of the skin and soft tissues using clean, absorbent porous clothing; control and prevention of sepsis helps in the management.

DIABETIC ULCER

Cause

- ◆ Increased glucose in the tissue precipitates infection.

Sites

- ◆ Foot/plantar aspect—is the most common site
- ◆ Leg
- ◆ Upper limb, back, scrotum, perineum
- ◆ Diabetic ulcer may be associated with ischemia
- ◆ Ulcer is usually spreading and deep

Management

- ◆ Control the sugar level
- ◆ Antibiotics
- ◆ Nutritional supplements
- ◆ Regular cleaning, debridement, dressing
- ◆ Once granulates, the ulcer is covered with skin graft or flap
- ◆ Toe/foot/leg amputation
- ◆ Microcellular rubber (MCR) shoes to prevent injuries; care of foot

TUBERCULOUS ULCER

It is due to M.tuberculosis. It is usually due to cold abscess later forming ulcer in the neck, chest wall, axilla and groin.

- It can also be primary tuberculosis of the skin (commonly in face)
- Ulcer can be single or multiple; oval or rounded; with undermined edge (due to progression of disease outwards underneath and healing inwards by skin), painful and tender with caseating material on the floor.
- Ulcer is usually not deep. Regional lymph nodes may be enlarged matted, firm, and nontender

Management

- ✓ Anti TB

BURN

Outline

1. Classification of burn
2. Pathophysiology
3. Burn wound assesement
4. Burn patient care



1. Classification of burn

- ❑ Thermal
 - Flame—damage from superheated, oxidized air
 - Scald—damage from contact with hot liquids
 - Contact—damage from contact with hot or cold solid materials
- ❑ Chemical
 - Contact with noxious chemicals
- ❑ Electrical
 - Conduction of electrical current through tissues

2. Pathophysiology

Local changes

- Burn causes coagulative necrosis of the epidermis and underlying tissues
- Zones of cutaneous injury can be divided in to 3
 - ❖ Zone of coagulation
The necrotic area of a burn where cells have been irreversibly damaged at the time of injury
 - ❖ Zone of stasis
 - Surrounding the necrotic zone
 - Moderate degree of insult with decreased tissue perfusion/due to vascular damage
 - Either survive or progress to coagulative necrosis
 - ❖ Zone of hyperemia
Contains clearly viable tissue from which the healing process begins and is generally not at risk for further necrosis.

NB

Depth of burn depends on

- Temperature
- Duration of exposure

Systemic Changes

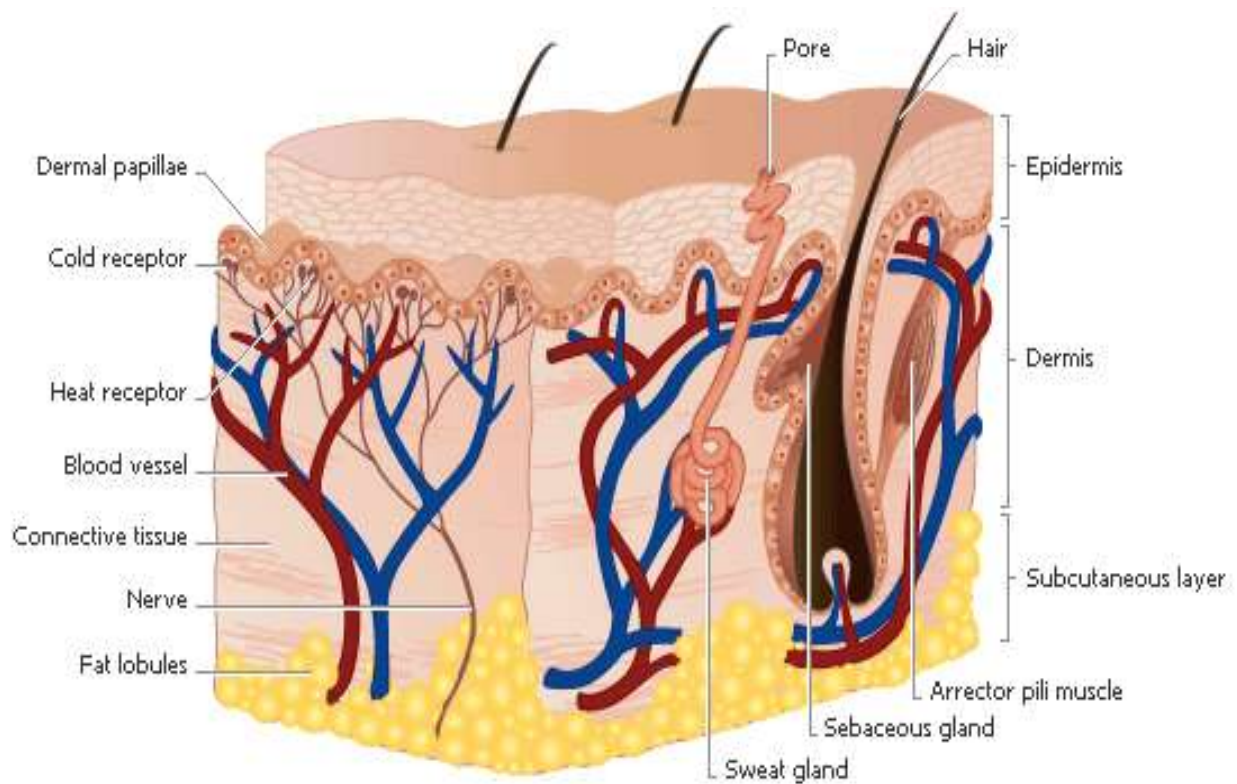
- Inflammation and Edema
 - Generalized edema occurs in response to changes in Starling forces in both burned and unburned skin
 - Effect on renal system
 - Diminished blood volume and cardiac output result + Stress-induced hormones and mediators → reduce renal blood flow → acute tubular necrosis and renal failure
 - Early resuscitation decreases renal failure and improves the associated mortality rate
- Effects on the Gastrointestinal System
 - The gastrointestinal response to a burn is highlighted by mucosal atrophy, changes in digestive absorption, and increased intestinal permeability
 - Effects on the Immune System
 - Burns cause a global depression in immune function. Burned patients are then at great risk for a number of infectious complications, including bacterial wound infection, pneumonia, and fungal and viral infections
- Hypermetabolism
 - Characterized by tachycardia, increased cardiac output, elevated energy expenditure, increased oxygen consumption, proteolysis and lipolysis, and severe nitrogen loss, develops after severe burns

3. Burn wound assesement

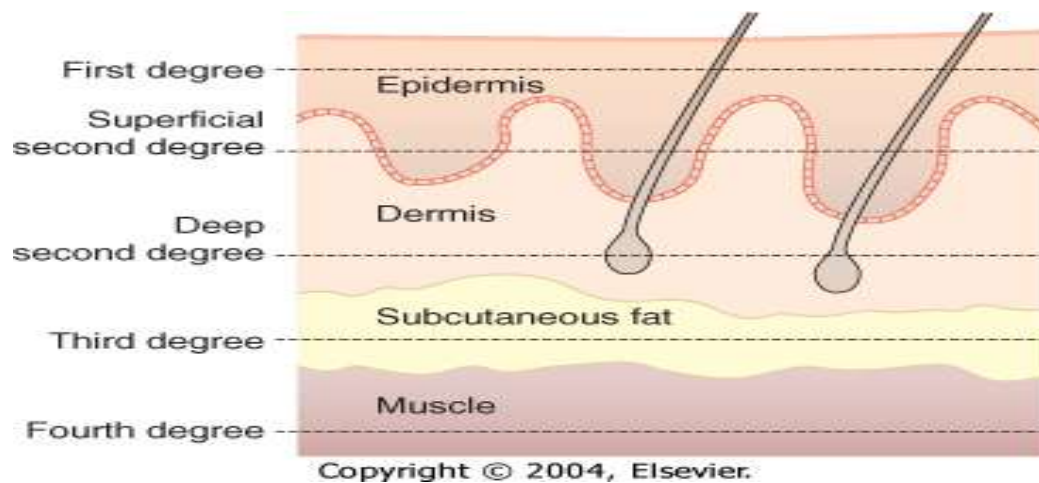
1. Burn depth

Accurate depth determination is critical because wounds that will heal with local treatment are treated differently from those requiring operative intervention

2. Burn size

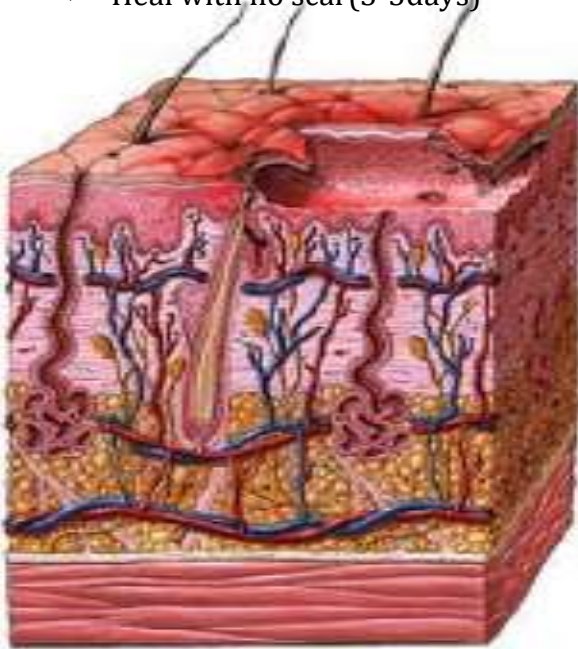


1. Burn depth



1st degree /superficial burn

- ✓ Only epidermis involved
- ✓ Painful, pink, blanch but no blister
- ✓ Heal with no scar(3-5days)



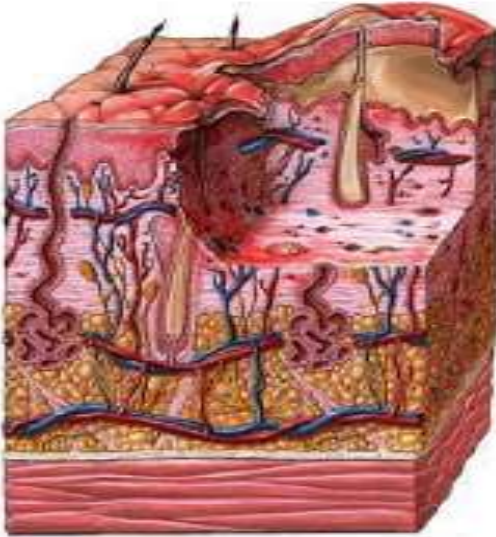
1st degree burn



Sun burn

Superficial second degree/partial thickness burn

- ✓ Epidermis + up to papillary dermis
- ✓ Extremely painful, blister, blanch with weeping
- ✓ There may be slight skin discoloration after healing (7-28days)



2nd degree burn



Deep second degree burn/partial thickness burn

- ✓ Epidermis + up to reticular dermis
- ✓ More pale, mottled, do not blanch to touch, sensation reduced
- ✓ Heals with severe scarring



3rd degree /full thickness burn

- ✓ Epidermis+ dermis + subcutaneous tissue
- ✓ Leathery, painless, and non blanching
- ✓ Requires excision with skin graft



3rd degree burn



Fourth degree burn

- ✓ Epidermis + dermis + subcutaneous tissue + muscles & bones
- ✓ Black, charred appearance
- ✓ Common in electrical burn



Summary

Degree		Extent	Characteristics	Remark
First degree or superficial		Localized to the Epidermis	Erythema,blanch to touch,painful	Eg.Sunburns Doesn't result in scars
Second degree	Superficial	Epidermis + portion of dermis(papillary)	Erythema,blanch,painful,often blister and weeping	There may be slight skin discoloration after healing
	Deep	Epidermis + dermis(up to reticular dermis)	More pale,mottled,do not blanch to touch,not sensitive to touch but sensitive to pin prick	Heals with sever scarring
Third degree		Full thickness(epidermis +dermis), no epidermal or dermal appendages remain	Hard,leathery,eschar,painless and nonblanching	Require excision with skin grafting
Fourth degree		Involving organs beneath the skin such as muscle or bones	Black, charred appearance	Common in electrical burn

Burn size

TOTAL BODY SURFACE AREA/TBSA

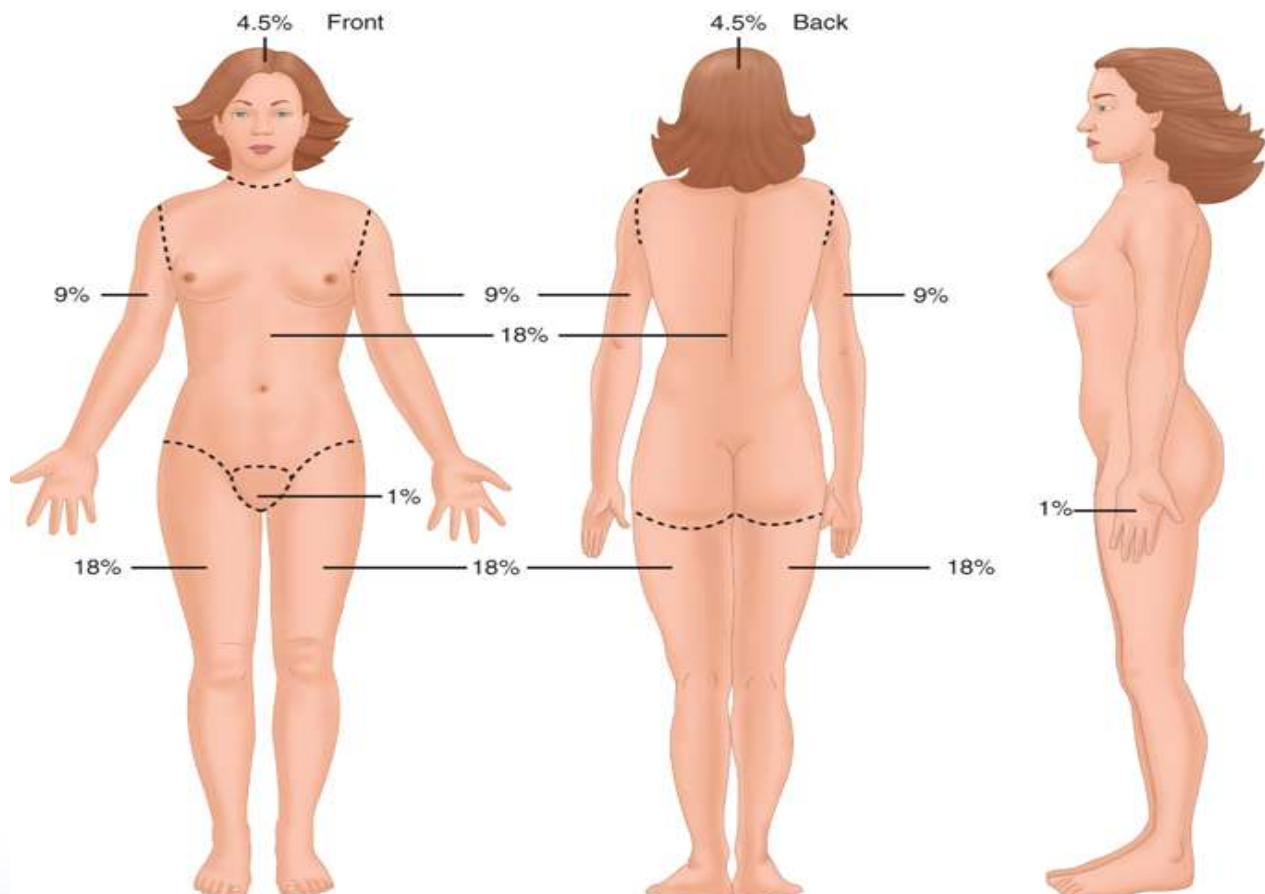
- A) **Rule of nines** divides the body – adequate for initial assessment for adult burns
- B) **Lund and Browder Chart** is the most accurate because it adjusts for age

NB

Superficial burns are not involved in the calculation

a) Rule of 9

- Head & Neck = 9%
- Each upper extremity (Arms) = 9%
- Each lower extremity (Legs) = 18%
- Anterior trunk = 18%
- Posterior trunk = 18%
- Genitalia (perineum) = 1%



b) Lund & Browder chart

Lund-Browder Chart									
Area	0-1 Years	1-4 Years	5-9 Years	10-14 Years	15 Years	Adult	% 2nd	% 3rd	% TOTAL
Head	19	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Ant. Trunk	13	13	13	13	13	13			
Post. Trunk	13	13	13	13	13	13			
R. Buttock	2.5	2.5	2.5	2.5	2.5	2.5			
L. Buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Genitalia	1	1	1	1	1	1			
R.U. Arm	4	4	4	4	4	4			
L.U. Arm	4	4	4	4	4	4			
R.L. Arm	3	3	3	3	3	3			
L.L. Arm	3	3	3	3	3	3			
R. Hand	2.5	2.5	2.5	2.5	2.5	2.5			
L. Hand	2.5	2.5	2.5	2.5	2.5	2.5			
R. Thigh	5.5	6.5	8	8.5	9	9.5			
L. Thigh	5.5	6.5	8	8.5	9	9.5			
R.L. Leg	5	5	5.5	6	6.5	7			
L.L. Leg	5	5	5.5	6	6.5	7			
R. Foot	3.5	3.5	3.5	3.5	3.5	3.5			
L. Foot	3.5	3.5	3.5	3.5	3.5	3.5			

From the Medical University of South Carolina Children's Hospital Pediatric Burn Intake Form. (Charleston, SC)

NB

palm method--small or patchy burns can be approximated by using the surface area of the patients palm. The entire (including fingers) is approximately 1% and is used in pediatric burn patients

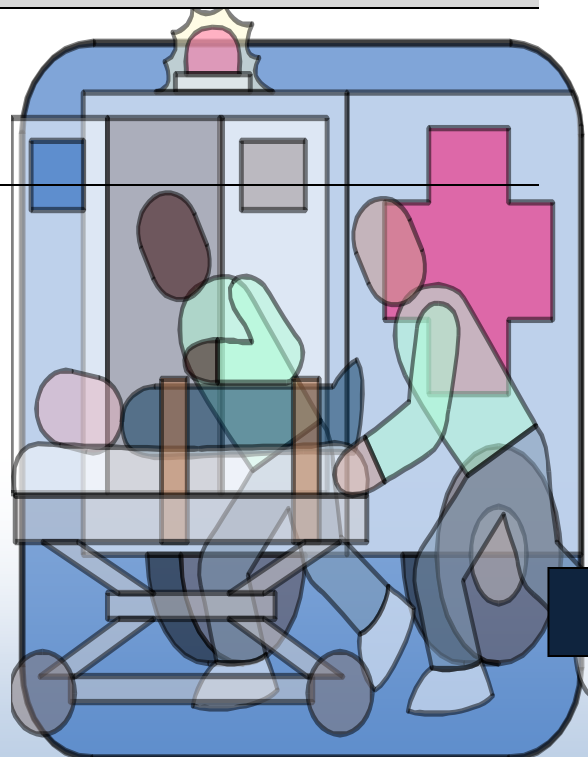
4. Burn patient care

- I. Prehospital care
- II. Hospital care

Prehospital care

- ☐ Thermal
 - Stop, drop & roll
- ☐ Chemical
 - Dilute chemicals
 - Remove clothing
- ☐ Electrical
 - Remove from source

***ABC of life



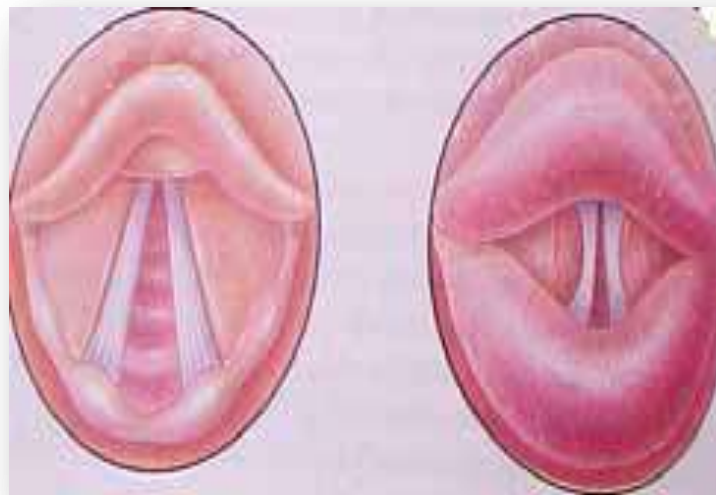
Indications for admission to burn center

- ☐ Burns involving the face, hands, feet, genitalia, perineum, or major joints
- ☐ Partial-thickness burns greater than 10% TBSA
- ☐ Third-degree burns in any age group
- ☐ Electrical burns, including lightning injury
- ☐ Chemical burns
- ☐ Suspected Inhalation injury
- ☐ Burn injury in patients with complicated pre-existing medical disorders
- ☐ Burned children in hospitals without qualified personnel for the care of children
- ☐ Burn injury in patients who will require special social, emotional, or rehabilitative intervention

Hospital care

Airway & breathing

- Airway injury must be suspected with facial burns, singed nasal hairs, carbonaceous sputum, and tachypnea.
- Upper airway obstruction may develop rapidly; the patient's respiratory status must be continually monitored to assess the need for airway control and ventilatory support.



- Progressive hoarseness is a sign of impending airway obstruction, and endotracheal intubation needs to be instituted early before edema distorts the upper airway anatomy.
- The chest is exposed in order to assess breathing; airway patency alone does not ensure adequate ventilation. Chest expansion and equal breath sounds with CO₂ return from the endotracheal tube ensure adequate air exchange.
- 100% O₂

NB

INHALATION INJURY

- One major factor contributing to death in burn injury patients is the presence of inhalation injury
- With inhalation injury, damage is caused primarily by inhaled toxins.

Circulation

Commonest types of fluid used in resuscitation

- Ringer's lactate or
- Hartmann's solution

These fluids

- ✓ Restore plasma volume
- ✓ Avoid microvascular ischemia
- ✓ Maintain vital organ function

The simplest and most widely used formula is the **Parkland formula**. This calculates the fluid to be replaced in the first 24 hours

❑ **$4 \times \text{TBSA}\% \times \text{Weight(Kg)} = \text{Volume(ml)}$**

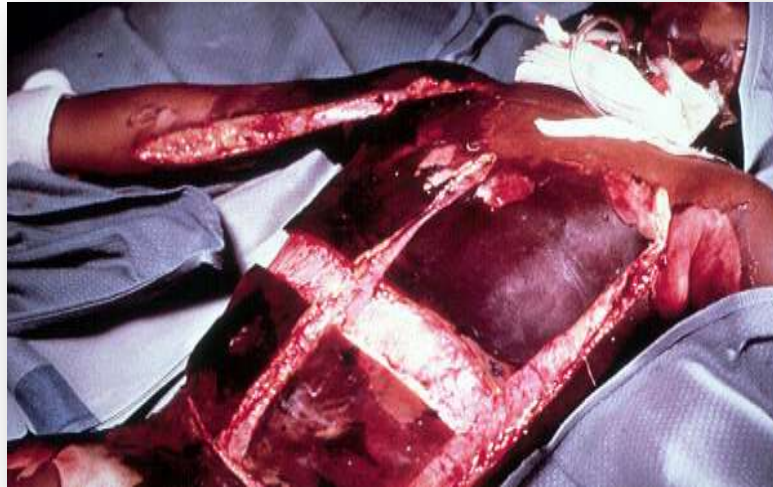
- ✓ Half this volume is given in the first 8 hours, and
- ✓ The second half is given in the subsequent 16 hours

- Donot give colloids in the first 24hours.
- Monitor the effectiveness of resuscitation by following Urine output, Blood pressure, and pulse rate
- Titrate to UOP of 0.5mL/kg/hr in adults and 1mL/kg/hr in children
 - If the urine output is below this, the infusion rate should be increased by 50%. If the urine output is inadequate and the patient is showing signs of hypo-perfusion (restlessness with tachycardia, cool peripheries and a high haematocrit)→then a bolus of 10 ml/kg body weight should be given.

Burn wound management

- Escharotomy

Circumferential full-thickness burns to the limbs require emergency surgery. The tourniquet effect of this injury is easily treated by incising the whole length of full-thickness burns.



- First-degree wounds are minor with minimal loss of barrier function. These wounds require no dressing and are treated with topical salves to decrease pain and keep the skin moist. Systemic NSAID agents given by mouth assist in pain control
- Second-degree wounds can be treated with daily dressing changes and topical antibiotics, cotton gauze, and elastic wraps
- Deep second-degree and third-degree wounds require excision and grafting for sizable burns, and the choice of initial dressing is aimed at holding bacterial proliferation in check and providing occlusion until surgery is performed.



Appropriate burn wound dressing

- protects the damaged epithelium
- minimizes bacterial and fungal colonization
- provides splinting action to maintain the desired position of function
- reduce evaporative heat loss and minimize cold stress
- provide comfort over the painful wound.

TAT

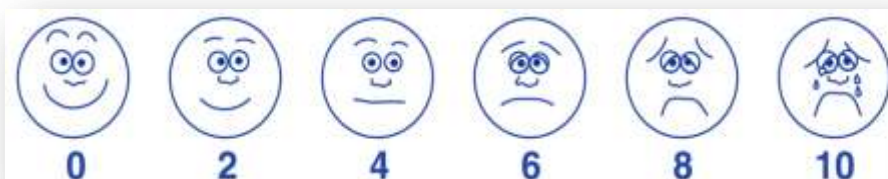
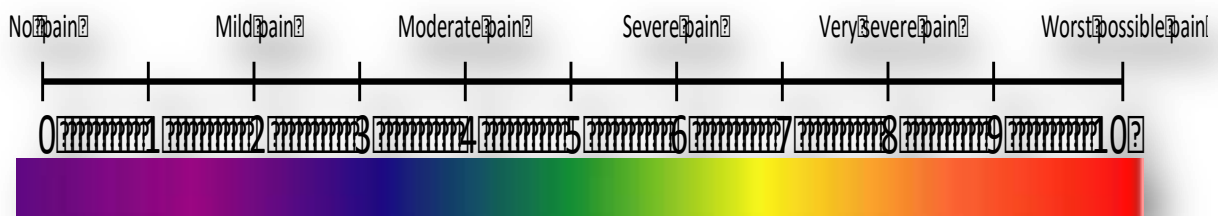
- Tetanus antitoxoid (TAT) should be updated for any burns deeper than superficial thickness

Pain management

- Pain severity scale

Scientifically validated pain scales:

- Numeric Pain Rating Scale
- Wong-Baker FACES Scale: for children who can talk
- Observation-FLACC Scale: for children who can't talk



©1983 Wong-Baker FACES™
Foundation. Used with permission.

- Manage the pain based on the score (see under Nephrolithiasis management part/ Debol long case)

GI Prophylaxis

- For prevention of stress ulcers/curling ulcer
 - Curling ulcer--Acute gastro duodenal ulcer.

Nutritional rehabilitation

- The response to injury known as hypermetabolism occurs dramatically after a severe burn

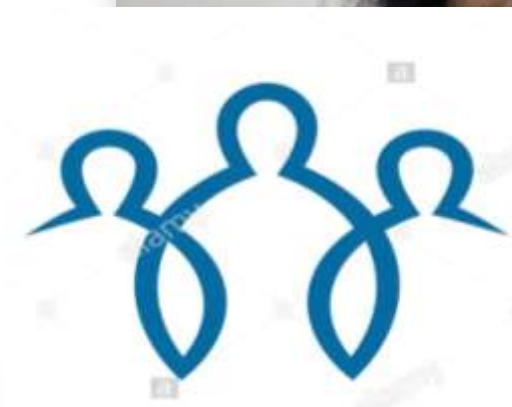
- Hypermetabolism → increased energy requirement, can be as high as 200% of the normal metabolic rate and returns to normal only with complete closure of the wound

The goals of nutritional support are to maintain and improve organ function and prevent protein-calorie malnutrition.

Physiotherapy



Psychological support



Non thermal burn injuries

Electrical burn injury

High voltage

- High-voltage injuries damage by flash (external burn) and conduction (internal burn)
- Myocardium may be directly damaged without pacing interruption
- Limbs may need fasciotomies or amputation
- Look for and treat acidosis and myoglobinuria



Low voltage

- Low-voltage injuries cause small, localized, deep burns
- They can cause cardiac arrest through pacing interruption without significant direct myocardial damage

- ❖ Electrical burns are more severe at the point of exit



- ❖ Follows path of least resistance towards ground
 - ✓ Nerve→bloodvessels→muscle→skin →tendon→fat and then bone
- ❖ Electrical energy is converted to heat in direct proportion to the resistance of the tissue
- ❖ Disrupts body's electrical activities
- ❖ Initial evaluation consists of cardiopulmonary resuscitation if ventricular fibrillation is induced



- ❖ Initial evaluation consists of cardiopulmonary resuscitation if ventricular fibrillation is induced.
 - ✓ Thereafter, if the initial electrocardiographic findings are abnormal or there is a history of cardiac arrest associated with the injury, continued cardiac monitoring is necessary along with pharmacologic treatment of any arrhythmias. The most serious derangements occur in the first 24 hours after injury
- ❖ Evaluation of the circulation to distal vascular beds because immediate escharotomy and fasciotomy may be required.
- ❖ vigorous hydration
 - ✓ Muscle damage results in the release of hemochromogens (myoglobin), which are filtered in the glomeruli and may result in obstructive nephropathy→AKI
- ❖ If the muscle compartment is extensively injured and necrotic such that the prospects for eventual function are dismal, early amputation may be necessary
- ❖ Delayed Effects →Neurologic deficits may occur
 - ✓ Such as cortical encephalopathy, hemiplegia, aphasia, and brainstem dysfunction have been reported up to 9 months after injury...

Chemical burn

Determine type of agent

- Strength
- Concentration
- Duration of contact
- Extent of penetration
- Mechanism of action

Alkaline materials worse than acid

- Acids cause coagulation necrosis
- Alkalis cause liquefaction necrosis allowing deeper penetration

Estimation of size and extent difficult



- Speed is essential in the management of chemical burns. For all chemicals, lavage with copious quantities of clean water needs to be performed immediately after removing all clothing. Dry powders must be brushed from the affected areas before irrigation. Early irrigation dilutes the chemical.
- Operative debridement, if indicated, takes place as soon as a patient is stable and resuscitated.
- After adequate lavage and debridement, burn wounds are covered with antimicrobial agents or skin substitutes.
- Once the wounds have stabilized with the indicated treatment, they are taken care of similar to any loss of soft tissue. Skin grafting or flap coverage is performed as needed.

SHOCK

Shock is defined as a failure to meet the metabolic demands of cells and tissues and the consequences that ensue.

Forms

Strict adherence to a classification scheme may be difficult from a clinical standpoint

- Hypovolemic Shock
- Obstructive Shock
- Cardiogenic Shock
- Neurogenic shock
- Septic shock
- Traumatic shock

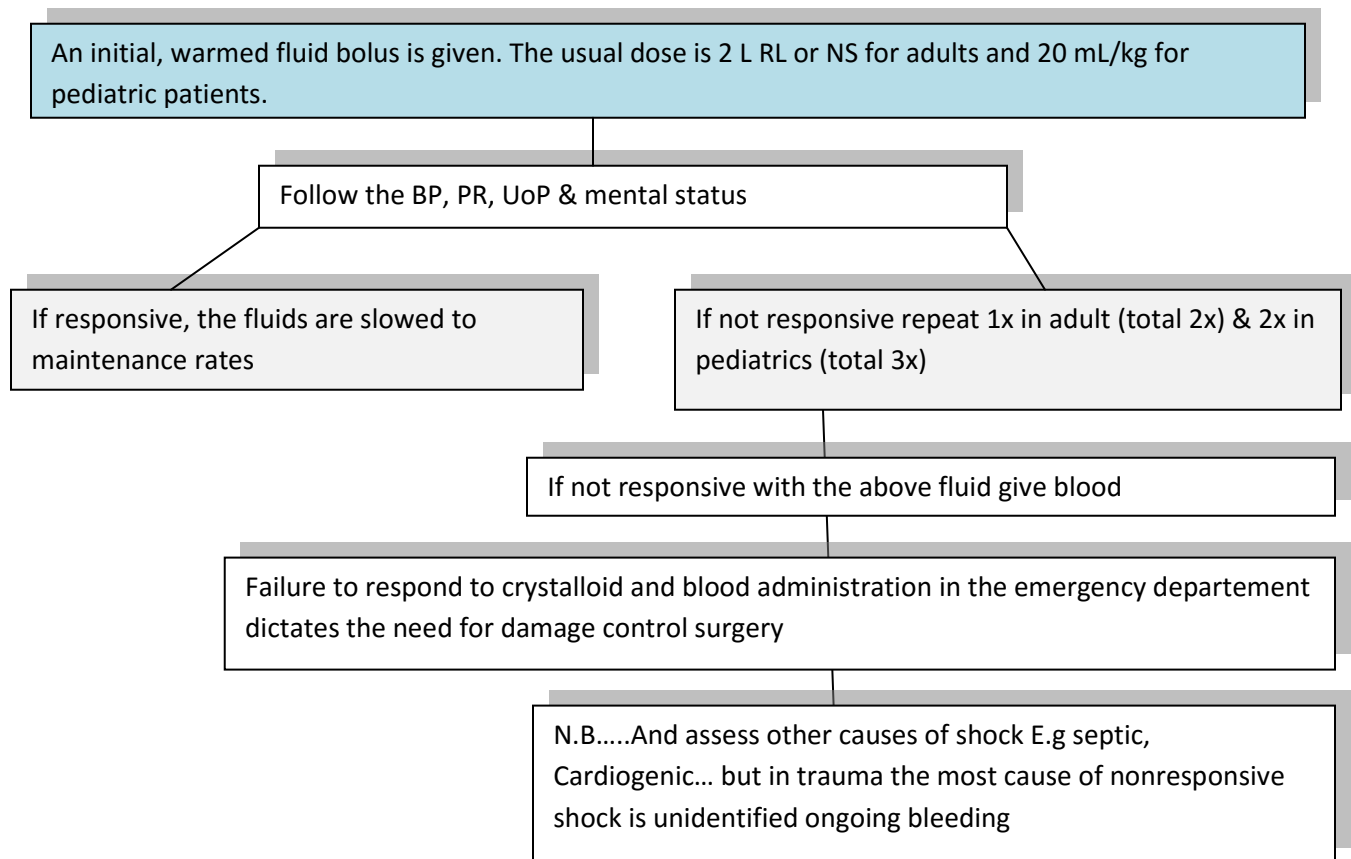
Hypovolemic shock

- ❖ most commonly encountered in surgical practice
- ❖ It can be non hemorrhagic or hemorrhagic shock
- ❖ Classified into four

PARAMETER	CLASS			
	I	II	III	IV
Blood loss (mL)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15	15–30	30–40	>40
Heart rate (bpm)	<100	>100	>120	>140
Blood pressure	Normal	Orthostatic	Hypotension	Severe hypotension
CNS symptoms	Normal	Anxious	Confused	Obtunded

Management of hemorrhagic shock

- The first step in the initial management of shock in trauma patients is to recognize its presence.
- The second step in the initial management of shock is to identify the probable cause of the shock state.
 - ❖ Mostly this is associated with trauma so first apply ATLS



- ✓ NGT
 - prevent aspiration
 - Prevent bradycardia
- ✓ Catheterization
 - Follow-up
- ✓ Analgesic

Based on the initial fluid resuscitation

	RAPID RESPONSE	TRANSIENT RESPONSE	MINIMAL OR NO RESPONSE
Vital signs	Return to normal	Transient improvement, recurrence of decreased blood pressure and increased heart rate	Remain abnormal
Estimated blood loss	Minimal (10%–20%)	Moderate and ongoing (20%–40%)	Severe (>40%)
Need for more crystalloid	Low	Low to moderate	Moderate as a bridge to transfusion
Need for blood	Low	Moderate to high	Immediate
Blood preparation	Type and crossmatch	Type-specific	Emergency blood release
Need for operative intervention	Possibly	Likely	Highly likely
Early presence of surgeon	Yes	Yes	Yes

How do differentiate nonresponsive causes of shock

Hemorrhagic shock	Cardiogenic shock
Central venous pressure (CVP) is decreased	CVP is increased

Cardiac tamponade	Tension pneumothorax
Tachycardia Distant heart sound Distended neck veins Breath sound normal	Absent breath sound Hyperresonant Distended neck veins

Condition	Pathophysiology
Tension pneumothorax	Compress heart → increased RV pressure finally decreased venous return
Massive hemothorax	Due to lost compression effect, like that of T.pneumothorax
Cardiac tamponade	Decreases the cardiac filling like that of T.pneumothorax

Septic shock

- Sepsis--evidence of an infection, as well as systemic signs of inflammation (e.g., fever, leukocytosis, and tachycardia).
- Severe sepsis--Hypoperfusion with signs of organ dysfunction
- Septic shock
 - Requires the presence of the above, associated with more significant evidence of tissue hypoperfusion and systemic hypotension.
 - Severe sepsis plus hypotension persisting ≥ 1 hour despite fluid resuscitation
- Hemodynamic changes are ...
 - Early septic shock (warm or hyperdynamic) characterized
 - ✓ Peripheral vasodilation,
 - ✓ Flushed and warm extremities, and
 - ✓ A compensatory elevation in cardiac output
 - ✓ Although an increase in venous capacitance diminishes venous return to the heart, cardiac output is maintained via tachycardia and the decrease in afterload due to systemic vasodilation
 - Late septic shock (cold or hypodynamic) characterized by
 - ✓ Impaired myocardial contractility due to local and systemic release of cardiac depressants,
 - ✓ Worsening peripheral perfusion, vasoconstriction, extremity mottling, oliguria, and hypotension

Management of septic shock

- ❖ If we diagnosed it from the out set
 - ABC of life
 - Stabilize respiration- airway and breathing
 - Severely obtunded & labored breathing -- require intubation and ventilation
 - Secure IV line or insert catheter(CVC)
 - 5-6 liters in 6 hours of therapy. i.e. initial 1-2L bolus in the first 1-2hour(least 30 mL/kg within the first 4 to 6 hours)
 - Assess V/S & presence/absence of pulmonary edema
 - Transfusion
 - Inotropic/Vasopressor: 2nd line
 - Dopamine--start with 2-4 μ gm/kg/min up to 30-50 μ gm/kg/min and escalate every 15-20 min maintain for at least 6 hours before tapering then discontinue
- ❖ Control of septic focus
 - Identification of septic focus: plus blood culture 2x
 - empirical antimicrobial agent in the 1st hour +/- surgical debridement

- ❖ Corticosteroids
 - hydrocortisone 50mg IV every 6 hr and
 - fludrocortisone 50µg orally once daily

Assessment of end points in resuscitation

- ❖ Generally those are classified as
 - ✓ Systemic (global)
 - Vital signs (BP, PR , UOP)
 - Lactate
 - Cardiac output
 - Base deficit
 - Oxygen delivery and consumption
 - ✓ Tissue specific
 - ✓ Cellular

BLOOD TRANSFUSION

Blood components

Banked Whole Blood

- Now rarely indicated and rarely available. With the new preservatives, the shelf life has been extended to 40 ± 5 days. At least 70% of the transfused erythrocytes remain in the circulation for 24 hours after transfusion and are viable. The hemolysis that occurs during storage is insignificant.

Fresh Whole Blood

- Refers to blood that is administered within 24 hours of its donation and is rarely indicated. Because of the time required for testing for infectious disease, it must be administered untested. One unit of platelet concentrate has more viable platelets than 1 unit of fresh blood. Fresh whole blood is a poor source of platelets and factor VIII.

Packed red cells

- Cells are spun down and concentrated.
- Each unit = 330 ml (Hct=50–70%)
- PRCs are stored in a SAG-M solution (saline–adenine–glucose–mannitol) to increase shelf-life to 5 weeks at $2-6^{\circ}\text{C}$.

Fresh-frozen plasma

- It is rich in coagulation factors & removed from fresh blood and stored at -40 to -50°C with a 2 years shelf-life. It is the first-line therapy in the treatment of coagulopathic haemorrhage.

Platelet

- Pooled platelet concentrate containing about 250×10^9 cells per litre. Platelets are stored on a special agitator at $20-24^{\circ}\text{C}$ and have a shelf-life of only 5 days.

Cryoprecipitate

- It is precipitate of FFP
- Rich in factor VIII and fibrinogen
- Stored at -30°C with a 2-year shelf-life
- Given in low-fibrinogen states or in factor VIII deficiency

Prothrombin complex concentrate

- Contain factors II, IX and X(factor VII may be included)
- Indicated for the emergency reversal of anti-coagulant (warfarin) therapy in uncontrolled haemorrhage

Indications for blood transfusion

- Acute blood loss, to replace circulating volume and maintain oxygen delivery; in case of hemorrhagic shock, major surgery, extensive burns.
- Perioperative anaemia, to ensure adequate oxygen delivery during the perioperative phase
- symptomatic chronic anaemia without haemorrhage or impending surgery

Complications from a single transfusion

ABO incompatibility

- Clinical presentation=hematuria,bilateral loin pain,fever,chills,rigor,oliguria(ATN)
- Rx=stop blood transfusion.send it to blood bank & recheck.
 - Repeat coagulation profile
 - Iv fluids,monitor UOP,U/A for Hgb
 - Diuretic- furosemide

Minor incompatibility reaction

- Due to extra vascular hemolysis(mild ,occur 2-21days)
- Clinical presentation-fever,malaise & jaundice
- Rx-supportive

Febrile reaction

- Due to sensitization to WBCs or platelets
- ↑ temperature but no hemolysis
- Use of 20-40mm filter or leukocyte depleted blood avoids it.

Allergic reaction

- Due to allergy to plasma products
- Clinical presentation-chills,rigor & rash
- Rx- antihistamines

Embolism

- Although air embolism has been reported as a complication, healthy individuals can tolerate large amounts of air injected intravenously at a rapid rate. The normal adult can tolerate an embolism of 200 mL of air. Smaller amounts, however, can cause alarming signs. Manifestations of venous air embolism include a rise in venous pressure, cyanosis, a "mill wheel murmur" over the precordium, tachycardia, hypotension, and syncope.
- Treatment - consist of placing the patient on the left side in a head down position with the feet up. Arterial air embolism may be visible in the retinal vessels or as blood flows from transected vessels. Plastic tubes used for transfusion can break off within a vein and embolize into the right atrium or pulmonary artery. Embolized catheters have been successfully removed.

Thrombophlebitis

- Prolonged infusions into a peripheral vein are associated with venous thrombosis. Intravenous infusions that last more than 8 hours are more likely to be followed by thrombophlebitis, with an increased incidence in the lower limbs.
- Treatment consists of discontinuation of the infusion and the application locally of warm moist compresses. Embolization from superficial thrombophlebitis or venous thrombosis is extremely rare.

Overtransfusion and Pulmonary Edema

- Overloading the circulation is an avoidable complication. It can occur with rapid infusion of blood, plasma expanders, and other fluids, particularly in patients with heart disease. To prevent the complication, the central venous pressure should be measured whenever large amounts of fluid are administered. Circulatory overload is

manifested by a rise in venous pressure, dyspnea, and cough. Rales can generally be heard at the lung bases.

- Treatment consists of diuresis, placing the patient in a sitting position, and, occasionally, venesection.

Syndrome of transfusion-related acute lung injury (TRALI)

- It is sometimes seen after transfusion and is characterized as mild to life-threatening. Noncardiogenic pulmonary edema is often accompanied by fever, rigors, and a "white out" chest x-ray.
- Treatment is discontinuation of any transfusion, notification of the transfusion service, and intensive pulmonary support, sometimes including intubation. This type of pulmonary edema does not respond to diuretics and is thought to be caused by infused donor antigranulocyte or anticlass I or II MHC antibodies.

Infection

- Bacterial infection - contamination of infused blood is rare and can be acquired from contaminated collection bags or poor cleaning of the donor's skin. Gram-negative organisms, especially coliform and *Pseudomonas* species, which are capable of growth at 4°C (39.2°F), are the most common cause. Clinical manifestations include fever, chills, abdominal cramps, vomiting, and diarrhea. There may be hemorrhagic manifestations and increased bleeding. If the diagnosis is suspected, the transfusion should be discontinued and the blood cultured. Emergency treatment includes oxygen, adrenergic blocking agents, and antibiotics.
- Hepatitis, HIV, malaria, syphilis.
 - It is mandatory to screen the blood for these diseases before transfusion.

Complications from massive transfusion

The term massive transfusion implies a single transfusion greater than 2500 mL or 5000 mL transfused over a period of 24 hours

- Coagulopathy
- Hypocalcaemia
- Hyperkalaemia
- Hypokalaemia
- Hypothermia
- Iron overload due to repeated transfusions over long periods of time /eg. Thalassaemia
 - Each transfused unit of RBCs contains 250 mg of elemental iron

FLUID & ELECTROLYTE IMBALANCE

BODY FLUIDS

- Total Body Water(TBW)
 - ✓ 60% in an average young adult male & 50% in young adult female
 - ✓ The highest percentage of TBW is found in newborns, with approximately 80% of their total body weight.

Classification of Body Fluid Changes

- Disorders in fluid balance may be classified into three general categories: disturbances in;
 - ❖ volume
 - ❖ concentration &
 - ❖ Composition
- Although each of these may occur simultaneously, each is a separate entity with unique mechanisms demanding individual correction

Disturbances in Fluid Balance

- ❖ Extracellular volume deficit is the most common fluid disorder in surgical patients and can be either acute or chronic.
 - Acute volume deficit is associated with cardiovascular and central nervous system signs (shock)
 - Chronic deficits display tissue signs, such as a decrease in skin turgor and sunken eyes (dehydration), in addition to cardiovascular and central nervous system signs.
 - Shock Vs dehydration???

FLUID AND ELECTROLYTE THERAPY

- ❖ The type of fluid administered depends on the patient's volume status and the type of concentration or compositional abnormality present.
- ❖ Both RL & NS are considered isotonic crystalloids and are useful in replacing GI losses and correcting extracellular volume deficits.

Crystalloids	colloids
✓ <i>Are balanced salt solutions</i> ✓ <i>use to restore extracellular volume by exerting significant hydrostatic effect on capillaries due to this pulmonary & GI wall edema is common</i> ✓ <i>Causes hemodilution due larger volume required</i> ✓ <i>Although it expands the intravascular volume, it also expands the interstitial space by approximately three times as much as the plasma.</i> ✓ <i>Cheap</i>	✓ <i>Contain larger molecules & remain in the intravascular compartment</i> ✓ <i>expand the plasma by increasing the colloid oncotic pressure</i> ✓ <i>requiring smaller volumes for resuscitation than crystalloids</i> ✓ <i>Allergic reaction is common</i> ✓ <i>Pulmonary edema possible if given in injured lung b.c it can extravasates</i> ✓ <i>Expensive</i>

Crystalloids

- ❖ Solutions of electrolytes and water are collectively referred to as crystalloids
- ❖ the most widely used fluids for parenteral administration
- ❖ Inexpensive, highly effective for fluid maintenance and replacement, and have outstanding safety profiles

Types of crystalloids

- RL (ringer lactate)
 - ✓ Slightly hypotonic in that it contains 130 mEq of lactate.
 - ✓ It is ideal for the replacement of existing fluid deficits when serum electrolyte concentrations are normal. why?
 - Because Lactate is used rather than bicarbonate because it is more stable in IV fluids during storage. It is converted into bicarbonate (used to buffer acid base imbalance) by the liver after infusion, even in the face of hemorrhagic shock.
- NS (normal saline /Sodium chloride(0.9%))
 - ✓ is mildly hypertonic, containing 154 mEq of sodium that is balanced by 154 mEq of chloride.
 - ✓ The high chloride concentration imposes a significant chloride load on the kidneys and may lead to a hyperchloremic metabolic acidosis.
 - So, it is an ideal solution, for correcting volume deficits associated with hyponatremia, hypochloremia, and metabolic alkaloids
- D5W (5% dextrose)
 - ✓ 50 g of dextrose per liter--supplies 200 kcal/L
 - Dextrose is always added to solutions containing <0.45% sodium chloride (hypotonic) to maintain osmolality and thus prevent the lysis of RBCs that may occur with rapid infusion of hypotonic fluids. The addition of potassium is useful once adequate renal function and urine output are established.
- 0.45% sodium chloride
 - ✓ useful for replacement of ongoing GI losses as well as for maintenance fluid therapy in the postoperative period.
 - ✓ This solution provides sufficient free water for insensible losses and enough sodium to aid the kidneys in adjustment of serum sodium levels.
 - ✓ This hypotonic solution causes lysis of RBCs--to prevent this add 5% dextrose (50 g of dextrose per liter)

Alternative Resuscitative Fluids

- ❖ Hypertonic saline
 - ✓ Hypertonic saline solutions (3.5% and 5%)--used for correction of severe sodium deficits
 - ✓ Hypertonic saline (7.5%)
 - has been used as a treatment modality in patients with closed head injuries.
 - It has been shown to increase cerebral perfusion and decrease intracranial pressure, thus decreasing brain edema.

- appears to increase intravascular volume in patients more quickly than isotonic solutions, and the total resuscitation volume requirement may be decreased
- reduces the systemic inflammatory response syndrome (SIRS) and may attenuate multiple organ dysfunction syndrome
- These cardiovascular and hemodynamic effects are short-lived, in general lasting from 60 to 120 minutes. The addition of colloids such as dextran or hetastarch can prolong the effect.
- Adverse effect
 - ❖ increased bleeding--because hypertonic saline is an arteriolar vasodilator
 - ❖ electrolyte abnormalities such as hypernatremia

Colloids

- ✓ also are used in surgical patients
- ✓ Due to their molecular weight, they are confined to the intravascular space, and their infusion results in more efficient transient plasma volume expansion.
- ✓ However, under conditions of severe hemorrhagic shock, capillary membrane permeability increases; this permits colloids to enter the interstitial space, which can worsen edema and impair tissue oxygenation
- ✓ Colloid solutions with smaller particles and lower molecular weights exert a greater oncotic effect but are retained within the circulation for a shorter period of time than larger and higher molecular weight colloids.
- ✓ Are limited in use because of their doubtful effectiveness, cost, and potential complication.

Four major types of colloids are available

- ✓ Albumin
- ✓ Dextran
- ✓ hetastarch, and
- ✓ Gelatin

- ❖ Albumin (molecular weight 70,000)
 - ✓ is prepared from heat-sterilized pooled human plasma.
 - ✓ It is typically available as either a 5% solution (osmolality of 300 mOsm/L) or 25% solution (osmolality of 1500 mOsm/L).
 - ✓ Because it is a derivative of blood, it can be associated with allergic reactions.
 - ✓ Albumin has been shown to induce renal failure and impair pulmonary function when used for resuscitation in hemorrhagic shock.
- ❖ Dextran
 - ✓ are glucose polymers produced by bacteria grown on sucrose media and are available as either 40,000 or 70,000 molecular weight solutions.
 - ✓ They lead to initial volume expansion due to their osmotic effect but are associated with alterations in blood viscosity.
 - ✓ Thus dextran are used primarily to lower blood viscosity rather than as volume expanders.

- ✓ Dextrans have been used, in association with hypertonic saline, to help maintain intravascular volume.
- ❖ Hetastarches
 - ✓ Administration of hetastarch can cause hemostatic derangements related to decreases in von Willebrand's factor and factor VIII
 - ✓ And its use has been associated with postoperative bleeding in cardiac and neurosurgery patients.
 - ✓ Hetastarch also can induce renal dysfunction in patients with septic shock and in recipients of kidneys procured from brain-dead donors.
 - ✓ Currently, hetastarch has a limited role in massive resuscitation because of the associated coagulopathy and hyperchloremic acidosis (due to its high chloride content).

CORRECTION OF VOLUME ABNORMALITIES

- Maintenance Fluid (MF) Therapy
 - ✓ It is the usual daily requirement. It to replace basal fluid requirement i.e the sensible and insensible losses
 - ✓ Calculation of MF replacement doesn't include replacement of either preexisting deficits or ongoing additional losses

Calculated as;

For 0-10 kg	Give 100 mL/kg/d
For the next 10-20 kg	Give an additional 50 mL/kg/d
For weight >20 kg ^b	Give 20 mL/kg/d

E.g. MF for 70kg pt ($10\text{kg} \times 100\text{ml} + 10\text{kg} \times 50\text{ml} + 50\text{kg} \times 20\text{ml}$) = 2500ml per day

- $2500\text{ml} / 3 = 833 \text{ ml over } 8\text{hrs}$
 - $1\text{ml} = 20\text{drops through IV line}$
 - $833\text{ml} = 16,660 \text{ drop}$
 - $8\text{hrs} = 8 \times 60\text{min} = 480 \text{ min}$
- If we want 16,660 drop (meaning 833ml) over 480min (meaning 8 hrs), we have to adjust by counting the drop to around 35 drops per minute. ($16,660 / 480$)
- ✓ For maintenance therapy, 1 to 2 mEq/kg per day of sodium is required & Potassium requirements are approximately 0.5 to 1 mEq/kg per day.
 - e.g. If sodium is replaced at 2 mEq/kg per day and potassium is replaced at 1 mEq/kg per day, a 70-kg patient requires 2,500 mL of water containing 140 mEq of sodium and 70 mEq of potassium.
- ✓ Short-term maintenance therapy generally does not require addition of calcium, phosphate, or magnesium
- ✓ Replacement of Ongoing Fluid Losses
 - Once volume deficits have been replaced and maintenance fluids have been calculated and given, the overall fluid balance of the patient can be

maintained by replacement of fluid losses beyond those considered to be maintenance

- Ongoing losses from nasogastric tubes, stomas, fistulas, and other measurable sources are recorded during the course of care and can be replaced in fairly straight forward fashion
- ✓ Intraoperative Fluid
 - Intraoperative replacement with isotonic solutions is usually accomplished at rates of 500 to 1,000 mL/h.
- ✓ Postoperative Fluid Therapy and Monitoring
 - is adjusted to the patient's volume status at the completion of the operative procedure, as well as to anticipated ongoing fluid losses
 - Isotonic solutions should be used for volume resuscitation during the early postoperative period.
 - It is best not to give potassium supplements during this period without indication. Why? Because surgery by itself causes Hyperkalemia due cell damage & stress.
 - Routine monitoring of postoperative fluid status consists
 - ✓ serial vital signs and urine output measurements
 - ✓ intake and output data are recorded
 - ✓ Weight should also be recorded daily. Because postsurgical rapid fluctuations in weight are generally related to changes in TBW
 - ✓ Urine specific gravity
 - ✓ greater than 1.010 to 1.012 indicates that the urine is being concentrated (relative to plasma)
 - ✓ less than 1.010 indicates that dilute urine is being produced
 - ✓ Both volume depletion and cardiac failure are accompanied by increased urine concentration and low urine outputs.
 - ✓ Urine specific gravity in the range of plasma (1.010 to 1.012) may indicate either adequate hydration or the inability of the kidneys either to dilute or concentrate the urine

Correction of Life-Threatening Electrolyte Abnormalities

Hypernatremia

- Hypernatremia is a less common problem in surgical patients than hyponatremia. Mainly due to concentration changes in body Fluids. I.e. sodium abnormality is mainly affected by body fluid status (Volume excess or deficits) than intake.
- It can happen in low, normal or high volume status

Clinical Features

- ❖ Symptomatic hypernatremia usually occurs only in patients with impaired thirst or restricted access to fluid, because thirst will result in increased water intake.
- ❖ Symptoms are rare until the serum sodium concentration exceeds 160 mEq/L but, once present, are associated with significant morbidity and mortality.

- ❖ symptoms are related to hyperosmolarity , central nervous system effects predominate
- ❖ Water shifts from the intracellular to the extracellular space which results in cellular dehydration
- ❖ range from restlessness and irritability to seizures, coma, ataxia, spasm and death

Treatment

- ❖ Treatment of hypernatremia usually consists of treatment of the associated water deficit.
- ❖ In hypovolemic patients, volume should be restored with normal saline before the concentration abnormality is addressed
- ❖ The formula used to estimate the amount of water required to correct hypernatremia is as follows:

$$\text{water requirement} = \frac{\text{desired change in } [Na^+]_s \times TBW}{\text{desired } [Na^+]_s}$$

- ❖ E.g. a 70-kg patient with a TBW of 42 L (TBW 60 %) has serum sodium concentration of 170 mEq/L. So what is his water requirement?
 - Given
 - ❖ current serum sodium =170 meq/l
 - ❖ TBW=42 L
 - ❖ normal sodium = 154 meq/l
 - Required= water requirement?
 - Solution
 - ❖ Desired change in Na=170-154=16 meq/l
 - ❖ Water requirement= 16x42/154=4.36 L
- ❖ How much the rate?
 - ✓ Slow for chronic hyponatremia. why?
 - In chronic hypernatremia, the cells in the brain gradually adapt by increasing intracellular osmotic solute content, thereby regaining cellular volume. These cellular changes are not readily reversed. A sudden decrease in extracellular sodium concentration, and therefore osmolality, results in cell , brain edema.
- ❖ The rate of fluid administration should be titrated to achieve a decrease in serum sodium concentration of no more than 1 mEq/h and 12 mEq/day for the treatment of acute symptomatic hypernatremia.
- ❖ Even slower correction should be undertaken for chronic hypernatremia (0.7 mEq/h),
- ❖ Oral or enteral replacement is acceptable in most cases, or IV replacement with half- or quarter-normal saline can be used
- ❖ Frequent neurologic evaluation as well as frequent evaluation of serum sodium levels also should be performed.

Hyponatremia

- ❖ Frequently seen in the post-op or post injury period when ADH is elevated as a component of the normal stress response to injury
- ❖ As a consequence of either sodium depletion or dilution
- ❖ Because volume expansion and hyponatremia diminish the effects of ADH on the collecting tubules--is usually self-limited under these circumstances, with serum sodium concentration rarely falling below 130 mEq/L unless exacerbated by exogenous free water administration.

Clinical Features

- ❖ Chronic hyponatremia is often asymptomatic until serum [Na⁺] falls below 110 to 120 mEq/L
- ❖ In acute drop serum [Na⁺] to the 120 to 130 mEq/L range may be symptomatic
- ❖ Symptoms related to the CNS result largely from cellular water intoxication, and permanent CNS injury can occur if it is left untreated.
- ❖ Central nervous system
 - ✓ Headaches, Confusion, Delirium, Coma, Seizure...
- ❖ Gastrointestinal Anorexia
 - ✓ Nausea & Vomiting
- ❖ Musculoskeletal Weakness
 - ✓ Fatigue, Muscle cramps...

Treatment

- ❖ Most cases of hyponatremia can be treated by free water restriction and, if severe by administration of sodium.
- ❖ If neurologic symptoms are present, 3%NS should be used to increase the sodium by no more than 1 mEq/L per hour until the serum sodium level reaches 130 mEq/L or neurologic symptoms are improved.
- ❖ Correction of asymptomatic hyponatremia should increase the sodium level by no more than 0.5 mEq/L per hour to a maximum increase of 12 mEq/L per day, and even more slowly in chronic hyponatremia.
- ❖ The rapid correction of hyponatremia is not recommended

Hyperkalemia

- Defined as a serum potassium concentration > the normal range (3.5 to 5.0 mEq/L)
- It is caused by
 - ✓ Excessive potassium intake--can be either from oral or IV supplementation, or from red cell lysis after transfusion
 - ✓ Increased release of potassium from cells--Acidosis and a rapid rise in extracellular osmolality from hyperglycemia or IV mannitol can raise serum potassium levels by causing a shift of potassium ions to the extracellular compartment.
 - ✓ Impaired potassium excretion by the kidneys—as in case of potassium-sparing diuretics, renal insufficiency/failure...
 - ✓ Because 98% of total body potassium is in the intracellular fluid compartment, even small shifts of intracellular potassium out of the intracellular fluid compartment can lead to a significant rise in extracellular potassium.

Clinical feature

- Clinical manifestations of hyperkalemia are primarily related to membrane depolarization related to intra & extracellular imbalance
- The most life-threatening manifestations are related to the cardiac effects
 - ✓ Arrhythmia, arrest
 - ✓ Peaked T waves (early), widened QRS complex, flattened P wave, prolonged PR interval, sine wave formation, and ventricular fibrillation.
- Neuromuscular manifestations of severe hyperkalemia include weakness progressing to flaccid paralysis
- GI... Nausea/vomiting, colic, diarrhea

Treatment goals

- protecting the cells from the effects of increased potassium (neutralization)
- reducing the total body potassium
- shifting potassium from the extracellular to the intracellular space
- For all patients, exogenous sources of potassium should be removed, including potassium supplementation in IV fluids and enteral and parenteral solution.
- Potassium removal--Is the definitive therapy
 - ✓ removed from the body using a cation-exchange resin such as Kayexalate that binds potassium in exchange for sodium
 - ✓ Kayexalate --usual oral dose is 40 g dissolved in 20 to 100 mL of sorbitol
 - ✓ Each gram removes approximately 1 mEq of potassium
- Shift potassium
 - ✓ regular insulin 5–10 units IV & Glucose 1 ampule of D50
 - ✓ Why D50 added...? To avoid insulin-induced hypoglycemia
 - ✓ Bicarbonate 1 ampule IV--MOA; the increase in extracellular pH shifts potassium into the cells
- Counteract cardiac--urgent & first Rx in severe cases
 - ✓ Calcium gluconate 5–10 mL of 10% solution
 - ✓ to counteract the myocardial effects of hyperkalemia
 - ✓ The effects are transient and usually last approximately 30 minutes

Hypokalemia

- Hypokalemia is much more common than hyperkalemia in the surgical patient.
- caused by
 - ✓ inadequate potassium intake
 - ✓ excessive renal potassium excretion
 - ✓ potassium loss in pathologic GI secretions, such as with diarrhea, fistulas, vomiting, or high nasogastric output
 - ✓ Intracellular shifts from metabolic alkalosis or insulin therapy.

Potassium decreases by 0.3 mEq/L for every 0.1 increase in pH above normal.

Symptoms

- ☐ Ileus, constipation
- ☐ Decreased reflexes, fatigue, weakness, paralysis
- ☐ Cardiovascular Arrest

Treatment

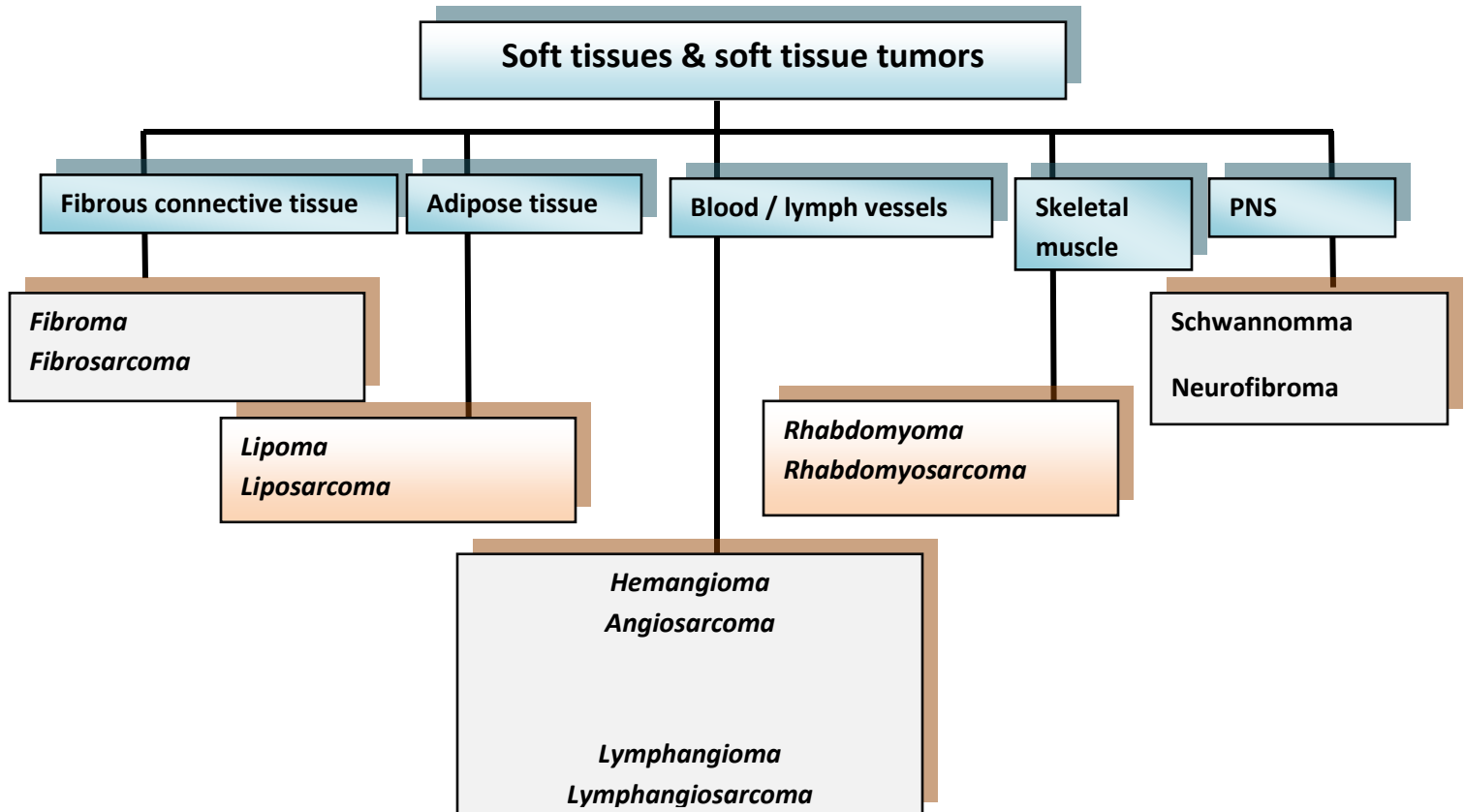
- ☐ consists of potassium repletion, the rate of which is determined by the symptoms
 - ✓ Oral repletion is adequate for mild, asymptomatic
 - ✓ If IV repletion is required, usually no more than 10 mEq/h is advisable in an unmonitored setting but in monitored setting can be increased to 40 mEq/h
 - ✓ Caution should be exercised when oliguria or impaired renal function is coexistent

SOFT TISSUE TUMORS

Soft tissue = supportive tissue of various organs & the non-epithelial, extra-skeletal structures exclusive of lymphohematopoietic tissues

NB

When you are asked to list DDx for a swelling...think of the structures that are found underlying/overlying the swelling like fibrous connective tissue, adipose tissue, vessels, skeletal muscles, nerves & bones. Then list the benign & malignant disease forms of each structure. E.g. fibroma, fibrosarcoma (for fibrous CT)



Predisposing factors

- Genetic factors
- Radiation
- Chronic lymphedema
- Environmental carcinogens
- Infections

Clinical presentation

- Mass
 - ✓ Common sign of soft tissue tumor
 - ✓ Usually painless
 - ✓ If it has rapid growth suspect malignancy
- See mass physical examination under short cases of Debol

SKIN GRAFT AND FLAP

Graft

- A tissue of epidermis and varying amounts of dermis that is detached from its own blood supply and placed in a new area with a new blood supply.

Flap

- Any tissue used for reconstruction or wound closure that retains all or part of its original blood supply after the tissue has been moved to the recipient location.

Classification of graft

1. Auto-graft--a tissue transferred from one part of the body to the other body part
2. Homograft(allograft)-- a tissue transferred from a genetically different individual of the same species
3. Xeno-graft--a tissue transferred from an individual of one species to other species

Indications

- Generally to achieve temporary or permanent cover
 - temporary cover—for open wound & to prevent infection
 - Permanent cover for
 - Skin loss secondary to;
 - post traumatic (avulsion and degloving injuries)
 - post-surgical (excision of burn wound)
 - infection
 - Mucosal loss (excision lesion of oral cavity or tongue)
 - Cosmetic reasons

Contraindication

- Beta hemolytic streptococcal Infection
- Infected wound with copious discharge
- Avascular wound

Types of skin grafts

Type	Definition
Split-thickness grafts	• superficial and some deep layers of skin(contain 100% of the epidermis & some part of dermis) • Split-thickness grafts are used for non-weight-bearing parts of the body • Donor sites: - thigh, buttock & abdomen • Can be of two type:- thin split thickness and thick split thickness
Full-thickness grafts	• Contains all of the layers of the skin including blood vessels • For weight-bearing portions of the body and friction prone areas such as, feet and joints. • Resistant to trauma with minimal secondary contractions • Donor sites: - groin and medial aspect of the arm
Pinch grafts	• quarter inch pieces of skin • will then grow to cover injured sites (grow even in areas of poor blood supply and resist infection)
Pedicle grafts	• skin used from the donor site will remain attached to the donor area (remainder is attached to the recipient site) • Blood supply remains intact at the donor location until the new blood supply has completely developed. • Mostly used for hands, face or neck areas of the body

Comparison between grafts

Type of Graft	Advantages	Disadvantages
Thin Split Thickness	- Best Survival - Heals Rapidly	- Least resembles original skin. - Least resistance to trauma. - Poor Sensation - Maximal Secondary Contraction
Thick Split Thickness	- More qualities of normal skin. - Less Contraction - Looks better - Fair Sensation	- Lower graft survival - Slower healing
Full Thickness	- Most resembles normal skin. - Minimal Secondary contraction - Resistant to trauma - Good Sensation - Aesthetically pleasing	- Poorest survival. - Donor site must be closed surgically. - Donor sites are limited.

Requirement for graft survival

- Bed must be well vascularized
- Contact between graft and recipient should be immobile
- Low bacterial count at the site

Factors that contribute for graft failure

- Malnutrition
- Sepsis
- DM, HIV, immune status, malignancy, CKD, liver failure
- Drugs: - steroids, chemotherapy, vasoconstrictors
- Recipient site of the graft like bone, tendon, infected wound & highly irradiated areas

Ideal donor site would provide: -

- Skin that is identical to the skin surrounding the recipient area in terms of Color, thickness, hair & texture.

ANESTHESIA

Anesthesia → temporary induced loss of sensation or awareness.

- It may include
 - analgesia (relief from or prevention of pain)
 - paralysis (muscle relaxation)
 - amnesia (loss of memory) or
 - Unconsciousness.

TYPES OF ANESTHESIA

1) General anesthesia

- ✓ To do most complex surgical procedures.
- ✓ Has 3 distinct phases
- ✓ Induction
 - For adults is usually achieved with the injection of intravenous medications. Propofol is commonly used because of its favorable recovery profile and short elimination half-life. But may induce bradycardia & hypotension. Other less commonly used drugs are etomidate & ketamine.
 - Etomidate : used when vasodilation and cardiac depression are undesirable
 - Ketamine: analgesia, preserve respiratory drive, bronchodilator and CVS stimulant.
- ✓ Maintenance
 - Achieved with volatile or intravenous (IV) anesthetics.
 - Volatile agents are popular because of their ease of delivery, reliable recovery, excellent safety profile and mode of onset. But can cause fulminant hepatic necrosis
- ✓ Emergence ("waking up")
 - Patient backs to a restored state of consciousness.
 - Autonomic hyper-responsiveness → short period of time in which patient's body is aware of emergence without full return to consciousness. Manifest as hypertension, tachycardia, bronchospasm, or laryngospasm. Short acting narcotics, beta blockers or lidocaine can blunt these responses

2) Peripheral nerve block

- ✓ For procedures involving the extremities.
- ✓ major relaxation and analgesia of the selected area without hemodynamic instability
- ✓ Also used for operative anesthesia or post-operative pain control.
- ✓ Commonly blocked nerve groups
 - Brachial plexus(interscalene block, infraclavicular block)
 - The sciatic nerve (posterior or lateral approach), and
 - The femoral nerve group (3 in 1 block)

3) Intravenous regional block(Bier block)

- is alternative to peripheral nerve block for extremity surgery, usually the hand or forearm. It can be applied in pain therapy (eg, chronic regional pain syndrome)

4) Neuraxial anesthesia

For patients undergoing surgery of the lower extremities or abdomen

- A) Spinal anesthesia
- B) Epidural Anesthesia

A) Spinal anaesthesia

- also called spinal b ,subarachnoid b ,intradural b and intrathecal block
- Involves the injection of a local anaesthetic into the subarachnoid space.

Indications

- Hemorrhoidectomy
- Hernia (inguinal or epigastric)
- Transurethral resection of the prostate and transurethral resection of bladder tumors
- Hysterectomy in different techniques used
- Caesarean sections
- Orthopedic surgery on the pelvis, hip, femur, knee, tibia, and ankle
- Vascular surgery on the legs
- Endovascular aortic aneurysm repair
- Nephrectomy and cystectomy in combination with general anaesthesia
- severe respiratory disease such as COPD as it avoids intubation and ventilation

Contraindications

- Non-availability of patient's consent
- Local infection or sepsis at the site
- Bleeding disorders, thrombocytopaenia, or systemic anticoagulation
- Space occupying lesions of the brain
- Anatomical disorders of the spine
- Hypovolemia
- stenosis valvular diseases
- allergy to anesthetics
- Multiple sclerosis-controversial...

Complications

- Hypotension (Neurogenic shock) - Due to sympathetic nervous system blockade.
 - treated with iv fluid and sympathomimetic drugs
- post spinal head ache --associated with size and type of spinal needle used
- Cauda equina injury--very rare, due to the insertion site being too high
- Cardiac arrest--very rare, usually related to the underlying medical condition of the patient
- Spinal canal hematoma--Urgent CT/MRI to confirm the dx followed by
 - urgent surgical decompression to avoid permanent neurological damage
- Epidural abscess--May present as meningitis or abscess.
 - Urgent CT/MRI confirms the dx followed by antibiotics and urgent surgical drainage

Limitations

- Limited to procedures involving most structures below the upper abdomen.
- To administer a spinal anesthetic to higher levels may affect the ability to breathe by paralysing the intercostal respiratory muscles, or even the diaphragm in extreme cases (called a "high spinal", or a "total spinal", with which consciousness is lost), as well as the body's ability to control the heart rate via the cardiac accelerator fibres.
- Also, injection of spinal anaesthesia higher than the level of L1 can cause damage to the spinal cord, and is therefore usually not.

<https://www.youtube.com/watch?v=RiqQleLPIBg>

Miscellaneous—watch examination of Varicose Vein

CHAPTER 22 -- VIVA TIPS

1. Basic surgical instrument
2. Basic radiological images
3. Case scenario (read long & short cases)

1. Basic surgical instruments



Artery forceps/ Haemostat



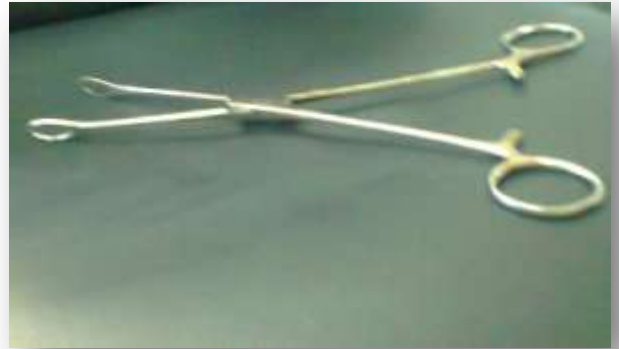
Blade holder



Knife/scalpel



Sponge forceps



Needle holder





**Toothed pick up/tissue
forceps**



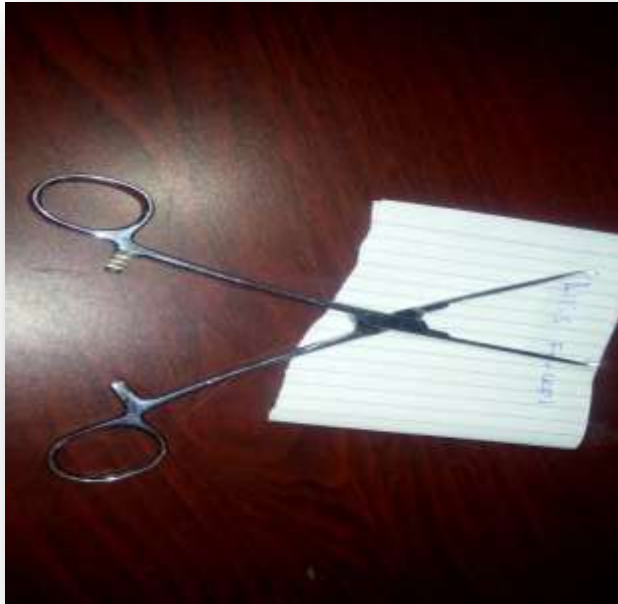
**Non-toothed pick up/tissue
forceps**



Stich scissor /straight



Tissue scissor /Curved



Alli's forceps



Babcock

Gally pot



Kidney dish



Towel clip

Curette





Kocher



Intestinal/smooth clamp



Maggille's forceps

GB/Gall bladder forceps



Dissector



Mastoid retractor

Cervical thongs traction



Lungenbek retractor



Abdominal wall retractor



Deaver retractor



Doines retractor

Bladder retractor



Abdominal spatula



Jiggle saw holder





Sigmoidoscope



Rectal tube

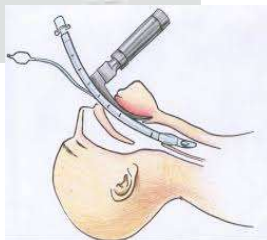




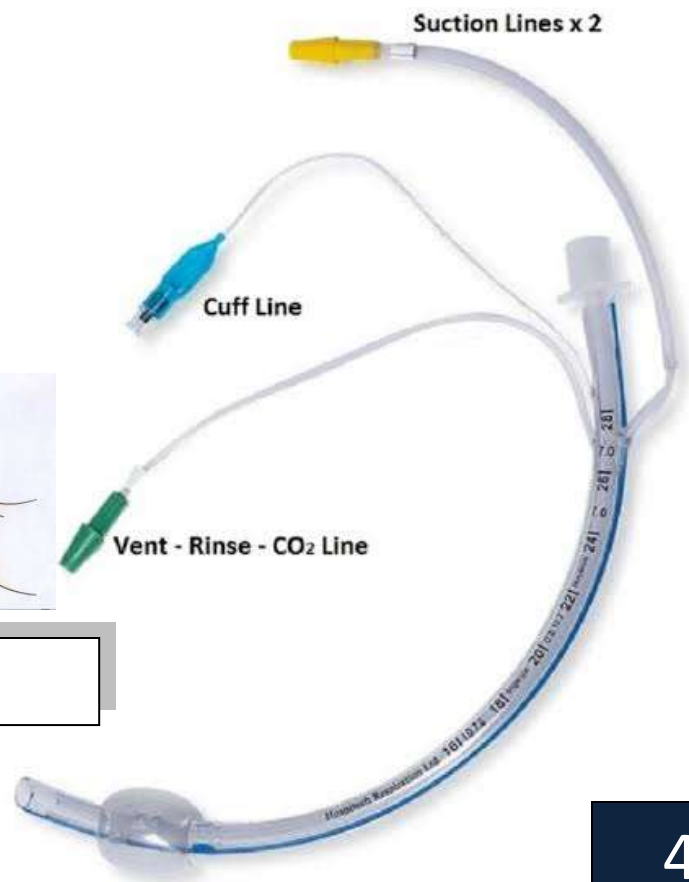
Oropharyngeal airway



Laryngoscope



Endotracheal tube

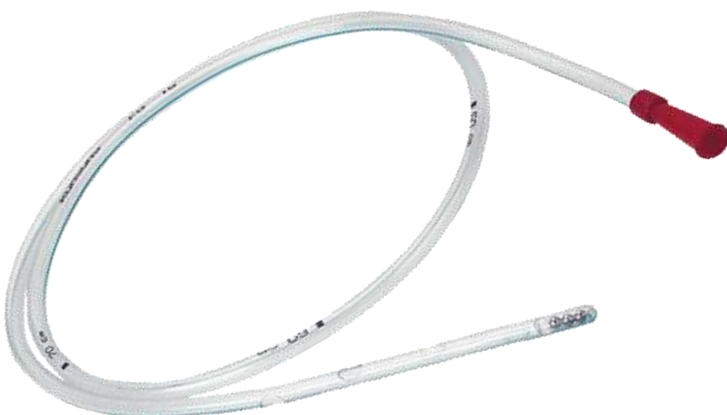




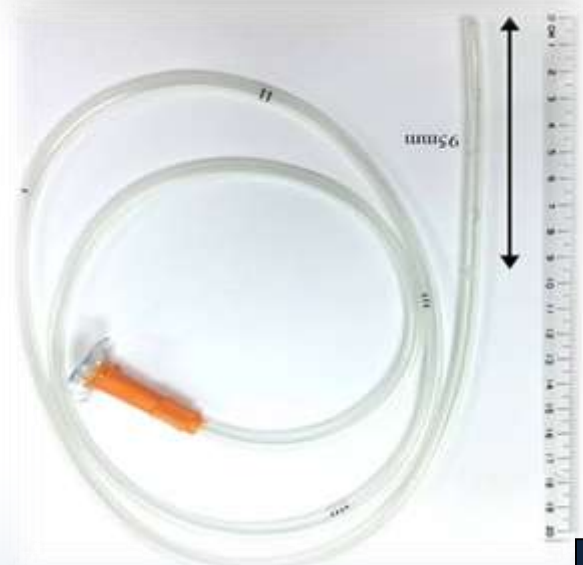
Ambu bag

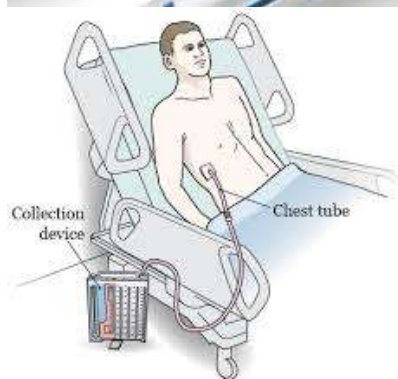
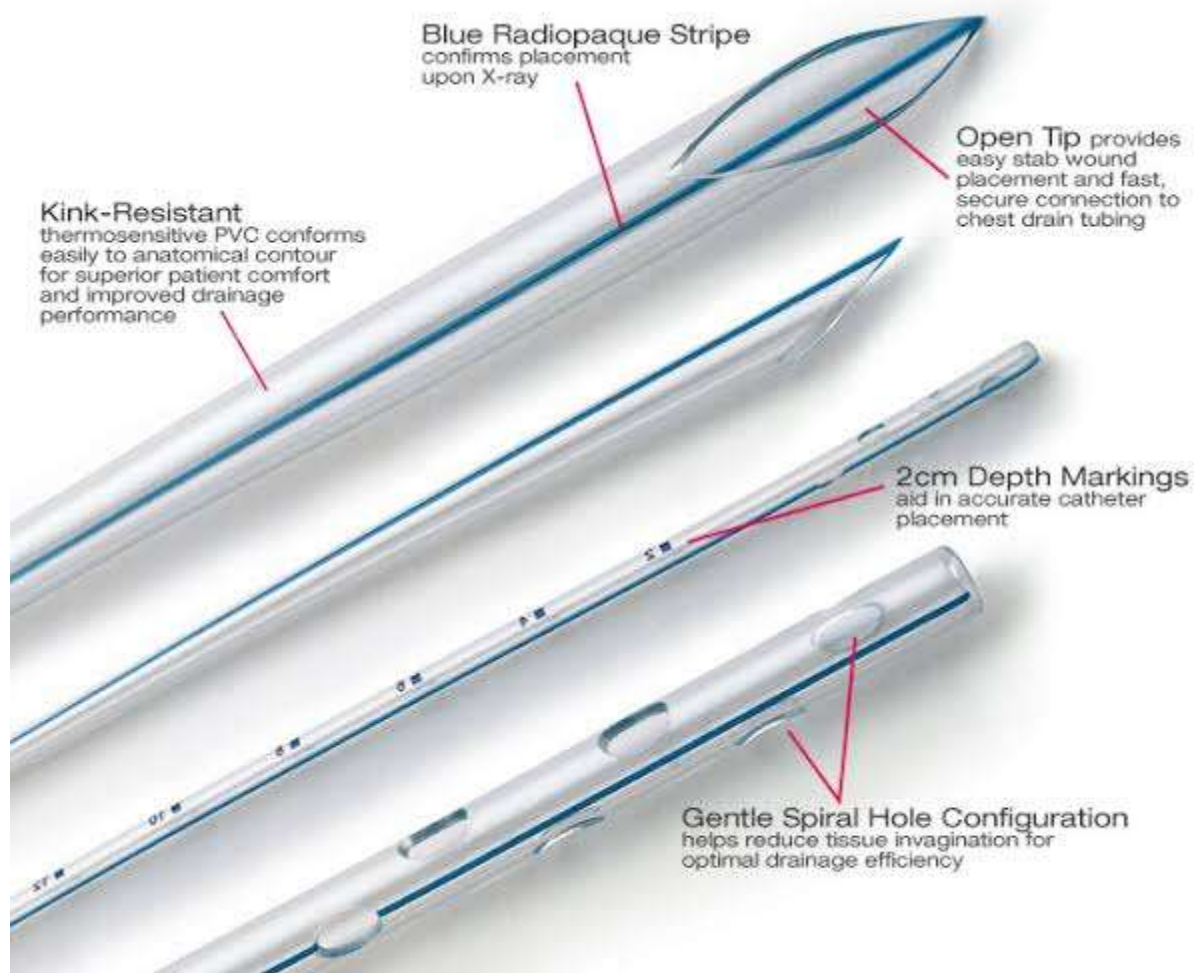


Laryngeal mask airway (LMA)

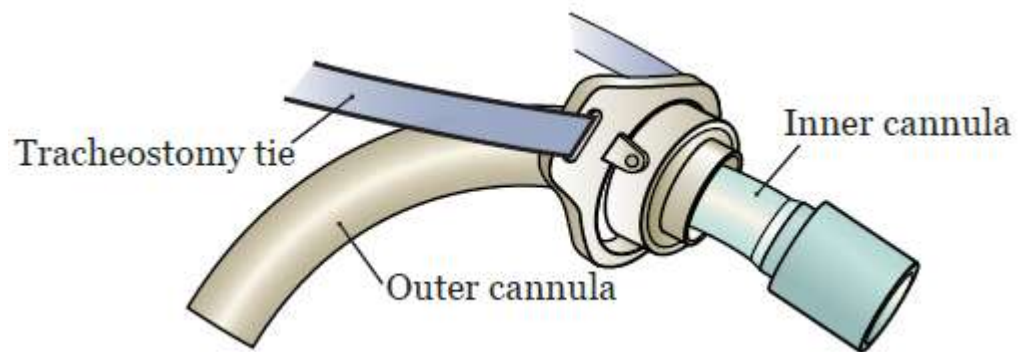


Naso gastric tube

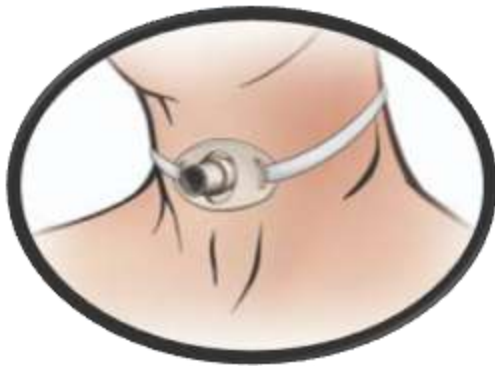




Chest tube/chest drainage catheter



Tracheostomy tube/Plastic

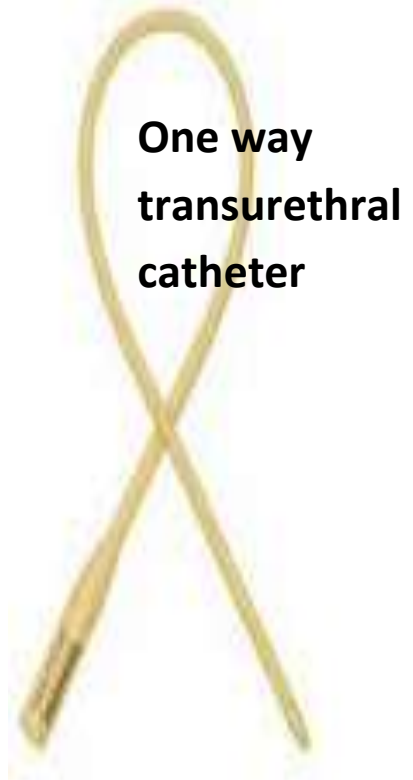


Tracheostomy tube/Metalic

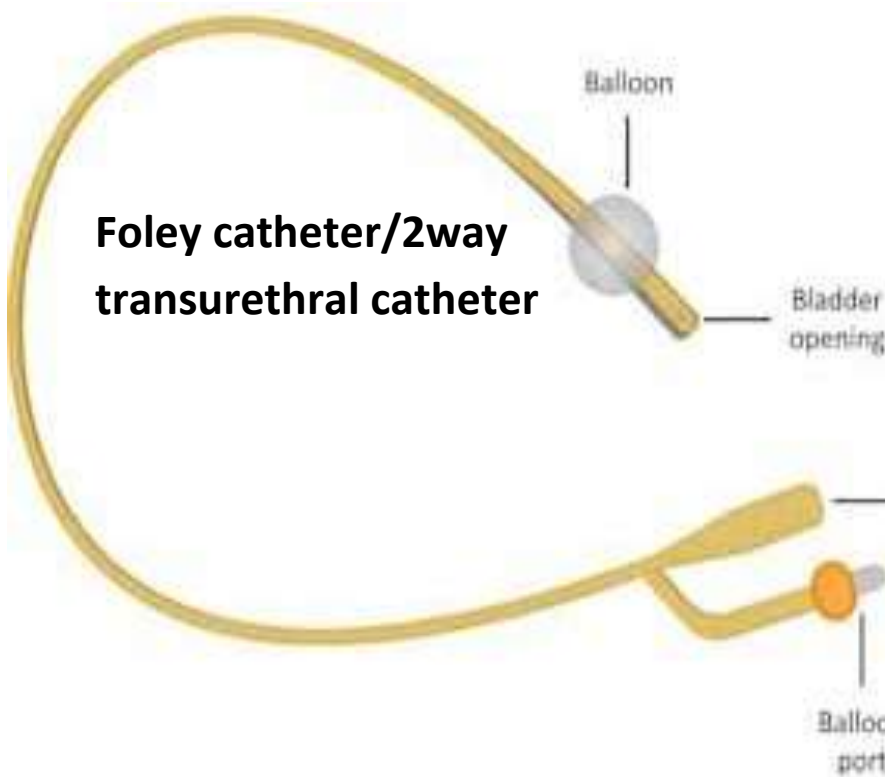
Urethral catheters



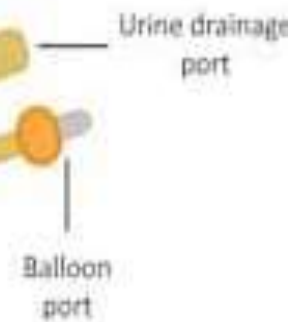
Condom catheter



One way transurethral catheter



Foley catheter/2way transurethral catheter



Three way transurethral catheter /irrigation





Clamps

**Rods of external
fixator**

**Pins of external
fixator**

External fixation

2. Basic radiological images



Foreign body ingestion/upper esophageal sphincter



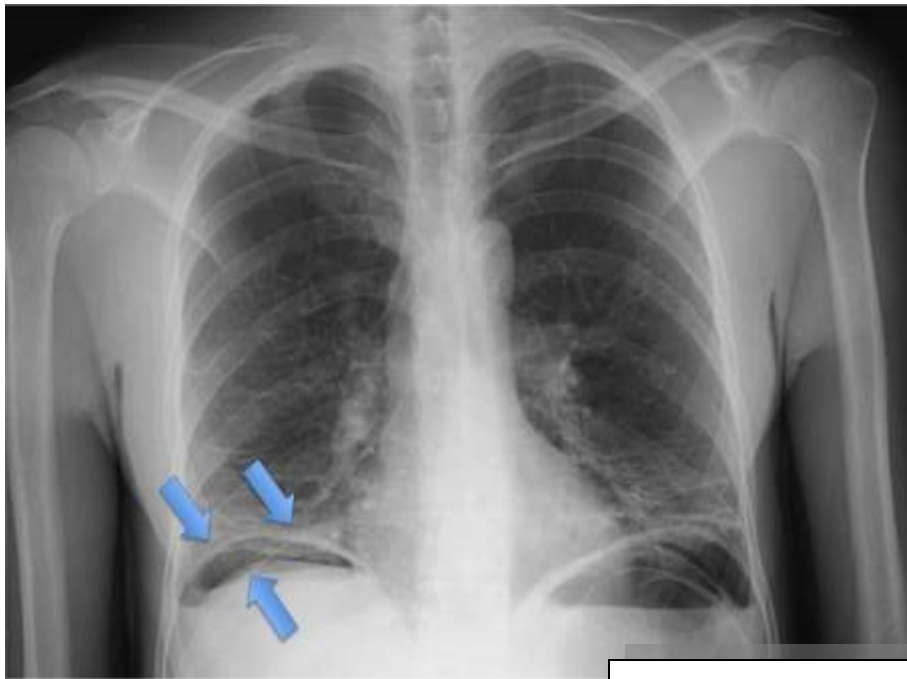
Large bowel obstruction



**Large bowel obstruction 2^o
sigmoid volvulus**



Small bowel obstruction



Perforated PUD



Intussusception



Intramedullary nailing (internal fixation)

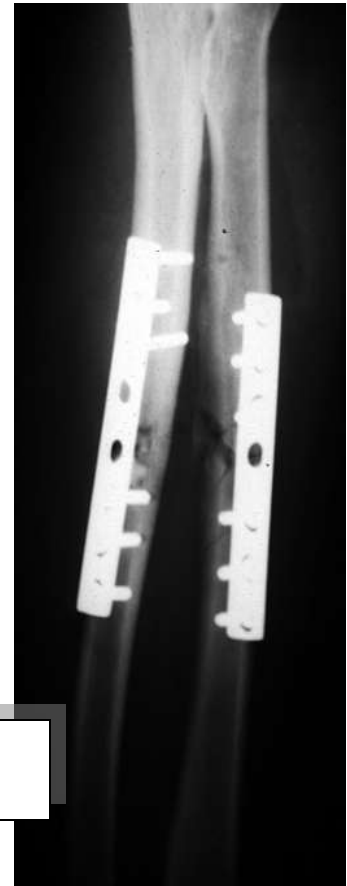
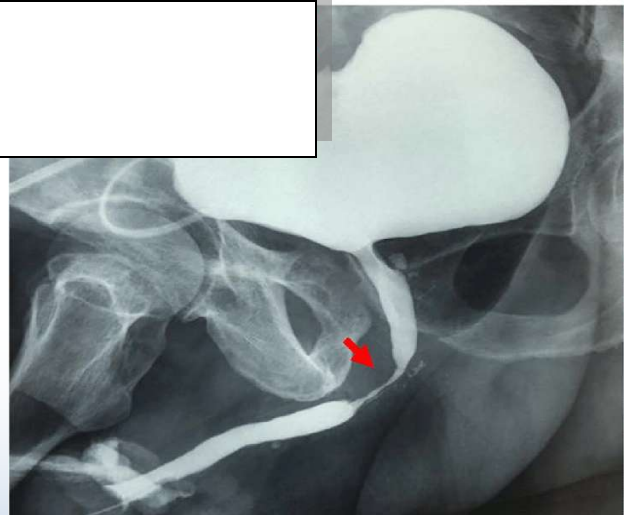


Plate & screw (internal fixation)

Urethral stricture



<https://www.youtube.com/channel/UCZ0IBTvCctutXxL462pe-RQ>

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