UPSC CMS → 2019

SURGERY MCQ MARATHON JULY 24

Medsynapse by Dr. Nikita

Gentleman of 56 years underwent laparoscopic left hemicolectomy for diagnosed left colonic carcinoma. Histopathology revealed the tumour to be invading submucosa and muscularis propria. Among the 16 regional lymph nodes harvested, 2 were positive for malignant deposits. His staging as per AJCC will be:

(a) T1, N1, M0
(b) T2, N1, M0
(c) T1, N0, M0
(d) T2, N1, M1

N1 - upto 3 LN pericolic

N2 - ≥4

Stage	Characteristics
Tumor	
T1	Tumor invades submucosa
T2	Tumor invades muscularis propria
T3	Tumor invades through muscularis propria into subserosa or nonperitonealized pericolic or perirectal tissues
T4	Tumor directly invades other organs or structures and/or perforates visceral peritoneum
Regional nodal metastasis	
NX	Regional lymph nodes cannot be assessed
N0	No nodal metastasis
N1	Metastasis in one to three pericolic or perirectal nodes
N2	Metastasis in four to more pericolic or perirectal nodes
N3	Metastasis in any node along course of a named vascular trunk and/or metastasis to apical node
Distant metastasis	
MX	Presence of distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

What is true about the management of a corrosive injury of oesophagus?

(a) Early skilled endoscopy is must

(b) Immediate surgery with oesophagectomy is advisable

(c) Broad spectrum antibiotics should be started as soon as possible xx no role

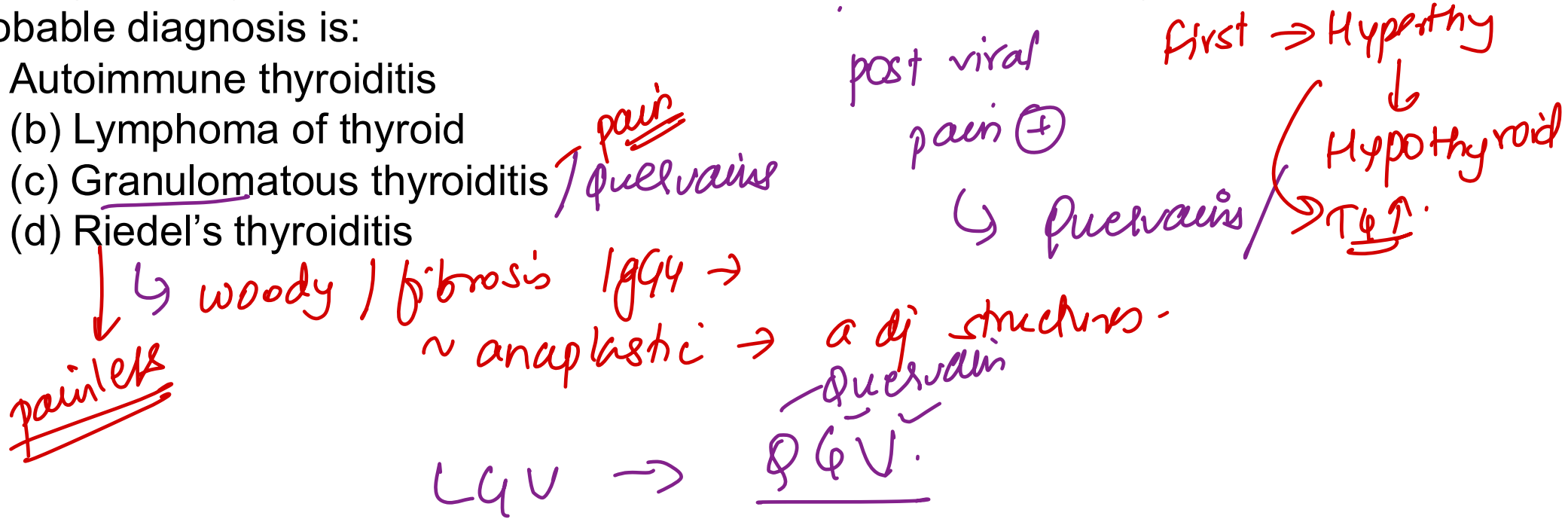
(d) Immediate NG tube insertion and gastric lavage should be preformed ✗

alkali → more injury
↳ liq. necrosis

acid → coag nec except HF
↳ fluid liq.

A few days following viral fever, a 50 year old female presented with pain in neck, fever, malaise and firm enlargement of both the lobes of thyroid. On investigation thyroid antibodies were normal & serum T4 was high normal. Probable diagnosis is:

(a) Autoimmune thyroiditis
(b) (b) Lymphoma of thyroid
(c) (c) Granulomatous thyroiditis
(d) (d) Riedel's thyroiditis



No increased relative risk of invasive breast carcinoma based on histopathological examination of benign breast tissue is for all of the following EXCEPT:

(a) Hyperplasia

(b) Periductal mastitis

(c) Squamous metaplasia

(d) Solitary papilloma of lactiferous sinus

TABLE 53.3 Relative risk of invasive breast carcinoma based on pathological examination of benign breast tissue (American College of Pathologists Consensus Statement).[a]

No increased risk	Adenosis, sclerosing or florid
	Apocrine metaplasia
	Cysts, macro and/or micro
	Duct ectasia
	Fibroadenoma
	Fibrosis
	Hyperplasia
	Mastitis (inflammation)
	Periductal mastitis
	Squamous metaplasia
Slightly increased risk (1.5–2 times)	Hyperplasia, moderate or florid, solid or papillary
	Papilloma with a fibrovascular core
Moderately increased risk (5 times)	Atypical hyperplasia (ductal or lobular)
Insufficient data to assign a risk	Solitary papilloma of lactiferous sinus
	Radial scar lesion

After Page and Dupont (1978) by kind permission of the *Journal of the National Cancer Institute*, USA.

[a] A combination with positive family history significantly increases the risks shown above.

Lesions with malignant potential include all of the following EXCEPT

A. Intraductal papilloma

B. B. Atypical ductal hyperplasia

C. C. Sclerosing adenosis → no risk

D. D. Atypical lobular hyperplasia

What is the correct order of the normal phases of wound healing?

(a) Proliferative phase → Haemostatic phase → Inflammatory phase → Remodelling phase

(b) (b) Haemostatic phase → Inflammatory phase → Proliferative phase → Remodelling phase

(c) (c) Destructive phase → Proliferative phase → Remodelling phase → Inflammatory phase

(d) (d) Remodelling phase → Proliferative phase → Destructive phase → Inflammatory phase

HiDe PR

. destructive

HiDe PR

NORMAL WOUND HEALING

This is variously described as taking place in three or four phases, the most commonly agreed being:

1 the inflammatory phase;
2 the proliferative phase;
3 the remodelling phase (maturing phase).

Occasionally, a haemostatic phase is referred to as occurring before the inflammatory phase, or a destructive phase following inflammation consisting of the cellular cleansing of the wound by macrophages (**Figure 3.1**).

HIDE PR

All of the following are risk factors for an increased risk of wound infection EXCEPT:

(a) Obesity ✓ (malnutrn)
(b) (b) Hypertension
(c) (c) Jaundice (metabolic) → DM / uremia
(d) (d) Cancer ✓ → immunosuppressn

Summary box 5.5

Risk factors for increased risk of wound infection

- Malnutrition (obesity, weight loss)
- Metabolic disease (diabetes, uraemia, jaundice)
- Immunosuppression (cancer, AIDS, steroids, chemotherapy and radiotherapy)
- Colonisation and translocation in the gastrointestinal tract
- Poor perfusion (systemic shock or local ischaemia)
- Foreign body material
- Poor surgical technique (dead space, haematoma)

Systemic Inflammatory Response Syndrome (SIRS) is characterized by all of the following EXCEPT:

(a) Hyperthermia (>38℃)

(b) (b) Platelet count (<1,00,000/mm3)

(c) (c) Tachypnoea (>20/min)

(d) (d) Hypothermia (<36℃)

TPR + WBC infecn

↓

>38

<36

TABLE 5.3 Definitions of systemic inflammatory response syndrome (SIRS) and sepsis.

SIRS is

Two of:

- hyperthermia (>38°C) or hypothermia (<36°C)
- tachycardia (>90/min, no β-blockers) or tachypnoea (>20/min)
- white cell count >12 × 10^9/litre or <4 × 10^9/litre

Sepsis is SIRS with a documented infection

Severe sepsis or sepsis syndrome is sepsis with evidence of failure of one or more organs: respiratory (acute respiratory distress syndrome), cardiovascular (septic shock follows compromise of cardiac function and fall in peripheral vascular resistance), renal (usually acute tubular necrosis), hepatic, blood coagulation systems or central nervous system

- SIRS
- sepsis
- severe sepsis / sepsis syndrome / potential

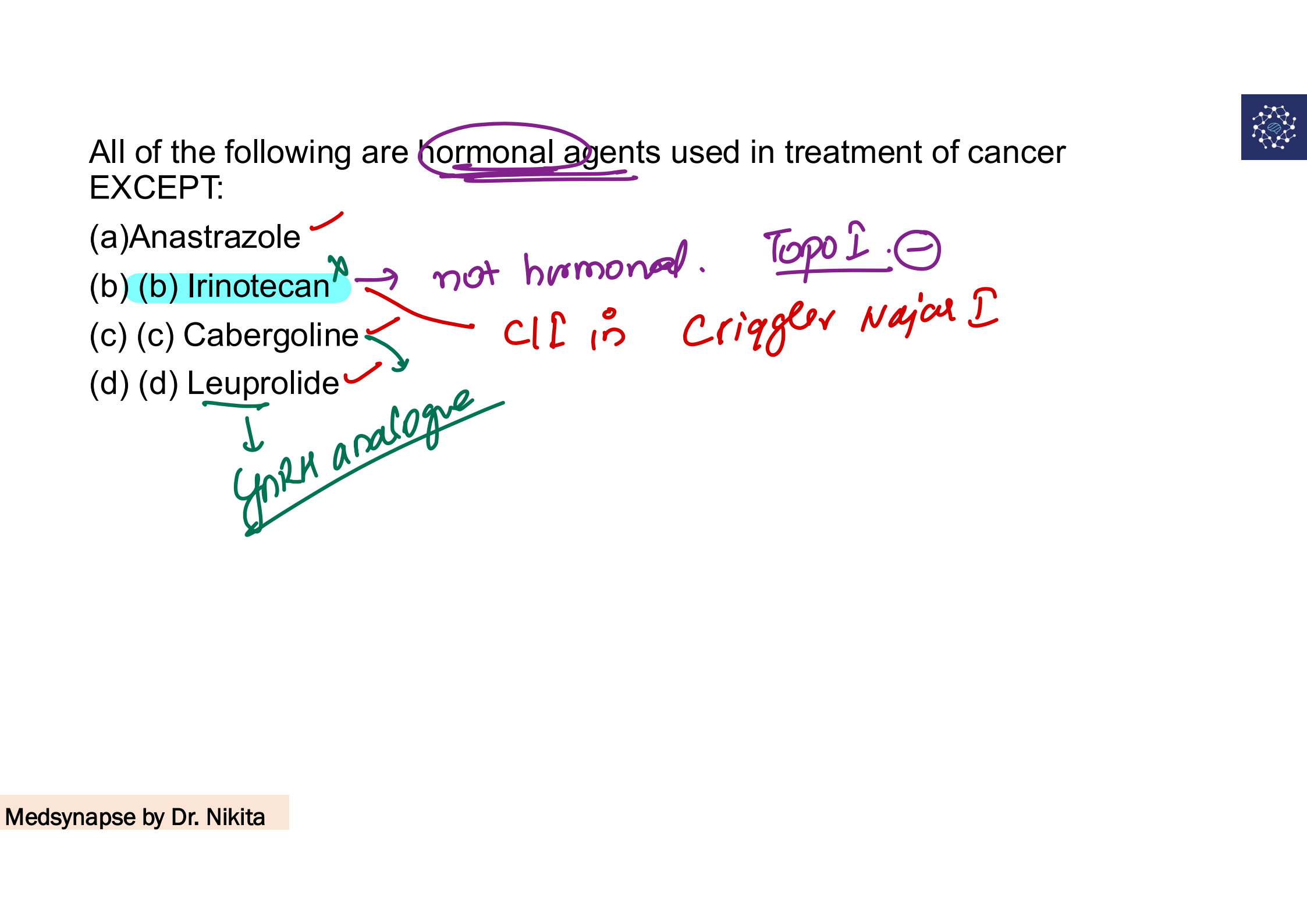
All of the following are hormonal agents used in treatment of cancer EXCEPT:

(a)Anastrazole

(b) (b) Irinotecan

(c) (c) Cabergoline

(d) (d) Leuprolide



Hormones			
	Tamoxifen	Blocks oestrogen receptors	Breast cancer
	Anastrazole	An aromatase inhibitor than blocks post-menopausal (non-ovarian) oestrogen production	Breast cancer
	Exemestane	An aromatase inhibitor than blocks post-menopausal (non-ovarian) oestrogen production	Breast cancer
	Letrozole	An aromatase inhibitor than blocks post-menopausal (non-ovarian) oestrogen production	Breast cancer
	Leuprolide	Analogue of gonadotrophin-releasing hormone, continued use produces downregulation of the anterior pituitary with consequent fall in testosterone levels	Prostate cancer
	Goserelin	Analogue of gonadotrophin-releasing hormone, continued use produces downregulation of the anterior pituitary with consequent fall in testosterone levels	Prostate cancer
	Buserelin	Analogue of gonadotrophin-releasing hormone, continued use produces downregulation of the anterior pituitary with consequent fall in testosterone levels	Prostate cancer
	Cabergoline	Blocks prolactin release, a long-acting dopamine agonist	Prolactin-secreting pituitary tumours
	Bromocriptine	Dopamine agonist, blocks stimulation of anterior pituitary	Pituitary tumours
	Cyproterone acetate	Blocks the effect of androgens	Prostate cancer
	Flutamide	Blocks the effect of androgens	Prostate cancer
	Nilutamide	Blocks the effect of androgens	Prostate cancer
	Bicalutamide	Blocks the effect of androgens	Prostate cancer

Which of the following statements regarding lymphoedema are correct?

1. Patients experience constant dull ache and even severe pain sometimes
2. Manual lymphatic drainage has a role
3. Primary lymphoedema is caused by congenital lymphatic dysplasia
4. Noone Milroy's disease is a type of primary lymphoedema → congenital

Select the correct answer using the code given below:

(a) 3 and 4 only

(b) 1 and 2 only

(c) 1, 2 and 3 only

(d) 1,2,3and4

TABLE 58.1 Aetiological classification of lymphoedema.

Primary lymphoedema	Congenital (onset <2 years old): sporadic; familial (Nonne–Milroy's disease)
	Praecox (onset 2–35 years old): sporadic; familial (Letessier–Meige's disease)
	Tarda (onset after 35 years old)
Secondary lymphoedema	Parasitic infection (filariasis)
	Fungal infection (tinea pedis)
	Exposure to foreign body material (silica particles)
	Primary lymphatic malignancy
	Metastatic spread to lymph nodes
	Radiotherapy to lymph nodes
	Surgical excision of lymph nodes
	Trauma (particularly degloving injuries)
	Superficial thrombophlebitis
	Deep venous thrombosis

1 → pitting
2 → non pitting
3 → sCin

lymphed
Turner
Brunner

TABLE 58.2 Clinical classification of lymphoedema.

Grade (Brunner)	Clinical features
Subclinical (latent)	There is excess interstitial fluid and histological abnormalities in lymphatics and lymph nodes, but no clinically apparent lymphoedema
I	Oedema pits on pressure and swelling largely or completely disappears on elevation and bed rest
II	Oedema does not pit and does not significantly reduce upon elevation, positive Stemmer's sign
III	Oedema is associated with irreversible skin changes, i.e. fibrosis, papillae

Primary Lymphedema :

Lymphedema congenita

0-2 yrs M >F

Can involve multiple limbs, face & genitalia Familial : Noone Milroy syndrome (FLT-4 gene)

Lymphedema praecox (M/c)

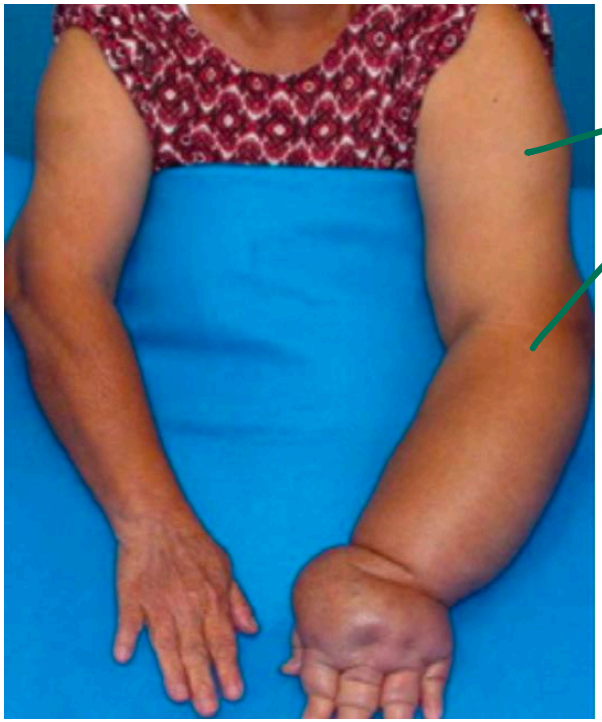
2-35 yrs F > M

Usually U/L till kee

Familial : Meig's disease (GJC gene)

Lymphedema tarda

After 35 yrs



post mastectomy

↓

Lymphangiosarcoma

Stewart-Treves

FAB

Surgical options for lymphoedema

- Bypass procedures
  - Lymphatic bypass, e.g. omental pedicle, the skin bridge (Gillies) and the ilieal mucosal patch (Kinmonth)
  - Lymphaticovenular anastomosis (LVA)
  - Lymph nodes to vein anastomosis (Kinmonth)
- Liposuction
- Limb reduction procedures
  - Sistrunk
  - Homans
  - Thompson
  - Charles

C TSH → lymphedema

MLD-nle.

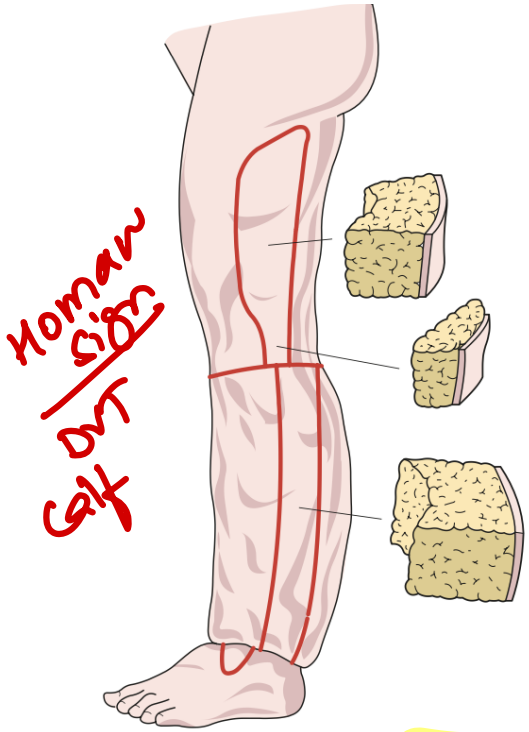


Figure 58.11 Homans' procedure involves raising skin flaps to allow the excision of a wedge of skin and a larger volume of subcutaneous tissue down to the deep fascia. Surgery to the medial and lateral

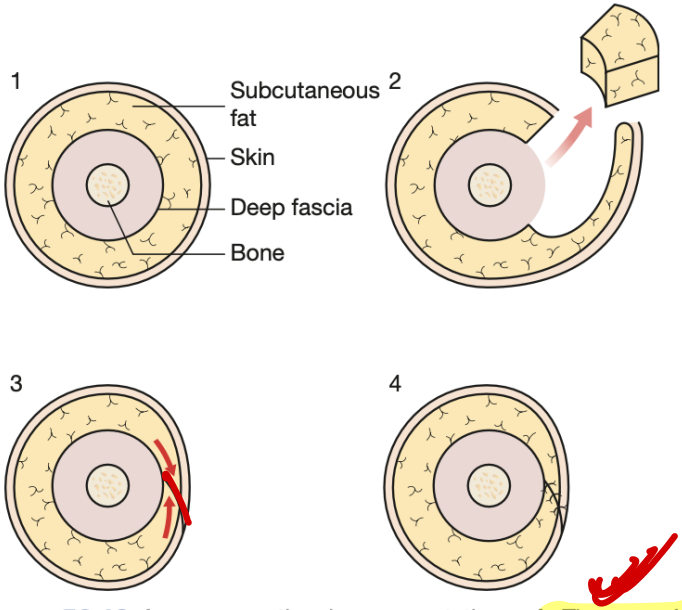


Figure 58.12 A cross-sectional representation of Thompson's reduction operation; red arrows illustrate the buried dermal flap sutured to deep fascia.

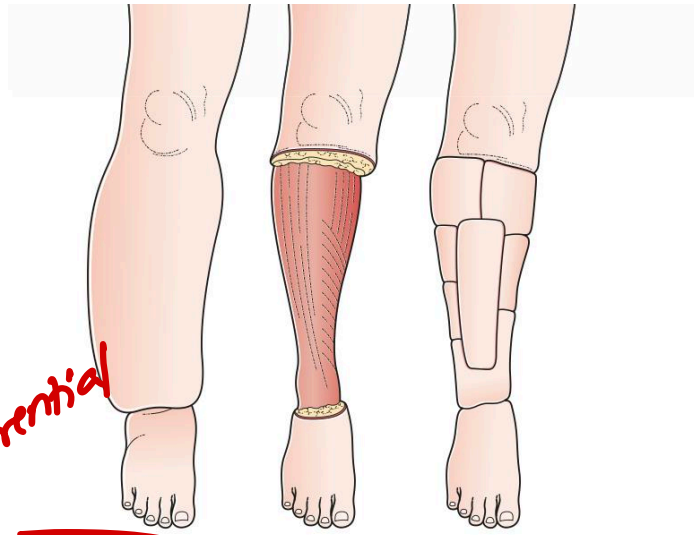


Figure 58.13 The Charles procedure involves circumferential excision of lymphoedematous tissue down to, and including, the deep fascia followed by split-skin grafting. This procedure gives a very poor cosmetic result but does allow the surgeon to remove very large amounts of tissue and is particularly useful in patients with severe skin changes.

Which of the following statements regarding Paget's disease of nipple are correct?

1. It represents benign pathology of nipple areola complex
2. It is eczema like condition of nipple and areola
3. Erosion of nipple is seen
4. Nipple biopsy is required for definitive diagnosis

Select the correct answer using the code given below:

(a) 1,2and3

(b) 2,3and4

(c) 1,3and4

(d) 2 and 4 only

* BIL breast Ca → lobular
* leaf → phyllodes

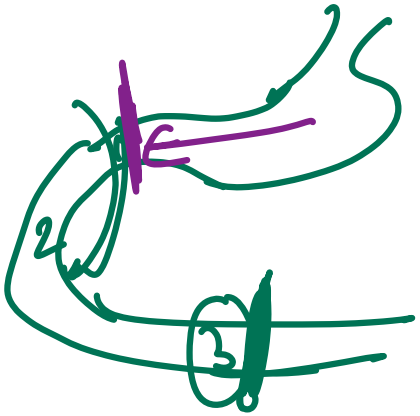
A gentleman of 36 years presented with a long history of upper abdominal pain which was periodic and often occurred early morning. For last 3 months, he is having projectile vomiting, which is non bilious, unpleasant in nature with undigested food materials. On examination he appears unwell, dehydrated and seemed to have lost weight. Probably he is suffering from:

(a) Gastric outlet obstruction

(b) Carcinoma stomach

(c) Gastro-oesophageal reflux with oesophagitis

(d) Superior mesenteric artery syndrome

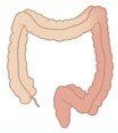
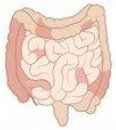
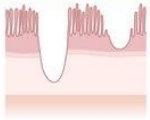
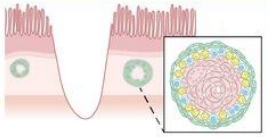
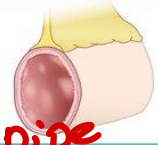
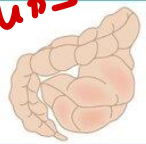
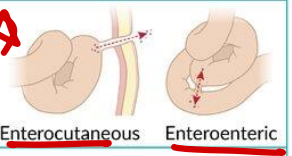
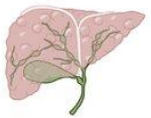
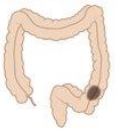


A 40 year old female patient presents with colicky abdominal pain associated with episodes of mild diarrhoea for last 6 months accompanied with intermittent fever and weight loss. There are multiple discharging sinuses on perineal examination. The most likely clinical diagnosis in this patient is:

(a) Amoebic colitis
(b) Crohn disease
(c) Ulcerative colitis
(d) Ileocaecal Tuberculosis

TABLE 75.1 Distinguishing ulcerative colitis (UC) and Crohn's disease (CD).

	UC	CD
Macroscopic		
Distribution	Colon/rectum	Anywhere in the gastrointestinal tract
Rectum	Always involved	Often spared
Perianal disease	Rare	Common
Fistula formation	Rare	Common
Stricture	Rare	Common
Microscopic		
Layers involved	Mucosa/submucosa	Full thickness
Granulomas	No	Common
Fissuring	No	Common
Crypt abscesses	Common	Rare

	Ulcerative colitis	Crohn disease
Location	• Continuous inflammation 	• Skip lesions 
Histology	• Crypt Abscess • Inflammation confined to submucosa 	• Transmural inflammation • Non caseating granulomas 
Colonoscopy	• Friable mucosa • Deep ulcerations • Loss of haustra 	• Creeping fat • Thickened cobblestone mucosa 
Stool	• Bloody diarrhea 	• Watery diarrhea (can be bloody) 
Complications	• Toxic megacolon 	• Fistulas 
Associated conditions	• Primary sclerosing cholangitis 	• Nephrolithiasis 
	• Colorectal cancer 	

SCIP

o smoking ↓ UC

↓

PSC

P-anca ⊕

Uncle → UC

crohns

asca ⊕

Q TH2 →

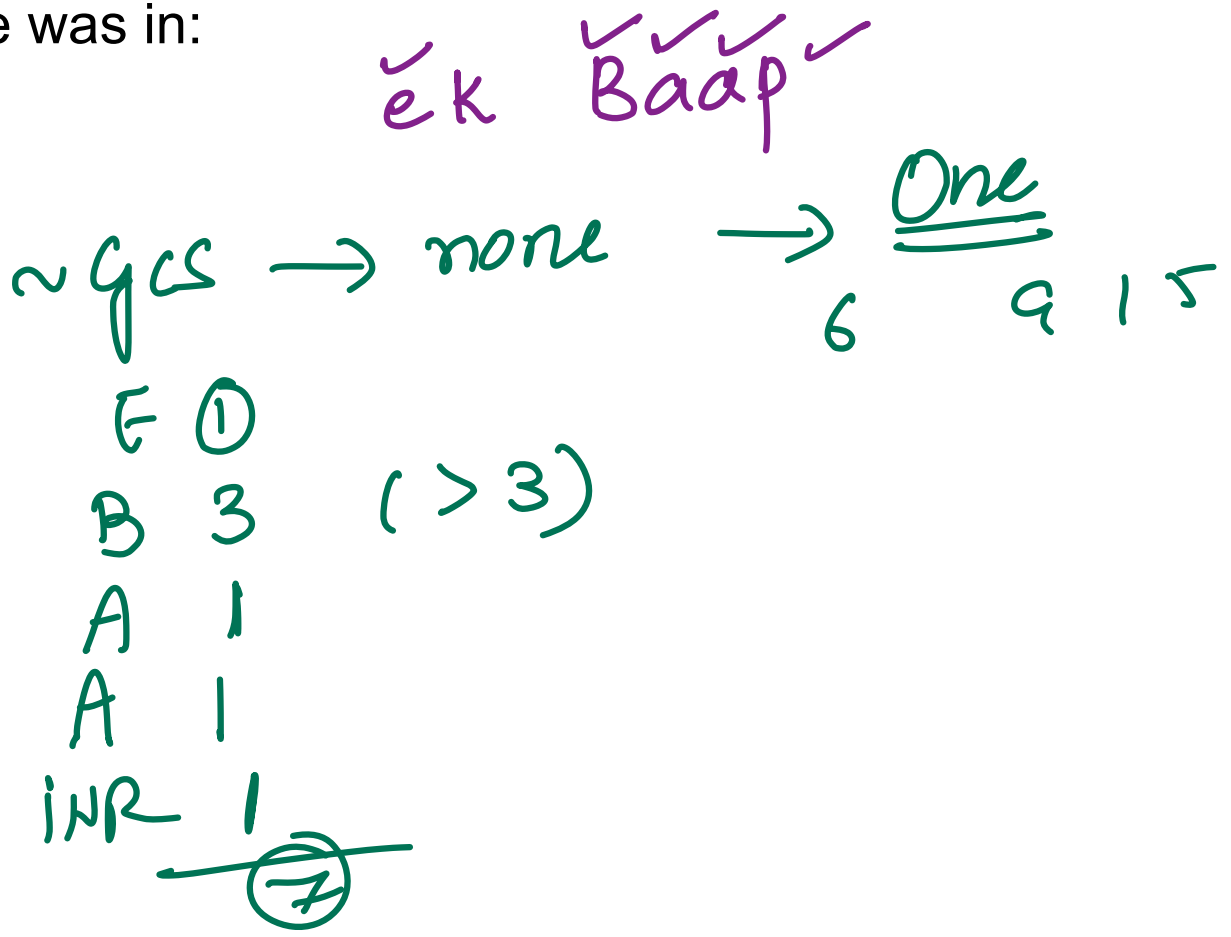
A gentleman of 48 years was being worked up for hepatocellular function. He had no history or signs of encephalopathy. His serum bilirubin was 5 mg%, serum albumin was 3.9 gm%, International normalized ratio was 1.6. On ultrasound no free fluid was detected inside abdomen. As per Child-Turcotte-Pugh (CTP) classification, he was in:

(a) CTP–A

(b) (b) CTP–B

(c) (c) CTP–C

(d) (d) CTP–D



Child-Turcotte-Pugh Classification for Severity of Cirrhosis

Clinical and Lab Criteria	Points*		
	1	2	3
Encephalopathy	None	Grade 1 or 2	Grade 3 or 4
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time			
Seconds prolonged	<4	4-6	>6
or			
International normalized ratio	<1.7	1.7-2.3	>2.3

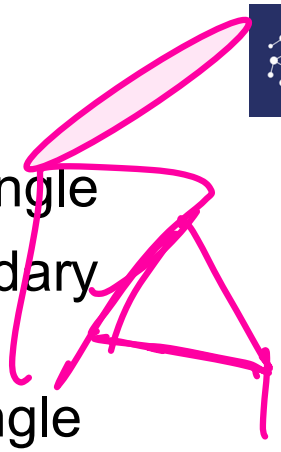
*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)

- **Class A** = 5 to 6 points
- **Class B** = 7 to 9 points
- **Class C** = 10 to 15 points

A 6 B 9 C 15

Which of the statements regarding Calot's triangle are correct?

1. Common hepatic duct forms the medial boundary of the Calot's triangle
2. Inferior surface of the right lobe of the liver forms the superior boundary of Calot's triangle
3. Right hepatic artery is usually found as a content of the Calot's triangle
4. Cystic duct and medial border of gall bladder forms the lateral border of Calot's triangle



Select the correct answer using the code given below:

(a) 1,2and3

(b) 2,3and4

(c) 1,3and4

(d) 1,2and4

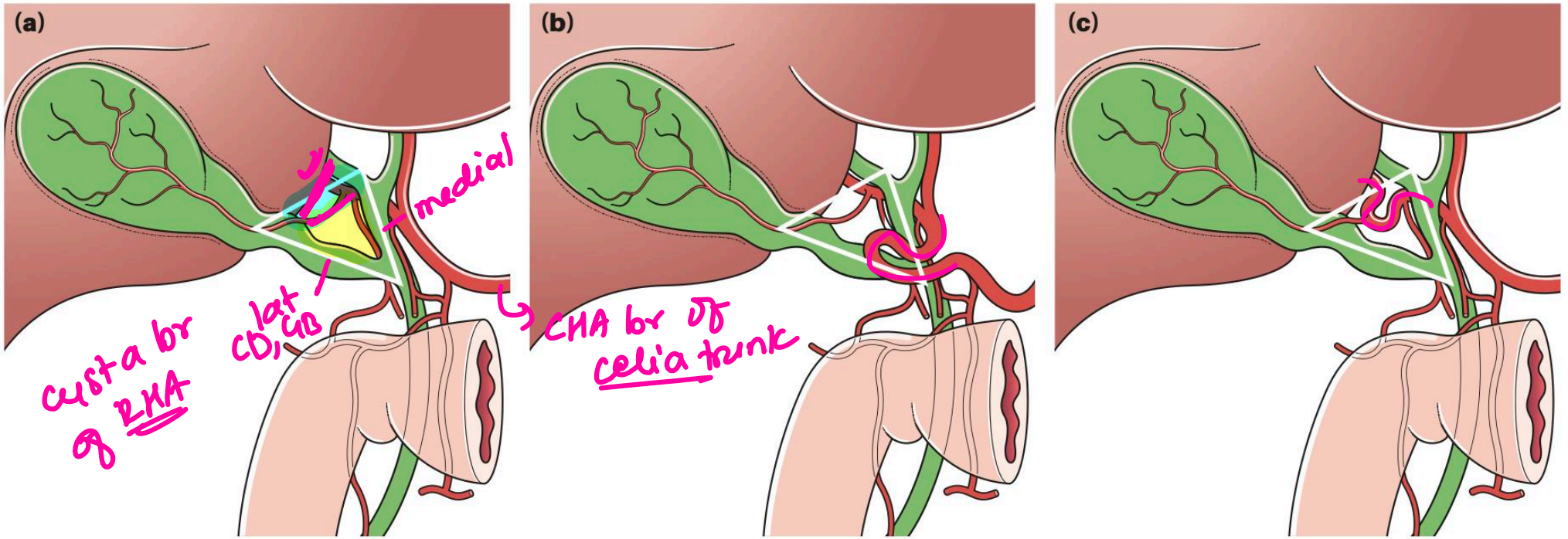


Figure 67.2 **(a)** Usual anatomy of Calot's triangle; **(b)** tortuous common hepatic artery; **(c)** tortuous right hepatic artery with short cystic artery; **(b)** and **(c)** are examples of so-called 'caterpillar turn' or 'Moynihan's hump', which if not recognised can lead to inadvertent arterial injury or bleeding during cholecystectomy.

Calot's triangle, or the hepatobiliary triangle, was initially described by Calot as the space bordered by the cystic duct inferiorly, the common hepatic duct medially and the superior border of the cystic artery. This has been modified in contemporary literature to be the area bound superiorly by the inferior surface of the liver, laterally by the cystic duct and the medial border of the gallbladder and medially by the common hepatic duct. It is an important surgical landmark as the cystic artery usually can be found within its boundaries and should be identified by surgeons performing a cholecystectomy to avoid damage to the extrahepatic biliary system

Consider the following statements regarding Opportunistic post-splenectomy infections (OPSI):

1. Haemophilus influenzae, Neisseria meningitidis and Streptococcus pneumonae are the most common causative agents
2. Risk is greatest in the patients who have undergone splenectomy for trauma
3. Risk is greatest within the first 2–3 years following splenectomy
4. Prophylactic vaccination should be done 2 weeks prior to elective splenectomy

Which of the statements given above are correct?

(a) 1,2and3

(b) 2,3and4

(c) 1,3and4

(d) 1,2and4

OPSI (Opportunistic Post splenectomy infections) :

MC organism : Encapsulated bacteria (Pneumococcus (MC), Meningiococcus, H. Influenzae).

More common in children > adults.

More common within 1st 2yrs of splenectomy.

High mortality.

More likely in splenectomy done for hematological conditions > trauma.

Prevention by vaccines :

- Elective Sx : 2 weeks before.
- Emergency Sx : Post op day 2.

Ventral hernia includes all EXCEPT:

(a) Epigastric hernia

(b) (b) Para-umbilical hernia

(c) (c) Lumbar hernia

(d) (d) Inguinal hernia

except ① inguinal ② femoral.

VENTRAL HERNIA

This term refers to hernias of the anterior abdominal wall. Inguinal and femoral hernias are not included, however lumbar hernia is included despite being dorsolateral. The European Hernia Society classification (2009) distinguished primary ventral from incisional hernia but did not include parastomal hernia, which is included in this section.

Summary box 64.13

Primary ventral hernias

- Umbilical
- Epigastric
- Spigelian
- Lumbar
- Traumatic

'Secondary' ventral hernias

- Incisional
- Parastomal

E H S

Incisional Hernia Classification

Midline	subxiphoidal	M1	
	epigastric	M2	
	umbilical	M3	
	infraumbilical	M4	
	suprapubic	M5	
Lateral	subcostal	L1	
	flank	L2	
	iliac	L3	
	lumbar	L4	
Recurrent incisional hernia?		Yes O	No O
length: cm		**width**: cm	

A young sports person presented in surgical emergency with the complaints of severe pain in the groin area, extending into the scrotum and upper thigh. The pain is debilitating and he is not able to exercise. On examination there is tenderness in the region of Inguinal canal and pubic tubercle. He is probably suffering from:

(a) Varicocele

(b) (b) Inguinal hernia

(c) (c) Sportsman hernia / Gilmore's / groin pain - tear of post wall muscle.

(d) (d) Femoral hernia

Diaphragmatic injury is suspected in a 50 year gentleman with history of blunt abdominal trauma, having a normal chest X-ray. He is best managed by:

(a) Diagnostic peritoneal lavage and proceed

(b) (b) Upper GI contrast study

(c) (c) CECT abdomen

(d) (d) Diagnostic laparoscopy

INI → NO blind insertn of chest tube

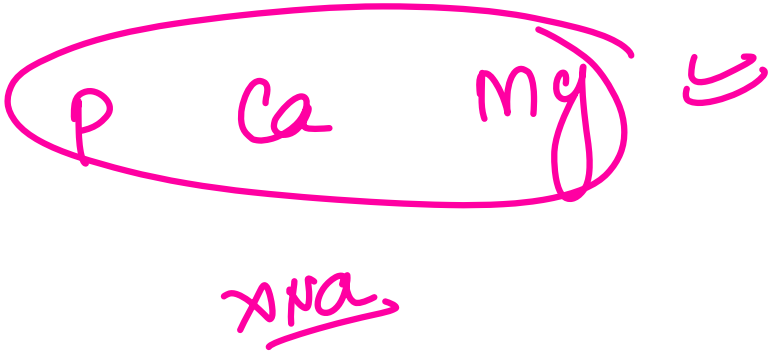
Refeeding syndrome seen after enteral or parenteral nutrition is characterized by all EXCEPT:

(a) Hypophosphatemia

(b) (b) Hypocalcemia

(c) (c) Hypomagnesemia

(d) (d) Hyponatremia



Summary box 25.5

Refeeding syndrome

Patient is considered to be at risk of developing refeeding syndrome with
EITHER

One or more of the following:

- BMI <16 kg/m^2
- Unintentional weight loss >15% within the last 3–6 months
- Little or no nutritional intake for more than 10 days
- Low potassium, phosphate or magnesium levels prior to feeding

OR

Two of more of the following:

- BMI <18.5 kg/m^2
- Unintentional weight loss >10% within the last 3–6 months
- Little or no nutritional intake for more than 5 days
- History of alcohol abuse or on medication, including insulin, chemotherapy, antacids or diuretics

The capillary refill time is prolonged in all types of shock EXCEPT:

(a) Hypovolaemic shock ⟶ cold clammy → symp. activn

(b) (b) Cardiogenic shock ↓CO

(c) (c) Septic shock — vasodil → warm

(d) (d) Obstructive shock ⟶ tamponade / PTx ↓CO.

opp Hypvol → warm dry → Neurogenic

↓

B HR↓ BP↓

Capillary refill

Most patients in hypovolaemic shock will have cool, pale peripheries, with prolonged capillary refill times. However, the actual capillary refill time varies so much in adults that it is not a specifc marker of whether a patient is shocked, and patients with short capillary refill times may be in the early stages of shock. In distributive (septic) shock, the peripheries will be warm and capillary refill will be brisk, despite profound shock.

TABLE 2.1 Cardiovascular and metabolic characteristics of shock.

	Hypovolaemic	Cardiogenic	Obstructive	Distributive
Cardiac output	Low	Low	Low	High
Systemic vascular resistance	High	High	High	Low
Venous pressure	Low	High	High	Low
Mixed venous saturation	Low	Low	Low	High
Base deficit	High	High	High	High

★ High → MVO2 → Septic / distributive
High → CO (vasodil")

High → JVP → cardiog / obstructive

Penile cancer :

Squamous cell carcinoma.

Premalignant conditions :

- Bowens disease of the shaft.
- Erythroplasia of Queyart (Glans).
- Balanitis xerotica obliterans.
- Genital warts : HPV.
- Leukoplakia.

C/f :

- Growth over penis.
- Inguinal lymph nodes enlarged : 50 % cases may be due to infection.

M/c cause of death : Erosion of femoral/iliac vessel due to involved lymph nodes.
Dx : Bx of the lesion.

Staging : Jacksons Staging

T1: Skin involved
T2: Corpora Involved
T3 :Urethra involved
T4: Adjacent structures involved

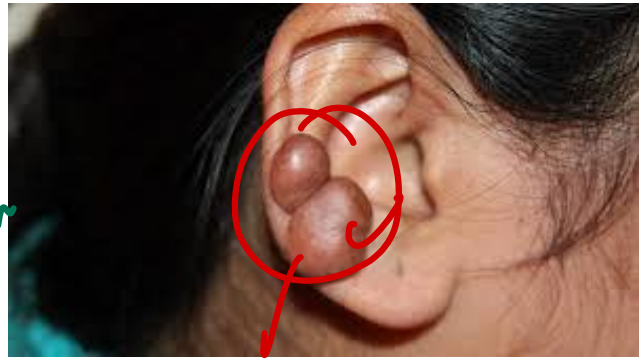
Management :
In situ carcinoma Topical 5 - Fluorouracil

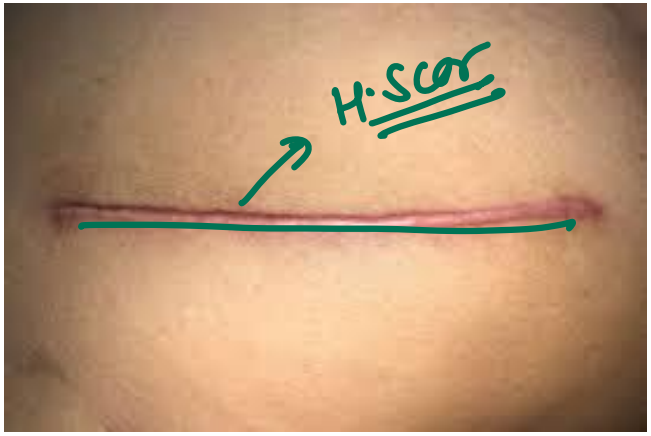
Distally placed - Distal/ Partial Penectomy : Margin- 0.5cm
Proximally placed - Total amputation of the penis & Urethral opening in perineum

Sentinel Lymph node biopsy

Characteristic	Keloids	Hypertrophic Scars
Trauma	Non-severe (acne, folliculitis)	Burns, incision
Body sites	Chest, upper back, earlobe	Any
Symptoms	Erythema, itch, pain	Erythema, itch
Exploration	Beyond the limit of the trauma	Limited to the initial wound
Treatment	Combined therapies with frequent recurrence	Good response
Surgical excision	Contraindicated due to recurrence	Without recurrences, could be considered a treatment

Hypertrophic scars	Keloid scars
Raised, linear, firm lesion	Raised, hard dermal outgrowth
Develop within 3 months of injury	Develop up to or even beyond 1 year postinjury
Remain within the boundaries of the original wound site	Spread outside wound margins, invading normal skin
Common postburn injury	Any form of dermal injury
Increased collagen type III	High in collagen type I
Not typically associated with pigmented skin	Associated with certain ethnicities (higher in darkly pigmented skin)
Not commonly familial	Strong familial tendency
Affect any age	Common in young age
Can regress spontaneously	Do not regress spontaneously





Aberrations of normal wound healing such as prolonged infammation can result in excessive scar tissue, for example hypertrophic and keloid scars. These abnormal scars contain excess collagen, which is arranged in a disorganised pattern in keloid scars as opposed to a parallel pattern in hypertrophic scars.

Hypertrophic scars do not extend beyond the boundary of the original incision or wound and eventually regress. They are more common in areas of increased tension, wounds crossing tension lines, deep dermal burns and wounds left to heal by secondary intention (longer than 3 weeks).

Keloid scars extend beyond the boundaries of the original incision or wound , do not spontaneously regress and are difcult to treat. The aetiology is unknown but genetic predisposition is implicated. They often occur as a result of relatively minor tra