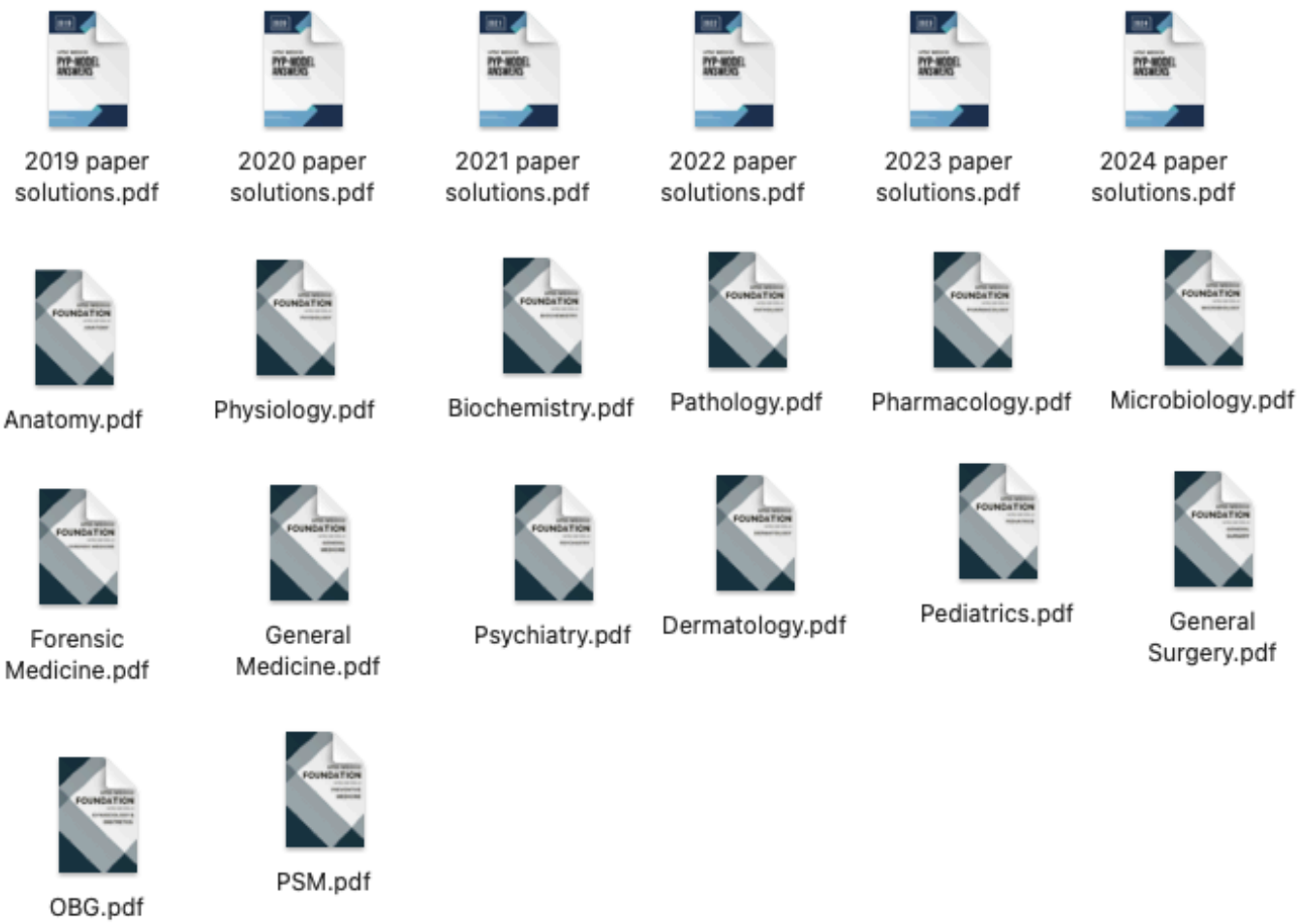
UPSC MEDICO
FOUNDATION

NOTES EDITION 1.0

SAMPLE NOTES

HIGHLIGHTS

- 100% syllabus coverage
- Perfect indexing (Page no)
- Colored images/Tables and flowcharts: Treatment algorithms
- PYQ details
- Subject wise notes
- Previous Year Paper Model Answers (2019-2024)
- In accordance with the videos (SYNCHRONIZED)
- UP TO DATE NOTES (reference from standard text books: latest edition)
- UPDATED National health programs (latest from MoHFW)
- Printable A4 size format
- Total 20 pdfs (14 subject wise pdfs (Total 1747 pages) and 6 previous year paper model answers: 2019-2024 (Total 901 pages)



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ANATOMY

SAMPLE NOTES

SYLLABUS (page no.)

- Applied anatomy including blood and nerve supply of upper and lower limbs (3, 12)
- Joints of shoulder, hip and knee. (20, 26, 32)
- **Gross anatomy, blood supply and lymphatic drainage of**
    - Tongue (42)
    - Thyroid (46)
    - Mammary gland (50)
    - Stomach (53)
    - Liver (57)
    - Prostate (63)
    - Gonads (65)
    - Uterus (68)
- **Applied anatomy of**
    - Diaphragm (76)
    - Perineum (80)
    - Inguinal region (80)
- **Clinical anatomy of**
    - Kidney (89)
    - Urinary bladder (99)
    - Uterine tubes (104)
    - Vas deferens (106)
- **Embryology:**
    - Placenta and placental barrier. (107)
    - Development of heart (117) , gut (128), kidney, uterus, ovary, testis and their common congenital abnormalities.(129)
- **Central and Peripheral Autonomic Nervous System:**
    - Gross and clinical anatomy of ventricles of brain, circulation of cerebrospinal fluid (131)
    - Neural pathways and lesions of cutaneous sensations, hearing and vision (132)
    - Cranial nerves distribution and clinical significance (139)
    - Components of autonomic nervous system. (162)

Applied anatomy including blood and nerve supply of joints of shoulder, hip and knee

PYQs

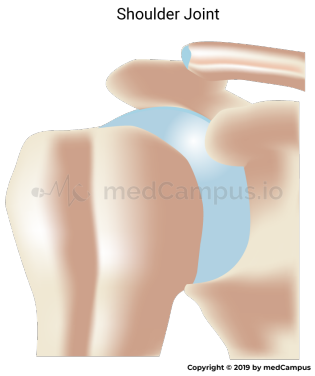
SHOULDER JOINT

- Describe the shoulder joint under the following headings: (2012)
    1. Type of joint (Classification of joint)
    2. Movements occurring at this joint
    3. List main ligaments
    4. Name the muscles acting at this joint (Do not describe each muscle)
    5. Applied anatomy of dislocation of shoulder joint
    6. What is meant by the term "frozen shoulder"?
- Describe the shoulder joint under the following headings (2013)
    1. Type
    2. Movements
    3. Muscles
    4. Applied anatomy
- Describe the shoulder joint under the following headings: (2017)
    1. Movement
    2. Blood Supply
    3. Ligaments
- Describe shoulder joint under the following headings: (2020)
    1. Type and articulating surfaces
    2. Movements and muscles responsible for each
    3. Painful arc syndrome
- Discuss the attachments and applied aspects of the rotator cuff muscles of the shoulder joint. (2022)

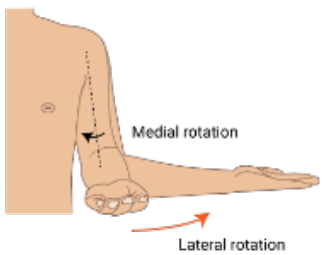
SHOULDER JOINT

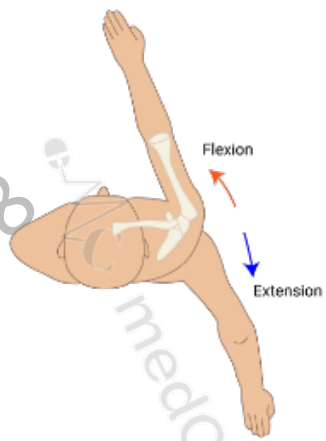
Type of joint

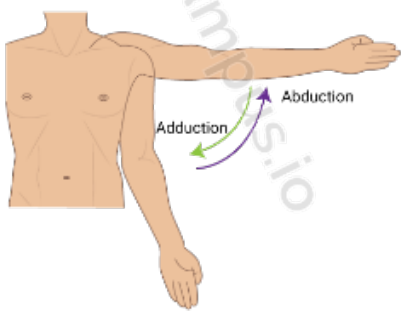
Synovial ball-and-socket joint between the head of the humerus and the glenoid cavity of the scapula

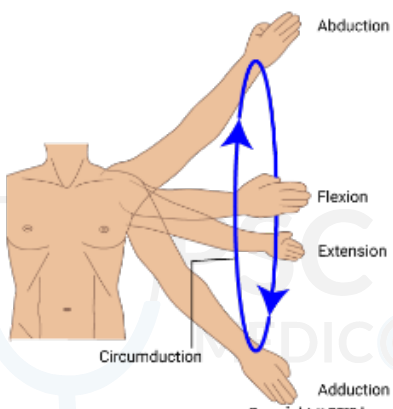


Movements	Main muscles	Accessory muscles
1. Flexion	(i) Clavicular head of the pectoralis major (ii) Anterior fibres of deltoid	(i) Coracobrachialis (ii) Short head of biceps
2. Extension	(i) Posterior fibres of deltoid (ii) Latissimus dorsi	(i) Teres major (ii) Long head of triceps (iii) Sternocostal head of the pectoralis major
3. Adduction	(i) Pectoralis major (ii) Latissimus dorsi (iii) Short head of biceps (iv) Long head of triceps	(i) Teres major (ii) Coracobrachals
4. Abduction	(i) Deltoid (ii) Supraspinatus (iii) Serratus anterior (iv) Upper and lower fibres of trapezius	
5. Medial rotation	(i) Pectoralis major (ii) Anterior fibres of deltoid (iii) Latissimus dorsi (iv) Teres major	(i) Subscapularis
6. Lateral rotation	(i) Posterior fibres of deltoid (ii) Infraspinatus (iii) Teres minor	



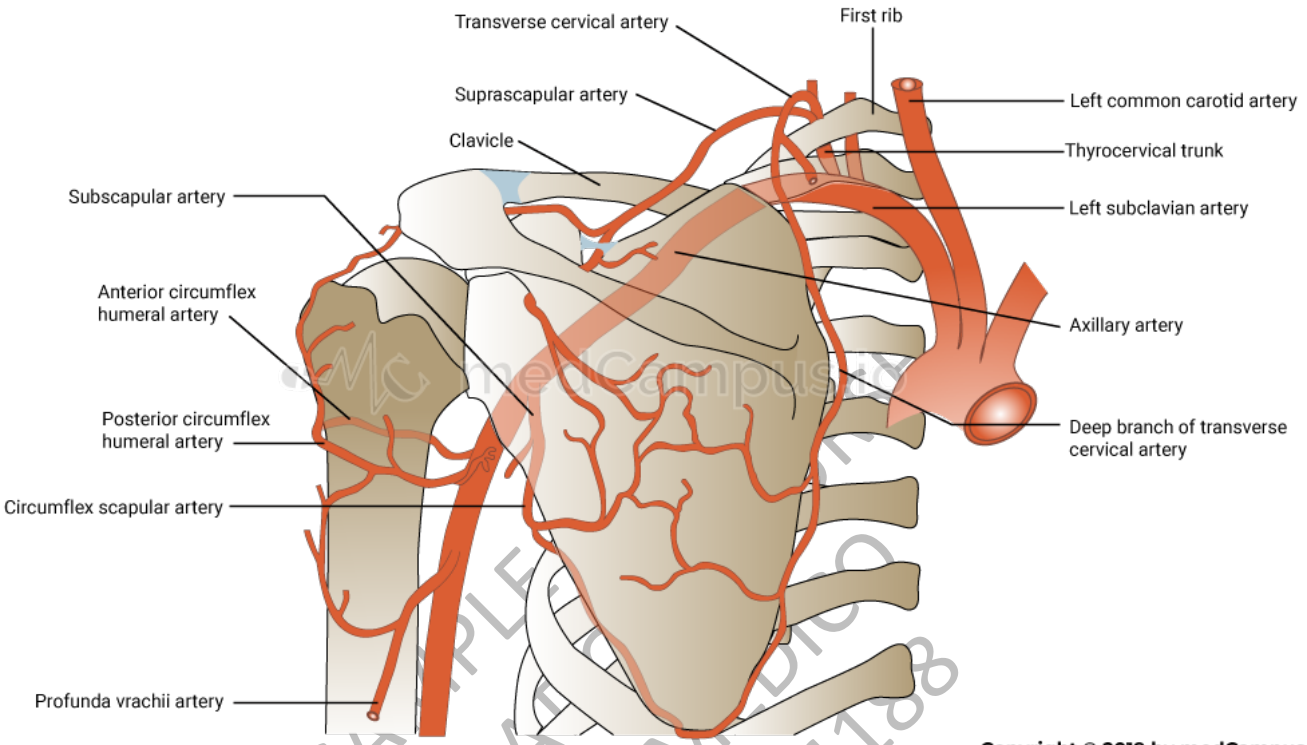






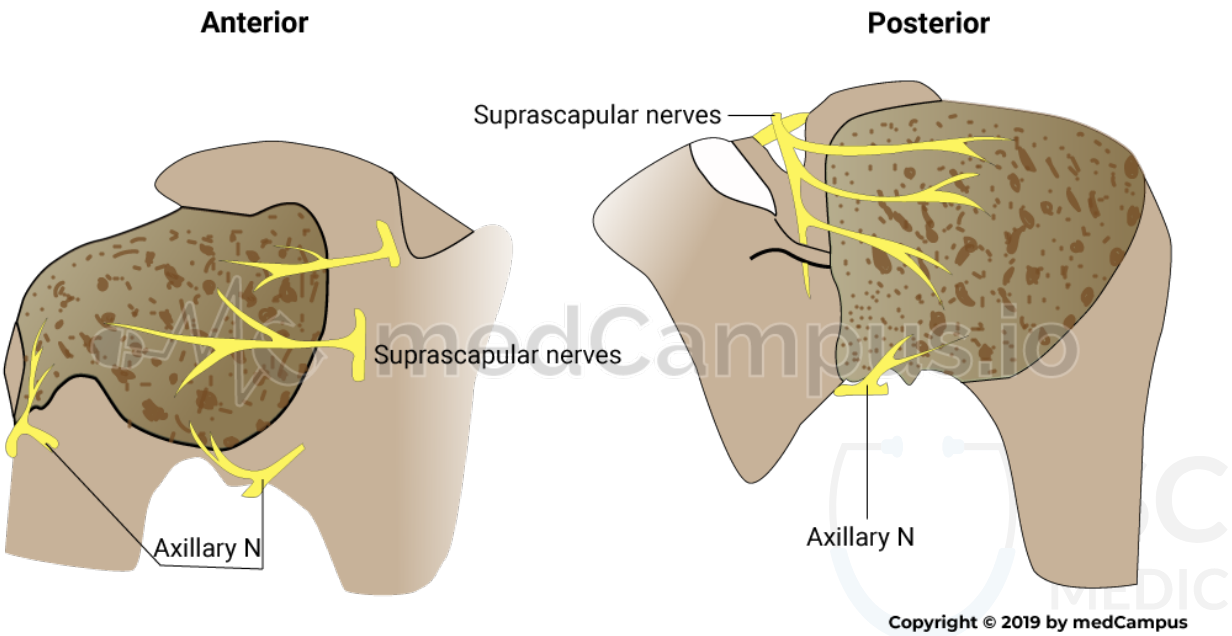
Blood supply

- Anterior and posterior circumflex humeral arteries (branches of the axillary artery)
- Suprascapular artery (branch of the thyrocervical trunk of the subclavian artery)
- Venous drainage into the subclavian and axillary vein



Nerve supply

- Innervation is provided by the **axillary**, **suprascapular** and **lateral pectoral** nerves.



Supports

Capsule:

a fibrous capsule envelops the entire joint

Reinforced superiorly by the rotator cuff muscles and the long head of the biceps brachii

Weakest inferiorly

Glenoid labrum: a soft tissue rim surrounding the glenoid fossa of the scapula

Ligaments

- **Coracoacromial ligament**

Extends from the coracoid process to the acromion process of the scapula

Forms a tunnel, known as the coracoacromial arch, that prevents the superior displacement of the head of the humerus from the glenoid cavity

- **Coracohumeral ligament**

Extends from the coracoid process to the greater and lesser tubercles of the humerus

Strengthens the anterior part of the joint capsule

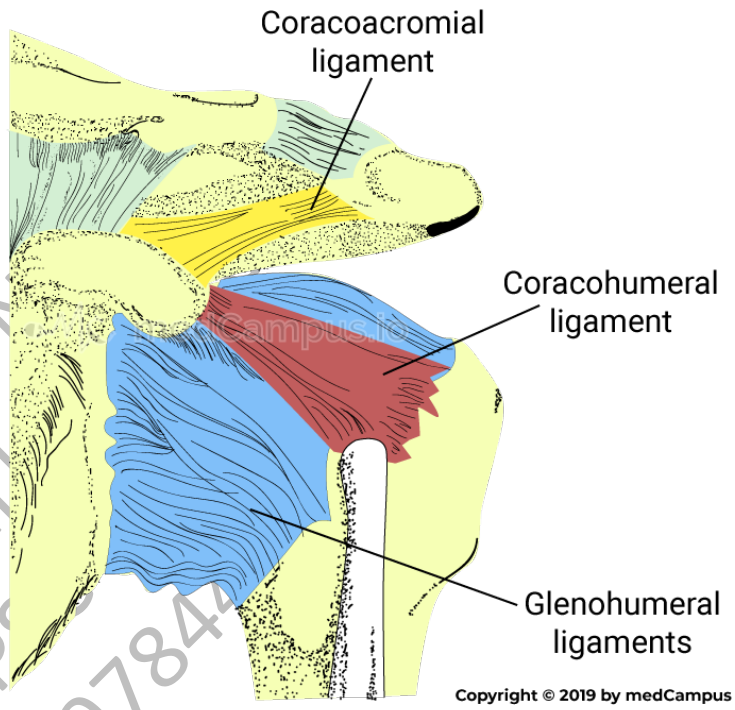
- **Glenohumeral ligament**

strengthens the joint capsule between the supraglenoid tubercle of the scapula and the anatomical neck of the humerus

- **Transverse humeral ligament**

Extends from the greater tubercle to the lesser tubercle of the humerus

Bridges the bicipital groove and holds the biceps tendon in place

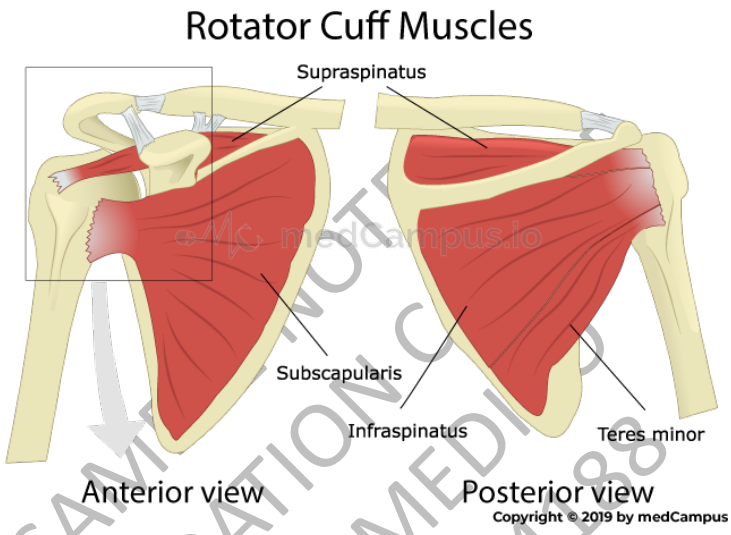


Rotator cuff muscles

The tendons of subscapularis, supraspinatus, infraspinatus and teres minor fuse with the lateral part of the joint capsule to form the 'rotator cuff'.

These short muscles collectively produce a compressive force during active glenohumeral movements which maintains congruent contact between the head of the humerus and the glenoid fossa, helps to resist skid, and checks excessive translation.

The rotator cuff also provides strong lateral stability and prevents this part of the lax capsule from being nipped during joint movements.



Clinical Anatomy

Rotator cuff disease

The subacromial space is defined inferiorly by the humeral head, and superiorly by the anterior edge and inferior surface of the anterior third of the acromion, coracoacromial ligament and acromioclavicular joint, forming the coracoacromial arch.

It is occupied by the supraspinatus tendon, subacromial bursa, tendon of the long head of biceps brachii, and the capsule of the shoulder joint.

Rotator cuff disease is a painful condition with a multifactorial aetiology in which severe or chronic impingement of the rotator cuff tendons on the undersurface of the coracacromial arch is often a significant factor.

The cuff normally impinges against the coracoacromial arch when the humerus is abducted, flexed and internally rotated. This is known as the impingement position.

The supraspinatus tendon is anatomically affected most by the impingement, which interestingly also coincides with an area of reduced vascularity in this tendon.

Severe impingement can be caused by thickening of the coracoacromial arch, by inflammation of the cuff from disorders such as rheumatoid arthritis, or as a result of prolonged overuse in the impingement position, as in cleaning windows.

Clinical Anatomy

Painful arc syndrome

When associated with a tendinopathy from age-related degenerative changes within the tendon, impingement may be associated with partial or complete tears of the cuff.

Clinically, this condition causes tenderness over the anterior portion of the acromion, and pain which typically occurs on abducting the shoulder between 60° and 120° (the painful arc).

Frozen shoulder (adhesive capsulitis)

Inflammatory process causing fibroblastic proliferation of joint capsule leading to thickening, fibrosis, and adherence of the capsule to itself and humerus.
Associated conditions-Diabetes, autoimmune thyroiditis, depuytrens contractures
Presentation-Symmetric loss of active and passive rotation of movements (external rotation is most common finding). Self limited disease

Glenohumeral joint dislocations

- The glenohumeral joint is the most frequently dislocated joint in the body.
- It is most unstable anteroinferiorly, which explains why the vast majority of dislocations are anterior, and occur when the arm is forced backwards when it is in abduction, external rotation and extension.
- Clinically, a dislocated shoulder loses its normal contour, and the acromion process, rather than the greater tubercle, becomes the most lateral bony structure.
- The axillary nerve and artery may be injured during dislocation, and this can lead to inability to abduct the shoulder as a result of paralysis of deltoid together with an area of anaesthesia over the distal part of the muscle (sometimes referred to as the 'badge area' of skin), as well as ischaemic changes in the limb. Posterior dislocation is rare and typically occurs when violent movements produce marked internal rotation and adduction, e.g. in epileptic seizures or electric shock.

Gross anatomy, blood supply and lymphatic drainage of liver

PYQs

- Write in brief about hepatic segments (2021)
- Define Porto caval anastomosis. What are their sites? Also write the anastomosing vessels on each site with its applied anatomy. (2011)
- Describe in detail about hepatic spaces in relation to peritoneal reflection. Add a note on its applied importance. (2017)
- Describe the formation and tributaries of portal vein. List the sites of portocaval anastomosis. (2021)

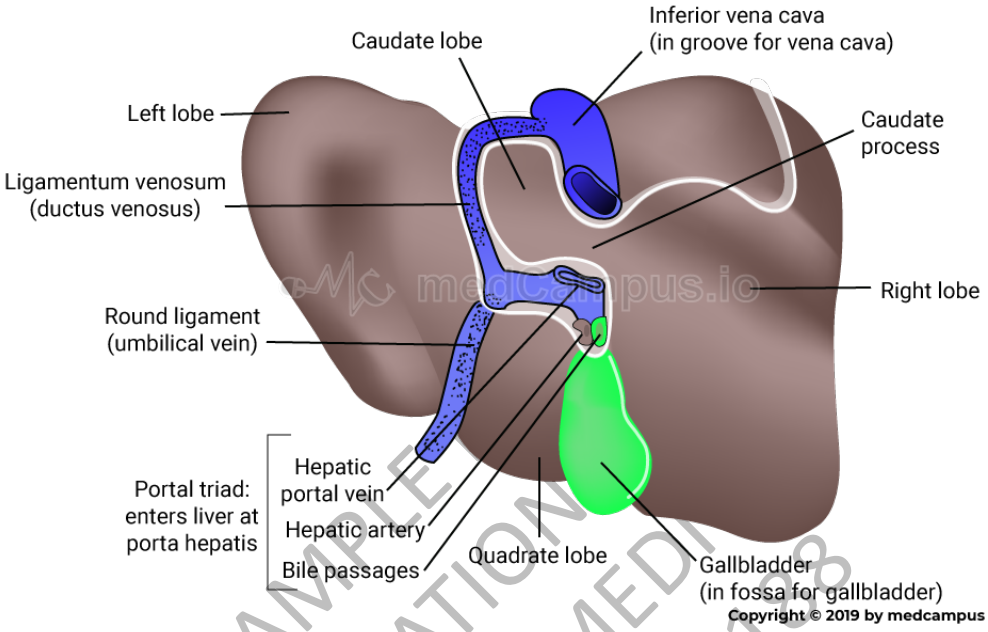
GROSS ANATOMY OF LIVER (Blood supply and lymphatic drainage)

LOCATION

- Liver is a wedge shaped, large solid gland situated in the right upper quadrant of the abdominal cavity.
- It occupies whole of the right hypochondrium, greater part of epigastrium and extends into the left hypochondrium reaching upto the left laterla line.

EXTERNAL FEATURES

- It has five surfaces (1) Anterior (2) Posterior (3) Superior (4) Inferior and (5) Right
- **Out of these inferior surface is well defined.**



- Liver is divided in to right and left lobes by the attachment of falciform ligament anteriorly and superiorly; by the fissure for the ligamentum teres inferiorly; by the fissure for the ligamentum venosum posteriorly.
- Right lobe is much larger than left lobe of the liver.

Caudate lobe	**Quadrate lobe**
Right lobe of the liver Caudate lobe on the posterior surface. **Its boundaries are** **Right-Groove for the inferior vena cava** **Left-Fissure for ligamentum venosum** **Inferior-Porta hepatis**	Right lobe of the liver contains quadrate lobe on the inferior surface. It is rectangular in shape. **Its boundaries are** **Anterior-Inferior border of liver** **Posterior-Porta hepatis** **Right-Fossa for gall bladder** **Left-Fissure for ligamentum teres**

Porta hepatis

- Deep transverse fissure (5cm long), situated on the inferior surface of the right lobe of the liver.
- It lies between caudate lobe above and quadrate lobe below and in front.
- **Contents**-Portal vein, hepatic artery and hepatic plexus of nerves enter into it. Right and left hepatic ducts and few lymphatics leave it.

Omental tuberosity or tuber omentale-Present in the inferior surface of left lobe of liver.

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PATHOLOGY

SAMPLE NOTES

SYLLABUS (Page no.)

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Pathogenesis and histopathology of carcinoma of breast

- Classify carcinoma of the breast and write in brief about histopathology of each type (1996).
- Discuss the histopathologic features that play a role in the prognosis of breast carcinomas (2004).
- Discuss the role of FNAC and biopsy in the diagnosis of neoplasms of the Breast. Briefly mention the different histopathologic types of breast carcinoma. (2010)
- What are the possible contributing factors in pathogenesis of carcinoma breast? Describe briefly histopathology of different types of breast carcinoma. (2013)
- Describe risk factors identified for breast carcinoma. Discuss molecular subtypes of invasive breast carcinoma. (2018)
- Describe the molecular mechanisms of carcinogenesis of breast carcinoma. Describe the salient histopathological features of invasive carcinoma of no special type. (2022)
- Describe the microscopic features of breast cancer. Enumerate any five major prognostic factors.(2024)

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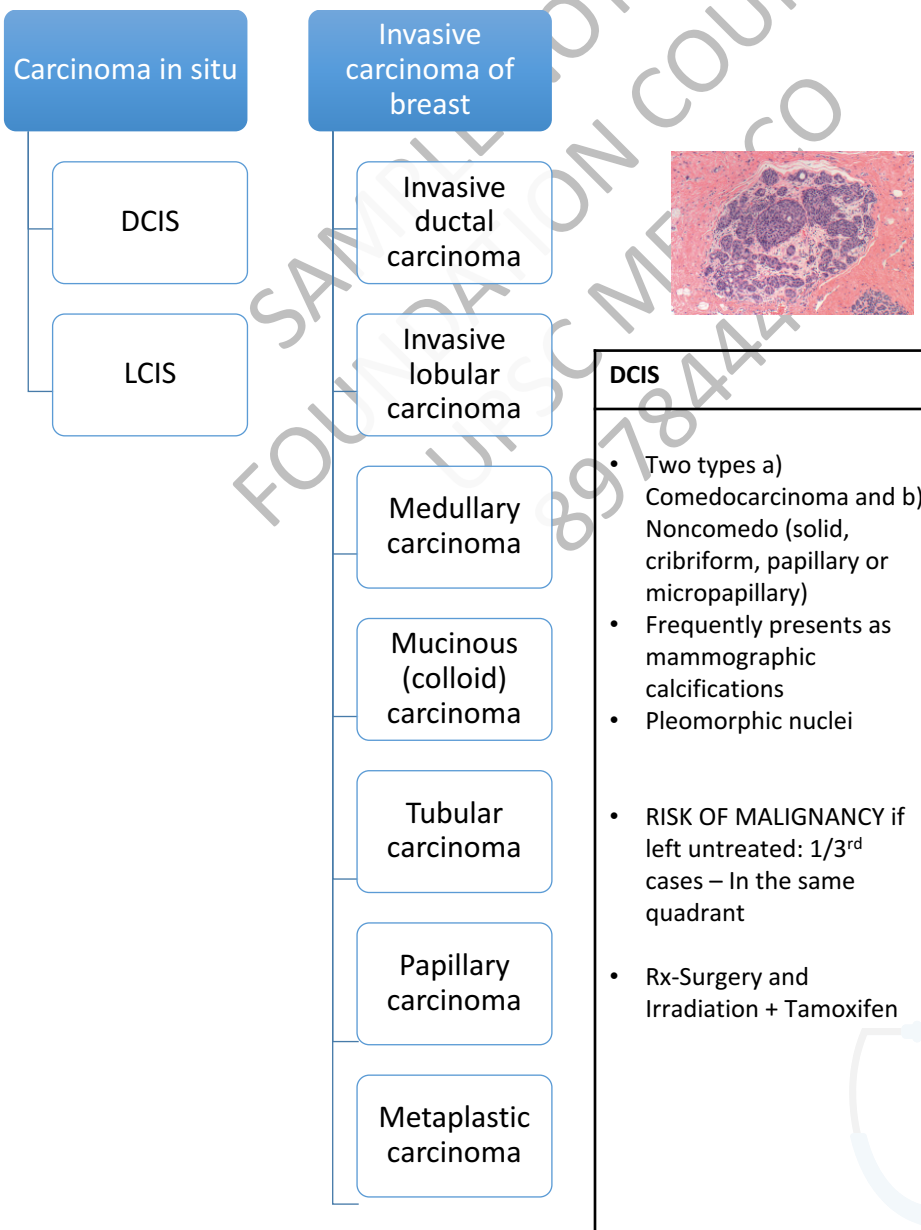
Carcinoma Breast

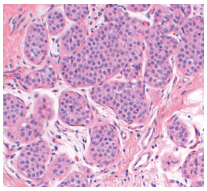
Breast carcinoma is the most common malignancy of women globally (excluding nonmelanoma skin cancer) and causes the majority of cancer deaths in women

Almost all breast malignancies are adenocarcinomas (>95%).
The most common location of tumors within the breast is in the upper outer quadrant (50%), followed by the central portion (20%). About 4% of women with breast cancer have bilateral primary tumors or sequential lesions in the same breast.

CLINICAL/MOLECULAR CLASSIFICATION/MOLECULAR OR GENE PROFILING: LUMINAL CLASSIFICATION

Histopathological types of Breast Carcinoma





DCIS	LCIS
• Two types a) Comedocarcinoma and b) Noncomedo (solid, cribriform, papillary or micropapillary) • Frequently presents as mammographic calcifications • Pleomorphic nuclei	• Incidental finding in biopsy • No calcifications • Signet ring cells are commonly seen • Monomorphic, have bland, round nuclei, and are found in loosely cohesive clusters within the lobules.
• RISK OF MALIGNANCY if left untreated: 1/3rd cases – In the same quadrant	RISK OF MALIGNANCY if left untreated: 1/3rd cases – same or contralateral breast
• Rx-Surgery and Irradiation + Tamoxifen	• Rx- Close clinical and radiologic follow-up, chemoprevention with tamoxifen or, less commonly, bilateral prophylactic mastectomy.

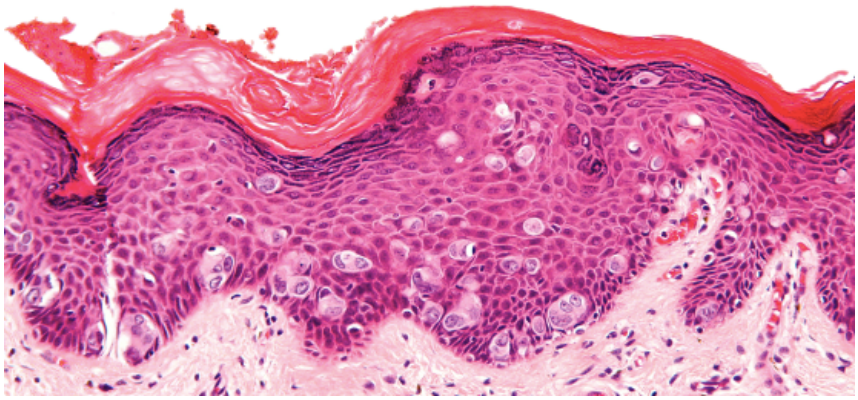
Invasive Breast Carcinoma

Histological type	Features
Invasive ductal carcinoma	• Most common type of breast carcinoma • Gross features-Hard tumor with irregular borders, with a characteristic grating sound when cut or scraped • Five major patterns of gene expression noted (Luminal A, B, Normal, Basal line, Her 2 positive) • **Paget disease of nipple is infiltration of skin of nipple by underlying intraductal carcinoma**
Invasive lobular carcinoma	• Frequently bilateral • Signet ring cells are common
Medullary carcinoma	• Gross –soft, fleshy, well circumscribed • Solid syncitium like sheets of large cells
Mucinous (colloid carcinoma)	• Small islands of cells with large lakes of mucin
Tubular carcinoma	• Well formed tubules
Papillary carcinoma	• Tumors with papillary architecture
Metaplastic carcinoma	• Adenocarcinomas/squamous cell carcinomas/Sarcoma

Paget's disease of Nipple

Paget's disease represents the superficial manifestation of an **underlying malignant lesion** in Breast.
Nipple Eczema Should be Biopsied. (Often U/L)

Large Ovoid cells with abundant clear pale cytoplasm in malpighian layer known as Paget cells seen



BREAST CARCINOMA - CLASSIFICATION

A. Noninvasive

1. Ductal carcinoma in situ
2. Lobular carcinoma in situ

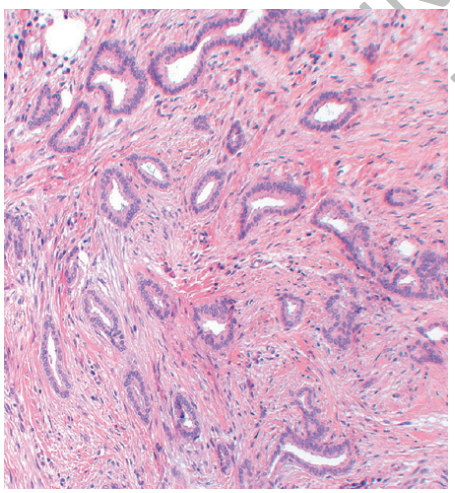
B. Invasive

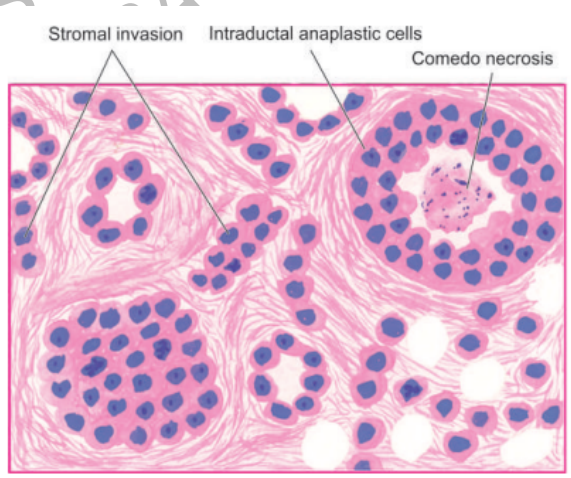
1. **Invasive ductal carcinoma (includes all carcinomas that are not of a special type)—70% to 80%**
2. Invasive lobular carcinoma— ~10% to 15%
3. Carcinoma with medullary features— ~5%
4. Mucinous carcinoma (colloid carcinoma) — ~5%
5. Tubular carcinoma— ~5%
6. Other types

Invasive carcinoma of no special type

Invasive ductal carcinoma is a term used for all carcinomas that cannot be subclassified into one of the specialized types described below

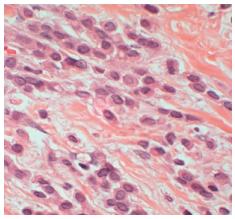
- Most common type of breast carcinoma (70%-80%)
- Associated with DCIS.
- **Gross features**-Hard tumor with irregular borders with a characteristic **grating sound when cut or scraped**
- **Microscopic appearance**
  - Tumors with well-developed tubules and low-grade nuclei
  - Tumors consisting of sheets of anaplastic cells
  - Desmoplastic response.
- **HORMONAL ASSAY** - About 50% to 75% of ductal carcinomas are ER positive, 20% are HER2 positive, and 15% are negative for both ER and HER2



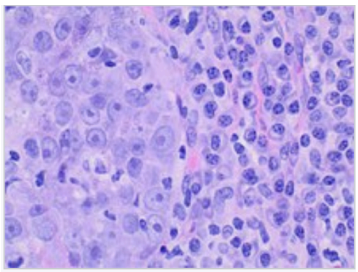


Invasive lobular carcinoma— ~10% to 15%

Bilateral involvement
Monomorphic, have bland, round nuclei, and are found in loosely cohesive clusters within the lobules.
The cells invade stroma individually and often are aligned in **"single-file"**
No desmoplastic response
Noncohesive tumor cells that invade as linear cords of cells and induce little stromal response.



Frequently spread to cerebrospinal fluid, serosal surfaces, gastrointestinal tract, ovary, uterus, and bone marrow.
Almost all are ER/PR + : GOOD PROGNOSIS
HER/NEU rare

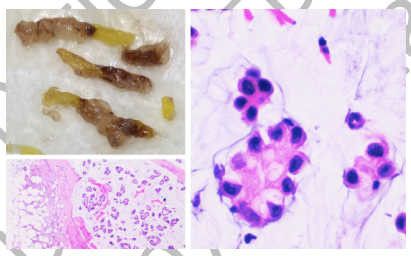


On histopathology, medullary breast carcinoma is characterized by groups of tumor cells with syncytial appearance (that is, seemingly fused cytoplasms, at left). There is typically also a lymphocytic and plasma cell infiltrate (right).[5]

Carcinoma with medullary features

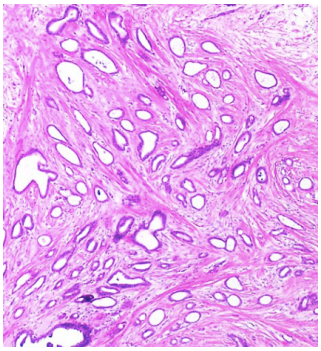
Triple negative/BRCA1
Round masses mimick benign tumors
Sheets of large anaplastic cells (SYNCITIUM) associated with
pronounced lymphocytic infiltrates (Better prognosis)
composed predominantly of T cells
Mutations of E-cadherin

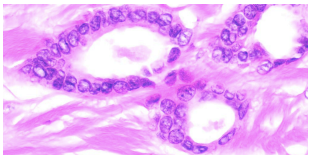
Mucinous carcinoma (colloid carcinoma)



ER+/HER2 –
Abundant amounts of extracellular mucin
Soft gelatinous tumors

Tubular carcinoma
ER+/Her2-
Small irregular mass
The tumor cells are arranged in well formed tubules and have low-grade nuclei. Lymph node metastases are rare, and the prognosis is excellent.
More than 90% of the tumor composed of small, ovoid or angulated tubules with open lumina





Others – Papillary/Metaplastic/Inflammatory carcinoma

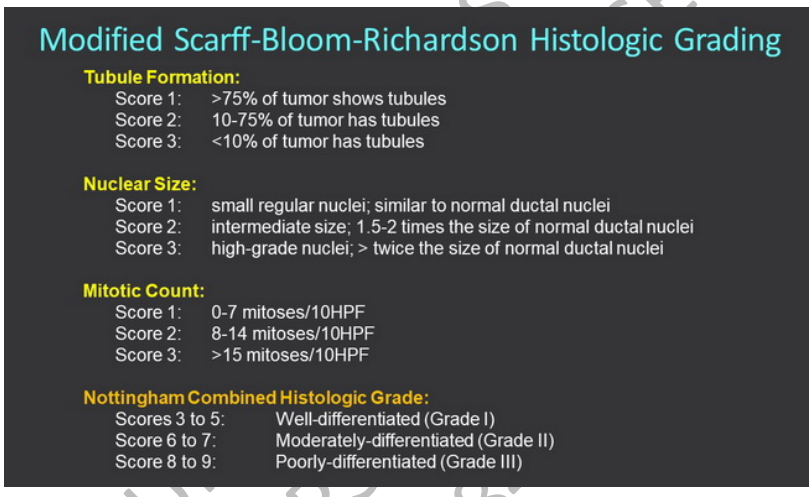
INFLAMMATORY CARCINOMA

- **CLINICAL PRESENTATION:** swollen erythematous breast without a palpable mass
- **True inflammation is absent :** poorly differentiated cells obstructing lymphatics-mimicking inflammed appearance.
- **POOR PROGNOSIS**

MODIFIED BLOOM RICHARDSON SCORE/NOTTINGHAM SCORING SYSTEM

Grading of ductal carcinoma of breast is performed on the **invasive component**. It is an estimation of the **degree of differentiation** of the tumor. The currently used histologic grading system is known as the **Elston-Ellis modification of Scarff-Bloom-Richardson grading system** OR **Nottingham Combined Histologic Grade**.

Histologic grade is a **powerful prognostic indicator**.

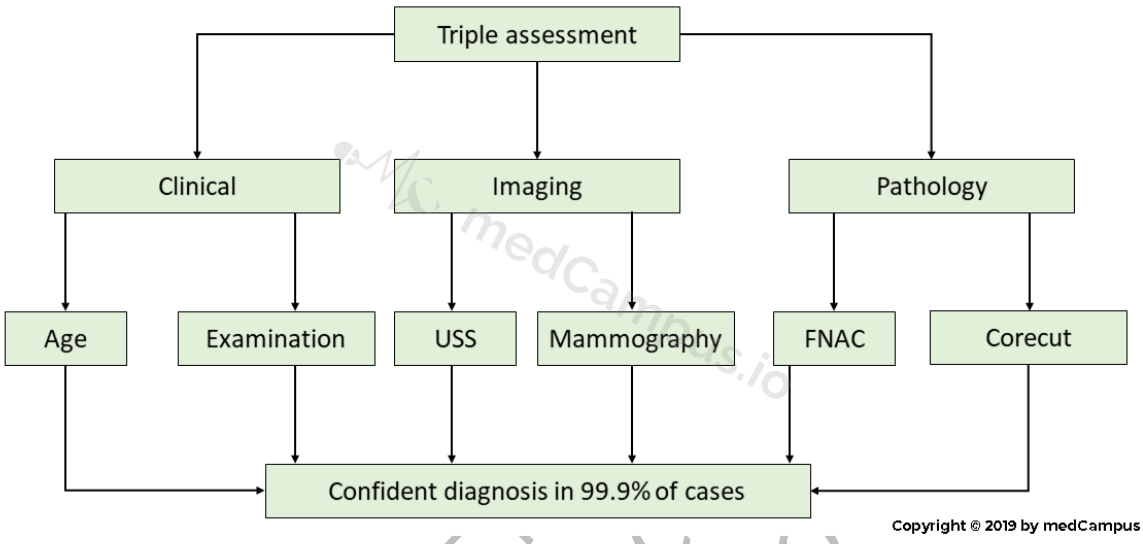


Better prognosis	**Worst prognosis**
Medullary carcinoma, Tubular carcinoma, Colloid carcinoma Invasive lobular carcinoma	Invasive ductal carcinoma – Poorly differentiated Inflammatory carcinoma
LUMINAL A/B	Triple negative, Her2 Neu+, Claudin type
Nottingham score 3-7	Nottingham score 8-9
Axillary LN spared	Axillary LN involvement
Metastasis -	Metastasis +

Triple Assessment

- In any patient who presents with a breast lump or other symptoms suspicious of carcinoma, the diagnosis should be made by a combination of **clinical assessment, radiological imaging and a tissue sample taken for either cytological or histological analysis**, the so-called triple assessment.

- **The positive predictive value (PPV) of this combination should exceed 99.9%.**



Biopsy/FNAC

FNAC	NEEDLE BIOPSY
Procedure Cytology is obtained using a 21G or 23G needle and 10 mL syringe with multiple passes through the lump with negative pressure in the syringe. The aspirate is then smeared on to a slide, which is air dried or fixed. Advantages • Least invasive technique of obtaining a cellular diagnosis and is rapid and very accurate if both operator and cytologist are experienced. Disadvantages • False negative results can occur • invasive cancer cannot be distinguished from in situ disease	Procedure Histology can be obtained under local anesthesia using a spring-loaded core needle biopsy device. Advantages • Definitive preoperative diagnosis • Differentiates between DCIS and Invasive disease • Tumor can be stained for receptor status Disadvantages • Invasive

Breast Carcinoma - Risk Factors

Hormonal factors (hyperestrogenemia is the risk factor)

- Early menarche
- Late menopause
- First full-term pregnancy >35 years
- Nulliparity
- Obesity (post menopausal)

Higher socioeconomic status, high fat diet, alcohol intake

- Age: Incidence increases with increasing age.
- Sex: more common in females.
- Personal history and family history of breast cancer
- Race, ethnicity (white women have increased risk compared to women of other races)
- History of radiation exposure
- Dense breast

Genetic factors:

Mutations in tumor suppressor genes

- *BRCA 1*
- *BRCA 2*
- *TP53*
- *CHEK2*

Hereditary syndromes

- Li Fraumeni syndrome
- Cowden's syndrome
- HNPCC syndrome
- Peutz-Jegher's syndrome
- Ataxia telangiectasia

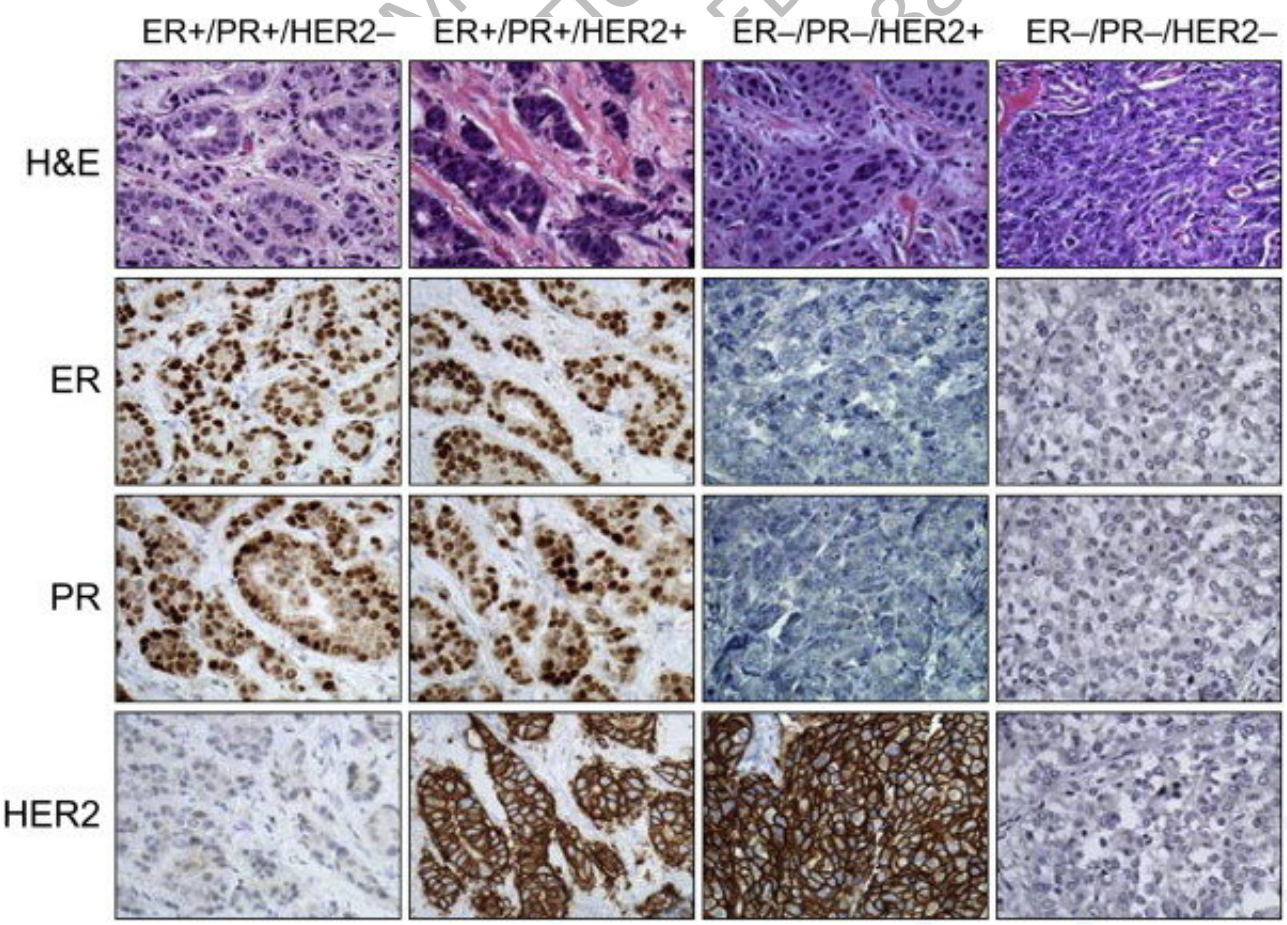
Table 19.6 Factors Associated With Development of Invasive Carcinoma

Factor	Relative Risk[a]	Absolute Lifetime Risk[a]
Women with no risk factors	1.0	3%
First-degree relative(s) with breast cancer[b]	1.2–9.0	4%–30%
Germline tumor suppressor gene mutation (e.g., *BRCA1* mutation)	2.0–45.0	6% to >90%
Menstrual History		
Age at menarche <12 years	1.3	4%
Age at menopause >55 years	1.5–2.0	5%–6%
Pregnancy		
First live birth <20 years (protective)	0.5	1.6%
First live birth 20–35 years	1.5–2.0	5%–6%
First live birth >35 years	2.0–3.0	6%–10%
Never pregnant (nulliparous)	3.0	10%
Breast-feeding (slightly protective)	0.8	2.6%
Benign Breast Disease		
Proliferative disease without atypia	1.5–2.0	5%–6%
Proliferative disease with atypia (ALH and ADH)	4.0–5.0	13%–17%
Carcinoma in situ (ductal or lobular)	8.0–10.0	25%–30%
Ionizing radiation	1.1–1.4	3.6%–4.6%
Mammographic density	3.0–7.0	10%–23%
Postmenopausal obesity and weight gain	1.1–3.0	3.6%–10%
Postmenopausal hormone replacement	1.1–3.0	3.6%–10%
Alcohol consumption	1.1–1.4	3.6%–4.6%
Alcohol consumption	1.1–1.4	3.6%–4.6%

[a]Relative risk is the likelihood of developing cancer compared to a woman with no risk factors—whose relative risk is 1.0. Absolute lifetime risk is the fraction of women expected to develop invasive carcinoma without a risk reducing intervention. For women with no risk factors, there is about a 3% chance of developing invasive breast cancer.
[b]The most common family history is a mother who developed cancer after menopause. This history does not increase the risk of her daughters.

Breast Carcinoma - Molecular Profiling (St. Gallen Classification)

Type	Receptor status	Level of protein Ki-67	Prognosis	Management
Luminal A	ER/PR positive Her2neu negative	Low levels Ki-67 <14%	Low grade, grow slowly and have the best prognosis. Well-differentiated Most frequent (50-72%), the lowest risk of recurrence and relapse.	*Third generation aromatase inhibitors (Anastrazole) *Selective estrogen receptor modulators (Tamoxifen)
Luminal B	ER/PR positive Her2neu positive	High levels Ki-67 > 14%	Grow slightly faster than luminal A and their prognosis is slightly worse Poorly differentiated, frequent bone metastasis	*Tamoxifen/anastrazole *Plus neoadjuvant chemotherapy
HER2/NEU positive	ER/PR negative Her2neu positive	Any level	Grows faster than luminal cancers and have a worse prognosis. Associated with TP53 mutations. Very aggressive with high proliferative index.	*Herceptin (Transtuzumab-monoclonal antibody)
Basal-like (Triple negative)	ER/PR negative Her2neu negative	CK5/6+ EGFR+	More common in woman with BRCA1 gene mutation Large tumor, poorly differentiated, high mitotic index and tumor necrosis Visceral metastasis is often seen (liver, lung and CNS, etc.) Worst prognosis	*Chemotherapy *PARP inhibitors-olaparib



Recent Developments

Oncotype Dx
Mammaprint
EndoPredict
PAM50

- Using these molecular tests, a recurrence score (0 – 100) is calculated.
- If the score is low, then there is no advantage of adding chemotherapy.
- If the score is high, then chemotherapy should be added.

Oncotype Dx	Mammaprint
Is a 21-gene assay	Is a 70-gene assay
Used for T1,2 N0 M0 breast cancers – (Node negative , metastasis negative)	Used for T1,2 N0 M0 breast cancers – (Node negative , metastasis negative)
Can only be used in hormone (ER, PR) receptor–positive cancers	Can be used in both hormone receptor (ER, PR) positive and hormone receptor– negative cancers

Claudin-low breast cancer is a molecular type of breast cancer originally identified by gene expression profiling and reportedly associated with poor survival.

Claudin-low breast cancer is typically negative for ER, PR, HER2, claudin 3, claudin 4, claudin 7 and E-cadherin.

Claudin-low tumors identified with [illegible] immunohistochemical panel, were associated with **young age of onset, higher tumor grade, larger tumor size, extensive lymphocytic infiltrate and a circumscribed tumor margin.** Patients with claudin-low tumors had a worse overall survival when compared to patients with luminal A type breast cancer.

Interestingly, claudin-low tumors were associated with a low local recurrence rate following breast conserving therapy.

Table 19.7 Summary of the Major Biologic Types of Breast Cancer

Feature	ER Positive/HER2 Negative	HER2 Positive (ER Positive or Negative)	Triple Negative (ER, PR, and HER2 Negative)
Overall frequency	50%–65%	20%	15%
Typical patient groups	Older women; men; cancers detected by screening; germline *BRCA2* mutation carriers	Young women; germline *TP53* mutation carriers	Young women; germline *BRCA1* mutation carriers
Ethnicity			
European/American	70%	18%	12%
African/American	52%	22%	26%
Hispanic	60%	24%	16%
Asian/Pacific Islander	63%	26%	11%
Grade	Mainly grade 1 and 2	Mainly grade 2 and 3	Mainly grade 3
Complete response to chemotherapy	Low grade (<10%), higher grade (10%)	ER positive (15%), ER negative (>30%)	30%
Timing of relapse	May be late (>10 years after diagnosis)	Usually short (<10 years after diagnosis)	Usually short (<8 years after diagnosis)
Metastatic sites	Bone (70%), viscera (25%), brain (<10%)	Bone (70%), viscera (45%), brain (30%)	Bone (40%), viscera (35%), brain (25%)
Similar group defined by mRNA profiling	Luminal A (low grade), luminal B (high grade)	Luminal B (ER positive), HER2-enriched (ER negative)	Basal-like
Common special histologic types	Lobular, tubular, mucinous, papillary	Apocrine, micropapillary	Carcinoma with medullary features
Common somatic mutations	*PIK3CA* (40%), *TP53* (26%)	*TP53* (75%), *PIK3CA* (40%)	*TP53* (85%)

PATHOGENESIS : GENETIC (or) Molecular / Hormonal / Environmental factors

HORMONAL : ESTROGEN stimulate the production of growth factors, such as transforming growth factor-α, platelet-derived growth factor, fibroblast growth factor, and others, which may promote tumor development through paracrine and autocrine mechanisms.

ENVIRONMENTAL :

- The risk is significantly higher in the Americas and Europe than in Asia and Africa.
- Incidence and mortality rates are five times higher in the United States than in Japan.

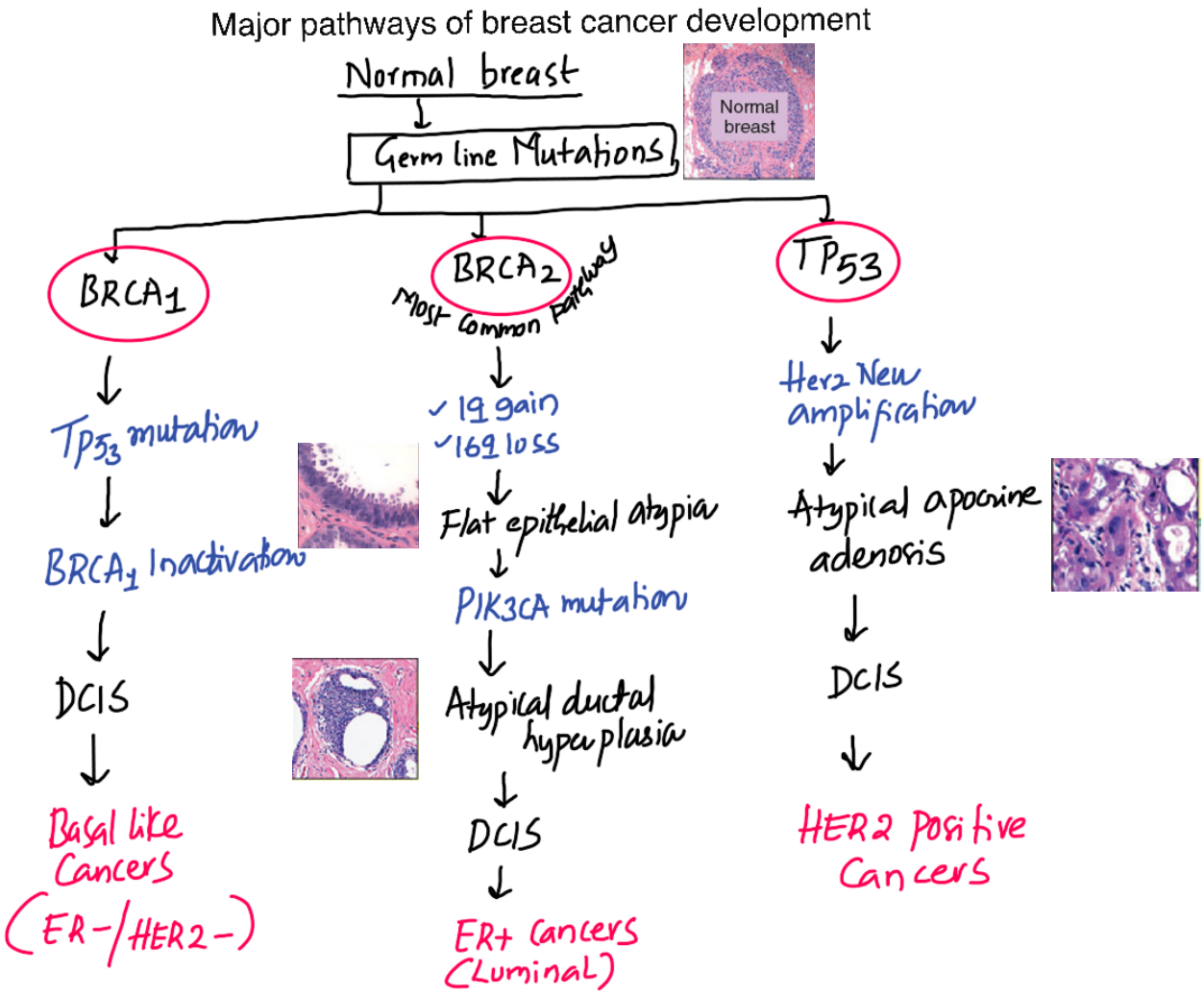
Environmental influences are suggested by the variable incidence of breast cancer in genetically homogeneous groups (e.g., Japanese women living in Japan and the United States)

- Some risk factors must be modifiable because migrants from low incidence to high-incidence areas tend to acquire the rates of their new home countries.
- Diet, reproductive patterns, and breastfeeding practices are thought to be involved.
- **In line with this, breast cancer rates appear to be rising in parts of the world that are adopting Western habits.**

Molecular mechanisms of carcinogenesis of breast carcinoma

Stepwise acquisition of driver mutations in the epithelial cells of the duct/lobular system

A common clinically important driver mutation in breast cancer is amplification of the HER2 gene



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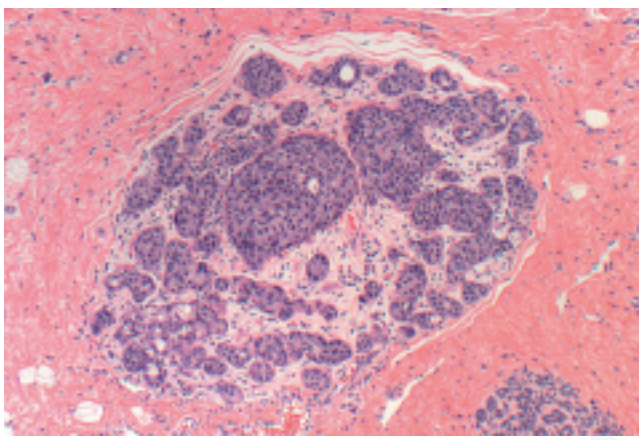

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NOTES EDITION 1.0

GENERAL MEDICINE

SAMPLE NOTES

General Medicine Syllabus (Page no)

Infectious diseases: Etiology, clinical features, diagnosis and principles of management (including prevention) of

- Typhoid (3)
- Rabies (6)
- AIDS (10)
- Dengue (30)
- Kala-azar (37)
- Japanese Encephalitis (45)
- Tuberculosis (51)

Heart and Lung : Etiology, clinical features, diagnosis, and principles of management of:

- Ischemic heart disease (62)
- Pulmonary embolism (75)
- Bronchial asthma (79)
- Pleural effusion (87)

GIT and Kidney: Etiology, clinical features, diagnosis, and principles of management of:

- Malabsorption syndromes (92)
- Acid peptic diseases (99)
- Viral hepatitis (107)
- Cirrhosis of liver (119)
- Glomerulonephritis (135)
- Nephrotic syndrome (140)
- Pyelonephritis (145)
- Renal failure (151)
- Renovascular hypertension (157)

Endocrinology, Hematology and CNS: Etiology, clinical features, diagnosis, and principles of management of:

- Complications of diabetes mellitus (162)
- Coagulation disorders (169)
- Leukemia (184)
- Hypo and hyperthyroidism (201)
- Meningitis and encephalitis (209)

Imaging in medical problems (214)

- Ultrasound
- Echocardiogram
- CT scan
- MRI

Etiology, clinical features, diagnosis and management of ischemic heart disease

- Enumerate the different clinical presentations of Acute Myocardial Infarction. How will you manage cardiogenic shock (1997)?
- Describe the clinical features, investigations and management of Acute Myocardial Infarction (1999)
- Describe the symptoms and sign of acute MI. How will you investigate the case and what positive finding will you get in reports? Describe the treatment in brief. (2007)
- What is unstable angina? How will you manage a patient with unstable angina? (2012)
- Enumerate various coronary risk factors. Describe the management of acute myocardial infarction in the emergency room. (2011)
- Discuss the pharmacological strategies available for the post-myocardial infarction management. (2013)
- A 60-year-old chronic smoker, obese gentleman who had been suffering from diabetes for last five years, not on regular treatment, developed 'Ghabarahat' and sudden breathlessness and perspiration.
    (i) How would you work up this case? Draw a flow chart.
    (ii) Enumerate the line of treatment in case ECG indicates acute inferior wall myocardial infarction.
    (iii) Which lifestyle measures would you suggest to this person? (2014)
- Describe the management of acute anterior wall myocardial infarction. (2016)
- What advice would you give to a 50-year-old diabetic male for the prevention of Ischaemic heart disease? (2019)
- Discuss the management of acute myocardial infarction (2020)
- A sixty-year-old diabetic presented to the emergency department with acute onset, central squeezing type of chest pain, which is severe in intensity and not relieving even after taking rest.
    (i) What is the most probable diagnosis?
    (ii) How will you investigate the case to reach the diagnosis?
    (iii) Outline the steps in the management of the case? (2021)
- A sixty-year-old male develops central chest pain while walking uphill. The pain is squeezing in character, radiating to left arm, that relieves on taking rest. Discuss in short about the evaluation and treatment of this case. (2024)

FOCUS AREAS:

1) Coronary risk factors
2) PREVENTION OF Ischemic Heart Disease (Life style measures)
3) Clinical presentations of IHD (Symptoms and Signs)

Management of

- Stable angina
- Unstable angina
- Acute MI (Anterior wall MI, Inferior Wall MI)
- Post MI
- Cardiogenic shock

#unasked

4) Prinzmetal's angina

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Etiology, clinical features, diagnosis and management of ischemic heart disease

ISCHEMIC HEART DISEASE:

Approach to Chest Pain

D/D

- Ischemic heart disease
- Pericarditis (pleuritic sharp pain, retrosternal, pericaridal friction rub)
- Aortic dissection (Tearing or ripping, knife like, often radiating to back, Hypertension, loss of peripheral pulses)
- Pulmonary embolism (Pleuritic or heaviness: Great masquerader, dyspnea, tachypnea, and tachycardia)
- Pulmonary hypertension (Pressure type, substernal, Signs of increased venous pressure)
- Pneumonia or pleuritis (Unilateral, pleuritic, cough, fever, rales, rub)
- Spontaneous pneumothorax (Pleuritic, decreased breath sounds)
- Esophageal reflux/peptic ulcer (Burning type, epigastric)
- Costochondritis (ACHING)
- Gall bladder disease (Colicky)
- Cervical disc disease (Aching plus numbness)
- Herpes zoster (Sharp, burning dermatomal distribution)
- Psychological (Variable)

Evaluation = (History+Physical examination)

HISTORY:

Ask for quality of pain, provoking or alleviating factors and associated symptoms.
Past medical history is useful in assessing the patient for risk factors for coronary atherosclerosis and VTE.
H/O Connective tissue disease (MARFAN SYNDROME)
DIET HISTORY

PHYSICAL EXAMIANTION

1) Assess for patients clinical stability
2) General appearance (LEVINE SIGN), BODY HABITUS
3) Vital sigs (TACHYCARDIA, HYPOTENSION)
4) Lung examination : Breath sounds
5) Cardiac : JVP (N) , S3/S4, Murmurs, Pericardial friction rubs
6) Abdominal : Abdominal tenderness
7) Musculoskeletal

INVESTIGATIONS (Helps in diagnosis and guides treatment)

1) **ECG**
2) **Chest X ray**
3) **2D echo**
4) **Cardiac biomarkers**
5) **Provocative tests for ischemia : Stress testing: Exercise ECG**
6) **CT angiography**
7) **MRI**

Etiology, clinical features, diagnosis and management of ischemic heart disease

Risk factors

Factors not amenable to preventive measures (Non-Modifiable)

- Increasing age
- Make sex
- Family history of premature IHD

Factors which can be minimized by preventive measures

- Diet (+ association with consumption of saturated fats and sugars and -association with consumption of dietary fiber)
- Lipid metabolism (Serum cholesterol levels)
- **Hypertension**
- **Diabetes mellitus**
- **Obesity**
- Hypercoagulability of blood (High plasma levels of factor VII, factor VIII and fibrinogen)
- Hyperhomocysteinemia
- Physical inactivity
- Smoking
- Alcohol (moderate drinking is protective-raises HDL levels, relieves mental stress)
- Socioeconomic factors (Upper class)
- Psychological stress (Type A personality-aggressive, striving, ambitious, restless person bothered with deadlines)
- **Lack of Sleep (Latest update)**

AHA's Life's Essential 8

- **Eat better**
- **Be more active**
- **Manage blood sugar**
- **Manage weight**
- **Control cholesterol**
- **Manage blood pressure**
- **Quit tobacco**
- **Get healthy sleep**

Life style changes to prevent IHD

A diet low in saturated and trans-unsaturated fatty acids and a reduced caloric intake to achieve optimal body weight	**Effective control of Hypertension** DASH diet (Dietary approaches to stop Hypertension)
Weight loss in case of obesity	Maintain normal weight for adults (BMI 20-25 kg.m2) Reduce salt intake to < 100mmol/day (<6g Nacl) Engage in regualr aerobic physical exercises
Regular exercise Vigorous exercise (Involving an energy expenditure of 5kcal/minute or more is more beneficial than overall energy output at low intensity effort	**Effective control of Diabetes mellitus**
Cessation of smoking Complete abstinence of tobacco product usage	**Proper management of Dyslipidemia** Weight loss Diet Statins

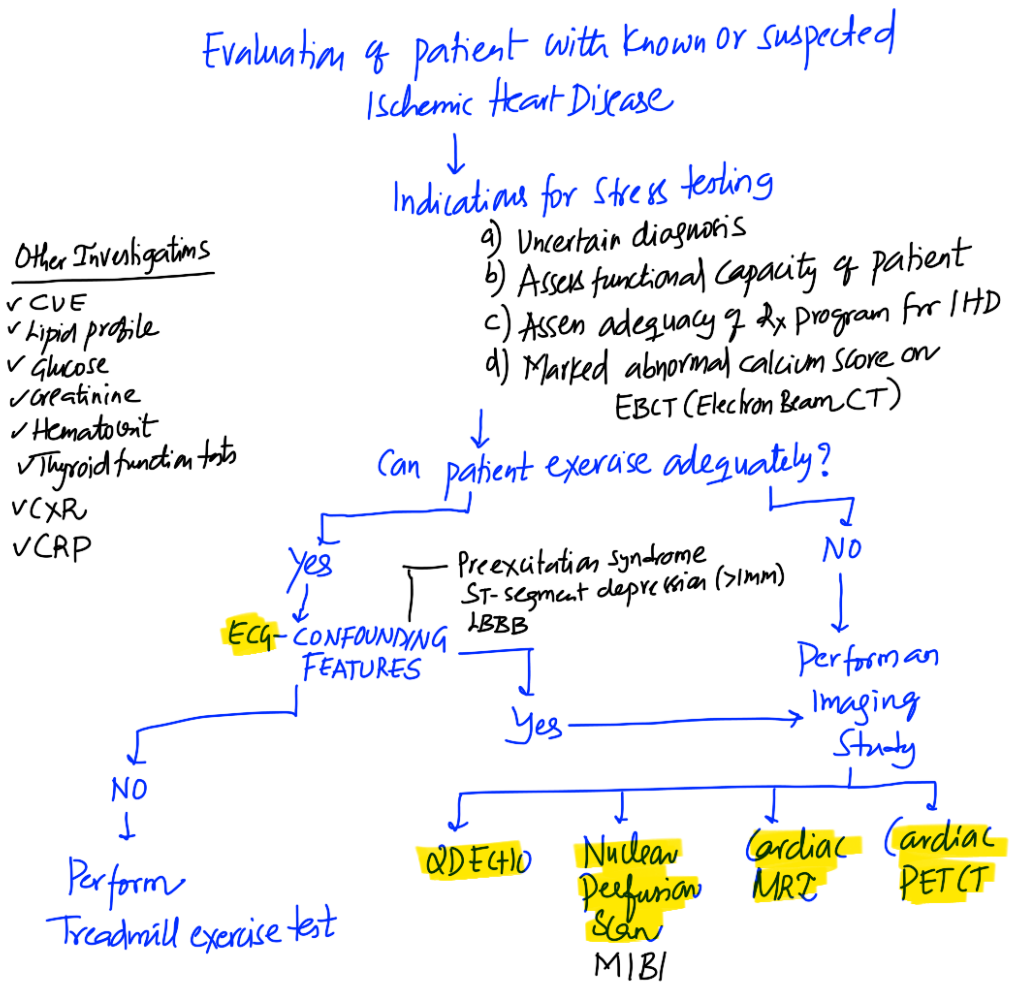
STABLE ANGINA PECTORIS

HISTORY

Ask for quality of pain, provoking or alleviating factors and associated symptoms.
Past medical history is useful in assessing the patient for risk factors (Diabetes, hypertension, smoking, hyperlipidemia) for coronary atherosclerosis and VTE.
Angina equivalents : Dyspnea, nausea, fatigue, and faintness.
Examine for peripheral arterial disease
Family history of premature IHD

PHYSICAL EXAMINATION

Often normal in asymptomatic patients
Search for evidence of atherosclerotic disease at other sites (Abdominal aortic aneurysm, carotid arterial bruits, and diminished arterial pulses in the lower extremities), Xanthelasmas and xanthomas.
Check for ankle brachial index
Palpation: Cardiac enlargement
Auscultation: Arterial bruits, s3,s4+, murmurs



Treatment of Stable angina pectoris

1) Explanation of the problem and reassurance
2) Identification and treatment of aggravating conditions (LVH, Aortic valve disease, HOCM, Obesity, hyperthyroidism and anemia)
3) Adaptation of activity : Physical conditioning, regular program of isotonic exercise.
4) Treat risk factors: Hyperlipidemia, hypertension, diabetes. Quit smoking, weight loss, diet advice
5) **DRUG THERAPY**
    - **Commonly used antianginal drugs are**
        - **Nitrates: nitroglycerin, isosorbide dinitrite and isosorbide 5-mononitrate (symptomatic relief)**
        - **Beta blockers (Once daily dosing: reduce myocardial oxygen demand)**
        - **CCBs : Amlodipine, Dilitazem and verapamil (used when beta blockers are contraindicated)**
        - **Ranolazine in resistant cases**
    - **Chronic administration of ASPIRIN (75-325mg per day oral)**

6) Revascularization (PCI/CABG) : in presence of unstable phases of disease, intractable symptoms, severe ischemia or high risk coronary anatomy, Diabetes and impaired LV function.

Life styles changes to prevent IHD

A diet low in saturated and trans-unsaturated fatty acids and a reduced caloric intake to achieve optimal body weight
Weight loss in case of obesity
Regular exercise Vigorous exercise (Involving an energy expenditure of 5kcal/minute or more is more beneficial than overall energy output at low intensity effort)
Cessation of smoking Complete abstinence of tobacco product usage
Effective control of Hypertension DASH diet (Dietary approaches to stop Hypertension) Maintain normal weight for adults (BMI 20-25 kg/m2) Reduce salt intake to < 100mmol/day (<6g NaCl) Engage in regualr aerobic physical exercises
Effective control of Diabetes mellitus
Proper management of Dyslipidemia Weight loss Diet Statins

Management of patient with IHD

↓

Initiate medical therapy

[Nitrates (SOS) + β blockers + ASA]

↓

Assess for High Risk features

a) Acute coronary syndrome presentation
b) Low exercise capacity
c) Ischemia at low work load
d) EF <40%
e) Large area of Ischemic Myocardium

No → Exertional Symptoms Controlled → Yes

Yes → NO → Refer for Coronary Arteriography

Yes → Continue Medical therapy : conduct periodic Stress assessment

Coronary Arteriography

Anatomy suitable for Revascularization → Yes

- Two vessel disease includes Proximal left descending Coronary artery
- Three vessel disease
- Impaired global LV dysfunction (EF<50%)

→ CABG

- Single vessel disease
- Two vessel disease + (N) LV function

→ PCI

Not suitable (small diffusely diseased vessels)

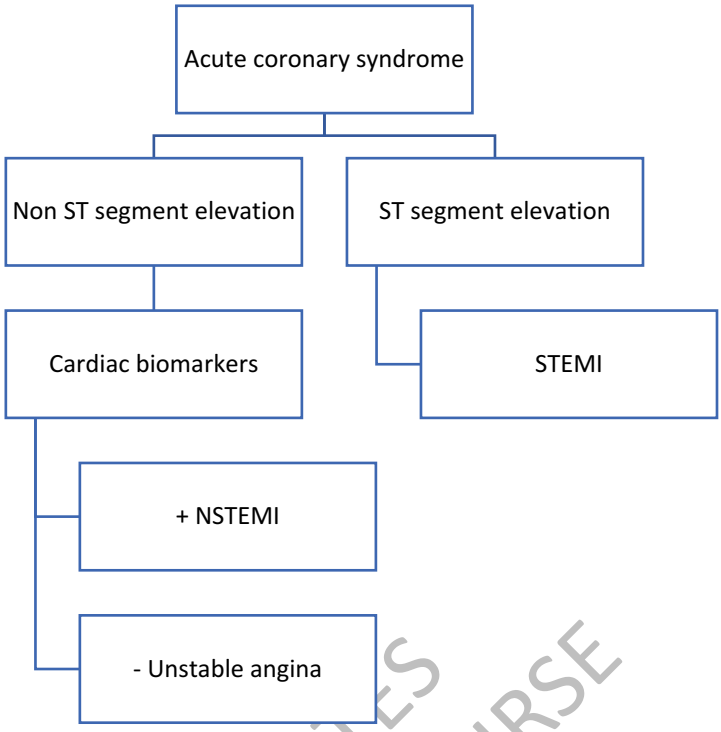
↓

Consider Unconventional treatments

↓

Enhanced

a) External Counterpulsation
b) Experimental therapies
   → Stem cell therapies
   → Cardiac repair with Small non-coding RNA



Acute Myocardial Infarction-Clinical features

Precipitating factors-vigorous physical exercise, emotional stress, or a medical or surgical illness.

Cardinal symptoms

- Chest *Pain-heavy, squeezing* and *crushing* occurs at rest, is usually more severe, and lasts longer than stable angina. Typically, the pain involves the central portion of the chest and/or the epigastrium, and, on occasion, it radiates to the arms. Less common sites of radiation include the abdomen, back, lower jaw, and neck.
- It is often accompanied by weakness, sweating, nausea, vomiting, anxiety, and a sense of impending doom.
- The proportion of painless events is greater in patients with diabetes mellitus, and it increases with age.
- In the elderly, STEMI may present as sudden-onset breathlessness, which may progress to pulmonary edema.
- **Uncommon presentations**
    a) Sudden loss of consciousness
    b) confusional state
    c) a sensation of profound weakness
    d) the appearance of an arrhythmia
    e) evidence of peripheral embolism
    f) an unexplained drop in arterial pressure.

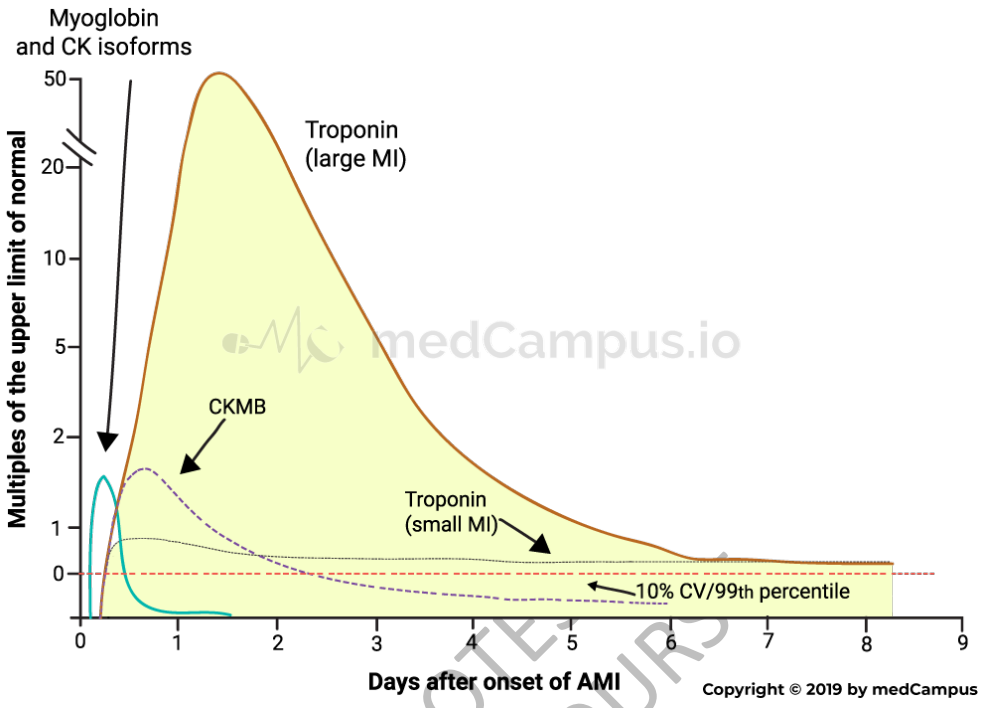
General appearance

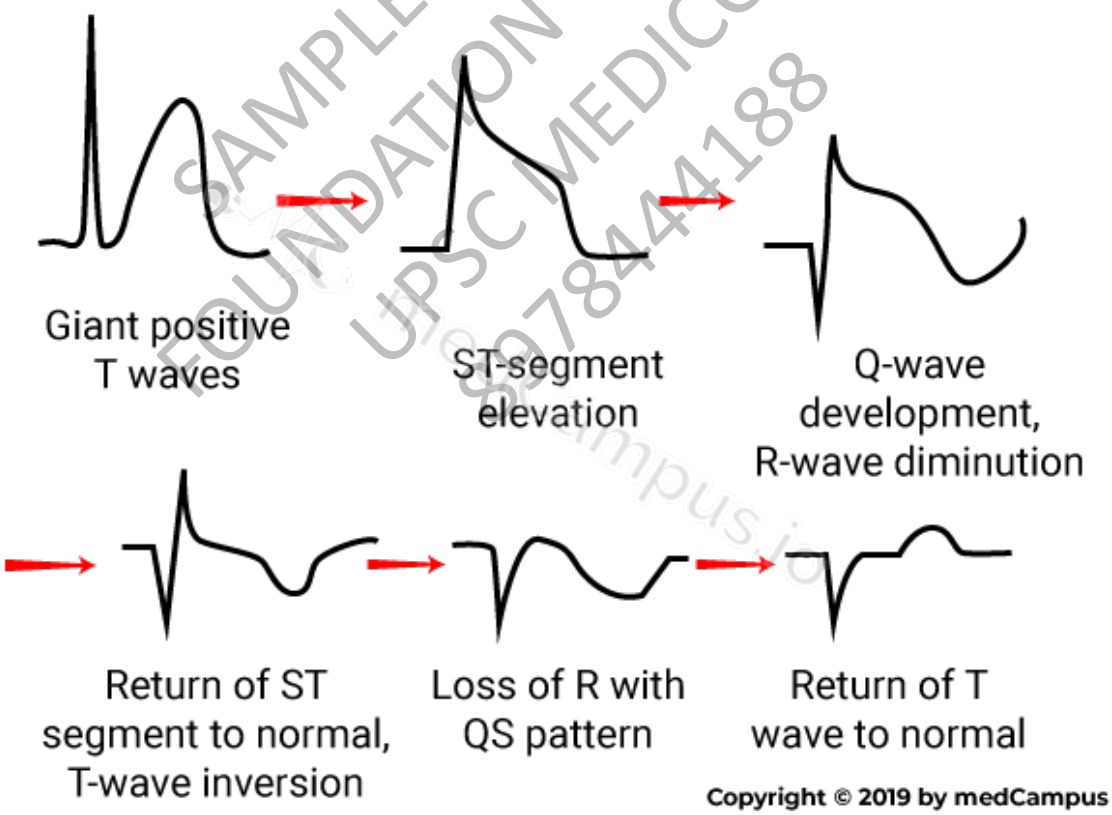
- Most patients are anxious and restless, attempting unsuccessfully to relieve the pain by moving about in bed, altering their position, and stretching.
- Pallor associated with perspiration and coolness of the extremities occurs commonly.

Signs

- Within the first hour of STEMI, about one-fourth of patients with anterior infarction have manifestations of sympathetic nervous system hyperactivity (tachycardia and/or hypertension), and up to one-half with inferior infarction show evidence of parasympathetic hyperactivity (bradycardia and/or hypotension).
- Physical signs of ventricular dysfunction include fourth and third heart sounds, decreased intensity of the first heart sound, and paradoxical splitting of the second heart sound.
- A transient mid-systolic or late systolic apical systolic murmur due to dysfunction of the mitral valve apparatus may be present.
- A pericardial friction rub may be heard in patients with transmural STEMI at some time in the course of the illness, if they are examined frequently.
- The carotid pulse is often decreased in volume, reflecting reduced stroke volume.
- Temperature elevations up to 38°C may be observed during the first week after STEMI.
- The arterial pressure is variable
- Lung crepitations
- Raised JVP

ECG	Serum cardiac biomarkers	Cardiac imaging
ST segment elevations T wave inversions Pathological Q waves • Infarction of the anterior wall is caused by obstruction of the LAD or its branches. Depending on the extent of anterior wall infarction, it results in ECG changes in I, aVL, and/or anterior wall leads (V_{1-6}). • Infarction of the Inferior wall is caused by obstruction of the LCX or RCA or their branches, and ECG changes are seen in leads II, III, and aVF.	• Troponins T and I (TnT and TnI normally are not found in the circulation; however, after acute MI both are detectable within 1 to 2 to 4 hours, with levels peaking at 48 hours and remaining elevated for 7 to 10 days. • CK-MB activity begins to rise within 2 to 4 hours of MI, peaks at 24 to 48 hours, and returns to normal within approximately 72 hours. • The serum lactate dehydrogenase (LDH) level rises above the reference range within 24 hours of MI, reaches a peak within 3-6 days, and returns to the baseline within 8-12 days. • Myoglobin is released more rapidly from infarcted myocardium than troponin and CK-MB and may be detected as early as two hours after an acute myocardial infarction.	Two-dimensional echocardiography • Identifies presence of right ventricular (RV) infarction, ventricular aneurysm, pericardial effusion, and LV thrombus Radionuclide imaging techniques • Myocardial perfusion imaging with [99mTc]- sestamibi, which is distributed in proportion to myocardial blood flow and concentrated by viable myocardium, reveals a defect ("cold spot") in most patients during the first few hours after development of a transmural infarct. **Cardiac MRI** • MI can be detected accurately with high-resolution cardiac MRI using a technique referred to as late enhancement. • A standard imaging agent (gadolinium) is administered and images are obtained after a 10-20 min delay.





Management of Unstable angina/NSTEMI

Reperfusion therapy has no role in management of Unstable angina

- **Admit**
- **Bed rest**
- **Continuous monitoring in specialized cardiac units**
- **Oxygen support**

Anti-ischemic treatment

Nitrates

- Contraindicated in hypotension, severe RV infarction/patient on PDE-5 inhibitors
- Route-sublingual or IV infusion
- NTG 0.4 mg every 15 minutes (Max dose-3 tablets) to control ischemic pain.
- 5-10 microgram/min infusion
- Side effects Headache and hypotension

Beta blockers

- Contraindicated in inferior wall MI (due to bradycardia and hypotension), asthma and copd (non-selective)
- Cardioselective-metaprolol 25-50 mg every 6 hours

Calcium channel blockers

- Nifedipine
- Avoided in cases of pulmonary edema and LV dysfunction

Morphine sulphate

- Contraindicated in cases of hypotension
- 5mg IV stat followed by 2 mg IV every 10 minutes

Anti-thrombotic therapy

Antiplatelet therapy

ORAL

- Aspirin 325 mg stat f/b 75-100mg/day
- Clopidogrel 300-600 mg stat f/b 75mg/day

PARENTERAL

- Abciximab
- Eptifibatide
- Tirofiban
- Cangrelor

Anticoagulant therapy

- Unfractionated heparin (UFH) bolus (70-100 u/kg) f/b infusion of 12-15 U/kg/hr
- Enoxaparin SC
- Fondaparinux SC 2.5mg
- Bivalirudin

At first contact start DUAL ANTIPLATELET THERAPY (Aspirin+Clopidogrel) and Anticoagulation (Enoxaparin or fondaparinux or Bivalirudin)

During hospitalisation-if patient is on medical treatment continue with antiplatelet and anticoagulant treatment is continued.

Invasive strategy is followed in patients with high risk factors, ST segment deviation, and /or positive biomarkers

- Cardiac catheterization followed by PCI or CABG based on coronary anatomy.

Long term management

Aspirin 75-100mg/day-Indefinite

Clopidogrel 75mg/day for next 12 months

Management of STEMI

ST segment elevation indicates transmural infarction. Aim is to limit the size of infarct. **Reperfusion therapy is the key.**

General management

- Assess vitals and maintain hemodynamics
- Establish IV lines
- Cardiac monitoring-admit in Cardiac ICU
- Measure spo2 and maintain oxygen saturations
- Morphine to decrease anxiety (5mg IV stat followed by 2 mg IV every 10 minutes). Morphine is contraindicated in inferior wall MI (Alternatives inj.pethidine or inj.phenergan)
- Immediately start DAPT (Dual antiplatelet therapy)
    - Aspirin 325 mg stat f/b 75-100mg/day-Indefinite
    - Clopidogrel 300-600 mg stat f/b 75mg/day for next 12 months
- Sublingual nitrates-NTG 0.4 mg every 15 minutes (Max dose-3 tablets) to control ischemic pain.

Reperfusion therapy (Should be started as immediately as possible)

- **If the Patient is presented to PCI capable hospital**
    - Send to Cath lab immediately for primary PCI (percutaneous intervention)-stent insertion.
    - First medical contact to device time should be less than or equal to 90 minutes.
    - Diagnostic angiogram done.
        - Medical therapy in case of Prinzmetal angina
        - PCI if coronary anatomy allows
        - CABG if coronary anatomy is not supportive

- **If the patient presents to PCI incapable hospital**
    - Anticipated DIDO time (Door in Door out time) < or = 30 minutes and FMC-Device time is < or = 120 minutes-**Diagnostic angiogram**
    - **Anticipated FMC-Device time > 120 minutes-Start fibrinolytic therapy with in 30 minutes**
        - Streptokinase is replaced with tPA in view of allergic manifestations
        - Look for any contraindications
        - **tPA dosage**-15 mg bolus followed by 50 mg IV over the next 30 minutes followed by 35 mg over the next 60 minutes
    - After failed thrombolysis RESCUE PCI can be considered.
    - In case of recurrence URGENT PCI can be considered.
    - If thrombolysis is successful, after recovery ELECTIVE PCI or CABG are considered.

Pharmacological strategies available for the post-myocardial infarction management (Management of patients who survive acute MI)

Management of complications

- *Severe hypotension due to heart failure*-Vasopressors
- *Pulmonary edema*-Diuretics
- *Arrhythmias-VF (most common)*-Antiarrhythmic/Defibrillations/Cardiac pacing
- *Dressler syndrome (Post-myocardial infarction syndrome)* – Autoimmune inflammatory reaction- High dose aspirin/Corticosteroids

Long term management (CARDIAC REHABILITATION)

- Aspirin (75-100mg/day) to be continued indefinite and Clopidogrel (75mg/day) for one year
- Inj. Heparin (UFH) to prevent *Thromboembolism*
- Beta-blockers (Metoprolol 50 mg oral BD) *to reduce anxiety and cardiac work load*
- ACE inhibitors to prevent *cardiac remodeling and further complications*
- Statins for *control of hyperlipidemia*
- **Supportive treatment**
    - Reassurance
    - Complete bed rest
    - Deep breathing exercises
    - Walking
    - Avoid straining (Laxatives to relieve constipation)

Prinzmetal's variant angina (PVA)

It is a syndrome of severe ischemic pain that usually occurs at rest and is associated with transient ST-segment elevation.
It is caused by focal spasm of an epicardial coronary artery with resultant transmural ischemia and abnormalities in left ventricular function that may lead to acute MI, ventricular tachycardia or fibrillation, and sudden cardiac death.

Patients with PVA are generally younger and, with the exception of cigarette smoking, have fewer coronary risk factors than do patients with Other forms of ACS

Coronary angiography demonstrates transient coronary spasm as the diagnostic hallmark of PVA.

Treatment
Nitrates and calcium channel blockers are the main therapeutic agents.
Aspirin may actually increase the severity of ischemic episodes, possibly as a result of the sensitivity of coronary tone to modest changes in the synthesis of prostacyclin.

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NOTES EDITION 1.0

PREVENTIVE MEDICINE

SAMPLE NOTES

SYLLABUS (Page no.)

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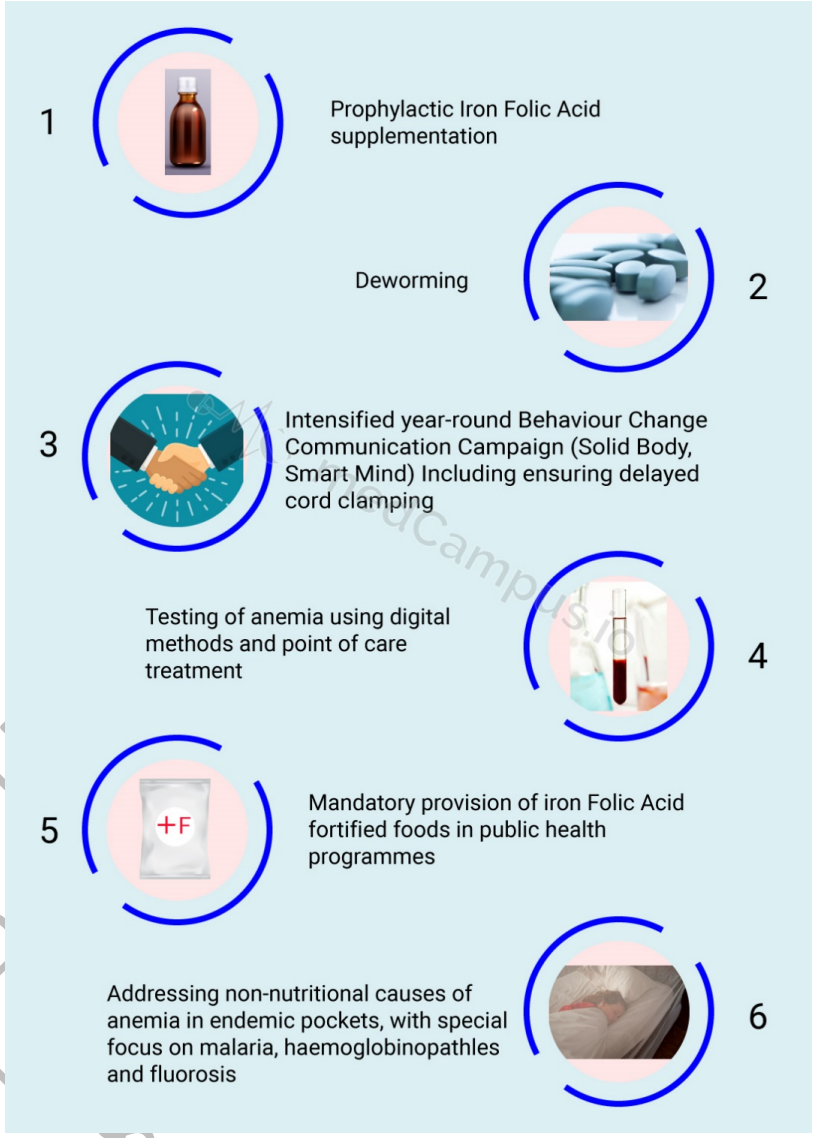
The Anemia Mukt Bharat- intensified Iron-plus Initiative (POSHAN ABHIYAN)

The Anemia Mukt Bharat- intensified Iron-plus Initiative aims to strengthen the existing mechanisms and foster newer strategies for tackling anemia.

It focusses on **six target beneficiary groups, through six interventions and six institutional mechanisms** to achieve the envisaged target under the POSHAN Abhiyan.

The reduction of anemia is one of the important objectives of the POSHAN Abhiyaan launched in March 2018.

Complying with the targets of POSHAN Abhiyaan and National Nutrition Strategy set by NITI Aayog, the Anemia Mukt Bharat strategy has been designed to reduce prevalence of anemia by 3 percentage points per year among children, adolescents and women in the reproductive age group (15–49 years), between the year 2018 and 2022.



SIX INTERVENTIONS

The Anemia Mukt Bharat strategy is a universal strategy and will focus on the following interventions:

- **Prophylactic Iron and Folic Acid supplementation**-Given to children, adolescents, women of reproductive age and pregnant women.
- **Deworming**-Albendazole 400 mg is used.

Intensified year-round Behaviour Change Communication Campaign (Solid Body, Smart Mind) focusing on four key behaviours

1. Improving compliance to Iron Folic Acid supplementation and deworming
2. Appropriate infant and young child feeding practices
3. Increase in intake of iron-rich food through diet diversity/quantity/frequency and/or fortified foods with focus on harnessing locally available resources
4. Ensuring delayed cord clamping after delivery (by 3 minutes) in health facilities

- **Testing and treatment of anemia**-using digital methods and point of care treatment, with special focus on pregnant women and school-going adolescents
- **Mandatory provision of Iron and Folic Acid fortified foods** in government-funded public health programmes.
- **Intensifying awareness, screening and treatment of non-nutritional causes of anemia** in endemic pockets, with special focus on malaria, haemoglobinopathies.

ANEMIA MUKT BHARAT
6×6×6 STRATEGY

- Children, 6–59 months of age
- Adolescent girls and boys (10–19 years of age)
- Pregnant women
- Children, 5–9 years of age
- Women of reproductive age (20–24 years of age)
- Lactating mothers (of 0–6 months child)

6 Beneficiaries

6 Institutional Mechanisms

- National Anemia Mukt Bharat Unit
- Intra Ministerial Coordinaion
- Strengthening Supply Chain and Logistics
- Convergence with Other Ministries
- National Centre of Excellence and Advanced Research on Anemia Control
- Anemia Mukt Bharat Dashboard and Digital Portal – One Stop Shop for Anemia

POSHAN Abhiyaan (National Nutrition Mission), targets to reduce Stunting, undernutrition, Anemia (among young children, women and adolescent girls) and reduce low birth weight by 2%, 2%, 3% and 2% per annum respectively. Recently, the Ministry for Women and Child Development inaugurated **Poshan 2.0** and urged all Aspirational Districts to establish a **Poshan Vatika (nutrition garden)** during the Nutrition Month **(Poshan Mah)** from 1st September.

POSHAN 2.0

It is an umbrella scheme covering the Integrated Child Development Services (ICDS) (Anganwadi Services, Poshan Abhiyan, Scheme For Adolescent Girls, National Creche Scheme).

It was announced in Union Budget 2021-22 by merging supplementary nutrition programmes and the POSHAN Abhiyaan.

It was launched to strengthen nutritional content, delivery, outreach and outcome, with renewed focus on developing practices that nurture health, wellness and immunity to disease and malnutrition in the country.

Poshan Maah:

Month of September is celebrated as POSHAN Maah since 2018 to improve nutritional outcomes for children, adolescent girls, pregnant women, and lactating mothers.It includes a month-long activities focussed on antenatal care, optimal breastfeeding, Anaemia, growth monitoring, girls education, diet, right age of marriage, hygiene and sanitation and eating healthy (Food Fortification).

The activities focus on Social and Behavioural Change Communication (SBCC) and are based on Jan Andolan Guidelines.

SBCC is the strategic use of communication approaches to promote changes in knowledge, attitudes, norms, beliefs and behaviours.

Poshan Vatika:

It's main objective is to ensure supply of nutrition through organically home grown vegetables and fruits simultaneously ensuring that the soil must also remain healthy.

Plantation drives for Poshan Vatikas would be taken up by all the stakeholders in the space available at anganwadis, school premises and gram panchayats.

About
Poshan Tracker App

POSHAN TRACKER:

The 'Poshan Tracker' is a mobile based application rolled out by the Ministry of Women and Child Development, Government of India on 1st March 2021 through National e-Governance Division (NeGD), Poshan Tracker is an important governance tool. Technology under Poshan Tracker is being leveraged for dynamic identification of stunting, wasting, under-weight prevalence among children and last mile tracking of nutrition service delivery.

- Job-aid to the Anganwadi worker for efficient delivery of services along with reflection of their efforts.
- Critical and beneficiary-centric service delivery Application under POSHAN Abhiyaan
- Promote real time data with analytics.

Pre-school Education

Poshan Tracker helps in effective management of pre-school education activities. In remote and socio-economically backward areas, it brings young children aged 3-6 years together at the Anganwadi Centre, where different activities relating to physical, cognitive, social, emotional, creative development of children are facilitated by the Anganwadi Worker.

Real time recording and Monitoring

The Poshan Tracker enables real-time monitoring and tracking of all AWCs and beneficiaries on defined indicators. The objective is to provide a 360-degree view of the activities of the Anganwadi Centre (AWC), service deliveries of Anganwadi Workers (AWWs) and complete beneficiary management.

Growth Measurement

Children of different age groups are monitored on monthly basis by AWWs. Nutritional indicators like Stunting, Wasting and Underweight prevalence are measured as per WHO standards. Regular growth monitoring helps AWWs to provide relevant and timely assistance to children.

Home Visit Alerts

The Poshan Tracker generates home visit alerts, notifying about scheduled visits to the critical last-mile beneficiaries. These alerts help ensure timely and consistent follow-ups and assessments, enabling efficient monitoring and implementation of nutrition and health counselling. This management information system helps generating and documenting positive impact.

Services Delivered

Tracking of services like Take Home Ration (THR) and Hot Cooked Meal (HCM), vaccination for infants and pregnant women, at a click of a button. The dashboard helps to impart transparency on services delivered to the end beneficiaries.

MILLETS

India at the helm of the G20 presidency, is spreading the millet revolution. (International year of millets 2023)

The term "millet" is used for smaller grains which are ground and eaten without having the outer layer removed

Major millets include sorghum (Jowar) , Bajra (pearl millet) , and Ragi (finger millet).

Minor millets/Pseudocereals include little millet, foxtail millet, proso millet, barnyard millet, and kodo millet.

Nutritional value of Millets (Values per 100mg)

	Jowar	Bajra	Ragi
Protein (g)	10.4	11.6	7.3
Fat (g)	1.9	5.0	1.3
Carbohydrate (g)	72.6	67.5	72.0
Minerals (g)	1.6	2.3	2.7
Calcium (mg)	25.0	42.0	344.0
Iron (mg)	4.1	8	3.9
Thiamine (mg)	0.3	0.3	0.2
Riboflavin (mg)	1.3	0.25	0.18
Niacin (mg)	3.1	2.3	2.3
Energy (Kcal)	349	361	328

Millet	**Health benefits**
Jowar	Helps to control heart problems, obesity and arthritis. Rich in potassium, phosphorus, and calcium with sufficient amounts of iron, zinc and sodium. Being targeted as a means to reduce malnutrition globally. Contains high content of leucine-consumption of high quantities leads to pellagra.
Bajra	Contains significant amounts of B-group vitamins and minerals such as calcium and iron.
Ragi	Cheapest millet, rich in calcium. Ideal for diabetics
Foxtail millet (Kangani/Tangun)	Rich in omega-3 and 6 fatty acids, it rejuvenates nerves and helps to cure epilepsy. Steadily releases glucose with out affecting the metabolism of the body, hence the prevalence of diabetes is reduced. Good source of magnesium. Rich in dietary fiber.
Barnyard millet (Sanva)	Appropriate food for patients intolerant to gluten.

Millets survive in less moisture condition. They have shorter cultivation cycles. Millets are drought resistant.
Millets are called superfoods.

National Framework for Malaria Elimination in INDIA

The NFME 2016–2030 was developed in close alignment with the Global Technical Strategy for Malaria 2016–2030, Action and Investment to defeat Malaria 2016–2030 and the Asia Pacific Leaders Malaria Alliance Malaria Elimination Roadmap.

VISION
Eliminate malaria nationally and contribute to improved health, quality of life and alleviation of poverty.

GOALS
In line with the WHO Global Technical Strategy for Malaria 2016–2030 (GTS) and the Asia Pacific Leaders Malaria Alliance Malaria Elimination Roadmap, the goals of the National Framework for Malaria Elimination in India 2016–2030 are:
• Eliminate malaria (zero indigenous cases) throughout the entire country by 2030; and
• Maintain malaria–free status in areas where malaria transmission has been interrupted and prevent re-introduction of malaria.

OBJECTIVES
The Framework has four objectives:
1.Eliminate malaria from all 26 low (Category 1) and moderate (Category 2) transmission states/union territories (UTs) by 2022;
2.Reduce the incidence of malaria to less than 1 case per 1000 population per year in all states and UTs and their districts by 2024;
3.Interrupt indigenous transmission of malaria throughout the entire country, including all high transmission states and union territories (UTs) (Category 3) by 2027; and
4.Prevent the re-establishment of local transmission of malaria in areas where it has been eliminated and maintain national malaria-free status by 2030 and beyond.

STRATEGIC APPROACHES

1. Programme phasing

• Malaria elimination in India will be carried out in a phased manner because various parts of the country differ in their malaria endemicity due to differences in their eco-epidemiological settings, socioeconomic conditions, health system development and malaria control accomplishments.

• Malaria incidence in high transmission areas (Category 3) must be lowered first before it is possible and rational to investigate each case.

• States/UTs will be subdivided into four categories with annual parasite incidence (API) as the primary criteria, and the annual blood examination rate (ABER) and slide positivity rate (SPR) as secondary criteria.

• Category-specific milestones and targets will be set up and strategies implemented subsequently.

Category of districts	**Definition**
Category 0: Prevention of re-establishment phase	States/UTs with zero indigenous cases of malaria.
Category 1: Elimination phase	States/UTs (15) including their districts reporting an API of less than 1 case per 1000 population at risk .
Category 2: Pre-elimination phase	States/UTs (11) with an API of less than 1 case per 1000 population at risk, but some of their districts are reporting an API of 1 case per 1000 population at risk or above.
Category 3: Intensified control phase	States/UTs (10) with an API of 1 case per 1000 population at risk or above.

2. District as the unit of planning and implementation

• Apart from the category to which they belong, each state/UT will be allowed to further classify their districts so that even if a state/UT is not yet in the elimination phase, but has some districts with an API below 1 case per 1000 population at risk, those may be considered eligible for initiating elimination phase activities provided they meet the secondary criteria.

• In addition, states/UTs may also sub-classify districts into community health centres, community health centres into primary health centres, primary health centres into sub-centres, and sub-centres into villages for localized planning and implementation.

3. Focus on high transmission areas

• The majority of malaria is being reported from states in the eastern, central and north-eastern part of the country, such as Odisha, Chhattisgarh, Jharkhand, Madhya Pradesh, Maharashtra, Tripura and Meghalaya.

• Most of these states are characterized by widespread hilly, tribal, forested and conflict-affected areas which are pockets of high malaria transmission.

• An aggressive scaling up of existing interventions, intensification of all malaria control activities and innovative strategies and partnerships will be carried out in these high endemic pockets to rapidly reduce malaria morbidity and mortality.

4. Special strategy for P. vivax elimination

• According to the World Malaria Report 2015, more than 80% of the global P. vivax burden is contributed by 3 countries including India .

• This serious challenge to malaria elimination efforts within the country will require special measures to be undertaken, such as good quality microscopy to detect all P. vivax infections, operational research to estimate prevalence of G6PD deficiency in the population, appropriate vector control measures and ensuring good compliance to 14-day radical treatment with primaquine in affected individuals.

• These measures are in line with the WHO Control and Elimination of Plasmodium vivax Malaria – Technical Brief.

MILESTONES AND TARGETS

By end of 2016

•All states/UTs have included malaria elimination in their broader health policies and planning frameworks.

By 2020

•Transmission of malaria interrupted and zero indigenous cases and deaths due to malaria attained in all 15 states/UTs under Category 1 (elimination phase) in 2014 (base year).
•All 11 states/UTs under Category 2 (pre-elimination phase) in 2014 enter into Category 1 (elimination phase).
•Five states/UTs under Category 3 (intensified control phase) in 2014 enter into Category 2 (pre-elimination phase).
•Five states/UTs under Category 3 (intensified control phase) in 2014 reduce malaria transmission but continue to remain in Category 3.
•An estimated reduction in malaria of 15–20% at the national level compared with 2014.
•Additionally, progressive states with strong health systems such as Gujarat, Maharashtra and Karnataka may implement accelerated malaria elimination programmes to achieve interruption of transmission and demonstrate early elimination followed by sustenance of zero indigenous cases.

By 2022

•Transmission of malaria interrupted and zero indigenous cases and deaths due to malaria attained in all 26 states/UTs that were under Categories 1 and 2 in 2014.
•Five states/UTs which were under Category 3 (intensified control phase) in 2014 enter into elimination phase.
•Five states/UTs which were under Category 3 (intensified control phase) in 2014 enter into pre-elimination phase.
•An estimated reduction in malaria of 30–35% at the national level compared with 2014.

By 2024

•All states/UTs and their respective districts reduce API to less than 1 case per 1000 population at risk and sustain zero deaths due to malaria while maintaining full functional malaria surveillance to track, investigate and respond to each case throughout the country.
•Transmission of malaria interrupted and zero indigenous cases and deaths due to malaria attained in all 31 states/UTs.
•Five states/UTs which were under Category 3 (intensified control phase) in 2014 enter into elimination phase.

By 2027

•The indigenous transmission of malaria in India interrupted.

By 2030

•The re-establishment of local transmission prevented in areas where malaria has been eliminated.
•The malaria-free status maintained throughout the nation.

Steps taken by Indian government to eliminate KALA – AZAR from the country by 2023

"India is committed to eliminating Kala Azar from the country by 2023.

Elimination is defined as reducing the annual incidence of Kala-azar to less than 1 case per 10,000 population at the sub-district (block PHCs).

PROBLEM STATEMENT

- About 90% of global cases of kala Azar were reported from eight countries: Brazil, Eritrea, Ethiopia, India, Kenya, Somalia, South Sudan and Sudan in 2021,with India contributing 11.5% of total cases reported globally.
- 632 (99.8%) endemic blocks have already achieved elimination status (<1 case/10,000).
- Only one block (Littipara) of Pakur district, Jharkhand is in the endemic category (1.23 case/10,000 population).

Government has taken several steps towards elimination of Kala-Azar by 2023.

National roadmap for Kala-azar elimination has been circulated to states with goals, objectives, strategies, timelines with activities and functions at appropriate level. This document has been developed for focused intervention at national, state, district and sub-district and village levels.

- Pucca houses through Pradhan Mantri Awas Yojana (PMAY)
- Rural electrification
- **Timely testing, treatment**
  Treatment of Kala-azar patient with single day single dose Liposomal Amphotericin B (AmBisome) injection has improved treatment compliance.
  Continuous free supply of AmBisome is ensured by WHO.
  Regular supply of diagnostic kit (rapid diagnostic test) and drugs in states
- **Periodic high-level review**
  On monthly basis Kala-azar elimination is being reviewed by Prime Minister's Office (PMO) as well as higher officials of Ministry of Health and Family Welfare.
- **Incentives through award distribution for states/districts/blocks**
- **Incentives to Patients and Health workers**
  An amount of 500 Rs. is given to each Kala-azar patient, and Rs. 2,000 is given in case of Post-Kala-azar Dermal Leishmaniasis (PKDL) from Government of India to compensate the loss of wages.
  Rs. 300 is given to the ASHA or health volunteer for bringing Kala- azar suspected case to health facility as well as to ensure complete treatment.
  Moreover, ASHA is also being paid Rs. 200 during indoor residual spray for social mobilization and community acceptance to allow spray in their rooms.
- Vector management
- Health education

SUPPORT

Government of India is directly working with development partners such as Bill Melinda Gates Foundation (BMGF), KalaCORE Consortium, Rajendra Memorial Research Institute (RMRI), National Centre for Disease Control (NCDC) and World Health Organization (WHO) to eliminate Kala Azar from India.

ABOUT NTDs

NTDs (Neglected Tropical Diseases) are now recognized in the SDGs for health with target 3.3 articulated as "the end of NTDs" by 2030, to be measured by "number of people requiring interventions against" these diseases among which leishmaniasis is included.

World Health Organization (WHO) launched an implementation roadmap for accelerating work on NTDs during a gathering in London, resulting in the London Declaration on NTDs. The "Uniting to Combat NTDs – Ending the Neglect and Reaching 2020 Goals" campaign generated an unprecedented renewed focus on these diseases.

This roadmap targets 10 diseases, including visceral leishmaniasis (VL), for elimination by year 2020 and the WHO has been rallying regional processes for strategic and integrated activities towards the goal with notable progress to date.

Current LF overview in INDIA
333 endemic districts in 2023

136 districts cleared TAS 1 & in Post MDA surveillance
176 districts reported more than 1% microfilaria (mF) rate and are under MDA

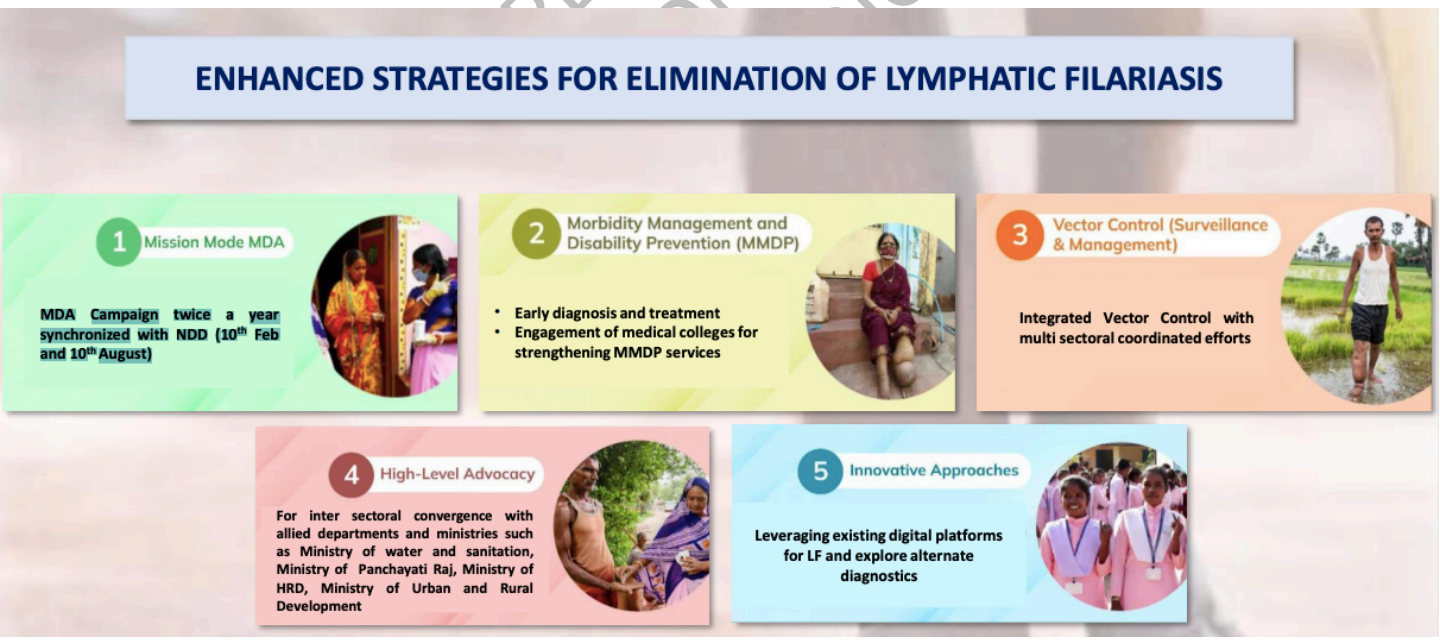
75% of MDA districts are from 5 states of Bihar, Jharkhand, UP, Odisha and chhattisgarh.
5.5 lakhs Lymphedema cases till 2022 (provisional)
1.5 lakhs Hydrocele cases till 2022 (provisional)

Building India's Roadmap to Eliminate Lymphatic Filariasis by 2027

For India, LF is not a Neglected Disease, but a Priority Disease for Elimination

National Center for Vector Borne Disease Control (NCVBDC) in collaboration with Global Health Strategies (GHS) and its partners organized a National Symposium on Building India's Roadmap to Eliminate Lymphatic Filariasis by 2027 - three years ahead of the global target, through the five-pronged roadmap.

A renewed five-pronged strategy for elimination of LF is as follows:
1. Multi-drug administration (MDA) Campaign twice a year synchronized with National Deworming Day (10th Feb and 10th August)
2. Early diagnosis and treatment; engagement of medical colleges for strengthening Morbidity management and disability (MMDP) services
3. Integrated Vector Control with multi sectoral coordinated efforts
4. For inter sectoral convergence with allied departments and ministries
5. Leveraging existing digital platforms for LF and exploring alternate diagnostics



ABOUT MDA : MASS DRUG ADMINISTRATION
- Mass Drug Administration of single dose medication for 5 years or more to the eligible population (except pregnant women, children below 2 years of age and seriously ill persons) to interrupt transmission of the disease.
- MDA started as mass campaign from 2004. Initially with single dose of DEC only.
- In the year of 2007 with DEC + Albendazole co-administration
- Form 2018 Triple Drug Therapy (IDA) i.e. **DEC + Albendazole + Ivermectin** is launched initially in five endemic districts. Since elimination target is approaching first all the left out districts which are yet to achieve elimination will be brought under IDA.

Joint Monitoring Mission LF elimination (JMM)

Joint Monitoring Mission for Elimination of Lymphatic Filariasis Program, India was conducted from November 14-25, 2022.
As part of JMM, expert teams were constituted to visit the States of Assam, Bihar, Jharkhand, Telangana, and UP.

Each State Team visited 2 districts, 2 PHCs, and 2 villages in each PHC during the period of field visits beginning from 16th to 22nd November 2022.
Each team consisted of Team Leaders and experts in the field of LF, entomology, social scientists, and procurement specialists in some states.
Teams were accompanied by the NCVBDC officers as National Observers.

A few key recommendations by the team were:

- States to timely initiate and plan the preparatory phase in good compliance with MDA.
- Strong political and administrative leadership needs to be leveraged for effective intersectoral and multistakeholder collaboration.
- Medical colleges to be effectively engaged in the LF programme.
- Rollout of **MMDP (Morbidity Management and Disability Prevention)** training in a cascaded manner across districts.
- Hydrocele backlog to be completed with help of medical colleges and district hospitals.
- **Linking of financial assistance schemes for disability due to lymphatic filariasis.** For example, Andhra Pradesh has approved financial assistance of Rs. 5000/- for bilateral elephantiasis Grade-IV patients, Telangana is providing a monthly pension of Rs. 2016/- to Grade-III onwards filariasis affected persons and Tamil Nadu is providing Rs. 1000/- for Grade-IV lymphatic filariasis cases.

LATEST UPDATE: New regimen for the treatment of MDR TB adopted by MOHFW

WHO recommended a new **6-month BPaLM regimen** for treating MDR TB in 2022. The regimen consisting of bedaquilline, pretomanid, linezolid and moxifloxacin has been now adopted by the MOHF W as a more effective option than the previous BpaL regimen.

Indications:

1. MDR/RR-TB or pre-XDR-TB (MDR/RR-TB and resistance to fluoroquinolones).
2. Those with confirmed pulmonary TB and extrapulmonary TB (except for TB involving CNS, osteoarticular, and miliary TB).
3. Patients >14 years of age.

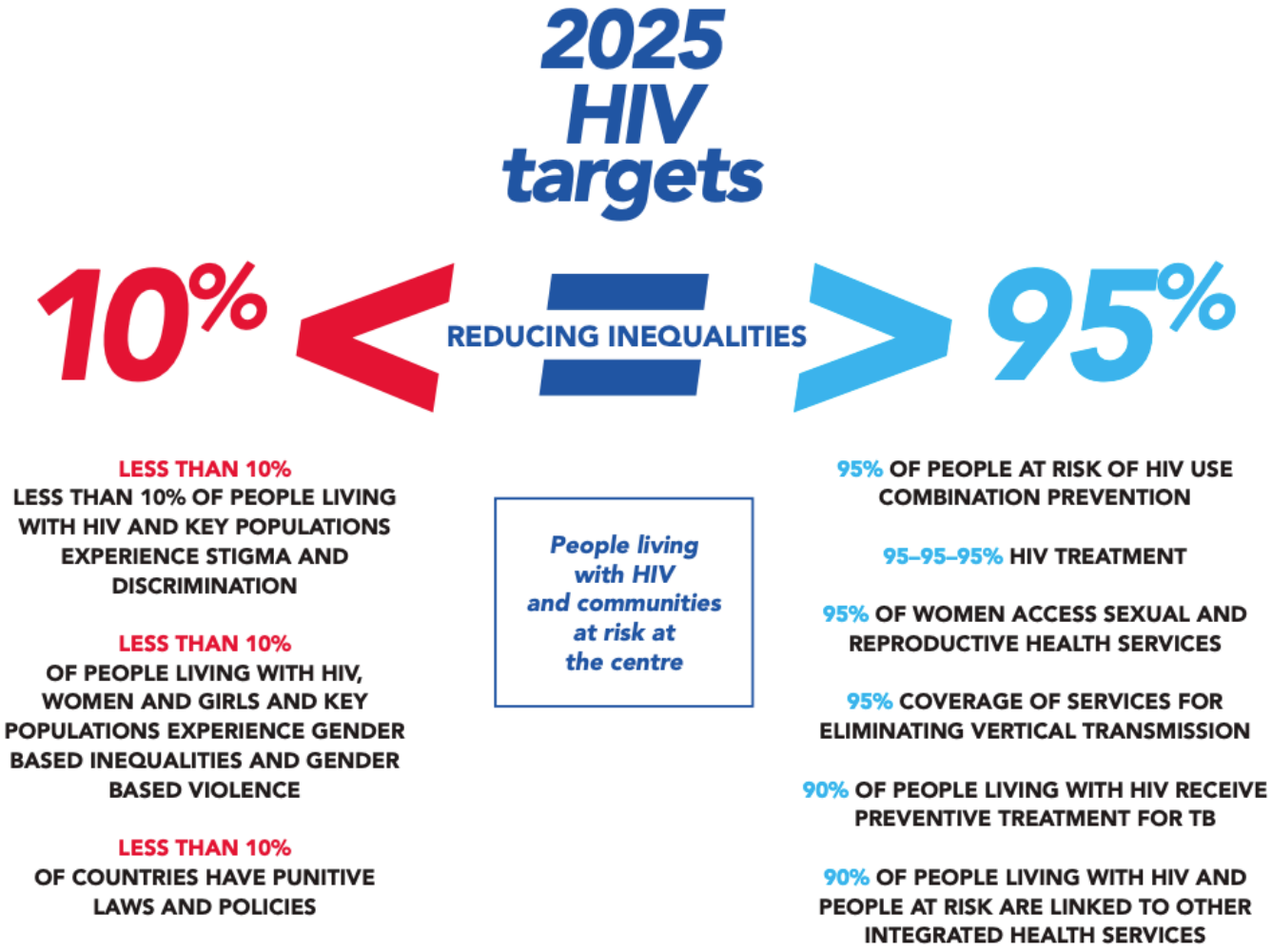
Dosage:

* Bedaquilline 100 mg tablet: 400 mg OD for 2 weeks f/b 200 mg 3 times/week OR 200 mg daily for 8 weeks f/b 100 mg daily
* Pretomanid 200 mg tablet OD
* Linezolid 600 mg tablet OD
* Moxifloxacin 400 mg tablet OD

Precautions:

* Linezolid should be continuously monitored due to risk of anemia, thrombocytopenia, optic neuritis, and peripheral neuropathy.
* Pretomanid has limited safety and hence the regime is contraindicated in pregnant and lactating women
* Individuals with CVS disease, elevated liver enzymes, or very low BMI

GLOBAL AIDS STRATEGY 2021-2026



National AIDS Control Programme V

National AIDS and STD Control Programme (NACP) Phase-V is a Central Sector Scheme fully funded by the Government of India with an outlay of Rs 15471.94 crore.

The NACP Phase-V will take the national AIDS and STD response till Financial Year 2025-26 towards the attainment of United Nations' Sustainable Development Goals 3.3 of ending the HIV/AIDS epidemic as a public health threat by 2030 through a comprehensive package of prevention, detection and treatment services.

The Phase-V builds upon the **gamechanger initiatives of the HIV/AIDS Prevention and Control Act (2017)**

- Test and Treat Policy
- Universal Viral Load Testing
- Mission Sampark
- Community-Based Screening
- Transition to Dolutegravir-based Treatment Regimen etc

And introduces newer strategies consolidating and augmenting the gains. This include

- Setting-up of Sampoorna Suraksha Kendras (SSK) for providing services through a single window model for those "at risk" for HIV and STI covering prevention-test-treat-care continuum. It includes a holistic set of services customized as per clients' needs, with strong linkages and referrals within and outside of health system.

Mission Sampark" - to bring back People Living with HIV who have left treatment after starting Anti Retroviral Treatment (ART).

National AIDS and STD Control Programme Phase-V (2021-2026) : Anchoring the national response towards ending the AIDS epidemic.

NACP Phase-V is a Central Sector Scheme, fully funded by the Government of India, with an outlay of Rs 15471.94 crore.

The NACP Phase-V aims to reduce annual new HIV infections and AIDS-related mortalities by 80% by 2025-26 from the baseline value of 2010.
The NACP Phase-V also aims to attain dual elimination of vertical transmission, elimination of HIV/AIDS related stigma while promoting universal access to quality STI/RTI services to at-risk and vulnerable populations.

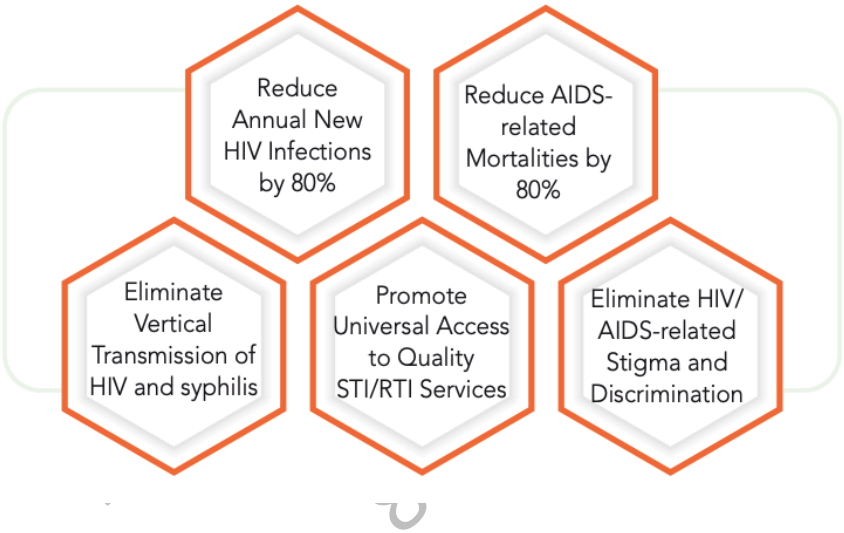
The specific objectives of the NACP Phase-V are as below:

a. **HIV/AIDS prevention and control**

i. 95% of people who are most at risk of acquiring HIV infection use comprehensive prevention
ii. 95% of HIV positive know their status, 95% of those who know their status are on treatment and 95% of those who are on treatment have suppressed viral load
iii. 95% of pregnant and breastfeeding women living with HIV have suppressed viral load towards attainment of elimination of vertical transmission of HIV iv. Less than 10% of people living with HIV and key populations experience stigma and discrimination

b. STI/RTI prevention and control

i. Universal access to quality STI/RTI services to at-risk and vulnerable populations
ii. Attainment of elimination of vertical transmission of syphilis



Sampoorna Suraksha Strategy

We have reached a critical junction which requires concerted efforts to attain 95-95-95 targets by 2025 and ultimately achieve the goal of ending the epidemic by 2030. To achieve this ambitious goal, intensified preventive efforts are needed to accelerate reduction in the new infections to further curb new HIV infections and avert AIDS-related deaths at much higher rates. In order to reach out the population "At Risk" for HIV and STIs with a comprehensive preventive services delivery package, NACO has envisaged a new form of "Immersion Learning Model" of Service delivery as 'Sampoorna Suraksha Strategy' under Global Fund Grant 2021-24 with the goal of arresting the spread of HIV by effectively targeting and tailoring interventions to prevent HIV transmission.

NACO has rolled out the Sampoorna Suraksha Strategy as an immersion learning model at 150 selected facilities in 150 districts of 20 States under Global Fund Grant 2021-24. These facilities are being re-modelled and branded as Sampoorna Surakhsa Kendra (SSK). The learnings from implementation will be used to modify, tweak the strategy and strengthen the outreach strategies and service delivery mechanism under NACP –V in future.

Key Features of PMSMA

- PMSMA is based on the premise — that if every pregnant woman in India is examined by a physician and appropriately investigated at least once during the PMSMA and then appropriately followed up — the process can result in reduction in the number of maternal and neonatal deaths in our country.
- Antenatal checkup services would be provided by OBGY specialists / Radiologist/physicians with support from private sector doctors to supplement the efforts of the government sector.
- A minimum package of antenatal care services (including investigations and drugs) would be provided to the beneficiaries on the 9th day of every month at identified public health facilities (PHCs/ CHCs, DHs/ urban health facilities etc) in both urban and rural areas in addition to the routine ANC at the health facility/ outreach.
- Using the principles of a single window system, it is envisaged that a minimum package of investigations (including one ultrasound during the 2nd trimester of pregnancy) and medicines such as IFA supplements, calcium supplements etc would be provided to all pregnant women attending the PMSMA clinics.
- While the target would reach out to all pregnant women, special efforts would be made to reach out to women who have not registered for ANC (left out/missed ANC) and also those who have registered but not availed ANC services (dropout) as well as High Risk pregnant women.
- OBGY specialists/ Radiologist/physicians from private sector would be encouraged to provide voluntary services at public health facilities where government sector practitioners are not available or inadequate.
- Pregnant women would be given Mother and Child Protection Cards and safe motherhood booklets.

- **One of the critical components of the Abhiyan is identification and follow up of high risk pregnancies. A sticker indicating the condition and risk factor of the pregnant women would be pasted onto MCP card for each visit:**
    - **Green Sticker- for women with no risk factor detected**
    - **Red Sticker – for women with a high-risk pregnancy**
    - **Blue Sticker – for pregnancy-induced hypertension**
    - **Yellow Sticker – for co-morbidities (like DM, STDs, hypothyroidism)**

- A National Portal for PMSMA and a Mobile application have been developed to facilitate the engagement of private/ voluntary sector.

- **'IPledgeFor9' Achievers Awards have been devised to celebrate individual and team achievements and acknowledge voluntary contributions for PMSMA in states and districts across India.**
-

2024

UPSC MEDICO

PYP-MODEL ANSWERS

UPSC Medical Sciences Optional 2024 QUESTION PAPER

PAPER I (Anatomy/Biochemistry/Physiology)
SECTION A

1.(a) Describe the radial nerve under the following headings :
(i) Origin and course
(i) Branches and muscles supplied
(iii) Applied aspects (10)
1.(b) Describe the development of ventricles of the heart. Add a note on Fallot's tetralogy. (10)
1.(c) Explain the 'Mucosal Block Theory' for iron absorption. Discuss briefly the complications of excess of iron deposition in the body. (10)
1.(d) Give the sequence of events at the neuromuscular junction during the transmission of a nerve impulse. Name the muscles most commonly affected by myasthenia gravis. (10)
1.(e) Describe the components of the nervous system which are concerned with 'conscious alert state' that makes perception possible. (10)

2.(a) A middle-aged female notices a lump in the midline of the neck. She notices it moves with swallowing. The surgeon gives a tentative diagnosis of goitre.
Describe the thyroid gland under the following headings :
(i) Gross anatomy and relations
(ii) Blood supply and lymphatic drainage
(iii) Surgical anatomy of thyroid gland (5+5+5=15)
2.(b) Explain why vitamin D can be considered to be a hormone'. Describe the role of vitamin D in calcium homeostasis. (10)
2.(c) Discuss the sources and Recommended Daily Allowance (RDA) of vitamin B12. Briefly discuss the absorption of vitamin B12 in the GIT and the clinical manifestations of the disorder of absorption of vitamin B12. (10)
2.(d) Define the Frank-Starling law. State the significance and causes of shift of Frank-Starling curve to right and left. (10)
2.(e) What is the role of baroreceptors and chemoreceptors in the regulation of blood pressure? (5)

3.(a) Describe the uterus under the following headings :
(i) Gross anatomy
(ii) Ligaments and supports
(iii) Relations of uterus
(iv) Blood supply
(v) Lymphatic drainage
(vi) Applied aspects (20)
3.(b) i. Which antibiotics and toxins inhibit protein synthesis in prokaryotes and eukaryotes? Briefly explain the mechanism of action of each of them. (10)
3.(b) ii. What are ribozymes? Explain briefly the role of any one ribozyme in protein synthesis. (5)
3.(c) i. Enumerate the major hormonal causes of dwarfism. Give their characteristic features. (10)
3.(c) ii. Describe the principal events during oogenesis in brief. (5)

4. (a) (i) Describe the facial nerve under the following headings :
1. Functional columns and nuclei of origin
2. Course and branches
3. Bell's palsy (4+4+2=10)
4.(a) (ii) Differentiate between indirect and direct inguinal hernia (5)
4. (b) (i) Define renal clearance. What key features should be present in a compound for it to be considered as a 'gold standard' for measurement of renal clearance? Explain why urea is not considered as a 'gold standard' for this. (10)
4. (b) (ii) Briefly describe the role of Restriction Fragment Length Polymorphism (RFLP) in DNA fingerprinting (10)
4. (c) (i) Give the physiological basis of anaemia in kidney and liver disease. (5)
4. (c) (ii) Describe the role of eosinophils in control of allergy reactions. (5)
4. (c) (iii) Describe the role of platelets in haemostasis. (5)

Paper I
Section B

5. (a) (i) Discuss the antiviral spectrum and therapeutic uses of acyclovir. (5)
5. (a) (ii) Doxorubicin is an antibiotic. Enumerate its role in cancer chemotherapy and its adverse effects. (5)
5. (b) What is hypersensitivity? Enumerate different hypersensitivity reactions along with examples. Define type I hypersensitivity reaction and write its role in health and disease. (10)
5. (c) Define cancer. Describe in detail the effects of cancer-related genes on cell growth. (2+8=10)
5. (d) A 50-year-old male presented with a history of chest pain, polyuria and polydipsia since last 5 years. Investigations showed HbAlc level of 12%, cardiac enzymes were normal, while urinalysis showed proteinuria.
(i) What in the most likely diagnosis?
(ii) Describe the microscopie findings.
(iii) What in the pathogenesis? (2+4+4=10)
5. (e) Define injury. Discuss the microscopic and histochemical methods which can determine the age of injury. (10)

6. (a) (i) Describe the microscopic features of breast cancer. Enumerate any five major prognostic factors. (5+5=10)
6. (a) (ii) Enumerate any five differences between primary tuberculosis and secondary tuberculosis. (10)
6. (b) (i) Explain why primaquine is used for radical cure of malaria. (5)
6. (b) (ii) Explain why albendazole is termed as broad-spectrum oral antihelminthic. (5)
6. (c) (i) What is candidiasis? What are its different presentations and etiological causes? Give the laboratory diagnosis of a case of invasive candidiasis. (2+4+4=10)
6. (c) (ii) What is shigellosis? What are its causative organisms and their modes of pathogenicity? Give the laboratory diagnosis of a case. (2+4+4=10)

7. (a) (i) Define death due to hanging. What are the probable causes of death in hanging? (10)
7. (a) (ii) What are the findings in a case of judicial hanging? (5)
7. (b) (i) A 55-year-old female presented with hamatemesis. On physical examination, she was afebrile and pale. No organomegaly was noted. Serological tests for hepatitis B were positive.
1. What is the most likely diagnosis?
2. Describe the microscopic findings.
3. What is the pathogenesis? (2+4+4=10)
7. (b) (ii) Describe the clinical features and microscopic findings in acute lymphoblastic leukaemia. (5+5=10)
7. (c) (i) State the role of diuretics in the management of hypertension. (5)
7. (c) (ii) Discuss how excess dose of paracetamol causes acute hepatocellular toxicity and how you will manage the condition. (5)
7. (c) (iii) Elaborate the advantages and disadvantages of Sodium-Glucose Cotransporter-2 (SGLT-2) inhibitors in the management of diabetes mellitus. (5)

8. (a) (i) What is enteric fever? What are its causative agents? Give a detailed presentation of a case according to the time of the disease and the respective tests used for diagnosis. (2+2+3+3=10)
8. (a) (ii) Draw a diagram of HIV virion depicting the various antigens and proteins. Give the serological pattern in an HIV infection according to the time of presentation. List the various diseases associated with AIDS. (4+4+2=10)
8. (b) (i) What is virtual autopsy? State its methodology. What are its merits? (10)
8. (b) (ii) Define brain death. Describe its medicolegal importance. (10)
8. (c) (i) Discuss why infliximab is considered as an immunosuppressant. Mention its therapeutic uses. (5)
8. (c) (ii) Describe briefly the pharmacological characteristics of natriuretic peptides and their clinical uses. (5)

UPSC Medical Sciences Optional 2024 QUESTION PAPER

PAPER II
SECTION A (General Medicine/Dermatology and Paediatrics)

1. (a) Discuss in short the role of a Chest X-ray in the diagnosis of pulmonary tuberculosis. (10)
1. (b) Describe the clinical features for diagnosing a case of Depression. (10)
1. (c) What are the key differences between Kwashiorkor and Marasmus?
Which is the easiest method which can help in the early detection of Protein Energy Malnutrition (PEM) in children? (10)
1. (d) During the initial phase of stabilization in a severe acute malnourished child, map out the dietary plan. State the type, amount and frequency of feed that the child requires and for how long that would be necessary. In this phase, what is the vitamin and mineral supplementation given? (10)
1.(e) In a confirmed case of scabies in an adult :
(i) What are the primary manifestations of the disease and what is the pattern of distribution of lesions on the body ?
(i) What are the complications seen in scabies? (5+5=10)

2. (a) (i) Discuss in short about the different modalities used in the diagnosis of Extra-Pulmonary Tuberculosis. (10)
2. (a) (ii) Describe the clinical features of malabsorption syndrome. (10)
2. (b) (i) Write in brief the ten steps of Baby-friendly Hospital Initiative (revised 2018). (10)
2. (b) (ii) Write the advantages of breast-feeding. (10)

2. (c) A young adult female develops asymptomatic depigmented chalky white macules and patches with no sign of inflammation over face and around body orifices.
(i) What is the diagnosis ?
(i) What are the associated findings seen in this disorder ?
(iii) How is this disorder classified ?
(iv) Describe the clinical course of the disease. (3+4+4+4=15)

3. (a) Describe the clinical features, diagnosis and treatment of Kala-azar. (20)
3. (b) What are the types of vaccines currently in use against Pneumococcus organisms? State the National Immunization Schedule for administering Pneumococcal Vaccine in infants. Enumerate the diseases that the Pneumococcal Vaccine can safeguard against. (15)
3. (c) A young female patient develops acute inflammatory papules and vesicles all over her scalp and tips of ears following repeated use of hair dye.
(i)What is the diagnosis?
(i) How can the diagnosis be confirmed ?
(iii) How will this condition be treated ? (5+5+5=15)

4. (a) A sixty-year-old male develops central chest pain while walking uphill. The pain is squeezing in character, radiating to left arm, that relieves on taking rest. Discuss in short about the evaluation and treatment of this case. (20)
4. (b) (i) Enumerate the causes of respiratory distress in a newborn. How would you differentiate between respiratory distress of respiratory origin and that of cardiac origin? (8)
4.b. (ii) Write the complications of cyanotic congenital heart diseases. (4)
4.b. (iii) How will you manage a one-year five-month old child presenting with severe respiratory distress with a history of cough and fever for 5 days? (8)
4. (c) (i) What is the meaning of the term lichenoid' ? (ii) Name the disease that is a prototype of lichenoid reaction. (iii) Describe the clinical features of the disease.
(3+3+4=10)

SECTION B (General Surgery/OBG/PSM)

5.a.(i) Enlist conditions having an increased risk of malignant disease in bone and cartilage.
5.a.(ii) Briefly mention classification of bone tumours. (5+5=10)
5. (b) A 55-year-old male patient underwent subtotal gastrectomy for carcinoma stomach. Briefly describe early and late complications of this procedure. (10)
5.c.(i) A 25-year-old infertile woman presents with menorrhagia. USG (Ultrasound) pelvis revealed multi-fibroid uterus, largest measuring 3 x 3 cm. Describe the evaluation and management of Fibroid Uterus in the above patient.
5.c.(ii) Describe recent classification of Abnormal Uterine Bleeding (AUB). Briefly discuss the endometrial pattern in various types of Abnormal Uterine Bleeding.
(5+5=10)
5. d. (i) A young newly married couple wants advice on contraception.
Describe the various methods of contraception which are suitable for them. (5)
5. d. (ii) Enlist the various methods of female sterilization and complications of tubectomy. (5)
5. (e) (i) Describe the 'yellow' category of biomedical waste in terms of types of waste, types of bags or containers to be used, and treatment and disposal options.
5. (e) (ii) Comment upon 'incineration' as a method of biomedical waste management. (5+5=10)

6. a. (i) A 22-year-old Unbooked Primigravida at 38 weeks of gestation presents to Emergency with labour pains. How would you evaluate the patient for obstetric triaging and further management of labour ?
6. a. (ii) Discuss the clinical features, diagnosis and management of Rupture Uterus following obstructed labour.
(10+10=20)
6. b. (i) Write the clinical features and diagnostic work-up in a case of carcinoma rectum. (5)
6. b. (li) Briefly mention Dukes' staging for this condition. (5)
6. b. (iii) Enumerate surgical options for this disease. (5)
6. (c) In the context of HIV/AIDS control and the National AIDS Control Programme in India, comment upon the following:
(i) 95-95-95 targets (3)
(ii) Categorization of districts (4)
(ii) TB-HIV coordination to reduce mortality (8)

7 (a) A 50-year-old male presented with a 3 cm nodule in the left lobe of thyroid gland with a hard left cervical lymph node. Fine Needle Aspiration Cytology (FNAC) from the thyroid nodule revealed orphan Annie-eyed nuclei.
(i) What is the diagnosis in this case? How can this condition be managed surgically ?
(ii) Enumerate different prognostic scoring systems for this condition.
(iii) What are the post-operative complications of total thyroidectomy ?
(8+5+7=20)
7 (b) (i) List the various sources of health information. (5)
7 (b) (ii) Describe the limitations of hospital records as a source of health information. (5)
7 (b) (iii) Write in brief the use of pictograms for presenting health information data. (5)
7.c. (i) What are the signs and symptoms of Pelvic Inflammatory Disease (PID) ?
(ii) What are the complications of PID?
(iii) How do you manage a 28-year-old woman, P1L1 with unilateral Tubo-ovarian abscess ? (5+5+5=15)

8. (a) (i) What are the different types of epidemiological studies? (ii) What are the possible sources of control in case-control studies? (iii) List the advantages of case-control studies as compared to cohort studies. (6+6+8=20)
8.b. Define Antenatal Care. What are its objectives? What is the schedule of antenatal clinic visits that a mother is expected to follow during the course of her pregnancy? What are the advantages and disadvantages of domiciliary midwifery service'? (15)
8.c.(i) Enumerate causes of hematuria in a 60-year-old male (5)
8.c.(ii) Briefly describe the management of carcinoma prostate in a 60 year old male (10)

MS OPTIONAL 2024 Paper
PAPER I SECTION – A
Anatomy

1.(a) Describe the radial nerve under the following headings :
(i) Origin and course
(i) Branches and muscles supplied
(iii) Applied aspects (10)

Describe the effects of damage to radial nerve at the level of deltoid tuberosity. (2014)

RADIAL NERVE
ORIGIN
The radial nerve originates from the posterior cord of the brachial plexus. It is one of the five branches of brachial plexus. The radial nerve is made up of fibers from the anterior rami of the fifth, sixth, seventh, and eighth cervical nerves, and the first thoracic nerve (C5-T1).

COURSE
The radial nerve's course is from the axilla to the hand:

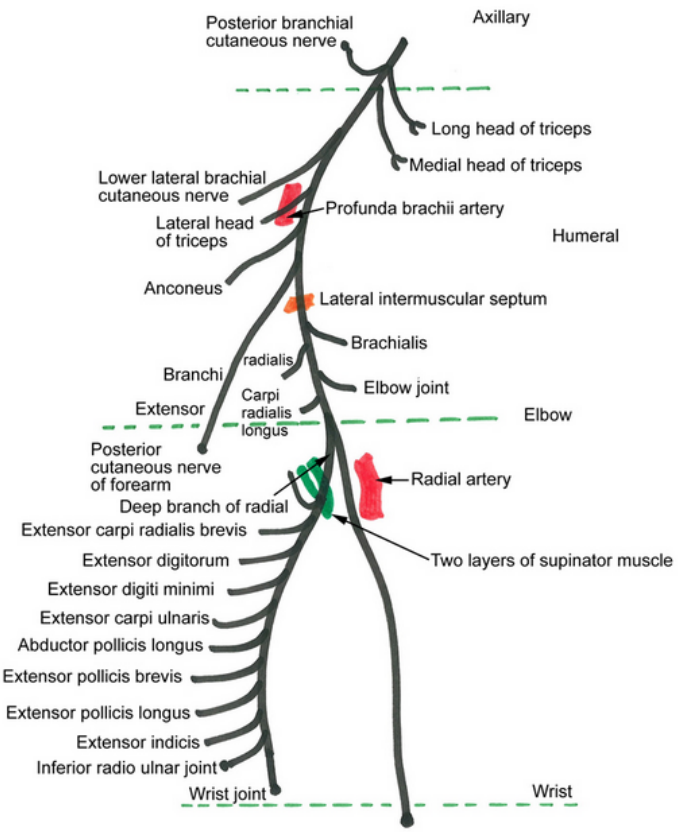
- **Axilla**
- The radial nerve exits the axilla behind the brachial artery. It runs behind the third part of the axillary artery, against the subscapularis, teres major, and latissimus dorsi muscles.
- **Arm**
- The radial nerve descends through the triangular interval, between the teres major, long head of triceps brachii, and humerus. It then enters the radial groove, where it runs between the medial and lateral heads of the triceps.
- **Cubital fossa**
- The radial nerve passes through the lateral intermuscular septum and enters the anterior compartment of the arm. It reaches the cubital fossa, where it's located between the brachioradialis and extensor carpi radialis longus muscles laterally, and the brachialis muscle medially.
- **Forearm**
- The radial nerve divides into superficial and deep branches in the forearm. The superficial branch provides sensory innervation to the back of the hand, including the web of skin between the thumb and index finger.

Clinical Anatomy

Lesions of the radial nerve at its origin from the posterior cord in the axilla may be caused by pressure from a long crutch (crutch palsy). Compression of the nerve against the humerus occurs if the arm is rested on a sharp edge such as the back of a chair ('Saturday night palsy').
Both these injuries cause weakness of brachioradialis with wasting and loss of the reflex.

There is both wrist and finger drop due to weakness of wrist and finger extensors, as well as weakness of extensor pollicis longus and abductor pollicis longus. There may be sensory impairment or paraesthesiae in the distribution of the superficial radial nerve. However, nerve overlap means that only a small area of anaesthesia usually occurs on the dorsum of the hand between the first and second metacarpal bones.

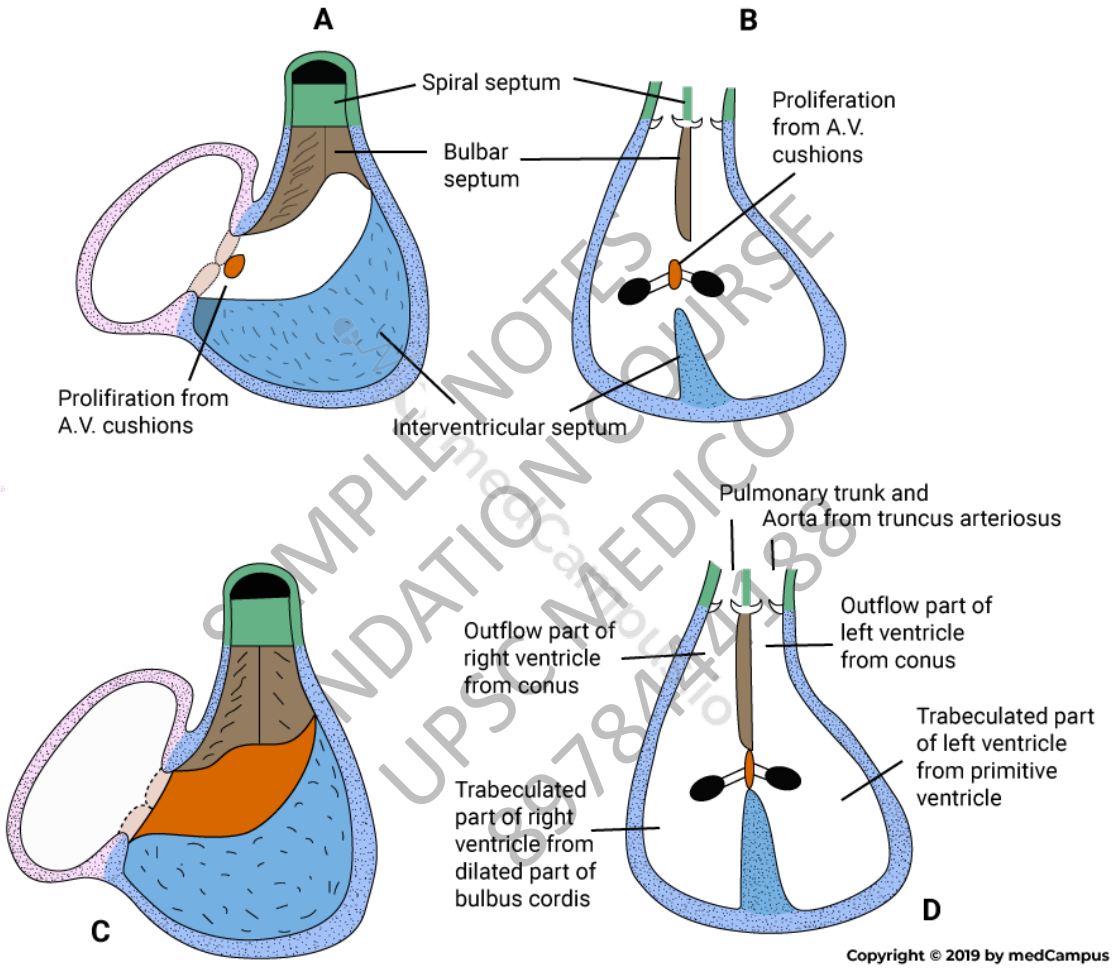
BRANCHES



1.(b) Describe the development of ventricles of the heart. Add a note on Fallot's tetralogy. (10)

Describe the development of heart chambers. Mention the common congenital anomalies and explain the developmental reasons for them. (2010)
Describe the development of the interventricular septum and write briefly about the congenital anomaly of the heart related to this structure. (2022)

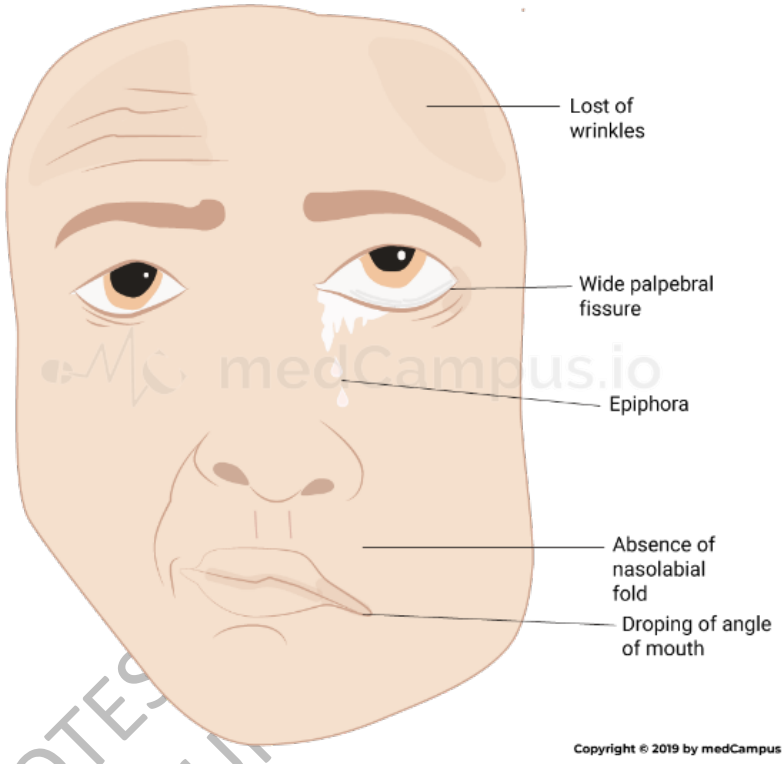
DEVELOPMENT OF VENTRICLES OF HEART



Developmental components of Ventricles
Right ventricle
a. Rough part—proximal portion of bulbus cordis
b. Smooth part—the conus cordis or middle portion of bulbus cordis.
Left ventricle
a. Rough part—whole of primitive ventricular chamber.
b. The conus cordis or the middle portion of bulbus cordis forms the smooth part.
Interventricular septum
a. Thick muscular in lower part by the two ventricles.
b. Thin membranous in upper part by fusion of inferior atrioventricular cushion and right and left conus swelling. Membranous part not only separates the two ventricles, but also separates right atrium from left ventricle.

Bells palsy

It is defined as idiopathic, peripheral facial paralysis or paresis of acute onset. Patient is unable to close his eye.
On attempting to close the eye, eyeball turns up and out Bell's phenomenon). Saliva dribbles from the angle of mouth.
Face becomes asymmetrical. Tears flow down from the eye. Pain in the ear may precede or accompany the nerve paralysis.



Treatment

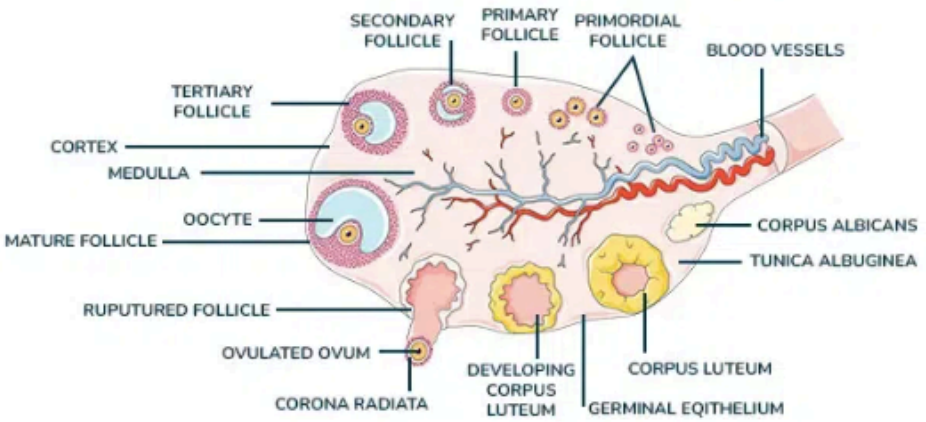
General	1. Reassurance 2. Relief of ear pain by analgesics 3. Care of the eye. Eye must be protected against exposure keratitis. 4. Physiotherapy or massage of the facial muscles.
Medical	**Prednisolone** is the drug of choice. If patient reports within 1 week, the adult dose of prednisolone is 1mg/kg/day divided into morning and evening doses for 5 days. Patient is seen on the 5th days. If paralysis is incomplete or is recovering, dose is repeated during the next 5 days. If paralysis remains complete, the same dose is continued for another 10 days and thereafter tapered in next 5 days. (Total of 20 days).
Surgical	**Nerve decompression** relieves pressure on the nerve fibres and thus improves the microcirculation of the nerve.

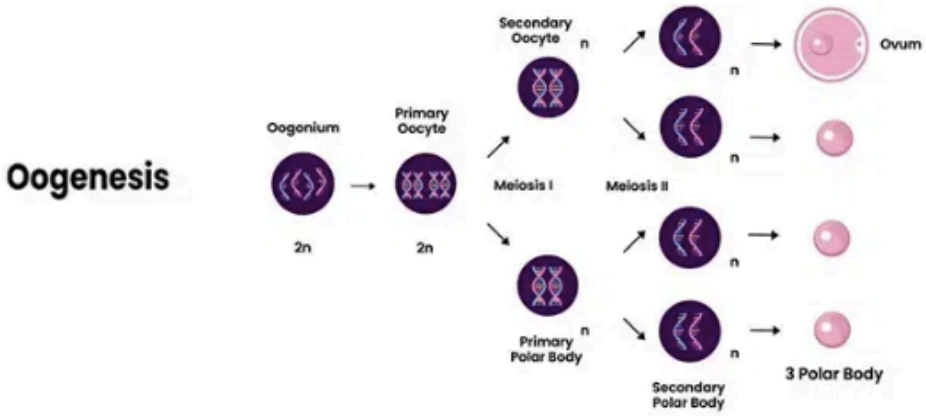
3.(c) ii. Describe the principal events during oogenesis in brief. (5)
What changes take place in the ovary, uterus, vagina and breast during a menstrual cycle (2003)?
Explain the changes which takes place during luteal phase of menstrual cycle? (2018)
Describe the key events occurring in the ovarian cycle with reference to their hormonal basis. (2021)

OOGENESIS

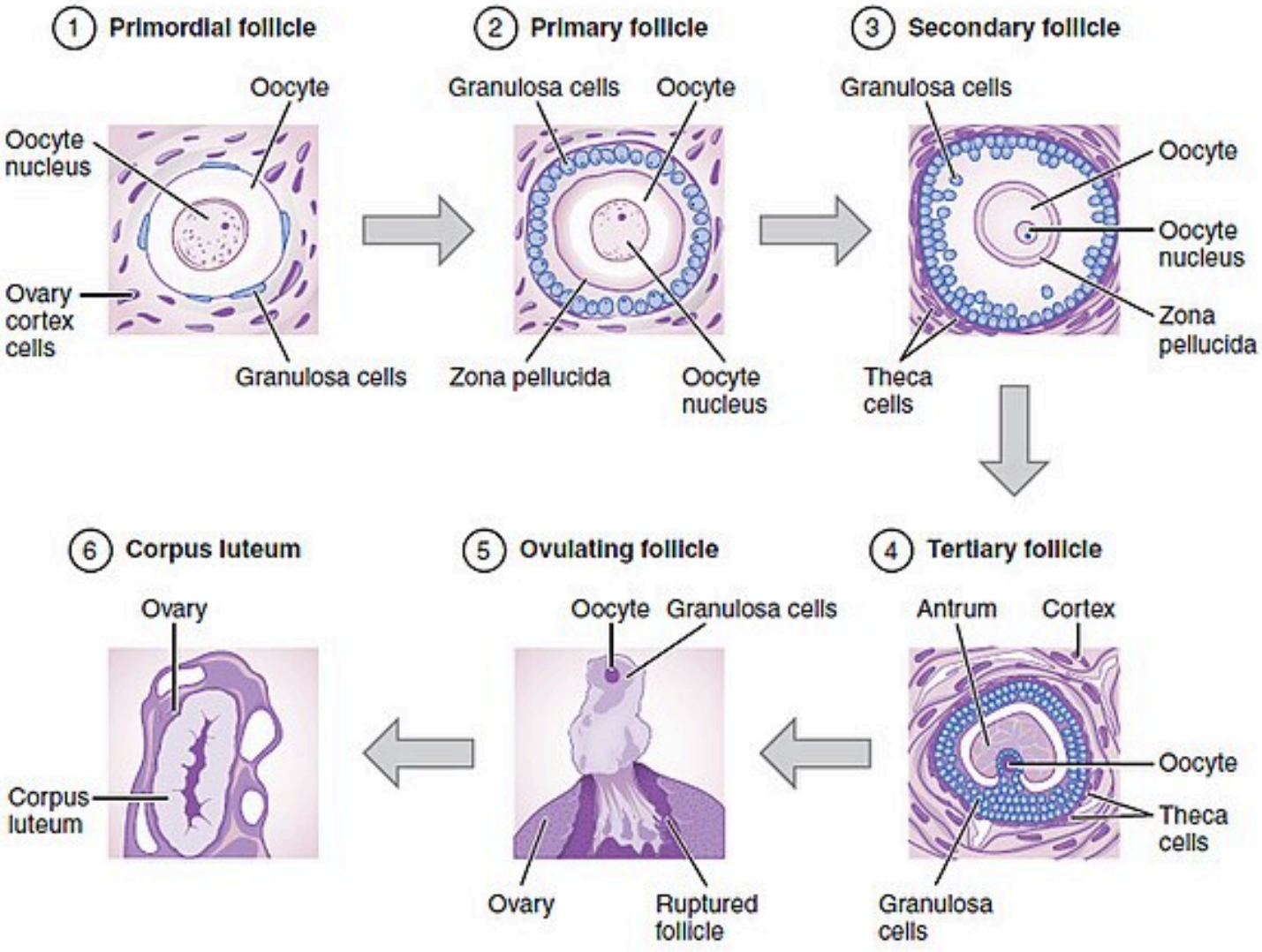
In the female, maturation from primitive germ cell (PGCs) to mature gamete, which is called **oogenesis, begins before birth.**

- In the female, PGCs form **oogonia.**
- After repeated mitotic divisions, some of these arrest in prophase of meiosis I to form **primary oocytes.**
- By the seventh month, many oogonia have become atretic, and only primary oocytes remain surrounded by a layer of **follicular cells** derived from the surface epithelium of the ovary. Together, they form the **primordial follicle.**
- At puberty , a pool of growing follicles is recruited and maintained from the finite supply of primordial follicles.
- Thus, every month, 15 to 20 follicles begin to grow, and as they mature, they pass through three stages:
  1) **primary** or **preantral**
  2) **vesicular or antral,** and
  3) **mature vesicular or graffian follicle.**
- The primary oocyte remains in prophase of the first meiotic division until the secondary follicle is mature. At this point, a surge in **luteinizing hormone (LH)** stimulates preovulatory growth: Meiosis I is completed, and secondary oocyte and polar body are formed.
- Then, the secondary oocyte is arrested in metaphase of meiosis II approximately 3 hours before ovulation and will not complete this cell division until fertilization.





Folliculogenesis

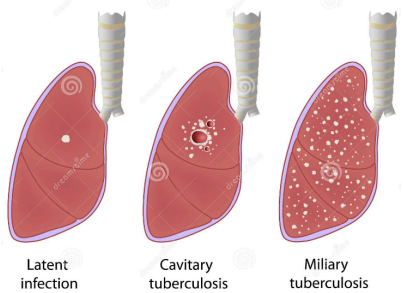


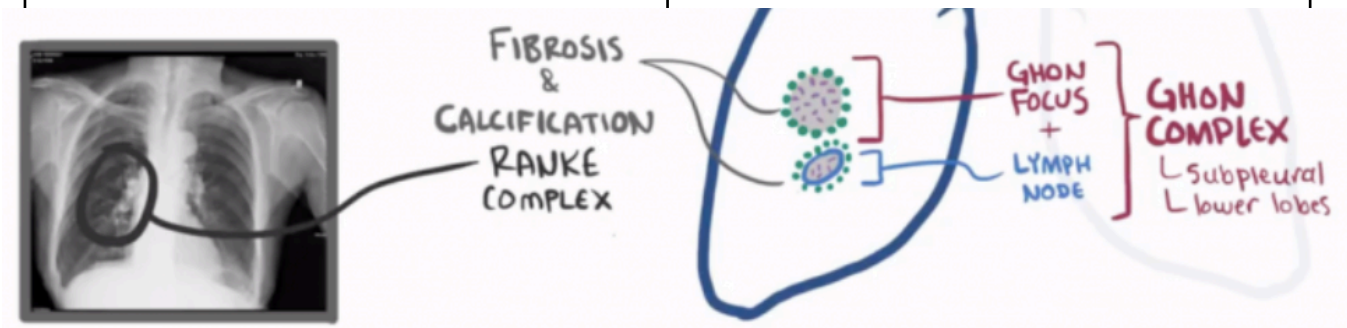
MS OPTIONAL 2024 Paper
PAPER I SECTION – B
Pathology

6. (a) (ii) Enumerate any five differences between primary tuberculosis and secondary tuberculosis. (10)

- What do you understand with the terms miliary tuberculosis and disseminated tuberculosis? What are the complications of miliary tuberculosis? (2007)

Primary tuberculosis vs Secondary tuberculosis

Primary Tuberculosis	Secondary Tuberculosis
Defintiion **Primary tuberculosis** is the form of disease that develops in a previously unexposed and therefore **unsensitized patient.**	**Definition** **Secondary tuberculosis** is the pattern of disease that arises in a **previously sensitized host.** **Also called Reactivation Tuberculosis.**
Incidence About 5% of those newly infected acquire significant disease.	**Incidence** Only a few patients (<5%) with primary disease subsequently develop secondary tuberculosis.
Location: Primary tuberculosis almost always begins in the lungs. The inhaled bacilli usually implant in the **distal air spaces of the lower part of the upper lobe or in the upper part of the lower lobe.** They are typically close to the pleura.	**Location:** Secondary pulmonary tuberculosis is **classically localized to the apex of one or both upper lobes**. The reason is obscure but may relate to high oxygen tension in these areas.
Pathogenesis: 1) Foci of scarring in lungs in healthy individuals may reactivate later when immunity fades 2) **Progressive primary TB/Non-reactive** (absence of caseating granulomas) in immunocompromised, malnourished	**Pathogenesis:** a) **Reactivation** 1) May appear shortly after primary tuberculosis 2) More commonly arises **from reactivation** of dormant primary lesions many **decades after initial infection**, particularly when host resistance is weakened. b) **Reinfection** 1) Either because the protection afforded by the primary disease has waned 2) Or because of exposure to a large inoculum of virulent bacilli.
Morphology: **Ghon focus:** 1-1.5 cm area of gray white inflammatory consolidation close to pleura **in lower part of upper lobe or in upper part of the lower lobe.** **Ghon complex:** Ghon focus plus nodal involvement **Ranke complex:** Progressive fibrosis and calcification of Ghon complex: detected radiographically **Progressive Primary TB:** - Irregular cavity lined by caseous material that is **poorly walled off by fibrous tissue** - Pleural effusions, tuberculous empyema or obliterative fibrous pleuritis	**Morphology:** 1-2 cm small focus of consolidation in **apical pleura** Localized lesions due to preexisting hypersensitivity (walled of lesions) Less prominent involvement regional lymph nodes **CAVITATION**  Latent infection Cavitary tuberculosis Miliary tuberculosis



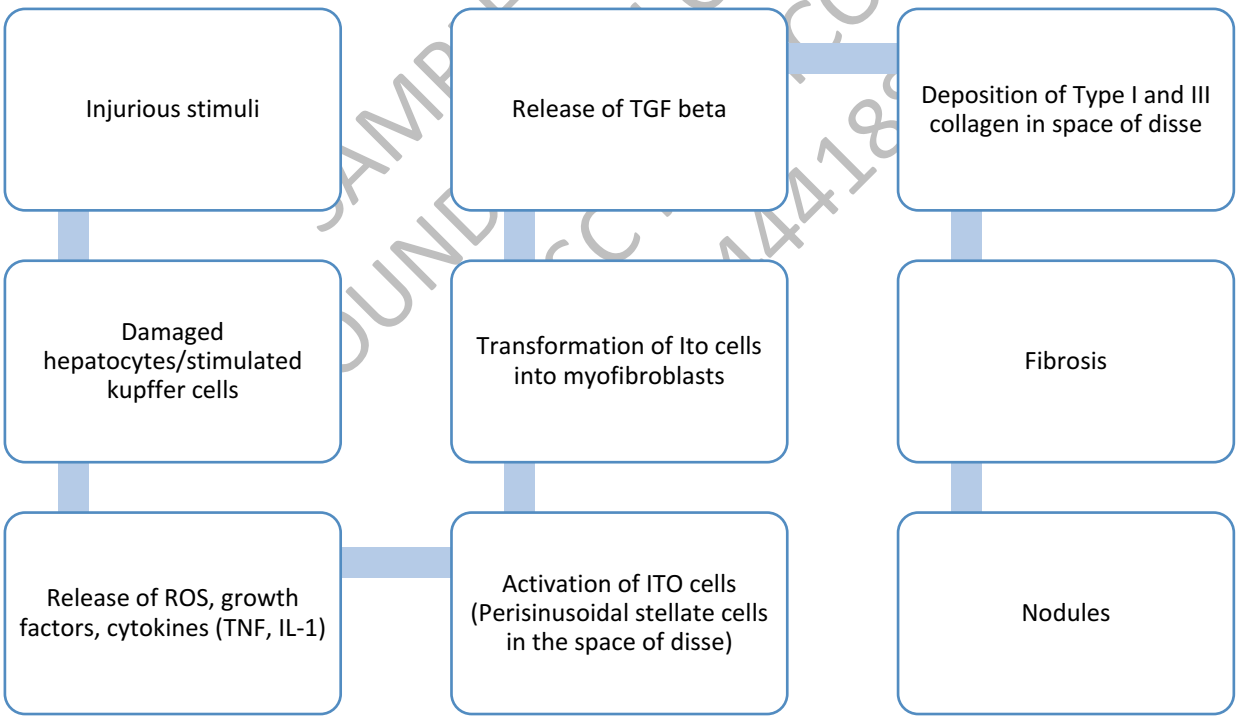
7. (b) (i) A 55-year-old female presented with hematemesis. On physical examination, she was afebrile and pale. No organomegaly was noted. Serological tests for hepatitis B were positive.
1. What is the most likely diagnosis?
2. Describe the microscopic findings.
3. What is the pathogenesis? (2+4+4=10)

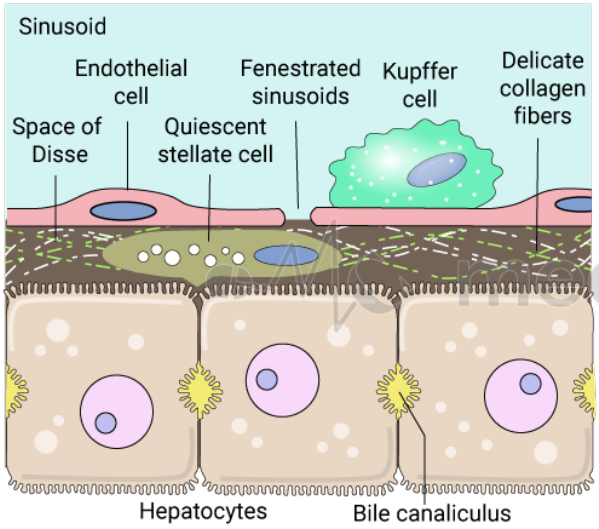
- Write in brief about aetiopathogenesis and histopathology of alcoholic cirrhosis (1994).
- Define cirrhosis of liver. Classify cirrhotic conditions of liver. Discuss the aetiopathogenesis and pathology of primary biliary cirrhosis (2000 & 2010)
- Describe etiopathology of hepatic cirrhosis (2003).
- Define cirrhosis. Enumerate four common causes of cirrhosis. (2020)
- Enumerate two important causes of cirrhosis, Describe the key histopathological features of cirrhosis. (2022)
- A 55-year-old female presented with hamatemesis. On physical examination, she was afebrile and pale. No organomegaly was noted. Serological tests for hepatitis B were positive.
  1. What is the most likely diagnosis?
  2. Describe the microscopic findings.
  3. What is the pathogenesis? (2024)

Case scenario: 55 year old female c/o hematemesis O/E pallor, inv: Hep B +

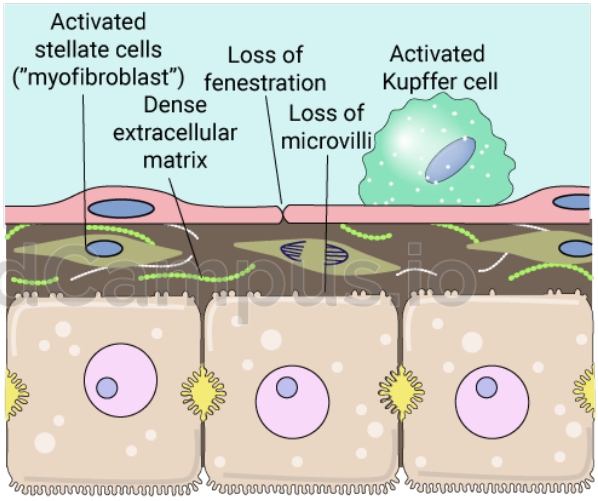
Likely diagnosis: c/o viral hepatitis presenting with hematemesis signifying esophageal varices due to PORTAL HYPERTENSION caused by CIRRHOSIS OF LIVER

Pathogenesis



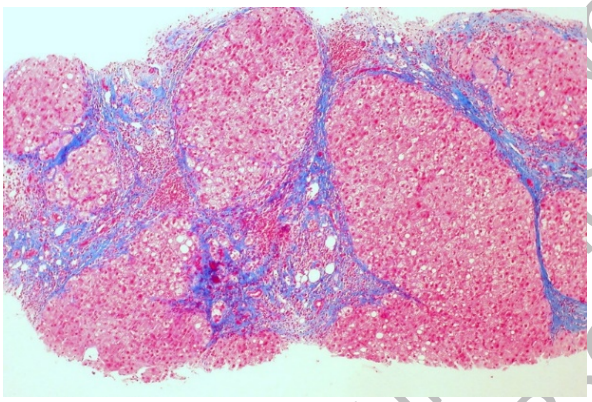


NORMAL LIVER



LIVER FIBROSIS

Histopathology



Fibrous septa-delicate bands or broad scars around multiple adjacent lobules.
Parenchymal nodules, encircled by these fibrous bands. Regeneration by preexisting long-lived hepatocytes and newly formed hepatocytes from stem cells (canals of hering)

MS OPTIONAL 2024 Paper
PAPER I SECTION – A
Microbiology

6. (c) (i) What is candidiasis? What are its different presentations and etiological causes? Give the laboratory diagnosis of a case of invasive candidiasis. (2+4+4=10)

- Pathogenesis, laboratory diagnosis and prophylaxis of Candidiasis (2009)
- Discuss morphology, pathogenicity and laboratory diagnosis of Candidiasis. (2013)
- Pathogenesis and laboratory diagnosis of candida albicans (2015)
- What are the factors responsible for invasive candida infection? Briefly describe the laboratory diagnosis of bloodstream candida infection. (2017)
- Enumerate infections causing Candida species. Suggest a laboratory approach for the diagnosis of invasive candidal infections (2019)

CANDIDIASIS

CANDIDOSIS/CANDIDIASIS/MONILIASIS is an infection of the skin, mucosa, and rarely of the internal organs, caused by yeast like fungus, CANDIDA ALBICANS and occasionally by other candida species.

Predisposing factors:
1) Extreme ages, pregnancy
2) Immunosuppression
3) Broadspectrum antibiotic therapy
4) Diabetic mellitus
5) Febrile neutropenia
6) Zinc or Iron deficiency

CLINICAL PRESENTATIONS
a) Skin (Cutaneous candidosis)
- Intertriginous – Effects groin, perineum, axilla and inframammary folds. Erythematous scaling or moist lesions with sharply demarcated borders.
- Paronychial – Seen around the nail, in occupations that lead to frequent immersion of the hands in water.

b) Mucosa
- Vaginitis – Acidic discharge (Thick curdy), seen commonly in pregnancy
- Oral Thrush – Seen in bottle fed infants, old and debilitated. Creamy white patches appear over tongue or buccal mucosa that leave a red oozing surface on removal.

c) Eye (Ocular candidosis)
- Fungal corneal ulcer (Pain, photophobia and blurred vision – less marked than bacterial ulcer)
- Chorioretinitis (Oval yellow-white or white colonies retinal lesions without vitreal involvement)
- Endophthalmitis (Vitritis and is often described as "fluff balls" or "string-of-pearls" within the vitreous body)

d) Intestinal candidosis
- Sequelae to oral antibiotic therapy characterized by Diarrhea which do not respond to treatment

e) Bronchopulmonary candidosis
- A rare complication of pre-existing pulmonary or systemic disease

f) Allergic candidosis : CANDIDID

f) Systemic manifestations
- Septicemia (Mainly by albicans, glabarata)
- Endocarditis
- Meningitis

CANDIDA SPECIES

C stellatoidae, C tropicalis, C pseudotropicalis, C krusei, C guillieromondii, C paraspilosis, C viswanathii, C glabarata, C dubliniensis

SPECIES IDENTIFICATION

Candida albicans can be identified from other candida species by GROWTH CHARACTERISTICS, SUGAR ASSIMILATION and FERMENTATION TESTS

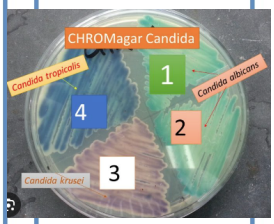
1) Formation of chlamydospores on corn meal/Rice meal agar at 20 degree c (Dolmaeu plate culture)
2) Chrom Agar-Light green color colonies
3) Reynolds Braude phenomenon (Germ tube test) : Also positive for dubliniensis

Laboratory diagnosis of INVASIVE CANDIDIASIS

Specimen	Transfer media	Direct microscopy	Culture media	SPECIES IDENTIFICATION
•**Skin scrapings from the lesions** •**Blood** •**Urine** •**Sputum** •**Catheters**		•Gram positive oval budding yeast cells (4-6 micrometers in size) •Other stains-PAS/Metahmine silver stain	•**Creamy white smooth pasty colonies with typical yeasty order** SDA	•GERM TUBE TEST •specific for candida albicans •It is also called Reynolds Braude phenomenon

Serological diagnosis	Molecular diagnosis	Other tests
•Antigen detection-cell wall mannan antigen •Antibody detection-ELISA •Enzyme detection-Enolase/aspartate proteinase	•Matrix-assisted laser desorption–ionization–time-of-flight mass spectometry (MALDI-TOF MS) •PCR	•Upper GI endoscopy

Formation of chlamydospores on corn meal/Rice meal agar



Chrom Agar-Light green color colonies

Galactomannan test

Factors Responsible for Invasive Candida Infection

- Antibacterial agents
- Abdominal and thoracic surgery
- Indwelling intravenous catheters
- Cytotoxic chemotherapy
- Hyper alimentation fluids
- Indwelling urinary catheters
- Parenteral glucocorticoids
- Severe burns
- HIV-associated low CD4+ T cell counts
- Immunosuppressive agents for organ transplantation
- Respirators
- Neutropenia
- Low birth weight (neonates)
- Diabetes

Beta - Glucan Assay

When the pretreated sample is mixed with the LAL solution, (1→3)-β-D-glucan in the sample activates Factor G, which initiates the cascade reactions and causes gelation.
The time taken for the transmittance to reach the threshold value is also measured. This time is defined as gelation time (Tg).

HOW TO FIND OUT HEMATOGENOUS DISSEMINATION TO MULTIPLE ORGANS

- The most challenging aspect of diagnosis is determining which patients with *Candida* isolates have hematogenously disseminated candidiasis.
- For instance, recovery of *Candida* from sputum, urine, or peritoneal catheters may indicate mere colonization rather than deep-seated infection, and *Candida* isolation from the blood of patients with indwelling intravascular catheters may reflect inconsequential seeding of the blood from or growth of the organisms on the catheter.
- Despite extensive research into both antigen and antibody detection systems, there is currently no widely available and validated diagnostic test to distinguish patients with inconsequential seeding of the blood from those whose positive blood cultures represent hematogenous dissemination to multiple organs.

- **Many studies are under way to establish the utility of the β-glucan test; at present, its greatest utility is its negative predictive value (~90%).**
- **Meanwhile, the presence of ocular or mucocutaneous skin lesions is highly suggestive of widespread infection of multiple deep organs.**

MS OPTIONAL 2024 Paper
PAPER II SECTION – A
Dermatology

1. (e) In a confirmed case of scabies in an adult :
(i) What are the primary manifestations of the disease and what is the pattern of distribution of lesions on the body ?
(i) What are the complications seen in scabies? (5+5=10)

- Describe the clinical manifestations and treatment of scabies (2004).
- Mention the complications of scabies (2012)

PRIMARY MANIFESTATIONS OF SCABIES/PATTERN OF DISTRIBUTION OF LESIONS :

Insidious onset : Intense noctural pruritus 4-6 weeks after initial infestation.
Carriers are asymptomatic
Physical examination reveals excoriations and eczematous dermatitis

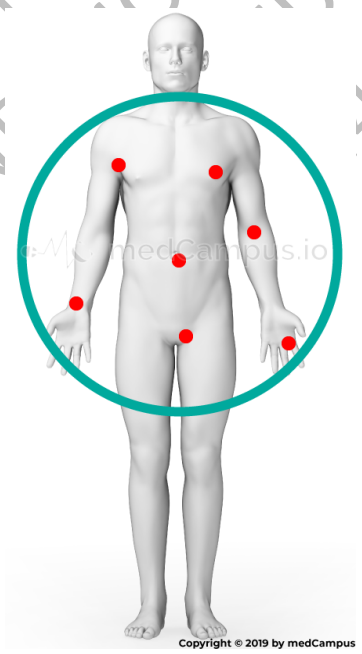
Pathognomic lesion in case of Scabies is Burrow.

Burrows represent the intraepidermal tunnel created by the moving female mite. They appear as serpiginous, grayish, threadlike elevations in the superficial epidermis, ranging from 2-10 mm long.

It usually involves-Finger webs, Wrist, Axillae, Areola, Umbilicus, Lower Abdomen, Genitalia, and Buttocks.

Except in infants, the face, scalp, neck, palms, and soles are usually spared.

Marking these areas form an imaginary circle known as CIRCLE OF HEBRA



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PAPER II SECTION – A
Pediatrics

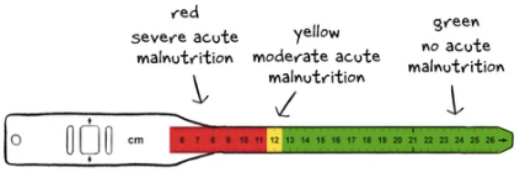
Growth charting is only one method of growth monitoring. There are other indicators such as height for age, weight for height and arm circumference.
Arm circumference is independent of age and particularly useful when age is not known.

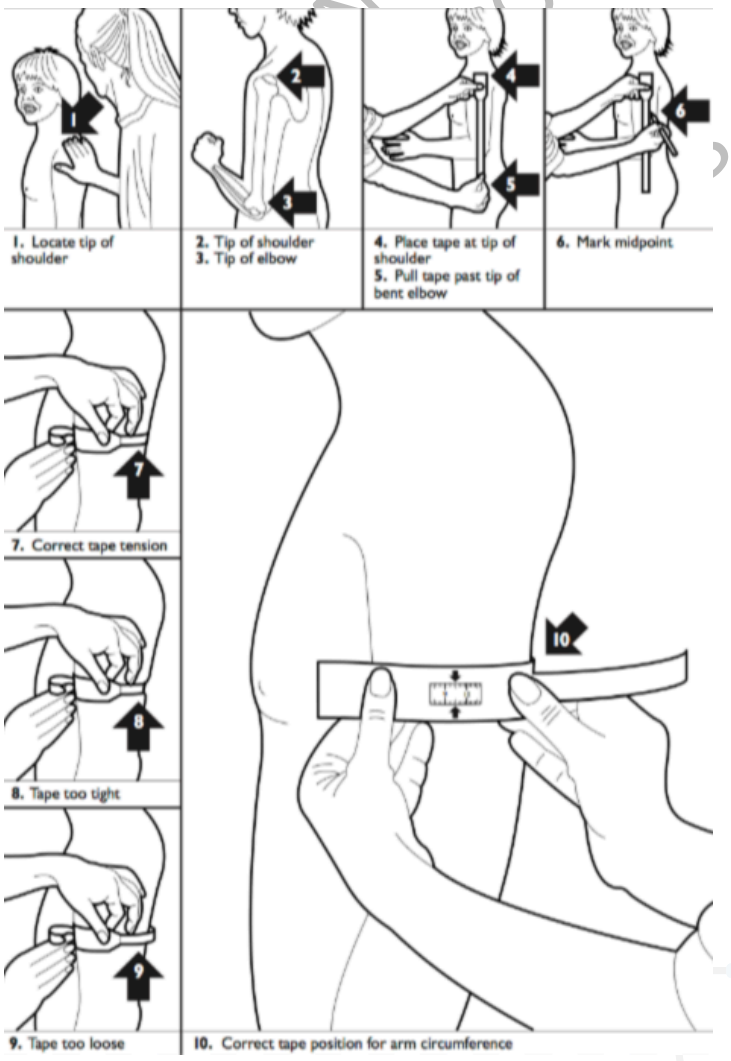
ARM CIRCUMFERENCE:

- It yields a relatively reliable estimation of the body's muscle mass, the reduction of which is one of the most striking mechanisms by which the body adjusts to inadequate energy intakes. Arm circumference cannot be used before the age of one year; between ages one and five years, it hardly varies.
- An arm circumference exceeding 13.5 cm is a sign of a satisfactory nutritional status, between 12.5 cm and 13.5 cm indicates mild-moderate malnutrition and below 12.5 cm: SEVERE MALNUTRITION

- To measuring the mid-upper arm circumference, first mark apoint midway between the tip of acromian process of scapula and the olecranon of ulna, while the child holds the leftarm by his side.
- It should be ensured that the tape is just tight enough to avoid any gap as well as avoid compression of soft tissues.

Shakir tape





MS OPTIONAL 2024 Paper
PAPER II SECTION – B
General Surgery

Diagnosis

Biopsy and histological analysis

MC histology-Adenocarcinoma (well-differentiated, moderately differentiated and undifferentiated types)

Poor prognosis

- Vascular and perineural invasion
- Presence of infiltrating margin
- Presence of tumor budding
- Signet ring carcinomas (primary mucoid carcinomas)

- Assess the surgical fitness of the patient
- Assess the extent of spread of the tumor

  -Local spread(Endoluminal ultrasound and MRI)

  -Metastasis(CT chest, abdomen and pelvis, PET CT WHOLE BODY)

Staging

Duke' s staging (3 stages)

A: Growth limited to rectal wall (15%) (excellent prognosis)

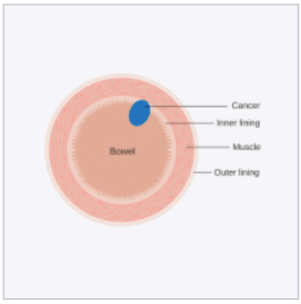
B: Growth extends to extrarectal tissues (35%) (reasonable prognosis)

C: Secondary deposits in regional lymph nodes (50%) (poor prognosis)

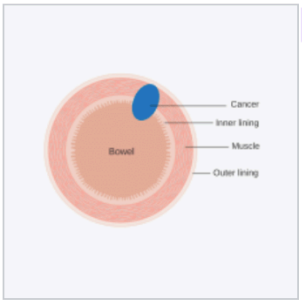
C1: Local pararectal lymphnodes alone are involved

C2: Nodes accompanying the supplying blood vessels to their origin from the aorta are involved. This does not take into account cases that have metastasised beyond the regional lymph nodes only by way of the venous system.

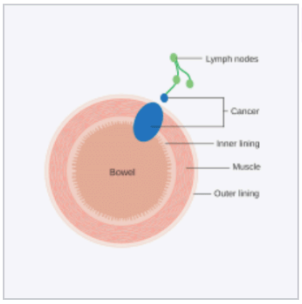
D: Distant metastasis (not a part of dukes staging original)



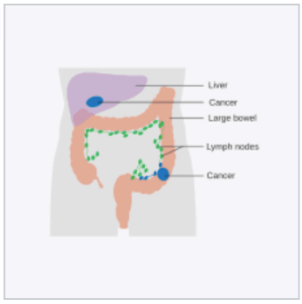
Dukes stage A bowel cancer; the cancer is only in the inner lining of the bowel.



Dukes stage B bowel cancer; the cancer has invaded the muscle.



Dukes stage C bowel cancer; the cancer has invaded the nearby lymph nodes.



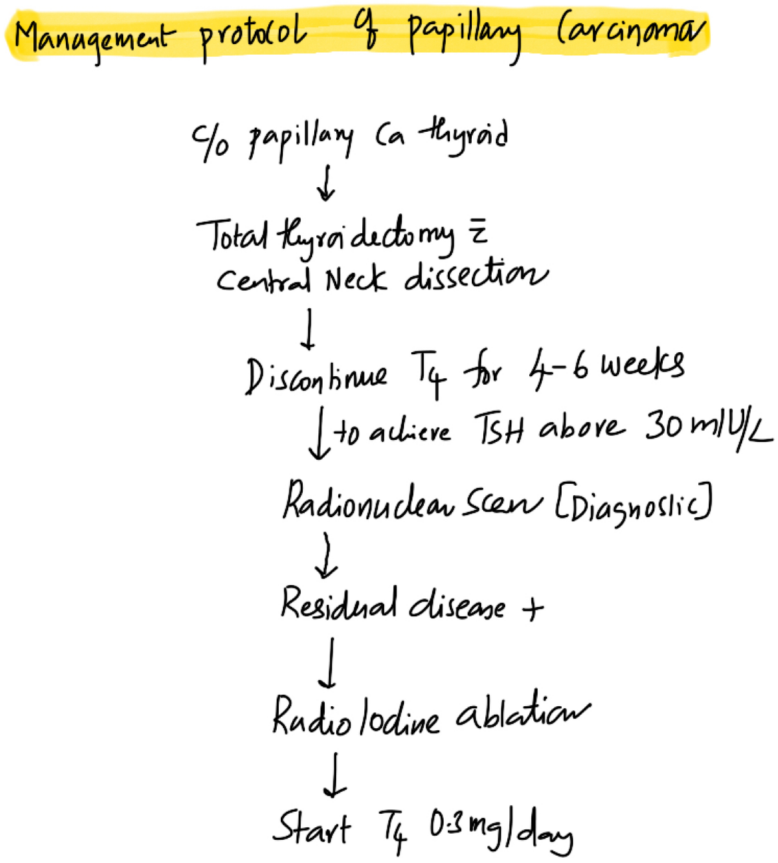
Dukes stage D bowel cancer; the cancer has metastasized.

Well-differentiated thyroid cancers >1cm (T1b or larger) – SURGICAL EXCISION

- **CHOICE OF SURGERY**

  Intrathyroidal cancers >1 cm and <4 cm (T1b and T2 tumors) in the absence of metastatic disease - **Unilateral (lobectomy) or bilateral (near total thyroidectomy)**

  Risk of recurrence and metastatic disease – Bilateral thyroid surgery (Near total thyroidectomy) + Radioiodine ablation



PROGNOSTIC CRITERIA

AMES scoring

A: Age less than 40 years—better prognosis
M: Distant metastasis—poor prognosis
E: Extent of tumour extracapsular spread—poor prognosis
S: Size less than 4 cm, good prognosis

AGES scoring
A: Age less than 40 years better prognosis
G: Grade of the tumour—high grade—poor prognosis
E: Extracapsular spread—poor prognosis
S: Size less than 4 cm, good prognosis

MACIS scoring
M: Metastasis
A: Age
C: Completeness of resection
I: Invasion
S: Size

AJCC 8th edition TNM Staging (AGE CUT OFF 55 years)

ENDOMETRIAL CHANGES

0—5 (10) Prolr

Ovulatory: Irregular Ripening, Irregular Shedding (LH ↑ FSH); Anovulatory: Est

OVULATORY DUB

Secretory changes +

1] Irregular shedding of Endometrium
[Mixture of Secretory + proliferative Endometrium]
on 5th day of Menstruation
Sampling of Endometrium

2] Irregular Ripening
patchy area of secretory changes amidst proliferative Endometrium

Prior to or soon after spotting : Sampling time

Anovulatory DUB

cystic glandular
3] Hypertrophy + Hyperplasia (premenopause)

SWISS CHEESE PATTERN
[Small + Large glands]
↓
Small + Large holes of Swiss cheese

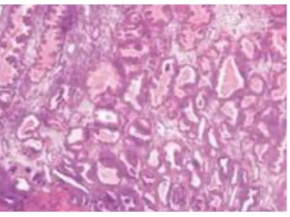
Secretory changes [−]

4] Atrophic pattern [Menopause]

Endometrial Patterns in DUB

[60%]

Normal secretory changes



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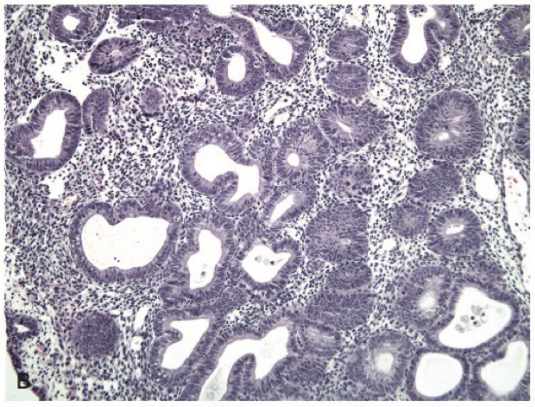
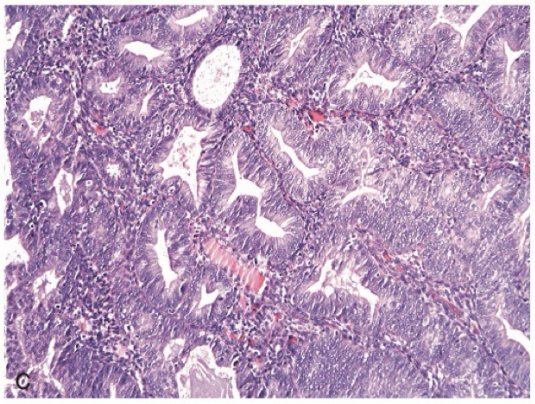
Hyperplastic Endometrium

Without atypia

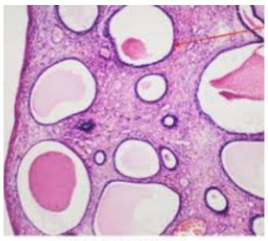


With atypia



[10%]

- Irregular shedding
- Irregular ripening
- Atrophy



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