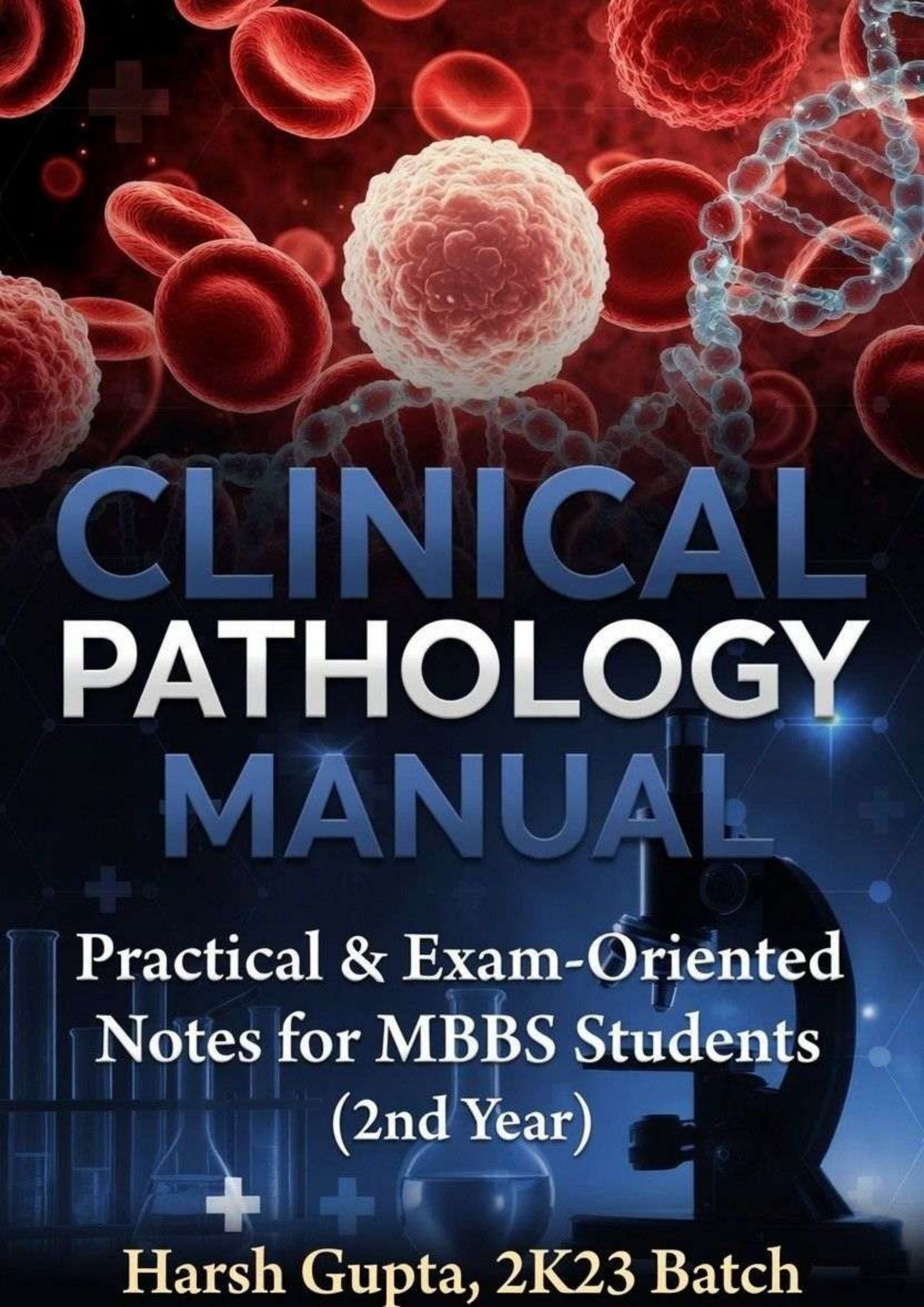

CLINICAL PATHOLOGY MANUAL

Practical & Exam-Oriented Notes for MBBS Students (2nd Year)

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BOOK AT A GLANCE

TABLE OF CONTENTS

QUESTIONS LIST:

SECTION 1: BASIC HAEMATOLOGY

Chapter 1: Haemoglobin Estimation & Red Cell Indices

Q1. Haemoglobin Estimation
Definition:
Haemoglobin estimation is the measurement of the concentration of haemoglobin (Hb) in blood, which reflects the **oxygen-carrying capacity of blood** and is a key investigation in haematology.

Normal Values:
- **Males:** 13–17 g/dL
- **Females:** 12–15 g/dL
- **Newborns:** 16–18 g/dL

Methods of Estimation:
1. Sahli's (Acid Haematin) Method
- **Principle:**
  Haemoglobin is converted to **acid haematin** by N/10 HCl and matched with a standard comparator.
- **Advantages:** Simple, cheap, rapid
- **Disadvantages:**
  - Less accurate
  - Subjective (visual color matching errors)

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2. Cyanmethemoglobin Method (ICSH reference method)
- **Principle:**
  Hb is converted to **cyanmethemoglobin** using potassium ferricyanide and potassium cyanide, and absorbance is measured at 540 nm.
- **Advantages:**
  - Most accurate and reliable
  - Standard reference method
  - Suitable for calibration of automated analyzers

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3. Automated Hematology Analyzers
- Based on **spectrophotometry / electrical impedance**
- Provide rapid Hb estimation along with full blood count (CBC)

Clinical Significance:
- **Low Hb → Anaemia** (iron deficiency, chronic disease, hemolysis, etc.)
- **High Hb → Polycythaemia, dehydration**
- Helps in:
  - Diagnosis of anaemia type
  - Monitoring treatment response
  - Preoperative and routine screening

Key Exam Line:
Cyanmethemoglobin method is the **gold standard for haemoglobin estimation**.

Q2. Red Blood Cell (RBC) Indices
Definition:
RBC indices are calculated values derived from **RBC count, haemoglobin (Hb), and haematocrit (Hct)**. They help in:
- Morphological classification of anaemia
- Differentiation of microcytic anaemias
- Assessment of RBC size and haemoglobin content

Types of RBC Indices:
1. Mean Corpuscular Volume (MCV)

$$\frac{Hct \times 10}{RBC\ count}$$

- **Normal: 80–100 fL**
- **Significance:** Indicates **size of RBC**

Interpretation:
- ↓ MCV → Microcytic anaemia
- Normal MCV → Normocytic anaemia
- ↑ MCV → Macrocytic anaemia

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2. Mean Corpuscular Haemoglobin (MCH)

$$MCH = \frac{Hb \times 10}{RBC\ count}$$

- **Normal:** 27–32 pg
- **Significance:** Hb content per RBC

---

3. Mean Corpuscular Haemoglobin Concentration (MCHC)

$$MCHC = \frac{Hb \times 100}{Hct}$$

- **Normal:** 32–36 g/dL
- **Significance:** Concentration of Hb inside RBC

Interpretation:

- ↓ MCHC → Hypochromia (iron deficiency, thalassaemia)
- Normal MCHC → Normochromic RBCs

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4. Red Cell Distribution Width (RDW)

- **Normal:** 11.5–14.5%
- Measures **variation in RBC size (anisocytosis)**

Interpretation:

- ↑ RDW → Iron deficiency anaemia (early marker)
- Normal RDW → Thalassaemia trait

Mentzer Index

$$Mentzer\ Index = \frac{MCV}{RBC\ count}$$

Interpretation:

- 13 → Iron Deficiency Anaemia
- < 13 → ß-Thalassaemia trait

Morphological Classification of Anaemia (based on MCV):

- **Microcytic (<80 fL):**
  - Iron deficiency anaemia
  - Thalassaemia
  - Anaemia of chronic disease
- **Normocytic (80–100 fL):**
  - Acute blood loss
  - Haemolytic anaemia
  - Chronic disease
- **Macrocytic (>100 fL):**
  - Megaloblastic anaemia (B12, folate deficiency)
  - Liver disease
  - Hypothyroidism

Key Points (Exam-Oriented):

- **MCV → best index for RBC size classification**
- **RDW ↑ → earliest indicator of iron deficiency**
- **MCHC ↓ → hypochromic anaemia**
- **Mentzer index → differentiates IDA vs thalassaemia trait**

Mnemonic:

- **MCV = Cell Size**
- **MCH = Hb per cell**
- **MCHC = Hb concentration**

Chapter 2: Peripheral Blood Smear & DLC

Q3. Differential Leukocyte Count (DLC)

Definition:

Differential Leukocyte Count (DLC) is the **percentage distribution of different types of white blood cells (leukocytes)** in peripheral blood.

It is an important part of haematological examination that helps in assessing **quantitative and qualitative changes in WBCs**.

Normal DLC (Adult Peripheral Blood):

- **Neutrophils:** 40–70%
- **Lymphocytes:** 20–40%
- **Monocytes:** 2–10%
- **Eosinophils:** 1–6%
- **Basophils:** 0–1%

Method of DLC:

1. **Peripheral blood smear preparation**
   - Staining: Leishman / Wright stain
2. **Microscopic examination**

   - Oil immersion lens used
3. **Cell counting**
   - 100–200 WBCs are counted
4. **Calculation**
   - Percentage of each type of leukocyte is calculated
5. **Automated analyzers**
   - Provide DLC but **manual smear review is essential** for abnormal cells

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Clinical Significance:

1. Neutrophilia
- Acute bacterial infections
- Acute inflammation
- Stress response
- Corticosteroid therapy

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2. Neutropenia
- Viral infections
- Bone marrow suppression
- Aplastic anaemia
- Drug toxicity

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3. Lymphocytosis
- Viral infections (e.g., infectious mononucleosis)
- Tuberculosis (chronic infections)

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4. Lymphopenia
- HIV/AIDS
- Steroid therapy
- Immunodeficiency states

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5. Monocytosis
- Tuberculosis
- Chronic infections
- Malaria
- Leukemias

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6. Eosinophilia
- Allergic disorders (asthma, rhinitis)
- Parasitic infestations
- Skin diseases
- Hodgkin lymphoma

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7. Basophilia
- Rare finding
- Seen in **myeloproliferative disorders**, especially:
  - Chronic Myeloid Leukemia (CML)

Advantages / Importance:
- Helps in diagnosis of infections and inflammatory conditions
- Detects allergic and parasitic diseases
- Important in diagnosis of haematological malignancies
- Detects abnormal/immature WBCs (e.g., blasts in leukemia)

Q4. Leishman Staining

Definition:
Leishman stain is a **Romanowsky-type stain** used for staining **peripheral blood smears, bone marrow smears, and parasite detection**. It provides detailed visualization of blood cell morphology and blood parasites.

Principle:
Leishman stain is based on the Romanowsky effect.
- **Basic dye (Methylene blue / Azure):**
  Stains acidic structures → **nucleus, RNA** → blue to purple
- **Acidic dye (Eosin):**
  Stains basic structures → **hemoglobin, cytoplasm** → pink to red
- Combined effect gives **differential staining of blood cells and parasites**

Procedure:
1. Prepare a thin **air-dried blood smear**
2. Flood smear with **Leishman stain (in methanol)** → acts as fixative
3. Allow staining for **1–2 minutes**
4. Add buffered water (pH ~6.8) and mix gently

Histopathology

TABLE OF CONTENTS

SECTION 11. SKIN & SOFT TISSUE

Benign Tumors

37. Lipoma
38. Hemangioma
39. Squamous Papilloma

Malignant Tumors

40. Basal Cell Carcinoma

SECTION 12. BONE & JOINTS

41. Osteoclastoma (Giant Cell Tumor)

SECTION 13. NERVOUS SYSTEM

42. Schwannoma

PRACTICAL EXAM APPROACH FOR HISTOPATHOLOGY SLIDES

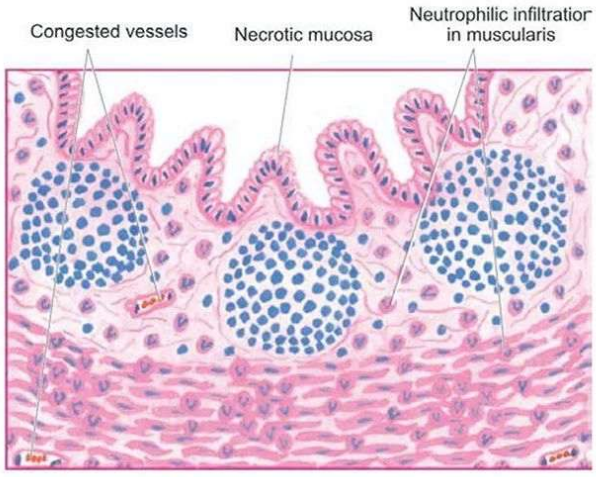
For every slide write:

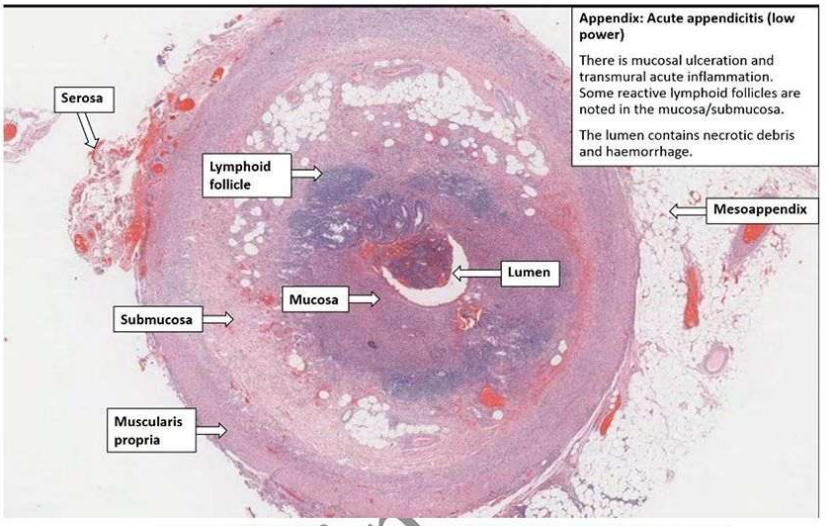
1. *Identification*
2. *Key microscopic features*
3. *Diagnosis*
4. *Clinical significance*

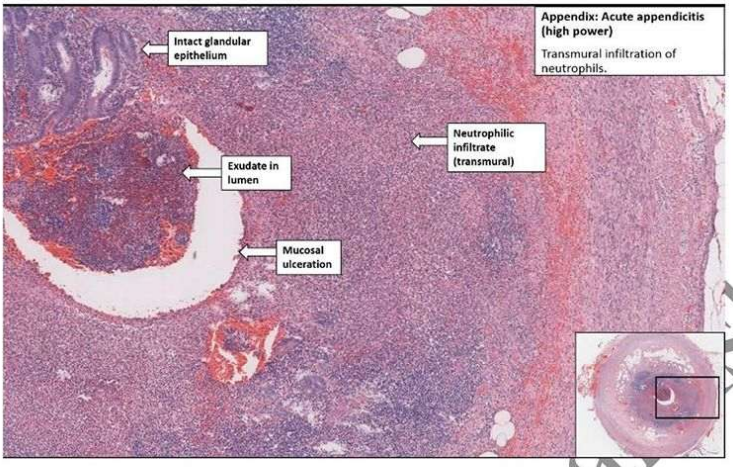
BASIC RULE - In H&E stain:

- **Nuclei → blue-purple**
- **Cytoplasm / muscle / collagen → pink**
- **Neutrophils → small dark blue-purple dots**
- **RBC congestion → bright pink-red areas**
- **Necrosis → dirty pink granular area with loss of nuclei**

1. Acute appendicitis







Concept:

Step 1: Recognize Appendix Wall Layers

From lumen to outward:

1. **Mucosa**
2. **Submucosa**
3. **Muscularis propria** ← (MOST IMP)
4. **Serosa**

Diagnostic feature:

<u>Neutrophilic infiltration of muscularis propria</u>

How neutrophils look:

- Tiny dark blue-purple cells

Where seen?

- Between muscle fibers ,In mucosa ,In submucosa ,Extending into muscularis

Normally :Muscle layer looks smooth pink.

In **appendicitis**:

- pink muscle gets interrupted by many **blue dots**

That means neutrophils infiltrating muscle.

In detail:

Histopathology of Acute Appendicitis

Mucosal Changes

- Mucosal neutrophils
- Focal superficial ulceration *(**not specific** for acute appendicitis)*

<u>Diagnostic Feature: Neutrophilic infiltration of muscularis propria</u>

Progressive / Severe Acute Appendicitis: Dense neutrophilic exudate

Acute Suppurative Appendicitis: Formation of focal abscesses within appendiceal wall

Acute Gangrenous Appendicitis

Due to vascular compromise: Ischemia & Hemorrhagic ulceration

Complications: Rupture / perforation / Suppurative peritonitis
Stages: (**imp**)
Normal appendix
→ Early acute appendicitis
→ Suppurative appendicitis
→ Gangrenous appendicitis
→ Perforation and peritonitis.

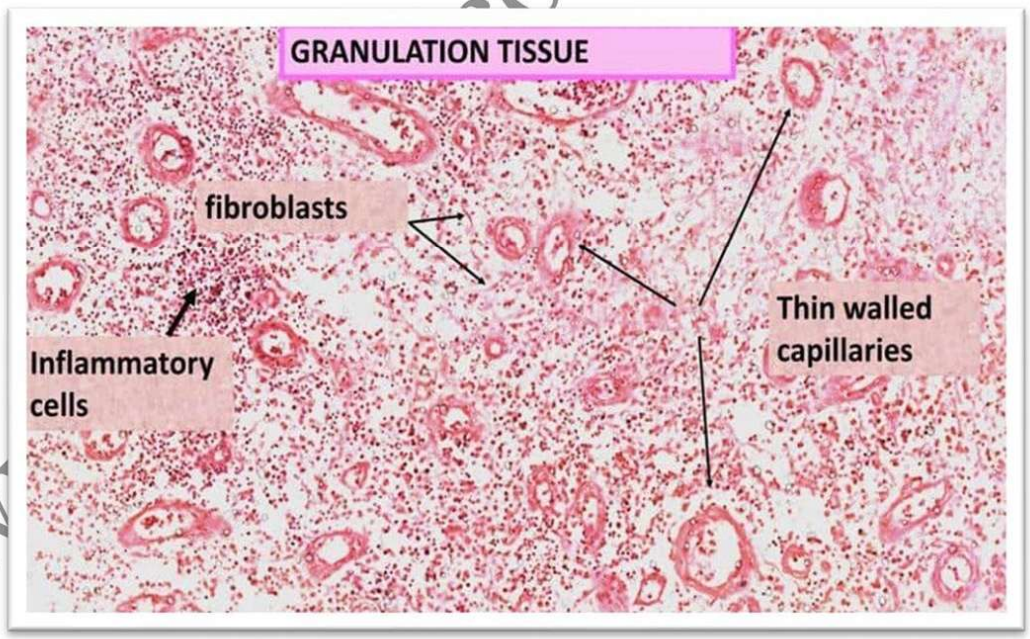
IN EXAM

Identification H&E stained section of appendix (appendicitis)
Key Microscopic Features
- Luminal exudate (neutrophils + fibrin)
- Dense transmural neutrophilic infiltrate (muscularis & serosa)
- Mucosal ulceration
- Intact glandular epithelium in areas

Diagnosis Acute suppurative appendicitis
Clinical Significance Indicates established bacterial infection; high risk of perforation if untreated. Requires urgent appendicectomy

2. Granulation tissue

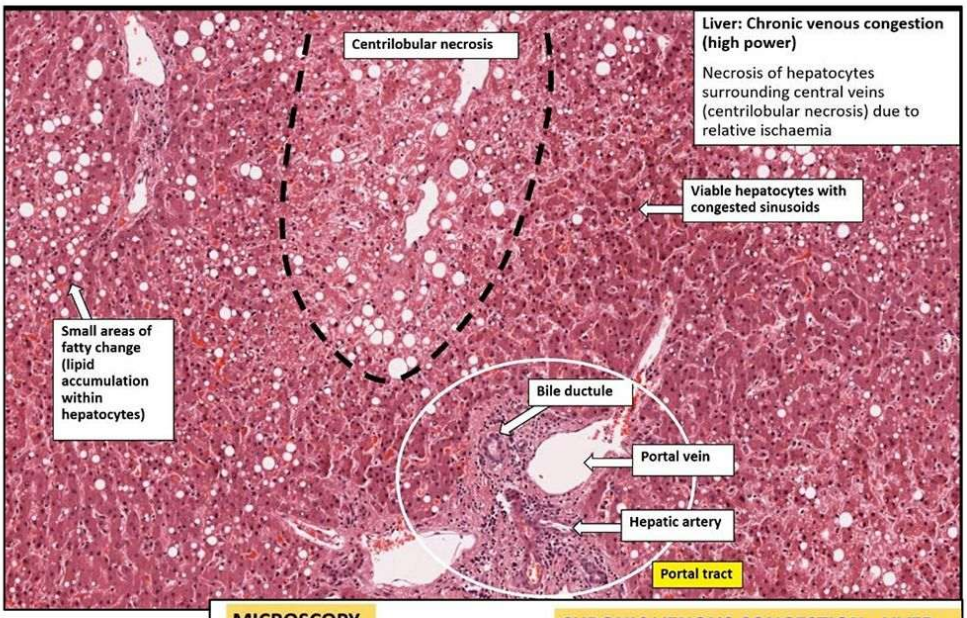
Concept:
Granulation tissue is the pink-red, granular, highly vascular connective tissue that forms in the proliferative phase of wound healing (starts ~day 3–5). It fills the defect in healing by secondary intention. Histologically defined by new capillary formation(angiogenesis), proliferating fibroblasts, and a loose edematous stroma with mixed inflammatory cells. It gradually matures into a scar (fibrosis)



In slide:
- **Newly formed capillaries** → The most prominent round/oval structures with bright red blood cells (RBCs) inside (thin endothelial lining).
- **Fibroblasts** → spindle-shaped cells with oval nuclei and pink cytoplasm; they are actively proliferating and producing collagen.

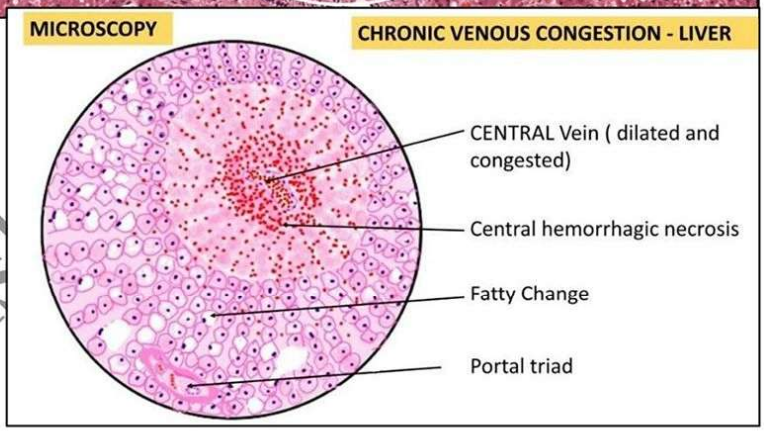
SECTION 2: HEMODYNAMIC DISORDERS

11. Nutmeg Liver



Nutmeg liver (also called chronic passive venous congestion or cardiac hepatopathy) is not a disease but a **pattern of liver injury** caused by long-standing **right-sided heart failure**, constrictive pericarditis, or Budd-Chiari syndrome. Blood backs up into the hepatic veins → centrilobular (zone 3) congestion, sinusoidal dilatation, hemorrhage, and hepatocyte necrosis. The cut surface grossly looks like nutmeg (dark red congested areas alternating with pale/yellow periportal areas).

On microscopy, the contrast between congested centrilobular zones and spared periportal zones is diagnostic.



How to identify:

1. **Low Power – Recognise Liver** (10 seconds) See classic liver architecture (& hexagonal cells) with central veins and portal tracts.
2.
3. **Look for the "Zebra" or "Mottled" Pattern** ← Notice **dark red/purple congested areas around central veins** alternating with **pale areas around portal tracts** → classic nutmeg appearance.
4. **High Power – Confirm the Zones**
   - Central vein area → dilated sinusoids full of RBCs + hepatocyte necrosis (zone 3)
   - Portal tract area → hepatocytes look normal/viable (zone 1)

IDENTIFICATION H&E STAINED SECTION OF LIVER – HIGH MAGNIFICATION
KEY MICROSCOPIC FEATURES

- CENTRILOBULAR NECROSIS OF HEPATOCYTES
- MARKEDLY CONGESTED & DILATED SINUSOIDS
- VIABLE HEPATOCYTES AROUND PORTAL TRACTS
- PORTAL TRIAD PRESERVED

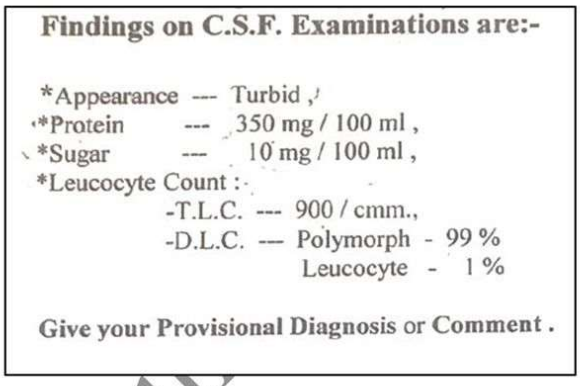
DIAGNOSIS CHRONIC PASSIVE VENOUS CONGESTION OF LIVER (NUTMEG PATTERN)
CLINICAL SIGNIFICANCE EXPLAINS ELEVATED LIVER ENZYMES AND JAUNDICE IN HEART FAILURE PATIENTS

DIAGNOSIS

1.Cerebrospinal fluid (CSF) examination of a patient revealed the following findings:

- Appearance: **Turbid**
- Protein: **350 mg/100 mL**
- Sugar: **10 mg/100 mL**
- Total leukocyte count (TLC): **900/cmm**
- Differential leukocyte count (DLC):
  - Polymorphs: **99%**
  - Lymphocytes: **1%**

Give your **provisional diagnosis/comment** based on the above CSF findings.


Findings on C.S.F. Examinations are:-

*Appearance --- Turbid ,
*Protein --- 350 mg / 100 ml ,
*Sugar --- 10 mg / 100 ml ,
*Leucocyte Count :-
-T.L.C. --- 900 / cmm.,
-D.L.C. --- Polymorph - 99 %
Leucocyte - 1 %

Give your Provisional Diagnosis or Comment .

Provisional Diagnosis:
Acute Pyogenic (Bacterial) Meningitis
You can write in exam style:
CSF picture is suggestive of acute pyogenic meningitis with neutrophilic pleocytosis, raised protein, and markedly decreased glucose.
Key supportive findings:

- **Appearance: Turbid** → indicates heavy cellular exudate/pus
- **Protein: 350 mg/100 ml** ↑ → markedly elevated due to inflammation and increased permeability
- **Sugar: 10 mg/100 ml** ↓ → very low CSF glucose, typical of bacterial infection
- **TLC: 900/cmm** ↑
- **DLC: Polymorphs 99%** → neutrophilic predominance, characteristic of pyogenic meningitis

2.A 60-year-old male presented with gradually increasing weakness, fatigue, and enlargement of multiple superficial lymph nodes.
Laboratory investigations revealed the following findings:

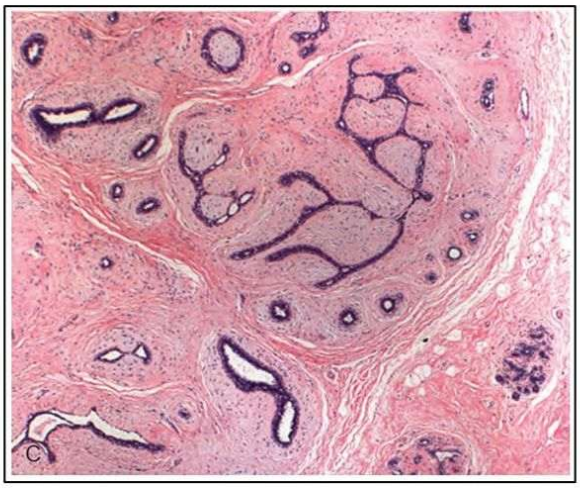
- Hemoglobin: **10.0 g/dL**
- RBC count: **4.2 million/cmm**
- Platelet count: **0.8 lakh/cmm**
- Total WBC count: **3.0 lakh/cmm**

Differential WBC count:

- Lymphocytes: **90%**
- Lymphoblasts: **10%**

Give your **provisional diagnosis/comment** based on the above findings.

Provisional Diagnosis:
Chronic Lymphocytic Leukemia (CLL)



4.A 24-year-old female presents with a gradually increasing breast lump associated with mild pain. There is no history of nipple discharge, redness, fever, or weight loss. On examination, a 2 × 2 cm, **round, well-defined, mobile breast lump** with firm to **rubbery** consistency is noted. A biopsy was performed and histopathological slides were prepared. Carefully examine the given specimen/photograph/slide and answer the following questions.
A. What is the most likely diagnosis? Is the lesion benign or malignant?
B. Describe the important microscopic (histological) features of this lesion.
C. Does this lesion increase the risk of breast carcinoma in the patient?

A. Diagnosis: Fibroadenoma of breast. It's a **benign** breast tumor.
Two points suggesting the diagnosis

1. **Young age with painless mobile breast lump**
    - Common in adolescent and young females
    - Lump is well-defined, freely mobile ("breast mouse"), firm/rubbery
2. **Slowly growing, well-circumscribed lesion**
    - No pain, discharge, fever, redness, or weight loss
    - Benign clinical behavior

B. Four important histological features

1. **Well-circumscribed / encapsulated lesion**
2. **Proliferation of both stromal and glandular components**
3. **Compressed slit-like ducts** due to stromal proliferation
4. **Fibrous stroma surrounding ducts** (intracanalicular or pericanalicular pattern)

C. Does it increase the risk of carcinoma?
Simple fibroadenoma usually does NOT significantly increase the risk of breast carcinoma.

5. A **10-year-old** boy was referred to the hospital for evaluation of **short stature**. He had grown only 2 cm in height over the past 2 years. On examination, **no abnormality** was detected apart from short stature.
Laboratory investigations revealed the following serum growth hormone levels:

- After vigorous exercise: **4 mU/L**
- During documented insulin-induced hypoglycaemia: **4 mU/L**

Answer the following questions:
A. What is the most likely diagnosis?
B. Mention any three other causes of short stature.
C. Enumerate the mediators regulating growth hormone secretion.

A. Diagnosis
The findings suggest **Growth Hormone Deficiency (Pituitary dwarfism)**.
Basis:

Specimen

Gross Specimen Viva – Quick Guide

Before You Speak

- *Pick up the jar confidently.*
- *Rotate it and look at all sides. (Most imp.)*
- *Spend 10–15 seconds observing before speaking. (even if you know)*
- *Do not blurt out the diagnosis immediately, even if you recognize it*

Standard Opening: "This is a jar-mounted, formalin-preserved gross pathological specimen. It appears to be a specimen of the (organ identified after careful observing)."

Sequence that must be followed:

Below are the must remember points

- **Type of Specimen (Jar mounted etc that line you said in opening)**
- **Identification of Organ (identified after observing the specimen- said in opening line)**
- **Gross Description**: Size, Shape, External Surface, Colour, Consistency
- **Cut Surface**
- **Description of Lesion**
- **Diagnosis**
- **Supporting Gross Features**

Below points are for outstanding viva

- **Etiology / Causes**
- **Pathogenesis** *(if asked)*
- **Microscopy** *(if asked) – Very imp. Don't tell any microscopic property in a gross specimen until asked.*
- **Clinical Features** *(if asked)*
- **Complications**
- **Investigations** *(if asked)*
- **Differential Diagnosis** *(if asked)*
- **Management** *(rarely asked in pathology practicals)*

INDEX

13. Lipoma

1. Crohn Disease (Intestine)

ONE LINER

"This is a jar-mounted, formalin-preserved gross specimen of the small intestine, most likely the terminal ileum. The bowel wall is markedly thickened and the mucosa shows linear serpiginous ulcers with a characteristic cobblestone appearance. Segmental involvement (skip lesions), luminal narrowing due to strictures, and creeping fat over the serosal surface are noted. These gross features are suggestive of Crohn disease, a chronic transmural inflammatory bowel disease."

1. Type of Specimen
Jar-mounted, 10% formalin-preserved gross pathological specimen.

---

2. Identification of Organ
Small intestine (terminal ileum ± ileocecal region/colon).

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3. Gross Description
Size
- Segment of intestine with **thickened bowel wall**.

Shape
- Irregular due to **strictures**.

Colour
- Mucosa appears **reddish with ulcerated areas**.

External Surface
- **Serosal surface shows creeping fat** (mesenteric fat extending over the bowel).
- May show **serositis**.

Consistency
- **Firm/rubbery** due to transmural fibrosis and muscular hypertrophy.

---

4. Cut Surface
- **Linear (serpiginous) ulcers** running along the long axis of the bowel.
- **Cobblestone appearance** due to alternating ulcerated and normal mucosa.
- **Thickened intestinal wall**.
- **Luminal narrowing (stricture)**.
- Deep **fissures** may be seen extending into the bowel wall.

---

5. Description of Lesion
- **Segmental involvement (skip lesions)** with sharply demarcated diseased areas separated by normal bowel.
- **Transmural inflammation** involving the entire bowel wall.
- Deep fissures may form **fistulae** or lead to **perforation**.
- Creeping fat may cover the serosal surface.

---

6. Diagnosis
Crohn Disease (Regional Enteritis).

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7. Supporting Gross Features
- **Skip lesions**.
- **Cobblestone mucosa**.
- **Linear/serpiginous ulcers**.
- **Transmural bowel wall thickening**.
- **Stricture formation**.

- **Creeping fat**.

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8. Etiology

Exact cause is unknown. It results from:

- Genetic susceptibility
- Dysregulated immune response
- Environmental factors
- Altered intestinal microbiota

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9. Microscopy

- **Transmural chronic inflammation**.
- **Crypt abscesses**.
- Crypt distortion and branching.
- **Non-caseating granulomas** (hallmark; present in ~35% of cases).
- Deep fissuring ulcers.
- Submucosal fibrosis and muscular hypertrophy.

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10. Clinical Features

- Chronic diarrhea
- Right lower abdominal pain
- Weight loss
- Fever
- Perianal fistulae/abscesses
- Malabsorption (especially with ileal disease)

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11. Complications

- Intestinal strictures causing obstruction
- Fistula formation (enteroenteric, enterovesical, enterocutaneous)
- Perforation
- Abscess formation
- Malabsorption (especially vitamin B12 deficiency)
- Increased risk of intestinal adenocarcinoma (long-standing disease)

⭐ **Viva Pearls (Most Important)**

Sites of involvement

- **Terminal ileum** (most common)
- Ileocecal region
- Colon
- Any part of the GIT from mouth to anus

Hallmark Gross Features

- **Skip lesions**
- **Cobblestone appearance**
- **Creeping fat**
- **Transmural inflammation**
- **Strictures**

Hallmark Microscopic Feature

- **Non-caseating granulomas** (when present, highly characteristic)

2. **ULCERATIVE COLITIS (Gross Specimen)**

ONE LINER

"This is a jar-mounted, formalin-preserved gross specimen of the large intestine. The colonic mucosa shows diffuse, continuous ulceration with loss of the normal mucosal pattern and multiple broad-based inflammatory pseudopolyps. There are no skip lesions, fistulae, or transmural thickening. These gross features are suggestive of ulcerative colitis, a chronic inflammatory disease confined to the mucosa and submucosa of the colon."

Spotters

Pathology Spotters: Laboratory Instruments and Accessories
Instructions for Students: In the spotter exam, you have 40-50 seconds per item. Identify the instrument/reagent by its appearance, note key markings/colour/features, and write 2–3 lines on its uses. In viva, examiners commonly ask: "What is this?", "Uses?", "How is it used?", "Principle?", "Advantages/Disadvantages".

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1. Wintrobe Tube Identification: Glass tube ~110 mm long, internal bore 3 mm, closed at one end. Graduated 0–10 cm on both sides (ascending on one side, descending on the other). Holds ~1 ml blood. **Uses:**

- Erythrocyte Sedimentation Rate (ESR) by Wintrobe method.
- Packed Cell Volume (PCV/Hematocrit).
- Preparation of buffy-coat smear (for LE cells, abnormal cells in aleukemic leukemia). **Viva Tip:** Anticoagulant used is EDTA; read after 1 hour.

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2. Westergren Pipette Identification: Long glass tube ~300 mm (or 200 mm marked), internal bore ~2.5 mm, open at both ends, graduated 0–200 mm from top to bottom. Usually supplied with a stand/pipette.
Uses:

- Standard method for ESR determination (more sensitive than Wintrobe). Blood is diluted with sodium citrate (4:1 ratio). **Viva Tip:** Preferred over Wintrobe for routine ESR; read at 1 hour.

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3. Salah Bone Marrow Aspiration Needle Identification: Short, stout needle, Usually 16–18G, with a sharp bevelled tip.
Parts of the needle: Trocar, Cannula, Adjustable side guard (This is important part, which helps in adjusting the depth of penetration of the needle)
Uses:

- Bone marrow aspiration (sternum/iliac crest) for cytology, leukemia diagnosis, megaloblastic anemia, etc.
- Not for trephine biopsy.
- **Imp-** Guard prevents over-penetration; aspiration yields liquid marrow.

Experiments

TABLE OF CONTENTS – PRACTICAL EXPERIMENTS

4.Experiment: Benedict's Test for Reducing Sugar in Urine

Aim

To detect the **presence of reducing sugar (glucose)** in urine by **Benedict's test**.

Principle

In an **alkaline medium**, **reducing sugars** reduce **blue cupric ions (Cu^{2+})** of Benedict's reagent to **yellow-red cuprous oxide (Cu_2O)** on boiling. The color of the precipitate varies from green to brick-red depending on the concentration of reducing sugar present.

.

Experiment (Procedure)	Observation	Inference
Take 5 mL Benedict's reagent in a test tube and boil. Add 8–10 drops (0.5 mL) urine, boil for 2 minutes, then allow to cool.	**Blue solution**	Negative (No reducing sugar)
	Green ppt.	Trace sugar (≈0.5%)
	Yellow ppt	+ (≈1%)
	Orange ppt	++ (≈1.5%)
	Brick-red ppt	+++/++++ (≥2% reducing sugar; strongly positive)

Normal Result: Negative (Blue solution; no glucose in urine).
Positive in: Diabetes mellitus, renal glycosuria, galactosemia, fructosuria.

Composition of Benedict's Reagent

Component	Function
Copper sulfate ($CuSO_4$)	Provides **Cu^{2+}** ions (oxidizing agent)
Sodium carbonate (Na_2CO_3)	Provides **alkaline medium**
Sodium citrate	Keeps copper ions in solution by preventing precipitation of copper hydroxide

6. Experiment: Modified boiling test for protein (Qualitative Test for Protein in Urine)

Aim

To detect the **presence of protein (mainly albumin)** in urine by the **Heat and Acetic Acid Test**.

Principle

Proteins coagulate on heating. On adding **acetic acid**, phosphates dissolve, while **coagulated proteins remain**, confirming the presence of protein (albumin).

CLINICAL PATHOLOGY MANUAL

For MBBS Second Year Students

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