

PATHOLOGY PRACTICAL FOUNDATION BLOCK









- Describe the pathological changes (both macro and micro) • which can occur, and are seen in the diseases and lesions studied in the foundation block.
- Identify the clinical manifestations of each pathological lesion.
- Correlate the morphological features with the clinical manifestations.
- seen in the lesions and diseases studied.
- Differentiate between the normal structure and the • pathological changes of the given tissue.



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CELL INJURY

1.FATTY LIVER (STEATOSIS)

| Cross | A | | DrOI |
|-------|---|--|------|
| | | | |

| Normal liver | Ste | |
|---|--|--|
| Features -the color is brown. -the surface is smooth. | Features: -slightly enlarg -pale yellow a - greasy. | |
| | | |

nce

eatosis

ged. appearance.



Causes:

1 - chronic alcoholism (the most common cause)
2 - Morbid Obesity.
3 - hepatitis C.

Fatty change is reversible type injury ; by strict diet and treatment of hyperlipidemia.



1.FATTY LIVER (STEATOSIS)

HistologicalAppearance

The lipid accumulates in the hepatocytes as vacuoles(macrovesicles).

Because: The lipid accumulates when lipoprotein transport is disrupted and/or when fatty acids accumulate.







The Stain: By Haematoxylin and Eosin. (H&E).

The nuclei displacement to the periphery.



Gross Appearance

Wedge-shaped pale area of coagulative necrosis in the renal cortex



2.COAGULATIVE NECROSIS(KIDNEY)

Causes:

ischemia and infarction of the kidney due to Obstruction of the artery.



2.COAGULATIVE NECROSIS(KIDNEY)

HistologicalAppearance

- 1. Coagulative necrosis (arrow) of glomeruli, tubules and interstitial tissue with loss of cell nuclei.
- proximal convoluted tubules. The **PAS stain** colors the brush border of 2. The haemorrhagic zone (star) at the periphery of the infarct (arrow) shows dilated and congested blood these structures a deep pink-lavender. vessels and cellular infiltrate by neutrophils, red blood • pale-staining collecting duct stands out in contrast to the abundant proximal tubules cells and lymphocytes (curved arrow)



The majority of the tubules seen here are



2.COAGULATIVE NECROSIS(SPLEEN)



Two large infarctions (areas of coagulative necrosis) are seen in this sectioned spleen.

Note: They may ask about the definition and the types of necrosis



2.COAGULATIVE NECROSIS: HEART (MYOCARDIUM)

HistologicalAppearance

Features:

1) Many nuclei undergo: Pyknosis (shrunken and

dark) \rightarrow karyorrhexis (fragmentation) \rightarrow

karyolysis (dissolution)

2) The cytoplasm and cell borders are not recognizable.

3) inflammatory cells can be seen (neutrophils).



Features:

- 1)The nuclei of the myocardial fibers are being lost.
 2) The cytoplasm lost its structure because no
 - well-defined crossstriations can be seen.



3.LIQUEFACTIVE NECROSIS (BRAIN)

GrossAppearance

Features:

the effected area Looks

-cystic or cavity formation (found in the upper right quadrant of the visual field).

- -creamy
- -yellow center
- -liquefactive necrosis.





Causes:

-Often it is associated with focal bacterial or fungal infections. -irreversible cell injury Brain infarction leading to ischemia is the most common cause of such type of lesions



3.LIQUEFACTIVENECROSIS (BRAIN)

- Gliosis



3.LIQUEFACTIVENECROSIS (LIVER ABSCESS)

Histological Appearance

- The liver shows a small abscess here filled with many <u>neutrophils</u>.
- This abscess is an example of **localized** liquefactive necrosis.





4.CASEOUS NECROSIS (LUNG)

GrossAppearance

Tuberculosis of the lung

Features:

large area of
caseous necrosis
containing yellowwhite cheesy debris.



Causes: caused by Mycobacterium Tuberculosis.



4.CASEOUS NECROSIS (LUNG)

Histological Appearance

Features:

- Multiple caseating granulomas
- with giant cells
- And caseous necrosis.

Granuloma consist of :



1 - Epithelioid macrophage 2. Giant cells (Langhan's) 3. rim of lymphocytes. Pink area with no nucleus

> Note: preserved alveolar spaces at the margins of the field.





5.FIBRINOID NECROSIS (ARTERY)

Histological Appearance

Features:

The wall of the artery shows a circumferential bright pink area of necrosis with inflammation (neutrophils with dark nuclei). which appears smudgy and acidophilic/eosinophilic.



Causes:

- Immune mediated diseases (autoimmune diseases)
- malignant hypertension.



6.FAT NECROSIS

Gross Appearance

In mesentery

Features:

The areas of white chalky deposits represent foci of fat necrosis with calcium soap formation (saponification) at sites of lipid breakdown in the mesentery

Fat necrosis of the mesentery (by lipase) in a case of acute pancreatitis Numerous round white fat necrosis.

Fat necrosis can also be seen in female breast 🕝





Causes:

- in acute pancreatitis.
- can also be seen in breast fat and other fatty areas due to traumatic injury.



6.FAT NECROSIS

HistologicalAppearance

fat necrosis in the fat surrounding the pancreas FAT NECROSIS (ANOTHER PICTURES) Features:

- The fat cell (adipocytes) are necrotic.
- The necrotic fat cells have vague cellular outlines.
- have lost their peripheral nuclei.
- their cytoplasm has become pink and amorphous





NORMAL ADIPOCYTES





CALCIFICATION

dystrophic calcification

deposition of calcium in dead or dying tissues

serum calcium levels are NORMAL

calcium metabolism is **NORMAL**

Seen in areas of **necrosis or damage**

| metastatic calcification: |
|--|
| deposition of calcium in normal and healthy tissue. |
| serum calcium levels are elevated HIGH |
| calcium metabolism is UBNORMAL |
| seen in <mark>hypercalcemia</mark> |

7.DYSTROPHIC CALCIFICATION (AORTIC)

Gross Appearance

Features:

- It is markedly narrowed (stenosis).
- 2. The (semilunar cusps) are thickened and fibrotic, and behind each cusp are irregular masses of piled-up dystrophic calcification.





Causes:

1.Deposition of calcium. 2.Normal calcium metabolism. 3.Seen in aging or damaged heart valves (e.g. athersosclerosis).



7.DYSTROPHIC CALCIFICATION (AORTIC)

Histological Appearance

Features:

- Fibrosis with some lymphocytes and dystrophic calcification.
- (A) hematoxylin and eosin; 1.25×
 objective magnification , and siderosis .
- 3. (B) Berlin blue 40× objective magnification.

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7.DYSTROPHIC CALCIFICATION (STOMACH)

Histological Appearance

Features:

1. At the far left is an artery with calcification in its wall. 2.There are an irregular bluish-purple deposits of calcium in the submucosa.



7.DYSTROPHIC CALCIFICATION (SKIN)

Gross Appearance

Features:

- Multiple erythematous hard papules in linear configuration on the extensor aspect of the arm.
- 2) Within the lesion there were several 2-5 mm white calcifications.



7.DYSTROPHIC CALCIFICATION (SKIN)

Histological Appearance

Features:

- 1. Irregular blue granular nodule or deposits of calcium in the dermis surrounded by fibrous.
- inflammatory cell like 2. histiocytes and also multinucleated giant cells (called as foreign body giant cell reaction)



Atrophy Shrinkage in the size of the cell

Causes of Atrophy :

- Decreased workload
- Loss of innervation
- Diminished blood supply
- Inadequate nutrition
- Loss of endocrine stimulation
- Aging

Metaplasia

Changing from one type of cell to another.

Hyperplasia

An increase in the number of the cells resulting in increase in the size of the organ.

Hyperatrophy

An increase in the size of cells resulting in increase in the size of the organ.

8.ATROPHY OF THE (BRAIN, TESTIS)

GrossAppearance

Cerebral atrophy in a patient with Alzheimer disease.

Features:

- The gyri are narrowed.
- The sulci are widened.
- particularly pronounced toward the frontal lobe region.

The testis

Features:

Left : Normal testis.

Right : the testis has undergone atrophy and is much smaller than the

normal testis.





Causes:

the most common cause is Alzheimer disease

Atrophied Testis

Causes: people who take steroids.





9.LEFT VENTRICULAR HYPERTROPHY

GrossAppearance

Features:

- myocardial fibers :
 - 1- doesn't increase of number.
 - 2- Size increase (hypertrophy) response to an Increased workload .
- Left ventricle is grossly thickened.
- cross section view (up) and longitudinal section view (down) of the heart.

The heart is from a severe **hypertensive** patient.

Compensatory hyperplasia can also be seen in organ as prostate, bladder, female breast and uterus.







Normal ventricular



Causes:

- Patients with severe chronic hypertension caused by atherosclerosis.
- Hypertrophic cardiomyopathy.



10.PROSTATIC HYPERPLASIA



10.PROSTATIC HYPERPLASIA

HistologicalAppearance

- Nodular hyperplasia of glandular and fibromuscular stromal tissue.
- Each nodule shows large number of glands of variable sizes lined by tall columnar epithelium and some are cystically dilated.

Here is one of the nodules of hyperplastic prostate

- glands.



there are many glands along with some intervening stroma. The cells making up the glands are **normal in appearance**, but there are just too many of them.

Eosinophilic hyaline corpora amylacea is present in some



11.SQUAMOUS METAPLASIA

Gross Appearance

Normal Uterine Cervix

Features:

1. Smooth

2. glistening mucosal surface, with small rim of vaginal cuff from this hysterectomy specimen.
3.The cervical is small and round, typical for a nulliparous woman.
4.The OS will have a fish-mouth shape after one or .

more pregnancies.



Histological Appearance

Normal and Dysplastic Cervical Squamous Epithelium

Features:

The normal cervical squamous epithelium transforms to dysplastic changes with underlying chronic inflammation

Normal



11.SQUAMOUS METAPLASIA

Histological Appearance

Endocervical Squamous Metaplasia:

Features:

A section of endocervix shows the normal columnar epithelium at both margins and a focus of squamous metaplasia in the center.



11.SQUAMOUS METAPLASIA

Laryngeal Squamous Metaplasia Features:

Metaplasia of laryngeal respiratory epithelium has occurred here in a smoker.

 The chronic irritation has led to an exchanging of one type of epithelium (the normal respiratory epithelium at the left) for another (the more resilient squamous epithelium at the right)







Thrombo-embolic disorder

PART2

Background information

HEMOSTASIS & THROMBOSIS

- Thrombosis: is a process by which a thrombus is formed.
- A thrombus: is a solid mass of blood constituents which
- Develops in artery, vein or capillary. •
- It is intravascular coagulation of blood and it can cause significant interruption to blood flow.
- Thrombi may develop anywhere in the cardiovascular system, the cardiac chambers, valve surface, arteries, veins, or capillaries. They vary in size and shape, depending on the site of origin.
- Thrombi in the vein are called venous thrombi. Thrombi in the artery are called arterial thrombi. When arterial thrombi arise in heart chambers or in aorta they are termed mural thrombi.
- Thrombi can grow. The propagating/growing tail of the thrombi is weak and is prone to fragmentatio creating an **embolus**

Deep Veins of the Le



Background information

HEMOSTASIS & THROMBOSIS MORPHOLOGY OF THROMBUS

- A thrombus is made up of fibrin, platelets & red blood cells and some inflammatory cells.
- When formed in the heart or aorta, thrombi may have laminations produced by alternating of pale and dark layers, called **lines of zahn** the pale layers contain platelets mixed with fibrin. The darker layers contain red blood cells.









Background information

HEMOSTASIS & THROMBOSIS EMBOLISM

- •An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.
 - •Almost all emboli represent some part of a dislodged thrombus, hence the commonly used term thromboembolism.
- The emboli ultimately lodged in vessels too small to permit further passage, resulting in partial or complete vascular occlusion leading to ischemic necrosis of distal tissue (infarction). Depending on the site of origin, emboli may lodge in the pulmonary or systemic circulations resulting in a **pulmonary embolus** or **systemic embolus**.





HEMOSTASIS & THROMBOSIS Background information PULMONARY THROMBOEMBOLISM

- •Here the embolus get lodged in the pulmonary vasculature.
- Depending on size of embolus, it may get stuck and block the main pulmonary artery or block the bifurcation of the pulmonary trunk (saddle embolus) or pass out into the smaller, branching arterioles of the pulmonary circulation.
- Most pulmonary emboli (60% to 80%) are clinically silent because they are small. Sudden death or cardiovascular problems occurs when 60% or more of the pulmonary circulation is obstructed with emboli.
 - Embolic obstruction of small end-arteriolar pulmonary branches may result in infarction $^{\odot}$.

bins Basic Pathology 8e - www.studentconsul
1. ORGANIZING THROMBUS

Gross Appearance

Histological Appearance

Name of thrombus: Organizing thrombus in a case of pulmonary embolism



1.ORGANIZING THROMBUS WITH LINES OF ZAHN

Histological Appearance

- Microscopic appearance of: pulmonary thromboembolus
- Location:
- large pulmonary artery.
- There are interdigitating areas of pale pink and red that form the "lines of Zahn" characteristic for a thrombus.



"lines of Zahn" represent layers of **RBCs**, platelets, and fibrin which are laid down in the vessel as the thrombus forms.



1.ORGANIZING THROMBUS WITH LINES OF ZAHN



1.ORGANIZING THROMBUS WITH LINES OF ZAHN



The interdigitating areas of pale pink and red within the organizing embolus form the "lines of Zahn" (arrow) characteristic of a thrombus.

Organizing thrombus : Cross section of blood vessel shows:

- inflammatory cells.
- Recanalization can be seen at one side.

Histological Appearance

The lumen is occluded by thrombus which consists of alternate layers of platelets with fibrin thread and clotted blood (line of Zahn). Organization can be seen at the periphery of thrombus which includes formation of small capillaries & fibroblasts with chronic

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2. PULMONARY EMBOLUS WITH INFARCTION



عادة ما يحدث عندما تتكون تجلطات في أورده الساق(غالبا تحدث من قل الحركه او اطاله الجلوس) وتنفصل لتنتقل و تستقر في الشرايين الرئويه مسببه انسداد كامل او جزئي لها

3. MYOCARDIAL INFARCTION

Gross Appearance



- area of fresh myocardial infarction (arrows) in the left ventricle.
- Initially the area of fresh infarct appears red.
- The area of infarct becomes well defined by **2 to 3 days** with a central area of yellow discoloration surrounded by a thin rim of hemorrhage.
- There is also some **left ventricular hypertrophy**.

Clinical features: - Arrhythmias (عدم (انتظام ضربات القلب - ventricular, تمدد الأوعية)aneurysm (الدموية - Rupture of myocardium (تمزق (عضلة القلب - Cardiac tamponade (hemorrhage in the pericardium lead to restricted) حالة يحدث فيها تجمع للسوائل أو) (الدم حول القلب اذا تبون تقرونه للاستزادة (\cdot) TEAM435 والاحتياط



Gross Appearance



(left ventricular hypertrophy).

Changes in myocardial Infarction

| Time | Gross changes | Microsco |
|-------------|--|----------------------|
| 0-4 hours | None | None |
| 4-12 hours | Mild Mottling (hemorrhagic look) | Coagula |
| 12-24 hours | Dark Mottling | More coo neutroph |
| 1-7 days | Yellow infarct center with surrounding red borders | Neutroph come to |
| 1-2 weeks | Yellow infarct center with red gray borders | Granulat |
| 2-8 weeks | Scar | Collagen |

Background information

opic changes

tion necrosis

agulation necrosis; ils come in

nils die, macrophages eat dead cells

ion tissue

and fibrosis



4. MYOCARDIAL INFARCTION (Acute myocardial infarct histological appearance)

Histological Appearance

after 24 hours

- there is a neutrophilic infiltrate at the border of the infarct.
- Viable myocardium is at the left, and neutrophils are seen infiltrating the necrotic muscle.

Note: the nuclei are not clearly visible in most of the necrotic cells.

at day 3

necrosis of cardiomyocytes, infiltrated by a heavy neutrophilic infiltrate (arrow).

The neutrophils release enzymes that help dissolve dead cell bodies which will be phagocytized by macrophages. With time the neutrophils begin to die and replaced by an influx of macrophages.



4. INFARCTION OF THE SMALL INTESTINE Histological Appearance

<u>early healing changes (3 weeks post MI)</u> - granulation tissue (growth of capillaries and fibroblasts)

- the collagen is being laid down to form a scar.

The non-infarcted myocardium is present on the left and upper part of the picture.

2 months post MI (healed) there is replacement of the necrotic cells by a dense collagenous scar.

- The myocardium shows fibrosis with collagenization (scar).
- Residual viable red myocardial fibers are present.







(۲۵ منخص من تيم ۲۵) Microscopic / Histological appearance



•Pic (1) : Transmural myocardial infarct at 2 weeks.

•Pic (2) : Acute myocardial infarct. This 3-4 day old infarct shows necrosis of myocardial cells and

is infiltrated with polymorphonuclear (Neutrophil) leukocytes.

- Picture 1-2 (early stage and middle stage) :
- Loss of nucleus and cross saturations.
- Debris of necrotic tissue.((الخلايا الميتة)
- Neutrophil (pic 2).





 1- Patchy coagulative necrosis of myocardial fibers. The dead muscle fibers are structureless and hyaline with loss of nuclei & striations.

2- Chronic ischemic fibrous scar replacing dead myocardial fibers .
3- The remaining myocardial fibers show enlarged nuclei due to ventricular

Hypertrophy.

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4. INFARCTION OF THE SMALL INTESTINE

infarcted small bowel loops

viable bowel

Intestinal infarction / <u>severe ischemia (important)</u>

- light pink viable bowel.
- (note: the forceps extend through an internal hernia in which a loop of bowel and mesentery has been caught. This is one complication of adhesions from previous surgery. The trapped bowel has lost its blood supply



4. INFARCTION OF THE SMALL INTESTINE



Features:

- Mucosal erosions / ulceration (a complete loss of the mucosal epithelium).
- **Broad areas of hemorrhage necrosis**
- moderate inflammatory infiltrate is present.

Histological Appearance

intestinal infarction typically begins in the villi, which are end vasculature without vanastomoses.





Inflammation

Redness (Rubor)

the five basic signs

INFLAMMATION

Inflammation is the tissue reaction to cellular injury.

> steps of the inflammatory response

Types of inflammation







- The process of exudation, aided by endothelial cell contraction and vasodilation, which typically is most pronounced in venules.
- Collection of fluid in a space is a transudate. If this fluid is protein-rich and has many cells then it becomes an exudate.





Acute inflammation



1.INFLAMMATION WITH NECROSIS



2. ACUTE FIBRINOUS PERICARDITIS

Normal pericardium







2. ACUTE FIBRINOUS PERICARDITIS

RIGHT FIBRINOUS PERICADITIS 25X & 100X H&E STAIN inal College of Medicine Collection Webslide Enterprise Slide #MS P074

Note a considerable number of erythrocytes trapped in the mesh of fibrin threads.

Histological Appearance



- The fibrinous exudate is seen to consist of pink strands of fibrin gutting
 - from the pericardial surface at the upper right.
 - The exudate on the surface is shown enlarged in the inset.



2. ACUTE FIBRINOUS PERICARDITIS



3. ACUTE APPENDICITIS

Normal appendix



- yellow to tan exudate.
- Hyperemia.
- pale tan serosal surface.

against the background of the caecum

Acute Fibrinous Pericarditis

periappendiceal fat superiorly, rather than a smooth, glistening

- The organ is enlarged and sausage-like (botuliform).
- red inflamed mucosa with its irregular luminal surface. It does not show late complications, like transmural necrosis, perforation, and abscess formation



3. ACUTE APPENDICITIS



HPF Scattered Neutrophils in the crypt epithelium.

Histopathology This slide shows the muscle layer of the appendix which is permeated with numerous polymorphonuclear leukocytes

Histological Appearance





4. ACUTE CHOLECYSTITIS

Gross



Caused by impacted gall stone in cystic duct.

Histological Appearance

- Infiltration of the mucosa and submucosa of by many neutrophils.
- Vascular congestion.



4. SKIN PILONIDAL SINUS

-Redness -swelling -sinus opening

A pilonidal sinus is a sinus tract which commonly contains hairs. It occurs under the skin between the buttocks (the natal cleft) a short distance above the anus. Usually runs vertical between the buttocks and rarely occurring outside the coccygeal region.

Gross

- Surgical excised pilonidal sinus tracts. -Fistula : an inflammatory
- tract lined by
- inflammatory granulation
- tissue, has tow opening and lies between to
- organs.







4. SKIN PILONIDAL SINUS Histological Appearance



The lumen of the sinus and wall contain large number of hair shafts with foreign body giant cells, chronic inflammatory cells (lymphocytes, macrophages, neutrophils)

chronic inflammation

5. CHRONIC CHOLECYSTITIS WITH STONES

- thickness of gall bladder wall.
- abundant polyhedric stones.
- Small papillary tumor in the cystic duct.



Irregular mucosal folds and foci of ulceration in mucosa. Wall is penetrated by mucosal glands which are present in muscle coat (Rokitansky-Aschoff sinuses). All layers show chronic inflammatory cells infiltration and fibrosis





The mucosa is atrophic, with a single layer of flattened epithelium. There is proteinaceous fluid adherent to the mucosal surface, with some bile stained orangebrown crystals toward the upper left in the lumen. The lamina propria shows fibrosis and contains a mononuclear cell infiltrate (small dark blue nuclei). The muscle is hypertrophiedcompared to normal gallbladder



6. BRAIN ABSCESS





Cavity contains neutrophils, necrotic cells, bacteria and fibrinous material



CT of a cerebral abscess. There is a liquefactive center with yellow pus surrounded by a thin wall. Abscesses usually result from hematogenous spread of bacterial infection, but may also occur from direct penetrating trauma or extension from adjacent infection in sinuses

Histological Appearance

Center of the abscess:

Cavity contains neutrophils, necrotic cells, bacteria and fibrinous material



7. GRANULATION TISSUE

Histological Appearance



1 - repair tissue occurs after inflammation Pink homogenouscollagen fibers may be 2- consist of new blood vessels, macrophage and fibroblast which form identified collagen fibers. Function? To start healing process.





GRANULOMAS









COLONIC BILHARZIASIS - HPF

COLON BIOPSY OF BILHARZIASIS. FIBROSING FOREIGN BODY GRANULOMA AGAINST THE MIRACIDIUM-CONTAINING OVUM OF S. MANSONI IS <u>OBSERVED IN THE</u> <u>SUBMUCOSAL LAYER</u> (H&E).

BILHARZIASIS OF THE URINARY BLADDER

SCHISTOSOMA HAEMATOBIUM. URINARY BLADDER BIOPSY <u>SHOWING BILHARZIASIS</u>

EGGS

1.BILHARZIAL GRANULOMAS



S. JAPONICUM IN THE HEPATIC PORTAL TRACT

S. JAPONICUM EGGS IN HEPATIC PORTAL

TRACT



2. CUTANEOS LEISHMANIASIS



GROSS APPEARANCE

LEISHMANIASIS IS CAUSED BY PARASITIC INFECTION, MAINLY BY PARASITES OF THE LEISHMANIA GENUS WHICH ARE CARRIED BY A BLOOD-SUCKING INSECT KNOWN AS THE SANDFLY.



HISTOLOGICAL APPERANCE



CUTANEOUS LEISHMANIASIS

HISTOLOGICAL VIEW SHOWS <u>MARKED CELLULAR</u> INFILTRATION AND PARASITES

(LEISHMAN BODIES) WITHIN MACROPHAGES



CUTANEOUS LEISHMANIASIS

THE BLOOD FILM SHOWS MACROPHAGES CONTAINING LEISHMANIA AMASTIGOTES, EACH WITH A PROMINENT KINETOPLAST (SEEN AS A DARKENED SPOT NEXT TO THE LARGER NUCLEUS) AND NO FLAGELLA (IN CONTRAST WITH THE PROMASTIGOTE FORM).



3.Tuberculosis of the lung

GROSS Appearance

Pulmonary TB-Caseous Necrosis

Features:

*The granulomas have areas of <u>caseous</u>

necrosis

producing a sub-pleural lesion called

a Ghon's focus.

• Ghon's complex: The early Ghon's focus + the lymph node lesion

The Ghon's complex



Initial (primary) infection with Primary-tupperculosis: the pattern seen with initial infection with tuberculosis in children. Reactivation, or secondary tuberculosis: is more typically seen in adults.



caused by Mycobacterium tuberculosis

Pulmonary TB - Ghon's Complex

Miliary TB of the Lungs *Can occur when TB lung lesions erode pulmonary veins Results in: hematogenous dissemination of tubercle bacilli * Miliary spread limited to the lungs can occur following erosion of pulmonary arteries by TB lesions lung lesions.







Edge of a granuloma is shown here

1] composed of the necrotic elements of the granuloma as well as the infectious organisms. [2] inflammatory component: epithelioid cells, lymphocytes, and fibroblasts



A stain for Acid Fast Bacilli is done (AFB stain or ziehl-neelsen stain) to find the mycobacteria

• The mycobacteria stain as red rods, as seen here at high magnification.



rounded outlines contains several Langhan's giant cells. . Granulomas are composed of transformed macrophages called epithelioid cells along with lymphocytes ., plasma cells, and fibroblasts

The pyknotic nuclei of epithelioid cells in the center of the granuloma (apoptotic bodies) are a precursor of necrosis.


4.TUBERCULOUS LYMPHADENITIS GROSS Appearance

Tuberculous Lymphadenitis – Cut Section

Section of a lymph node with connective tissue capsule and lymphoid tissue



Tuberculous Lymphadenitis

Chronic granulomatous lymphadenitis: secondary to tuberculosis

*Enlarged right cervical lymph nodes *Discharging sinus





4.TUBERCULOUS LYMPHADENITIS Histological Appearance

Tuberculous Lymphadenitis

The granulomas consists of : 1-epithelioid cells 2-few langhan's giant cells (large cell with multiple peripheral nuclei) 3-peripheral rim of lymphoc Epithelioid/histiocytes Giant Cells Lymphocytesytes



Tuberculous Lymphadenitis

Many round and oval tubercles/ granulomas with or







NEOPLASIA - BENIGN



1. ADENOMATOUS POLYP (RECTUM/COLON)

POLYP: FINGER LIKE PRODUCTION.





A microscopic comparison of normal colonic mucosa on the left and that of an adenomatous polyp (tubular adenoma) on the right is seen here. The neoplastic glands are more irregular with darker (hyperchromatic) and more crowded nuclei





SMALL ADENOMATOUS POLYP (TUBULAR ADENOMA) ON A SMALL STALK IS SEEN MICROSCOPICALLY TO HAVE **MORE CROWDED, DISORGANIZED GLANDS THAN THE** NORMAL UNDERLYING COLONIC MUCOSA. <u>GOBLET</u> **CELLS ARE LESS NUMEROUS** AND THE CELLS LINING THE **GLANDS OF THE POLYP HAVE HYPERCHROMATIC NUCLEI**

> Adenoma on top, no goblet cells on top.



THIS ADENOMATOUS POLYP HAS A HEMORRHAGIC SURFACE (WHICH IS WHY THEY MAY FIRST BE DETECTED WITH STOOL **OCCULT BLOOD SCREENING) AND A LONG NARROW STALK.** THE SIZE OF THIS POLYP--ABOVE 2 CM--MAKES THE **POSSIBILITY OF MALIGNANCY MORE LIKELY, BUT THIS POLYP PROVED TO BE BENIGN**









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are soft, non tender, and mobile if it is small size.

and lobulated

HISTOLOGICAL VIEW



Summary lipoma : mature adipose tissue , benign, soft , yellow , most are superficial.

Small lipomas are mobile.

This picture shows an area of fat necrosis within a lipoma. The masses are comprised primarily of <u>mature adipocytes</u>. Histiocytes present within these areas should not be mistaken for lipoblasts











Hemangioma of the skin

Features:

-A tumour mass in the dermis .

-consists of large number of vascular spaces of varying shapes. -separated by connective tissue stroma



Gross Appearance



Capillary Hemangioma of the skin – LPF

Histological appearance

Features:

- -Vascular spaces are lined by the flattened endothelial cells .
- -some contain blood.
- -Delicate connective tissue stroma separated the capillary vascular

spaces.







The stain : By H/E





Histological appearance

Features:

-usually on an extremity .

-complicated by thrombocytopenic purpura. tract.



Cavernous Hemangioma of Skin – HPF

-Blue rubber bleb nevus syndrome: cavernous hemangiomas of the skin and gastrointestinal



4.Teratoma (dermoid cyst) of the ovary





Gross Appearance

Features :

-filled with greasy material (keratin and sebaceous secretions) and shows tufts of hair. -The rounded solid area at the bottom is called Rokitansky's protruberance. -Microscopically, showed foci of neural tissue



Ovary: Mature Cystic Teratoma





Ovary: Mature Cystic Teratoma

Histological appearance

They consist of epidermis, <u>hair follicles</u>, sweat and sebaceous glands and neuroectodermal derivatives

Stratified Squamous epithelium with underlying sweat glands, sebaceous glands, hair follicles, columnar ciliated epithelium, mucous and serous glands and structures from other germ layers such as bone and cartilage, lymphoid tissue, smooth muscle and brain tissue containing neurons and glial cells



NORMAL Ovary

PATHPEDIA.COM





5. INTRADERMAL NEVUS(MOLES)



Causes:

- 1- exact reason is unknown
- 2- maybe Somatic mutations in RAS.



Histological

Features:

-small round or spindle shaped nevus cells

in the upper dermis.

few melanophages in the upper

Intradermal Nevus - <u>LPF</u>









Appearance

-cells contain varying amount of brown melanin pigment. No junctional activity.

Intradermal Nevus - <u>HPF</u>



6. MULTIPLE UTERINE LEIOMYOMATA

| Gross | |
|--|---------------------------------|
| Features: <pre> the multiple Smooth muscle tumors Seen in submucosal-intramural- subserosal. </pre> | Features: tumour uterus w |
| | |

Appearance

mass in the muscle coat of vithout a definite capsule.

Cause:

noncancerous growths of the muscle tissue of the uterus(Benign Tomur)





Normal Tissue



Features:

-well-differentiated. than the normal. the tumor mass.



Histological Appearance Features: -spindle shaped with the leiomyoma is hardly appears different elongated nuclei and eosinophilic cytoplasm. Bundles of smooth muscle are interlacing in



7. ENCHONDROMA OF THE (FIBULA)

Gross(X-RAY)



Appearance



Features:

intramedullary bone expansion.







Histological

Features:

cells irregularly distributed.

pale blue homogenous matrix.

in pairs or in tetrads.

Few bony trabeculae included in tumour.

Enchondroma - LPF



Features:

Condrocyte nuclei tend to be small. round and hyperchromatic nuclei. Irregular purple granules within the matrix represent calcifications





NEOPLASIA – MALIGNANT



1.ADENOCARCINOMA OF THE LARGE INTESTINE

Gross

the sigmoid colon

Features:

arising in a villous adenoma.
 Polypoid and fungating lesion showing a hemorrhagic area on its surface
 Lap test to confirm the diagnosis: occult blood in feces and carcinoma embryonic antigen (CEA)



Appearance

Features:

*exophytic(خارجي التنبت) in its growth pattern

(يعني قاعد ينمو من الاطراف الخارجية)



Cause:

Rare disorder called FAP (Familial adenomatous polyposis)

If not treated...it will cause this cancer



1.ADENOCARCINOMA OF THE LARGE INTESTINE

Histological appearance

Features:

• A moderately differentiated colonic adenocarcinoma arising from a villous adenoma



Histological

Adenocarcinoma of the Colon - LPF

Features:

Acini are lined by one or several layers of neoplastic cells with papillary projection.



Appearance

Adenocarcinoma of the Colon - HPF

Features:

 neoplastic glands have crowded nuclei with hyperchromatism and pleomorphism.
 No normal goblet cells are seen.









GROOS APPERANCE

Leiomyosarcoma



Cut surface of this leiomyosarcoma showing <u>ill defined pale</u> and <u>soft</u> large fleshy mass with <u>hemorrhage</u> and <u>necrosis</u>.

Leiomyosarcoma of Small Intestine



- Large mass showing ill defined borders.
- Pale firm and partially hemorrhagic cut surface with focal necrosis.



Histological

<u>Leiomyosarcoma – HPF Microscopy</u>

Features:

 The cell of origin is smooth muscle cell , Immunohistochemical staining for the malignant cells are desmin and actin.
 Marked atypia and cellularity with multiple mitoses present. Classic features of leiomyosarcoma including <u>cigar shaped</u> <u>nuclei</u> and <u>arrangement of cells in fascicles are seen</u>.



Appearance

Leiomyosarcoma of the Uterus - HPF Features: Pleomorphic malignant cells Multinucleation. Mitotic figures or mitosis. No normal goblet cells are seen.





3- Squamous cell carcinoma of the skin

00



SQUAMOUS CELL CARCINOMA - GROSS



SQUAMOUS CELL CARCINOMA (SCC) IS THE SECOND MOST COMMON CANCER OF THE SKIN A SORE THAT DOES NOT HEAL OR ANY CHANGE IN AN EXISTING MOLE, WART, OR SKIN LESION CAN POINT TO SCC. THERE MAY BE AN ULCER OR REDDISH SKIN PLAQUE THAT GROWS VERY SLOWLY, MAY BLEED OCCASIONALLY (ESPECIALLY IF LOCATED ON THE LIP), MAY HAVE AN ULCERATED CENTER WITH RAISED, HARD EDGES, MAY HAVE A PEARLY QUALITY WITH TINY BLOOD VESSELS, IS COMMONLY PRESENT ON SUN-EXPOSED AREAS (BACK OF HANDS, LIP

(USUALLY A SMALL ULCER WHICH WILL NOT HEAL AND BLEEDS SPORADICALLY, EARSAND THE SCALP.





Squamous Cell Carcinoma - Histopathology





The normal squamous epithelium at the right merges into the squamous cell carcinoma at the left, which is infiltrating downward. The neoplastic squamous cells are still similar to the normal squamous cells, but are less orderly.

Here is a moderately differentiated squamous cell carcinoma in which some, but not all, of the neoplastic cells in nests have pink cytoplasmic keratin

Squamous Cell Carcinoma - HPF



At high magnification, this squamous cell carcinoma demonstrates enough differentiation to tell that the cells are of squamous origin. The cells are pink and polygonal in shape with intercellular bridges. The neoplastic cells show pleomorphism, with hyperchromatic nuclei. A mitotic figure is present near the center





Squamous Cell Carcinoma - HPF



A mitotic figure is seen here in the center, surrounded by cells of a poorly differentiated squamous cell carcinoma, with pleomorphic cells that have minimal pink keratinization in their cytoplasm. In general, mitoses are more likely to be seen in malignant neoplasms



The dermis is infiltrated by masses of well differentiated neoplastic squamous cells separated by fibrous tissue stroma with chronic inflammatory cells. Tumour cells show pleomorphism, hyperchromatism and many mitotic figures . Pinkish laminated keratin pearls (epithelial cell nests) are present in the center of some cell masses

