Cell Injury

Lecture 2,3& 4

PATHOLOGY

TEAM 433

Red is definitions. Orange is listings. Green is examples.Blue is hyperlinks. Purple is notes to

NOTE THAT YOU MUST BE FAMILIER WITH"THE CELL STRUCTURE".

Objectives:

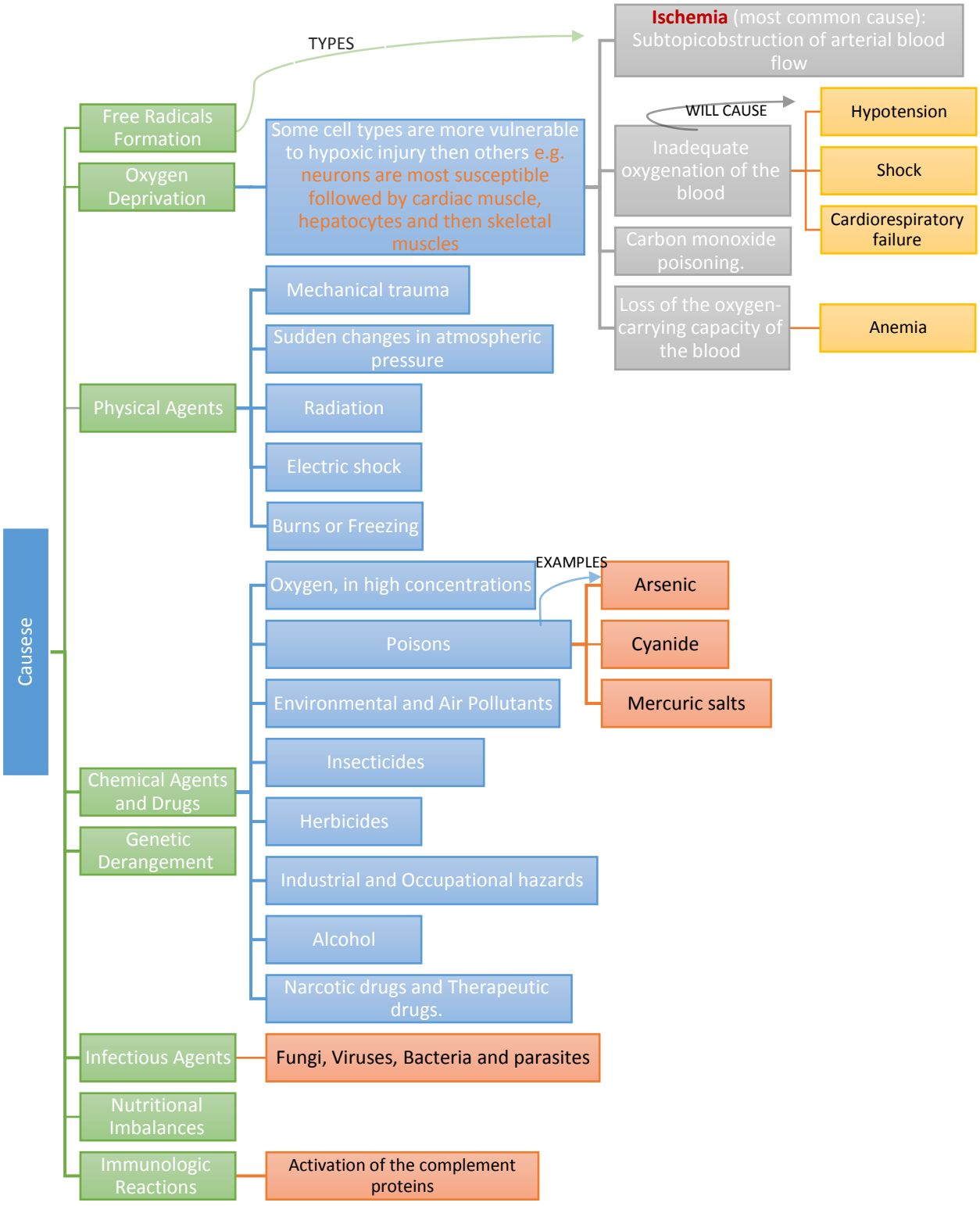
At the end, you should be able to:

- **Define cell injury.**
- **Describe the causes and the mechanism of cell injury.**
- **Differentiate between reversible and irreversible cell injuries.**
- **Differentiate between Necrosis and Apoptosis.**
- **Knowing each type of necrosis.**
- **Define Cell adaptation and its types and differentiate between them.**
- **Define Accumulation and describe it.**

Cell injury:

Cell injury can result in death of individual cells, tissue or organ failure and/or death of the organism.

Causes of cell injury:



Ischemia:

It is a poor blood supply to a certain organ, and that will lead to hypoxia or hypoxemia.
Hypoxemia: low oxygen concentration in the blood.
Hypoxia: low oxygen concentration in an organ or in the body.

Immunological reaction:

(E.g. activation of the complement proteins)
Complement proteins secreted by the liver. Those proteins are activated when there is an antibody or antigen reactant.

How does ischemia effect the cell?

It causes an increase in cytosolic calcium concentration. Increased Ca_2 in turn activates a number of enzymes that cause damage to the cell. Therefore, the cell will break down by the increased enzymes.

Free Radicals Formation(IMPORTANT)

Free radical, most of the time it is an oxygen atom, which has lost electron from its outer orbit (O^-).

When it loses an electron, it becomes unstable and**interacts with any biochemical reaction within the cell**, which will lead to disruption in the cell.

It can be produced in cells in response to a variety of processes including **radiation** OR **normal metabolic oxidation reactions** and **drug metabolism process.**

Types of free radicals:

I- Super oxide (O^-).
II- Hydrogen peroxide (H_2O_2).
III- Hydroxyl ion (OH^-)

These types formed according to the tissue or cell injury.

NOTE:the human body does not effected due the normal metabolic oxidation reaction, and the because of radical-scavenging

1- Antioxidants.
2. Enzymes, which break down hydrogen peroxide and superoxide anion

Any imbalance between free radical-generating and radical-scavenging systems results in oxidative stress causing cell injury.

Mechanisms of Cell injury:

Cell Membrane Damage	Mitochondrial Damage	Ribosomal Damage	Nuclear Change	Depletion of ATP
This disruption will cause imbalance in homeostasis. The disruption causes by: 1- Immunological reaction. 2- Certain drugs or toxins. 3- Free Radicals Formation.	Mitochondria is necessary for aerobic respiration of the cell. And it can be damaged by: 1- Drugs and toxins, especially cyanide which is lethal and attack the liver cells but its mechanism is attacking the mitochondria. 2- Free Radicals Formation.	Ribosomes are necessary for protein synthesis, it is found excessive in the liver cells. It can be damaged by: 1- Alcohol. 2- Drugs or toxins. E.g. Barbiturates.	It can be damaged by: 1- Viruses. 2- Radiation (which forms free radicals) 3- Free Radicals Formation.	ATP is required for normal function within the cell. ATP is produced in two ways. 1- Oxidative phosphorylation of ADP in mitochondria. 2- The glycolytic pathway, which generate ATP in absence of oxygen using glucose derived from body fluids or from glycogen.

Response of cell Injury:

1. reversible	2. irreversible
• Cytoplasmic eosinophilia • less ATP and Protein synthesis • Mitochondrial and ER swelling • Cytoskeletal damage, Cytoplasmic swelling and vacuolation. • DNA damage **damage can be reversed**	• Severe Mitochondrial vacuolization • Sever Damage of plasma membrane • Swelling of lysosomes • Nuclear damage : 1. Pyknosis (shrinkage) 2. Karyolysis (dissolution) 3. Karyorrhexis (break down) **irreversible**

Two phenomena consistently characterize irreversibility. They are: 1) The inability to reverse mitochondrial dysfunction even after removal of the original injury. 2) Profound loss in membrane function.

Irreversible cell injury or CELL DEATH: (cell death:is the ultimate result of cell injury, *irreversible* cell injury)

There are two types of irreversible cell injury:

1- **Necrosis.**
2- **Apoptosis.**

The enzymes used in this degradation are derived either:
1. from the lysosomes of the dead cells themselves, in which case the enzymatic digestion is referred to as ***autolysis***
2. from the lysosomes of immigrant leukocytes, during inflammatory reactions referred to as ***heterolysis.***

Necrosis:

Necrosis: Death and degradation of cells from severe environmental insult. Itoccurs after ischemia and chemical injury(Only occurred by a pathological reason)
Necrosis is characterized by changes in cytoplasm and nuclei of the injured cell.
Cytoplasmic major change → Increasing of eosinophilia (pink staining from the eosin dye)
Nuclear Changes:

Pyknosis → Karyorrhexis → Karyolysis

Remember:

Pyknosis: nuclear shrinkage.
Karyorrhexis: the nucleus undergoes fragmentations.
Karyolysis: the dissolution (تحلل) of the nucleus.

With the passage of time (a day or two), the nucleus in the necrotic cell totally disappears.

Types of cell necrosis

1- Coagulative.
2- Liqufactive.
3- Gangrene.
4- Fibrinoid.
5- Caseous.
6- Fat.

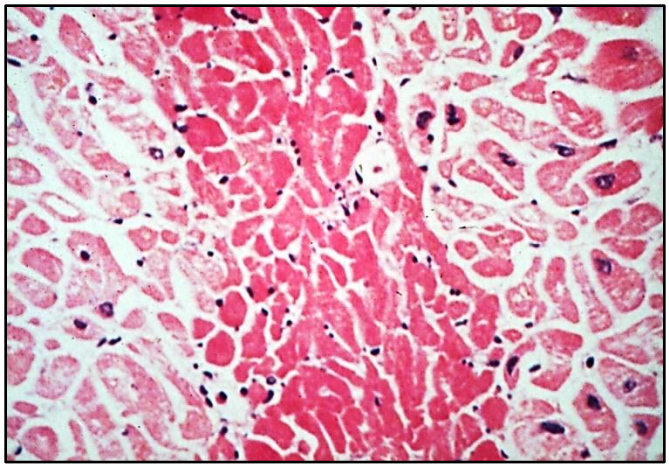
1-Coagulative necrosis: **Coagulative necrosis is irreversible cell damage the most important cause for it is local Ischemia. Typically seen in kidney and heart.**

Seen in all tissues except the brain.

Examples:

A- Myocardial infarction:

Under the microscope, the damaged cells have no nuclei (karyolysis) so the cells are dead.



in the wall of the left ventricle there is a large area containing hemorrhage and areas of soft yellowish tissue which is the necrotic cells ⟹ irreversible cell injury.

Coagulative cell injury:is cell necrosis (irreversible cell damage). It is an acquired condition might be caused by smoking, diabetes, overweight etc.

The Mechanism:

Local ischemia → Coagulative Necrosis → Irrevesible cell damage → Necrosis (Cell Death)

You can diagnose a patient with myocardial infarction through:

1- Increasing in troponini (protein) level.
2- CPK-MB test, (i.e. phosphocreatine kinase MB type), its increasing indicates to myocardial infarction.

Symptoms of myocardial infarction:

Chest pain, low blood pressure.

B- Infarction of kidney:

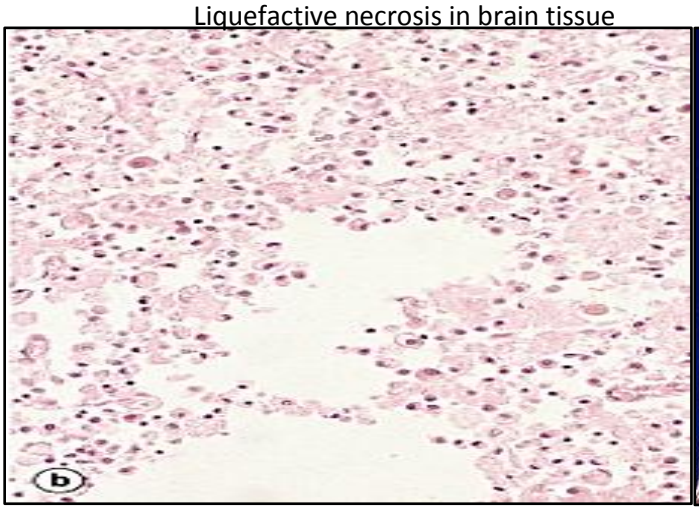
Glomerulus is dead and there are nuclear fragments (karyorrhexis of the nucleus) and karyolysis and pathological apoptosis.

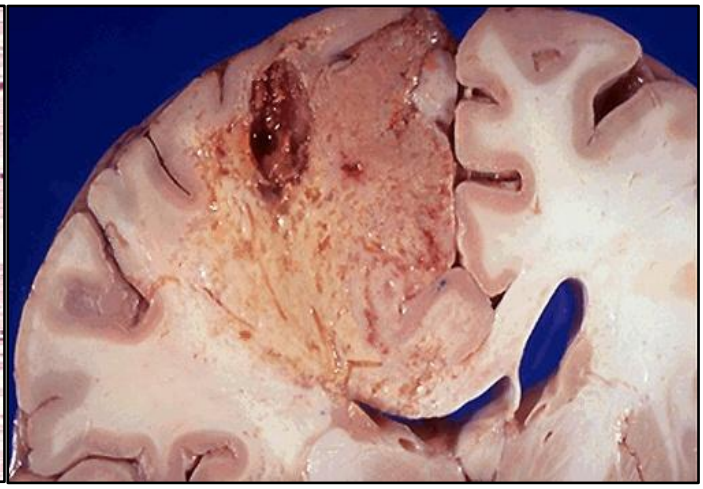
2-Liquefactive necrosis: **Occur in organs which are rich in water, It is a result from a release of hydrolytic lysosomal enzymes.**

It usually seen in the brain.

Example:

Liquefactive necrosis in brain tissue



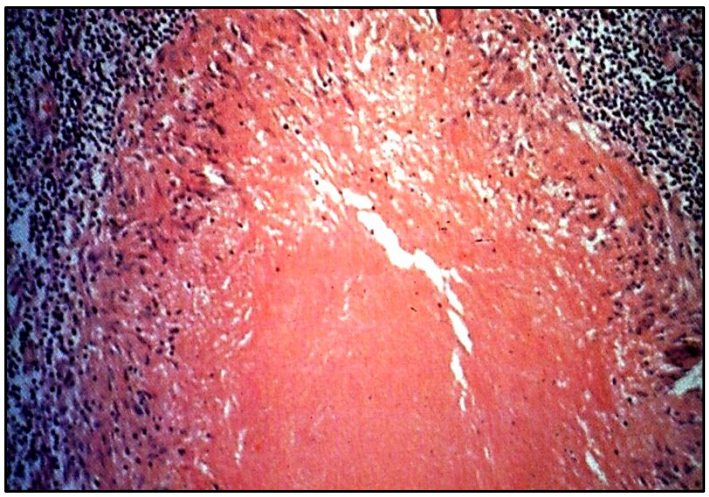


-Brain infarction:

Within few minutes the brain get irreversible cell damage (necrosis) while other organs can last for few hours before they die, susceptibility in necrosis differs between organs.

-Another example of Liqufactive cell necrosis is abscessالصديد caused by bacterial infection so the tissue becomes very friable
مفتت.

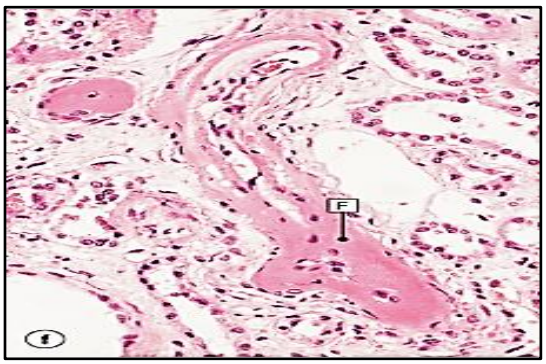
3- Caseous necrosis: **Only seen in TB (tuberculosisالدرن), it appearance is like cheese or white creamy material which is accumulation of the dead tissue and it is called Granuloma it can be found also in lymph node.**



Symptoms of swollen lymph node:

Swelling in lymph node, high temperature.

TB It is both high incidence and prevalence.



4-Fibrinoid necrosis: **Always happen in the blood vessels. It happens when the blood vessels get Vasculitis.(التهاب الأوعية الدموية)**

Vasculitis: **Is an Infection in blood vessels it is always because immunological reasons.**

The Mechanism:

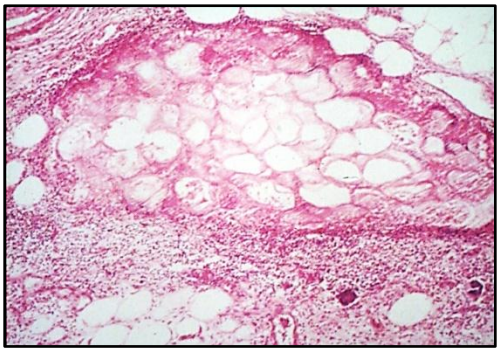
Formation of antigen and antibody → precipitating the immune complexes in the wall of blood vessels → Inflammatory reaction → cell injury

5-Fat necrosis:

Caused by:

A- Acute pancreatitis.

B- Trauma in fatty tissues.



Fat Necrosis

The Mechanism:

Inflammation in pancreas → Cell injury → irreversible cell change → Rapture of cytoplasmic membrane → Cell release its content to outside "Enzymes"

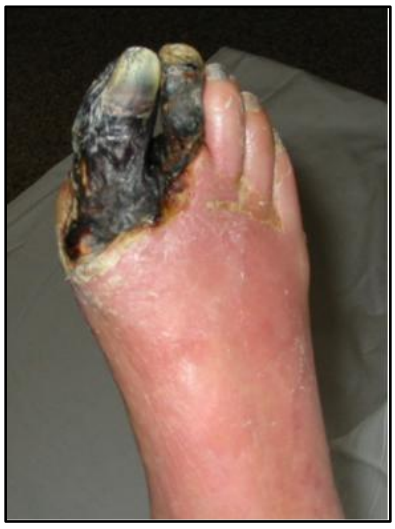
Two important enzymes get released from the pancreas, Amylase AND Lipase, these two enzymes are lypolytic. As a result of this process, we will get fat necrosis. This reaction (the necrotizing) called Saponification(التصبن). This reaction will attract calcium, which will cause calcification.

6-Gangrenous necrosis:

There are three type of gangrene: dry, wet and gas.

The first one is dry gangrene. ⟶

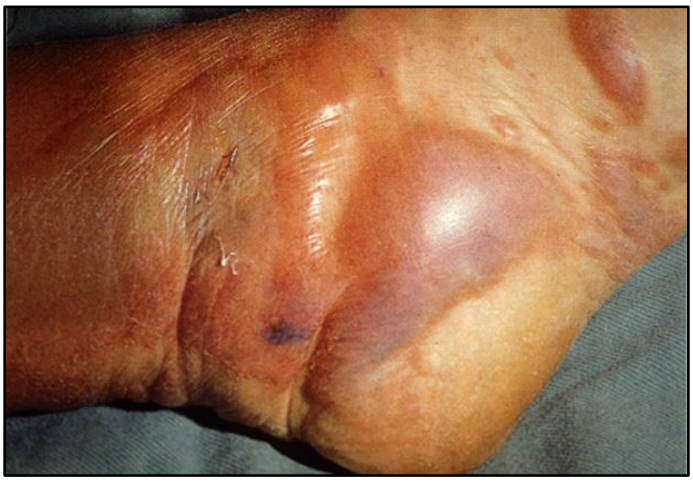
Case example: A woman has diabetes for 15 years. Heavy weight and she said she did not feel anymore the two dark toes All the cells in these toes are dead. Why? Because there is obstruction of the blood vessels.
why? Because she has atherosclerosis,
why she has very severe atherosclerosis? Because the diabetes she has will make the atherosclerosis worse and accelerate it.



The second is Wet gangrenes.

caused by Clostridiumgerms. It is rare, and only at battlefields. (*e.g. civil war in Syria*) The mechanism is when the wound get soiled.

The third one is gas gangrene. ⟶



Caused by a bacterial exotoxin-producing clostridial species, which are mostly found in soil and other anaerobes causing petrifaction, and it can be known or diagnosed by the smell.

The yellowing parts are swelling cause by gas gangrene. Gas gangrene is also rare.

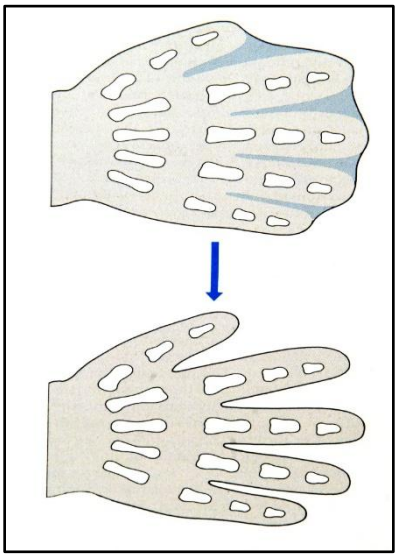
- **Autolysis: is disintegration of cells or tissues by autologous enzymes, it can be seen in cells after death of the organism and in some pathologic conditions**
- **Caseous: is derived from the cheesy white gross appearance of the area of necrosis**
- **Granuloma:the body tries to wall off and kill the bug with macrophages)**

Apoptosis:

Apoptosis is programmed cell death.
Apoptosis means "falling off".(when cell decide to kill itself).
It occurs normally/physiologically or pathologically unlike necrosis which occurs only pathologically.
The cell's plasma membrane remains intact, but its structure is altered in such a way that the apoptotic cell sends signal to macrophages to phagocytose it.

Physiologic Situations:

- **Embryogenesis (excess skin between the fingers and toes).**
- **During breastfeeding.**
- **Menstruation.**
- **Red blood cells after 120 days.**
- **Lymphocytes.**



Pathologic Conditions:

- **Viral diseases.**
- **Cancer.**
- **Ducts of salivary glands.**

Mechanism of Apoptosis:

- **Cell shrinkage.**
- **Chromatin condensation.**
- **Formation of cytoplasmic blebs and apoptotic bodies.**
- **Phagocytosis of apoptotic cells or cell bodies, usually by macrophages.**

Apoptotic cell dies and get digested by its own enzymes (Caspases) and converted to small fragments, then the adjacent (القريبة) cell will engulf these fragments.

Important enzymes of apoptosis

There are enzymes activate cell to kill itself.

1- Caspases

2- Endonucleases

Apoptotic bodies: can be disposed by the phagocytosis either by macrophages or uniquely by neighboring normal cells.

Features	Necrosis	Apoptosis
Damaged cells number	More	Less
Cell size	Enlarged	Reduced (Shrinkage)
Nucleus	Pyknosis → Karyorrhexis → Karyolysis	Fragmentations into nucleosomes size fragments
Plasma membrane	Disrupted	Intact
Adjacent inflammation	Frequent	No
Physiologic or pathologic role	Invariably (Always) Pathologic	Often physiologic

Cell adaption:

A new steady state and preserving viability and function.
The principles adaptive response are *hypertrophy*, *hyperplasia*, *atrophy,metaplasia, Hypoplasia AND aplasia.*

NOTE: HYPOPLASIA AND APLASIA RELATED TO DEVOLOPMENT DISORDER THAN BEING AN ADAPTIVE RESPONSE.

***Hypertrophy:*increasing in the cell size, which will lead to increased organ size.**

***Hyperplasia:* increasing in the numbers of cells, which will lead also increasing in organ size.**
It takes place if the tissue contains cell population capable of replication.

***Atrophy:*shrinkage in the size of the cell because of loss of cell substances, or loss cell number, which will lead to shrinkage of an organ.**

- **All of the above could occur by both Pathological and Physiological reasons.**
- **Hypertrophy and hyperplasia can occur together and the leads to an enlarged (hypertrophic) organ.**

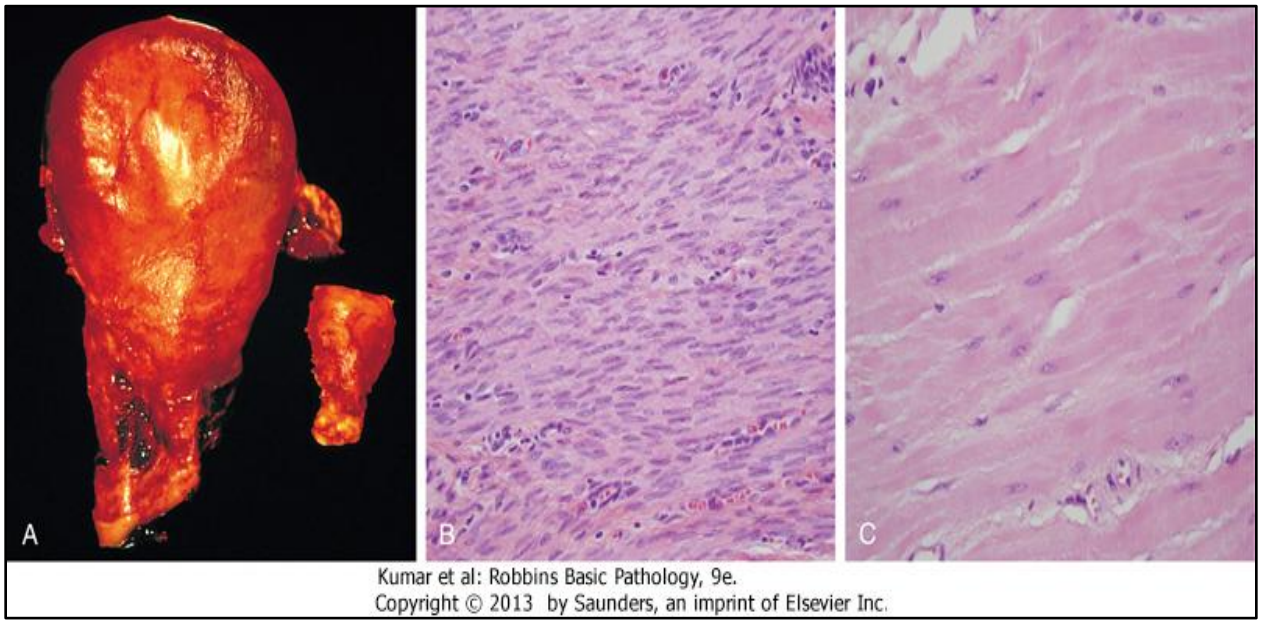
***Metaplasia:* A change in which one adult cell type is replaced by another adult cell type.**
***Hypoplasia:*Abnormal development of organ. (Refers that the organ does not reach its full size)e.g. partial lack of growth and maturation of gonadal structures in Turner or Klinefelter syndrome.**

Aplasia: **the failure of cell production. e.g.during fetal growth, aplasia can lead to agenesis of organs and aplasia in bone marrow (pathological) can cause aplastic anemia.**

Agenesis: Failure of development of an organ during embryonic growth.

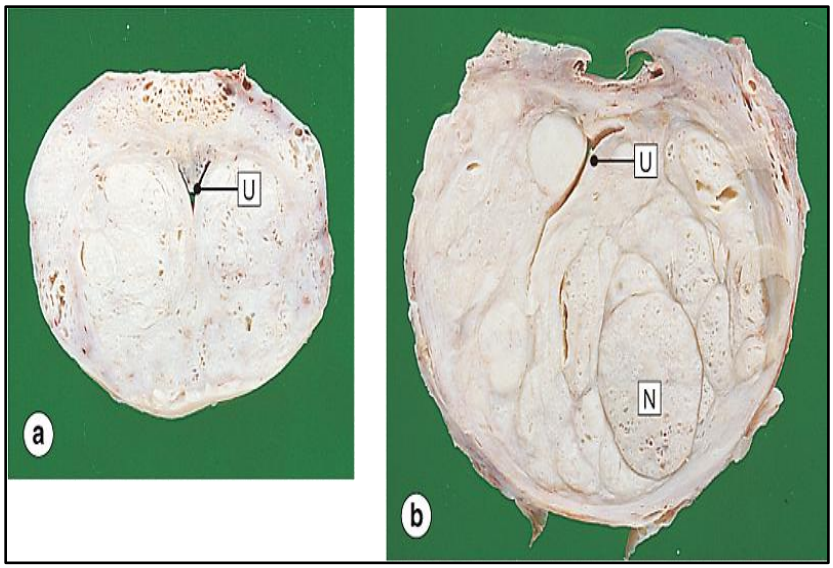
Examples:

1- Hypertrophy:



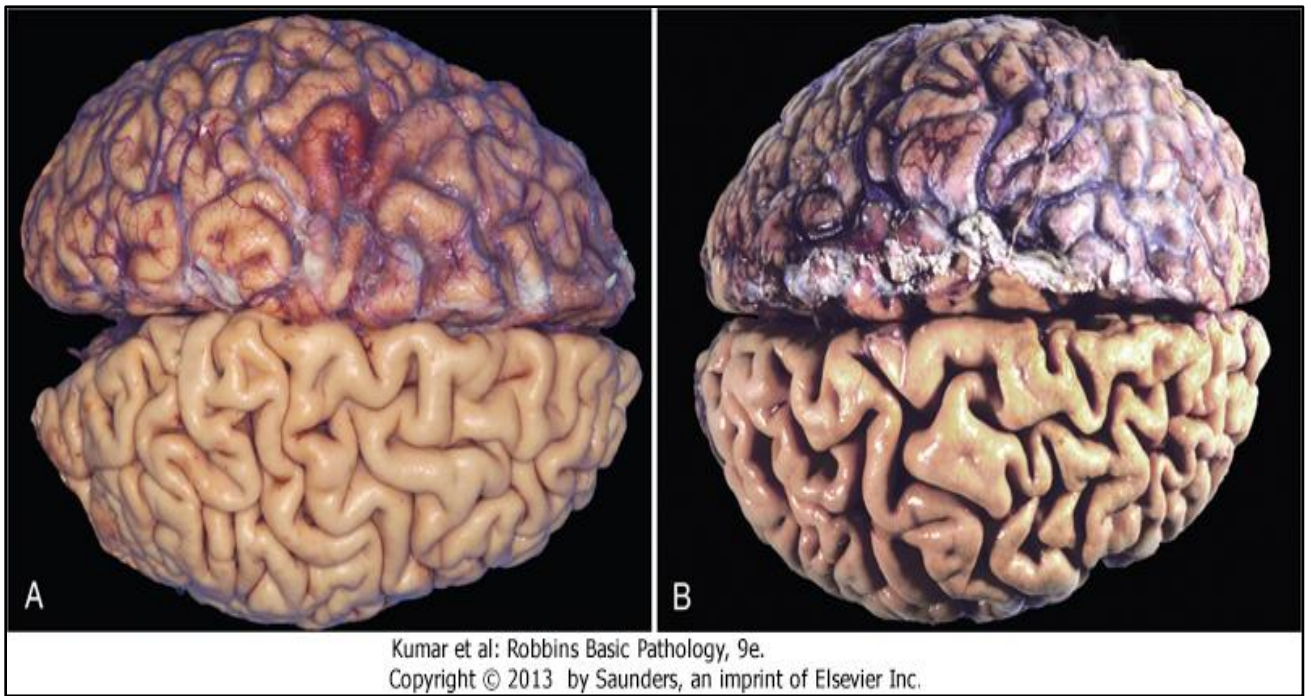
Physiologic hypertrophy of the uterus during pregnancy. A, Normal uterus (right) and a graviduterus (left). B, smooth muscle cells from a normal uterus. C, Large, plump hypertrophied smooth muscle cells from a gravid uterus.

2- Hyperplasia:



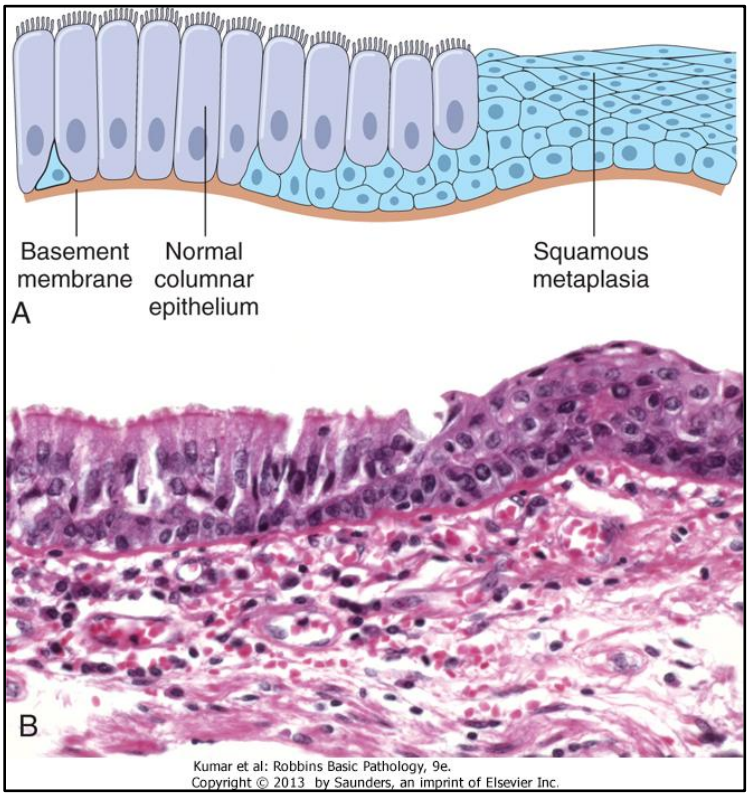
A) Normal prostate gland.
B) Nodular hyperplasia of prostate gland.

3- Atrophy:



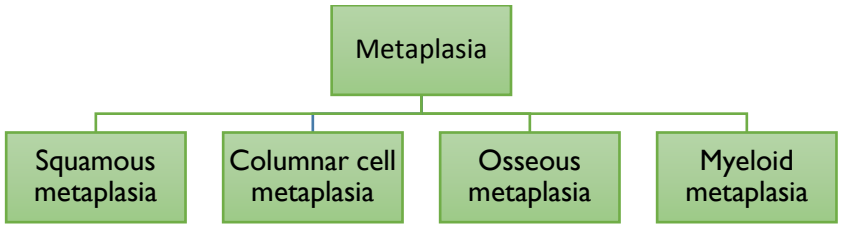
Atrophy as seen in the brain. A, Normal brain of a young adult. B, Atrophy of the brain in an 82-year-old man with atherosclerotic disease. Atrophy of the brain is due to aging and reduced blood supply.

4- Metaplasia:



Metaplasia of normal columnar (left) to squamous epithelium (right) in a bronchus. (A) Schematically (B) Histologically.

Types of metaplasia:



Metaplasia	Squamous metaplasia	Columnar cell metaplasia	Osseous metaplasia	Myeloid metaplasia (extramedullary hematopoiesis)
The replacement of one mature type of cells by another mature type of cells.	In cervix: replacement of columnar epithelium at the squamocolumnar junction of the cervix by squamous epithelium.	The replacement of the squamous lining of the esophagus by columnar cells	The formation of new bone at sites of tissue injury.	The proliferation of hematopoietic tissue in sites other then the bone marrow such as liver or spleen.
A reversible change provided the causative stress factor is removed	In respiratory tract : The columnar epithelium of the bronchus is replaced by squamous cell	It is seen in chronic gastric reflux in which the normal stratified squamous epithelium of the lower esophagus undergoes metaplastic transformation to columnar epithelium	Cartilaginous metaplasia may also occur.	
Type of cellular adaptation		This change is called as Barrett's oesophagus		

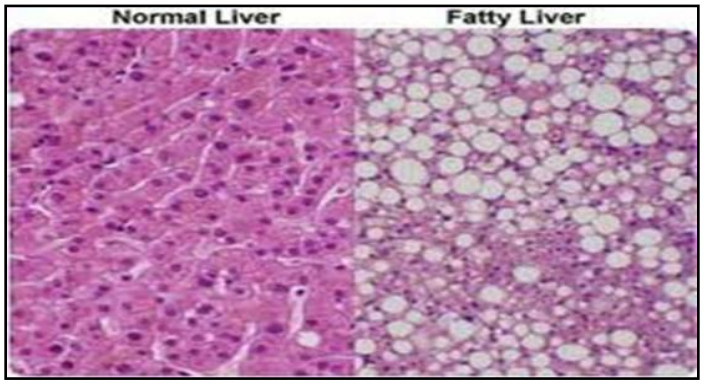
	Hypertrophy	Hyperplasia	Atrophy
Pathological Causes	- Increased resistance e.g. Cardiac enlargement that occurs with hypertension. - Physical obstruction e.g. hypertrophy in bladder smooth muscle caused by enlarged prostate gland.	- Excessive hormonal or growth factor stimulate. - Cell destruction.	- Denervation (lack of nerve stimulation). - Reduce blood supply, e.g. shrinkage of brain. - Inadequate nutrition, wasting muscle in starvation. -Decreased workload.
Physiological Causes	- Increased workload e.g. bodybuilding. - Hormone Stimulation e.g. The pregnant uterus.	- Hormonal, e.g. Female breast at puberty. - Cell loss, e.g. regeneration of the liver after partial hepatectomy.	- Reduction of endocrine stimulation.

ACCUMULATIONS

1- ***Fatty change*(Steatosis) is the abnormal accumulation of triglycerides (usually seen in the liver).**

The causes of steatosis include:

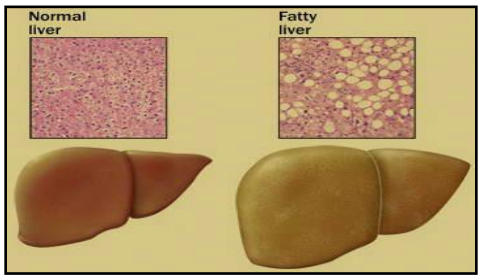
- **Toxins/poisoning.**
- **Protein malnutrition.**
- **Diabetes mellitus.**
- **Obesity.**
- **Anoxia.**
- **Alcohol abuse.**
- **Starvation.**
- **Pregnancy.**
- **Anorexia nervosa.**



Accumulation of lipids
All major classes of lipids can accumulate in cells: Triglycerides, Cholesterol/Cholesterol esters, and phospholipids.

Morphology of Steatosis:

-Gross appearance: Liver with progressive accumulation enlarges and becomes increasingly yellow and greasy.
-Light microscopy: fatty cysts – nucleus appears smaller.
Lipids appear like empty bubbles.



2- **Accumulation of cholesterol: Accumulation of cholesterol in the form of intracellular vacuoles, are seen in several pathologic processes e.g. atherosclerosis.**

3- **Accumulation of pigments:Pigments are colored substances, they can be exogenous, coming from outside the body, or endogenous, synthesized within the body itself.You should also know that a pigment is a sign of a disease.**

A- Endogenous Pigments

I- **Lipofuscin:** is an insoluble pigment, also known as wear-and-tear or aging pigment. It is seen in heart muscle and in the liver.
It caused by aging process and does not have harmful effect.

II- **Melanin:** is responsible for the color of our skin. The function of melanin is to prevent the harmful effects of UV light.
Cause:will happened due to accumulations of melanin in colon (Not harmful).

III- **Bilirubin:**is a yellow colored pigment that the liver produces when red blood cells are broken down and recycled.
Signs of accumulation of bilirubin:**1-**Yellowish green skin (Jaundice).**2-** Yellow eye.**3-** Urea is red because of the absence of bilirubin.
Causes of this accumulation: **1-** Pre-Hepatic: Hemolysis.
2- Hepatic: Viral hepatitis.
3- Post-Hepatic: Obstruction of bile.

IV- **Haemosiderin:**is a hemoglobin-derived, golden yellow-to-brown found in the lysosomes within the cell cytoplasm. It is composed of aggregates of partially degraded ferritin that is protein-covered ferric oxide and phosphate when there is an excess of iron, iron-containing pigment in cells.
Morphology of Haemosiderin Iron can be visualized in tissues by the Pearl. Prussian blue, in which it appears blue-black.

Primary Haemochromatosis(GENATIC & Rare): Is an inherited disease in which there is excessive accumulation of iron and widespread deposition of haemosiderin in the tissues especially the liver, pancreas and skin. The iron is toxic to the tissues and leads to fibrosis of the liver (cirrhosis) and pancreas (leading to diabetes mellitus).
Secondary Haemochromatosis(NON GENATIC & COMMON): Accumulations of irons in cells due to high frequent of blood transfusion.

B- Exogenous Pigments The most common exogenous pigment is carbon, coal dust and Tattooing

4- Calcification:

1- **Dystrophic:** Accumulations of calcium in blood vessels due to disease or genetic conditions or necrosis. E.g., Fat necrosis attract calcium that will lead to dystrophic calcification.

2- **Metastatic:** Occurs within normal tissues because of Hypercalcaemia. Hypercalcaemia can be caused by:

A- Hyperparathyroidism.
B- Vitamin D intoxication.
C- Metastatic malignant tumors.
D- Milk-alkali syndrome.
E- Aging.

Morphology of pathologic calcification (dystrophic or metastatic, both look the same):
Ca deposition occurs anywhere in the body. In the course of time, **heterotopic**(is the process by which bone tissue forms outside of the skeleton) bone may be formed due increasing of calcification.
Psammoma body: is a type of dystrophic calcification made up of round collections of calcium. They are seen in papillary cancers in the body (e.g. thyroid, ovary, and kidney) and in meningioma of the brain.

	Dystrophic calcification	Metastatic calcification
The deposition of calcium	In locally in dying or injured tissue	Normal tissues
Calcium level	Normal	Hypercalcaemia
Calcium metabolism	Normal	Abnormal
Seen in	-In the atheromas of advanced atherosclerosis. -In aging or damaged heart valves. -Tuberculosis. -In fat necrosis. -Psammoma body. -Areas of trauma.	Throughout the body, but especially in kidneys, lungs,gastric mucosa and vasculature.

5- Amyloid:

Amyloid is found in the liver, it is produced to react against inflammation.

1- **Primary amyloidosis (AL):**

Plasma cells produce immunoglobulin that has two type of chains Heavy and Light.

So, tumor in plasma cells may cause increasing in number of light chains which produce amyloid (AL)

2- **Secondary amyloidosis (AA):**

Caused by the presence of other disease like TB.

NOTE: TUMOR IS AN ABNORMAL REPRODUCTION OF CELLS.

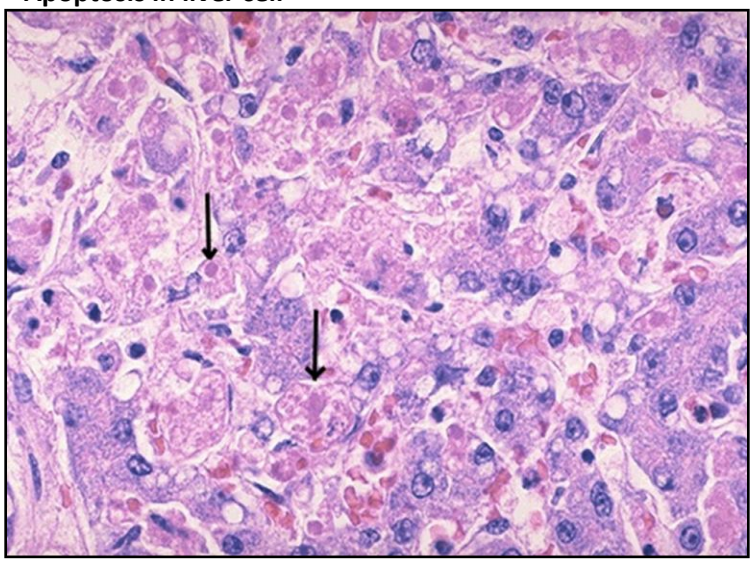
SUMMARY AND IMPORTANT NOTES:

- **MORPHOLOGY:**

It is a branch of bioscience dealing with the study of the form and structure of organisms and their specific structural features and the changes that could be occur on it.

Other Examples for apoptosis:

Apoptosis in liver cell



Apoptosis in epidermis.

Definitions

Necrosis: is the type of cell death that occurs after ischemia and chemical injury, and it is always pathologic.

Apoptosis: occurs when a cell dies through activation of an internally controlled suicide program.

Accumulation It is high abnormal amounts of various substances.

Cell adaption: A new steady state and preserving viability and function.

Intracellular Accumulations

Pigment: The substance may accumulate in either the cytoplasm or the nucleus.

An abnormal substance increasing: 1- Exogenous mineral or products of infectious agents.
2- Endogenous such as a product of abnormal synthesis or metabolism.

A normal cellular constituent accumulated in excess
e.g.: water, lipids, proteins, and carbohydrates.

Types of necrosis:

Types of necrosis	Seen in	Microscopic	Gross	Due to	Other
Coagulative	See this in infarcts in any tissue (except brain)	Cell outlines are preserved (cells look ghostly), and everything looks red	tissue is firm	Due to loss of blood	
Fat	Seen in acute pancreatitis , breast fat	shadowy outlines of necrotic/dead fat cells; sometimes there is a bluish cast from the calcium deposits, which are basophilic	chalky, white areas	release of activated pancreatic lipases into the substance of the pancreas and the peritoneal cavity	damage cells release lipases , the released fatty acids combine with calcium to produce calcium soaps (fat saponification)
Liquefactive	seen in hypoxic death of cells within CNS	lots of neutrophils and cell debris	tissue is liquidly and creamy yellow because of the presence of dead white cells and is called pus	Due to lots of neutrophils around releasing their toxic contentsthe tissue. The result is liquid viscous mass.	characteristic of infections especially bacterial. transformation into liquid vascous mass
Caseous	Seen in tuberculosis infection.	Granuloma (amorphous pink granular debris surrounded by a collar of lymphocytes and macrophages).	Gross : White, soft, cheesy-looking (“Caseous”) material		1.The lung and lymph nodes are commonly involved and patients present with fever, night sweats and respiratory symptoms 2.type of coagulative necrosis
Fibrinoid	Seen in immune mediated vascular damage, malignant hypertension.	Vessel walls are thickened and pinkish-red.	changes too small to see grossly		Is marked by the deposition of fibrin like proteinaceous material in the arterial walls, which appears smudgy and acidophilic
Gangrenous	When an entire limb loses blood supply and dies (usually the lower leg).	-dry gangrene: there is coagulative necrosis from the loss of blood supply -wet gangrene: there is superimposed bacterial infection, and then liquefactive necrosis develops.	Skin looks black and dead; underlying tissue is in varying stages of decomposition and foul smells.		- This isn’t really a different kind of necrosis.Is a term used by surgoens. It is usually applied to a limb, generally the lower leg, that has lost its blood supply and has undergone coagulation necrosis. - The limb has to be amputated - The bacteria is usually gram positive Clostridia species

Q1: What is necrosis?
Death and degradation of cells from severe environmental insult.

Q2: What is apoptosis?
Programmed, energy- dependent cell death.

Q3: What are the six types of necrosis?
Coagulation, Caseous, liquefaction, gangrenous, fibrinoid and fat.

Q4: What nuclear change take place in necrosis?
1- Pyknosis (Condensation of chromatin).
2- Karyorrhexis (Fragmentation of nucleus).
3- Karyolysis (Lysis of nucleus).
4- Nuclear loss.

Q5: What are the steps involved in apoptosis?
1- Cell shrinkage.
2- Chromatin condensation.
3- Formation of cytoplasmic blebs and apoptotic bodies.
4- Phagocytosis of apoptotic cells or cell bodies, usually by macrophages.

Q6: What is Steatosis (Fatty Change)?
It is the abnormal accumulation of triglycerides inside parenchymal cells.

Q7: What type of necrosis is seen in tuberculosis granulomata?
Caseous necrosis.

Q8: What is pus?
Product of liquefaction necrosis: dead cell debris, PMNs, monocyte, lysosomal enzymes in an exudative, purulent soup.
PMNs stands for (Polymorphonuclear Neutrophils).

Q9: In which type of necrosis is tissue architecture preserved?
Coagulation necrosis.

Q10: What are the three types of gangrene?
1- Wet gangrene which associated with liquefaction necrosis.
2- Dry gangrene which associated with coagulation necrosis.
3- Gas gangrene.

Q11: What is the “wear-and-tear” pigment?
Lipofuscin. The breakdown product of lipids which accumulate in atrophic cells of elderly people as “brown atrophy”

Q12: Name three important free radicals.
1- Superoxide $O2^{-}$.
2- Hydrogen peroxide (H2O2).
3- Hydroxyl radical OH^{-}.

Q13: Immunological reactions cause cell injury, how so?
By activation of compliment protein.

Q14: How does a free radical cause a cell injury?
By interacting with biochemical reaction within the cell.

Q15: Name one of the toxins that effect the ribosomes in the liver cells.
Barbiturates.

Q16: what is the most common cause of coagulative necrosis?
Ischemia.

Q17: How can you diagnose a patient with myocardial infarction?
1- Increasing in troponin.
2- Increasing in CPK-MB

Q18: Where does liquefactive necrosis occur?
In rich water organs.

Q19: Which organ is usually effected by liquefactive necrosis?
The Brain.

Q20: What causes Caseous necrosis?
Tuberculosis.

Q21: Where does Fibrinoid necrosis take place in?
Blood vessels.

Q22: Two important enzymes released by pancreas when it gets a sudden inflammation, what are they?
1-Lipase. 2- Amylase.

Q23: Where does Lipofuscin take place in?
In the heart muscle.

Q24: What is the function of melanin?
Prevent the harmful effects of UV light.

Q25: What is the most common exogenous pigments?
Carbon or coal dust.

Q26: What are the difference between dystrophic and metastatic?
1- Dystrophic: Occurs within diseased tissues, with unknown reason.
2- Metastatic: Occurs within normal tissues because of Hypercalcaemia

For any question or a feedback, please connect us through this e-mail:
Pathology433@gmail.com