**Lec 2 Pathophysiology Dr.Baydaa Hameed**

**Morphological patterns of cell injury :**

**Reversible cell injury**

It is called also **degeneration** .It is accumulation of normal substances (glycogen ,water) or abnormal (Amyloid) inside the cell due to injury agent. classified into two main groups

1-those with primary change in the **cell.**

2-Those with **interstitial** accumulation compressing the cell.

**Classification of degeneration :**

**1-primary changes in the cell.**

1. Intracellular accumulation of water (cloudy swelling ,hydropic & vacuolar changes ).
2. Intracellular accumulation of fat.(fatty change)
3. Intracellular accumulation of CHO.
4. Intracellular accumulation of proteins.

**2-interstitial accumulation compressing the cells**.

1. Amyloid
2. Hyaline change.(hyaline degeneration)
3. Mucinous change.(Mucoid degeneration)
4. Fatty infiltration.(fatty degeneration)

**Intracellular accumulation of water :**

1-cloudy swelling :results from impaired cellular regulation for Na , when Na enters the cell ,water will follow to maintain the isoosmotic condition →cellular swelling .

This type of cellular degeneration occur in paranchymus organs such as liver, kidney and heart.

Etiology: 1-physical like heat in burn 2-chemical 3-bacterial

Hydropic change occur where water transfer is most active e.g. in renal tubular epithelium after I.V administration of hypertonic glucose or in hyperkalemia

**Intercellular accumulation of lipid**

**Fatty change**: is the accumulation of neutral fat within parenchynal cells. Seen in the liver, heart, skeletal muscle, kidney and others.

Why is it common in the liver?

Because the liver plays central role in fat metabolism. The fatty change may be mild reversible or producing severe irreversible cell injury and death. This depends on the cause and amount of fat accumulation.

**Etiology (cause)**

* Starvation
* Obesity
* Malnutrition
* Alcoholism
* Diabetes mellitus
* Chronic illness like T.B
* Late pregnancy
* Liver toxin
* Drugs estrogen, steroid, tetracycline

**Interstitial accumulations**

1. **Hyaline change**

It is glassy homogenous, eosinophilic material, could be seen intra or extra cellular and it is not specific substance. This type of degeneration occur after necrosis of tissue.

1-connective tissue: blood vessels fused together& it seems homogeneous mass.

2-Epithelial tissue: kidney, liver

**2**-**Fatty degeneration(stroma fatty infiltration** )

It differs from fatty change, it is the deposition of mature adipose cells in the stromal connective tissue. In obese patient.

**3-Amyloidosis**

Amyloid is fibrillar material which is laid down in the tissues, usually extracellularly associated with chronic inflammation.

**4-Mucoid degeneration**

This type is derived from mucus which is mucin like substances with jelly appearance. Normally mucin is secreted by goblet cells of columnary epithelium of respiratory tract &gastrointestinal tract.

**5-Fibrinoid degeneration**

The deposition of fibrin like protineaceous material in the arterial walls. Often this type of degeneration associated with immune mediated vascular damage.

**Irreversible cell injury:**

**Necrosis**

Is sequence of morphological changes that follows cell death in a living tissue and always associated with inflammation two important changes leads to irreversible cell injury, they are:

1. Cell digestion by lytic enzymes
2. Denaturation of proteins

**Cytoplasmic changes**

1. Appears homogenous
2. Intensely eosinophilic because A- loss of basophilic effect of RNA. B- binding of eosin to denatured proteins
3. Sometimes vaculuolation or calcification.

**Nuclear changes:**

1. **Pyknosis** ( condensation of nuclear chromatin)
2. **Karyolysis** ( dissolution of nuclear chromatin )
3. **Karyorrhexis** (fragmentation)

**Morphological types of necrosis**

**1-Coagulative necrosis**:( when denaturation predominates )

***Cause:*** sudden cut in blood supply in all tissues except the brain and less often from bacteria and chemical agents . e.g. myocardial infarction ,ischemia (local anemia)

**2-Liquefactive necrosis**

It frequently occurs in brain tissues and results from break down of neurons by released lysosomal enzymes resulting in formation of pockets of liquid, debris and cyst like structures in the brain tissue.

**Cause:** ischemia bacterial infection (autolysis)

**3-Caseous necrosis**

**Caseous Necrosis: -** is a good example of structureless necrosis. It is common in tuberculosis and is characterized by central area of necrosis which is soft, friable and surrounded by an area with a cheesy, crumbly appearance.

**4-Fat necrosis:** are two types

1. **Enzymatic :** occur in pancreas, caused by enzymatic digestion.

Pathogenesis: lipase activation and liberation will digest the fat and split it into

**Glycerol &F. a** which will unite with calcium → chalky white material.

1. **Traumatic :** occur in female breast caused by trauma

Pathogenesis trauma → adipocyte will rupture and liberate fat which will act as foreign material

Stimulate inflammatory reaction. The macrophage will surround the area and ingest the fat ( lipid laden macrophage), later fibrosis will occur this will stimulate carcinoma.

**5-gangerous necrosis**

It is death of tissue + putrefaction. It is coagulative necrosis + liquefactive if the coagulative necrosis is predominant, then it is a dry gangrene

If the liquefactive necrosis is predominant then it is wet gangrene.

1-**Wet gangrene**

**Site:** moist areas ( lung, vulva, diabetic foot)

**Cause:** block of an artery or vein with stasis of blood + bacterial growth

\* rapid development

\* NO line of demarcation between the normal and abnormal areas .the affected part is swelled

\* there may be a spread of infection to the circulation (septicemia)

2- **dry gangrene**

**Site:** distal parts of the foot ( arterial sclerosis)

\* it develops slowly

\* NO bacterial growth

\* there is a line of demarcation in which the dead area appears black color. The affected part is shrinked

3- **gas gangrene (wet gangrene )**

**Cause:** the infection is with gas forming m.o. e.g. clostridia which enters by :

Open wound or as complication of colonic surgery clostridia produce various toxins that lead to necrosis and edema and usually associated with systemic manifestations.