Date:...../...../20.....

NECROSIS

M T W T F S

→ Always pathological.

→ "Series of morphological changes in a lethally / irreversibly injured cells ——."

* changes occurs due to:
  ** Denaturation of Intracellular Structural + Functional Proteins.
  ** Enzymatic digestion of injured cells → Auto + Hetero lysis.
  ** Disruption of cell membrane.
     ↳ Intracellular components come out to Extracellular compartment → affects Sorrounding cells → Elicit → Inflammation.

→ These changes doesn't occurs suddenly rather they take many hours to occur.

→ If a Person dies early ie. 1-2hrs → there will be no evidance of Necrotic changes (e.g: If a Person → Coronary Artry blockage → → dies in (2) hrs → After death → no signs of necrotic changes in his myocardium)
(But due to membrane disruption → cardiac specific enzymes may leak out & enter General circulation → & can be detected as early as (2) hrs. These enzymes are C-Troponin-T, C-Troponin-I, CK-MB etc)

* Eosin → Pink (↓ Cytoplasm) * Basophilia / Blue → Hemitoxilin (↓ Nucleus, Ribosome)

★ Cytoplasmic Changes: (Light Microscope)

* → During Necrosis → cell ↑ becomes (more) (Eosinophilic) due to: (cytoplasm)
  * cytoplasmic proteins → denatures → take more Eosin.
  * Ribosome dis Integrates → Basophalia → decreases

(P-T-O)

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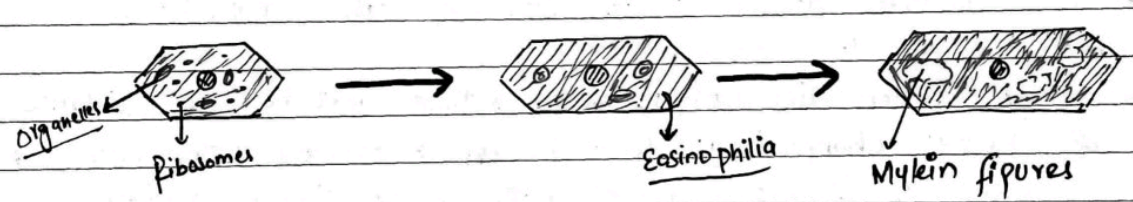
* During Stress → Glycogen rapidly converted into Glucose → → Glycogen granules → disappear → So Granular appearance of cytoplasm → vanishes → Glassy / Homogenous appearance.

* Also multiple organelles → disappears from cytoplasm → → empty spaces → Moth Eaten Appearance.

* Cell membrane / organelle's membrane → remain whorl like → → called → Myelin Figures
(Myelin Figures → either engulfed by marcophages or if they remain for long time → calicification occurs → Dystrophic (In T-B) ← ← calcification.)



Ultrastructural Changes: (under Electron microscope)

* Plasma Memb- → Ruptures → Both of cell & organelle's.

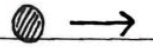
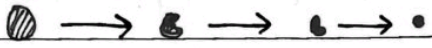
* Mitochondria swells up having dense amorphous bodies (Enzymes clumped)

* Organelle's memb- → Small multiple Myelin figures.

* Amphous Bodies in cytoplasm. (Protiens Denatured)

Darsi Notes

Nuclear Changes :

1. Nucleus → Fades away → Karyolysis
   
2. Nucleus → Condenses → Intensly condenses → Pyknosis
   
3. Nucleus → Condenses initially → Fragments → Karyorrhexis
   

Types Of NECROSIS

1. Coagulative Necrosis :

→ " When cells → lethally injured → their structural & functional protiens undergo denaturation Simultaneously —

e.g :

* Myocardial Infaraction

→ Hallmark of Coagulative Necrosis → cells maintain their Basic Arcticture & outline atleast for few days.

* Severe Ischemia / Hypoxia to many tissues except Brain →
  → causes Coagulative Necrosis.

● Ischemia → denaturation of Structural + Functional (enzyme) Protiens → → enzymatic dipestion → doesn't occurs → So cell Arcticture → maintain for few days → Nucleus disappear → ANucleated cells wd Eosinic cytoplasm. Then Neutrophils + Macrophages → Inflammatory Reaction. Fibrocytes → Fibroblast → Collagen formation → Scar → formed.

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②. Liquifactive Necrosis :

→ "There is denaturation of structural protiens but there is intense Enzymatic Digestion (Autolysis + Heterolysis) ——— ."

Heterolysis → Leukogenic
→ Microbes → Pus producing / Pyogenic

→ Tissue dissolution → faster than Repair.

* Pus → classical Example.

* In Brain → Liquifactive necrosis occurs → Not Associated wd Microbe → but due to Ischemia → but reason is not sure.

* ③ Suggestions are here:

① . structural Protiens → less in CNS.
② . Lysosomal Enzymes → more Intense in CNS.
③ . Phospholipids → more in CNS.

PUS :

→ "Area of Liquifactive Necrosis w/c consists of: (Alive + Dead + Dying) Local cells + (Alive + dead + dying) microbes + (Alive + Dead + Dying) Neutrophils → All floating in protien-rich Exudate ——— ."

* Another example of Liqu- Necrosis → Abcess
→ Abcess is localized pus in deep tissues
→ Central core → dead Neutrophils + Microbes + cells → Then Layer of Healthy Ntrphils → then Blood vessels → then Proliferating cells + Fibroblasts.