Toppersnotes
Unleash the topper in you

STAFF NURSE

NATIONAL RURAL HEALTH MISSION

DEPARTMENT OF MEDICAL, HEALTH & FAMILY WELFARE RAJASTHAN

VOLUME – 5

PATHOLOGY, SOCIOLOGY & FIRST AID

SIERRA INNOVATIONS

CONTENT

PATHOLOGY

PATHOLOGY

Pathology [To study of suffering]

Pathos → Suffering

Logos → To study

Introduction of phathology :→ four aspect of disease that form pathology :→

(1) Etiology [Causes]
(2) Pathogenesis [Machism of disease development]
(3) Morphology [Structural change in cells or organ]
    (1) Macroscopic (2) Microscopic
(4) Clinical significants [Functional consequence of morphological changes]

* Pathology can be divided into two groups
    (1) General
    (2) Systemic

(1) General :→ Basic reaction of cells two abnormal stimuli.

(2) Systemic:→

* Reaction of organs to abnormal stimuli.

* Father of modern pathology → Rudolf Virchow (विरचो)

* Hematoxylins & Eosin staining [H & E staining]:→ It is

* most common staining method used in pathology.

* staining method involve application of Hematoxylins which is provide colour to nucles of cells as Blue.

* After wards staining with eosin which provide coloured to most part of cytoplasm as pink, orge orange or Red [Depand on cells]

* Result of H & E staining:→

(1) Nucleus → Blue
(2) Muscles → Pink
(3) RBC → Cherry (चेरी) Red
(4) Cytoplasm → Pink

GENERAL PATHOLOGY

* CELL INJURY *

Definition:→

* Normal cell have a narrow range of function & structure which is able to handle normal physiological demands it is known as normal Homeostasis [Stoppage of bleeding]
* But some excessive physiological stress or pathological stress or pathological stimuli brings number of physiological & morphological cellular adpatation which maintain cellular viability (Cells को जिन्दा रखने की क्षमता)
* When adapative response to stimuls are exceeded (बहुत ज्यादा), sequence of events occured known as cell injury.

Types of cell injury

(1) Reversible Cell injury

(2) Irreversible cell injury

** Cell injury is reversible up to certain point but if stimuli persist (लगे रहना), cells reaches to point of no return it is known as irreversible or cell death.

Causes of cell injury:→

(1) Oxygen deprivation / Hypoxia
(2) Physical agent
(3) Chemical agent
(4) Immunological Reaction
(5) Infectious agent
(6) Genetic Disease
(7) Nutritional Imbalance

(1) Oxygen deprivation / Hypoxia:→

* Oxygen deficiency leads to defect in Oxidative respiration.

* Ischemia is more dengers to hypoxia because ischemia compromise avalibility of metabolic substrate.

(2) Physical Agent:→ Exterm [बहुत ज्यादा] temp [Burn], cold, radiation & electric shock, trauma

(3) Chemical Agent:→ O2 in high consntration arsenic cyanide, mercury salt air pollutants, insecticide, Herbicide [खरपतवार को नष्ट करने के लिए use में], Carbon mono-oxide, alcohol & Narcotic drug

(4) Immunological Reaction:→ derangements in the immune mechanisms

(5) Infectious Agent:→
* Bacteria
* Virus
* Fungal
* Protozoa

(6) Genetic Disease:→
* Down syndrome
* Sickle cell anaemia

(7) Nutritional Imbalance:→
Protein calorie malnutrition (PCM), vitamin deficiency.

* BIOCHEMICAL MACHNISM IN CELL INJURY
(क्रमी)

(1) ATP Depletion:→
ATP is required in protein synthesis membrane transport of molecule & lipogenesis
(Lipid के बनने की क्रिया)

ATP is formed by two methods:→

(1) Oxidative Phosphorylation
⇓
ATP is formed is present of O_2
⇓
(Mitochondrial में.

(2) Glycolytic pathway
⇓
ATP is formed is absence of O_2
⇓
All cells में

* ATP depletion & use ATP synthesis are common consequence of ischemic & toxic injury

(2) O2 derived Free Radicals:→

* Cell generate energy by reducing molecular oxygen to water. During this process some amount of reduced reactive O_2 is formed. [Mitochondria]

* Reactive oxygen damage lipids, protein & nucleic acid

* There is balance b/w free radical & radical (सफाई करना) ←Scavanging system

* Imbalance b/w these two system leads to oxidative stress.

(3) Intra cellular calcium & loss of calcium Homeostasis:→

* Most of intracellular calcium present in mitochondria and Endoplasmic reticulum

* There is use consntration of calcium in extracellular than intracellular

* Due to ischemia or toxin, their is ↑sed Ca concentration in cytoplasm with release of Ca from mitochondria & endoplasmic reticulum.

* ↑sed Ca concentration leads to activation of enzymes [Phospholypase, Endonuclease, proteiase, ATPase]

(4) Defect in membrane permeability:→

* Loss of selective membrane permeability of mitochondria & cytoplasmic membrane also leads to cell injury.

(5) Irreversible mitochondrial damage:→

* Direct or Indirect, mitochondria are important target of all type of injurious stimuli

* Cells are dependent on oxidative metabolism for longer term survival, so irreversible damage to mitochondria leads to cell death

* Mitochondria damaged by ↑se calcium concentration in cytoplasm & phospholypase enzyme.

* Due to damage of mitochondria, their

is mitochondrial permeability transition [MPT] formed in inner mitochondrial membrane which is indicator of cell death

REVERSIBLE CELL INJURY [RCI]
(सुधार योग्य)

* I[st] point of attack of hypoxia is cessation of oxidative phosphorylation & depletion of ATP.

* Depletion of ATP has following effects :->

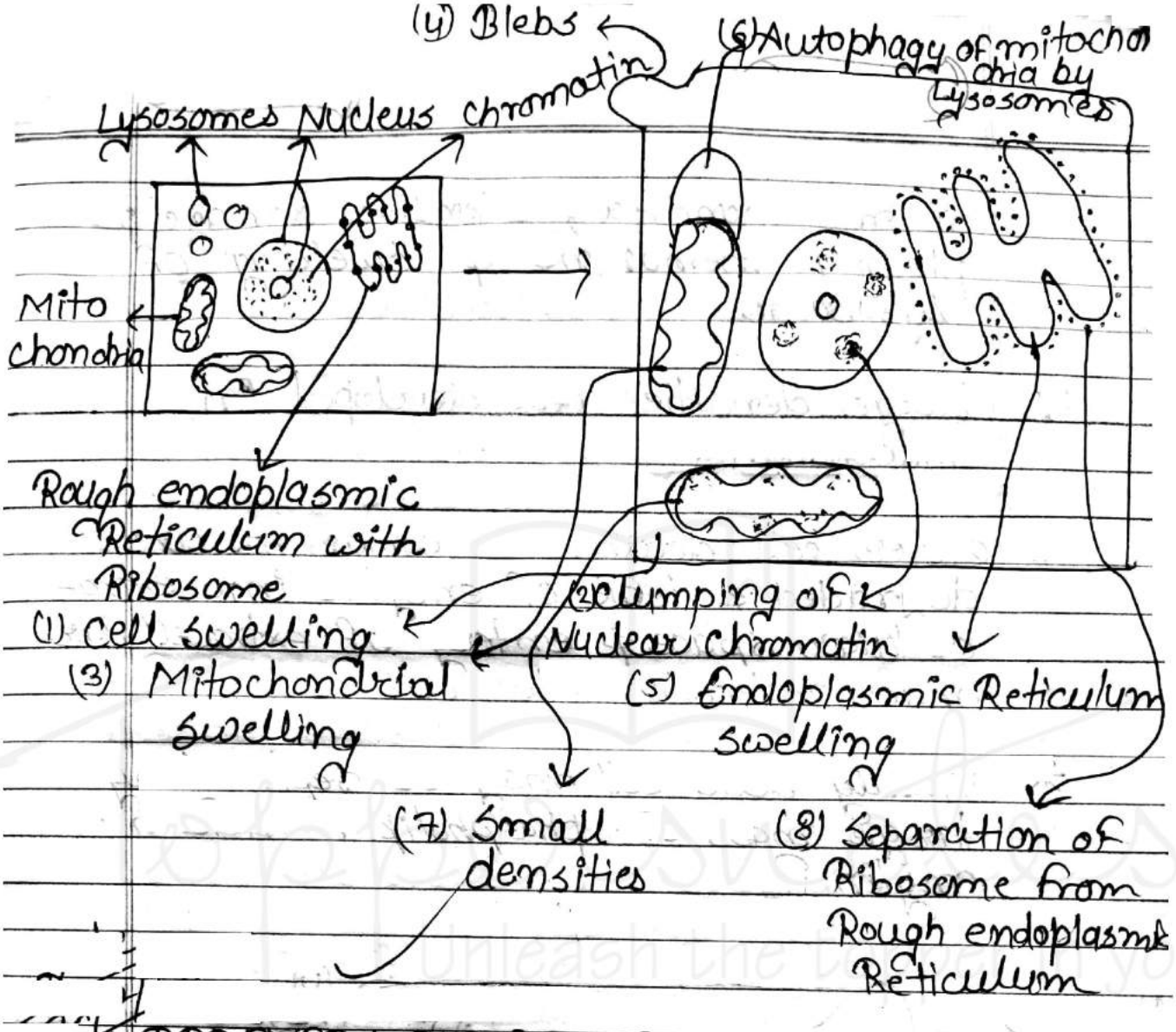
1) Activity of sodium potassium ATPase Enzyme is reduce so sodium accumulate in intracellular & K diffused outside the cell.

→ ↑sed Na in cell leads to ↑se ~~gai~~ gain of water which leads to cell swelling, endoplasmic reticulum swelling, loss of microvilli, blebs, myelin figures.

2) ↑se glycolysis leads to ↓se PH [Due to lactic acid formation] leads to clumping of nuclear Chromatin [DNA + Histone protein → Chromatin]

3) Separation of ribosomes from rough endoplasmic Reticulum

4) ↓se protein synthesis & lipid deposition.



* IRREVERSIBLE CELL INJURY [ICI]

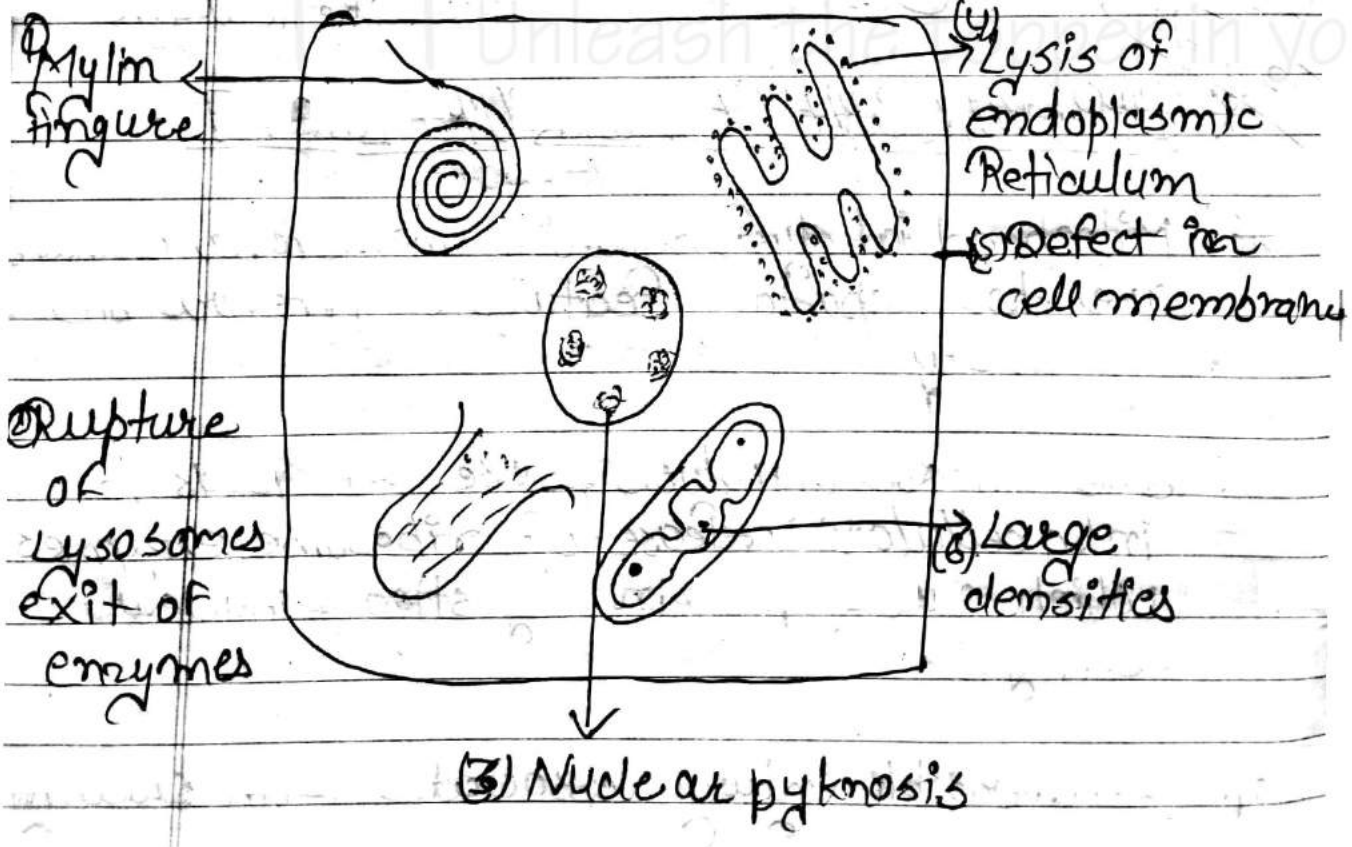
* Mitochondrial dysfunction & membrane damage are two important feature of irreversible cell injury
* Due to use glycolysis & use pH leads to intracellular release of ribosome enzymes which causes protein digestion & nuclear changes.
* Irreversible injury associated with swelling

of mitochondria, extensive damage to plasma membrane & swelling of Lysosome

* Large densities ~~also~~ developed in mitochondrial

* After cell death, cell components are degraded & leak of enzyme into extracellular space & entery of extracellular Macromolecule into cell

* Finally dead cell may be replaced by large mass of phospholipid in form of mylin figure



(Apopiosis)

[Programmed cell death]

It is form of cell death aim to remove unwanted host cell by systemic manner (तरीका)

Apoptosis is controlled by various gene product

Eg :→

(1) Programme destruction of cells during embryogenesis

(2) Hormone depended like endometrial cell breakdown in menstrual cycle

(3) Cell injury in viral hepetitis

(4) Injurious stimuli like heat, radiation hypoxia & anticancers drugs

Biochemical feature :→

(1) Protein cleavage :→ Protein hydrolysis occur through activation of protiage known as ~~eat~~ 'caspase'

(2) Protein Cross linking :→
It convert cytoplasmic protein into shrunken (सिकुड़ना) that may break into apoprotic bodies.

(3) DNA breakdown :→
Apoprotic cell show breakdown of DNA into large pieces by endonuclease

(4) Phagocytic Recognition →
Apoprotic cell have some specific molecule which ~~heall~~ help ~~erat~~ early ~~reg~~ recognition of cells by macrophage

* Morphology of Apoptosis :→
Following feature are ~~sarpe~~ seen under ~~an~~ electro micro :→

(1) Cell Shrinkage :→
Cell is smaller in size & organells are tightly packed

(2) Chromatin condensation :→
It is most characterstic feature of ~~apopo~~ apoptosis

(3) Cytoplasmic blebs & apoptotic bodies:→ Apoptotic cell show number of apoptotic bodies composed of cytoplasm & tightly packed organells

(4) Phagocytosis of apoptotic cells by adjacent parenchymal cell & ~~the~~ macrophages

[↑se Red colour → eosinphilia
↑se Blue colour → Basophilia]

V. MOST

(Necrosis)

* **Definition →**
* It is number of morphological changes that follow cell death in living tissue results from progressive degradations of injured cell by enzyme is known as necrosis.

* Morphological changes of necrosis occured due to two process :→

(1) Enzymatic Digestion of cells.
(2) Denaturation of protein.

* **Morphology of Necrosis :→**

*1] Due to loss of RNA leads to normal blue colour to nucleus & ↑se Red

*2] Cell have glassy appearance due to loss of glycogen particals

*3] Cytoplasm became vacoulated due to enzyme digestion

*4] Basophilia of chromatin is reduced due to DNAase activity.

[Chromatin ~~DNA~~ का reduce होना → Karyolysis]

* 5] Pyknosis :→
* Nucleus shrink (सिकुड़ना) in size.

* 6] Karyorrhexis :→
* Pyknotic nucleus undergoes fragmentation of nucleus disappear in 1 or 2 days.

* TYPES OF NECROSIS :→

Necrosis have five types :→

(1) Coagulative Necrosis
(2) Liquefactive Necrosis
(3) Gangrenous Necrosis
(4) Caseous Necrosis
(5) Fat necrosis

(1) Coagulative Necrosis :→ [Structured Necrosis

* It is occur due to protein denaturation.
* Preservation of basic out line of cells for some days.

Eg :→
* Myocardial Infraction (MI)

* It is occur in hypoxic death of all tissue except brain
* cause → Ischemia + hypoxia
* Organ involved → heart, kidney, spleen