

BATU-EXAM

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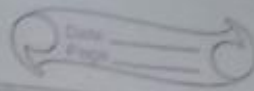
at MET Bhujbal Knowledge City

Pharmaceutical Organic Chemistry I Department

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Tutorial-2

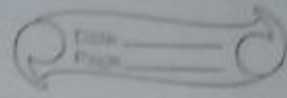


- * Introduction to basic terms of pathophysiology disease:
- 1) Pathophysiology
The changes that occur in biological function which caused by disease is called as 'pathophysiology'.
 - 2) Etiology
Etiology means study of causes / reasons of diseases.
 - 3) Injury
Injury is the variety of stresses that cause changes in cells internal or external environment.
 - 4) pathogenesis Ischemia
Loss of blood supply which is a major cause for cell injury is called 'pathogenesis Ischemia'.
 - 5) Hypoxia
Loss of oxygen supply which is a major cause for cell injury is called 'Hypoxia'.
 - 6) Necrosis
It is Irreversible cell injury and eventual cell death due to pathological processes are termed as 'Necrosis'.

7) Homeostasis

It is a Ability of human body to maintain the constant internal environment by maintaining & balancing a pH, temperature, Acid-base level.

Tutorial-3



* Give Detail about:

- 1) Atrophy
- 1) Atrophy is decrease in cell size due to loss of cell substance.
- 2) Atropic cells are living although they function at sub-normal level.
- 3) Cellular atrophy causes ↓ in organ size [atrophy of organ].
- 4) The stimuli causing organ atrophy are same that cause apoptosis. Therefore, organ atrophy is the total outcome of cellular atrophy & apoptosis.
- 5) Like apoptosis, atrophy may be triggered by both physiologic as well as pathologic stimuli but both types of stimuli induce similar fundamental cellular changes.
- 6) The stimuli which may lead to Atrophy, include ↓ work-load, loss of innervations, ischemia, inadequate nutrition, deficiency or lack of endocrine secretion & aging.
- 7) Mechanistically, atrophy may result from imbalance of protein synthesis & breakdown. Thus, reduce protein synthesis & increased protein breakdown may cause atrophy individually or together.
- 8) A small ↑ in protein breakdown also involved in cellular atrophy.
- 9) Deficiency of hormones, which promote protein anabolism & excess of catabolic hormones, which promote protein anabolism

↳ excess of catabolic hormones may both lead to atrophy.

10) These hormones include insulin, glucocorticoids & thyroid-stimulating hormone.

↑ in no. autophagic vacuoles also one of mechanism of atrophy.

11) Atrophic cells sometimes exhibit membrane-bound residual bodies, which are autophagic vesicles containing cell debris resistant to destruction.

eg. Lipofuscin.

ii) Hypertrophy

1) ↑ in cell size due to ↑ synthesis of cell substance is called 'Hypertrophy'.

2) Thus protein & cytoskeletal elements are synthesized in greater amounts by hypertrophied cell.

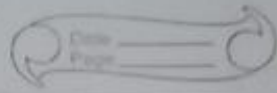
3) ↑ in cell size (cellular hypertrophy) reflects in ↑ in organ size (organ hypertrophy).

The most important fact about hypertrophy that is does not involve ↑ in cell no.s (mitosis), or oedema.

4) Both physiologic & pathologic processes may cause hypertrophy. Endometrial hypertrophy under influence of estrogens is e.g. of physiologic hypertrophy although it not pure hypertrophy.

5) Since, estrogens stimulate endometrial proliferation also.

6) myocardial hypertrophy in patients of chronic



high blood pressure - e.g. of pathologic stimulus causing Hypertrophy.

- 7) The stimuli that trigger Hypertrophy are
 - 1) Functional demand (work-load) & excessive stimulation by trophic hormones.
 - 8) Trophic hormones act by \uparrow rate of transcription of genes, thereby \uparrow protein synthesis. Hormones bring about this effect by binding to specific cytoplasmic or nuclear receptors.
 - 9) \uparrow Functional demand (like Hypertension & muscular exercise) exerts stretch on muscle, which act as mechanical trigger for activation of one or more genes.
 - 10) The result is \uparrow synthesis of proteins, cytoskeletal elements & organelles leading to hypertrophy.
 - 11) In hypertensive myocardial hypertrophy, apart from mechanical trigger, another trophic trigger is also involved.
 - 12) The mechanical trigger somehow activates α -adrenergic receptors, which seem to play role in resulting hypertrophy.
- iii) Hyperplasia
- 1) \uparrow in no. of cells in tissue or organ is called 'Hyperplasia'.
 - 2) Hyperplasia \rightarrow also \uparrow organ size, but unlike Hypertrophy, there is no \uparrow in cell size. Hyperplasia is closely related to hypertrophy & two often occur concurrently.

- 3) The end result of both is \uparrow in size of tissue or organ. Hyperplasia may also be physiologic or pathologic.
- 4) Physiologic hyperplasia occur as part of normal growth & development of body tissues and organs.
- 5) It can be of two types based on the trigger. Hormonal hyperplasia is proliferation of cells under influence of hormone.
- 6) Proliferation of female breast at puberty & during pregnancy is e.g. of physiological hormonal hyperplasia.
- 7) Removal of portion of tissue also act as a powerful trigger for hyperplasia. This type of Hyperplasia is called 'compensatory Hyperplasia'.
- 8) partial hepatectomy triggers proliferation of hepatocytes, which restores liver weight to original.
- 9) Residual parenchymal cells & perhaps other cells release some polypeptide growth factors, which stimulate mitosis that restores liver weight.
- 10) once parenchymal mass is restored, a different set of polypeptide growth inhibitors turn the proliferation off.
- 11) Pathologic hyperplasia may also be involved in carcinogenesis (if proliferation get out of control). However, hyperplasia is different from neoplasia in that former remains controlled & gradually suppressed

due to declining supply of triggering hormone or growth factor.

12) Proliferation of Fibroblasts & angiogenesis in process of wound healing, is another e.g. of pathologic hyperplasia.

iv) metaplasia

1) It is reversible change, in which one differentiated cell type (epithelial or mesenchymal) is replaced by another cell type.

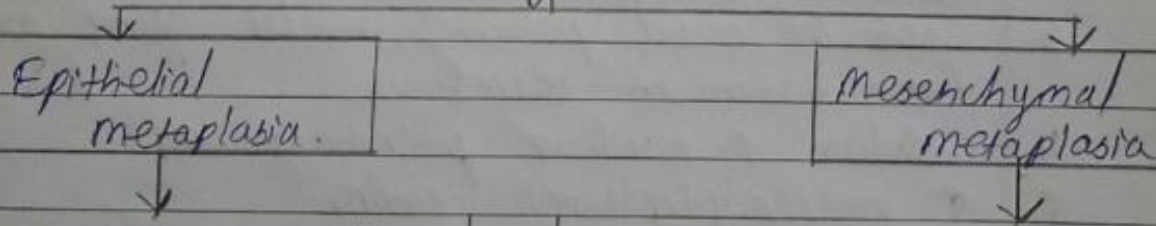
2) Mechanism:

Due to reprogramming of precursor cells, present in normal tissues.

3) Differentiation into new cell lineage.

By signals generated by various cytokines, growth factors & extracellular matrix components in the cell's environment.

4) metaplasia



1. Squamous metaplasia
 - Bronchus
 - Uterine Cervix.
 - Gall bladder
 - Renal pelvis
2. Columnar metaplasia
 - Barrett's esophagus
 - Intestinal & gastric metaplasia.

1. Osseous metaplasia
 - Arterial wall
 - myositis ossificans.
 - stroma of tumours.
 - cartilage of larynx in elderly.
2. Cartilagenous metaplasia.
 - Healing of fractures.

- 5) Other types of metaplasia:
- ① Apocrine metaplasia
 - ② Tubal metaplasia.
 - ③ Clear cell metaplasia.
 - ④ Mucinous metaplasia.
 - ⑤ Fat metaplasia.

v) Dysplasia

- 1) Dysplasia means 'disordered cellular development', often accompanied with metaplasia & hyperplasia, it is therefore also referred to 'typical hyperplasia'.
- 2) It occurs most often in epithelial cells. Epithelial dysplasia is characterized by cellular proliferation & cytologic changes.
- 3) These changes include:
 1. ↑ no. of layers of epithelial cells.
 2. Disorderly arrangement of cells from basal layer to surface layer.
 3. Loss of basal polarity, nuclei lying away from basement membrane.
 4. Cellular & Nuclear pleomorphism.
 5. ↑ nucleocytoplasmic ratio.
 6. Nuclear hyperchromatism.
 7. ↑ mitotic activity.
- 4) The two most common e.g.s. of dysplastic changes are uterine cervix & respiratory tract.
- 5) Dysplastic changes often occur due to chronic irritation or prolonged inflammation.

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