Medical Academic Team

Pathology sheet (2)

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MECHANISM OF ATROPHY:

Slide #30 (5:15):

Atrophy result from decreased protein synthesis because of reduced metabolic activity and decrease protein degradation in cells.

*the main goal is degradation of protein

**Extra information:

Ubiquitin: a compound found in living cells that plays a role in degradation of defective and unnecessary protein (it is a single poly peptide chain).

Slide #31 (6:34):

In many situations, atrophy is also accompanied by increased autophagy, resulting in increased numbers of autophagic vacuoles.

*the cases we will study from atrophy or various types of cell injury is autophagy.

*autophagic vacuoles are membrane-bound that contain fragment of cell component. The vacuoles ultimately fuse with lysosomes and their contents are digested by lysosomal enzymes.

Slide #32:

Autophagy is associated with various types of cell injury, and we will discuss it in more detail later.

*autophagy is not associated only with atrophy, any cell injury can lead to it.

Slide #33:

Metaplasia:

Is a change in the phenotype of cells. A reversible change in which one differentiated cell type (epithelial or mesenchymal) is replaced by another cell type.

*How that happen?
Some of the stem cells produce new cell type not the cell already differentiated.

*slide 34+35

*Example:  The most common epithelial metaplasia is **columnar ciliated** to **squamous**, as occurs in the respiratory tract in response to chronic irritation(happen to smokers people)

**squamous to other epithelial cell(to columnar ),in goblet cell in the large intestine as in Barrett esophagus.

Why do metaplasia happen ???

To adapt changes to survive.

*if metaplasia does not happen , the cell injury happen and then it may die ,metaplasia is one of the defence mechanism in our body.

EX:

* From squamous to columnar (barrett esophagus)

*From squamous to glandular (large intestine)

*may be from glandular to squamous

*the influence that predispose to metaplasia ,if persistant may initiate malignant transformation in metaplasitic epithelium .

*Connective tissue metaplasia is the formation of cartilage, bone, or adipose tissue (mesenchymal tissues) in tissues that normally do not contain these elements.

*This type of metaplasia is less clearly seen as an adaptive response, and may be a result of cell or tissue injury.

Slide #36:(15:27)
*Metaplasia does not result from a change in the phenotype of an already differentiated cell instead it is the result of reprogramming of stem cells.

*the new cells will resist the change that will lead to metaplasia (reprogramming of stem cell)

*slide 37: (16:18):

*In a metaplastic change, these precursor cells differentiate along a new pathway.  

*The differentiation of stem cells to a particular lineage is brought about by signals generated by cytokines, GF, and ECM components in the cells' environment.

*there are persistent (inflammatory irritation) in this tissue subsequently there are component ECM, GF and cytokines.

*cytokines: growth factors that secreted by certain cells of the immune system and have uneffect on other cells.

*note: thymus gland atrophy with increase age.

**OVERVIEW OF CELL INJURY AND CELL DEATH:**

**Slide #3: (20:07)**

*Reversible cell injury happen in early stages or mild forms of injuries (light effect)

*the functional and morphologic changes are reversible if the damaging stimulus is removed (how??)

*by therapeutic intention and when we have stopped the problem then the change will be reversible.

*The hallmarks of reversible injury are reduced oxidative phosphorylation with resultant depletion of energy stores (ATP), and cellular swelling caused by changes in
ion concentrations and water influx. (basically it is depletion of energy store (ATP) & cellular swelling).

Slide #4:

* Necrosis: uncontrollable
* Apoptosis: control, previously reprogramming to kill itself follow with rapid removal of cellular debris.
* Apoptosis: the damage may be the disability of the cell to repair or there are viruses infect the cell, the cell kill itself because there is no choice to resist (only by kill itself)
* Necrosis is always pathogenic.
* Apoptosis maybe associated with cell aging, defect in DNA or normal development and growth.

Slide #6 (24:39)

* Anemia: problem in RBCS, exactly in the capacity of them to transport oxygen.
* Hypoxemia: shortage of oxygenated blood in artery.
* Note: there are (16-18)% O2 in exhalation, the evidence is artificial respiration.

Slide #7 (28:55)

All kinds of chemical agent can lead to cell injury

Slide #8 (29:50)

* Autoimmune disease: neuro system losing its memory partially on one type of cell or tissue and consider it foreign and attack it.
* Reversible injury (early stages)

Slide #9 (31:45):

* Nutritional imbalances: not only protein-calorie deficiencies cause it, also obesity can cause it.
Slide #10

*Cellular develop biochemical alteration then structural changes, light microscopic with finally gross morphologic change in cell and tissue

Slide #11

Reversible injury (early stages)

*There are many changes:

*swelling  *fat deposition

Swelling:

If the stimulus abates will return to normaly.

Slide #12

In progressive injury:

*necrosis: breakdown of plasma membrane, organelles and nucleus, leakage of contents attraction of white blood cells & inflammation

-In necrosis: swelling, loss of integrity of cell membrane and condense chromatin.

-in apoptosis: shrinkage, there are apoptotic bodies, there are organelles, at the end the phagocytic cells are ready to engulfment the organelles

*there are phagocytosis because apoptosis is preprogramming cell death

*note:

Inflammation does not occur in apoptosis because apoptosis happen each second.

Slide #15

Under microscope, we can see deposition of fat droplet within the cell, organ or tissue itself. There are some situations lead to immensity of organ

EX:
In hepatic steatosis we can see hepatomegaly at the expense of accumulation of fat cells.

**Slide #17:**

In necrosis there is loss of nuclei

**Slide #18 (40:52)**

*L: lumen

*Mv: microvilli

**Slide #20 (44:41)**

Why is it important to study the type of necrosis?

To explain the disease because the type of necrosis depending on the type of disease.

*In most cases in necrosis, surrounding tissues and organs are inflammatory response.

*There are leukocytes because there are attraction or white blood cell because inflammatory response.

**Slide #22 (47:41)**

*If there is calcification in any cell, we must expect severe cell injury

*Dead cell become calcification

**GOOD LUCK**