patho lecture =5=

ADAPTATION

→ Reversible changes in the number, size, phenotype metabolic activity, Function of cell in response to chanes in their environment.

HAYPERTROPHY

→ ↑ in the size of cell → ↑ size of organ
→ cells have a limited capacity to divide.
→ no new cell, Just ↑ amount of ptn + organelles

1 physiologic :-

A ↳ stimulation
* estrogen stimulated smooth muscles
↳ enlargement of the uterus during preg...

B ↳ ↑ Demand
* in response to ↑ work load the stricted muscle
* Adult muscles have limited capacitiy to divide

2 pathologic :-

↳ hypertension or aortic valve disease
* only hyper trophy (limited capacity to divide)

* signal
↳ mechanical triggers
↳ soluable medicator (Growth Factor)

* mechanisms
↳ signal transition pathway → gene synthesis cellular ptn ← stimulate
↳ switch of contractile ptn From adult → Fetal, myosin → Fetal β-myosin slower, more economic

HAYPERPLASIA

→ ↑ in the number
→ cell capable of proliferation
→ may accure with hypertrophy

1 physiologic
- Hormonal :- proliferation of the glandular epithilium of the female brest at pregnancy & puberty.
- compensatory :- residual tissue grows after damage or resection of part an organ
  → stimuli → polypiptide growth Factor
  → liver eventuly restoring to its normal size

2 pathologic → excessive hormonal & Growth Factor.
↳ ↑ estrogen → endometrial hyper plasia → abnormal menstrual bleeding
↳ ↑ androgen → Benign prostatic hyperplasia.
↳ viral infection → causes skin warts & mucosal lesions. (papilloma - viruses)

ADAPTATION

ATROPHY

→ shrinkage in the size of the cell

→ diminshed function (not dead)

→ Causes of Atrophy :-
decreased workload , aging
loss of innervation. ↓ blood supply
inadequate nutrition, ↓ endocrine stimulation.

physiologic cause :-
↳ loss of hormone stimulation in menopause

pathologic cause :-
↳ denervation.
↳ ↓ blood supply → brain (narrow the gyri + widens the sulci)

The process of Atrophy result from
↳ ① decrease ptn synthesis
↳ ② increase ptn degradation by ubiquitin-proteasome

METAPLASIA

→ one adult cell type is replaced by another cell type
→ It arise by the reprogramming of stem cells
→ RS (SMOKING)
PSCE ⟶ squamous cell
important protective mechanism are lost
→ GI (GERD)
Squamous ⟶ columnar epithelium.
→ IF the metaplastic persistent.
may predispose malignant transformation
↳ LUNG CANCER malignant squamous cell
↳ ESOPHAGEAL CANCER columnar epithelium

3

INTRACELLULAR ACCUMULATION

→ The main pathway of abnormal intracellular accumulation
1. inadequate removal & degradation
2. excessive production endogenous
3. deposition of exogenous material.

FATTY CHANGE (steatosis)

→ accumilate triglycerides.
→ most seen **liver**
- heart, Kidney, skeletal muscles

→ caused by toxins, D.M, obesity anoxia, ptn malnutrition.
→ most common cause (in liver) Alcohol abuse & D.M

↓

CHOLESTEROL

→ Cause :- ↑ intake lipids
↓ metabolism of lipid

→ Atherosclerosis (the most impo.)

GLYCOGEN

→ Cause :- abnormalities in the metabolism of glucose or glycogen.
* poorly controlled D.M
→ glycogen accumilated in
- renal tubular
- Cardiac myocytes
- B cell of islet of langerhans.
→ glycogen storage diseases glycogen accumulated within cell ⊆ genatic disorder.

PIGMENTS

① Carbon (exogenous) (Anthracosis)
→ when inhaled → phagocytosed by alveolar macra
→ transported by lymphatic channels.
→ pulmonary & lymph nodes blacken

② lipofuscin (endogeneous)
→ brownish-yellow granular
→ accumulation in liver, heart, brain.
→ produced by free radical-catalyzed peroxidation of polyunsaturated lipid
→ marker of past free radical injury.
→ Brown atrophy → large amount.

③ Melanin (endogeneous)
→ brown-black by melanocyte in epidermis
→ screen against harmful UV radiation.
→ ** Other sources :- adjacen basal keratinocyte + dermal macrophage.

④ Hemosiderin
→ golden yellow – brown
→ when? excess of iron
→ iron ⊆ ptn (apoFerritin)
→ = micelles (E.M + L.M)
→ Prussian blue (for iron)
→ small amount of this pigment are normally Bone marrow, spleen, liver

* hemosiderosis excessive depusition of Hemosiderin

* hemochromatosis more extensive accumulation of iron