Cell injury

Response of cell to injury

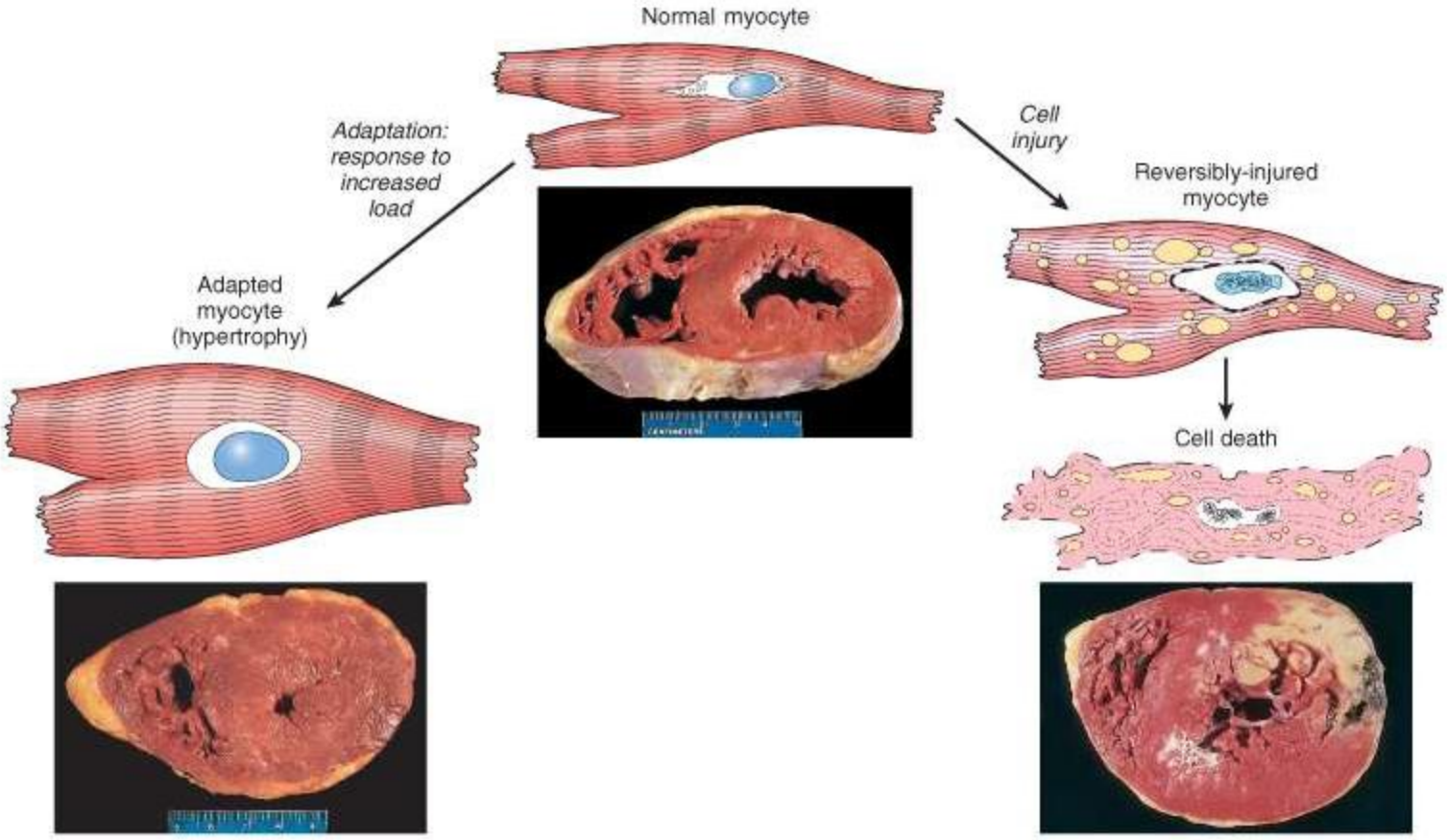
Response of cell to injury

Adaptation

Damage

- Reversible
- Irreversible

Adapted - Normal - Injured Cells



Cell injury

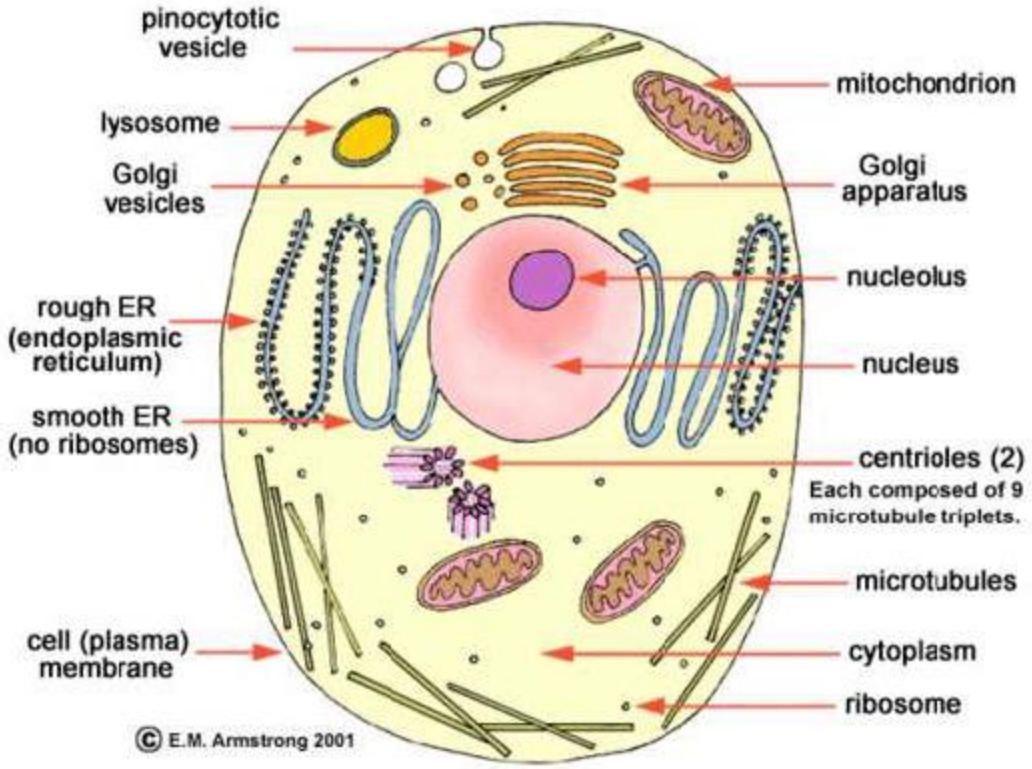
- **Reversible cellular morphological and functional changes accompanied with the accumulation of metabolites or other substances in a cell damaged by preceding injury.**
- **Resulting from metabolic disturbances due to mild injuries.**
- **Occurs in cells having high metabolic activity and rich in mitochondrial enzymes such as liver cells, Kidney tubules and cardiac muscles.**

*****NOTE**

Normal cell —(injuries)⟷ **Reversible changes (Degeneration)**

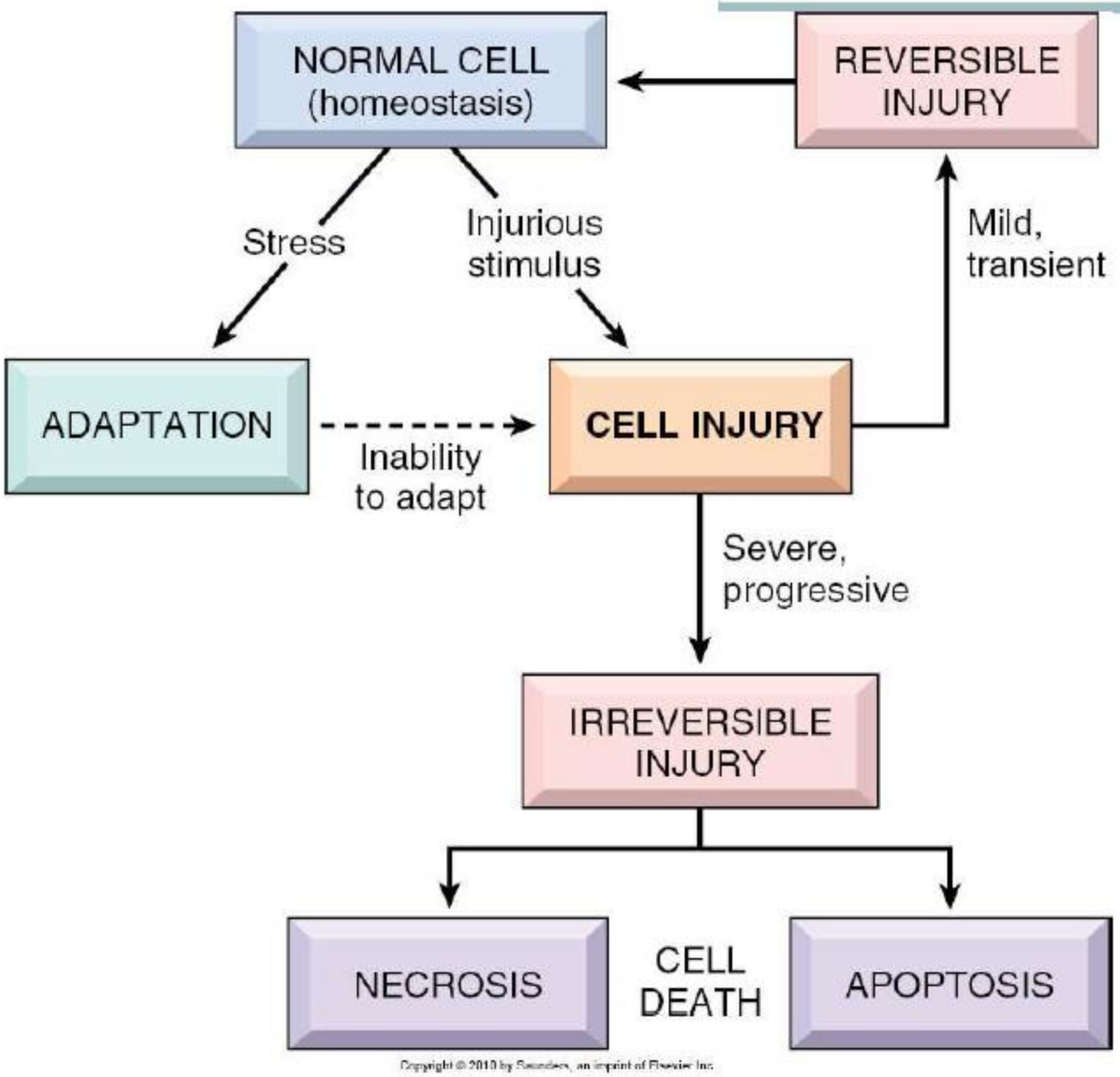
Normal cell —(injuries)→ **Irreversible changes (Necrosis)**

INTRODUCTION



- CELL INJURY:-

Cell injury is defined as a variety of stresses a cell encounters as a result of change in its internal and external environment.



Causes of Cell Injury

- Oxygen deprivation (hypoxia or ischemia)
- Physical Agents (trauma)
- Chemical agents and Drugs
- Infectious Agents
- Immunologic Reactions
- Genetic Derangements
- Nutritional Imbalances

Types of cell injury

Intracellular accumulations

- Water
- Proteins
- Fatty change
- Carbohydrates
- pigments

Extracellular accumulations

- Calcium
- Amyloid
- Uric acid & ureate crystals

Both intra&extracellular accumulation

- Mucin
- Hyaline

PROCESSES OF ACCUMULATIONS

1. **Production of a normal endogenous substance at normal or increased rate, but the rate of metabolism is inadequate to remove it.**

e.g., **fatty liver, reabsorption protein droplets in tubules**

of kidney

- **2- Accumulation of an abnormal endogenous substance due to defects in protein folding, transport & inability to degrade abnormal proteins efficiently**.
- e.g., accumulation of mutated proteins in liver cells

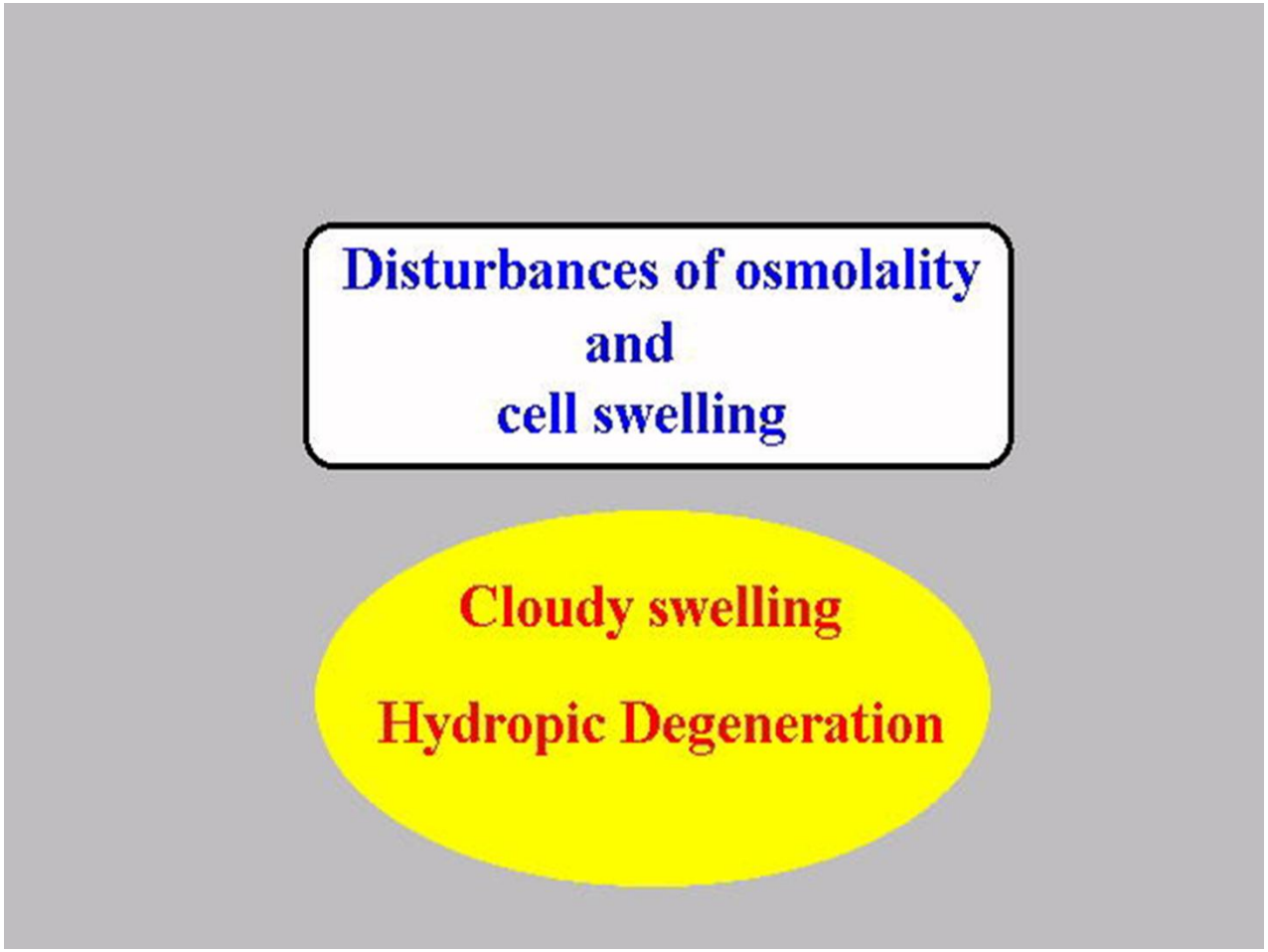
3-Accumulation of normal endogenous substance due to inherited defect in enzymes required for metabolism of the substance.

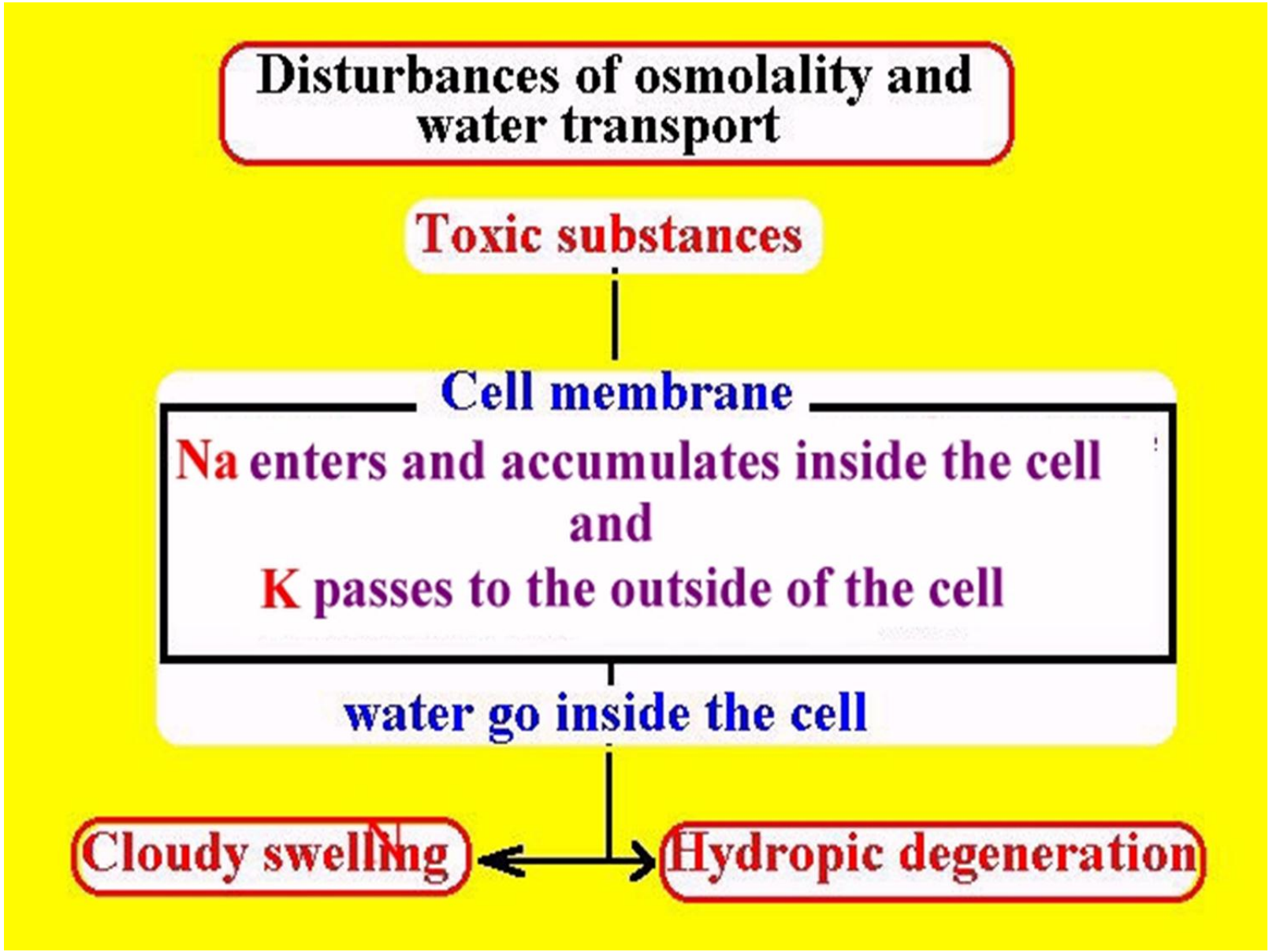
e.g., Lipid & Glycogen storage diseases

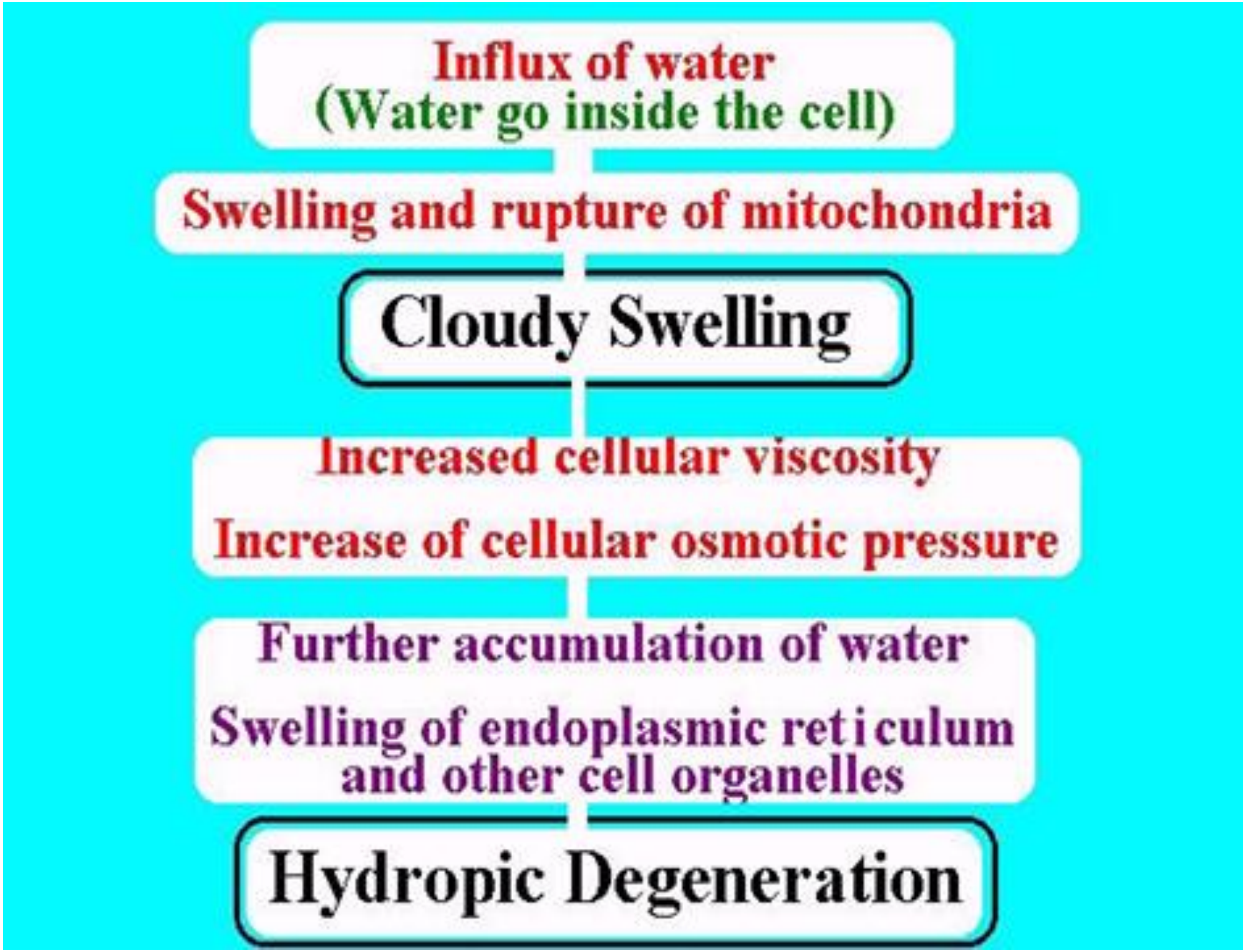
4. **Accumulation of abnormal exogenous substance due to unavailability of enzymatic & transport mechanisms to degrade & transport it to other sites.**

- e.g., Silicosis & Anthracosis

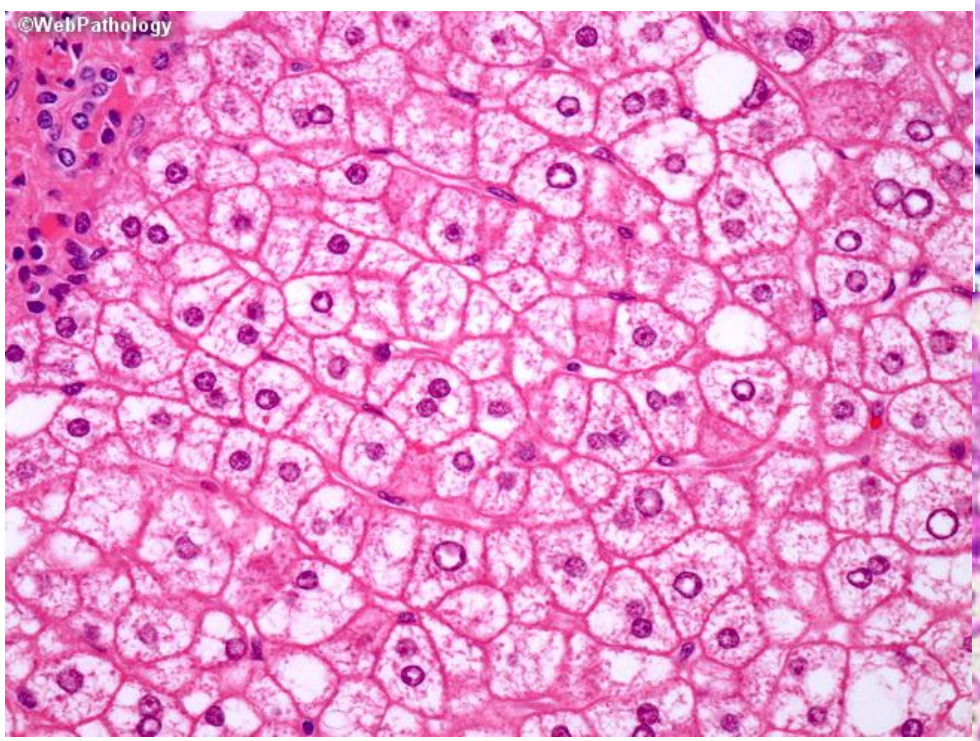
Water
(cloudy swelling & hydropic degeneration)

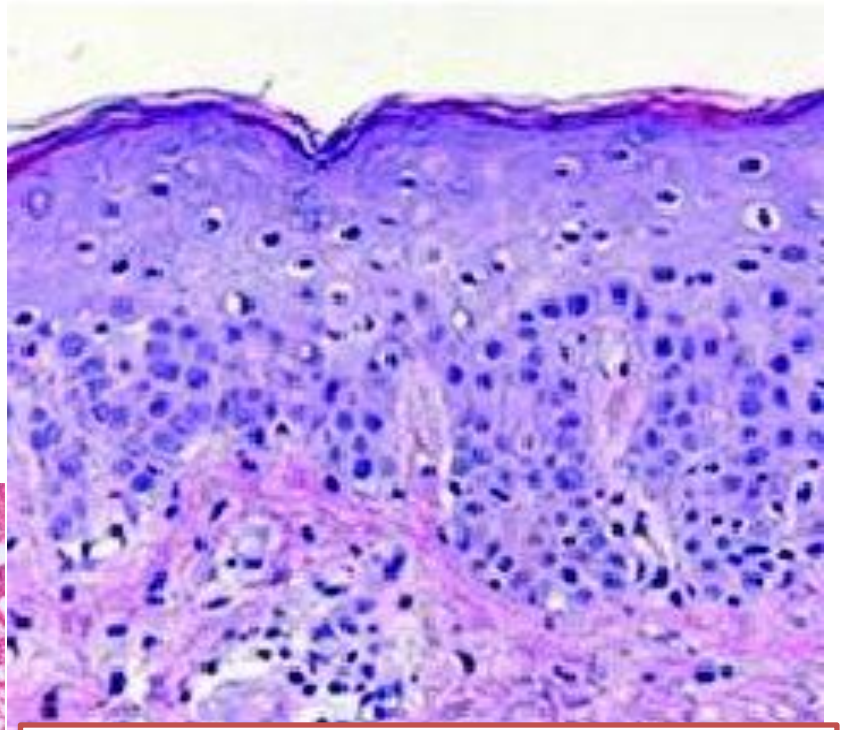



Disturbances of osmolality and water transport
Toxic substances
Cell membrane
Na enters and accumulates inside the cell
and
K passes to the outside of the cell
water go inside the cell
Cloudy swelling
Hydropic degeneration

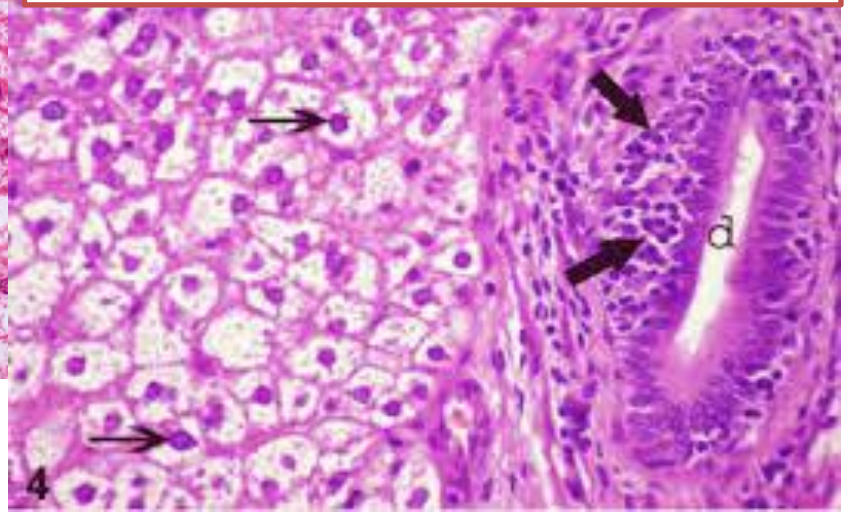

Influx of water
(Water go inside the cell)
Swelling and rupture of mitochondria
Cloudy Swelling
Increased cellular viscosity
Increase of cellular osmotic pressure
Further accumulation of water
Swelling of endoplasmic reticulum
and other cell organelles
Hydropic Degeneration

Cloudy swelling

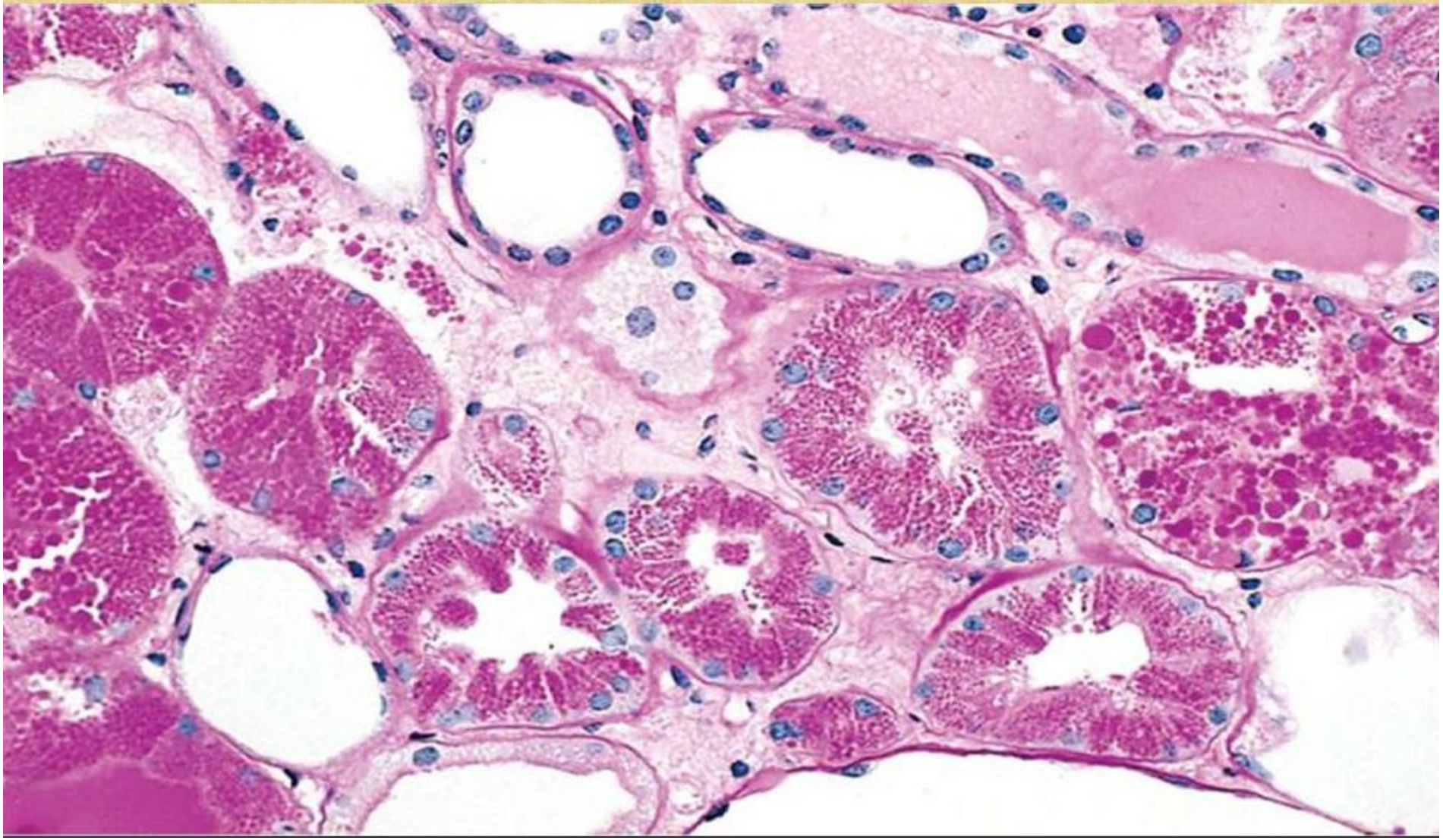


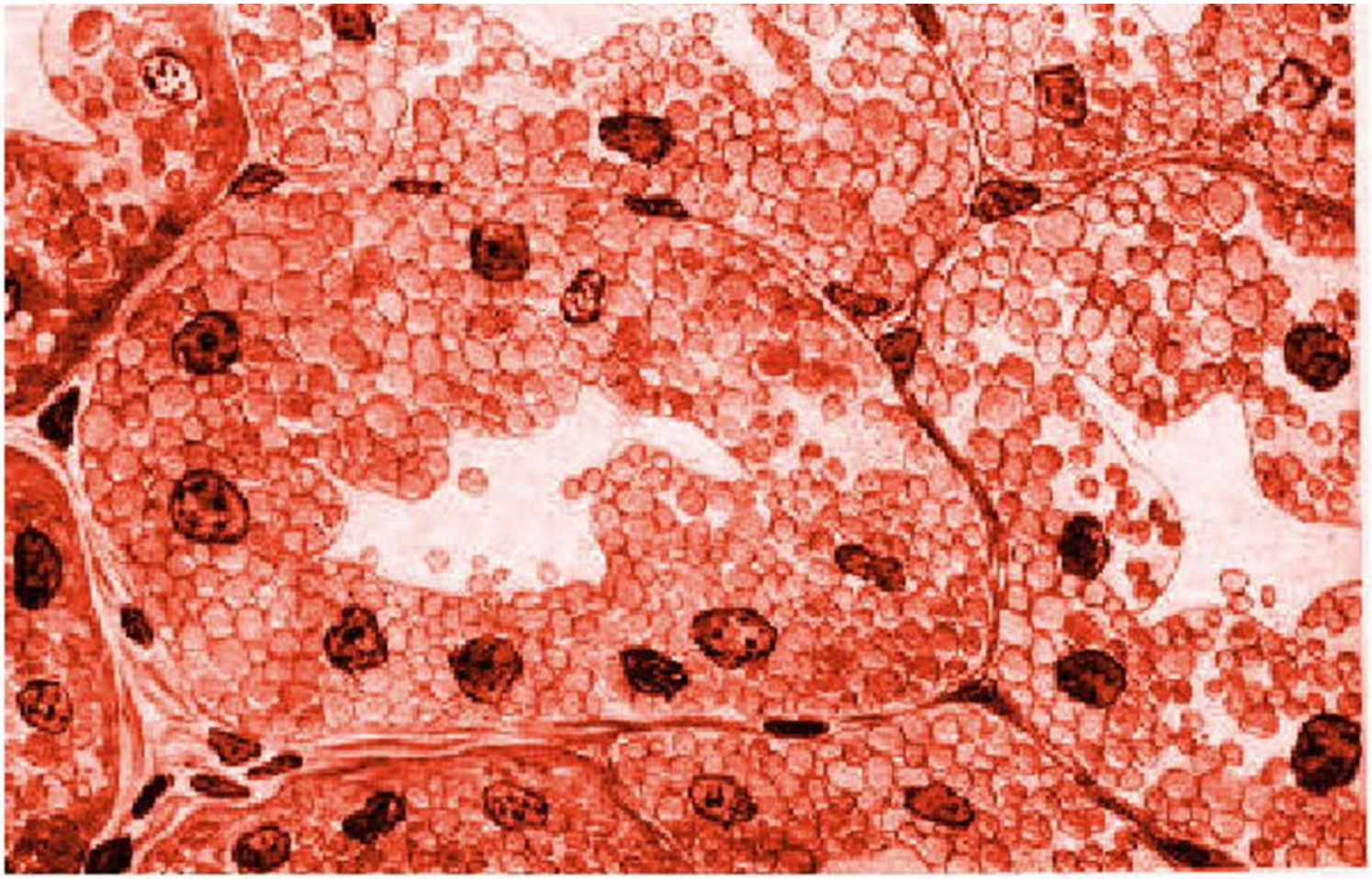


Hydropic degeneration



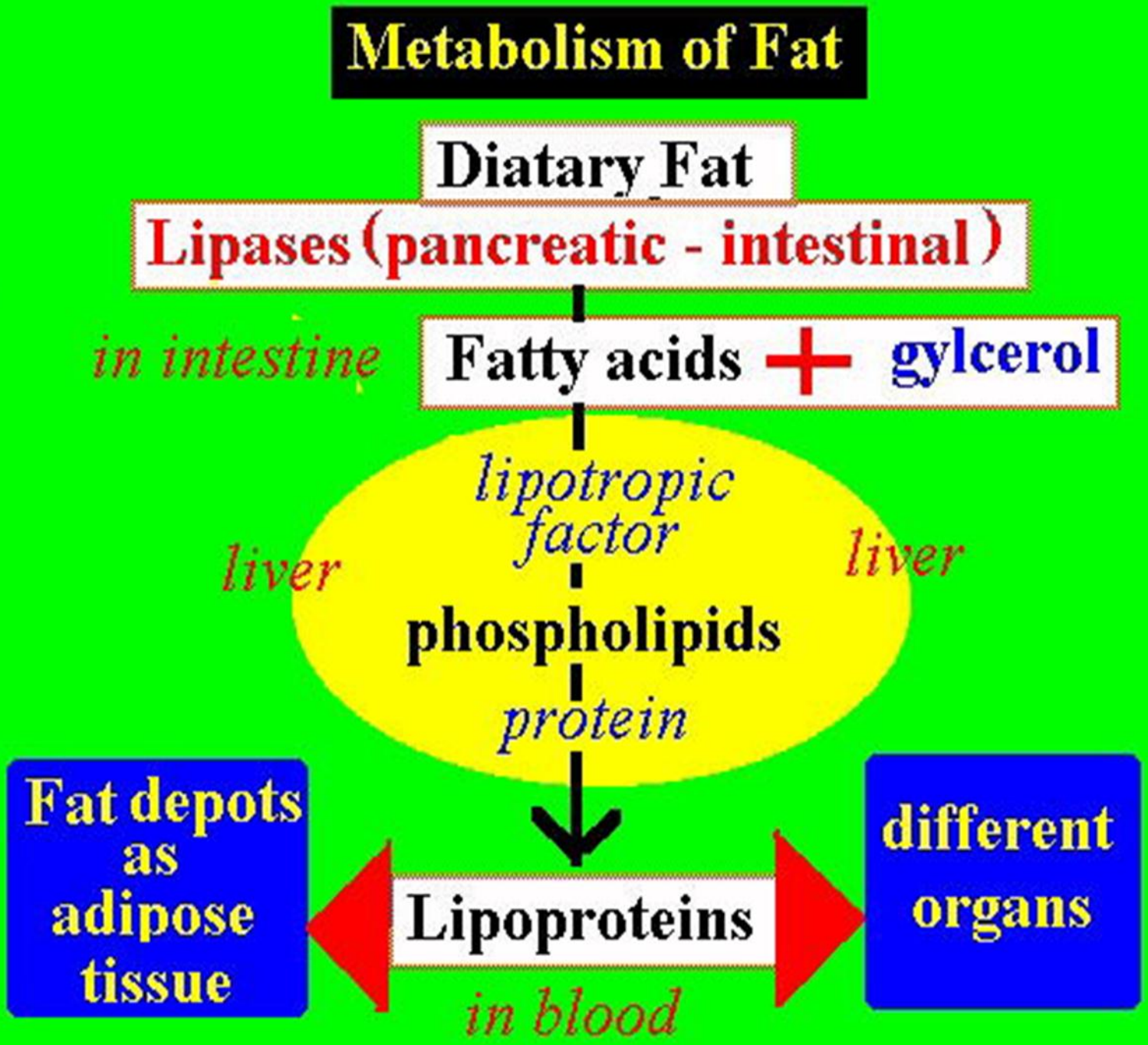
Protein reabsorption droplets in renal tubular epithelium





Hyaline droplet degeneration (kidney)

hyalinized droplets in degenerating renal tubules


Metabolism of Fat
Diatary Fat
Lipases (pancreatic - intestinal)
in intestine
Fatty acids + gylcerol
lipotropic factor
liver
liver
phospholipids
protein
Fat depots as adipose tissue
Lipoproteins
in blood
different organs

Fatty Change

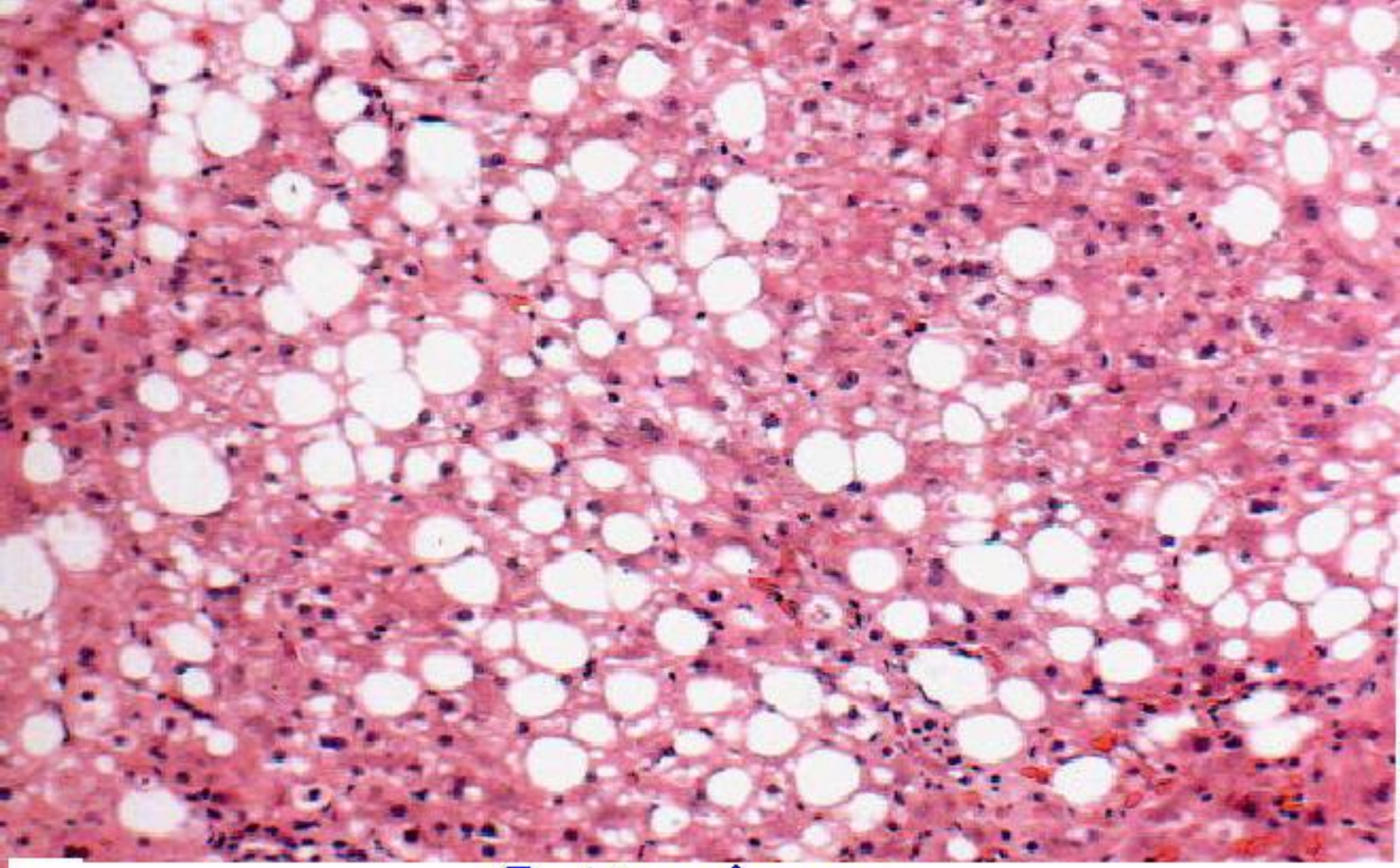
Pathogenesis

Appearance of neutral fat in the cell occur as a result of

Fat phanerosis
breakdown of cell organelles

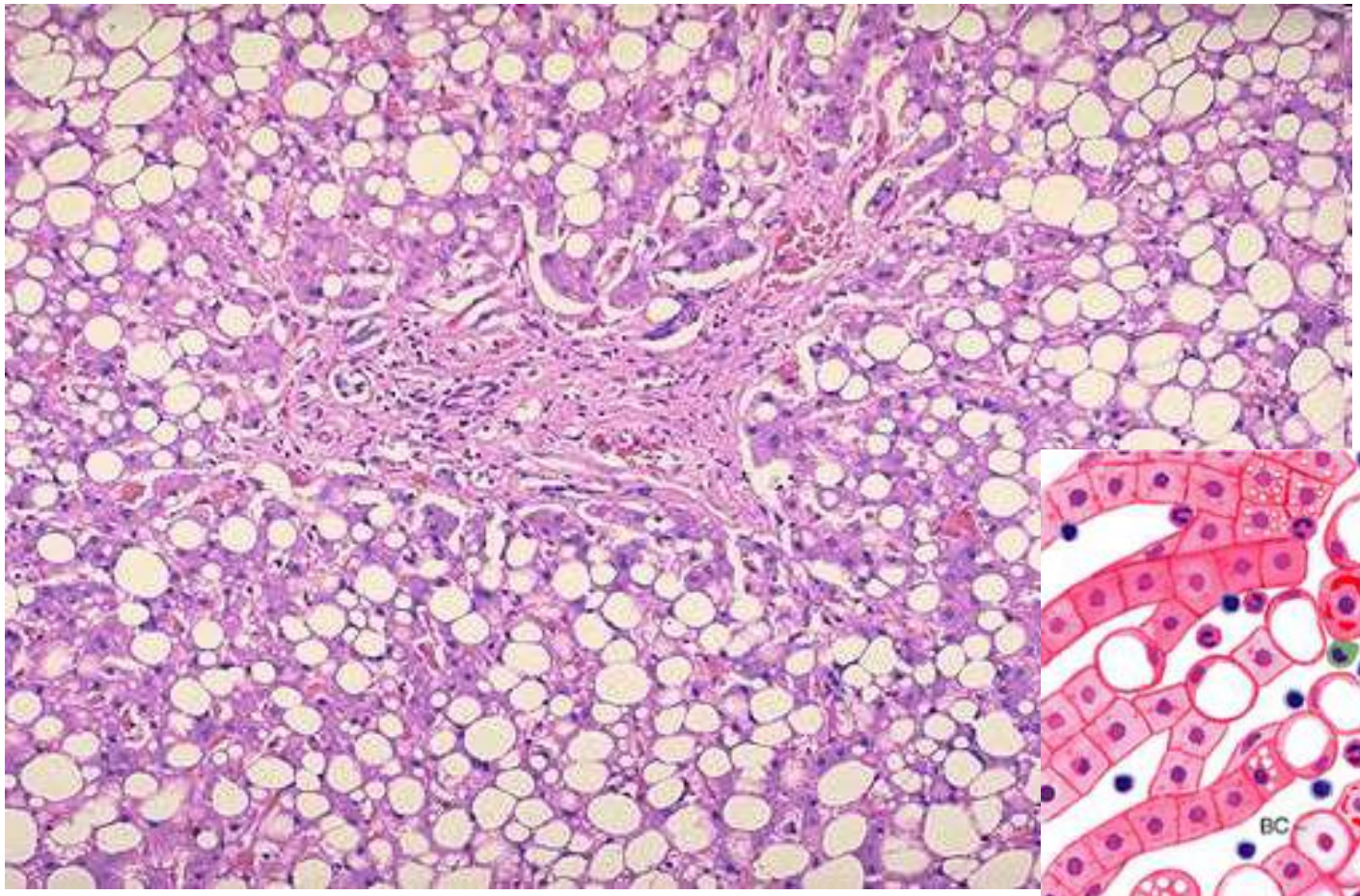
unmetabolizable fat due to
Impairment of protein synthesis
Increased mobilization of fat from fat depots
Deficiency of lipotropic factor
cell damage

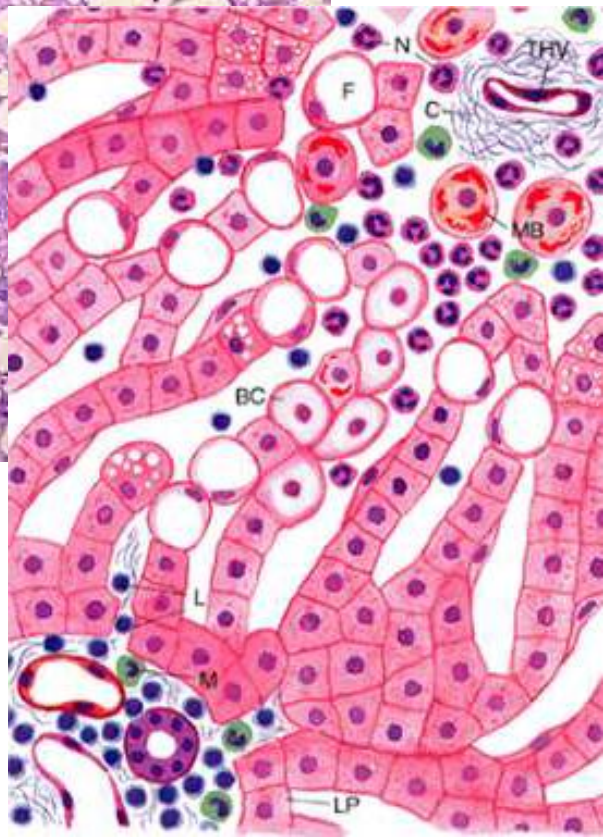
Liver
Fatty change



Fatty change

Fat cells have a signet-ring appearance due to accumulation of neutral fat in the cytoplasm



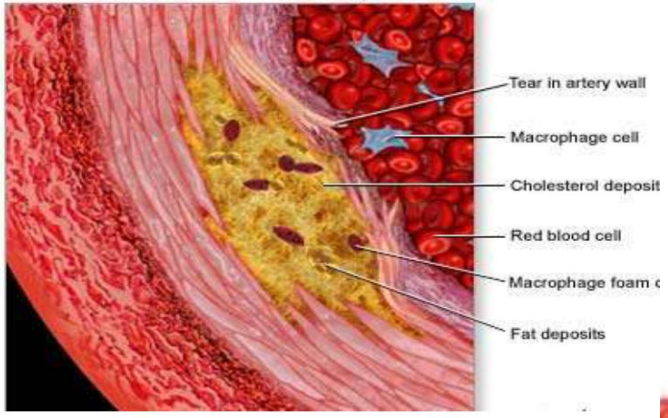

N
THV
F
C
MB
BC
L
M
LP

Fat stains

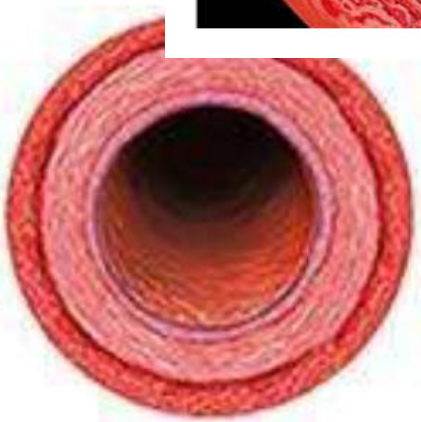
- Can be demonstrated in fresh unfixed tissue by frozen section

Special stains of fat	Stained the fat (frozen sections)
Sudan III	Yellow
Sudan IV (Scarlet red)	Orange
Oil red O	Red
Osmic acid	Black
Sudan black B	Black
Nile blue sulfate	i-Fatty acid: bluish
	ii-Neutral fat reddish

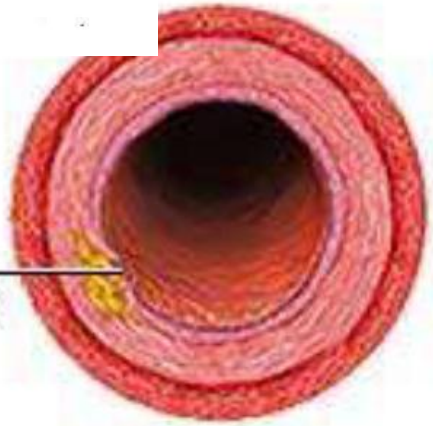
Cut-section of artery



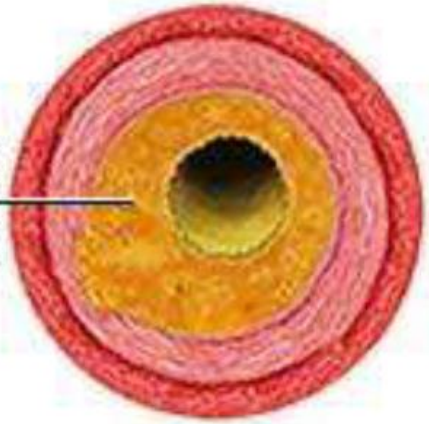
Normal cut-section of artery



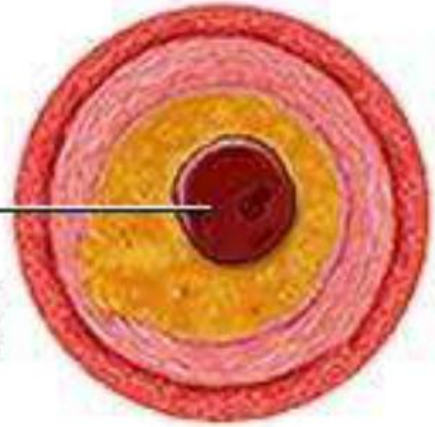
Tear in artery wall

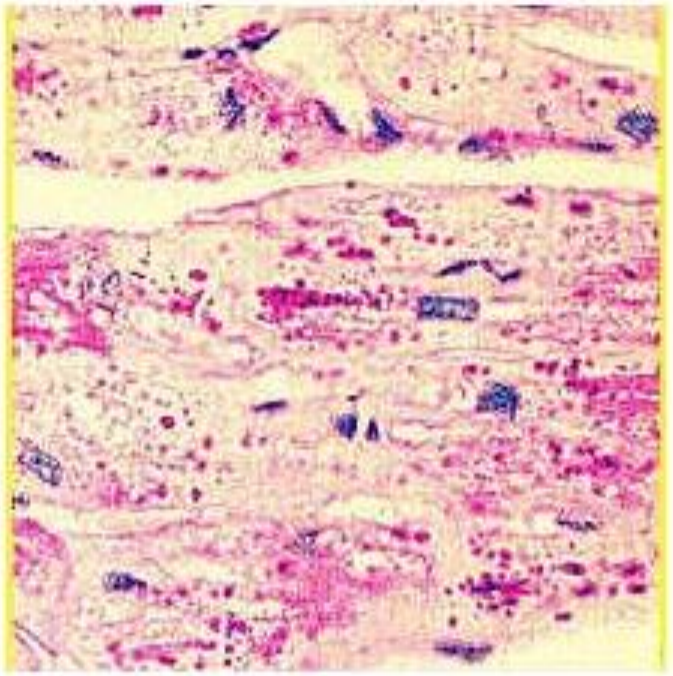


Fatty material is deposited in vessel wall



Narrowed artery becomes blocked by a blood clot



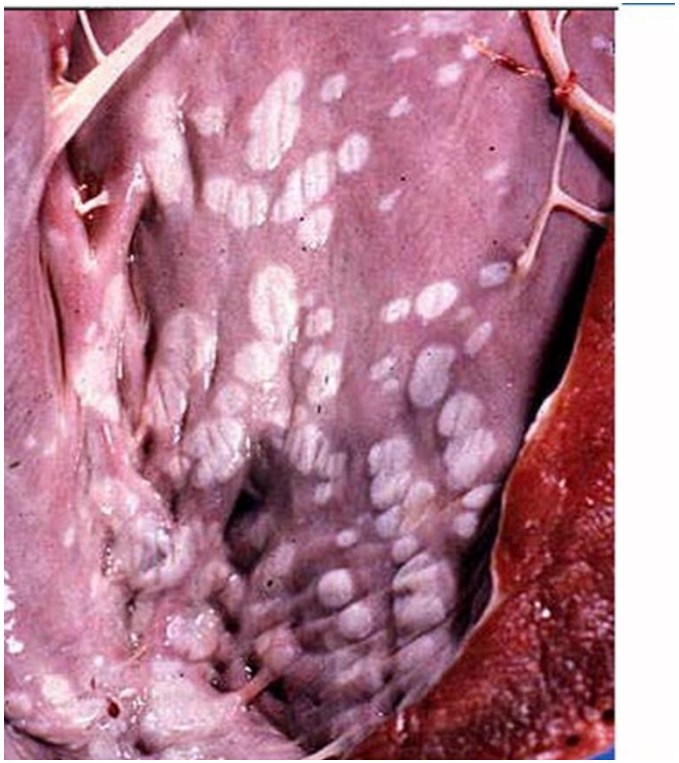


Glycogen storrage disease

(PAS stain)

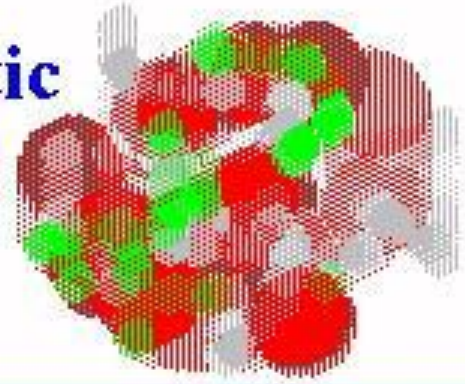
Pathological calcification

- **Extracellular deposition of calcium out side hard tissue (bone –teeth)**



Dystrophic Calcification

Necrotic tissue



Fatty acids
Mucopolysaccharides
glycogen
collagen
+

Calcium ions

Calcium salts

old abscess * parasitic cysts * thrombi * tumors
atherosclerosis * tuberculous nodules

Metastatic Calcification

low intake of Ca and P * Deficiency of vit. D

* Excessive excretion of Ca

lactating animals * diarrhoea * renal insufficiency

Hypocalcaemia

secondary hyperparathroidism

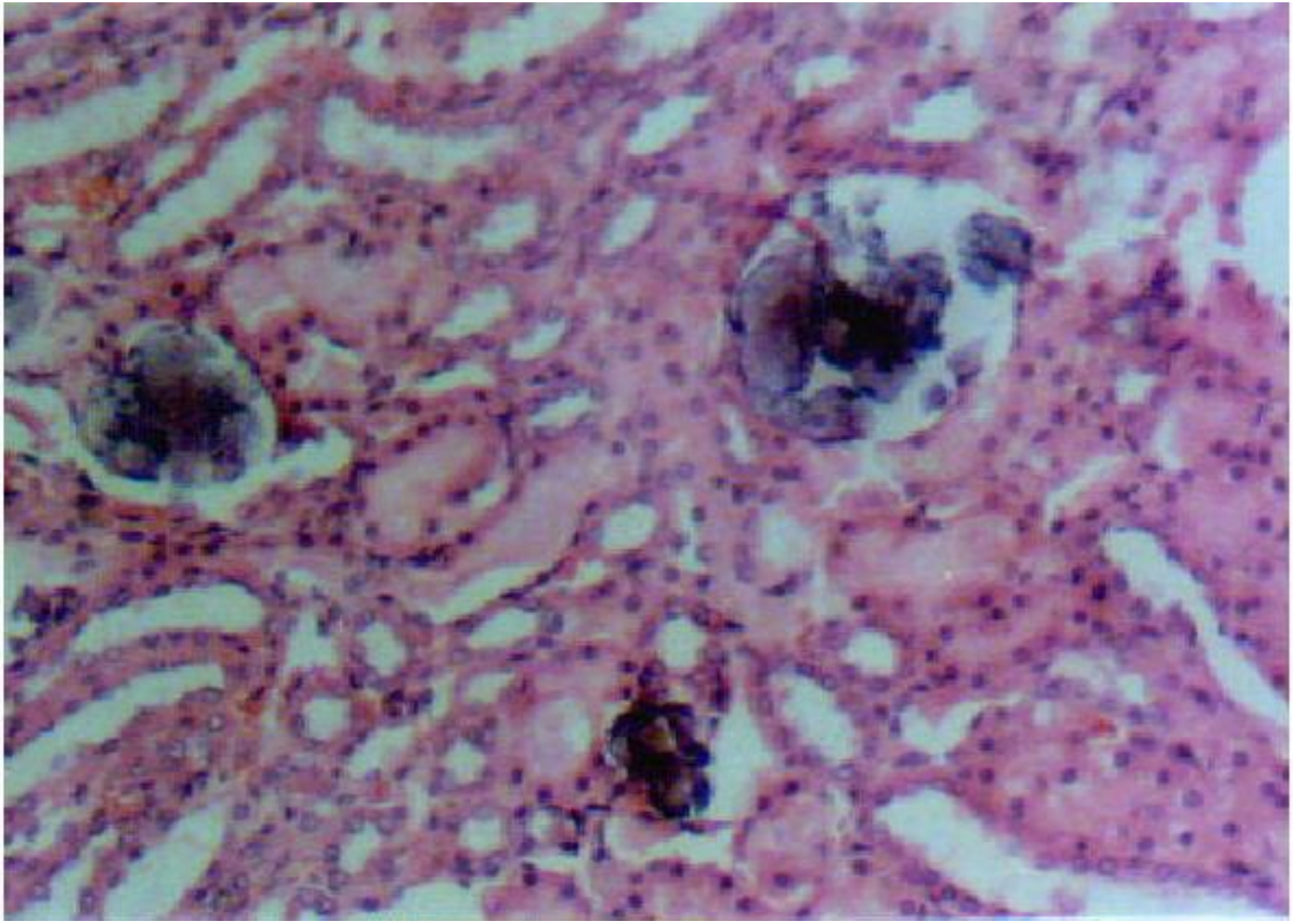
Hypervit. D
Excessive absorption of Ca

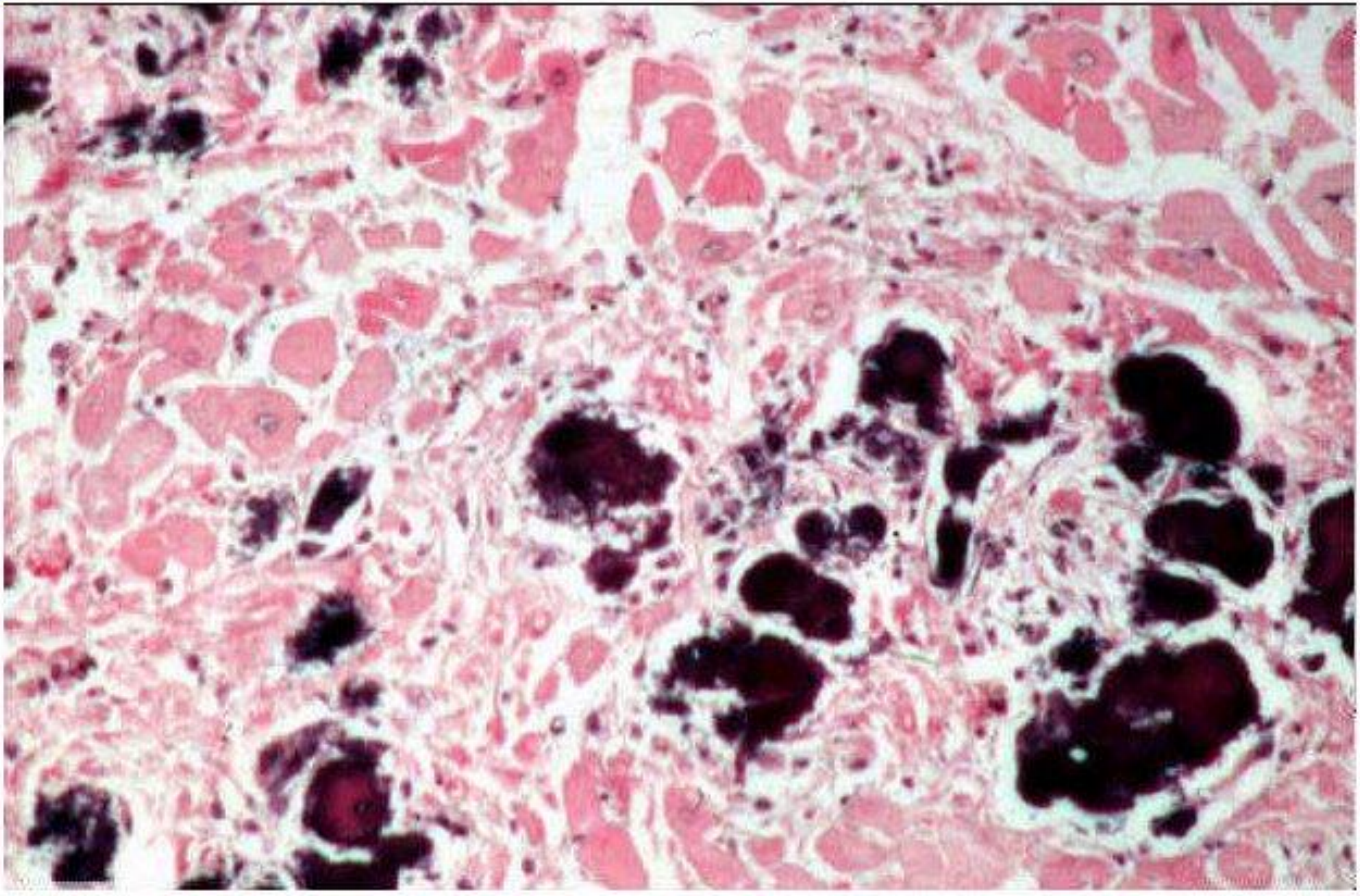
Bone

bone lesions
parathroid
adenoma

Hypercalcaemia

calcification of soft tissue





Disturbance of pigment metabolism

Endogenous pigment

- Bile pigment
- Haemoglobin*
- Haematin*
- Haemosidrin*
- Porphyrin
- Melanin
- Lipofuscin
- Ceroid pigment

Exogenous pigment

- Carbon particle
- Dust particles
- Carotenoids

* Iron-containing pigment

Jaundice (Icterus)

Presence of excessive amount of bilirubin (bile pigment) in the blood and discoloration of tissue

Haemolytic jaundice

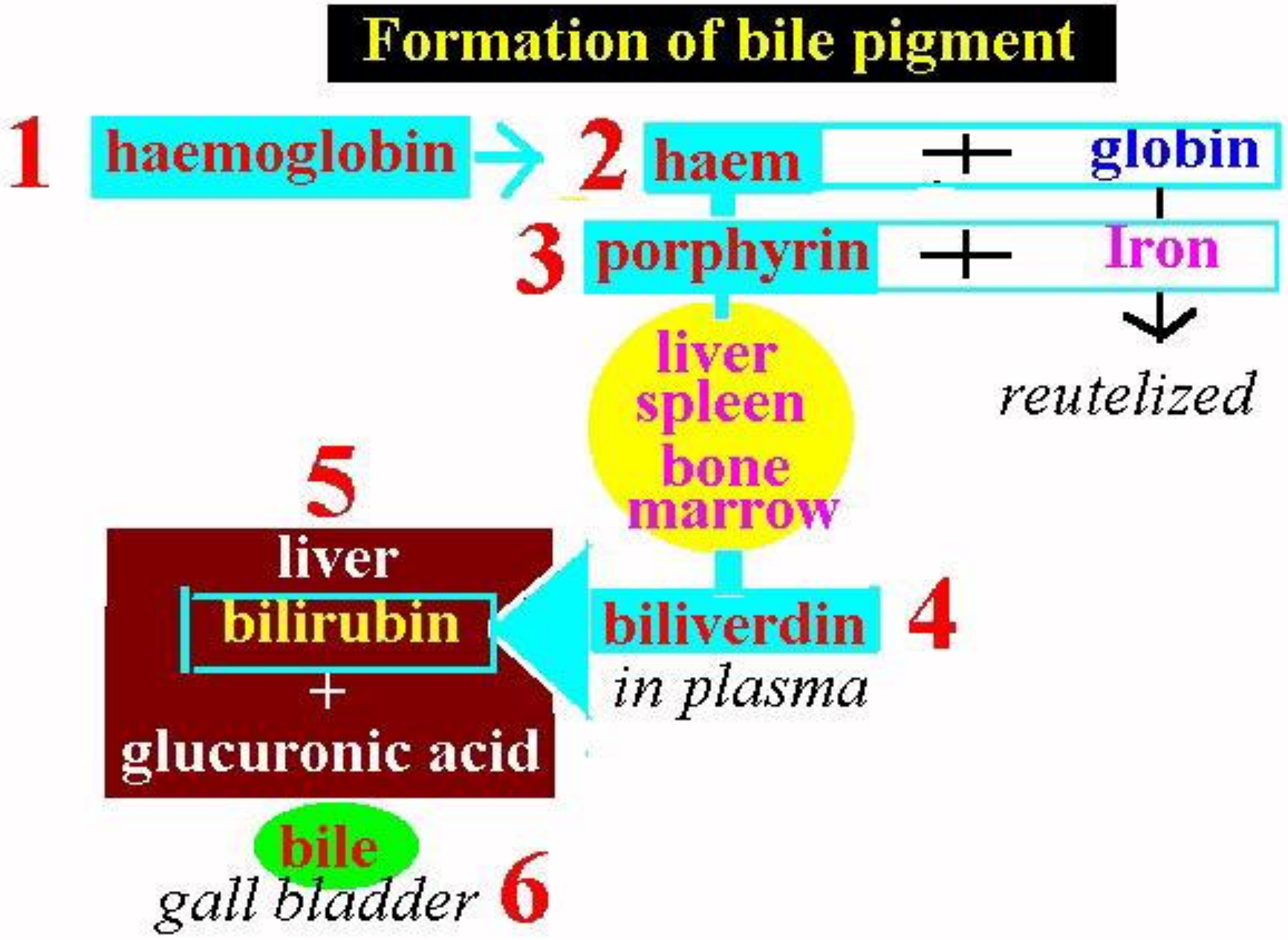
Increased production of bilirubin due to excessive haemolysis of red blood corpuscles

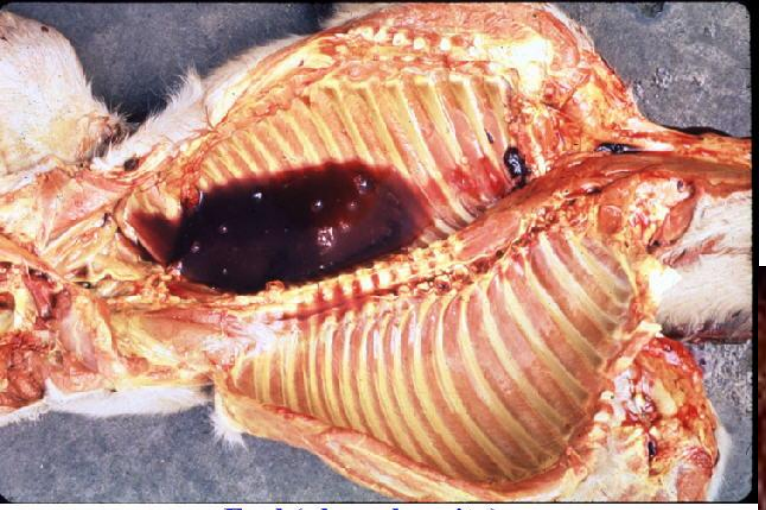
Toxic jaundice

Hepatic cells are damaged and can not perform their function leading to accumulation of bilirubin

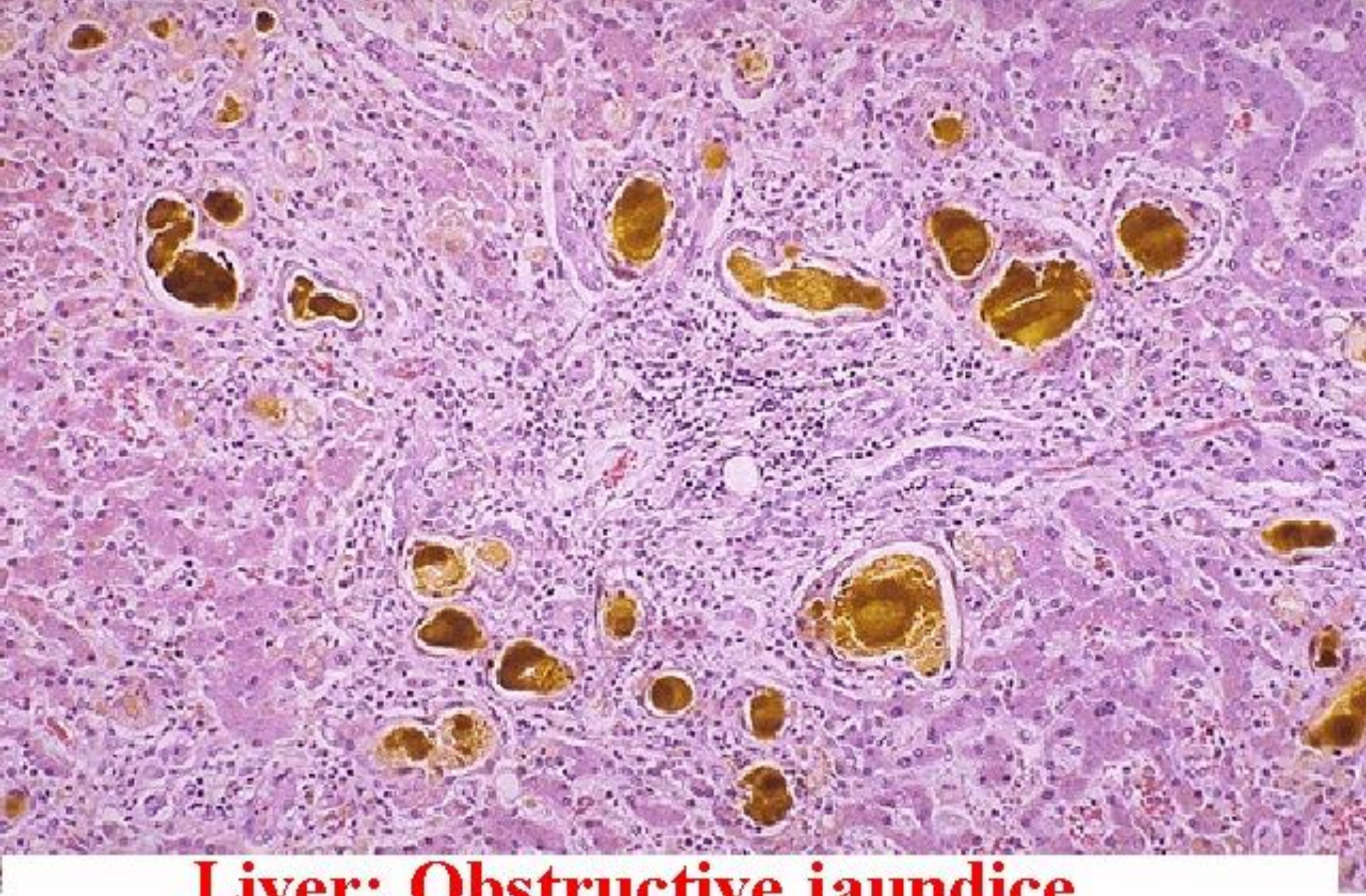
Obstructive jaundice

Obstruction of the normal flow of bile and reabsorption of retained bile in the blood


Formation of bile pigment
1
haemoglobin
2
haem
+
globin
3
porphyrin
+
Iron
reutelized
liver
spleen
bone
marrow
5
liver
bilirubin
+
glucuronic acid
biliverdin
4
in plasma
bile
gall bladder
6

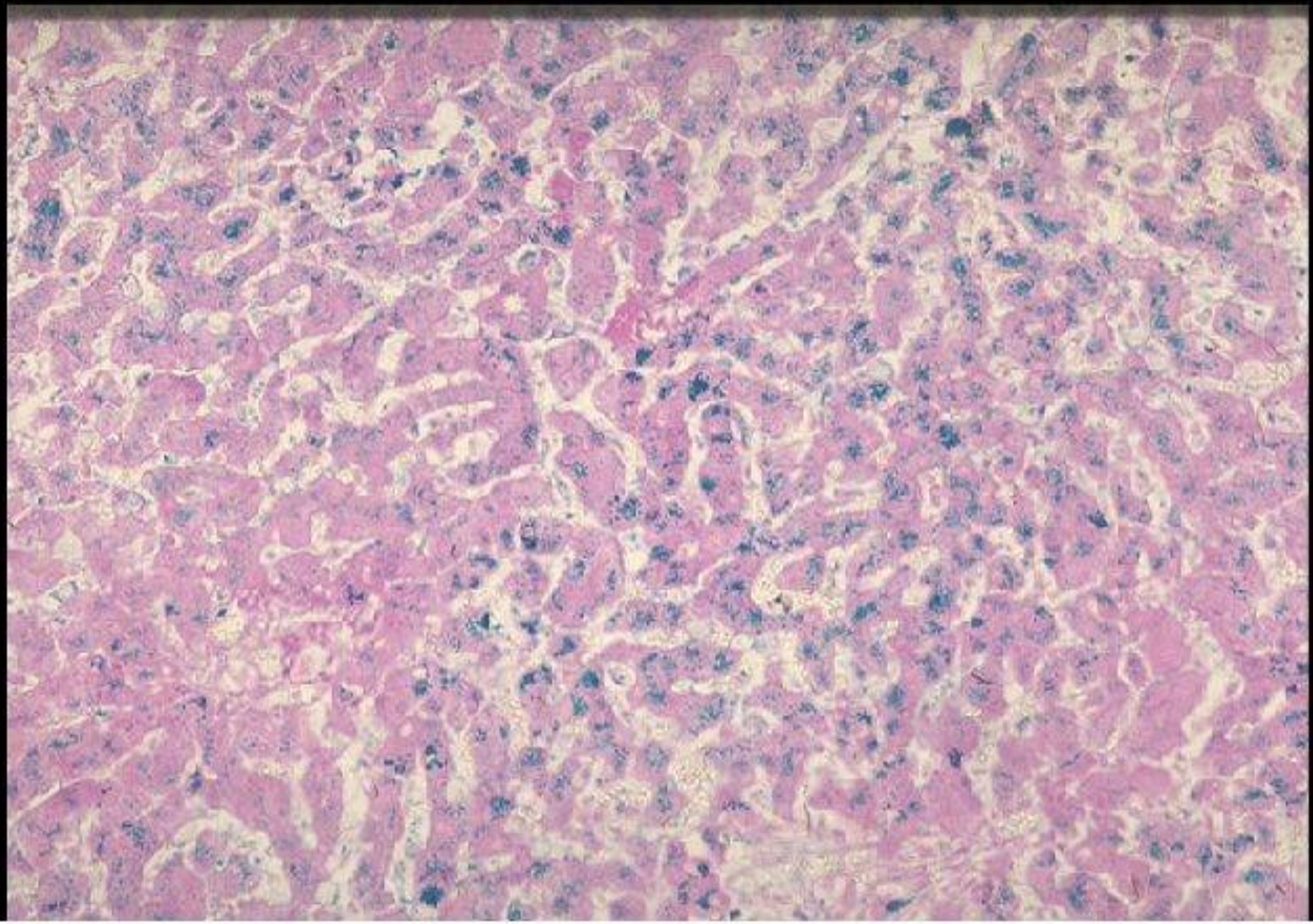


Foal (pleural cavity)
Icterus

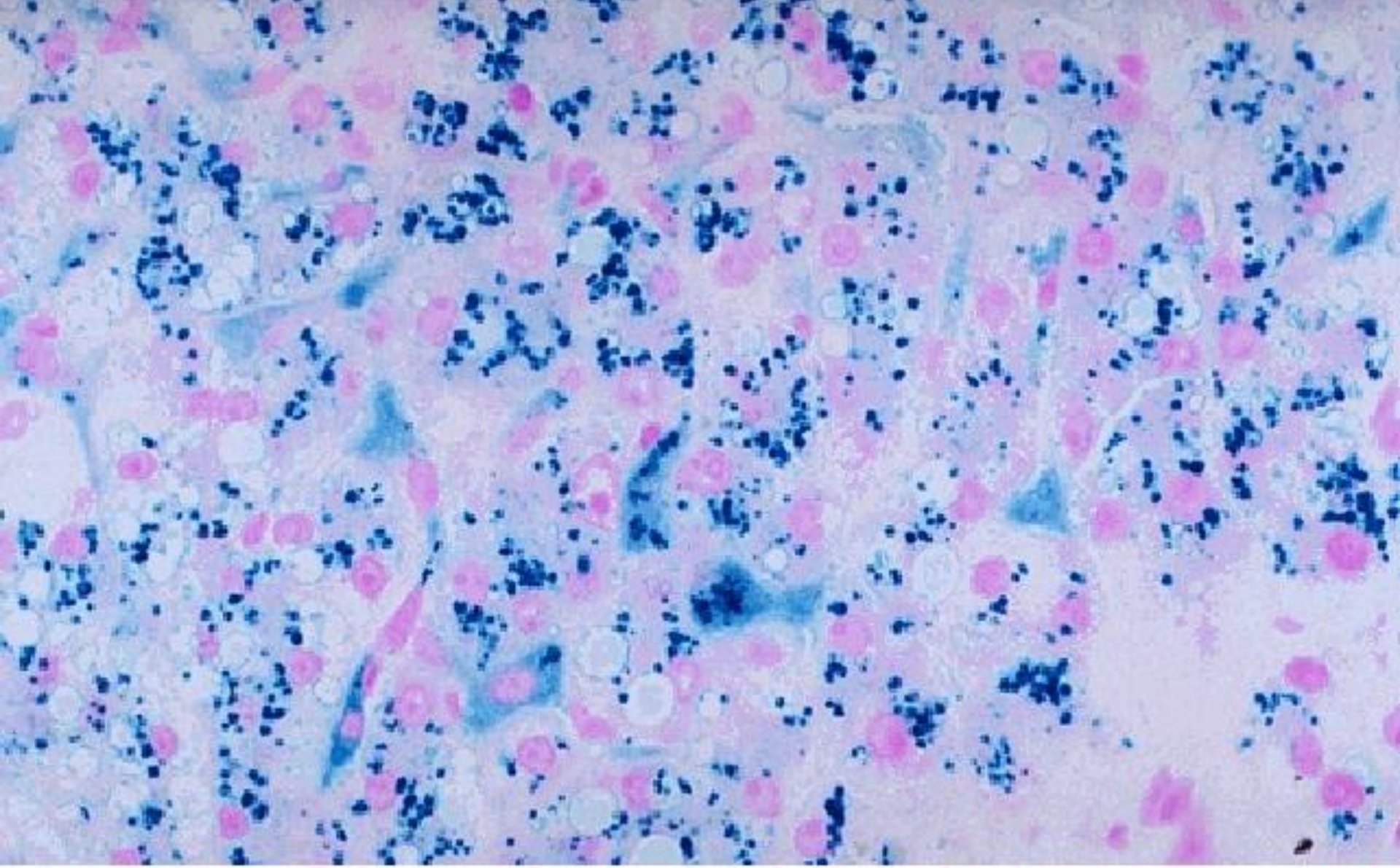


Liver: Obstructive jaundice

Presence of yellow-green globular material in small bile ductules in the liver



Liver: haemosiderosis



Liver: haemosidrosis

Accumulation of haemosidrin pigment in hepatocytes and Kupffer cells (Prussian blue stain)

Melanosis

Deposition of melanin pigment in abnormal sites in the body

Pathogenesis

overptoduction of melanin by melanocytes

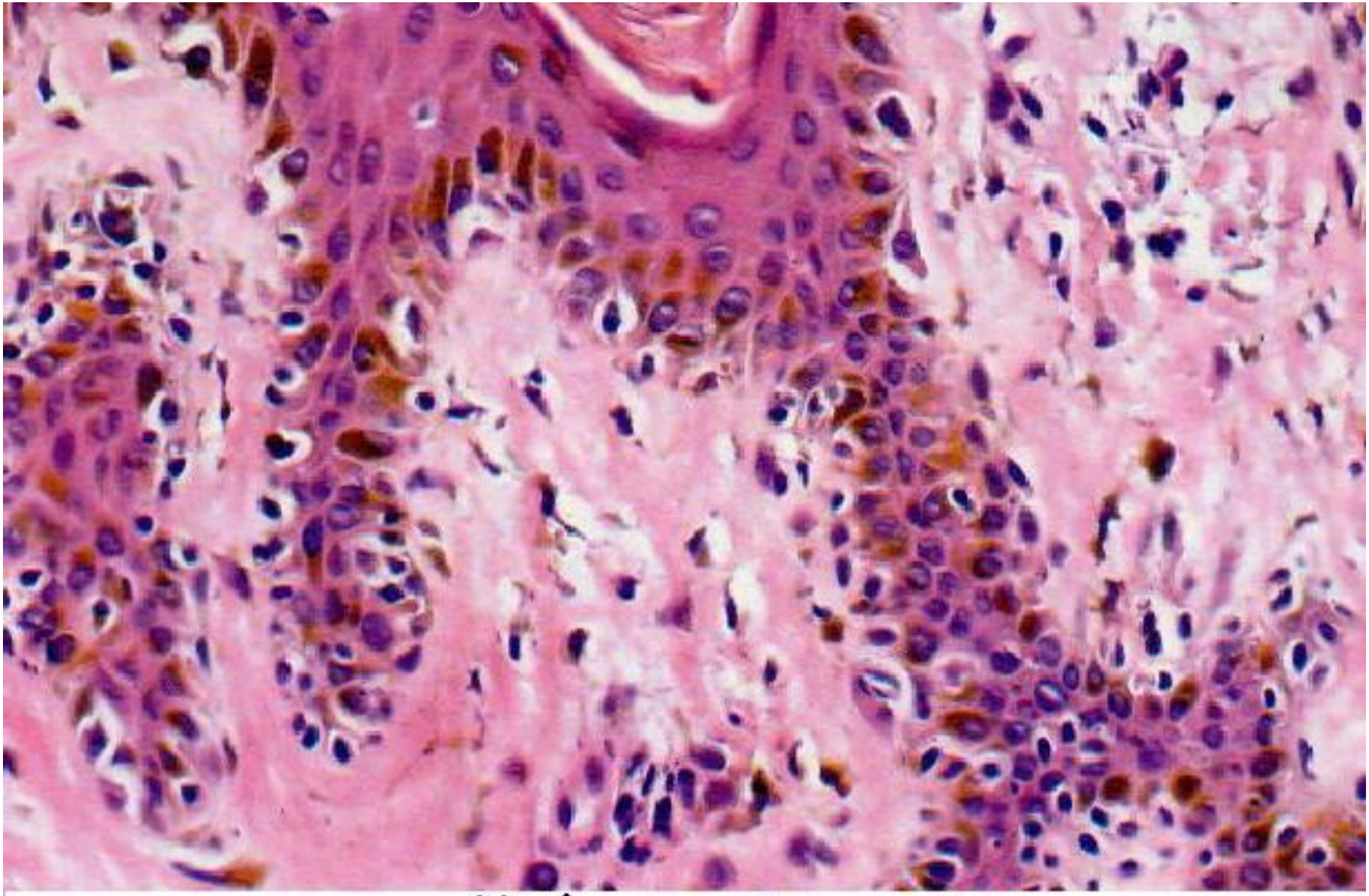
*

escape of the pigment outside the cells

*

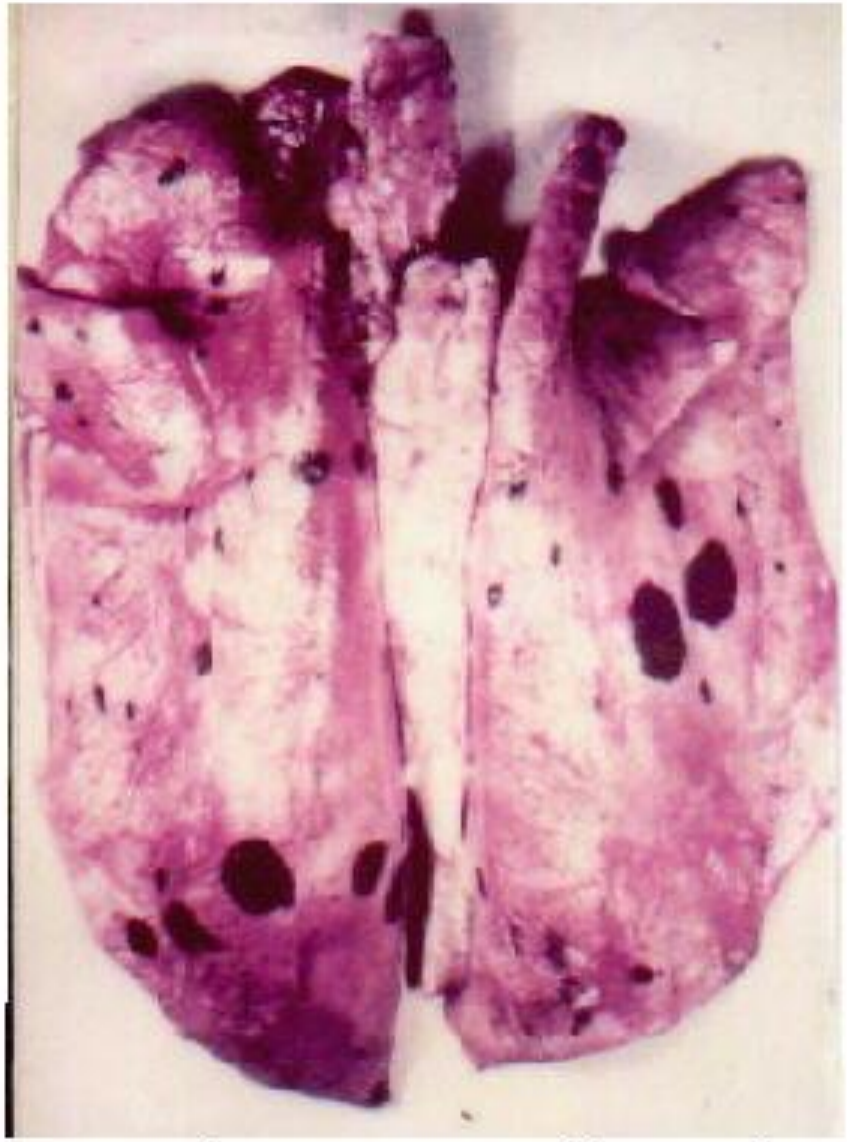
phagocytosis of the pigment by
- phagocytes (melanophores)

and their migration to different tissue and organs



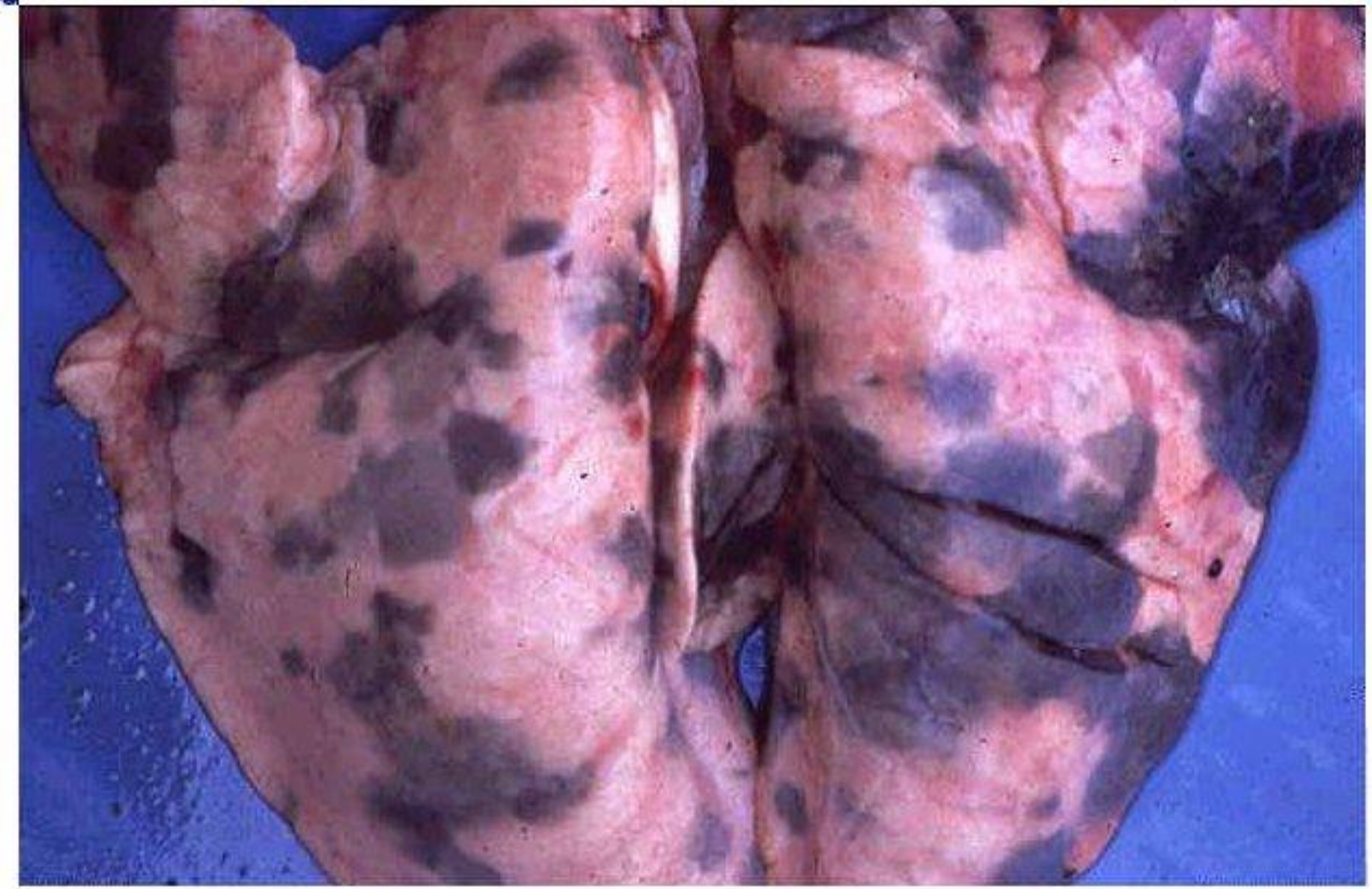
Melanin pigment

Brown-black granular pigment in the melanocytes of the epidermis and macrophages of the dermis



Melanosis (lung)

localized black areas in the pulmonary tissue



Lung (Pig): Normal melanosis

Pneumoconiosis

Deposition of mineral dusts in the lung

Types

Anthracosis Deposition of carbon particles (macrophages - connective tissue of alveolar septa)

Silicosis Deposition of silicon dioxide crystals

Asbestosis Deposition of asbestos (beads)

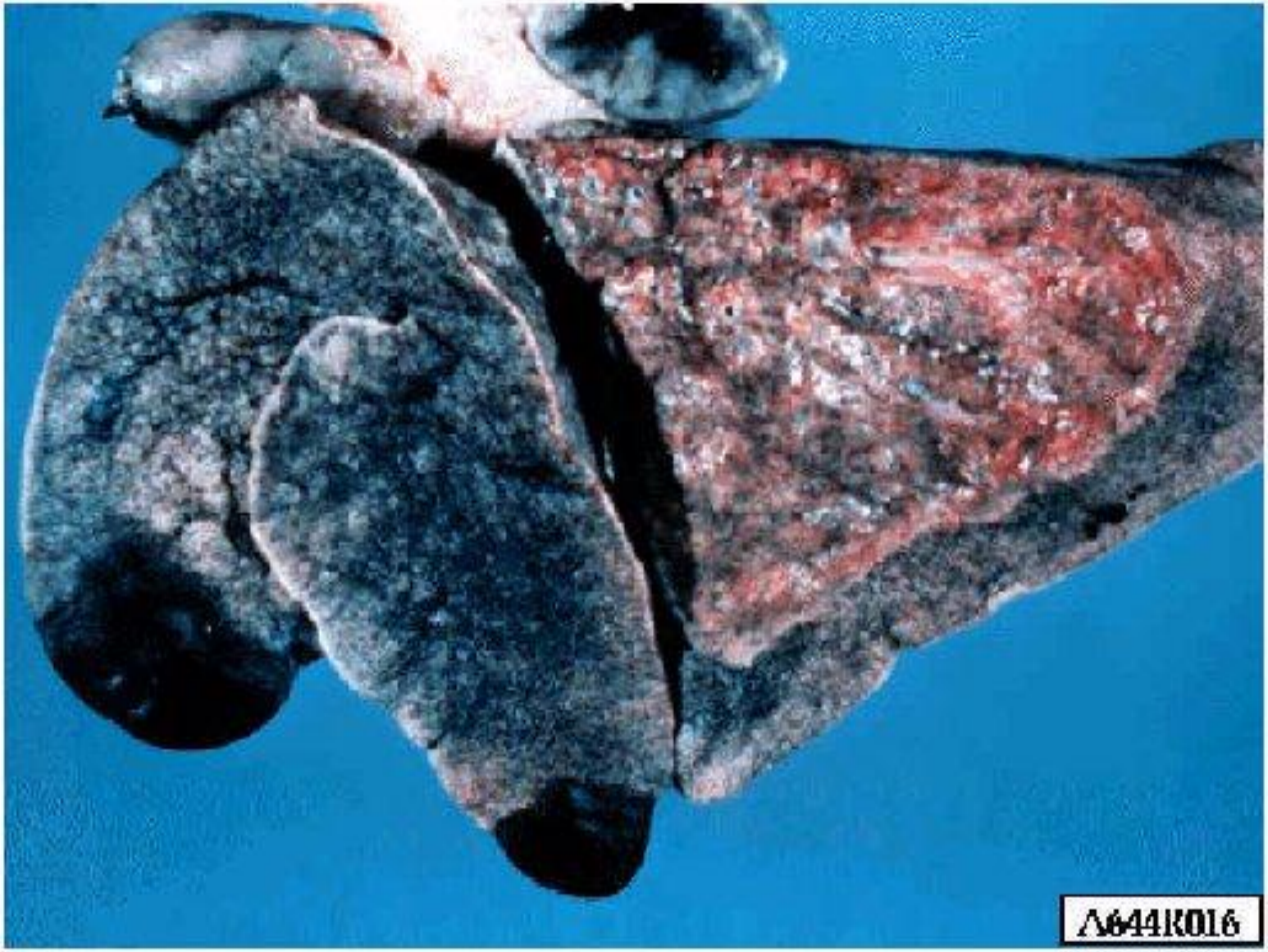
Siderosis Deposition of iron dust

Effect

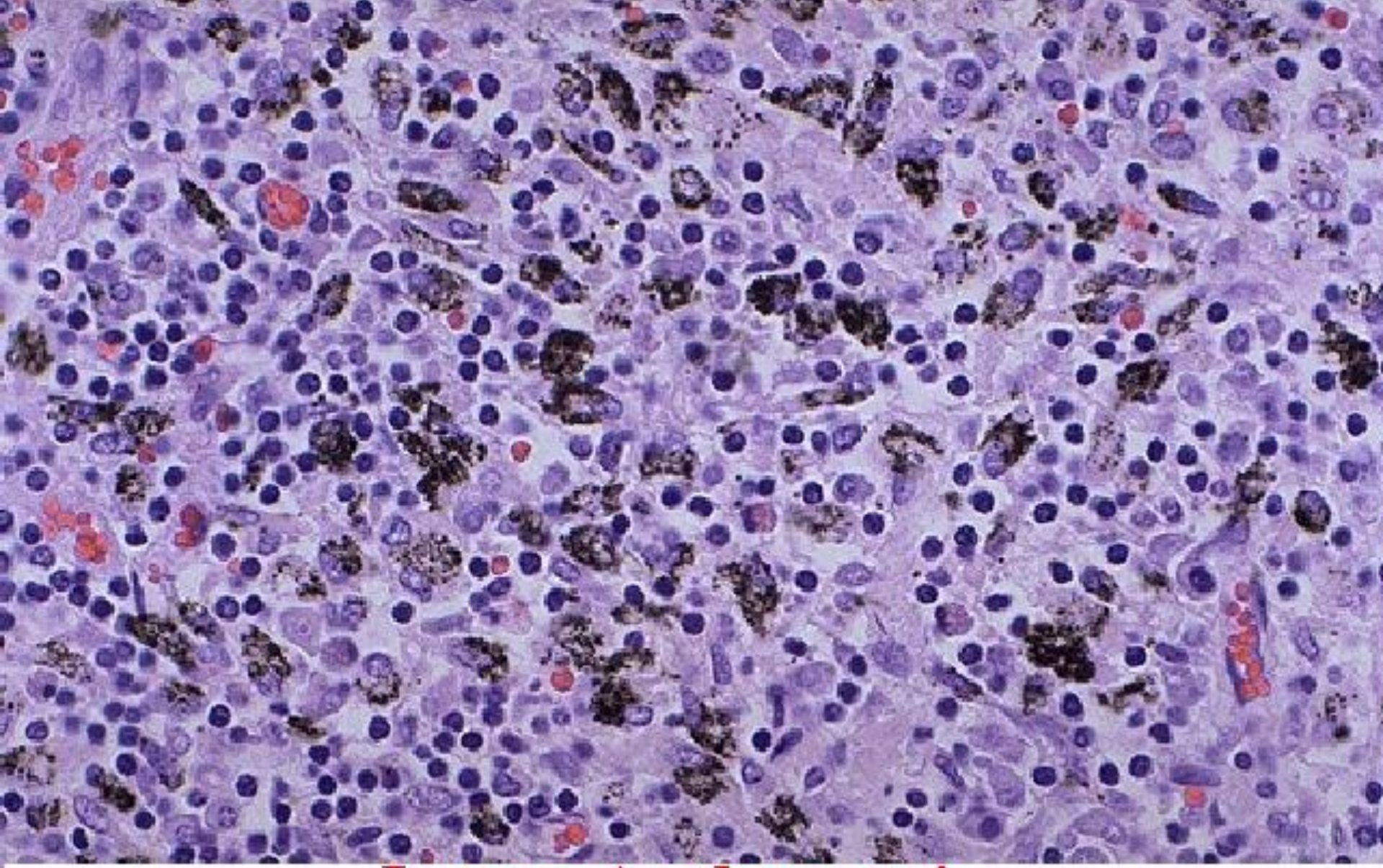
No effect (anthracosis)

Fibrosis (silicosis - siderosis)

Granulomatous reaction (asbestosis)



Lung
Anthracosis



Lung: Anthracosis

Deposition of carbon particles in the macrophages of hilar lymph node (of the lung)

Hyaline degeneration

It is intracellular and extracellular protein accumulation that display homogenous glassy pink appearance in H&E stain

Hyaline Change

Hyaline degeneration

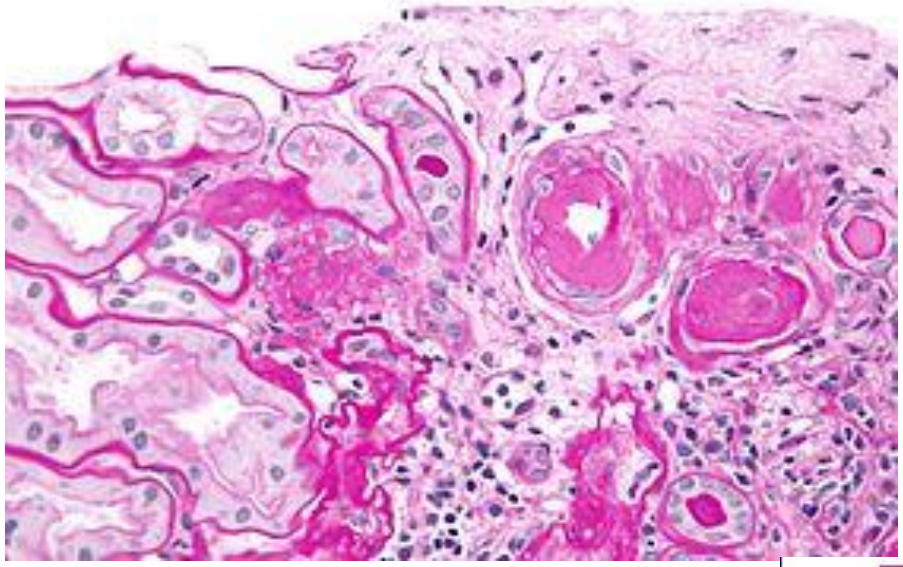
Tissue becomes grossly white, glassy, and dense and microscopically highly eosinophilic and homogenous

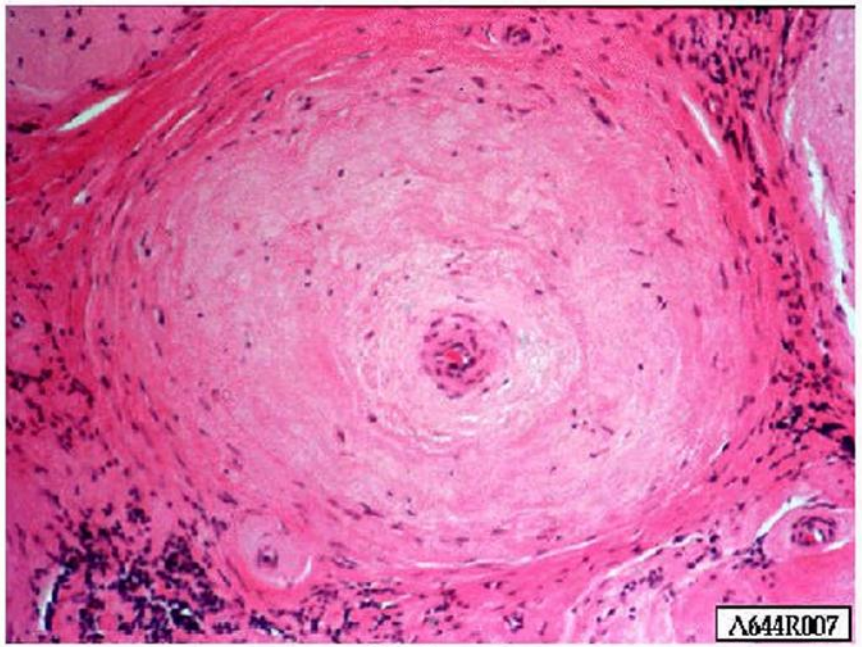
Connective tissue hyalinosis

- Old scars
- Corpus albicans
- Arteriosclerosis
- Chr.nephritis

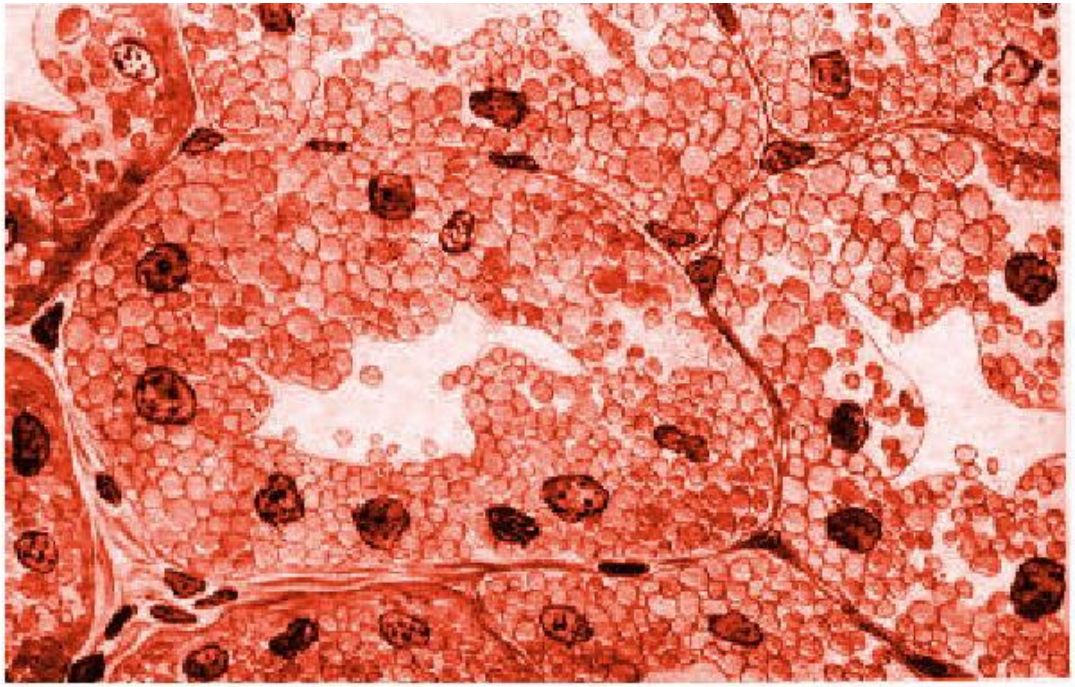
Cellular hyalinosis

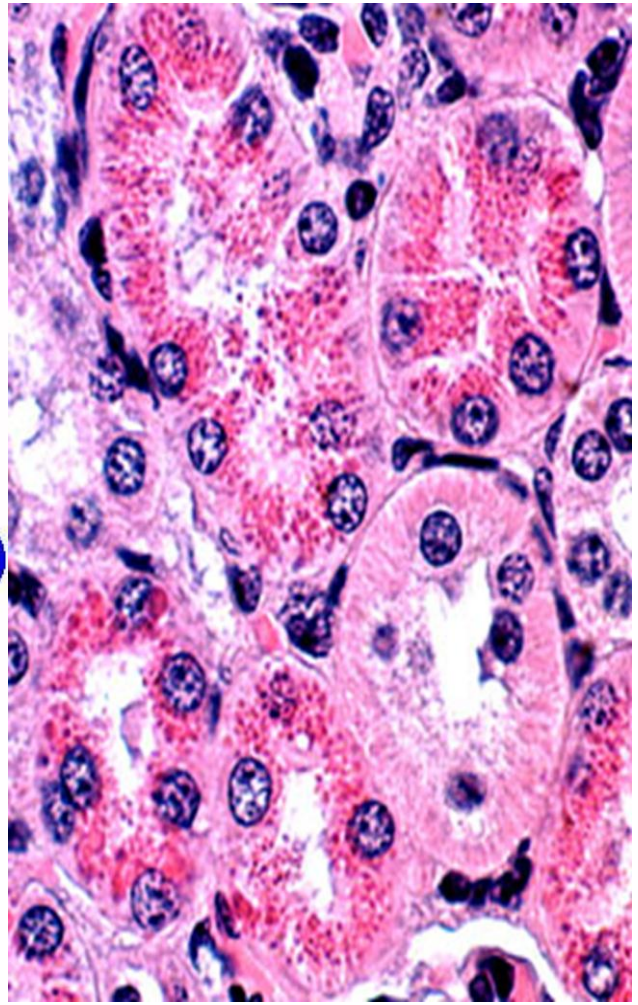
- Renal hyaline casts
- Corpora amylacea
- Russell's bodies
- Kratohyaline
- Muscles





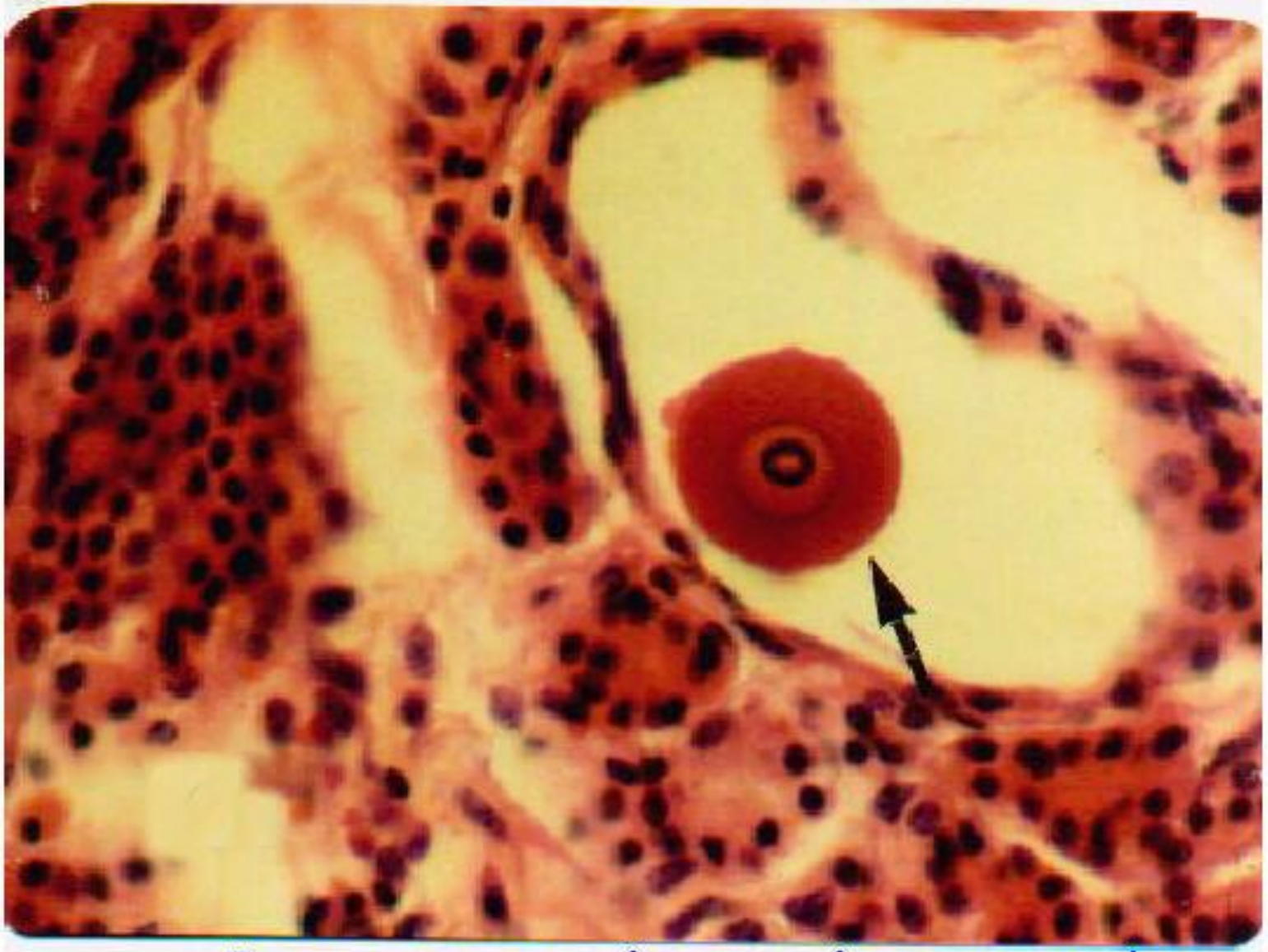
Spleen
Hyaline change of splenic artery





Hyaline droplet degeneration (kidney)

hyalinized droplets in degenerating renal tubules



Corpora amylacea (prostate)

Hyalinization of desquamated epithelial cells of the acini

Keratin Dystrophy

Hyperkeratosis

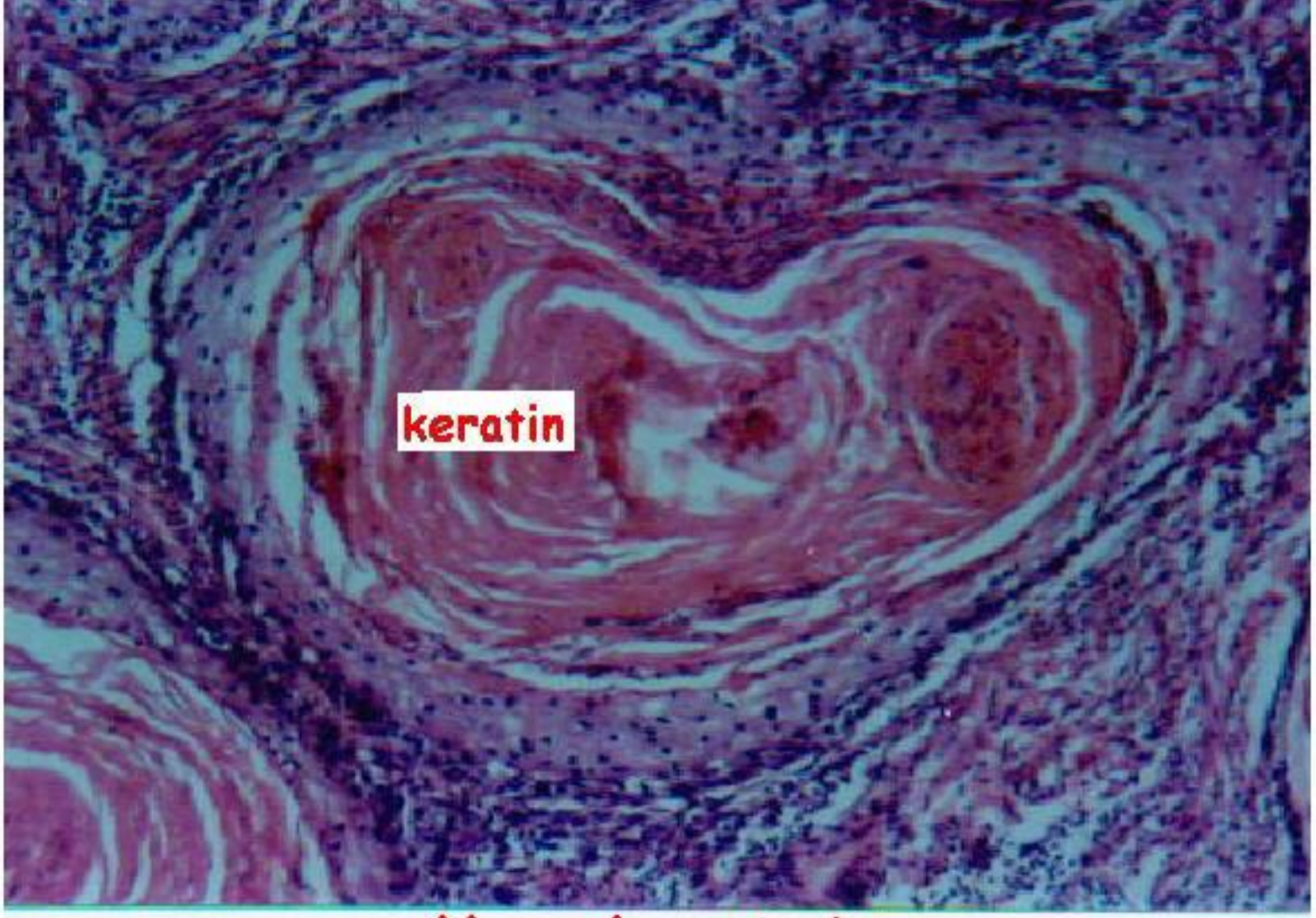
Excessive production
Thick str.corneum
Normal str.granulosum

Vit. A deficiency
Prolonged friction
Viral infection
Tumours
Poisoning

Parakeratosis

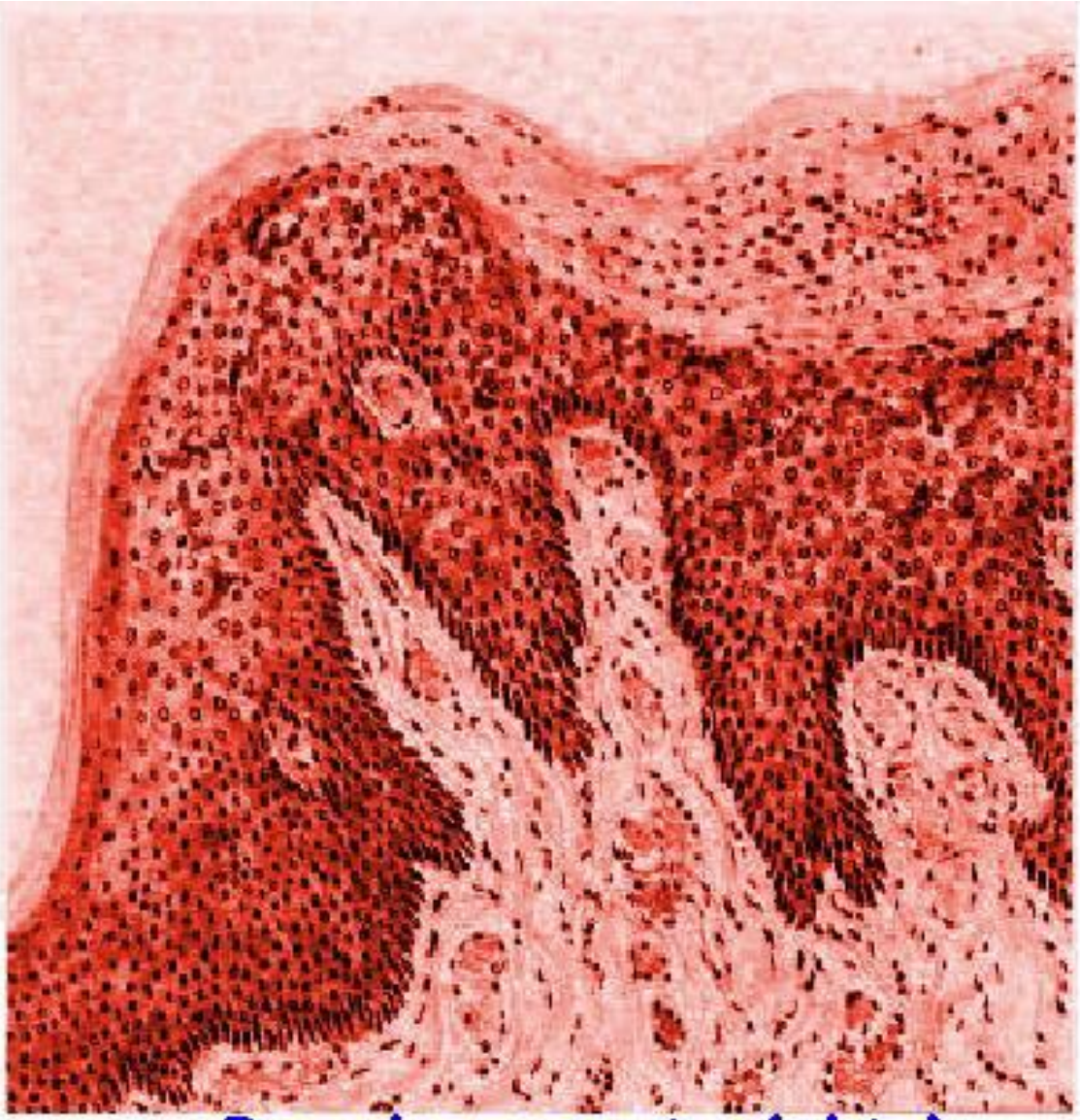
Deficient production
Thin str.corneum
Thickened str.granulosum

Zinc deficiency



Hyperkeratosis

Increased amount of keratin in squamous cell carcinoma



Parakeratosis (skin)

Increase in the thickness of stratum granulosum

Amyloidosis

It is a disease results from extracellular accumulation of abnormal protein (Amyloid)

Amyloidosis

Chronic wasting diseases

Chronic suppurative inflammation

Amyloid substance deposited outside the blood vessels

amyloid substance may be

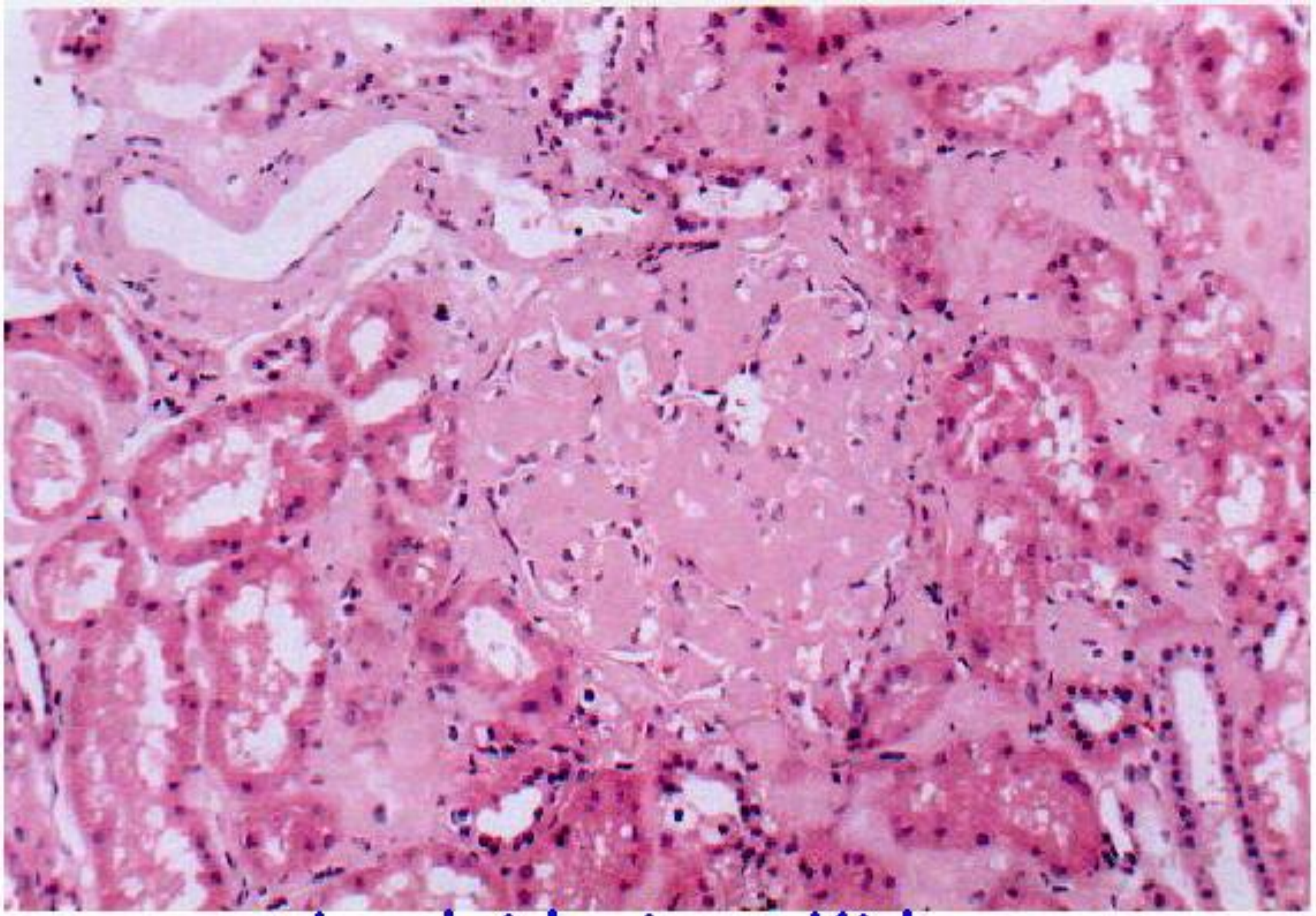
Abnormal immunoglobulins
Antigen-antibody complex
Abnormal plasma protein

Types

- Systemic amyloidosis
- Localized amyloidosis

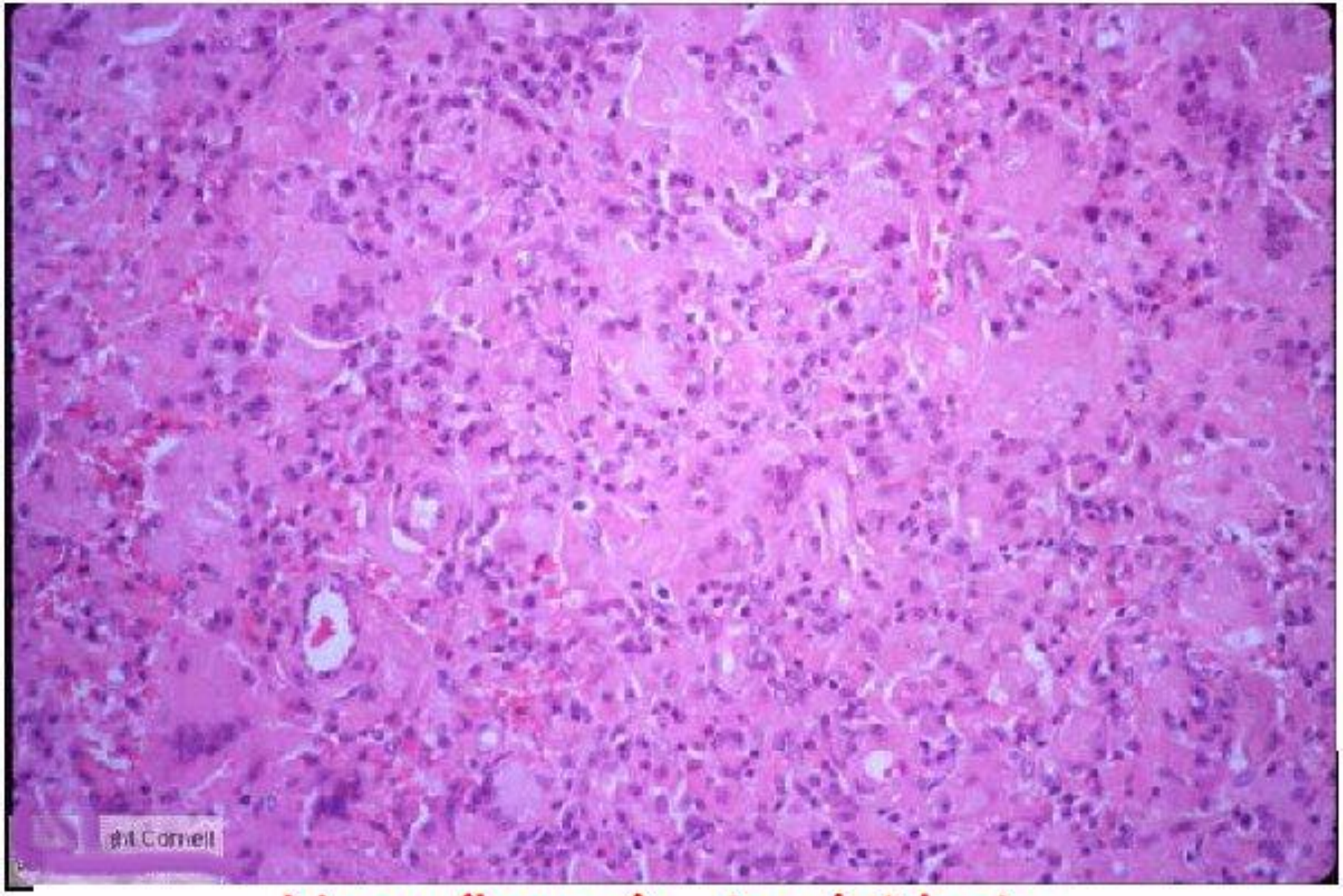
Clinical picture

- Heart failure
- Renal failure
- Splenomegaly
- Hepatomegaly and hepatic failure
- Diabetes mellitus



Amyloidosis - Kidney

Infiltration of amyloid substances in the glomerulus and interstitial tissue around blood vessels

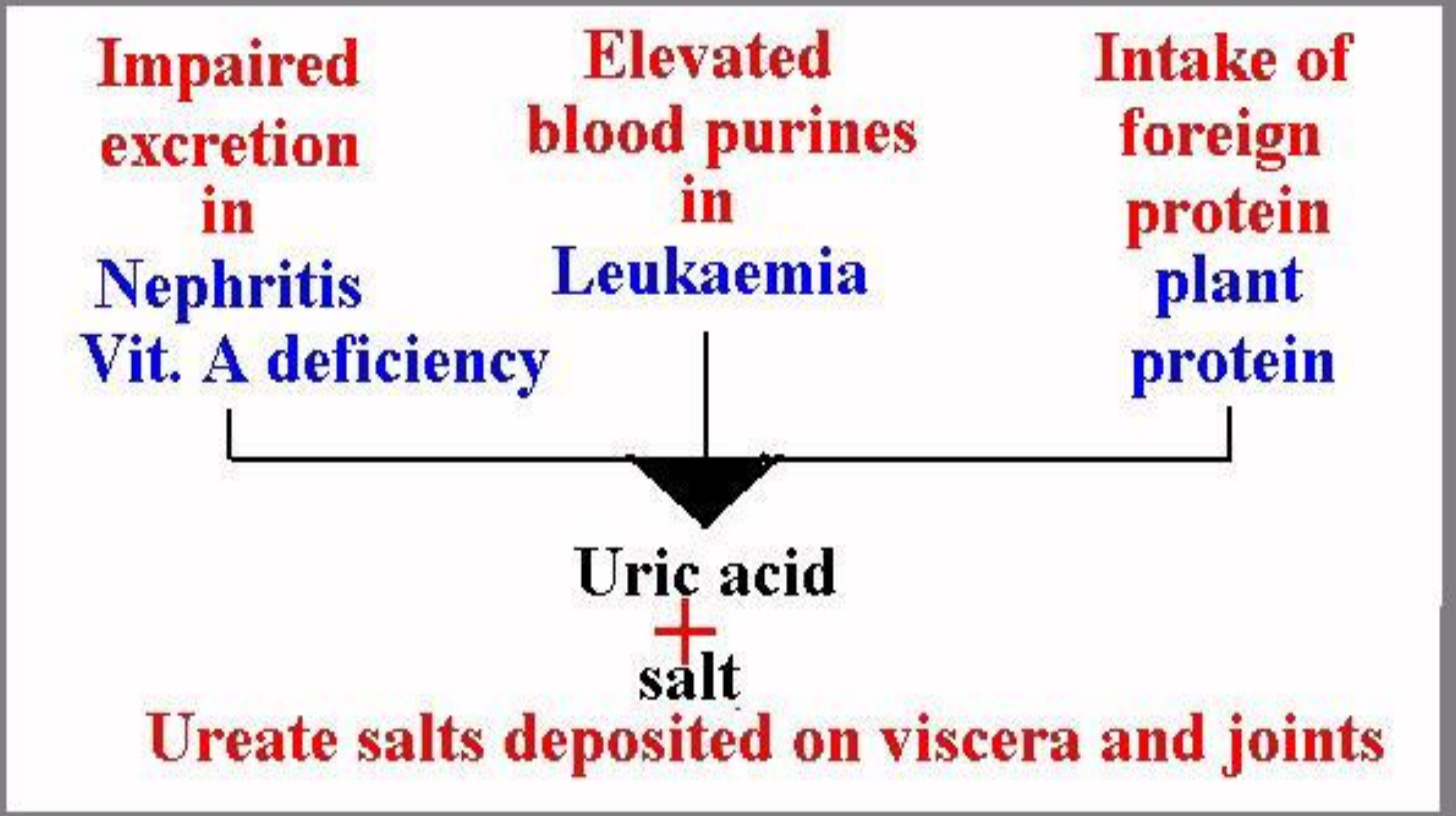


Nose (horse): Amyloidosis

Infiltration of pink material in the subcutaneous tissue with many multinucleated giant cells

Gout

Disturbance of nucleoportein metabolism



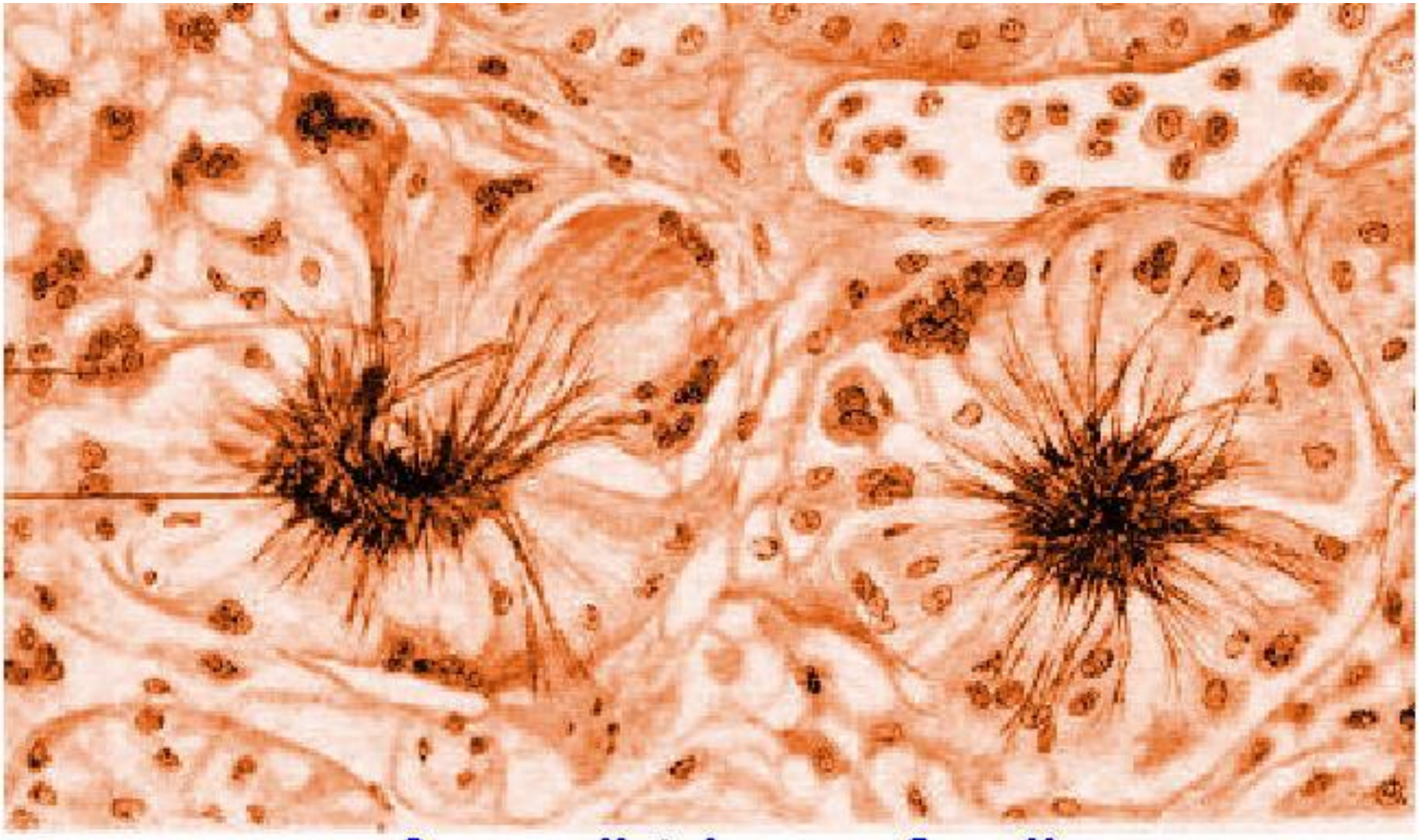
Gout

Clinically :

Acute arthritis

Soft tissue nodules

renal stone



Gout (kidney-fowl)

Needle-shaped ureate crystals in renal tubules

Mucin deposition

Intracellular (mucoid degeneration)

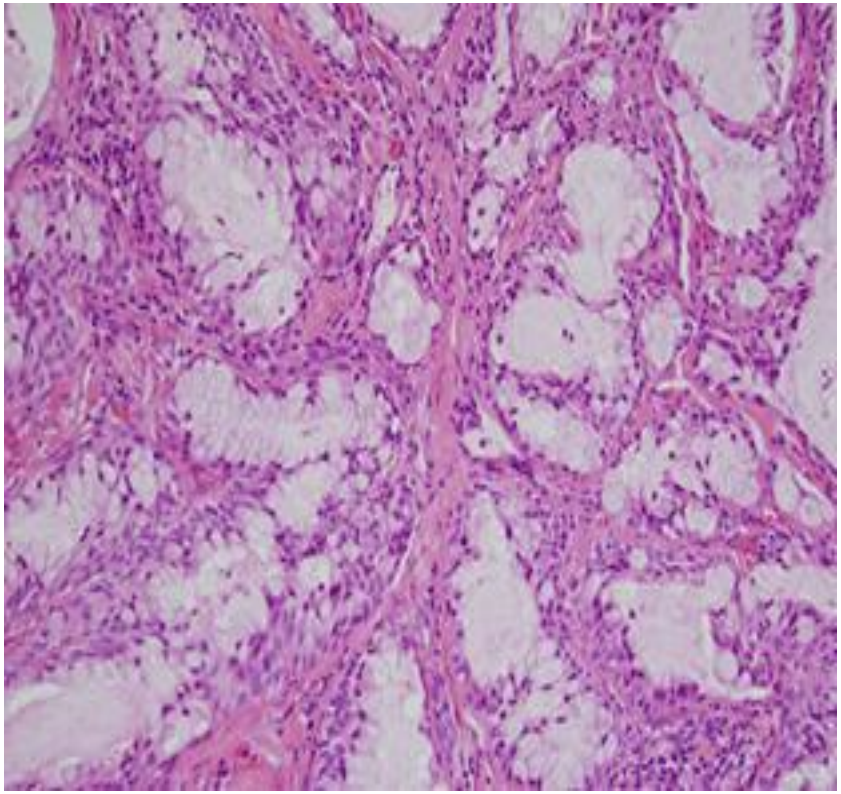
Extracellular (myxematous degeneration)

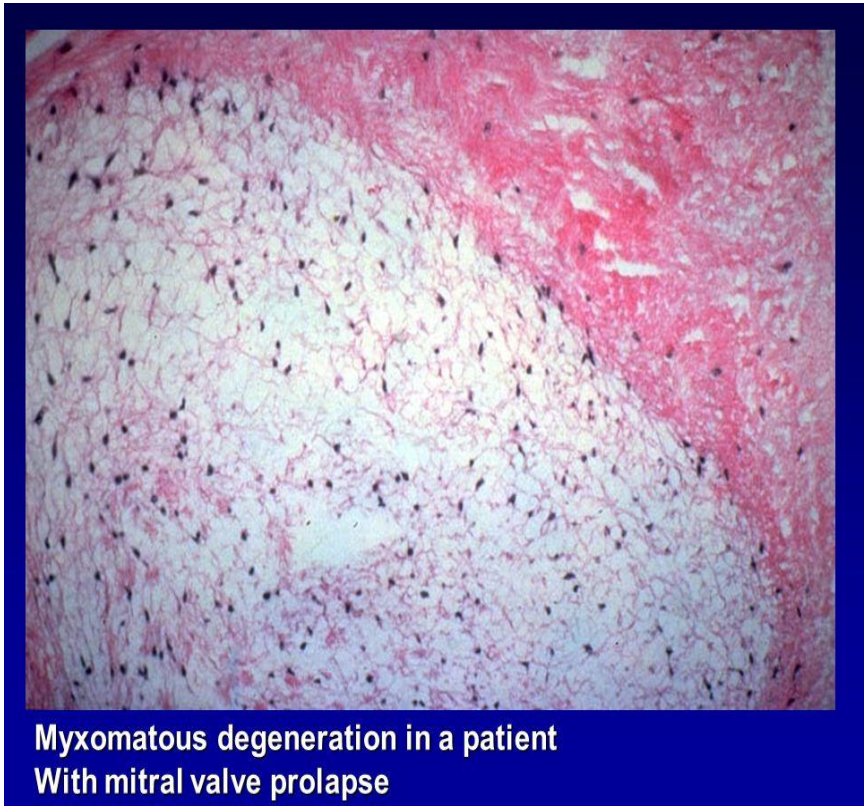
mucoid

- Accumulation of mucin inside cells which swell and become vacuolated with eccentric nuclei (**signet – ring cells**)
- Cell may rupture leading to extracellular pool of mucin (**ghost-like cells**)

myxematous

- **It is the accumulation of mucin between c.t. fibers**





Myxomatous degeneration in a patient
With mitral valve prolapse