V.I. Vernadsky Crimea Federal University
Medical Academy named after S.I. Geogievsky

Pathological Anatomy Introduction

Lecture # 1

International Medical Faculty
Pathological Anatomy Department

Head of the Department - PhD Kriventsov M.A.

PLAN OF THE LECTURE

1. **Introduction**
    1. Pathological anatomy and anatomic pathologist
    2. History
    3. Tasks of the pathological anatomy
    4. Biopsy, operational material, autopsy
2. **Damage (alteration) Definition, factors, adaptation limits.**
    1. Morphology of the damage (alteration)
    2. Definition, classification, mechanisms of degenerations
    3. Parenchymal (intracellular) degenerations
    4. Stromal vascular (extracellular) degenerations
    5. Disorders of hemoglobin derived pigments (porphyria, jaundice, hemosiderosis)
    6. Melanin
    7. Calcinosis

Pathological anatomy is the science that studies the structural bases of the disease at different levels of morphological organization

Anatomic pathologist (pathomorphologist) is a doctor who deals with the identification of disease based on the normal structure of the human body anatomy

History of the pathological anatomy

1) **In 1761 Italian author G. Morgagni wrote the first work on pathological anatomy "About the location and causes of diseases revealed through the incision".**

2) **Carl Rokitansky - a member of the Vienna and Paris Academy of Sciences. He created Europe's first department of Pathological Anatomy (in 1844). Rokitansky considered that the main cause of painful changes is a violation of the composition of fluids of an organism.**

Giovanni Battista Morgagni (25/02/1682 - 6/12/1771)

Carl von Rokitansky (19/02/1804 - 23/07/1878)

History of the pathological anatomy

3) **The founder of modern pathological anatomy is R. Virchow (1821—1902) - German researcher who created the doctrine of cellular pathology.**

Theoretic tasks of the pathological anatomy:

1) Study of the etiology, pathogenesis, morphology and morphogenesis of the diseases;
2) Study of pathomorphism of the diseases (medical, natural);
3) Study of outcomes and complications of the diseases;
4) Study of the mechanism of death (tanatogenesis);
5) Evaluation of the functioning and state of damaged organs.

Practical tasks of the pathological anatomy:

1) Control of accuracy and timeliness of clinical diagnosis;
2) Training of the attending physician;
3) Establishing clinical diagnosis in vivo (during the patient's life);
4) Monitoring the effectiveness of treatment (repeated biopsy);
5) Statistical records.

Approaches in the pathological anatomy

1) Post mortal study (autopsy);
2) In vivo (during the life) study (biopsy, operational material);
3) Experiment.

Methods of the pathological anatomy

- Macroscopic
- Microscopic (light microscope)
- Electron microscope
- Cytochemistry
- Histochemistry
- Immunohistochemistry (IHC)

Pathological Anatomy

General pathology studies typical pathological processes specific to a particular disease.

1. Damage of cells and tissues
2. Circulatory disorders
3. Regeneration and compensation processes
4. Inflammation
5. Tumors

Systemic pathology studies causes of diseases (**ethiology**), mechanism (**pathogenesis**), morphological basis of these mechanisms (**morphogenesis**) and mechanisms of death (**tanatogenesis**).

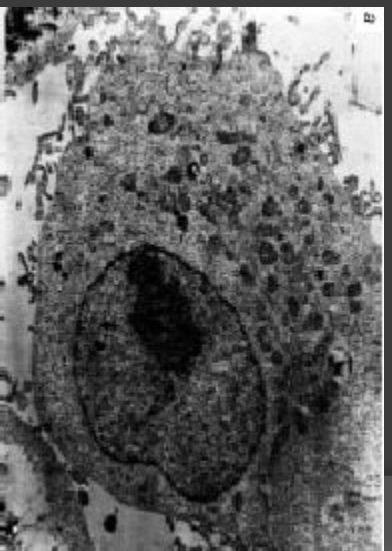
1. Ethiology, pathogenesis and morphology of diseases

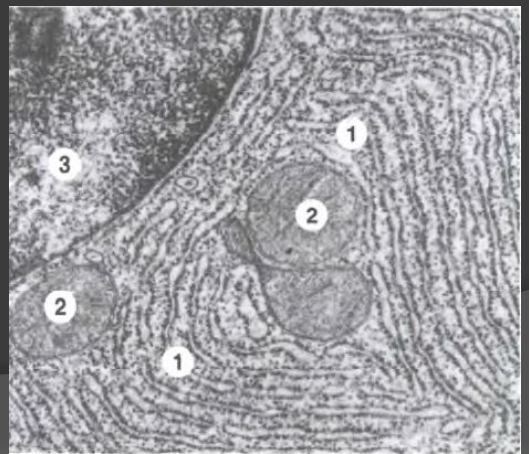
Damage

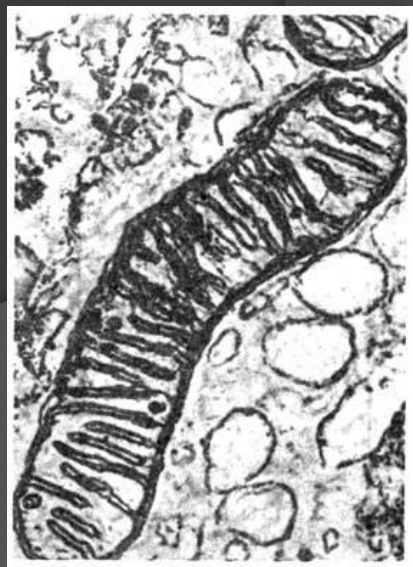
or alteration (from the Latin alteratio - change) is the changes in the structure of cells, intercellular substance, tissues and organs, which are accompanied by a violation of their life.

Adaptation limits (reversible/irreversible) depend on tissue type and its functional activity, strength and duration of exposure to the damaging factor.

Damage factors (physical and chemical factors, ischemia, infection, intoxication, immune response).







MORPHOLOGY OF THE CELLULAR DAMAGE

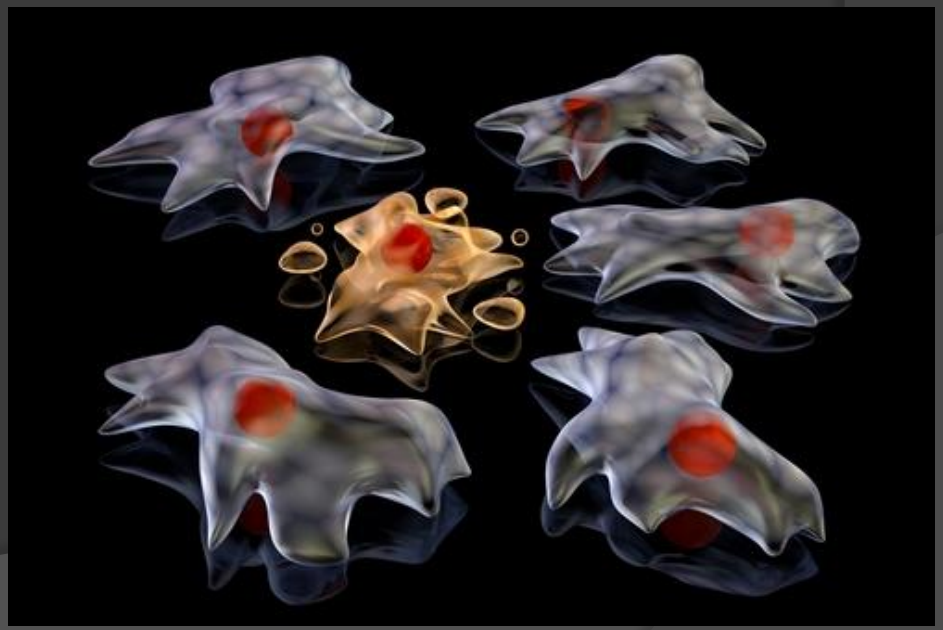
DEGENERATIONS

METABOLIC DISORDERS, LEADING TO CHANGES IN THE STRUCTURE

NECROSIS

APOPTOSIS

Death of cells, tissues, organs or body parts in live organism



DEGENERATION

Gr.: dys - violation; trophe - nutrition

MECHANISMS OF DEGENERATIONS

1. **Transformation** (ability of some substances turn into the other, which are close enough in structure and composition. For example, carbohydrates can be transformed into lipids)
2. **Decomposition** (break down of the intracellular structures)
3. **Perverted synthesis** (formation of abnormal substances, i.e. amyloid, alcoholic hyaline)
4. **Infiltration** (excessive penetration of a substance into the cell)

CLASSIFICATION

I. By localization

1. Intracellular (parenchymal);
2. Extracellular (stromal vascular, mesenchymal);
3. Mixed

II. By extent

1. General (systemic).
2. Local.

III. By etiology

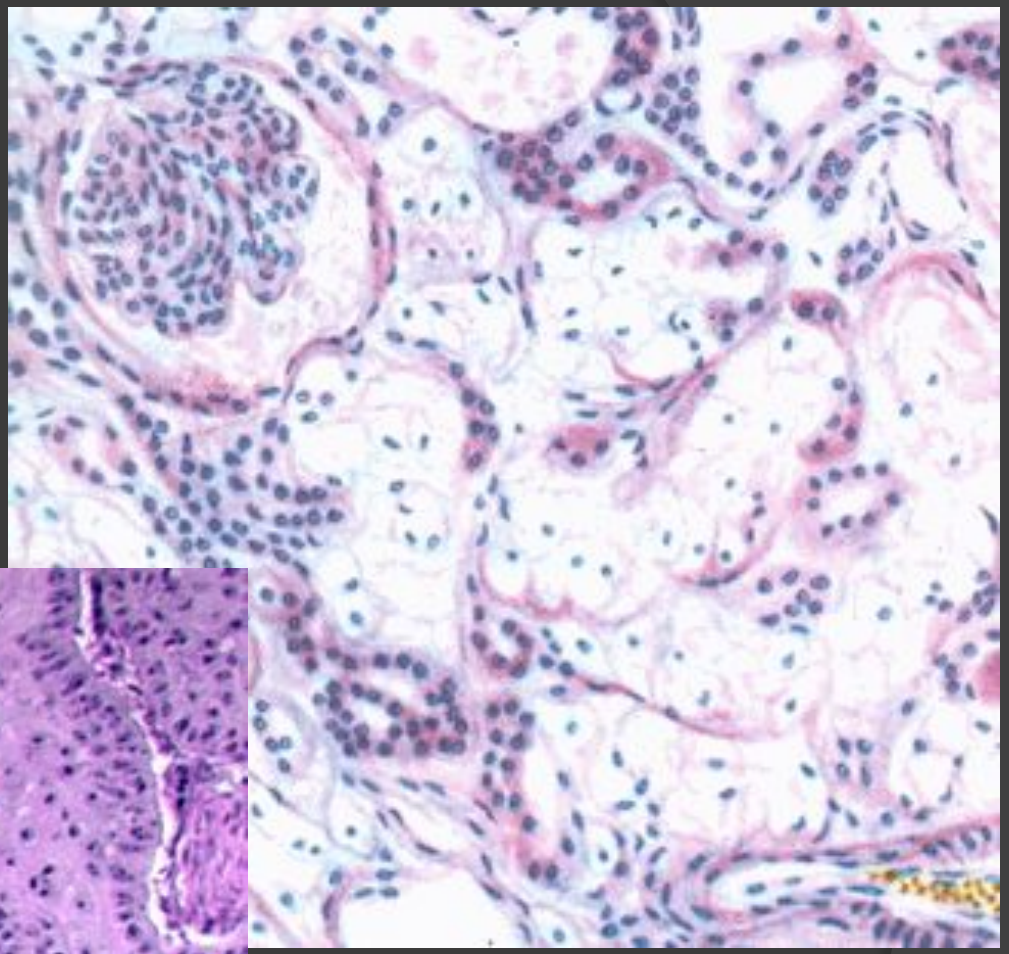
1. Acquired
2. Hereditary

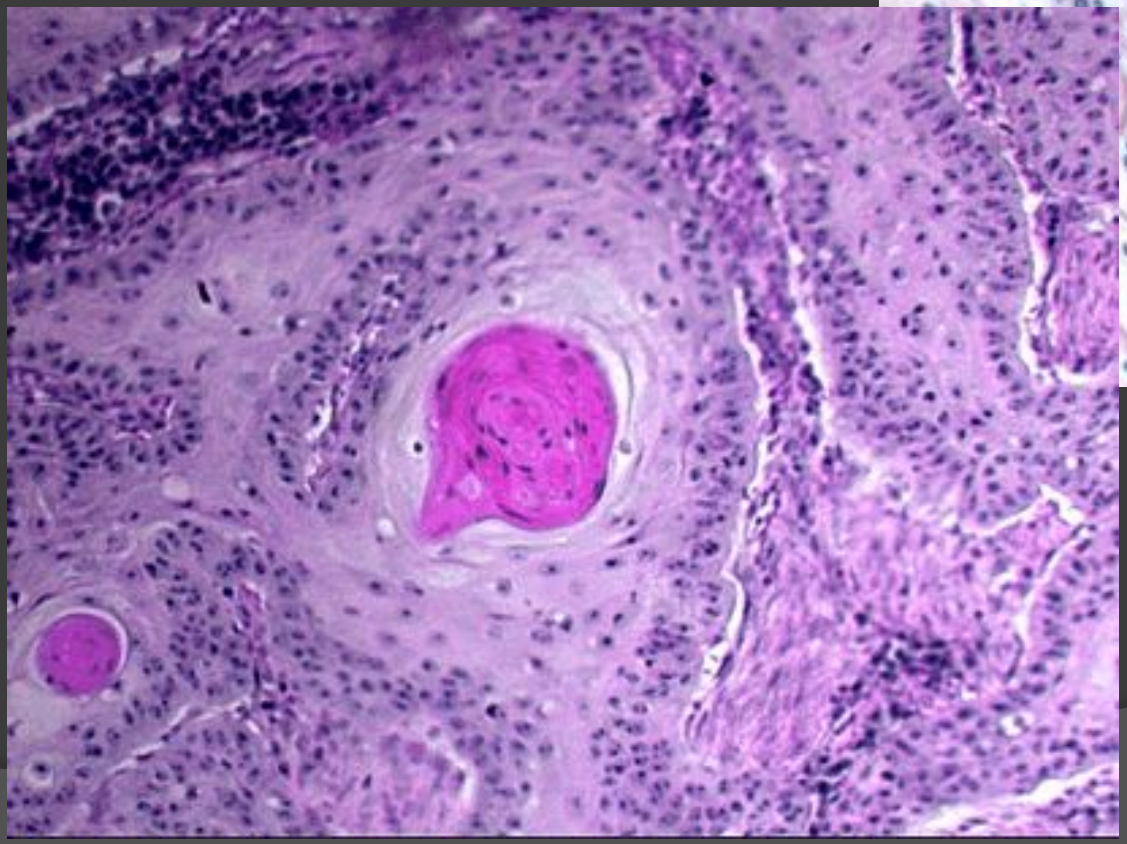
IV. By type of metabolic disorders

1. Protein;
2. Lipid (fat);
3. Carbohydrate;
4. Minerals.

INTRACELLULAR PROTEIN DEGENERATIONS

1. *Granular degeneration*
2. *Hyaline-drop degeneration*
3. *Hydropic degeneration*
4. *Keratinization degeneration*

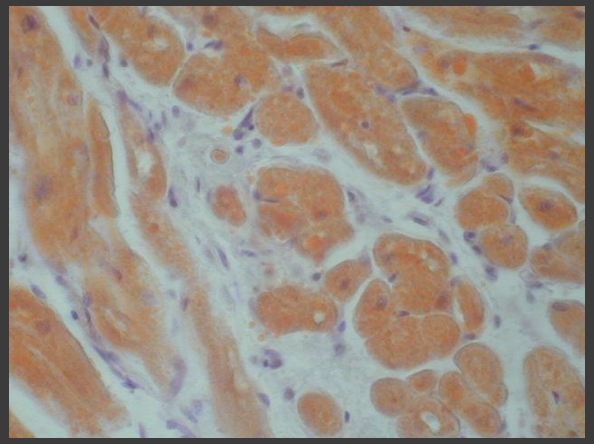


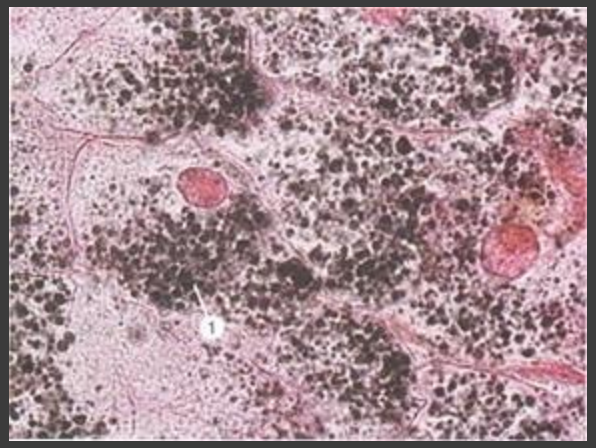


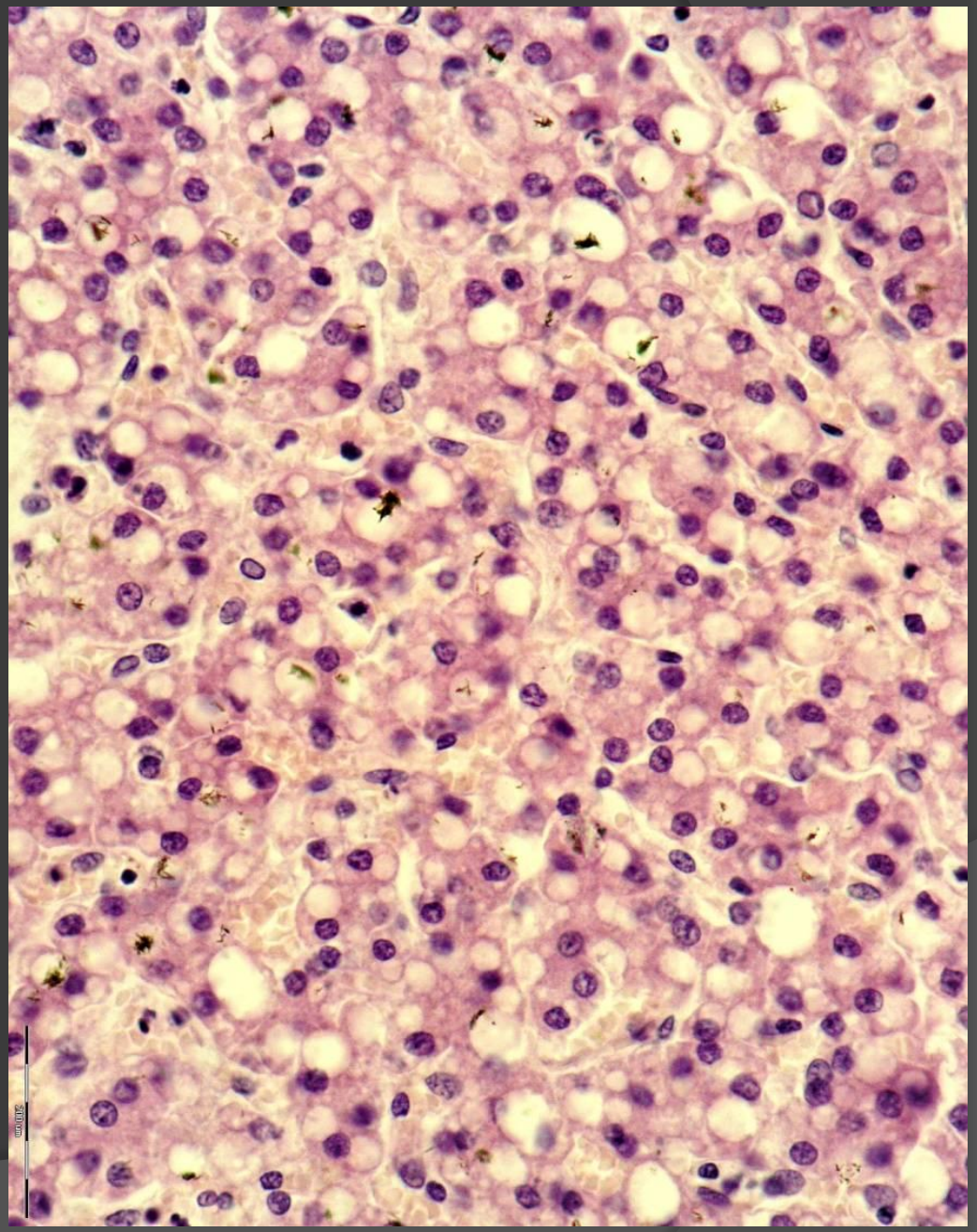
INTRACELLULAR FAT DEGENERATIONS

CAUSES:

1. *Hypoxia* (heart diseases, lungs and blood disorders)
2. Infections
3. Chronic intoxications







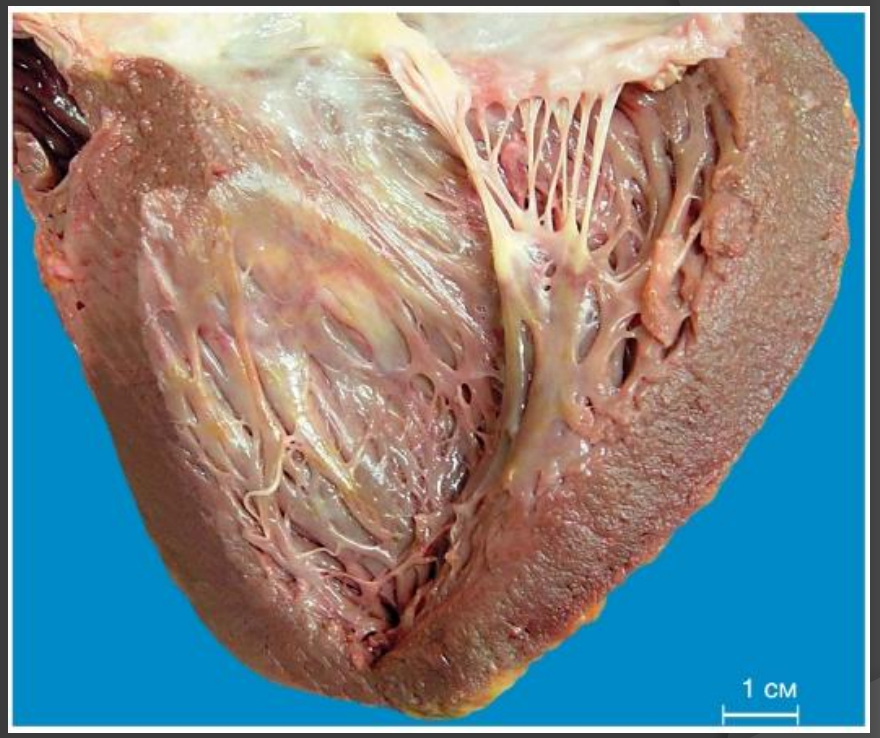
INTRACELLULAR FAT DEGENERATIONS

CAUSES:

1. *Hypoxia* (heart diseases, lungs and blood disorders)
2. Infections
3. Chronic intoxications

"Goose liver"

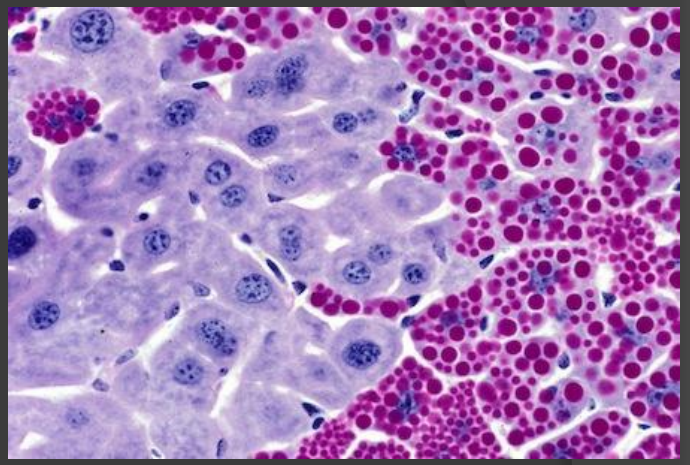
"Tiger's heart"

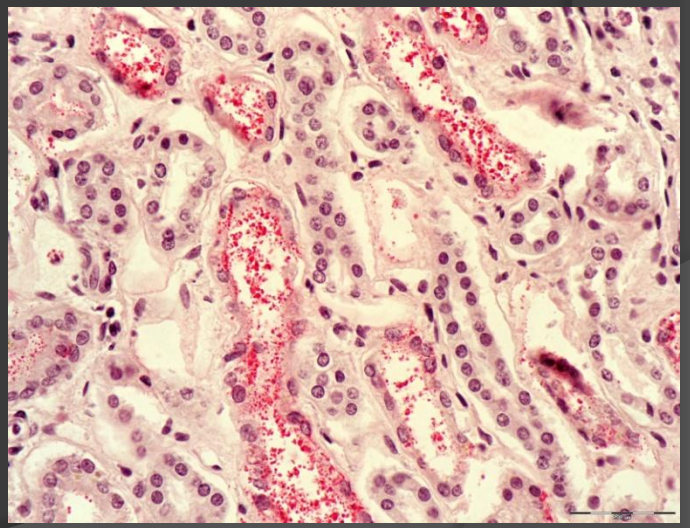


INTRACELLULAR CARBOHYDRATE DEGENERATIONS

- **Glycogen metabolic disorders**
- **Glycoproteins metabolic disorders**

Can be revealed using PAS-reaction
Glycogen is stained in red.

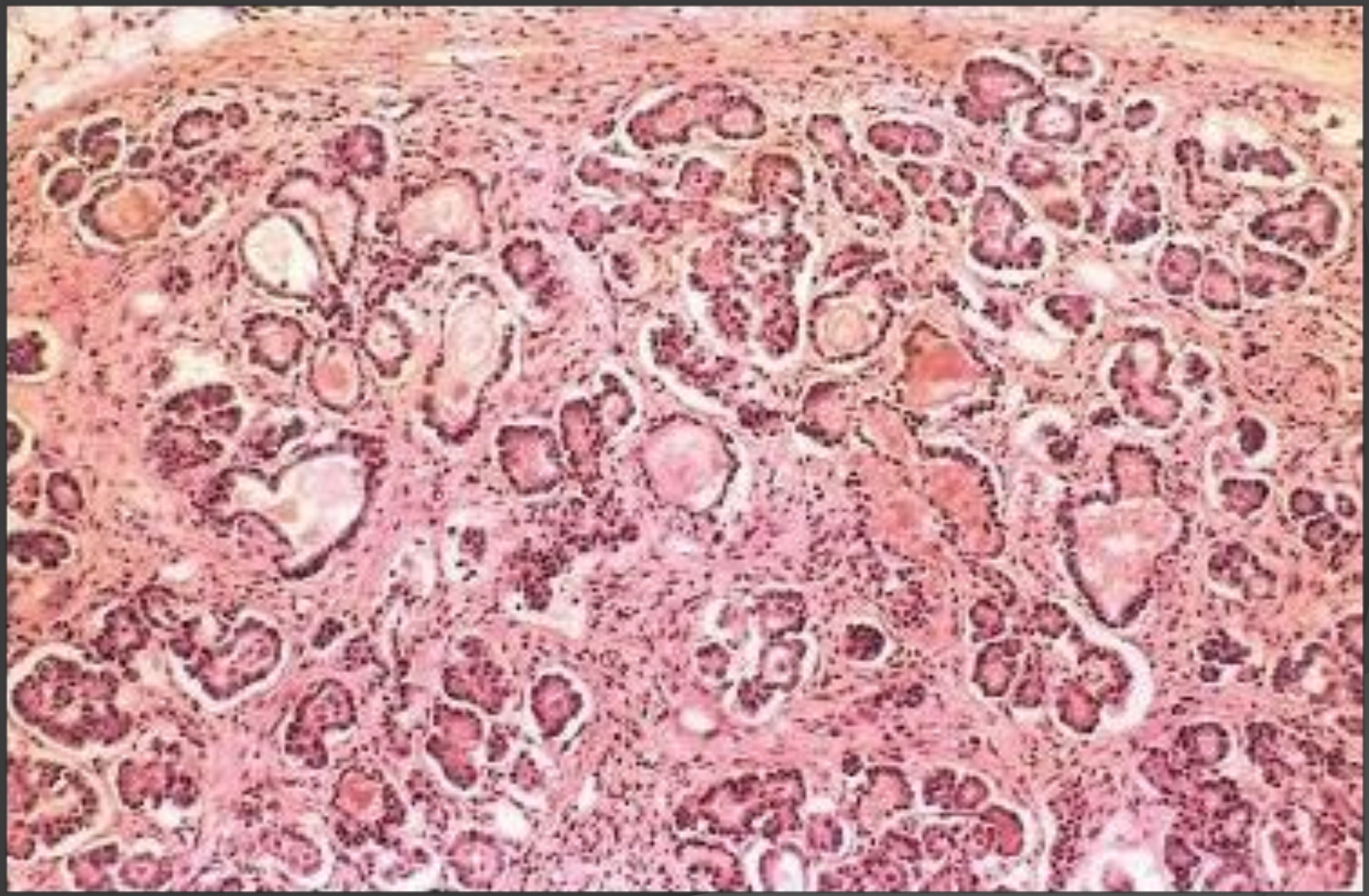




1. **Diabetes Mellitus**
2. **Glycogenosis.**
3. **Mucous degeneration of epithelium**

(catarral inflammation, mucoviscidosis [cystic fibrosis])

Cystic fibrosis of the pancreas



EXTRACELLULAR PROTEIN DEGENERATIONS

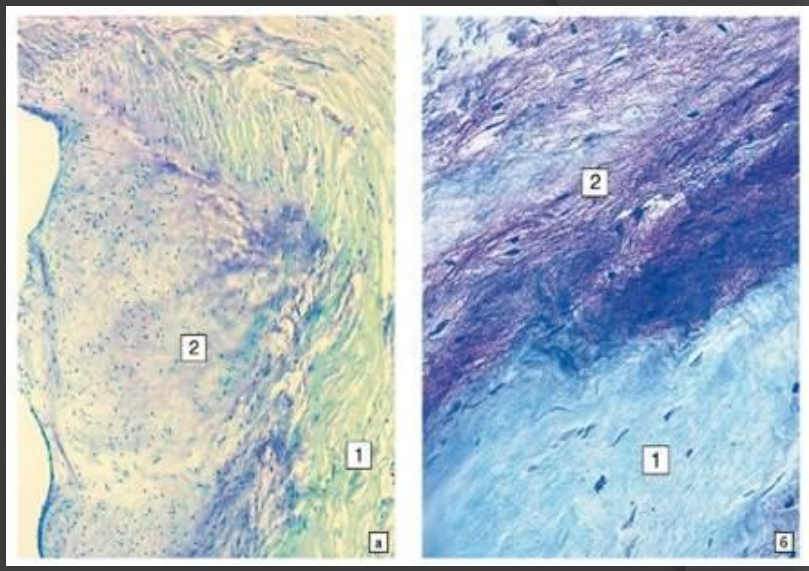
- Mucoid swelling
- Fibrinoid swelling
- Hyalinosis
- Amyloidosis

CAUSES:

-infectious diseases
-allergic diseases
- autoimmune diseases

Mucoid swelling

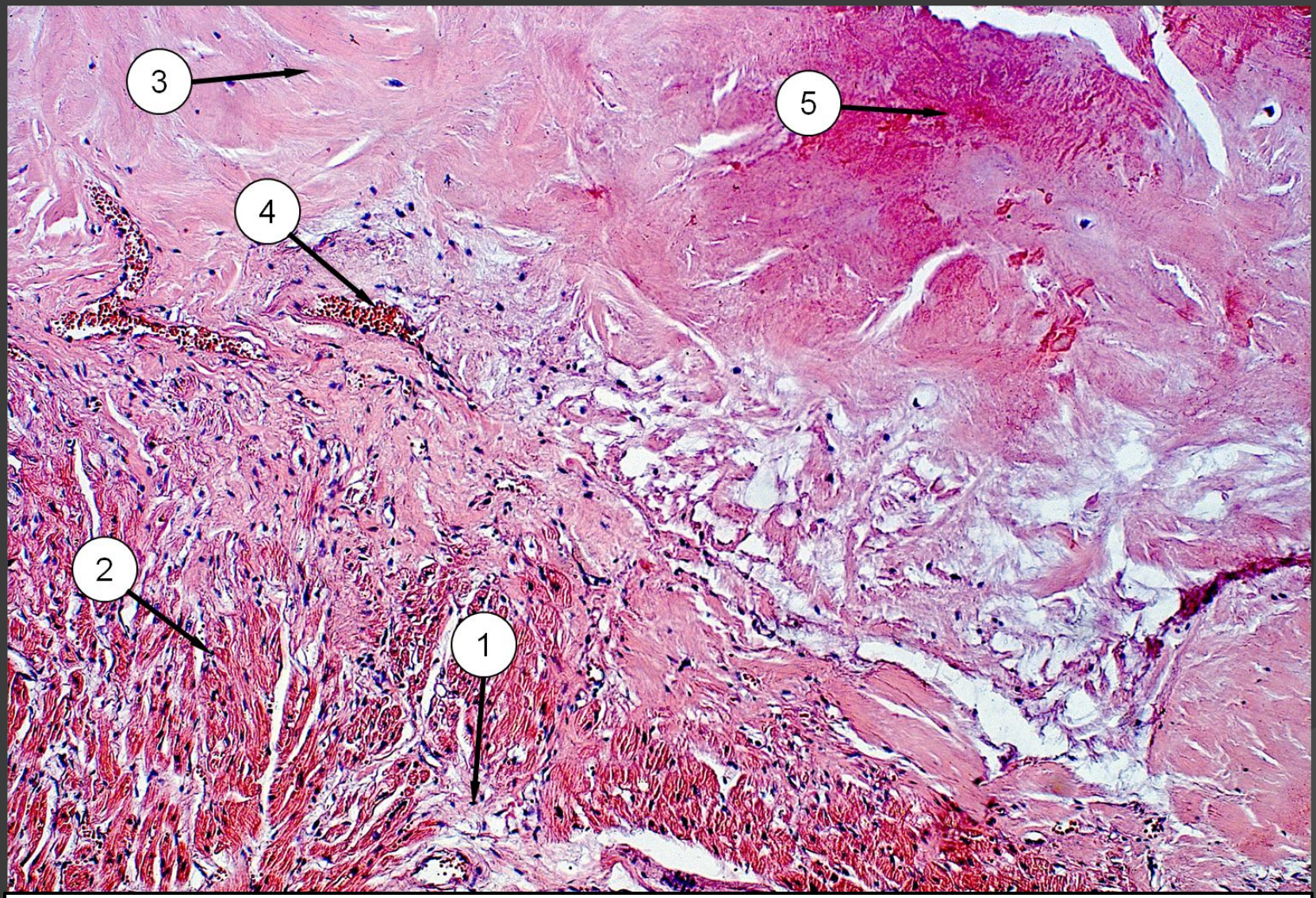
superficial and reversible desorganisation of the connective tissue. Accumulation of glycosaminoglycans by increasing the content of hyaluronic acid.



Fibrinoid swelling

deep and irreversible desorganisation of the connective tissue. Collagen breakdown, degradation of its material and fibers with increased vascular permeability and fibrinoid formation.

Fibrinoid changes



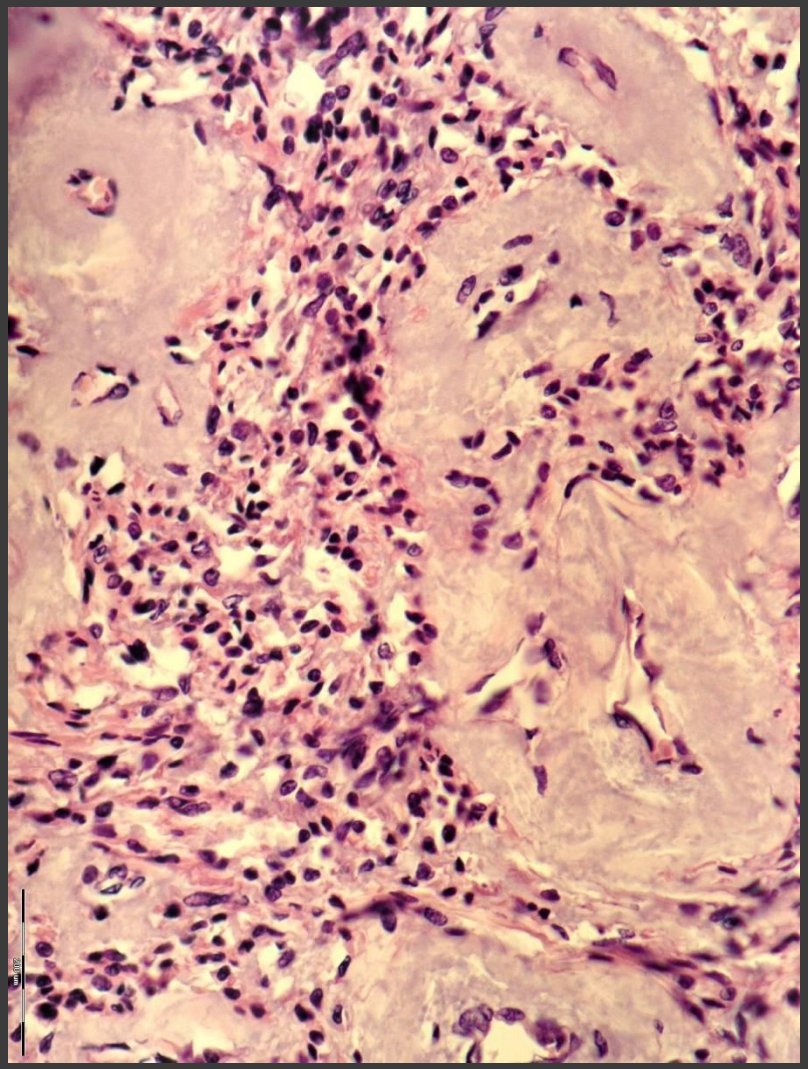
Hyalinosis

Degradation of connective tissue is accompanied by increased vascular permeability, degradation of collagen fibers and precipitation of plasma proteins.

Hyaline is the substance of complex chemical composition consisted of fibrin, immunoglobulins and proteins.

1. *Simple hyaline*
2. *Complex hyaline*
3. *Lipohyaline*

Hyalinosis

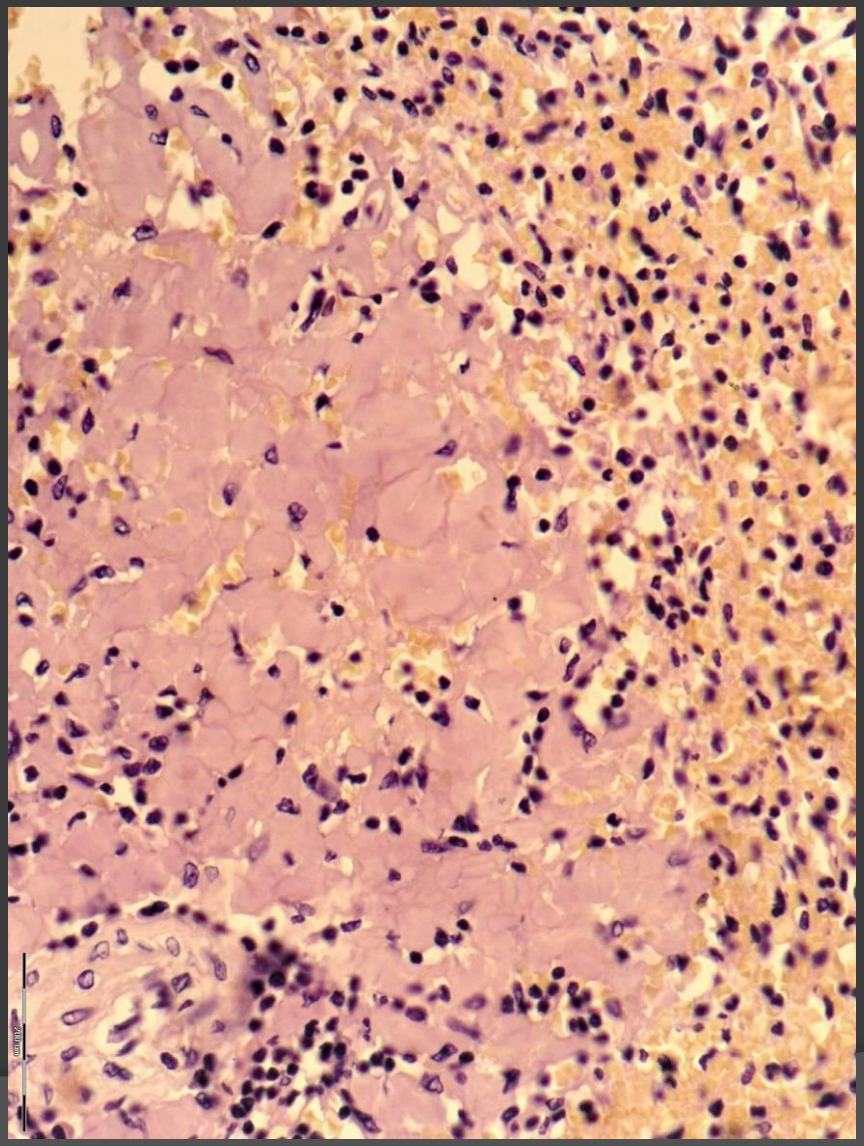


Hyalinosis of the spleen vessels in hypertension

Hyalinosis of the splenic capsule ("Glased spleen")

Amyloidosis

disease with the perverted synthesis of the substance called amyloid.



A mandatory condition for the development of secondary amyloidosis is a chronic inflammation.

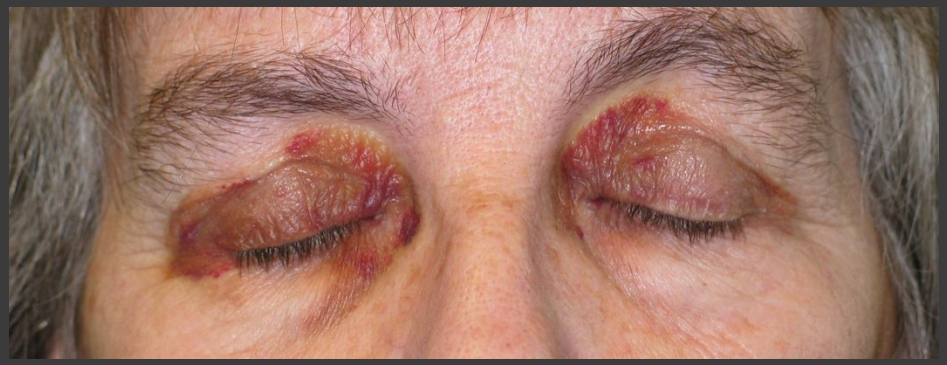
Most often amyloid deposits in liver, kidneys, spleen, adrenal glands (perireticular type)

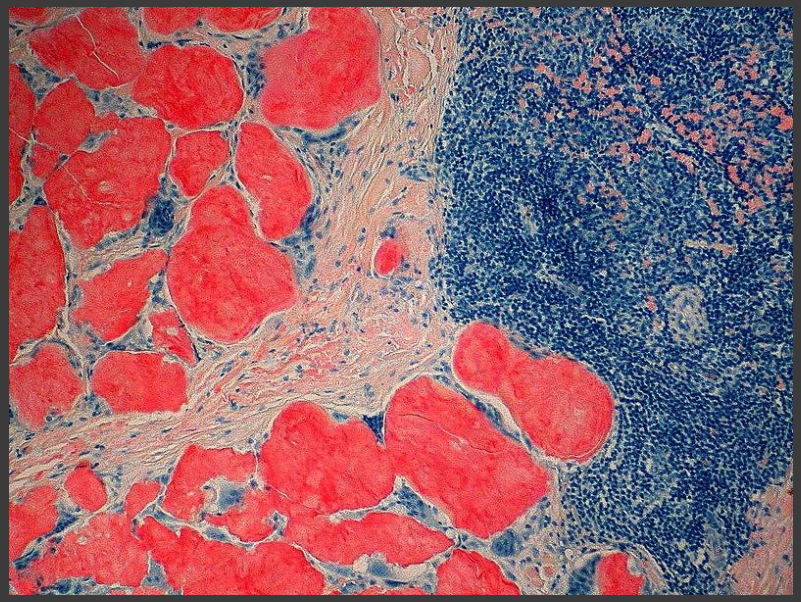
OR

In muscles, nervous system, heart (pericollagen type)

Amyloidosis

This term was proposed in 1853 by R. Virchow





Special staining methods on amyloid:

- Kongo red
- Iodine green
- Methyl violet

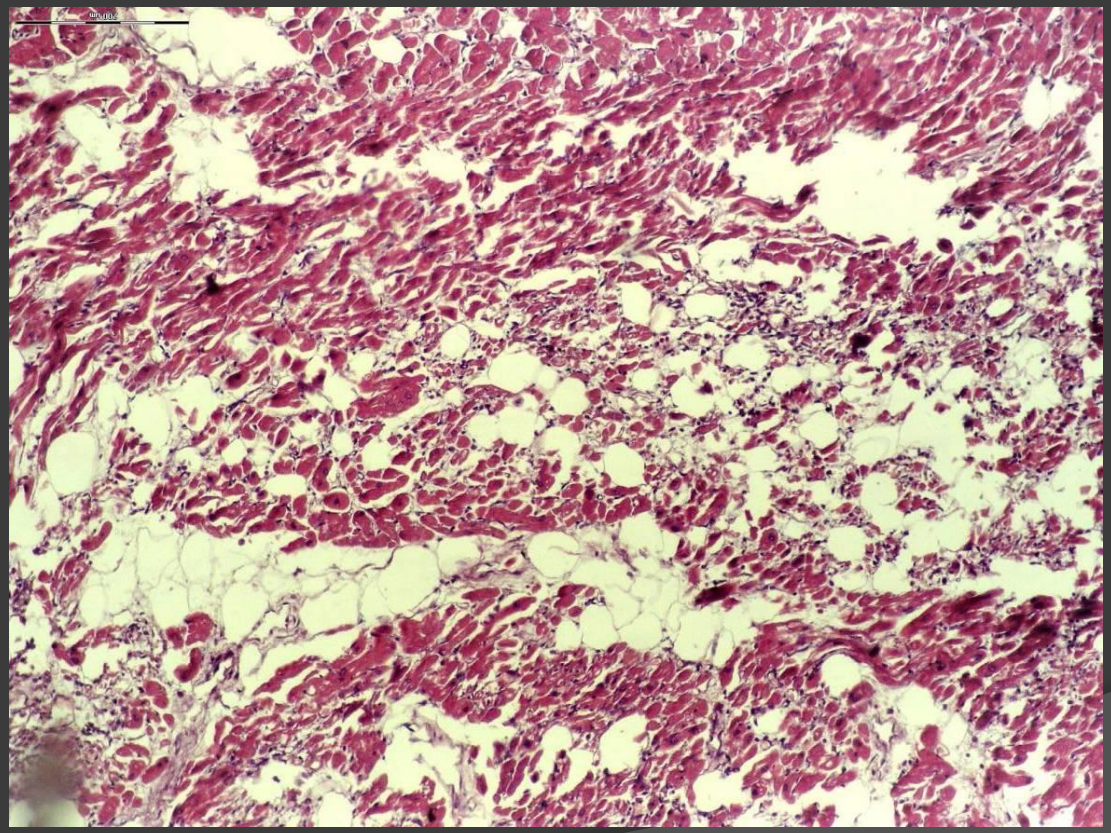
1. **Primary**
2. **Secondary**
3. **Idiopathic**
4. **Local tumor-associated**
5. **Senile**

EXTRACELLULAR LIPID DEGENERATIONS

General

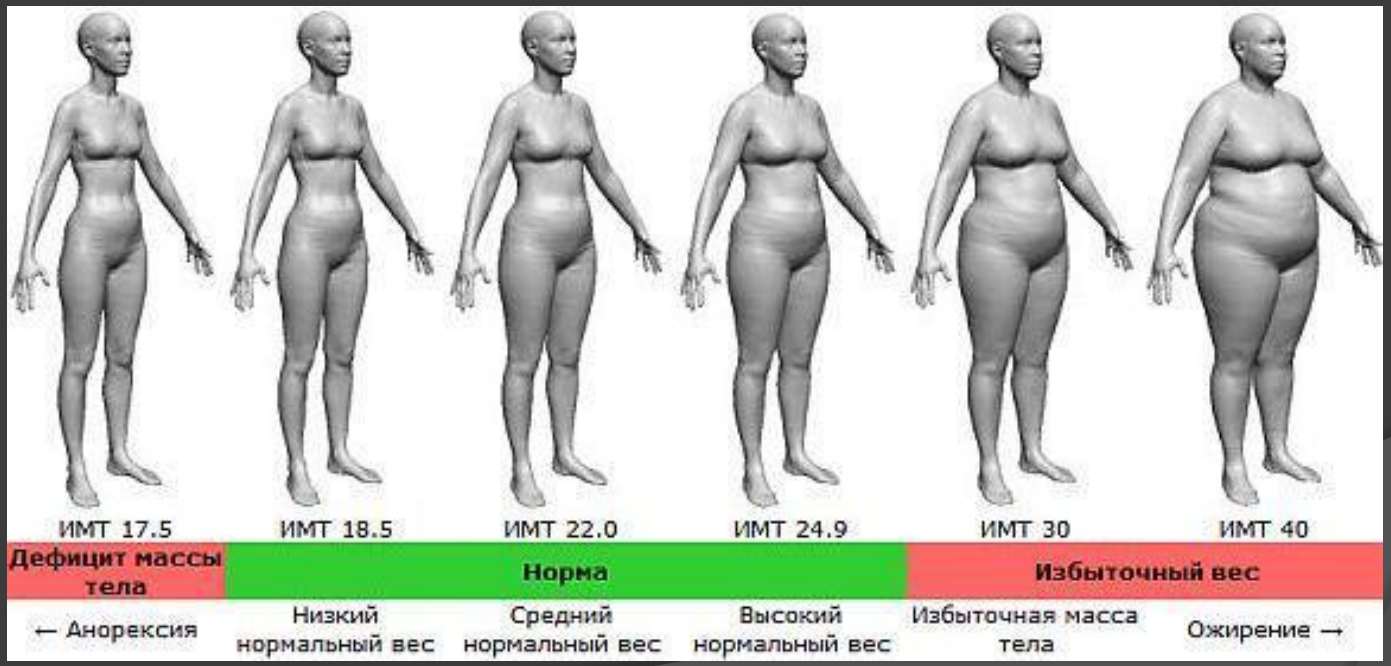
Local

Lipomathosis + deposition of lipid under the capsule of the organs



Degrees of obesity

I degree of obesity - overweight up to 30%;
II degree of obesity - overweight up to 50%;
III degree of obesity - overweight up to 99%;
IV degree of obesity - overweight over 100%;



Obesity

1. Hyperplastic
2. Hypertrophic
3. Mixed

General

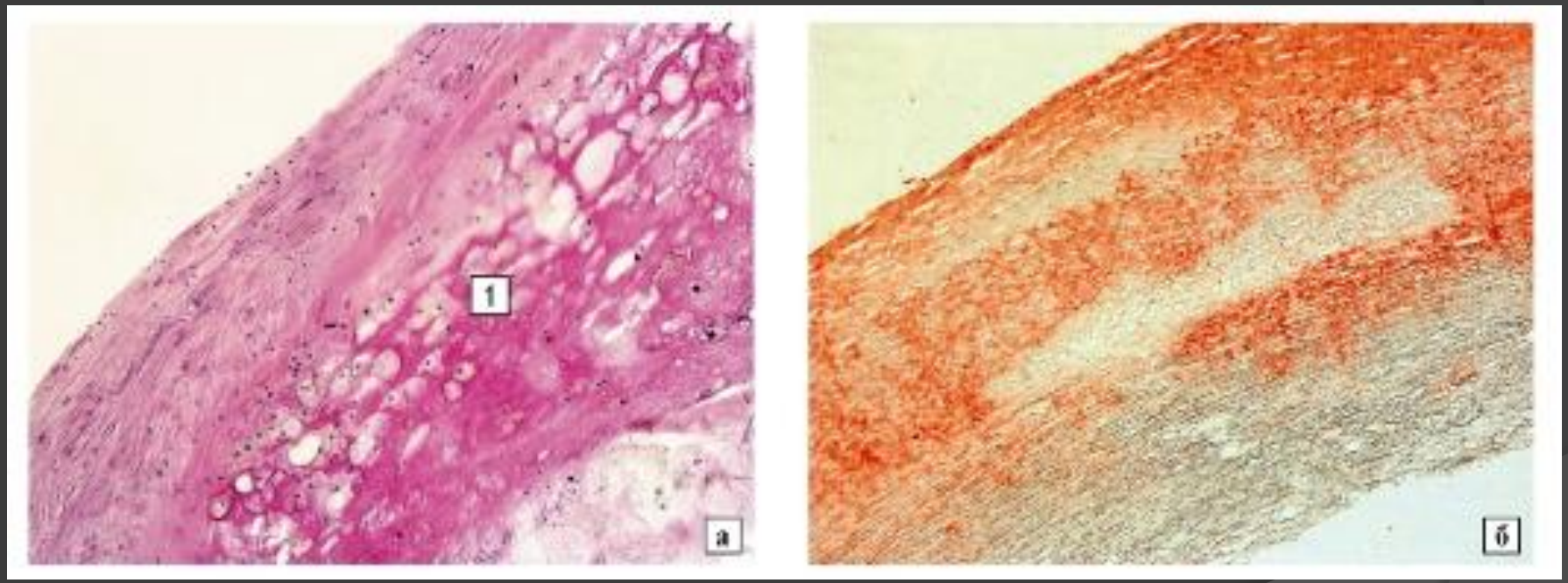
Symmetrical

Non symmetrical

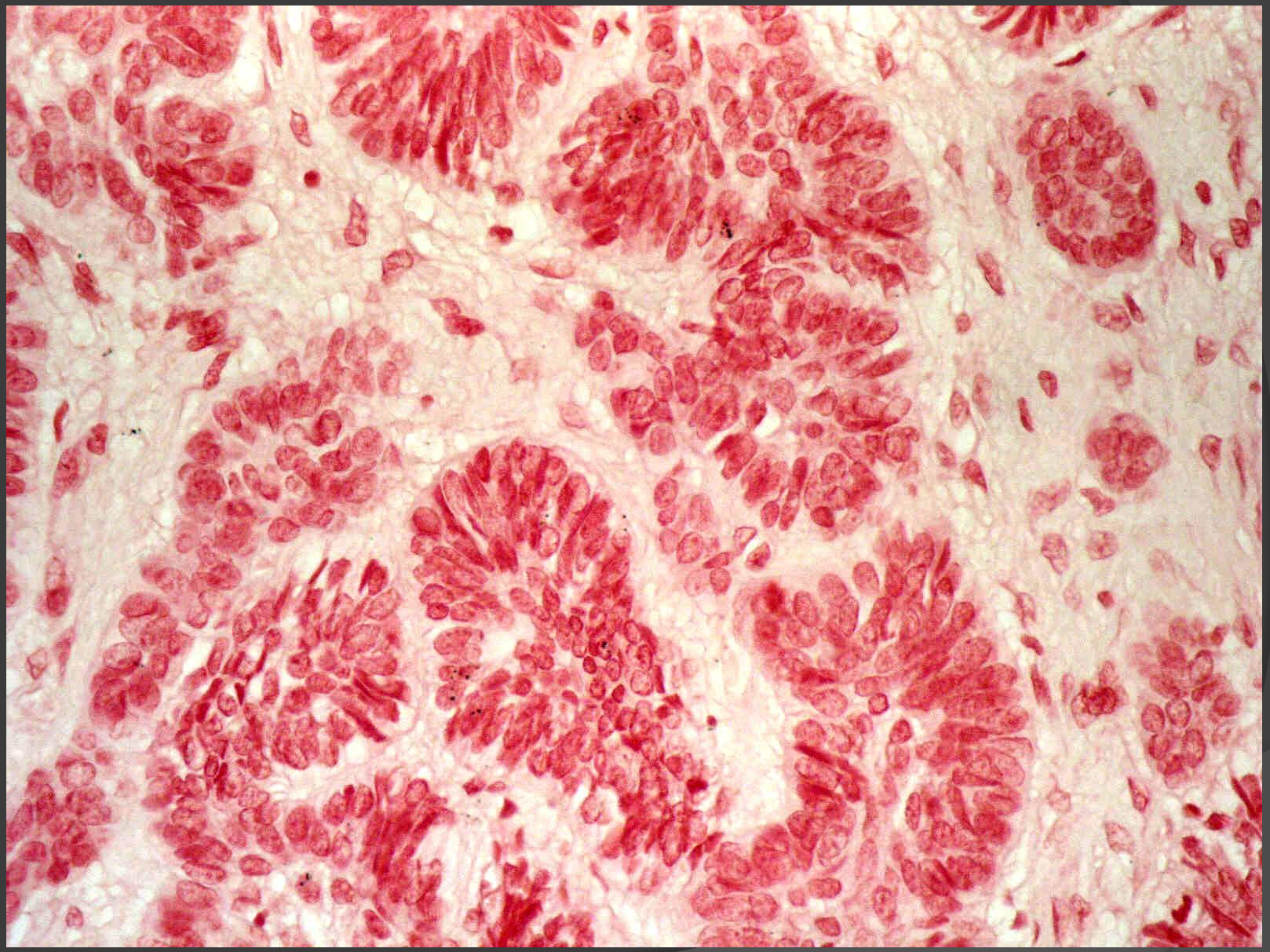
- Upper
- Middle
- Lower

ATHEROSCLEROSIS

chronic disease characterized by abnormalities in lipid and protein metabolism, which is manifested by the deposition of lipid complexes in the vascular wall



EXTRACELLULAR CARBOHYDRATE DEGENERATIONS



Pigments metabolism disorders

Chromoproteins - endogenous pigments

Hemoglobin - derivated	Protein - derivated (tyrosine)	Lipid - derivated
1. Ferritin 2. Hemosiderin 3. Bilirubin 4. Hematin 5. Hematoidin 6. Porfirin	1. Melanin 2. Adrenochrom 3. Pigment of the enterochromaffine cells	1. Lipofuscin 2. Lipochrom 3. Ceroid 4. Pigment of vitamin E deficiency

Pigments metabolism disorders

Hemoglobin - derivated

1. Ferritin
2. Hemosiderin
3. Bilirubin
4. Hematin
5. Hematoidin
6. Porfirin

Ferritin

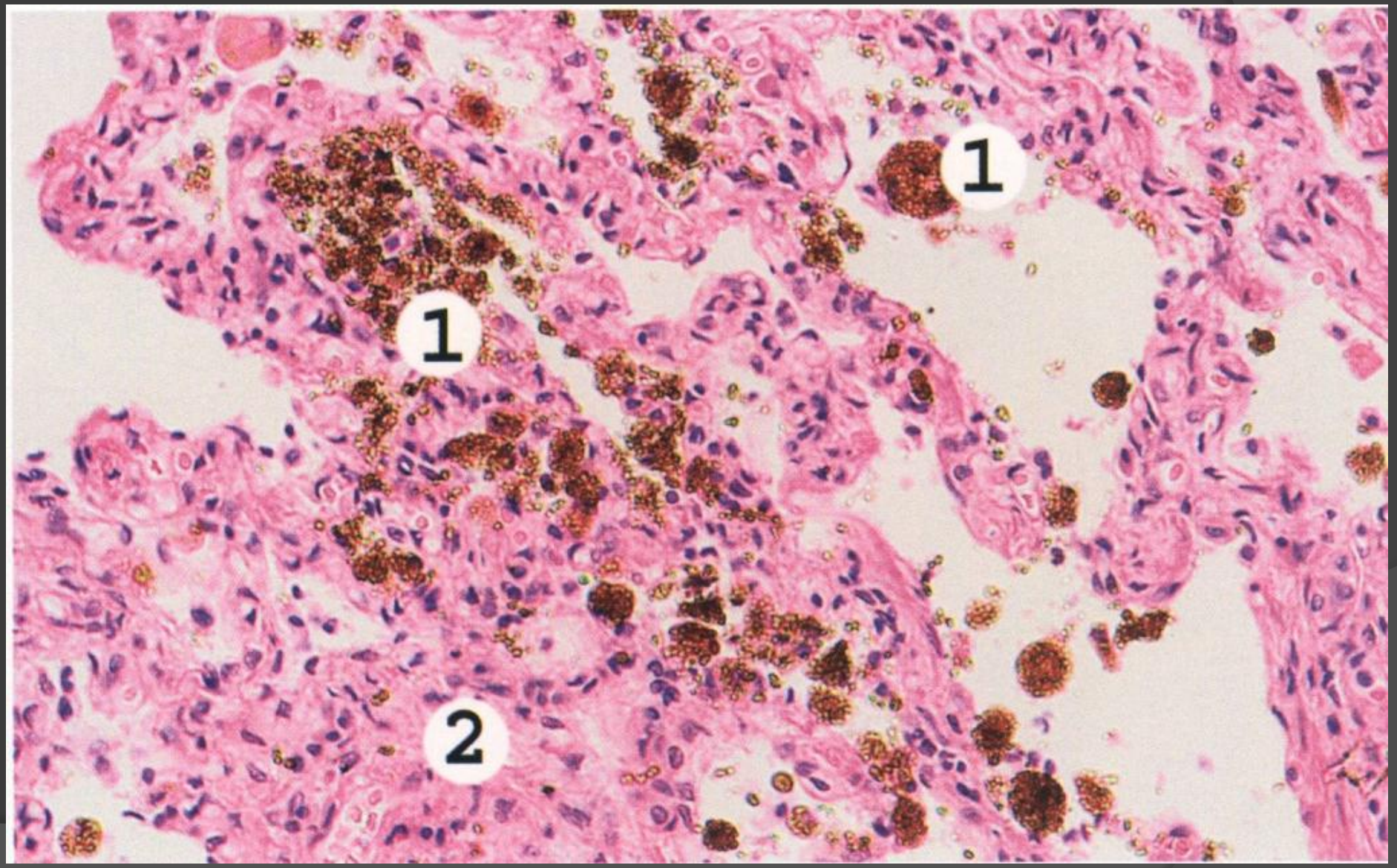
Hemosiderin is a polymer of ferritin. Hemosiderosis

Hematoidin — bright orange pigment lying freely in the central portions of hemorrhage.

Hematins: malaria, hydrochloric acid (hemin) and formalin.

Porfirins — hemoglobin tetrapyrrole ring without iron.

Brown induration of the lungs (hemosiderosis)



Jaundice

is an increase of bilirubin levels in blood, yellowing of the mucous membranes, sclera and skin

Hemolytic	Parenchymal	Mechanical
infectious diseases, hemolytic poisons, incompatible blood transfusion, blood system tumors	**damage of bilirubin capture by hepatocytes in liver diseases**	**violation of patency of the bile ducts**

Protein – derivated pigments (tyrosine)

1. MELANIN
2. Adrenochrome
3. Others

Thank you for attention!