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DYSTROPHIES

Laboratory manual of 3-ed year's students

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PARENCHYMATOUS DYSTROPHIES

Dystrophy (dys — disturbance, rophe — nutrition) — pathologic process, at basis of which lie the disturbances of tissue and cellular metabolism, which lead to the structural changes.

Classification of dystrophies:

1. Depending on the predominance of localization morphological changes:

- a) Parenchymatous.
- b) Strom-vascular.
- c) Mixed.

2. Depending form the disturbance of the exchange:

- a) Protein (disproteinoses).
- b) Fatty (lipidoses).
- c) Carbohydrate.
- d) Mineral.
- e) Mixed.

3. Depending on the prevalence of the process:

- a) local.
- b) General.

4. Depending on the origin:

- a) Acquired.
- b) Hereditary.

Morpho-Genetic mechanisms of dystrophies:

1. Infiltration.
2. Decomposition.
3. Distorted synthesis.
4. Transformation.

Decomposition — the disintegration of the endorsement — protein complexes of the structures of parenchymatous cell or protein — polysaccharide complexes of connective tissue.

Denaturing — damage of the native structure of protein under the action of any factors.

Coagulation (coagulation, thickening) — the passage of the colloidal solution into the state of ashes or gel. — softening, the melting of tissue.

Glycogenoses — hereditary carbohydrate dystrophy, at basis of which lie the disturbances of the exchange of glycogen.

Ichthyosis — (ichthyosis — fish scale) — the increased callusing of the significant sections of the skin. Leukoplacia — centers of callusing mucous membranes.

Tezaurismozy — (tesaurus — reserve) — the diseases, connected with the accumulation of metabolites in the cells and the tissue.

PARENCHYMATOUS DYSTROPHIES

URGENCY OF THE THEME:

The vital activity of any tissue is achieved as a result of a constant exchange of substances, in certain cases of the disturbance of metabolism the qualitative changes in the tissue or the organ are produced; the content of natural metabolites increases with this in the cell and interstitial substance or the substances of another chemical or physical composition appear. Such changes are called dystrophic. Dystrophy relates to the most ancient processes of phylogenies and accompanies many pathologic processes and diseases of children and adult.

Thus, dystrophic process is universal and is general pathological. It can be developed at different levels of the organization of the living: organ, tissue, cell and cellular ultra structures. The variety of reasons (alimentary, infectious and toxic, neuroendocrine disorders, defects of development of different systems) disrupts the regulator activity of central nervous and immune system, which changes the normal metabolism of proteins, fatty, carbohydrates and vitamins.

In the occupation it is proposed to study structural-pathogenetic changes in the organs and the tissue with disproteinosis, lipidoses and carbohydrate dystrophies; to dismantle the morpho-genetic aspects of the development of one or other form or another of parenchymatous dystrophies; to focus attention on the rare cases of the innate diseases of accumulation.

PURPOSES OF THE OCCUPATION

To study etiopathogenesis, structural-functional special features of parenchymatous dystrophies via the selection of the general characteristic of dystrophic process, their classification depending on the predominance of the disturbances of one or other form or another of exchange (protein, fatty, carbohydrate), on the localization of changes (cellular, extracellular, mixed) and on the prevalence of process (general and local).

Classification of parenchymatous dystrophies

1. Protein dystrophy

- a) Granular protein dystrophy.
- b) Hyaline-droplet dystrophy.
- c) Hydropic protein dystrophy.
- d) Horny dystrophy

2. Fatty dystrophy

3. Carbohydrate dystrophy

TASKS

1. To give a general characteristic of dystrophies.
2. Definition and classification of parenchymatous dystrophies.

3. Differentiation of parenchymatous (protein, fatty, carbohydrate) dystrophies on the basis of their macroscopic, microscopic and ultra structural characteristic.

4. To explain the mechanisms of the development of parenchymatous dystrophies in different organs under the effect of different reasons.

5. To estimate the functional value of parenchymatous dystrophies and their outcomes.

6. To know the relation between parenchymatous dystrophies and the development of diseases?

THE BASIC TRAINING QUESTIONS

1. Definition and classification of dystrophies.

2. Forms of protein dystrophies.

3. Morpho-genetic mechanisms of the development of dystrophies.

4. Morphological characteristic of granular, hyaline-drop, hydropic, fatty and horny dystrophies; reason and outcomes.

5. Etiology of the disturbances of the exchange of lipids, adipose dystrophy.

6. Mechanisms fatty dystrophy.

7. Morphological characteristic of parenchymatous lipidoses, the methods of development, outcomes.

ADDITIONAL MATERIALS ON THE THEME

MACRO-PREPARATIONS:

1. Turbid swelling of kidneys.

2. Tiger heart.

3. Fatty dystrophy of the liver.

MICROPREPARATIONS:

SLIDES:

1. The «Goose» liver.

2. Hyperkeratosis of the skin.

Parenchymatous dystrophies

The parenchymatous dystrophies of change as a result of the disturbances of the exchange of metabolites they appear predominantly in the highly specialized in functional of parenchymatous organs — heart, liver and kidneys.

Protein parenchymatous dystrophies

They are accompanied by appearance in the cytoplasm of the starts of protein nature and it is morphologically granular, hyaline-drop, hydropic and horny dystrophy. With the granular dystrophy the organs outwardly appear by those swollen, in the section they take the dim or turbid form («dim or turbid swelling»). Microscopically in the cytoplasm of cells small protein granules are determined. During the elimination of reason let us turn process; the passage into the hyaline-drop, hydropic dystrophy with the subsequent loss of cell is

possible during the continuation of the action of causal factor. The value of granular dystrophy is small; in the overwhelming majority weakening the function of the struck organ occurs.

Macro-preparation the «turbid swelling of kidneys». Kidneys are increased, flabby consistency, in the section they take the dim or turbid form, crust substance protrudes beyond the limits of connective-tissue capsule.

Micro-preparation «granular dystrophy of the epithelium of the convoluted tubules of kidneys» (stain H&E). The balls of kidneys are not changed. Cytoplasm of the epithelial cells of the convoluted tubules swollen, the opening of ducts is narrowed, the boundary between the cells illegible, nuclei are painted in the pale blue color. The protein small starts of pink color are revealed under a great increase in the cytoplasm of cells.

Macroscopically the organs do not change with the hyaline-drop dystrophy. Microscopically in the cytoplasm of cell appear the major drops of protein, hyaline- drop dystrophy leads to the loss of cell.

Macroscopically the organs do not change with the hydropic dystrophy. Microscopically in the cytoplasm of cell the vacuoles, filled with liquid, appear. Hydropic dystrophy can be completed by the (focal necrosis) or by the loss of cell (total necrosis).

Horny dystrophy is manifested in the form of the increased callusing (hyperkeratosis) or callusing mucous membranes (leukoplacia).

Fatty parenchymatous dystrophy (Lipidosos)

They are characterized by the disturbance of the exchange of cytoplasmic fat. They are morphologically manifested by the accumulation of the drops of fat in the cytoplasm of cells. For the development of lipids they are used painting with sudan dye III and IV, and also adapts scarlet and osmic acid. During the stain H&E in the histological preparations on the spot of the dissolved drops of the fat (fat it is dissolved in alcohols, xylene and so forth.) transparent vacuoles are visible. Most frequently adipose dystrophy is developed in the liver, the myocardium and the kidneys. Outcome is caused by gravity of the process: far visited disturbances lead to the loss of cells and tissues, with the associated reduction in the function of organ.

Macro-preparation «tiger heart». Heart is increased in the sizes, its chambers are dilated, the myocardium of flabby consistency, the endocardium and papillary muscles with the yellowish-white striation.

Macro-preparation «fatty dystrophy of the liver». Organ has the flabby consistency, it is increased in the sizes, in the section — yellow-brown or ocherous — yellow color (the «goose» liver).

Micro-preparation «fatty dystrophy of the liver» (stain H&E). On the periphery (predominantly) and in the centers of lobules are revealed the hepatic cells, in cytoplasm of which are contained the colorless vacuoles of different sizes. Vacuoles push aside nucleus to the periphery and the hepatocyte becomes similar to the fatty cell.

Micro-preparation «adipose dystrophy of the liver» (stain sudan III). In the cytoplasm of hepatic cells on the periphery of lobules an evidently large quantity of drops of fat, painted in the orange color.

Outcome is caused by gravity of the process: far visited disturbances lead to the loss of cells and tissue.

Value: the function of organ is reduced.

Micro-preparation «fatty dystrophy of myocardium» (stain sudan III). In the cytoplasm of the cardiomyocytes, located around venules and veins are the accumulations of the fat of yellow color; other muscle cells are free from the fatty esters. The transverse striation of muscle cells disappears; nucleus is wrinkled.

Carbohydrate parenchymatous dystrophy

In this type of dystrophy, the mainly distribute of glucose, leading to increase of glucose level — diabetic mellitus.

STROMAL-VASCULAR DYSTROPHIES (mesenchymal)

They are characterized by the disturbance of the exchange in the strom of organs or in the wall of blood vessels. They are also as parenchymatous dystrophy can be three types

Mesenchymal dystrophies are subdivided depending on the broken kind of metabolism on

1. Protin dystrophy

- a) Mucoidal swelling.
- b) Febrinoidal swelling.
- c) Hyalinosis.
- d) Amyloidosis.

2. Fatty dystrophy (lipedossis)

3. Carbohydrate dystrophy

THE BASIC EDUCATIONAL QUESTIONS

1. Classification of stromal and vascular dystrophies.
2. The reasons of development, morphogenesis, microscopic diagnostics and outcomes of mucoid swelling.
3. Fibrinoid swelling. Etiopathogenesis. Macro- and microscopic diagnostics. Outcomes.
4. Hyaline degeneration, hyalinosis. Classification. Types of hyaline. The pathoanatomical characteristic. Outcomes. Functional value.
5. Amyloidosis. Definition of amyloidosis. Classification.
6. Morphogenesis and pathogenesis of amyloidosis. Types of amyloid.
7. The reasons of amyloidosis.
8. Morphogenesis, macro- and microscopic characteristic of amyloidosis. Ways of amyloid deposition in an organism. Value of amyloidosis in clinic.

9. Histochemical reactions used for albuminous stromal and vascular dystrophies definition.

10. Infringement of neutral fats metabolism. Adiposity. Classification. The reasons and mechanisms of adiposity development. Outcomes.

11. Cachexy. The reasons. Kinds. The microscopic characteristic. Value for an organism.

12. Stromal and vascular carbohydrate dystrophies. Etiology. Morphological changes. Outcomes.

ADDITIONAL MATERIALS ON THE THEME

Macropreparations:

1. Sago spleen.
2. Hyalinosis of spleen capsule.

Micropreparations:

MATERIALS FOR STUDY

Terminology

1. Amyloid (amylum — starch) — glycoprotein the basic component of which is fibrous protein.

2. Atherosclerosis (athere — pappy mass, scleras — infiltration; hardening) — the disease described by cholesterol and its esters storage in the walls of large arteries with the next fibrous tissue overgrowth and calcification.

3. Hyalinosis (hyalos — light) — a kind of dystrophy at which light, translucent masses of protein are formed outside of cells.

4. Hhistian (histio — a tissue) — is structurally functional unit of a connective tissue.

5. Idiopathic (idios — original, own; pathos — suffering) — primary, arising for no apparent reason.

6. Cachexy (kakos — bad, hexis — a condition) — is a syndrome described like a strong exhaustion, physical weakness and the phenomenon of the general asthenia.

7. Interstitial (inter — between, stitere — to exist) — concerning to Interstitial spaces in tissues.

8. Keloid (kelis — a scar, kele — a tumour) — tumor like growth of a cicatricial connective tissue.

9. Fibrinoid — is the complex substance appearing in tissues at collagenic fibres destruction.

Practical part

Stromal and vascular (mesenchymal) dystrophies: Mesenchymal dystrophies develop as a result disturbances of metabolism and function in connective tissue and are discovered in stroma of organs and vessels walls.

We divide the following protein stromal and vascular disproteinoses: mucoid swelling, fibrinoid swelling, hyalinosis and amyloidosis. Quite often the first three versions of connective tissue disorganization which is observed at a great number of illnesses — rheumatic illnesses.

Mucoid swelling — is the superficial and convertible disorganization of a connective tissue. It is characterized by glycosaminoglycans accumulation in the basic substance of connective tissue which is caused by increased of stromal and vascular permeability and by exit of fine-dispersated plasma proteins (first of all albumins) into the interstitial space.

Mucoid swelling meets more often in arterioles' walls and arteries, valves of heart, glomeruli, parietal endocardium. Mucoid swelling is convertible process, however frequently passes in an irreversible condition — fibrinoid swelling.

Fibrinoid swelling. In its basis the destruction of connective tissue basic substance and fibers lays, accompanying with sudden increase of vascular permeability and coarse-dispersion plasma fibers exit, first of all fibrinogen, with the subsequent transformation into fibrin and then with special substance (glycoprotein) formation named as fibrinoid. Process is irreversible and comes to the end with fibrinoid necrosis, hyalinosis or sclerosis.

Hyalinosis. It is characterized by tissue accumulation of the translucent dense masses reminding hyaline cartilage. Arises in an outcome of fibrinoid swelling, plasmorrhagia, sclerosis and necrosis. The mechanism of hyaline formation develops of fibrous structures destruction and their following impregnation with fibrin and other plasma components (globulins, beta lipoproteins, immune complexes etc.)

Actually we divide hyalinosis of connective tissue and hyalinosis of vessels; these both kinds of hyalinosis can be systemic and local.

Known 3 types of hyaline:

- a) Common hyaline (arterial hypertension and atherosclerosis);
- b) Complex hyaline (with immune additives (at collagenoses).
- c) Lipohyaline (diabetes).

Macropreparation «spleen capsule hyalinosis». The spleen is enlarged, its capsule is thickened, of whitish color, translucent.

Micropreparation « Hyalinosis of connecting tissue in the scar ». There is a site with the sharp thickened collagenic fibres merging in homogeneous, eosinophilic mass In the skin scar. The amount of fibers is reduced, stromal cells are atrophic.

Amyloidosis. It is characterized by complex glycoprotein (amyloid) appearance in stroma of organs and vessels walls. Distinguish forms of amyloidosis depending on the causal factor: idiopathic (primary), hereditary (genetic), acquired (secondary), senile, local (tumor like).

Morphogenetically amyloidosis is distributed depending on the relation to connective tissue fibres: pericollagenically and perireticulary [parenchymatously].

Topographically we divide the following types of amyloidosis (depending from localization in organs): cardiopathic, nephropathic, neuropathic, hepatopathic, enteropathic, APUD-amyloidosis, systemic.

In general outcome of amyloidosis is unfavourable as in a result the atrophy and parenchymatous elements destruction develop in combining with sclerosis. Value of amyloidosis is determined by a degree of process development; expressed amyloidosis conducts to organs' function reduction.

Macropreparation «Spleen amyloidosis» (sago spleen). The spleen is increased in sizes, dense to the touch, its surface is smooth, a capsule is strained. On a cut section the surface is changed — on a background of dark — cherry pulp the increased follicles are seen which are looking like translucent grains reminding sago grains.

Micropreparation «kidneys amyloidosis» (stain Congo red). At painting with Congo red amyloid is painted in red color and it can be determined in mesangium and capillary loops of glomeruli, in interstitial vessels, in convoluted tubules basement membranes and reticular stroma. Additionally albuminous (hydropic, hyaline-drop) and fatty dystrophies could be found out In convoluted tubules epithelium.

Micropreparation «spleen amyloidosis». In spleen lymphatic follicles the deposition of homogeneous pink amyloid masses is marked. Cells of a pulp are atrophic, in a small amount are visible only in the center of follicles.

Stromal and vascular lipidoses are the structural changes connected to infringement of fat metabolism in fatty tissue and fatty depots, and also infringement of cholesterol and its ethers exchange in the walls of large arteries at an atherosclerosis.

Infringement of fat metabolism can have the general or local character, be accompanied by increase or reduction of fat quantity in fatty depots. The increase of fat quantity in fatty tissue leads to obesity.

Etiologically distinguish the following kinds of obesity:

- a) Alimentary;
- b) Cerebral;
- c) Endocrine;
- d) Hereditary.

Macroscopically distinguish:

- a) Symmetric type
- b) High level of obesity.
- c) Middle level obesity.
- d) Low level of obesity.

Depending on percent of body mass excess:

- I degree — 20–29 %;
- II degree — 30–49 %;
- III degree — 50–59 %.
- IV degree — more than 100 %.

Morphologically allocate the following types of obesity:

- a) Hypertrophic type;
- b) Hyperplastic type.

Adiposity of heart develops at the general adiposity of anyone genesis and results in its functional insufficiency.

Micropreparation « Adiposity of heart » (stain H&E). In specimens the foci of fatty tissue overgrowth are visible which are placed under the epicardium and in stroma, between cardiomyocytes. Cardiomyocytes are atrophic: the nuclei and cytoplasm are reduced. Places of brown pigment (lipofuscin) congestion in cardiomyocytes are marked.

Outcome: at the expressed adiposity a rupture of heart muscle with pericardial tamponade development can come.

Value: growth of fatty tissue under epicardium and in organs stroma leads to reduction of myocardial function that results in infringements of blood circulation.

Stromal and vascular carbohydrate dystrophies are connected to infringement of glycoproteins and glycosaminoglycans exchange. Structural changes at glycoproteins metabolism disorder result in mucin hypersecretion in the connective tissue. The changes caused by glycosaminoglycans accumulation lead to congenital enzymopathies (see topic «Tesaurismoses»).

Micropreparation «the mucoidal dystrophy of connective tissue». Connective tissue of specimens is swollen, mucus-like due to mucin distribution between fibres. Cells of connective tissue are dendritic, have star-shaped form.

THE MIXED DYSTROPHIES

URGENCY OF THE THEME

The mixed dystrophias are the structural changes, connected with the disturbance of the exchange of endogenous pigments and minerals, which can it was manifested by a number of the diseases: jaundice, gout, nephritic and bile- stones disease, the diseases, connected with the disturbance of the exchange of melanin, etc. Mastery of the materials of theme is the necessary prerequisite for the knowledge of laws governing the morphological reactions and their clinical manifestations with the development of pathologic processes and diseases. This is also necessary in the future professional activity of doctor for the clinical estimation of the conclusions of pathologist, for clinical diagnostics and treating the diseases, and also for the analysis of the sources of diagnostic errors in clinical practice.

PURPOSE OF THE OCCUPATION

Were studied etiopathogeneses, structural-functional special features of the mixed dystrophies via the selection of their general characteristic. It stopped at the classification of endogenous pigments (hemoglobinogenic, proteinogenic). Transferred the hemoglobinogenic pigments, which are formed in the human organism under the physiological conditions (hemosiderin, ferritin, bilirubin), also, with the pathologic states (hematoidine; hematin — malarial pigment, hydrochloric-acid hematin, formalin pigment; porphyrin), refined place and conditions for formation. It dismantled mechanisms and morphology of the disturbance of the exchange of the hemoglobinogenic pigments. Separately it stopped at hemosiderosis

(general and local), jaundices (mechanical, hemolytic, parenchymatous). From proteinogenic pigments (melanin, adrenochrome, pigment of the granules of enterochromaffin cells) in more detail follow it dismantled the disturbances of the exchange of melanin. It showed that they are manifested in the intensive formation of the pigment: extended (with Addison's disease) and local (pigment nevi, it spotted and melanoma), also, in its absence: extended (albinism) and local (vitiligo). With the disturbance of the lipidogenic pigments (lipofuscin, Steroid, hemofuscin, the pigment of the insufficiency of vitamin E) were studied the processes, which are accompanied by the excess accumulation of lipofuscin (brown atrophy of the myocardium, liver, innate lipofuscinoses). To students it follows were studied changes with the disturbance of the exchange of nucleoproteins (gout, the urate of infarction of kidneys in the newborns, urolithic disease). From the mineral dystrophias it dismantled the disturbances of calcium metabolism and the formation of stones. Were studied the classification of lime dystrophies on the localization and prevalences, by the mechanism of calcification (metastatical, dystrophic and metabolic). Follows were studied reason, localization and morphology of various forms of calcification. It became acquainted with different forms of stones, their localization, conditions for formation and complications of the presence of stones in the hollow organs.

TASKS

1. The determination of the mixed dystrophies, names and their forms.
2. The pigments, which relate to the groups hemoglobinogenic, proteinogenic and lipidogenic.
3. The forms of the disturbances of the exchange of chromoproteins (endogenous pigments) and explain the mechanisms of their development.
4. Distinguish the forms of the disturbances of the exchange of chromoproteins, lipoproteins, nucleoproteins on the basis of their macroscopic characteristic.
5. The functional disturbances of chromoproteins, lipoproteins, nucleoproteins.
6. Describe the forms of the disturbances of the exchange of minerals and microcells.
7. Describe the mechanisms of the development of the disturbances of the exchange of calcium (calcifications).
8. The macro- microscopic characteristic of calcinoses, disturbances of the exchange of minerals.
9. Mechanisms and structural special features of lithogenesis in different organs. The complications of the deposit of stones were distinguished.

ADDITIONAL MATERIALS ON THE THEME

Macro-preparations:

1. Stones of gall bladder. 2. Stones of kidney.
3. Mechanical jaundice in liver. 4. Lipofuscinoses of heart.
5. Brown induration of lung. 6. Metastases of melanoma into the liver.

Micro-preparations:

MATERIALS FOR THE CONTROL AFTER MASTERING OF THE THEME

Terminology

Albinism (albus — white) — weakening pigmentation, connected with the loss of the ability to manufacture melanin.

Hemosiderosis (haima — the blood, sideros — iron) — excessive formation and the deposit of product decomposed hemoglobin (hemosiderin) in the cloths.

Gout (podos — foot, agra — hunting «trap for the feet») — the disease, connected with the disturbance of the exchange of nucleoproteins, it is characterized by precipitation in the joints of salts of urate of sodium.

Siderophagia (sideros — iron, phagia — absorption) — the cells, which contain in the cytoplasm iron.

Practical part

The mixed dystrophies - these are the morphological manifestations of the disrupted metabolism, revealed both in parenchyma, and in the stroma of organs and tissues, that appear with the disturbance of the exchange of complex proteins — the endogenous pigments of chromoproteins, nucleoproteins, lipoproteins and minerals. Chromoproteins — painted substances of different chemical nature, which are synthesized in the organism. Endogenous pigmentations are usually connected with the excess accumulation of the pigments, which are formed within the standard, or with the accumulation of the pigments, which appear only under the conditions of pathology. Among the chromoproteins release the hemoglobinogenic, proteinogenic (tyrosinogenic) and lipidogenic pigments.

The hemoglobinogenic pigments are different derivatives of hemoglobin, which appear with synthesis or disintegration of erythrocytes. Within the standard are formed the ferritin, hemosiderin and bilirubin. Under the conditions of pathology are formed porphyrins, hematoidine and hematin.

Hemosiderin — aggregate of the molecules of ferritin, which is formed in the cell with the surplus of iron. With Perls reaction the granules of hemosiderin acquire the blue-green color (Berlin blue).

Hemosiderosis appears with the increased hemolysis — the destruction of erythrocytes. Can it was local and general. Local hemosiderosis appears with the extravascular hemolysis in the centers of hemorrhages and it decomposed erythrocytes. By an example of local hemosiderosis there can it was brown induration of lungs, which appears with the chronic venous stagnation in patients with the chronic diseases of heart (defects of heart, cardiosclerosis, etc.). Macro-preparation «brown induration of lungs» (hemosiderosis of lungs). Lungs are increased in the sizes, the dense consistency, reddish brown color, in the section with many reddish-brown starts and whitish layers.

Micro-preparation «brown induration of lung» (stain H&E and Perls reaction). During the stain H&E inside the cells (sideroblasts and siderophags) and

are extracellular visible the granules of brown pigment, which with Perls reaction acquire bluish-green color. Cells with the pigment are located in the stroma of lung, in the opening of bronchi and the cavities of alveoli. Inter-alveolar septa are thickened, sclerosed. The vessels of lung are full-blooded, extended.

Hemorrhages into the inter-alveolar septa are observed. Peribronchially and in the inter-alveolar septa are revealed connective-tissue layers.

Micro-preparation «hemorrhage into the brain» (stain H&E). Hemorrhage is represented by accumulation in the brain tissue of the hemolyzed and partially preserved erythrocytes.

On its periphery are visible the macrophages, loaded with brown pigment — hemosiderin. The substance of the brain in the center of hematoma is destroyed; it is edematous beyond its limits. In the region of hemorrhage the arterioles with the thickened hyalinized walls with the signs of plasmatorrhesis are visible. General Hemosiderosis appears with the intravascular hemolysis, which is developed with the diseases of the system of the blood, with the infectious diseases (malaria, sepsis, etc.), with the transfusions of incompatible blood and the rhesus- conflict. Slide Of «hemochromatosis of the liver».

Bilirubin — is formed in the cells with the decomposition of hemoglobin, gland it is not contained. In the hepatocytes it is occurred its conjugation — binding of bilirubin with the glucuronic acid, after which it is isolated with the bile. With the excess accumulation of bilirubin in the blood jaundice (it appears the icteric dyeing of the skin, scleras, and mucous membranes) develops. The following forms of jaundices are distinguished:

1. Nadpechenochnaya (hemolytic), appears with the intravascular hemolysis, in the blood increases the content of nonconjugated bilirubin.

2. Hepatic (parenchymatous) appears with the diseases of the liver (hepatites, hepatoses, cirrhoses), in the blood increases the content of conjugated bilirubin.

3. Podpechenochnaya (mechanical) appears during the obturation of bile tracts, in the blood increases the content of conjugated bilirubin. Macro-preparation the «liver with mechanical jaundice». The liver is increased in the sizes, its surface smooth- melted. In the section the tissue of the liver of greenish color. Are visible the sharply extended bilious ducts, filled with dark green bile. The fixing liquid is painted in the brown color. Micro-preparation the «liver with mechanical jaundice» (stain H&E In the preparation the evidently sharp expansion of bilious ducts and capillaries and overcrowding by their dark green bile. Epithelium of bilious ducts proliferating with the phenomena of vacuolization. It is located into several numbers.

Cytoplasm of hepatic cells is turbid, is grainy; in the hepatic cells it is observed the deposit of yellow-brown pigment (bilirubin). In the outcome biliar cirrhosis of the liver appears.

Value. It is occurred the disturbance of the functions of organ with the distortion of all forms of exchange of substances. Table «classification of jaundices».

Porphyryns — predecessors of hem, which have a structure of the locked tetrapyrrole rings, deprived of iron. The Porphyruses — State, with which it is, occurred the accumulation of the predecessors of porphyrin in the blood and the urine in connection with the disturbance of the synthesis of hem. The Porfiriuss can it was innate (genetically caused) and acquired (during the poisoning by lead, by barbiturates), avitaminoses (pellagra). Porphyryns are separated with the urine (porphirinuria), which the color of port is acquired, in the skin develop the erythema, dermatitis, pittings, depigmented scars, photophobia appears. Porphyryns are put aside in the spleen, they color the bones and teeth dark brown.

Hematoidine — pigment, in its structure close to bilirubin, is not contained gland. It is formed in the cells upon decay of hemoglobin in the sections of hemorrhages, with the loss of cells it is formed orange-red crystals in the necrotic detrite in the center of hematomas.

Hematins: malarial pigment (hemomelanin), hydrochloric-acid hematin (hemin), formalin pigment. Hemomelanin appears from hem with the actions of malarial plasmodium. Hydrochloric-acid hematin is formed under the influence on hemoglobin of the hydrochloric acid of gastric juice with erosions and stomach ulcers, coloring their bottom dark brown. Formalin pigment is formed in the tissues with the fixation by their acid formalin, has the form of brown grains and crystals, usually not connected with the elements of tissue, but the free at the opening venous vessels.

To the proteinogenic (tyrosinogenic) pigments has melanin, pigment of the granules of enterochromaffin cells, adrenochrome.

Melanin — pigment of reddish-brown-black color is synthesized in the specialized structures — melanosomes in the cells (melanocytes), from the tyrosine under the action of tyrosinase. The disturbance of the exchange of melanin is evinced by the development of the extended and local hyper-pigmentations and by hypo-pigmentation. The extended hyper-pigmentations develop with Addison's disease or with the pigment xeroderma. Local hyper-pigmentation are manifested in the form freckles, dark brown spots — lentigo, the benign melanocytic formations — nevi and malignant tumors — melanomas.

Macro-preparation the «metastases of melanoma in the liver». In the cloth of the liver the multiple formations of the rounded form of the dark brown and black color, which have clear outlines, are visible.

Micro-preparation «melanoma» (stain H&E). In the preparation of the retina of eye are visible the spindle-shaped lamellar and polymorphous cells of the melanocytes, which it is contained the granule of dark-brown pigment melanin, a large quantity of pigment lies extracellularly. The stroma of tumor is weakly developed. Extended hypopigmentoz — albinism is connected with the hereditary insufficiency of tyrosinase. Local hypo-pigmentations have name vitiligo.

To the lipidogenic pigments has lipofuscin, hemofustsin, pigment of the insufficiency of vitamin E, steroid. Lipofuscin — pigment of dark brown color,

the known also as pigment of aging, wear. It is formed via autophagia. Acquired lipofuscinoses is formed with the aging, hypoxia, scarcity in the food of proteins and vitamins, cachexia, increase in the functional activity of organs, endocrinopathies. Lipofuscin most frequently is accumulated in the cells of myocardium, liver, the skeletal muscles, which is accompanied by the development of the brown atrophy of organs. Macro-preparation Of «lipofuscinoses of heart» (brown atrophy of myocardium). Heart is reduced in the sizes. Lubricating cellulose under the epicardium he is, the channel of vessels is twisting. Heart muscle of brown color.

Micro-preparation «brown atrophy of the liver» (stain H&E). Hepatic cells and their nuclei are reduced in the sizes; spaces between the hepatic beams are extended. In the cytoplasm of the hepatocytes, especially the center of lobules, there are many small granules of brown pigment — lipofuscin. Some hereditary illnesses are accompanied by selective lipofuscin of individual organs.

The disturbance of the exchange of nucleoproteins is evinced by the development of gout, urolithic disease and urate of infarction.

Gout — chronic disease, which is characterized by periodic precipitation of urates in the small joints of arms and legs, in the tendons, periarticular cloth. The necrosis and inflammation with the development of connective tissue and the formation of the gouty lumps, which deform joints, appear as the answer. Micro-preparation «gouty lump» (stain H&E). In the sections of necrosis amorphous masses and crystals of urate of sodium are visible. Around the sections of necrosis — inflammatory infiltration with the gigantic polynuclear cells of the type of «Foreign bodies» and the development of connective tissue.

The disturbance of the exchange of calcium is manifested in the form calcinoses — precipitation of calcium from the dissoluble state and its deposit in the cells and the intercellular substance. On the basis of the mechanism of development they release metastatical, dystrophic and metabolic calcification.

1. Metastatical appears with the hyper-parathyroidism, the massive resorption of bone tissue (myeloma disease, the metastases of tumors in the bone, the plural breaks), with the overdose of vitamin D and the diseases of kidneys and is accompanied by hypercalcemia. Are damaged kidneys, myocardium, arteries, lungs, stomach? Slide «metastatical calcinosis of lung».

2. Dystrophic calcification appears locally in the tissues with the decreased vital activity and is revealed in the foci of chronic inflammation, the zone of necrosis, scar tissue, in the killed parasites, dead fruit with the extra-uterine pregnancy. The level of calcium in the blood is not changed. 3. metabolic — is connected with the instability of the buffer systems, which retain calcium in the dissolved state and by calcification — by increased sensitivity of cloths to calcium. Macro-preparation «stones in the kidneys». In renal pelvises the concrements of gray color with the uneven edges are visible. The cavities of wash-tubs, cups are sharply extended, the tissue of the kidney of istonchena, it is atrophic (hydro-nephrosis). Slide «nephrolithiasis». Macro-preparation the «stones in gall bladder». The cavity of gall bladder is filled with

many yellow-brown stones of average sizes. The wall of bubble is thickened, whitish color; it is soldered with the lower surface of the liver due to the inflammatory changes (associated cholecystitis).

Slides: the «plural stones of gall bladder», the «stone of gall bladder», the «stone of common bile duct».

Inspection questions

Parenchymatosis dystrophy

1. Definition and classification of dystrophies.
2. Forms of protein dystrophies.
3. Morpho-genetic mechanisms of the development of dystrophies.
4. Morphological characteristic of granular, hyaline- drop, hydropic, fatty and horny dystrophies; reason and outcomes.
5. Etiology of the disturbances of the exchange of lipids, adipose dystrophy.
6. Mechanisms fatty dystrophy.
7. Morphological characteristic of parenchymatous lipidoses, the methods of development, outcomes.

Mesenchymal dystrophy

8. Classification of stromal and vascular dystrophies.
9. The reasons of development, morphogenesis, microscopic diagnostics and outcomes of mucoidal swelling.
10. Fibrinoid swelling. Etiopathogenesis. Macro- and microscopic diagnostics. Outcomes.
11. Hyaline degeneration, hyalinosis. Classification. Types of hyaline. The path anatomical characteristic. Outcomes. Functional value.
12. Amyloidosis. Definition of amyloidosis. Classification.
13. Morphogenesis and pathogenesis of amyloidosis. Types of amyloid.
14. The reasons of amyloidosis.
15. Morphogenesis, macro- and microscopic characteristic of amyloidosis. Ways of amyloidal deposition in an organism. Value of amyloidosis in clinic.
16. Histochemical reactions used for albuminous stromal and vascular dystrophies definition
17. Infringement of neutral fats metabolism. Adiposity. Classification. The reasons and mechanisms of adiposity development. Outcomes.
18. Cachexy. The reasons. Kinds. The microscopic characteristic. Value for an organism.
19. Stromal and vascular carbohydrate dystrophies. Etiology. Morphological changes. Outcomes.

Mixed dystrophies

20. Mixed dystrophies. Determination.
21. Classification of endogenous pigments.

22. Hemoglobinogenic pigments.
23. Disturbance of the exchange of ferritin.
24. Disturbance of the exchange of hemosiderin. Mechanism of development and the morphology of general and local hemosiderosis.
25. Disturbance of the exchange of bilirubin. Jaundices. Forms of jaundices.
26. Etiology and the pathogenesis of different forms of jaundices.
27. Hematins, hematoidine, porphyrin. Reasons, the mechanism of their formation, structural special features.
28. Proteinogenic pigments.
29. Classification of the disturbances of the exchange of melanin.
30. Lipidogenic pigments. Reasons and pathogenesis, the morphology of the development of lipofuscinoses.
31. Disturbance of the exchange of nucleoproteins. Reasons for development, pathogenesis and morphological changes with the gout, the urolithic disease, the urate of infarction.
32. Disturbance of the exchange of minerals and microcells. Reasons, morphogenesis, path anatomy.
33. Disturbance of the exchange of calcium, classification of lime dystrophies.
34. Reasons and the signs of various forms of calcification, place of the deposit of lime metastases.
35. Lithogenesis. Forms of stones. Mechanisms of development.

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