LECTURE COMPLEX

Discipline: General Pathology
Discipline code: GP 2223
Name and code of the EP: 6B10115 - "Medicine"
Number of study hours/credits: 60hours/2 credits
Course and semester of study: 2 nd year; 5 semester
Lecture length: 4 hours

Shymkent, 2025

The lecture complex developed in accordance with the working program of the discipline (syllabus) "General Pathology" and discussed at the department meeting

Protocol No. 13 from " 26 " 06 2025 y.

Head of the Department _____ Sadykova A.Sh.

Lecture No 1

1. Topic: Management in general pathology

2. Objective: To characterize the goals, objectives, objects, methods and levels of pathological anatomy research; to give the concept of autopsy, biopsy, experiment as methods of studying pathological anatomy, to analyze the issues of their clinical significance in practical medicine.

3. Lecture abstracts

Subject, goals, objectives, objects, methods and levels of research of pathological anatomy. Cell and tissue death: pathology of the cell nucleus, mitosis and cytoplasm *Pathological anatomy is* a medical and biological science that studies the structural foundations of pathological processes, i.e. the material foundations of diseases. Pathological anatomy is an integral part of pathology and a link between theoretical and practical medicine, studies various aspects of disease. with this, is a scientific and applied discipline.

The tasks that modern pathological anatomy solves put it in a special position among medical disciplines. On the one hand, it is the theory of medicine, which, by revealing the material substrate of the disease, serves directly to clinical medicine, on the other hand, it is clinical morphology, which, while actively participating in the fate of the patient, at the same time serves the theory of medicine. Thus, pathological anatomy has a clinical and anatomical direction, therefore it is necessary for the study of other clinical disciplines.

The course of pathological anatomy includes 2 parts: general pathological anatomy and particular pathological anatomy. General pathological anatomy studies pathological processes that are manifestations of various pathological conditions or diseases. For example, cell pathology, dystrophies, necrosis, circulatory disorders, inflammation, regeneration, compensatory-adaptive and immunopathological processes, sclerosis and tumors. Particular pathological anatomy studies specific nosological forms or diseases.

The method of studying pathological anatomy is *the morphological method* - observation with the naked eye and with the help of optical devices.

Its varieties are: autopsy (autopsy, section, duction) - examination of the corpse at the macroscopic level in order to confirm and clarify the clinical diagnosis, identify a diagnostic error, establish the cause of death, the features of the course of the disease, identify the effectiveness of the use of medications, diagnostic manipulations, develop mortality and morbidity statistics; biopsy – intravital examination of organs and tissues for diagnostic purposes, examination of surgical material in order to confirm clinical diagnosis; experiment.

4 .Illustrative material: Electronic content.Topic№1

5. Literature: see Appendix No 1.(1.3.4.5.6.7)

6.Control questions (feedback)

What does pathological anatomy study?

1. The goals of pathological anatomy?

2. Methods, objects of research of pathological anatomy?

3. Pathology of the cell nucleus?

4. Cytoplasmic pathology?

Lecture No 2

1. Topic: Metabolic disorders

2. Objective: To give the concept of protein, fat and carbohydrate stromal-vascular dystrophies. Mixed, mineral dystrophies. Etiology, pathogenesis, morphogenesis, pathological anatomy.
mixed dystrophies, to explain the pathoanatomical signs, outcome and clinical significance of hemosiderosis, jaundice, hyper- and hypomelanosis; to analyze the issues of their clinical significance in practical medicine.

3. Lecture abstracts:

Hemosiderin is a polymer of ferritin, formed during the breakdown of heme, is a colloidal iron hydroxide bound to proteins, glycosaminoglycans and lipids of the cell. Cells that synthesize hemosiderin are called sideroblasts. Pigment in the form of granules is formed in the siderosomes of cells. Normally, hemosiderin is found in the reticular and endothelial cells of the spleen, liver, bone marrow, lymph nodes, in the intercellular

substance it is subjected to phagocytosis by siderophages. Hemosiderosis is an excessive formation of hemosiderin. In pathological conditions, hemosiderosis can be local and general. General hemosiderosis develops in pathological conditions associated with increased hemolysis. Causes of general hemosiderosis: 1. Anemia and hemoblastosis. 2. Poisoning with hemolytic poisons. 3. Infectious diseases (brucellosis, relapsing fever, malaria). 4. Transfusion of inocrucial blood and Rh conflict. Destroyed red blood cells, hemoglobin are used to build hemosiderin. Excessive formation of hemosiderin is accompanied by the activation of siderophages, which are the reticular and endothelial cells of the spleen, liver, bone marrow, lymph nodes, epithelial cells of the liver, liver, kidneys, lungs, sweat and salivary glands. However, siderophages do not have time to absorb hemosiderin, so it accumulates in the intercellular substance, the organs acquire a rusty-brown color.

Impaired formation and excretion of bilirubin is accompanied by jaundice. At the same time, the sclera, skin, mucous membranes and serous membranes of the body are stained yellow. There are three types of jaundice depending on the mechanism of its development: Hemolytic (prehepatic) jaundice**;** Parenchymal (hepatic) jaundice**;** Mechanical (subpenal) jaundice**.** Hemolytic (prehepatic) jaundice is associated with increased hemolysis. An increased amount of indirect bilirubin is captured by hepatocytes, but they are not able to utilize all the bilirubin, so its amount remains elevated. Hemolytic jaundice is observed in sepsis, malaria, relapsing fever, poisoning with hemolytic poisons, hemolytic disease of the newborn, transfusion of incompatible blood, hemoblastosis, systemic connective tissue diseases. Hemolytic jaundice is accompanied by a number of diseases associated with a defect in red blood cells (microspherocytosis, ovalocytosis), hemoglobinopathies (thalassemia, sickle cell anemia), vitamin B12 deficiency and hypoplastic anemia. Parenchymal (hepatic) jaundice is associated with damage to hepatocytes (in hepatitis, cirrhosis of the liver, intoxication). Affected hepatocytes do not capture bilirubin – its conjugation with glucuronic acid and excretion are disturbed. The content of indirect bilirubin in the blood increases. Parenchymal jaundice is also observed in hereditary intrahepatic disorders of bilirubin metabolism (enzymopathic hepatic jaundice). mechanical obstacles to the outflow of bile and its regurgitation. It occurs in the presence of stones in the bile ducts, cancer of the bile ducts, pancreatic head, Vater's nipple, bile duct hypoplasia, cancer metastases to the periportal lymph nodes and liver. Stagnation of bile in the liver leads to its outpouring into the parenchyma due to rupture of bile capillaries. As a result, foci of necrosis form in the liver tissue, which leads to their replacement by connective tissue and the formation of secondary biliary cirrhosis. At the same time, bile enters the blood, causing cholemia, general intoxication and related hemorrhagic syndrome (blood clotting disorders and multiple hemorrhages) and kidney damage. As a result, hepatic-renal failure develops.

Disorders of melanin metabolism can be widespread and local, congenital and acquired.

1. Melasma (widespread acquired hypermelanosis) is observed in Addison's disease, endocrine disorders (hypogonadism, hypopituitarism), vitamin deficiency, cachexia, intoxication with hydrocarbons pellagra, scurvy). In Addison's disease, tumor or tuberculous lesions of the adrenal medulla cause a lack of synthesis of the hormone adrenaline. Both substances - adrenaline and melanin are formed from tyrosine, as a result of which there is a compensatory conversion of tyrosine into melanin. Adrenaline deficiency stimulates the synthesis of ACTH, melanin in the melanosomes of melanocytes.

4 .Illustrative material: Electronic content.Topic№2

5. Literature:see Appendix No 1.(1.3.4.5.6.7)

6.Control questions (feedback)

1. Definition, classification of mixed dystrophies.
2. Specific coloration for the content of hemoglobinogenic pigments.
3. Hemosiderosis, types, characteristics, causes, pathogenesis, morphogenesis, macro- and microscopic signs, outcome.
4. Impaired bilirubin metabolism. Definition of jaundice, types of jaundice, etiology, pathogenesis, morphogenesis, macro- and microscopic signs, outcome.
5. Disorders of melanin metabolism. Determination of melasma and albinism, pathological anatomy.

Lecture No 3

1. Topic: Circulatory disorders

2. Objective: to explain the main causes, pathogenesis, pathological anatomy, outcomes, and clinical significance of arterial and venous plethora.

3. Lecture abstracts

: Arterial congestion (hyperemia) is an increase in blood supply to the organ and tissue due to increased arterial blood flow.

ν Arterial plethora can be: ν General, ν Local.

Venous plethora (venous stasis) is an increased blood supply to an organ or tissue due to a decrease or obstruction of blood outflow. Blood flow is not changed or decreased. ν Stagnation of venous blood leads to dilation of veins and capillaries, slowing down blood flow in them, and the development of hypoxia. ν Hypoxia is the main pathogenetic factor that determines changes in organs in venous plethora.

4 .Illustrative material: Electronic content.Topic№3

5. Literature: see Appendix No 1(1.3.4.5.6.7)

6.Control questions (feedback)

1. Classification of disorders of the fluid circulation system in the body.
2. Arterial hyperemia or congestion, definition, types of general and local arterial hyperemia.
3. Definition of venous hyperemia, types.
2. General venous plethora as a morphological manifestation of heart failure syndrome, definition of heart failure.
3. General acute venous plethora, causes, pathological anatomy (macro- and microscopic signs).
4. General chronic venous plethora, causes, pathological anatomy (macro- and microscopic signs).

Lecture No 4

1. Topic: Immunopathological processes. Morphology of immunogenesis disorders. Thymus pathology, Hypersensitivity reactions. Clinical significance

2. Objective: to explain the main causes, morphogenesis, pathological anatomy and clinical significance of immunopathological processes.

3. Lecture abstracts:

Thymus pathology. The thymus is the central organ of the immune system, as well as the endocrine gland. It is a link between the immune and endocrine systems. Its epithelial cells secrete specific hormones - thymopoietin, thymosin, which regulate T-lymphocytes in the thymus. Aplasia, hypoplasia and thymic dysplasia are congenital anomalies associated with underdevelopment and improper development of the organ. In aplasia, the thymus is absent at all. The production of thymic hormones is rarely reduced or absent. The organ is significantly reduced in size, there is no division into cortical and medulla, and the number of lymphocytes decreases. In pediatrics, immunodeficiency syndromes are of great importance - extreme manifestations of immunological deficiency. A distinction is made between primary immunodeficiency syndromes (IDS) associated with gene defects and are congenital, and secondary - associated with the disease (main) and treatment. Thymomomegaly: the normal structure is preserved, but the mass and volume of the thymic parenchyma increases, becomes higher than the age norm. Thymomomegaly can be congenital and acquired. Congenital - more often in children, in combination with other malformations of the nervous system, cardiovascular system, adrenal glands and gonads. Thymicolymfaticus status – reduced cellular immunity against the background of a decrease in the hormonal function of the thymus. Acquired thymomomegaly is observed in young adults with chronic adrenal insufficiency. Clinical manifestations are the same as in congenital immunodeficiencies. Thymic hyperplasia with lymphoid follicles is characteristic of autoimmune diseases. There is an expansion of intralobular spaces, B-lymphocytes, plasma cells accumulate in them, lymphoid follicles appear, as in lymph nodes. Most likely, this is secondary damage to the thymus in autoimmune diseases. Immediate hypersensitivity is morphologically manifested by acute immune inflammation: 1) develops rapidly; 2) alterative changes in the walls of blood vessels, the main substance, fibrous structures: - plasma impregnation, - mucoid and fibrinoid swelling, - fibrinoid necrosis, - formation of fibrinous-hemorrhagic exudate (neutrophils, fibrin, erythrocytes), 3) weak reprative reaction. Examples: Arthus phenomenon, BCG reaction, systemic lupus erythematosus, glomerulonephritis, croup pneumonia – immunoglobulins E play the main role. The object at which their actions are directed are target cells that undergo cytolysis (in tuberculosis, brucellosis, viral hepatitis, rheumatoid arthritis, periarteritis nodosa, etc.). Graft rejection reaction is the transplantation of an organ or tissue with an insufficient degree of compatibility. These organs or tissues are perceived by the body as "foreign" - antigen. An immune reaction begins to develop to this antigen - sensitization of the body, activation of T-killers: 1) infiltration of the transplant by lymphocytes and histiocytes; 2) proliferation of lymphocytes, histiocytes in the transplantation area; 3)

circulatory disorders and tissue edema; 4) the appearance of neutrophils and macrophages, killer T-cells, which lyse the cells of the transplant. Immunosuppressive drugs are used to reduce this reaction. Examples of autoimmune diseases: 1. Hoshimoto's thyroiditis is a true autoimmune disease. Autoantibodies to thyrocyte antigens and thyroglobulins appear. The thyroid gland is infiltrated by lymphocytes and plasma cells, the effector cells of the body act on the body's own structures of the thyroid gland, they are replaced by connective tissue, and lymphoid follicles are formed. 2. Systemic lupus erythematosus is a systemic autoimmune disease of the connective tissue with predominant damage to the skin, blood vessels and kidneys. 3. Dermatomyositis is a systemic autoimmune lesion of striated muscles, to a lesser extent smooth muscles and skin. 4. Scleroderma – autoimmunization leads to damage to the connective tissue and skin.

4 .Illustrative material: Electronic content.Topic№4

5. Literature see Appendix No 1.(1.3.4.5.6.7)

6.Control questions (feedback)

1. General characteristics of immunopathological processes, types.
2. Physiological (age-related) and accidental involution of the thymus, morphological characteristics.
7. Aplasia, hypoplasia and thymic dysplasia, morphological characteristics.
8. Thymomomegaly, clinical and morphological characteristics.
9. Thymic hyperplasia with lymphoid follicles, clinical and morphological characteristics.
10. Pathology of peripheral lymphoid tissue, causes, pathogenesis, morphological characteristics of hyperplasia of the spleen, lymph nodes.
11. Hereditary insufficiency of peripheral lymphoid tissue (spleen, lymph nodes), clinical and morphological characteristics.
12. Hypersensitivity reactions: mechanisms of development; hypersensitivity reactions of immediate, delayed type. Morphological characteristics. Examples.
13. Graft-versus-host disease, autoimmunization. Morphological characteristics. Examples.

"Appendix No1".

Reference citations:

1. Akhmetov , Zh. B. Pathologıialyq anatomy: okulyq [Pathological anatomy: okulyq]. - 4th bas.,óndelgenjánetolyqtyrylgan ; QR Densaýlyqsaqtaý min. oqý- ádisstemelik birlestiginde joǵary med.oqý oryndarynyń stýd. arnalǵan. - Moscow: "Litterra", 2016. - 792 bet p.-1-1--
2. Tusupbekova M. M. Klinicheskaya patomorphologiya: monografiya [Clinical pathomorphology: monograph]. - Almaty: Evero, 2016. - 184 p.-40--40-
3. Tusupbekova M. M. Morphological Atlas of General Pathological Processes: Textbook. - Almaty: Evero, 2016. - 164 p.-31--31-
4. Strukov, A. I. Pathologıialyq anatomy: okulyq /. - Moscow: GEOTAR - Media, 2015. - 984 bet. S-40-40--
5. Pathology. Ekitomdyq. 1 tom.:okulyq / Red. bass. M. A. Paltsev, Kaz. til. Oud. S. A. Apbasova. - Moscow: GEOTAR - Media, 2015. - 536 page : ill.-1-1--
6. Akhmetov Zh. B. Patologıialyq anatomy: okulyq [Pathological anatomy]. - ; QR densaýlyq saqtaý mınıstrligi. - Almaty: Evero, 2014. - 700 bet. S-200-200--
7. Esirkepov, M. M. Pathological Anatomy of the Genome: Textbook - Karaganda : IP "Publishing House AKNUR", 2013-40--40-

Further reading:

1. Povzun, S. A. Patologicheskaya anatomy v voprosakh i otvetakh [Pathological anatomy in questions and answers]. posobiye / S. A. Povzun. - 3rd ed., revised and supplemented; Rivers. SBEI HPO "First Mos. State Med. I. M. Sechenov University". - Moscow: GEOTAR - Media, 2016. - 176 p. --1--1-
2. Pathological anatomy. A guide to practical classes: a textbook / M-vo obraz. and Science of the Russian Federation. Rivers. I. M. Sechenov First Moscow State Medical University; Under. ed. by O. V. Zairatyants, L. B. Tarasova. - 2nd ed., ispr. Moscow: GEOTAR - Media, 2015. - 696 p. : ill.-1--1-
3. Pathologıialyq anatomy atlas: oku kuraly = Pathologicheskaya anatomy : atlas: ucheb. posobie - M. : GEOTAR - Media, 2014. - 1128 bet-1-1--

4. Strukov, A. I. Pathologıialyq anatomy: okulyq . - 5-bass., stereotype. - Moscow: GEOTAR-Media, 2013. - 984 bet. el. wholesale. CD-ROM-100-1--
5. Strukov, A. I. Pathologıialyq anatomy: okulyq - M. : GEOTAR-Media, 2013.-226-226--
6. Tusupbekova, M. M. Morphological Atlas of General Pathological Processes: Atlas. - Almaty: Evero, 2012.-10--10-
7. Tusupbekova M. M. Klinicheskaya patomorphologiya [Clinical pathomorphology]. - Almaty: Evero, 2012-10--10-
8. Kismanova, G. N. Jalpypathologıialyqprotsesternuskaýy: oqýquraly . - Almaty : Evero, 2010-123-123--
9. Strukov, A. I. Pathologıialyq anatomy (Jalpy bólimi): oqýlyq . - 2 basylym. – Aktobe : LLP "M. Style", 2010-200-200--
10. Strukov, A. I. Pathologıialyq anatomy (Jeke aurular bólimi. II bólim. 1st article) :oqýlyq. – Aktobe : JShS "M Style", 2010-200-200--
11. Strukov, A. I. Pathologıialyq anatomy (Jeke aurula rbólimi. II bólim 2-shi kitap) :oqýlyq . - 2 basylym. – Aktobe : LLP "M. Style", 2010-200-200--
12. Akhmetov Zh. B. Pathologıialyq anatomy [Pathological anatomy]. 1-shi kitap: oqý kýraly. - 2-bass.,.- Almaty, 2009-80-80--
13. Akhmetov Zh. B. Pathologiyalyk anatomy [Pathological anatomy]. 2-shi kitap :oqý kýraly. - 2nd bass., qaita óńd.. - Almaty, 2009.-3-3--
14. Paltsev M. A. Atlas po pathologicheskoy anatomy: uchebnik.-3-e izd. Stereo type.- M., 2007 -34--34-

Electronic literature:

1. Tusupbekova M. M. Morphologicheskiy atlas obshchepatologicheskikh protsessov [Morphological atlas of general pathological processes] : textbook. -Electron. textual dan. (1.03GB). - Almaty: Epigraph, 2016. --25--25-
2. Mitrofanenko, V. P. Patologıianyńnegizderi [Pathology]. School and college of the College. oqulyq = Fundamentals of pathology : / - Electron. textual dan. (154Mb). - Moscow: GEOTAR - Media, 2015. - 568b. p.-15-15--
3. Pathological anatomy [Pathological anatomy] : national hand. / Editor-in-Chief M. A. Paltsev. - Moscow: GEOTAR - Media, 2013. - 1264 p-16--16-
4. Strukov, A. I. Patologicheskaya anatomy [Pathological anatomy] : textbook - M. : GEOTAR - Media, 2013-20--20-
5. Strukov, A. I. Patologicheskaya anatomy [Pathological anatomy] : textbook . - 5th ed. Erased. - Electron. textual dan. (51.9 MB). - Moscow: Izd. group "GEOTAR-Media", 2011. - . el. wholesale. CD-ROM.-3--3-
6. Pathological anatomy [Elektronnyi resurs] : atlas: ucheb. posobiye / O. V. Zairatyants [i dr.] ; ed. by O. V. Zairatyants. -Electron. textual dan. (144 Mb). - Moscow: Izd. group "GEOTAR-Media", 2010. - 472 p. e-mail. wholesale. CD-ROM .--1--1-
7. Paukov, V. S. Patologicheskaya anatomy i patologicheskaya fiziologiya [Pathological anatomy and pathological physiology]. for colleges. -Electron. textual dan. (44.7 Mb). - Moscow: Izd. group "GEOTAR-Media", 2010. - 256 p. wholesale. CD-ROM-2--2-

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- information and legal system "Zan" - https://zan.kz/ru
- Medline Ultimate EBSCO
- eBook Medical Collection EBSCO
- Scopus - https://www.scopus.com/

In English

Main:

1. Kumar V., Abbas A.K., Aster C. Robbins Basic Pathology. – Elsevier, 2017.
2. Kumar V. Robbins and Cotran. Pathologic Basic of Disease. – Elsevier, 2015.
3. Klatt E.C., Kumar V. Robbins and Cotran. Review of Pathology. – Saunders, 2015.
4. Klatt E.C., Robbins and Cotran. Atlas of Pathology. – Saunders, 2014.

Additional:

1. Litvitsky P.F., Pirozhkov S.V., Tezikov E.B. Pathophysiology. Concise lec-tures, tests, clinico-patophysiological situations and clinico-laboratory cases: student manual. Moscow, GEOTAR-Media Publ., 2012.
2. Robbins and Cotran. Review of Pathology //Klatt E.C., Kumar V. – Third Edition. – Saunders Elsevier, 2010.