Discipline: "Biochemistry"

Course Code: Bio 2204

Title of the EP: 6B10115 Medicine

Amount of study hours/credits: 90 hours/3 credits

Course and semester of study: 3rd year,5semester

Lecture duration: 6 hours

Shymkent – 2025

The lecture complex was developed in accordance with the working program of the discipline (syllabus) "Biochemistry" and discussed at a department meeting.

Protocol No 1 dated 27.08. 2025 years

Head of Department, Acting Professor: _____ **Daurenbekov K.N.**

No. 1

1. Subject: Pathology of carbohydrate metabolism (diabetes mellitus, hypoglycemia, glycogenosis).

2. Objective: To introduce students to the fundamental biochemical mechanisms of carbohydrate metabolism disorders and their pathogenetic consequences. To examine the clinical and biochemical manifestations of diabetes mellitus, hypoglycemia, and glycogen storage diseases. To develop an understanding of the relationship between disruptions of enzymatic systems, regulatory mechanisms, and the development of pathological conditions.

3. Lecture abstracts. Carbohydrate metabolism plays a central role in providing the body with energy and maintaining the stable functioning of all organs and systems. Glucose is the primary and universal energy substrate, especially for tissues that cannot utilize other sources, such as the brain and red blood cells. Therefore, any disturbances in its metabolism impact vital functions. Carbohydrate metabolism pathologies are varied, but the most important examples include diabetes mellitus, hypoglycemic states, and glycogen storage diseases.

Diabetes mellitus is a chronic disease characterized by persistent hyperglycemia due to insulin deficiency or peripheral tissue insensitivity to it. This affects not only carbohydrate metabolism but also lipid, protein, and water-electrolyte metabolism, leading to systemic disorders. Type 1 diabetes is caused by autoimmune destruction of pancreatic β-cells, leading to absolute insulin deficiency. Type 2 is characterized by insulin resistance, where the hormone is produced, but tissues lose sensitivity to it. Biochemical manifestations of diabetes include persistent hyperglycemia, the presence of sugar in the urine, increased lipolysis with subsequent formation of ketone bodies, lipid imbalance, and a tendency toward metabolic acidosis. Clinically, this is accompanied by polyuria, thirst, weight loss, and, in the long term, the development of complications associated with damage to the blood vessels, kidneys, eyes, and nervous system.

Hypoglycemia is the opposite condition to hyperglycemia. It is characterized by a drop in blood glucose levels below a critical level, which threatens the brain's energy supply. The main causes of hypoglycemia can include an overdose of insulin or hypoglycemic medications, prolonged fasting, pancreatic tumors that produce excess insulin, and severe liver disease that disrupts glycogenolysis and gluconeogenesis. A lack of glucose leads to activation of the sympathoadrenal system, manifested by sweating, tachycardia, tremors, and anxiety. Further declines in sugar levels lead to impaired consciousness, seizures, and hypoglycemic coma. This condition is especially dangerous for children, as their brains require a constant supply of glucose.

Glycogenoses—hereditary enzyme deficiencies associated with impaired glycogen synthesis or breakdown—are a distinct group of pathologies. Since glycogen is the body's primary carbohydrate storage, its proper accumulation and utilization are critical for maintaining energy balance. Enzyme defects result in either excessive accumulation of glycogen in tissues or an inability to release glucose from it. Depending on the type of enzyme defect, the liver, muscles, or heart are affected. For example, in von Gierke disease, a lack of the glucose-6-phosphatase enzyme causes severe hypoglycemia and pronounced hepatomegaly. Pompe disease is associated with impaired glycogen breakdown in lysosomes, leading to its accumulation and damage to cardiac and muscle tissue. In McArdle disease, muscle phosphorylase deficiency manifests as exercise intolerance and muscle pain. The common biochemical result of these diseases is energy deficiency and disruption of the functioning of organs dependent on glucose and its derivatives.

Thus, carbohydrate metabolism pathology encompasses a wide range of disorders, varying in origin and mechanism, but sharing a common foundation—an imbalance between the body's energy needs and the ability to obtain it from glucose. Diabetes mellitus demonstrates how hormonal dysregulation can lead to chronic hyperglycemia and multiple complications. Hypoglycemia reveals the flip side—an acute, life-threatening deficiency of the energy substrate. Glycogenoses clearly illustrate the importance of enzymatic systems and hereditary factors in maintaining energy balance.

Understanding the biochemical basis of these diseases is essential for understanding their clinical manifestations, making a correct diagnosis, and developing effective treatments. Studying carbohydrate metabolism pathology gives students a holistic understanding of how disturbances at the molecular level lead to serious systemic consequences affecting all organs and tissues of the body.

4. Illustrative material:

PowerPoint presentation

5. *Literature:

6.Test questions:

1. How do the mechanisms of development of type 1 and type 2 diabetes differ?
2. Why is hypoglycemia dangerous for nervous tissue?
3. What biochemical disturbances are characteristic of glycogenoses?
4. How do the body's compensatory mechanisms manifest themselves in chronic hyperglycemia?

No. 2

1. Subject: Pathology of lipid metabolism (atherosclerosis, obesity, hyperlipoproteinemia).

2. Objective: To study the biochemical mechanisms of lipid metabolism disorders and their role in the development of atherosclerosis, obesity, and hyperlipoproteinemia. To examine the pathogenetic and clinical-biochemical manifestations of the main diseases associated with lipid metabolism disorders. To develop an understanding of the relationship between metabolic disorders and the development of systemic pathologies of the cardiovascular and endocrine systems.

3. Lecture abstracts: Lipid metabolism in the body is crucial, as lipids perform several vital functions. They serve as the body's primary energy reserve, participate in the construction of cell membranes, and act as precursors for hormones and signaling molecules. However, disruption of their metabolism leads to the development of a number of pathological conditions, of which atherosclerosis, obesity, and hyperlipoproteinemia are the most significant in medicine.

Atherosclerosis is the most common and socially significant disease associated with lipid metabolism disorders. Its development is based on an imbalance between the intake, transport, and utilization of cholesterol and lipoproteins. As low-density lipoprotein levels increase, they begin to penetrate the arterial wall, where they are oxidized and phagocytized by macrophages. The resulting foam cells initiate an inflammatory response, leading to thickening of the vascular wall and the formation of atherosclerotic plaques. Gradually, the vascular lumen narrows, impairing blood flow and leading to tissue ischemia. Biochemical changes include increased total cholesterol and increased concentrations of low- and very-low-density lipoproteins, while high-density lipoproteins decrease. All this reflects a shift in the balance toward atherogenic particles. Clinically, atherosclerosis is the leading cause of coronary heart disease, strokes, and chronic vascular insufficiency.

Obesity is a chronic energy imbalance in which dietary energy intake significantly exceeds energy expenditure. It is caused by the excessive accumulation of triglycerides in adipose tissue. Biochemically, this is accompanied by increased lipogenesis, decreased lipolysis, and disruption of hormonal regulation, primarily the action of leptin and insulin. Changes in adipose tissue lead to endocrine dysfunction: adipocytes begin to secrete various cytokines that promote chronic inflammation and the development of insulin resistance. This links obesity to metabolic syndrome, which includes hyperglycemia, hypertension, dyslipidemia, and a high susceptibility to thrombosis. The clinical consequences of obesity are not limited to cosmetic defects. It is directly linked to the development of type 2 diabetes, cardiovascular disease, fatty liver disease, and many other pathologies.

Hyperlipoproteinemias are a heterogeneous group of disorders characterized by elevated lipoprotein concentrations in the blood. They can be primary, associated with inherited enzyme and receptor defects, or secondary, arising from diabetes mellitus, obesity, alcoholism, and liver disease.

There are several types of hyperlipoproteinemia, which differ in the specific lipoproteins that accumulate. For example, familial hypercholesterolemia is characterized by a deficiency of low-density lipoprotein receptors, leading to significantly elevated cholesterol levels and the early development of atherosclerosis. Hypertriglyceridemias increase the levels of very low-density lipoproteins and chylomicrons in the blood, increasing the risk of pancreatitis and cardiovascular complications. A common pathogenetic link in all forms of hyperlipoproteinemia is impaired lipid transport in plasma, leading to their pathological deposition in tissues and vascular walls.

Despite their different mechanisms, all three of these pathologies share a common feature: they lead to an imbalance between the intake, utilization, and elimination of lipids, which disrupts energy and plastic metabolism throughout the body. Atherosclerosis demonstrates how excess atherogenic lipids damage the vascular system. Obesity reflects the consequences of chronic energy imbalance and develops into an endocrine-metabolic disease. Hyperlipoproteinemia demonstrates the importance of genetic and enzymatic factors in the regulation of lipid metabolism.

The significance of these pathologies extends far beyond biochemistry. Their study is of enormous practical importance, as they underlie the most common diseases in modern society—cardiovascular accidents, diabetes, and chronic liver disease. Understanding the biochemical mechanisms of lipid metabolism disorders helps physicians not only correctly diagnose the disease but also select the most effective approaches to treatment and prevention.

Thus, lipid metabolism pathology is one of the most important topics in medicine, as it combines fundamental knowledge of lipid biochemistry with clinical manifestations that are of great importance for public health.

4. Illustrative material:

PowerPoint presentation

5. *Literature:

6.Test questions:

1. How is lipoprotein metabolism disorder associated with the development of atherosclerosis?
2. Why is obesity considered not only as an energy disease, but also as an endocrine disease?
3. What changes in the lipid profile can be considered diagnostic markers of pathology?
4. What are the biochemical consequences of chronic hyperlipoproteinemia on blood vessels?

No. 3

1. Subject: Pathology of protein and amino acid metabolism (amyloidosis, dysproteinemia, hereditary diseases).

2. Objective: To introduce students to the fundamental biochemical mechanisms of protein and amino acid metabolism disorders and their consequences for the body. To examine the pathogenesis, clinical, and laboratory manifestations of amyloidosis, dysproteinemia, and hereditary amino acid metabolism disorders. To develop an understanding of the relationship between protein and amino acid metabolism disorders and the development of systemic diseases.

3. Lecture abstracts: Proteins and amino acids are the foundation of life: they form part of cellular structures, perform enzymatic and transport functions, participate in immune responses, and regulate metabolic processes. Disturbances in their metabolism impact the entire body, affecting virtually all levels of metabolism. Protein and amino acid metabolism disorders encompass a wide range of diseases, but amyloidosis, dysproteinemia, and hereditary diseases associated with enzyme system dysfunction are particularly significant.

Amyloidosis is a disease in which a pathological protein-polysaccharide complex called amyloid is deposited in tissues and organs. Its formation is associated with impaired coagulation and degradation of plasma proteins, most commonly immunoglobulins or acute-phase reactants. Amyloid has an abnormal fibrillar structure resistant to proteolysis, causing it to gradually accumulate in the

intercellular space, impairing organ function. Clinically, amyloidosis manifests itself in a variety of ways: from nephrotic syndrome due to kidney damage to heart failure due to amyloid deposition in the myocardium. From a biochemical perspective, it illustrates the dangers of the presence of a "foreign" protein that resists normal degradation processes.

Dysproteinemias are disturbances in the quantitative or qualitative composition of blood plasma proteins. They can result from both chronic inflammatory processes and malignant proliferation of immunoglobulin-producing cells. In some cases, a decrease in albumin is observed, leading to a decrease in oncotic pressure and the development of edema. In other cases, abnormal protein fractions—paraproteins—characteristic of myeloma appear in the blood. Biochemical analysis of dysproteinemias reveals changes in the electrophoretic profile of plasma proteins, which is of significant diagnostic value. These disturbances particularly clearly demonstrate the role of blood proteins in maintaining homeostasis, immune defense, and the transport of various compounds.

Hereditary diseases of amino acid metabolism are a group of disorders resulting from congenital defects in enzymes involved in the catabolism or synthesis of amino acids. A classic example is phenylketonuria, which is characterized by a lack of the enzyme phenylalanine hydroxylase. As a result, phenylalanine is not converted to tyrosine, and its excess and toxic metabolites accumulate in the body, damaging the nervous system and causing severe mental retardation. Another example is alkaptonuria, which is associated with a disorder of tyrosine metabolism. Patients develop a buildup of homogentisic acid, which darkens the urine and leads to pigment deposition in cartilage and joints. Another hereditary disorder is cystinuria, which impairs the reabsorption of certain amino acids in the renal tubules, leading to their loss in the urine and the formation of stones. All of these diseases illustrate the importance of enzymatic specificity: the absence of just one enzyme leads to severe consequences for the body.

Combining amyloidosis, dysproteinemias, and hereditary amino acid metabolism disorders, we see a common pattern: disruption of the normal structure or regulation of proteins leads to pathological conditions that can lead to disability and death. Proteins perform not only structural functions but also regulatory, transport, and protective functions. Therefore, the slightest changes in their metabolism immediately impact the body's vital functions.

Amyloidosis demonstrates how abnormal protein complexes can destroy tissue. Dysproteinemias demonstrate the critical importance of maintaining the balance of protein fractions in the blood. Hereditary amino acid metabolism disorders clearly demonstrate that genetic enzyme defects lead to severe metabolic disorders, which can often be prevented only with early diagnosis and dietary adjustments.

Thus, pathologies of protein and amino acid metabolism are not only of theoretical but also of enormous practical importance. Their study allows for a deeper understanding of the relationship between biochemistry and clinical practice, as well as the development of diagnostic and treatment skills for diseases that directly depend on disturbances at the molecular level.

4. Illustrative material:

PowerPoint presentation

5. *Literature:

6.Test questions:

1. Why is amyloid called a “foreign” protein?
2. How does the plasma protein spectrum change in dysproteinemia?
3. What mechanisms underlie hereditary diseases of amino acid metabolism?
4. What is the importance of laboratory biochemical methods in the diagnosis of amyloidosis?

No. 4

1. Subject:"Enzymopathies".

2. Objective: To introduce students to the biochemical nature of enzymopathies and the mechanisms by which they develop. To examine the main hereditary diseases associated with enzyme system disorders. To demonstrate the importance of early diagnosis of enzymopathies to prevent serious consequences.

3. Lecture abstracts: Enzymes are the primary biocatalysts of all biochemical reactions occurring in the body. Their activity determines the rate and direction of metabolic processes. Any disruption in the structure or function of an enzyme leads to metabolic changes, which underlie a large number of pathological conditions. Hereditary diseases associated with enzymes are collectively known as "enzyme disorders."

Enzyme disorders are a group of diseases resulting from congenital defects in the genes encoding enzymes. These defects result in the synthesis of enzymes with abnormal structures, reduced activity, or complete inactivation. As a result, normal metabolic pathways are disrupted: either toxic intermediates accumulate or a deficiency of end metabolites vital to the body occurs.

The biochemical nature of enzymopathies is explained by the direct link between genes and enzymes. A genetic mutation alters the amino acid sequence of a protein, leading to a disruption of its conformation and loss of catalytic activity. Sometimes, an enzyme is synthesized in normal quantities but is unable to perform its function due to the absence of a cofactor or a defect in the active site.

An example of a classic enzymopathy is phenylketonuria (PKU), which is associated with a deficiency of the enzyme phenylalanine hydroxylase. In this condition, the amino acid phenylalanine is not converted to tyrosine and begins to accumulate, forming toxic metabolites. In children, this leads to severe damage to the nervous system and mental retardation. However, with early diagnosis and adherence to a special diet, complications can be completely prevented.

Another example is Gaucher disease, a hereditary disorder characterized by a deficiency of the enzyme glucocerebrosidase. As a result, the lipid glucocerebroside accumulates in cells, leading to damage to the liver, spleen, and bones. These conditions are called storage diseases because their primary manifestation is the excessive deposition of a substrate that the enzyme cannot break down.

Alkaptonuria is another classic enzymopathy. It is associated with a deficiency of the enzyme homogentisinase, which is involved in tyrosine metabolism. This causes homogentisic acid to accumulate, darkening the urine and depositing in cartilage, causing its destruction.

Enzyme disorders can affect virtually all types of metabolism: carbohydrate, lipid, protein, and mineral. For example, in glycogen storage diseases, the breakdown or synthesis of glycogen is impaired, causing hypoglycemia, hepatomegaly, and muscle weakness in children. In galactosemia, the enzyme galactose-1-phosphate uridyltransferase is absent, leading to the accumulation of toxic galactose products and liver and brain damage.

Enzyme system disorders can manifest at various ages, from the first days of life to adulthood. In severe cases, the disease manifests in newborns with acute liver, brain, or heart damage. In milder forms, symptoms appear later, with the accumulation of toxic products.

Early diagnosis of enzyme deficiencies is particularly important. Neonatal screening—mass testing of newborns for a number of hereditary diseases—is currently used in many countries. For example, phenylketonuria and galactosemia are included in the mandatory list of tests. Early diagnosis allows for timely treatment, most often dietary therapy that limits the intake of toxic substrates.

Therapy for enzyme deficiencies is primarily aimed at reducing the accumulation of pathological metabolites and replenishing the deficiency of normal products. In some cases, enzyme

replacement therapy is used, in which the patient is given the missing enzyme. This approach is used, for example, in Gaucher disease.

Thus, enzymopathies are a clear example of how a single enzyme disorder can lead to severe systemic consequences. They demonstrate a direct link between genetic mutations, biochemical processes, and clinical manifestations. For physicians, understanding the mechanisms of enzymopathies is important not only for diagnosis but also for choosing the right treatment strategy and preventing complications.

4. Illustrative material:

PowerPoint presentation

5. *Literature:

6.Test questions:

1. Why are hereditary diseases often associated with enzyme defects?
2. What explains the accumulation of toxic products in enzymopathies?
3. What examples of enzymopathies can you give and what is their biochemical mechanism?
4. What is the importance of neonatal screening for the diagnosis of enzymopathies?
5. What modern approaches to the treatment of enzyme deficiencies do you know?

No. 5

1. Subject: Blood pathology (anemia, leukemia, jaundice, coagulopathy).

2. Objective: To introduce students to the biochemical and pathogenetic mechanisms of major blood diseases. To examine the clinical manifestations of anemia, leukemia, jaundice, and coagulopathies. To develop an understanding of the relationship between cellular and molecular disorders and systemic pathological processes.

3. Lecture abstracts: Blood is a unique tissue in the body, transporting oxygen, nutrients, hormones, and metabolic products. It plays a central role in immune defense and maintaining homeostasis. Disturbances in blood composition and function inevitably impact the health of all organs and systems. The most important examples of blood pathologies include anemia, leukemia, jaundice, and coagulopathy.

Anemias are a group of diseases characterized by a decrease in the red blood cell count or hemoglobin content, leading to tissue hypoxia. The causes of anemia are varied. Iron deficiency anemia occurs due to iron deficiency and impaired hemoglobin synthesis. B12 and folate deficiency anemia are associated with impaired DNA synthesis and manifest as megaloblastic changes. Hemolytic anemias occur with accelerated red blood cell destruction, accompanied by increased bilirubin and splenomegaly. Anemia of chronic disease develops due to impaired iron utilization and suppressed erythropoiesis. The common biochemical outcome of all anemias is a decrease in the oxygen-carrying capacity of the blood, which causes weakness, dizziness, tachycardia, and decreased performance.

Leukemias are malignant diseases of the hematopoietic system characterized by the uncontrolled proliferation of immature leukocytes. These cells lose their ability to differentiate normally, accumulate in the bone marrow, and displace normal hematopoietic lineages. This leads to anemia, thrombocytopenia, and immunodeficiency. Acute and chronic forms of leukemia are distinguished, differing in the rate of progression and morphological features of the cells. Biochemical changes include changes in enzymatic activity, impaired purine metabolism, and the development of hyperuricemia due to increased cell breakdown. Clinical manifestations include weakness, infections, bleeding, and enlarged lymph nodes and liver. Leukemias illustrate how cell cycle dysregulation leads to systemic blood disease.

Jaundice is a syndrome characterized by elevated bilirubin levels in the blood and a yellow discoloration of the skin and mucous membranes. Jaundice is divided into three main groups: suprahepatic (hemolytic), hepatic, and subhepatic (mechanical). In hemolytic jaundice, excessive red blood cell breakdown leads to the accumulation of unconjugated bilirubin. In hepatic jaundice, the uptake, conjugation, or excretion of bilirubin by liver cells is impaired, as occurs in hepatitis and cirrhosis. In mechanical jaundice, the cause is impaired bile outflow through the bile ducts, leading to the accumulation of conjugated bilirubin and bile acids. Biochemical diagnostics are based on the determination of bilirubin fractions, transaminase activity, and alkaline phosphatase. Jaundice is an important example of how pigment metabolism disorders reflect pathology of both the blood and the liver.

Coagulopathies are disorders characterized by abnormalities in the blood coagulation system. They can be congenital or acquired. A classic example of a congenital coagulopathy is hemophilia, which is associated with a deficiency of factors VIII or IX, in which fibrin clot formation is impaired. Acquired coagulopathies can be associated with vitamin K deficiency, liver disease, or excessive activation of the coagulation system in disseminated intravascular coagulation (DIC). Biochemically, coagulopathies are characterized by prolonged clotting time, decreased factor activity, and an imbalance between coagulation and fibrinolysis. Clinical manifestations include bleeding in various locations, hematomas, and hemorrhages into internal organs.

Thus, blood pathology encompasses a wide range of disorders that affect all cellular and plasma components. Anemias demonstrate the consequences of oxygen-transport deficiency, leukemias result from the malignant transformation of hematopoietic cells, jaundice reflects disturbances in hemoglobin and bilirubin metabolism, and coagulopathies demonstrate the critical role of the coagulation system in maintaining life. All these conditions clearly demonstrate that blood is not simply a transport system, but a complex and sensitive tissue, changes in which affect the entire body.

The study of blood pathologies is important not only for understanding biochemical processes, but also for practical medicine, since these diseases are among the most common and dangerous in clinical practice.

4. Illustrative material:
PowerPoint presentation
5. *Literature:
6.Test questions:
1. How does a decrease in hemoglobin affect tissue oxygen metabolism?
2. Why does immunodeficiency develop with leukemia?
3. What biochemical parameters can differentiate between different types of jaundice?
4. What is the difference between congenital and acquired coagulopathies?

No. 6

1. Subject: Pathology of connective and other tissues (collagenoses, osteoporosis, tissue amyloidosis).
2. Objective: To uncover the molecular and biochemical mechanisms underlying the development of connective tissue pathologies. To examine the pathogenesis, clinical manifestations, and laboratory diagnostics of collagenoses, osteoporosis, and amyloidosis. To develop an understanding of the role of connective tissue as a universal structure involved in maintaining the integrity of the body.
3. Lecture abstracts: Connective tissue is the fundamental structure of the body, providing mechanical strength, elasticity, and structural organization to organs and systems. It consists of cells (fibroblasts, osteoblasts, chondrocytes), intercellular matrix, collagen and elastic fibers, and matrix components. Connective tissue disorders disrupt not only local structures but also systemic processes,

as connective tissue binds and supports all tissues in the body. Key examples of such pathologies include collagenoses, osteoporosis, and tissue amyloidosis.

Collagenoses are a group of systemic diseases primarily involving connective tissue damage due to autoimmune disorders. These include systemic lupus erythematosus, scleroderma, dermatomyositis, and rheumatoid arthritis. The underlying pathogenesis is the formation of autoantibodies to components of the cell nucleus and matrix, which trigger immune complexes and inflammatory activation. This results in vasculitis, fibrosis, and collagen fiber degeneration. Biochemically, elevated levels of immunoglobulins, circulating immune complexes, and activation of proteolytic enzymes are observed. Clinical manifestations include polyarthralgia, skin rashes, and kidney, heart, and lung damage. Collagenoses clearly demonstrate how a disruption of immune regulation leads to the destruction of the body's supporting structure.

Osteoporosis is characterized by decreased bone density and deterioration of its microstructure. The underlying mechanism is an imbalance between bone resorption by osteoclasts and bone formation by osteoblasts. In osteoporosis, bone tissue loses calcium and organic matrix, making bones brittle and prone to fracture. Biochemical changes include increased levels of bone resorption markers, such as urinary hydroxyproline, and decreased calcium and phosphorus concentrations in bone. Hormonal factors are particularly significant: decreased estrogen levels in postmenopausal women, vitamin D deficiency, and hyperparathyroidism. Clinically, osteoporosis manifests as pathological fractures, decreased height, and spinal deformity. It is a classic example of a systemic connective tissue disorder, reflecting the interplay of metabolic, hormonal, and structural disturbances.

Amyloidosis is a disease in which a pathological protein, amyloid, accumulates in the intercellular space. This is a specific form of dysproteinosis associated with a disruption of protein structure and metabolism. Amyloid has a characteristic β-sheet structure, is resistant to proteases, and is deposited in various organs: the kidneys, heart, liver, and spleen. The pathogenesis of amyloidosis involves impaired plasma protein synthesis, their aggregation, and deposition in tissues. Depending on the cause, amyloidosis is classified as primary, secondary, and hereditary. Biochemically, amyloid consists of protein fibrils and glycosaminoglycans. Amyloid accumulation leads to cellular compression, impaired microcirculation, and parenchymal atrophy. Clinical manifestations depend on the location: kidney damage leads to nephrotic syndrome, heart damage leads to cardiomyopathy, and liver damage leads to hepatomegaly. Amyloidosis illustrates an example of a "foreign" protein disease, where the altered structure makes the protein toxic to tissues.

Thus, pathologies of connective and other tissues reflect a variety of injury mechanisms in the body. Collagenoses demonstrate autoimmune processes that lead to the destruction of the body's own structures. Osteoporosis demonstrates how an imbalance between matrix synthesis and degradation causes systemic tissue fragility. Amyloidosis is an example of a biochemical protein abnormality that compromises organ integrity. All these diseases highlight the pivotal role of connective tissue as a universal support for the normal functioning of the body.

The study of these pathologies is not only of academic but also of practical importance, as they are widespread and lead to disability. Biochemical research in this area allows for the identification of early signs of disorders and the development of modern treatments, including immunotherapy, hormonal drugs, and innovative approaches to tissue regeneration.

4. Illustrative material:

PowerPoint presentation

5. *Literature:

6.Test questions:

1. What mechanisms of autoimmune damage underlie collagenoses?
2. Why does osteoporosis develop more often in postmenopausal women?

3. How does amyloid impair organ function?
4. What biochemical markers can be used for early diagnosis of these pathologies?

Note: 5. *Literature:
main

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