AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

BIOCHEMICAL MECHANISMS OF ONCOGENESIS IN CHRONIC KIDNEY DISEASES

Halimboyev Hojiakbar Anvarjon ugli

Student of the group 209, the 2nd Faculty of Pediatrics, Medical Biology and Fundamental Medicine of TSMU

Tuychiyeva Mohiraxon Sultonali qizi

English teacher of the 1st category, 26th school, Beshariq district, Fergana region

Abdurashitov Murodjon Bahodir ugli

Student of the group 209, the 2nd Faculty of Pediatrics, Medical Biology and Fundamental Medicine of TSMU

Hikmatxuja Fatullayev Jasur ugli

Student of the 2nd Faculty of Pediatrics, Medical Biology and Fundamental Medicine of TSMU, group 208

Scientific supervisor: **Mukhammedova Sevara Nigmatulla qizi**

PhD, Assistant of the Department of Medical Informatics and Biophysics, Medical Biology, Medical and Biological Chemistry, Tashkent State Medical University

Abstract

This article analyzes the biochemical mechanisms of oncogenesis in chronic kidney disease. It was shown that inflammation, oxidative stress, and impaired cellular metabolism in kidney tissue in patients with chronic kidney disease can create conditions for oncogenesis. Modern science seeks not only to identify and explain developmental phenomena, but also to control biological processes using regulatory mechanisms. Therefore, the morphology of the study of tissue and organ relationships is an urgent and promising direction. In chronic renal failure, the level of vascular endothelial growth factor is dangerous due to a decrease in glomerular filtration rate and the appearance of vascular stiffness. Therefore, chronic renal failure not only leads to impaired functioning of all organs and systems of the body, but also causes morphological changes in the spleen, which allows it to be classified as a disease of medical and social importance.

Keywords

chronic kidney disease, renal functional reserve, glomerular filtration rate, antiplatelet agent, allthrombozepin, dipyridamole, edema, erythrocyturia, proteinuria, dysuria, nephrotic crisis, arthralgia, myalgia.

INTRODUCTION

Despite the fact that less than a quarter of the 21st century has passed, the problems of the course and treatment of chronic kidney disease (CKD) have not been adequately resolved.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

In the 21st century, chronic diseases, in particular chronic renal failure (CKD), remain an urgent problem for human health. Currently, the development of kidney diseases and their complex course, limitations in diagnosis and treatment require serious medical attention. Chronic kidney diseases may initially be asymptomatic, which makes them difficult to detect in a timely manner. During renal failure, the functional reserve of the kidneys decreases, the glomerular filtration rate decreases, as a result of which the body accumulates nitrogenous metabolites, electrolytes and other metabolic products. These processes are mainly associated with factors such as glomerulonephritis, pyelonephritis, drugs or metabolic diseases, including diabetes mellitus.

The pathogenesis of SBE includes several mechanisms: disruption of the hemostasis system and microcirculation, immune dysfunction, inflammation, and decreased ATPase activity. The disease is also characterized by changes in the balance of immunoglobulins, cytokines, and metabolic products in the blood. These changes affect not only the morphological and functional state of the kidneys, but also cause pathological changes in the cardiovascular, skeletal, and lymphoid systems, in particular, in the structure of the spleen. Microalbuminuria, proteinuria, and glomerular filtration rate (GFR) are important diagnostic indicators in identifying the early signs of the disease. At the same time, general blood and urine tests, biochemical and immunological tests are used to assess the condition of patients and determine the stages of the disease. Treatment is complex and includes rational nutrition, fluid and diet, drug therapy, as well as the practice of hemodialysis and peritoneal dialysis.

Relevance of the problem. In recent years, there has been an increase in kidney diseases. Today, the main diseases that cause SBE include diabetes mellitus, arterial hypertension, chronic glomerulonephritis, as well as a combination of these diseases.

It is known that the incidence of chronic kidney disease and renal failure varies widely across regions and remains a serious and urgent health problem. According to statistics, 1.2 million people died of chronic kidney disease worldwide in 2017. From 1990 to 2017, the global mortality rate from CKD among all ages increased by 41.5%, although there was no significant change in the age-standardized mortality rate. The global prevalence of CKD among all ages increased by 29.3% during this period.

It is worth noting that the clinical manifestations of chronic kidney failure develop with the loss of 70–75% of functionally active nephrons, since in this case their number is further reduced. The causes of this pathology are very diverse: these are congenital anomalies (polycystic kidney disease, hydronephrosis, renal hypoplasia) and acquired, undiagnosed inflammatory diseases (pyelonephritis, glomerulonephritis), drug-induced nephropathies (use of aminoglycosides, cytostatics), or the consequences of infection, metabolic diseases (diabetes mellitus), autoimmune diseases, etc.

Despite the high compensatory ability of the kidneys (even the remaining 10% of nephrons are able to maintain the water-electrolyte balance in the body), the early stages of chronic renal failure are manifested by quantitative disturbances in the electrolyte content of the blood, acidosis, impaired protein metabolism in the body and the retention of metabolic products: urea, creatinine, uric acid in the body, their increase in their amount. To date, more than 200 substances have been identified whose metabolism in the body is disturbed during renal failure.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

Pathological changes in renal function lead to a violation of the constancy of the internal environment of the body (homeostasis). With a decrease in the glomerular filtration rate and increased uremia, metabolism decreases, the transport and binding processes of many biologically active substances, including pituitary hormones, to target cells change. Thus, SBY leads to an increase in the level of prolactin, luteinizing hormone (LG) and follicle-stimulating hormone (FSG). In this group of patients, the concentrations of somatotropic hormone (SG), insulin-like growth factor-1 (IGF-1), thyroid-stimulating hormone (TTG), adrenocorticotropic hormone (ACTG), and vasopressin may remain within normal values or increase. Hemodialysis does not reduce the levels of prolactin, LG, FSG, while the concentrations of growth hormone, IGF-1, TTG normalize. The content of ACTG and vasopressin may remain unchanged or decrease.

Glomerulonephritis is an immuno-inflammatory disease of the renal glomeruli, which later spreads to the tubules and interstitial tissues and is accompanied by a gradual deterioration in kidney function.

It has latent, hematuric, hypertensive and nephrotic clinical forms. In the latent form, the patient may not even notice the course of the disease. On examination, proteinuria, microhematuria, leukocyturia are detected. The latent course can later turn into nephrotic or hypertensive forms. In the hematuric form, there are no external signs of the disease (edema, arterial hypertension). In the hypertensive form, the course of the disease occurs very slowly. In this form, symptoms characteristic of this disease such as "flies" flickering before the eyes, changes in the fundus, pain in the heart area, and left ventricular hypertrophy occur. The main clinical sign of the nephrotic form is proteinuria associated with severe damage to the renal filter, that is, the basal membrane and podocytes. As proteinuria increases, the amount of albumin in the blood decreases. As a result of hypoalbuminemia, there is a decrease in the oncotic pressure of the blood serum, which in turn causes the formation of edema.

Diagnostics: A general blood test reveals a slight increase in ECHT and a decrease in hemoglobin concentration. A general urine test shows proteinuria and hematuria. A biochemical blood test reveals signs of nephrotic or incomplete nephrotic syndrome - hypoalbuminemia, hypoproteinemia and dysproteinemia. In some patients, creatinine, uric acid and electrolytes (potassium, sodium) in the blood are higher than normal. Immunological tests detect antibodies to streptococci in the blood of patients with acute glomerulonephritis.

Treatment: Treatment includes a complex of measures such as rational nutrition, diet, and drug therapy. During the diet, salt, proteins, and fluid intake are limited in cases of edema. The amount of fluid drunk should be similar to diuresis. All patients suspected of acute glomerulonephritis should be immediately hospitalized and bed rest should be observed for 1-2 weeks. In severe cases, antibiotics are prescribed.

Pyelonephritis is a nonspecific inflammatory infectious disease of the kidneys, in which the parenchyma, calyx and calyx of the kidney are damaged.

Clinic: pain in the lower back is the most common complaint of patients, frequent urination, pain during urination, in chronic pyelonephritis, weakness, fatigue, headache, pain, loss of appetite are observed.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

Diagnostics: UTT, General blood test shows leukocytosis, a shift of the leukocyte formula to the left; in general urine test, alkaline pH (6.2-6.9) is observed.

Treatment: In severe cases, patients are treated in a hospital. To flush out microbes and toxins from the kidneys, it is prescribed to drink more fluids, and antibiotics are taken as the main treatment.

Acute kidney injury (AKI) is understood as a set of symptoms that occur as a result of a sudden violation of excretory function.

Clinic: In the clinical course, 4 periods are distinguished: initial, oligouric, polyuric, complete recovery of kidney function (but not always). In the 1st period, conditions such as shock, genolysis, poisoning are observed. In the 2nd period, diuresis decreases, reaching the level of anuria. In the 3rd period, due to electrolyte imbalance, the amount of urine excreted is 2-2.5 liters, sometimes more. In the 4th period, even if the symptoms of AKI are not observed, the symptoms characteristic of the disease may persist.

Diagnostics: The diagnosis is made based on creatinine levels in the blood and the amount of urine excreted. In addition, a general urinalysis is also performed.

Treatment: Treatment is aimed at eliminating etiological factors. Patients are immediately admitted to the intensive care unit or hemodialysis department. If there are contraindications to hemodialysis, peritoneal dialysis is performed.

Purpose of the study: three stages of acute lung injury in chronic renal failure are morphologically distinguished. The first of them is the early exudative stage (up to five days). It is characterized by capillary fragility, collapse of pulmonary alveoli, microthrombi, damage to the alveoli, infiltration of neutrophils, pulmonary edema, the presence of hyaline membrane and fibrin in the alveoli. The second stage is fibrino-proliferative (from six to ten days). Pulmonary edema gradually disappears and fibroblast proliferation begins. The third stage, which develops from the tenth day after the onset of acute lung injury, is characterized by the appearance of connective tissue (cells and fibers) in the foci of fibrous destruction. The main decompensating phenomenon in all stages is an increase in the permeability of the components of the aerohematoma barrier, which contributes to the development and progression of pulmonary edema. The development of acute emphysema is a compensatory mechanism. Atelectasis and dystelectasis occur when bronchioles are blocked by secretion, desquamation, and epithelial cells, and when type II alveolocytes, which are responsible for the synthesis and secretion of surfactant, are damaged, which contributes to the further development of structural changes in the lungs and the increase in hypoxia.

Risk factors for the development of SBE largely coincide with risk factors for the development of cardiovascular pathology, the most important of which are arterial hypertension and metabolic diseases, such as hyperglycemia, dyslipidemia, hyperuricemia, and obesity, as well as the toxic effects of drugs (analgesics, NSAIDs, nephrotoxic antibiotics, X-ray contrast agents), kidney damage, and smoking play an important role.

The initial signs of SBY are microalbuminuria (MAU) and a decrease in GFT, which is recommended to be calculated using the MDRD formula, the latter value is used to differentiate the stages of the disease and as a prognostic factor. MAU, proteinuria, a decrease in GFR < 60

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

ml/min. They are also considered independent risk factors for the development of cardiovascular diseases, therefore, patients with kidney pathology are included in the group of maximum cardiovascular risk. The central link in the cardiorenal relationship is the renin-angiotensin-aldosterone system (RAAS), endothelium-dependent factors and their antagonists - natriuretic peptides and the kallikrein-kinin system. Due to the activation of the renin-angiotensin and sympathetic nervous systems, the development of endothelial dysfunction and chronic systemic inflammation, damage to one of the organs initiates damage to the other, which leads to the development of cardiorenal syndrome (CRS).

Changes in the skeletal system are one of the severe complications of chronic uremia. In recent decades, there has been a steady increase in the number of patients with SBE worldwide, as well as a tendency to increase their life expectancy due to renal replacement therapy. With age, involutional anatomical and functional disorders occur directly in the kidneys themselves, which begin to appear at the age of forty. Among the many metabolic consequences of renal failure, electrolyte imbalance syndrome is one of the most important, which develops in almost 100% of cases in patients on program hemodialysis.

Adequate program hemodialysis, which provides partial relief of many metabolic diseases, as a rule, does not have a positive effect on phosphorus-calcium metabolism, today changes in bone tissue in chronic renal failure cannot be distinguished only by biochemical tests, but they are used to monitor the effectiveness of treatment. Thus, the concentration of calcium, phosphorus, alkaline phosphatase, PTG, as well as vitamin D and aluminum in the blood are regularly checked to monitor the progression of functional disorders into gross morphological changes in organs and tissues.

In recent decades, new clinical and experimental data have been obtained that deepen our understanding of the state of the immune system in chronic renal failure and possible pathways of immunopathogenesis of the disease. It is known that for the active phase of the disease, a characteristic and persistent change in the immunogram — a significant decrease in the level of immunoglobulins, especially IgG, in the blood — is associated with a violation of their synthesis and, to a lesser extent, with protein loss in the urine. A decrease in the concentration of IgA is less pronounced, and the levels of IgM and IgE are often increased. It has been established that a decrease in the production of IgG in B cells and an increase in the synthesis of IgM and IgE by B cells are caused by a violation of the regulation of T cells, in particular, a defect in the replacement of IgM by B cells.

According to Zueva T.V. et al., a high frequency of acute coronary syndrome, stroke, hemorrhagic complications, acute heart failure, atrial and ventricular fibrillation is associated with impaired renal function.

Lymphocytes from patients with chronic renal failure have a reduced response to polyclonal activators. This response is partially restored when the patient's cells are incubated with normal human serum, rather than autologous, which may indicate both a defect in lymphocytes and the presence of inhibitors in the serum that cause "immunological autosuppression." In vitro, the phenomenon of inhibition of blastogenesis of lymphocytes from healthy people has been observed when plasma from patients with renal disease is added, which also indicates the presence of an active circulating immunosuppressive factor in patients.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

One of the factors that generalize inflammation in chronic kidney disease is the activation of the production and secretion of various cytokines and their increase in the blood. At the same time, activated monocytes and tissue macrophages synthesize anti-inflammatory and pro-inflammatory cytokines, which ensure the recruitment of effector cells (neutrophils, macrophages) to the site of inflammation and cause their phagocytic, bactericidal activity and the initiation of an antigen-specific immune response. It should be noted that the protective role of anti-inflammatory cytokines is manifested when these mediators act locally at the site of inflammation, but excessive and generalized production of anti-inflammatory cytokines leads to the development of organ dysfunction.

In SBE, the level of vascular endothelial growth factor is a risk factor for reducing glomerular filtration rate and the development of vascular stiffness. An increase in the content of C-reactive protein is associated with an increase in the stiffness of the vascular wall, and the concentration of vascular endothelial growth factor is associated with hyperfibrinogenemia. It is clear that systemic atherosclerosis develops rapidly in SBE as a result of increased arterial stiffness.

Emanuel V.L. and his students studied the value of annexin-5 as a biochemical marker of the preclinical stage of atherosclerosis in patients with SBE who did not have clinical signs of atherosclerosis. The authors showed that a decrease in glomerular filtration rate is accompanied by an increase in annexin-5 levels, and they concluded that its concentration is associated with indicators of the blood lipid spectrum.

Although the exact molecular mechanisms that cause pathological damage to the renal system are still being studied, studies suggest that obesity is associated with chronic systemic low-grade inflammation of pro-inflammatory cytokines such as tumor necrosis factor (TNF)-alpha and IL-6, due to a decrease in the level of several anti-inflammatory cytokines produced by the spleen. In addition, IL-10 levels are reduced in obesity, possibly due to a decrease in CD20 on B cells that produce IL-10. A high-fat diet induces multiple morphological changes in the kidney, including glomerular hypertrophy, decreased nephrin levels, and increased desmin levels, which are compensated for by pro-inflammatory cytokines produced by the spleen, particularly IL-10. Thus, impaired splenorenal communication in the setting of obesity may be responsible for the development of SBE in patients with these underlying risk factors.

The results of the study show that under the influence of two different factors, the activities of Na + -K + -ATPase, Ca 2+ -ATPase, Mg 2+ -ATPase and Ca 2+ -Mg 2+ -ATPase in spleen tissues significantly decreased compared to the control group over several time periods. ATPase is a membrane-associated protein found in all cells, the function of which is to maintain a chemical and electrical gradient across the cell membrane. The energy that maintains this electrochemical gradient comes from the hydrolysis of ATP to ADP.

It is the energy stored in this gradient that drives membrane transport, cotransport and metabolic systems of other substances; ensures the movement of cations, anions, amino acids and glucose across the cell membrane; and maintains the vital functions of the cell, including the membrane electric potential, cell volume and intracellular concentrations of Na^+, K^+ and Ca^{2+}. The degree of cell damage is closely related to the activity of ATPase. Any substance or clinical condition that alters the normal cytomembrane ATPase activity inhibits the transport of ions between the interior and exterior of the cell, alters the electrochemical gradient, and leads to a

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

wide range of cellular dysfunction, resulting in increased organ and morphological damage. The data presented also indicate that inhibition of membrane pump activity is a pathogenesis of splenic injury in the development of SBE. In addition to direct damage from toxic substances, serious disturbances of the internal environment, such as fluid and electrolyte imbalance, accumulation of metabolic waste products, and impaired energy metabolism, may also be responsible for this pathogenesis.

The spleen is composed of two types of tissue: white pulp and red pulp. The white pulp contains lymphoid cells and white fibers that are involved in the body's immune defense. The red pulp contains red blood cells, white blood cells, and platelets, which are involved in the formation and breakdown of blood. The spleen has a rich network of blood vessels that are an integral part of its structure and provide a constant blood supply [7,8]. These vessels also serve to remove damaged or old cells from the blood.

Overall, the spleen performs important functions in the immune system and hematopoiesis, which makes it an important organ for maintaining health and well-being. Kidney failure can affect the spleen because the kidneys and spleen are closely linked. Kidney failure can cause changes in blood levels of iron, calcium, and other substances, which can affect the function of the spleen.

Currently, the main goal of specialists is to correct the pathogenetic links of SBE in various ways, slow down the progression of the disease, improve the quality of life of the patient and extend his life and the period before dialysis, which remains one of the urgent problems. A number of scientific studies are being conducted in the world to improve the microcirculation of the capillaries by coordinating the hemostasis system in patients with SBE and to introduce a gradient of renal functional reserve.

A review of the available literature revealed that there is a paucity of information on the effects of SBE on structural and morphological changes in the structure of the kidney, and some of it is outdated.

To treat chronic kidney disease (CKD), one should modify their diet to include foods that support kidney health and follow a meal plan developed by a registered dietitian.

- **Energy:** The recommended energy intake for people with CKD undergoing maintenance dialysis is 60 kcal/kg/day for 35 years and above and 30 kcal/kg/day for 35 years. We teach patients to have an adequate proportion of carbohydrates from sources such as cereals, millets, root vegetables, etc. For diabetic patients, only whole grains and high-fiber cereals are recommended; vegetables are recommended; and for non-diabetic patients, we allow simple carbohydrates (boiled potatoes, sweet potatoes, etc.) to maintain energy levels.
- **Limit salt intake:** To control blood pressure, the diet should contain no more than 2,300 milligrams of sodium per day. It is important to buy fresh food more often and cook it, as opposed to fast food, canned food, or frozen dinners, which are high in sodium. Avoid using salt when cooking, but use spices, herbs, or other sodium-free seasonings instead. You can always find out how much salt is in a food package by checking the nutrition label; anything over 20% of the daily value is too much salt; so try to use a low-sodium variety.
- **Protein Diet Management:** It is important to protect the kidneys by consuming the right amount and forms of protein. Excess protein intake can strain the kidneys. Therefore, it is important to consume small amounts of protein from both plant and animal protein sources.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

Such animal foods include poultry (such as chicken), fish and other seafood, red or white meat, including pork or beef, dairy products such as cheese or butter, etc. Eggs also fall into this category. For example, in terms of quantity, an average serving size for someone who eats something like beef would be two to three ounces, while another person who uses cheese might consider this amount to be half a cup of whole milk, which contains at least one slice of any sandwich made from anything other than what is mentioned here.

- **Eat heart-healthy foods:** When possible, choose healthier options such as grilling, broiling, baking, grilling, or roasting instead of deep-frying. Cook with nonstick cooking spray or olive oil instead of butter. Trim fat and skin from meats before eating. Avoid saturated and trans fats, and read food labels. Eat heart-healthy foods such as lean beef, skinless poultry, beans, potatoes, vegetables, fruits, fat-free or low-fat cheeses, milk, and yogurt.
- **Avoid a high-phosphorus diet:** If someone is trying to fight CKD, avoid foods and drinks that contain phosphorus to prevent weakening of the bones and blood vessels. Sometimes, too much phosphorus can lead to weak bones, itchy skin, and joint pain. Also, check the ingredients you buy from the butcher for phosphorus. There are some brands of deli meats that may be sprinkled with extra phosphorus, as well as fresh meat and poultry. Optimize potassium: Choose foods with optimal potassium content to facilitate nerve and muscle function. Low or high potassium levels can lead to kidney or heart problems in extreme cases. Make sure that potassium intake is minimized through the consumption of foods and drinks. Avoid using salt substitutes, as they may be high in potassium. It is also important to ensure that canned fruits and vegetables are drained.
- **Fluids:** Excessive fluid retention can lead to swelling, weight gain, and heart problems. To prevent such problems, it is important to monitor fluid intake and food sources, including gravies, sambar, rasam, soups, and porridge. Changes in blood pressure can also cause the heart to work harder.

CKD with Diabetes:

The American Diabetes Association (ADA) recommends eating fewer calories, increasing physical activity to promote weight management, and monitoring carbohydrate intake to maintain ABC (A1c, blood pressure, and cholesterol). A diet rich in carbohydrates, especially complex carbohydrates from fruits, vegetables, whole grains, legumes, and low-fat dairy, is recommended. A high-fiber diet (25-30 grams per day) can help control hemoglobin A1c and blood glucose levels.

CKD and Hypertension:

Blood pressure can be controlled by eating a healthy diet, which can help lower blood pressure in people with primary hypertension, as well as by following a diet with moderate sodium restriction, which is one of the most important causes of hypertension in the general population. Avoid canned foods, salted sauces, processed foods, and fast foods.

There is no single "best" food for healthy kidneys; some of the best foods for healthy kidneys include fruits, vegetables, fish, whole grains, and herbs. olive oil, bread, etc.

Conclusion. Modern science seeks not only to identify and explain developmental phenomena, but also to control biological processes using regulatory mechanisms. Therefore, the study of the morphology of tissues and organs is an urgent and promising direction. In chronic renal failure, the level of vascular endothelial growth factor is also dangerous due to a decrease in glomerular filtration rate and the appearance of vascular stiffness. Therefore, chronic renal failure not only leads to impaired functioning of all organs and systems of the body, but also causes morphological changes in the spleen, which allows it to be classified as a disease of medical and social importance.

AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 6. No 03. March ,2026

REFERENCES:

1. Abduraximov A. X., Ergasheva Z. A., Kasimova I. K. Korreksiya elektrolitnogo disbalansa pri khronicheskoy pochechnoy nedostatochnosti. Rezyume // *Akademik DS Seksenbaevti*. 2019. – T. 80. – S. 76.
2. Avezova D. B. Morfologicheskie izmeneniya legkikh 9-mesyachnykh belykh kris posle khronicheskoy pochechnoy nedostatochnosti // *TADQIQOTLAR.UZ*. 2024. – T. 38. – №1. – S. 210–218.
3. Aydarov Z. A. i dr. Khronicheskaya pochechnaya nedostatochnost i serdechno-sosudistye zabolevaniya: problema mezhdissiplinarnaya // *The Scientific Heritage*. 2020. – №49-2. – S. 10–17.
4. Aringazina A. M. i dr. Khronicheskaya bolezn pochek: rasprostranennost i faktori riska (obzor literatury) // *Analiz riska zdorovyu*. 2020. – №2. – S. 164–174.
5. Ahmedova N., Amonov M. Viyavlenie faktorov riska i optimizatsiya ranney diagnostiki khronicheskoy bolezni pochek // *Jurnal vestnik vracha*. 2020. – T. 1, №3. – S. 26–31.
6. Batyushin M. M. Khronicheskaya bolezn pochek: sovremennoe sostoyanie problemi // *Ratsionalnaya farmakoterapiya v kardiologii*. 2020. – T. 16, №6. – S. 938–947.
7. Dadashev A. Sh. i dr. Morfometricheskie osobennosti razlichnogo vida bifurkatsiy vnutriorgannogo arterialnogo rusla selezenki u lits raznogo pola i vozrasta // *Rossiyskiy mediko-biologicheskiy vestnik imeni akademika IP Pavlova*. 2024. – T. 32, №1. – S. 81–92.
8. Dadashev A. Sh. i dr. Morfometricheskie osobennosti raznogo tipa strukturnykh komponentov vnutriorgannogo venoznogo rusla selezenki // *Chelovek i ego zdorove*. 2024. – T. 27, №1. – S. 30–38.
9. Daminova M. A. Khronicheskaya bolezn pochek u detey: etiologiya, klassifikatsiya i faktori progressirovaniya // *Vestnik sovremennoy klinicheskoy meditsini*. 2016. – T. 9, №2. – S. 36–41.
10. Zufarov K.A. "Gistalogiya." – Toshkent: Ibn Sino nashriyoti, 2010.