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EFFECT OF CHRONIC KIDNEY FAILURE ON MORPHOLOGICAL INDICATORS OF THE SPLEEN

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ABSTRACT

Chronic kidney disease remains a serious public health problem worldwide, not only in terms of medical but also social and economic aspects. In recent years, there has been an increase in kidney disease. Today, the main diseases causing chronic kidney failure (CKD) are 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 kidney failure varies significantly in different regions and remains a serious and pressing health problem. According to statistics, 1.2 million people died from chronic kidney disease worldwide in 2017. From 1990 to 2017, the global mortality rate from cardiovascular diseases among all ages increased by 41.5%, although there were no significant changes in the age-standardized mortality rate. The global prevalence of CKD among all ages increased by 29.3% during this period.

Objective of the study: to conduct a comparative analysis of changes in morphological and morphometric parameters of the spleen in chronic renal failure at the same age.

Key words: kidneys, mucous membrane, mineral water, morphology.

INTRODUCTION

It should be noted that clinical manifestations of chronic renal failure develop with the loss of 70-75% of functionally active nephrons, as the condition of animals worsens, their number decreases even more. 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), and drug nephropathy, and the consequences of infection, metabolic diseases (diabetes mellitus), autoimmune diseases and others (1,7).

Despite the high compensatory capabilities of the kidneys (even the remaining 10% of nephrons are able to maintain water-electrolyte balance in the body), in the early stages of chronic renal failure, quantitative disturbances in the composition of electrolytes in the blood, acidosis, protein metabolism disorders in the body and an increase in the level of metabolic products: urea, creatinine, uric acid. Their retention in the body is manifested by an increase in their number. To date, more than 200 substances have been identified whose metabolism in the body is disrupted in renal failure (8,9).

Pathological changes in renal function lead to disruption of the constancy of the internal environment of the body (homeostasis). With a decrease in the glomerular filtration rate and an increase in uremia, metabolism decreases, the processes of transport and binding of many biologically active substances, including pituitary hormones, with target cells change. Thus, CRF leads to an increase in the level of prolactin, luteinizing hormone (LH) and follicle-stimulating hormone (FSH). In this group of patients, the concentrations of somatotropic hormone (SH), insulin-like growth factor-1 (IGF-1), thyroid-stimulating hormone (TSH), adrenocorticotropic hormone (ACTH) and vasopressin may remain within normal limits or be increased. Hemodialysis does not reduce the levels of prolactin, LH or FSH, but normalizes the concentrations of growth hormone, IGF-1 and TSH. ACTH and vasopressin levels may remain unchanged or decrease (4,6,12).

According to D.B. Avezova, three stages of acute lung injury in chronic renal failure are morphologically distinguished. The first of these is the early exudative stage (up to five days). It is characterized by capillary congestion, collapse of the pulmonary alveoli, microthrombi, alveolar damage, neutrophilic infiltration, pulmonary edema, and the presence of a hyaline membrane and fibrin in the alveoli. The second phase is fibrinoproliferative (six to ten days). Pulmonary edema gradually disappears and fibroblast proliferation begins. The third stage, developing 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 at all stages is an increase in the permeability of the air-hematoma barrier components, which contributes to the development and progression of pulmonary edema. The development of acute pulmonary emphysema is a compensatory mechanism.

Atelectasis and dyslectasis occur when the bronchioles are blocked by secretions, desquamation and epithelial cells, as well as when type II alveolocytes

responsible for the synthesis and secretion of surfactant are damaged, which contributes to the further development of structural changes in the lungs and increased hypoxia (2,11).

Today, the main goal of specialists is to correct the pathogenetic links of chronic renal failure in various ways, slow down the progression of the disease, improve the patient's quality of life, prolong his life and the period before dialysis, which remains one of the most pressing problems.

MATERIAL AND METHODS OF RESEARCH

To conduct a comparative analysis of changes in morphological and morphometric parameters of the spleen in chronic renal failure at the same age. Hematoxylin-eosin, Van Gieson and immunohistochemistry methods were used to study the obtained micropreparations.

RESULTS AND DISCUSSION

In chronic renal failure, the response of lymphocytes to polyclonal activators is reduced. In vitro, in renal failure, inhibition of lymphocyte blastogenesis is observed with the addition of plasma, which also indicates the presence of an active circulating immunosuppressive factor. The spleen of 6-month-old white outbred rats is surrounded on the outside by a serous membrane, a thin fibrous capsule called the splenic capsule, thickened and characterized by increased strength.

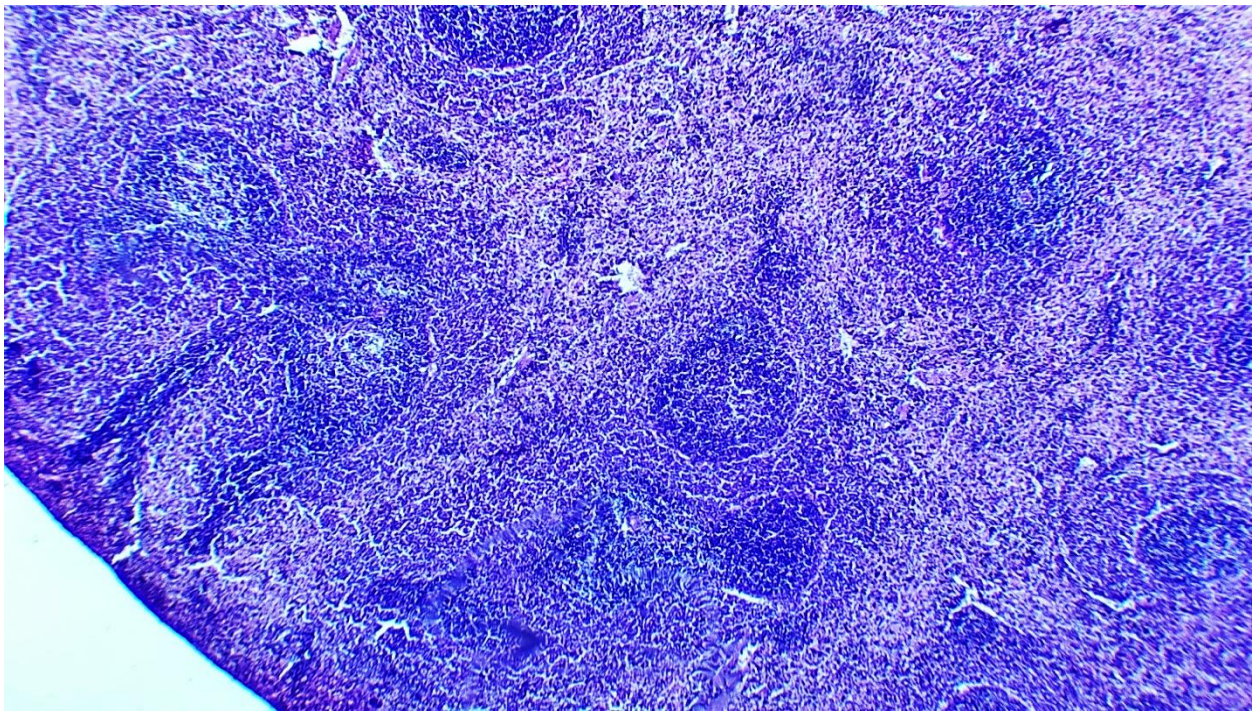

Figure 1. Morphological structure of spleen tissue. 1. Spleen capsule, 2. White pulp area, 3. Mantle and marginal area. 4. Collagen and elastic fibers of spleen trabeculae

Barriers called trabeculae extend from the capsule into the spleen. The spleen of 6-month-old albino rats is elongated and dark red in color because it is filled with blood. It is surrounded on the outside by a serous membrane and a fibrous capsule. Barriers called trabeculae extend from the capsule into the spleen.

The trabeculae and the walls of the blood vessels are thickened (a slightly pinkish area is visible with Van Gieson stain). The trabeculae divide the spleen into less clearly defined segments. The trabeculae are robust and thick in appearance. The trabeculae divide the spleen into less clearly defined segments. If the wall of the blood vessel is greatly thickened, the trabeculae and the walls of the blood vessels stain pale pink with Van Gieson stain. Between the trabeculae are white and red pulp. The volume of white pulp is small, and the volume of red pulp is 2-3 times greater than the volume of white pulp. In 6-month-old white outbred rats, the PALS area is of medium size. The PALS area is mainly populated by T-lymphocytes in large quantities, and B-lymphocytes also accumulate there eccentrically, forming the white pulp of the spleen. The central part of the lymphoid follicle of the white pulp is its proliferation center or reactive center, with a distinction made between the mantle-covered and marginal-coagulation areas. The central part is large and consists of B-lymphocytes, B-lymphoblasts, macrophages and plasma cells in the mantle zone, which is called the B-zone.

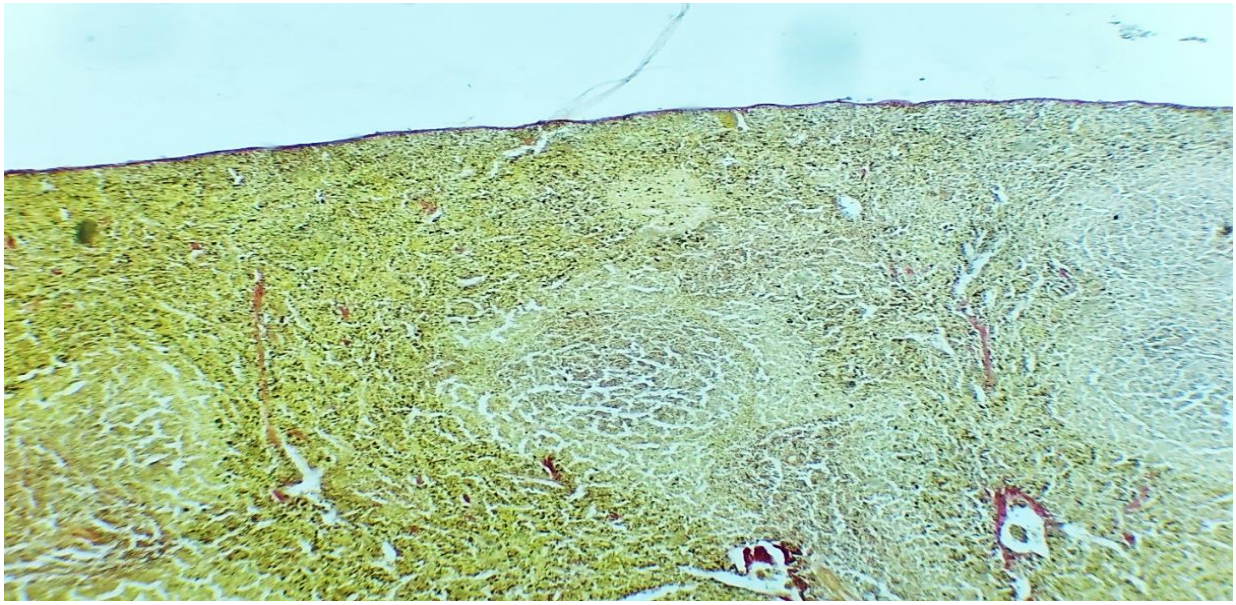

Figure 2. Morphological structure of spleen tissue. Red pulp area. 2. Thin splenic cords. 3. Central artery of the spleen. 4. Area of the periarteriolar lymphatic layer. 5. Collagen and elastic fibers of the splenic trabeculae.

The volume of the white pulp is small, and the volume of the red pulp is 2-3 times larger than the volume of the white pulp. As the disease progresses, T-lymphocytes accumulate around the artery (the T-PALS zone increases). B-

lymphocytes continued to accumulate around it, and the white pulp of the spleen increased. Among them, the reticular tissue grew into large sinusoidal blood vessels, also increasing the red pulp. The marginal zone also contains medium-sized T- and B-lymphocytes and a large number of macrophages - it performs the function of capturing and digesting antigens that have entered the blood, and is home to lymphocytes and lymphoblasts. This area is an important immune zone. The reticular stroma of the red pulp is expanded, the sinusoids of the blood vessels are expanded, the splenic cords located between these sinusoids are large. In the splenic strips, an increase in the number of B-lymphocytes and their derivative plasma cells is noted. In the reticular stroma of the red pulp, macrophages (derived from monocytes) are cells involved in phagocytosis, i.e., "splenocytes." The white pulp of the spleen consists of lymphatic follicles (Malpighian bodies). The central part of the lymphoid follicle is its proliferative or reactive center, a distinction is made between the mantle-covered and marginal-coagulation regions. The central part is large and consists of B-lymphocytes, B-lymphoblasts, macrophages, and plasma cells in the mantle zone, which is called the B-zone. The marginal zone contains increased T and B lymphocytes and a large number of macrophages - they perform the function of capturing and digesting antigens that have entered the blood, and are home to lymphocytes and lymphoblasts. This area is an important immune zone. The periarterial zone, located eccentrically in relation to the white pulp, around the central artery, is rich in T lymphocytes, macrophages and interdigital cells. The reticular stroma of the red pulp, sinusoidal blood vessels and the splenic cords located between these sinusoids are enlarged. The rods of the spleen are B lymphocytes and the plasma cells that originate from them. In the reticular stroma of the red pulp, the number of macrophages (derived from monocytes) increases, cells involved in phagocytosis, i.e. "splenocytosis".

Morphological and morphometric parameters of the spleen of a 6-month-old white mongrel rat infected with CRF. When chronic renal failure is experimentally created in white rats, hypoxic processes in the spleen increase and toxins accumulate, resulting in: uneven thickening of the capsule and trabeculae of the spleen, stagnation of blood in the vessels, increased permeability of the blood vessel wall, thickening of the fibrinoid, hyalinosis, and sclerosis-like changes. In the perivascular zone, pronounced edema and sclerotic changes are detected. (Stained pink with Van Gieson paint). The PALS zone has decreased (in size), while the number of mature T-lymphocytes in this area has decreased, and the number of young blast cells (T-lymphoblasts) has increased instead.

The white pulp zone is significantly reduced in volume: the central part of the lymphoid follicle or the proliferation focus - the reactive center - is enlarged,

proliferation of young lymphoblastic cells, blast transformation of T- and B-zone lymphocytes are revealed. The mantle zone and marginal zone are thinned. The number of mature T- and B-lymphocytes has decreased, and the number of macrophages has decreased in parallel. This process indicates that as a result of this, the immune system of the spleen is weakened.

Red pulp zone: It is evident that the sinusoidal spaces of the spleen are narrowed, the process of erythrocyte hemolysis has begun and this process is intensifying. The splenic cords continued to contract, and the number of blast cells increased. We see increased hemolysis in the perisinusoidal area, as well as wrinkling and atrophy of the red pulp. In chronic renal failure in white crossbred rats at the age of 6 months, hypoxic processes in the spleen intensify, which is caused by the accumulation of toxins, disruption of the water-electrolyte and protein balance: the splenic capsule and trabeculae continue to thicken unevenly. The blood vessels are filled with blood, and the changes in their walls, characteristic of hyalinosis and sclerosis, are somewhat enhanced. The area of the PALS is reduced, and the blast cells (T-lymphoblasts) are increased. Mature T-lymphocytes are present in small quantities.

Red pulp zone: narrowing of sinusoidal spaces, increased hemolysis of erythrocytes. In the splenic strips, a decrease in the number of blast cells and an increase in the number of plasma cells is noted. We see increased hemolysis in the perisinusoidal region, as well as wrinkling and atrophy of the red pulp. The white pulp zone is significantly reduced in volume: blast transformation of lymphocytes of the T- and B-zones of lymphoid follicles is revealed. The central part of the lymphoid follicle, or proliferation center, is a reactive center - increased proliferation of young lymphocyte cells. This indicates that the immune system is about to get out of control.

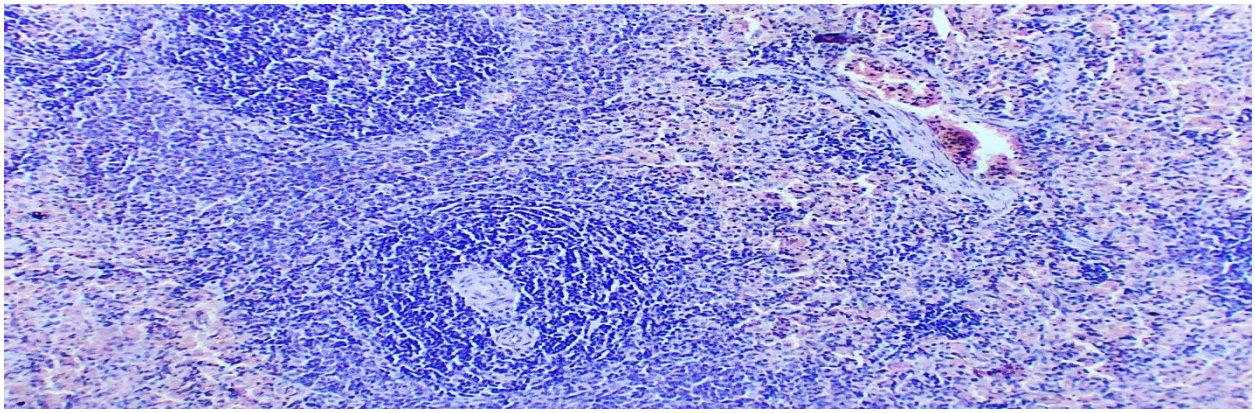

Figure 3. Morphological structure of spleen tissue. The area of white pulp is reduced in size: blast transformation of lymphocytes of T- and B-zones of lymphoid follicles. 2. The mantle and marginal area are reduced. 3. The red pulp shrinks – atrophies. 4. In the PALM area, the number of blast cells has increased. 5. Sclerosis of the vascular wall and perivascular zone..

Analysis of the obtained micropreparations revealed uneven thickening of the capsule surrounding the spleen tissue from the outside; these changes were observed in almost all micropreparations of the spleen. Characteristic changes were observed, such as a decrease in the number of lymphocytes in the white pulp of the spleen and an increase in the amount of blood in the blood vessels of the spleen, i.e. signs of congestion.

CONCLUSION

Macroscopic examination of spleen tissue in 6-month-old rats revealed darkening of the spleen color and rounding of its edges. Microscopically, thickening of the spleen capsule, characteristic changes in the white pulp of the spleen, and an increase in the amount of blood in the blood vessels of the spleen, i.e. signs of edema, were observed. Signs of adhesion of small blood cells to each other and to the wall of blood vessels were found inside the vessels. Analysis of morphometric parameters revealed an increase in the organometric dimensions of the spleen in the group with acute renal failure, namely, an increase in the mass of the spleen by 5% and an increase in the volume of the spleen by 1.7%.

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