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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

Илмий-рефератив, маънавий-маърифий журнал
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MORPHOLOGICAL CHANGES IN THE KIDNEY IN EXPERIMENTAL ULCERATIVE COLITIS

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✓ ***Resume***

Histological analysis of kidney tissue from 9-month-old white rats with ulcerative colitis revealed accumulation of glycosaminoglycans in the mesangial cells and matrix of the glomeruli, thickening of the basement membrane due to the accumulation of acid mucopolysaccharides, changes in the glomeruli as a result of exudative processes, an increase in mucopolysaccharides in the epithelial cells of the tubules, and accumulation of lymphocytes, plasma cells, and macrophages in the interstitial space. Morphological changes in the kidneys in ulcerative colitis may be associated with several factors, including autoimmune reactions and inflammation.

Keywords: kidney, ulcerative colitis, morphology, histochemical analysis, alcian blue stain.

EKSPERIMENTAL YARALI KOLITDA BUYRAKDAGI MORFOLOGIK O‘ZGARISHLAR

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✓ ***Rezyume***

Eksperimentda yarali kolit chaqirilgan 9 oylik oq zotsiz kalamushlar buyrak to’qimasi gistologik tahlil qilinganda glomerulalarning mezangial hujayralari va matriksida glikozaminoglikanlarning to‘planishi, bazal membranada kislota mukopolisaxaridlarining to‘planishi tufayli qalinlashish, glomerulalarda ekssudatsion jarayonlar natijasida o‘zgarishlar paydo bo‘lishi, kanalchalar epiteliy hujayralarida mukopolisaxaridlarning ko‘payishi, to‘qimalar o‘rtasida limfositlar, plazmatik hujayralar va makrofaglarning to‘planishi aniqlandi. Yarali kolitda buyraklardagi morfologik o'zgarishlar bir nechta omillar, jumladan autoimmun reaktsiyalar va yallig'lanish bilan bog'liq bo'lishi mumkin.

Kalit so’zlar: buyrak, yarali kolit, morfologiya, gistokimyoviy tahlil, alsian ko’ki bo’yog’i.

МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ ПОЧЕК ПРИ ЭКСПЕРИМЕНТАЛЬНОМ ЯЗВЕННОМ КОЛИТЕ

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✓ ***Резюме***

При гистологическом анализе ткани почек 9-месячных белых крыс с язвенным колитом выявлено накопление гликозаминогликанов в мезангиальных клетках и матриксе клубочков, утолщение за счет накопления кислых мукополисахаридов в базальной мембране, изменения клубочков. в результате процессов экссудации, канальцевого эпителия увеличение мукополисахаридов в клетках, лимфоцитах, плазматических клетках и макрофагах между тканями было решено накопить. Морфологические изменения почек при язвенном колите могут быть связаны с рядом факторов, в том числе с аутоиммунными реакциями и воспалением.

Ключевые слова: почка, язвенный колит, морфология, гистохимический анализ, краситель альциановый синий.

Relevance

According to the WHO, approximately 1.6 million people worldwide suffer from ulcerative colitis [WHO, 2023]. The maximum prevalence of UC in the world is currently 505/100,000 population (in Europe), and the incidence in different regions ranges from 0.6 to 24.3 per 100,000 population. The highest incidence of UC - 24.3/100,000 - is noted in Europe, 19.2/100,000 - in North America [Shelygin Yu.A., et al., 2023]. The prevalence of UC is higher in northern latitudes and in western regions. The incidence and prevalence of UC in Asia is lower, but is currently increasing. Caucasians suffer from the disease more often than representatives of the Negroid and Mongoloid races. The peak incidence occurs in the age range of 20-30 years, in some countries a second peak of incidence is noted at the age of 60-70 years. The incidence among men and women is approximately the same.

Ulcerative colitis (UC) refers to chronic inflammatory bowel diseases of unknown etiology with damage to the colon and putative multifactorial trigger components with an inadequate immune response in genetically predisposed individuals [1].

The etiology of UC has not been established. The disease develops as a result of a combination of several factors, including genetic predisposition, defects in innate and acquired immunity, disturbances in the intestinal microflora and the influence of environmental factors. About 100 genetic polymorphisms associated with UC have been described. Genetic determination leads to changes in the innate immune response, autophagy, disruption of the mechanisms of recognition of microorganisms, disruption of the epithelial barrier and, as a result, perversion of adaptive immunity. The key defect predisposing to the development of IBD is the impaired recognition of bacterial molecular markers (patterns) by dendritic cells, which leads to hyperactivation of signaling proinflammatory pathways. Also, with IBD, a decrease in the diversity of intestinal microflora is noted due to a decrease in the proportion of anaerobic bacteria, mainly Bacteroidetes and Firmicutes. Against this background, the development of IBD occurs under the influence of trigger factors, which include smoking, nervous stress, vitamin D deficiency, a diet low in dietary fiber and high in animal protein, intestinal infections, especially Clostridioides difficile infection and cytomegalovirus infection. The result of the mutual influence of genetic and predisposing factors is the activation of various subpopulations of T-lymphocytes: T-helpers 1, 2, 17 types and regulatory T-lymphocytes at different stages of inflammation, which leads to hyperexpression of proinflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukins 1, 12, 23, 17 (IL1, IL12, IL23, IL17) and others and cell adhesion molecules. As a result of these disorders, inflammatory lymphoplasmacytic infiltration and destruction of the colon mucosa with macroscopic changes characteristic of UC are formed. When determining the severity of ulcerative colitis in everyday practice, the Truelove-Witts classification is used [Truelove S.C. et al., 1955; Carter M.J. et al., 2004]. According to the activity of the attack, the disease is divided into mild (frequency of bloody stools up to four times a day and no systemic toxicity), moderate (frequency of bloody stools from four to six times a day and minimal signs of intoxication) and severe (frequency of bloody stools more than six times a day with signs of intoxication such as fever, tachycardia, anemia, and increased erythrocyte sedimentation rate [Lau A. et al., 2018].

The aim of the study was to determine the morphological changes in the kidney in experimental ulcerative colitis.

Materials and methods of the study

30 9-month-old white outbred rats of both sexes were selected for the study. All laboratory white outbred rats were combined in one vivarium. The animals were given sufficient water and fed a balanced diet. The importance of proper care and feeding of laboratory animals was taken into account in the preparation and conduct of experimental studies. The feeding regimen and diet were not violated, and hygienic rules were observed during feeding.

The first group of rats (intact) was the control group, and in order to compare the results with the other group, white outbred rats were fed a simple standard diet.

In the second group of our experiment, white outbred rats were induced to ulcerative colitis by rectally injecting 1 ml of 8% acetic acid through a special probe for 20 days. The experimental animals were killed in the morning, on an empty stomach, under ether anesthesia, by means of a momentary decapitation. After opening the abdominal cavity, the kidneys were removed for histological analysis.

Alcian blue staining is a histochemical staining method used to identify acidic mucopolysaccharides, such as acid mucopolysaccharides and glycosaminoglycans. These substances are found in many tissues, including tendons, mucous membranes, and the extracellular matrix of tissues. Alcian blue, when stained in an acidic environment (Ph 2.5 or Ph 1.0), binds to acid mucopolysaccharides through ion exchange and stains them blue.

Alcian blue staining procedure:

First, the tissue was fixed with formalin. This helps to preserve the tissue and strengthen its structure.

To study the tissue, it was passed through a series of alcohols and placed in an alcian blue solution. If staining in an acidic environment is necessary, attention should be paid to the pH value of the solution. The tissue was washed in distilled water or in a suitable solution.

The result of staining: glycogen, neutral mucopolysaccharides - red, acidic mucopolysaccharides - blue, nuclei - blue.

Alcian blue binds to acidic mucopolysaccharides, staining them blue. If these substances are increased, they appear more blue.

Therefore, an increase in acidic mucopolysaccharides as a result of staining in tissues with Alcian blue may indicate pathological conditions, including the diagnosis of mucopolysaccharidoses and other metabolic diseases.

Results of the study

In the control group, the proximal convoluted tubules are composed of a cylindrical epithelium with a clearly defined apical and basal sides, located in a row on the basement membrane. The convoluted tubule lumen is well visible, its shape and diameter depend on the histological section plane. In some cases, individual cellular elements are detected in the lumen of the tubules. When we magnify the epithelial cells, we see that their apical surface has a brush-like part and the cytoplasm is blurred in the basal part.

The epithelium of the distal convoluted tubules consists of cuboidal cells located on the basement membrane and does not have a brush-like part on the apical surface. Due to the absence of a brush-like part and the low height of the epithelium, the distal convoluted tubule lumen is larger in size than the proximal convoluted tubule lumen.

The tubular part of the nephron in histological sections is represented as round or oval-shaped hollow structures, the walls of which are formed by epithelial cells of various shapes located on the basement membrane. The shape of the tubular epithelial cells depends on the type of tubule and its location relative to the renal corpuscle.

The proximal convoluted tubule begins in the lumen of the capsule of the glomerulus and makes several turns near the renal corpuscle. The proximal convoluted tubules are thick-walled and consist of a narrow cavity. The walls of the proximal convoluted tubules are formed by cubic epithelial cells. The surface of these cells is covered with brush-like segments. The nuclei are round, located in the basal part of the cells, and intensively stain with basic dyes (see Fig. 1-2).

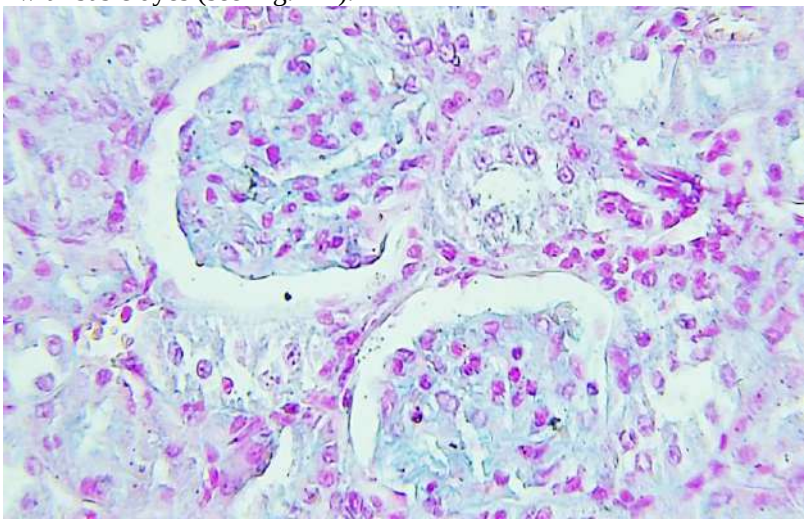

Figure 1. Microscopy of kidney tissue from 9-month-old outbred rats of the control group. Stained with alcian blue (magnified 400 times).

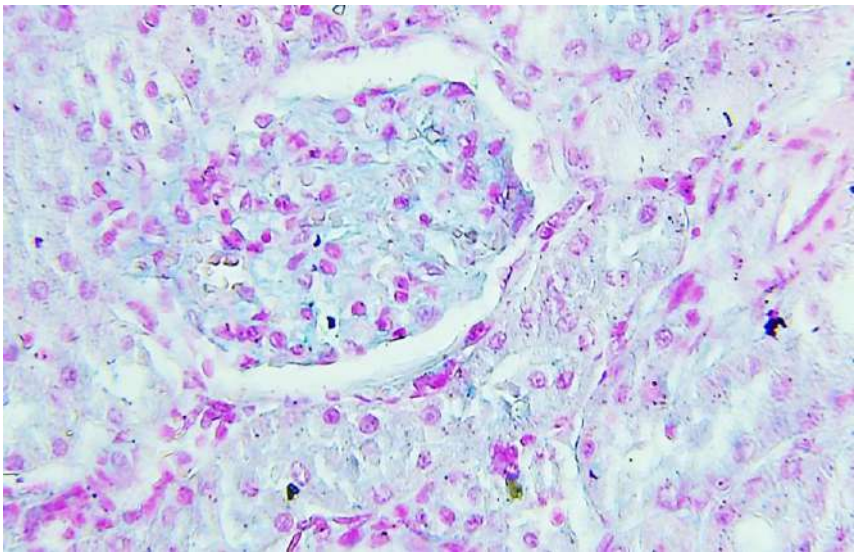

Figure 2. Microscopy of kidney tissue from 9-month-old outbred rats of the control group. Stained with Alcian blue (magnified 400 times).

The following changes were detected during histological analysis of kidney tissue from 9-month-old white outbred rats with ulcerative colitis in the experiment: Pathological changes in glomeruli, tubules, and interstitial tissue can be observed in histological preparations stained with Alcian blue. Alcian blue is used to show acid mucopolysaccharides and glycosaminoglycans. Therefore, the following morphological changes can be observed:

1. Glomeruli:

• Mesangial expansion: The accumulation of glycosaminoglycans in the mesangial cells and matrix of the glomeruli is visible. These areas are stained in blue.

• Basement membrane thickening: The basement membrane thickens due to the accumulation of acid mucopolysaccharides.

• Exudative and fibrotic changes: Changes occur in the glomeruli as a result of exudative processes.

2. Tubules:

• Mucoid degeneration: An increase in mucopolysaccharides is observed in the tubular epithelial cells, which are stained with a blue dye in the cell cytoplasm.

3. Interstitial tissues:

• Interstitial inflammation: An accumulation of lymphocytes, plasma cells, and macrophages is observed between the tissues. (Figure 3-4).

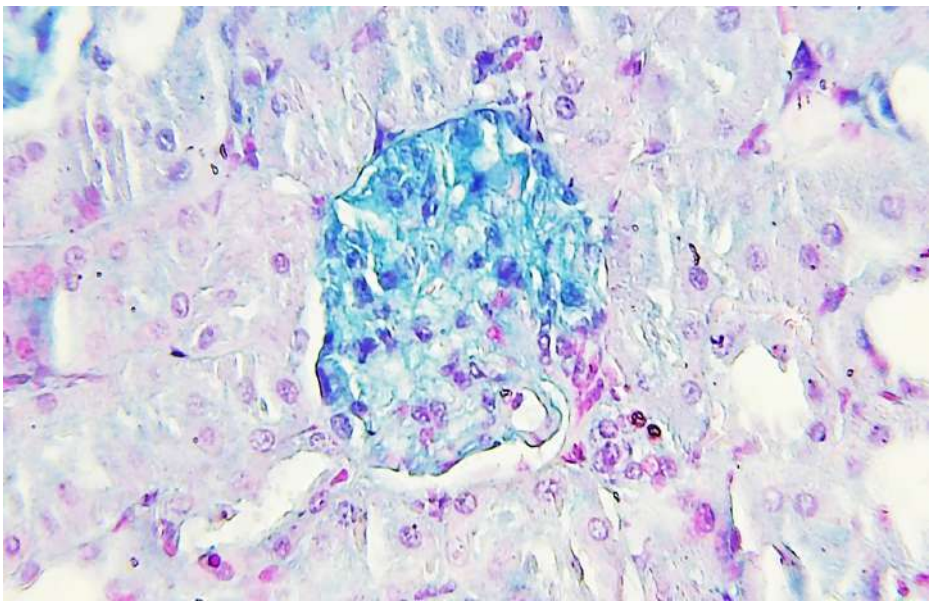

Figure 3. Microscopy of kidney tissue from 9-month-old outbred rats with experimentally induced ulcerative colitis. Stained with Alcian blue (magnified 400 times.)

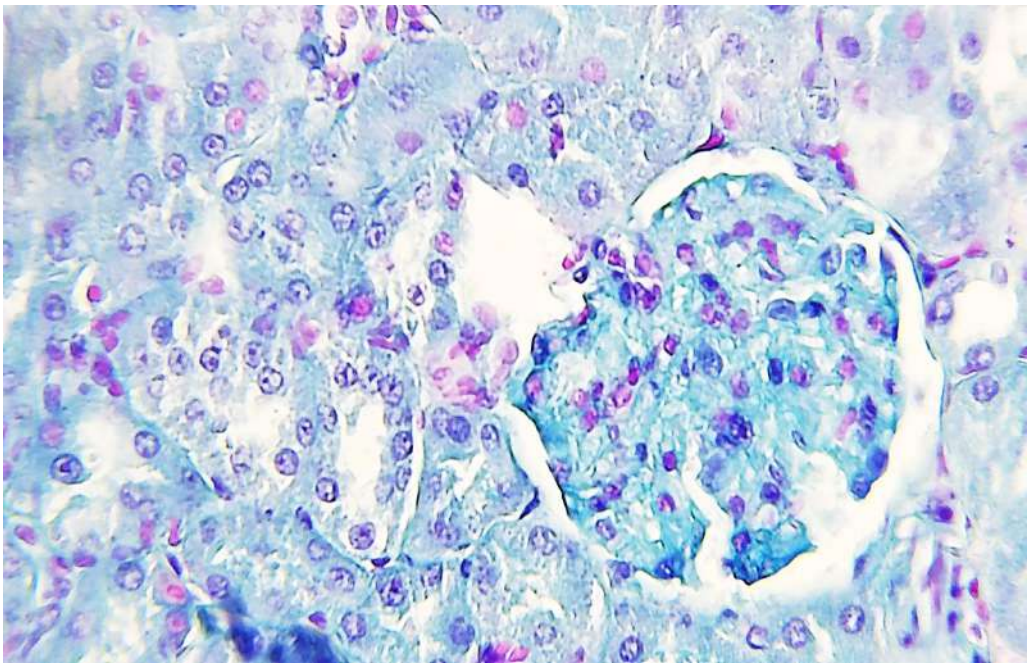

Figure 4. Microscopy of kidney tissue from 9-month-old outbred rats with experimental ulcerative colitis. Stained in alcian blue (magnified 400 times).

Conclusion

Histological analysis of kidney tissue from 9-month-old white rats with ulcerative colitis revealed accumulation of glycosaminoglycans in the mesangial cells and matrix of the glomeruli, thickening of the basement membrane due to the accumulation of acid mucopolysaccharides, changes in the glomeruli as a result of exudative processes, an increase in mucopolysaccharides in the epithelial cells of the tubules, and accumulation of lymphocytes, plasma cells, and macrophages in the interstitial space. Morphological changes in the kidneys in ulcerative colitis may be associated with several factors, including autoimmune reactions and inflammation. Morphological changes in the kidneys in ulcerative colitis are often the result of prolonged inflammation in the body and may also be associated with side effects of treatment. In order to timely diagnose and prevent renal complications, it is necessary to regularly monitor renal function in such patients.

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