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CLINICO-MORPHOLOGICAL CHANGES CHARACTERISTIC IN PATIENTS WITH NON-SPECIFIC ULCERATIVE COLITIS

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Abstract
Spectrum of morphological signs of nonspecific ulcerative colitis is wide and nonspecific. This makes it difficult to compare this disease with some group of diseases (intestinal tuberculosis, sarcoidosis, bacillary colitis, etc.). Therefore, in order to determine nonspecific ulcerative colitis, a more in-depth analysis is required, taking full account of clinical and morphological data.

Keywords: private layer of connective tissue, nonspecific ulcerative colitis, rectomonoscopy.

Currently, nonspecific ulcerative colitis (NUC) is a chronic ulcerative-inflammatory disease of the colon and rectal mucosa of unknown etiology, characterized by reparative processes and the growth of connective tissue. The medical and social importance of the problem of NUC is determined not only by the high incidence rate, but also by the uncertainty of its etiology, the multifactorial nature of its pathogenesis, and the non-specificity of treatment [4, 6, 15]. As we know, today endoscopic diagnosis and treatment methods of various gastrointestinal diseases are widely used in practice. Colonofibroscopy and rectoromonoscopy are the most effective methods for assessing the degree of severity of NUC and allow to obtain a biopsy from the intestine for histological examination. As a result of the histological examination, it is possible to make a comparative diagnosis of the disease of NUC from other diseases. Changes found in colonofibroscopy determine the form of the disease and the stage of the process. In some cases, it is characterized by hyperemia, granulation, porosity, and mucosal damage, while in other cases, it is characterized by loss and erosion of tissues, pseudopolyposis. In severe forms of the disease, a large area of the mucous membrane is damaged, the inner surface of the intestine bleeds, and discharges of pus and blood are observed. Inflammatory reaction in the mucosa is clearly expressed in non-specific ulcerative colitis as well as in infectious colitis caused by bacterial agents.

However, the development of large-scale abscesses and new tissue in the crypts indicates that the inflammatory process has become chronic [2,3, 8].

It is known that ulcerative colitis causes destructive changes in the intestinal walls. As a result of the wound-necrotic process, the absorption of products necessary for life from the intestinal wall is disrupted, endotoxicosis occurs, and local and systemic changes occur in the body. If patients with NUC are not treated properly in time, serious complications may occur, such as profuse bleeding, perforation, toxic dilatation of the large intestine, peri-intestinal cell abscess, malignancy of the process, septicemia, etc. [9]. In young and middle-aged patients, this disease is very severe, and in elderly patients, the disease is relatively mild. But the risk of malignancy increases in those who have been sick for 9-10 years. Therefore, in case of non-specific ulcerative colitis, first of all, it is necessary to make a correct diagnosis in time and prescribe a suitable complex treatment.

The analysis of literary sources shows that with the development of science in recent years, NUC poses a risk of developing a life-threatening colon tumor, causes the emergence of drug-resistant forms of the disease, and causes patients to lose their ability to work. This requires further scientific research to determine the appropriateness of comprehensive examinations and treatment measures aimed at achieving complete clinical remission of non-specific ulcerative colitis.

The purpose of the study. Determining the morphology of the large intestine and the nature of its changes in patients with nonspecific ulcerative colitis.

Research Material and Methods

The clinical examination of patients with CKD was carried out in the gastroenterology department of the 1st clinic of the Tashkent Medical Academy (TMA). 150 NUC patients treated in these departments were taken for the study.

NUC diagnosis was made based on modern descriptions (Khalif I.L., 2006; Belousova E.A., 2006) based on anatomical distribution of the process, endoscopic (colonoscopy) and histological examinations, patient complaints, anamnesis, objective examination and laboratory-instrumental examinations.

In addition to clinical examinations, complex laboratory and instrumental examinations were performed on all patients. According to the nature of the clinical course of the disease: acute (with lightning speed), chronic relapsing and chronic continuous forms; according to development: intermittent, remitting; according to severity: heavy, medium and light forms; according to the spread of the pathological process: total colitis, subtotal colitis and distal colitis or proctitis; according to the

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activity of the inflammatory process (based on rectomanoscopy data) - minimal, limited and maximally high; according to the characteristics of complications: local, systemic.

The following clinical-biochemical and instrumental examinations were included in the examination record: general blood analysis, biochemical blood analysis, rectoscopy, colonoscopy according to the instructions, irrigography, biopsy from the mucous membrane of the large intestine.

The severity of nonspecific ulcerative colitis was determined based on endoscopic (colonoscopy) and histological examinations, taking into account the following criteria: the number of daily defecations, rectal bleeding, according to the Schroeder index (Mayo Clinic, UC DAI).

Based on the obtained data (Table 1), it can be said that among men, the mild and medium severe form of the disease was observed equally (39.7%), and in women more severe form (44.8%) was observed.

Table 1 Distribution of patients with nonspecific ulcerative colitis by clinical form

Clinical forms	Total, n=150		Men, n=83		Women, n=67	
	абс.	%	абс.	%	абс.	%
Light	53	35,3	33	39,7	20	29,8
Medium heavy	63	42	33	39,7	30	44,8
Heavy	34	22,7	17	20,5	17	25,4

As can be seen from Table 2, the most frequent symptom of diarrhea was observed in 83.3% of patients. The occurrence of this symptom increased depending on the severity of the disease, that is, this indicator was 62.3% in the mild form, 100% in the severe form. Bleeding symptoms were severe in 100%. Abdominal pain along the colon was observed in 63.3% of patients, 100% in severe form, 49% in mild form. General weakness occurred in 81.3% of patients, body temperature increase in 22.7%.

Table 2 Distribution of patients with nonspecific ulcerative colitis according to clinical symptoms

Signs	Number of patients n=150		Light form n=53		Medium heavy n=63		Heavy form n=34	
	абс.	%	абс.	%	абс.	%	абс.	%
Diarrhea	125	83,3	33	62,3	58	92	34	100
Bleeding	109	72,7	21	39,6	54	85,7	34	100
Mucous-purulent discharge	16	10,7	11	20,7	5	7,9		–
Abdominal pain	95	63,3	26	49	35	55,5	34	100
General weakness	122	81,3	33	62,3	55	87,3	34	100
An increase in body temperature	34	22,7	2	3,8	10	15,8	22	64,7

All patients with nonspecific ulcerative colitis received anti-inflammatory steroid hormones and amino salicylates, immunomodulators, antispasmodics, and antidiarrheal drugs depending on the severity of the disease.

In a mild form (proctitis): prednisolone 20 mg per day, microclyma with hydrocortisone 125 mg or prednisolone 20 mg 2 times per day, for 7 days. Sulfasalazine 2 g or salazopiridazin 1 g or mesalazine (mezacol, salofalk and other analogs) 1 g was taken per day.

In the moderate form (proctosigmoiditis): prednisolone 40 mg per day, microclyse hydrocortisone 125 mg or prednisolone (20 mg) 2 times per day for 7 days. Sulfasalazine 2000 mg or salazolopyridazine 1000 mg or mesalazine (mezacol, salofalk and other analogues) 1000 mg per day and metronidazole 1000 mg per day were taken for 10-14 days.

In severe form (total): hydrocortisone 125 mg intravenously 4 times a day or prednisolone 30 mg intravenously 4 times a day for 5 days. Hydrocortisone 125 mg or prednisolone 20 mg rectally or drip (the drug was dissolved in a 0.9% solution of sodium chloride) was administered 2 times a day for 5 days.

In this scientific study, biopsies taken from the mucous membrane of the large intestine were morphologically studied in order to study the condition of the mucous membrane of the large intestine in patients with NUC.

Private inspection results. Colon. The primary inflammatory-ulcerative process begins in the rectum and sigmoid colon, and then spreads to the proximal parts of the gastrointestinal tract. Taking this situation into account, 26 NUC patients were taken and morphologically examined by taking biopsies from the mucosa of the large intestine during colonoscopic examination.

The results of microscopic examination of biopsies taken from colon cancer patients showed that pathomorphological changes differ depending on the level and stage of NUC activity. In the initial stage of the disease, swelling and redness with small hemorrhages were observed on the mucous membrane. The surface epithelium becomes necrotic in some places, forming ulcerated defects and is disorganized. The amount of goblet cells in the covering epithelium around the wound is reduced, the private connective tissue layer is strongly swollen and infiltrated with lymphocytes, plasma cells, neutrophils, and eosinophils (Fig. 1).

Histochemical examination revealed an increase in the amount of glycogen in the apical part of the crypts in the form of intense SHIK-positive material (Fig. 2). In the acute period of NUC, complete destruction of the crypts, pathological regeneration of the covering epithelium in the form of metaplasia and dysplasia, and proliferative infiltration and sclerosing of the submucosa are observed.

Signs of reparation in the form of nonspecific proliferative infiltration of cells of lymphohistiocytic origin are characteristic of chronic transition of nonspecific ulcerative colitis to chronic ulcerative process in the large intestine (Fig. 3). In this case, the strongest proliferation takes place in the injured area, which is a condition for the development of secondary infection and tissue autoimmunity, due to the loss of epithelial protection of the intestinal wall. In this regard, the immune response in chronic non-specific ulcerative colitis is characterized by the development of immunopathological inflammation with the predominance of plasmacytic infiltration. This process is always accompanied by a reaction in the form of hyperplasia of all morphofunctional areas of lymphoid follicles in the intestinal wall and the appearance of a germinal center.

It was found that in nonspecific ulcerative colitis, the inflammatory process is limited to the colon, that is, the smoothing of the crypts, blood filling, and many small ulcers are only in the mucosa of the colon. Only in some cases, large wounds with uneven edges are found. A pronounced proliferative inflammatory reaction of the submucosa is accompanied by necrosis, desquamation of the covering epithelium, the formation of ulcers, a decrease in goblet cells around them, and the presence of crypt-abscesses. In the histochemical examination, it is determined that the positive substance of SHIK is uneven in the crypts of the large intestine (Fig. 4), which indicates a decrease in the number of goblet cells. In this case, leukodiadesis in the surface epithelium, migration of leukocytes between intestinal gland crypts, their distal part closing and crypt abscesses appear. Violation of the epithelium of the wall of crypt-abscesses leads to the formation of small ulcerative defects.

Changes in the submucosa are slightly pronounced, except when the wound penetrates the submucosa. In the initial stage of nonspecific ulcerative colitis, lymphoid infiltration is mainly limited to the mucous membrane and submucosal surface layer. In atrophy of crypt-abscesses, the submucosal layer thickens due to obvious inflammatory infiltration of lymphohistiocytic cells (Fig. 5). When the disease becomes continuous, the mucous membrane atrophies, reactive epithelial hyperplasia develops, and the number of goblet cells with a small amount of glycogen content decreases (Fig. 6). Diffuse lymphoid and plasma cell infiltration is detected in the submucosa (Fig. 7).

In the long course of non-specific ulcerative colitis, all stages of the destructive ulcer and inflammatory process are manifested in the mucous and submucous layers of the large intestine. Therefore, the mucous membrane is not completely restored. In patients, only in the period of clinical remission, the activity of non-specific ulcerative colitis decreases, atrophy of the mucous membrane, the formation of crypt-abscesses,

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lymphoid and plasmatic infiltration of the submucosal layer are detected microscopically. In this case, the unevenness of the positive substance of SHIK is determined by the crypt-epithelium (Fig. 8).

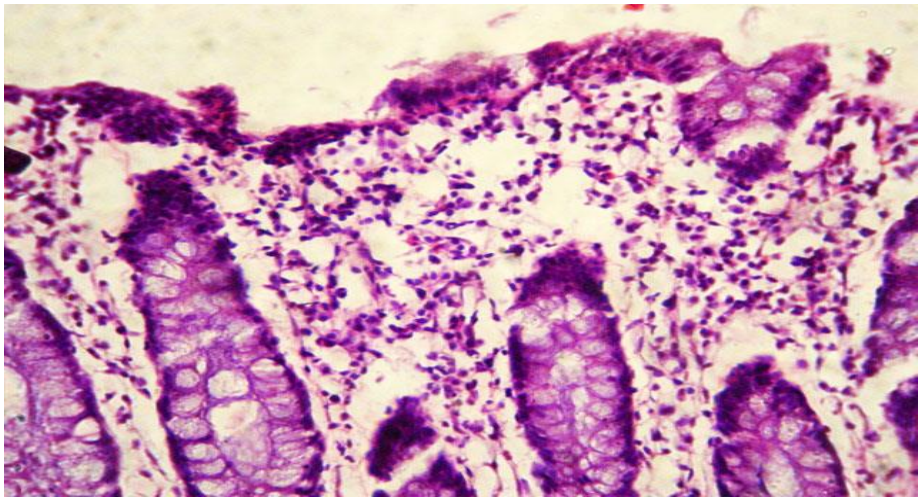

Figure 1. Photomicrograph of histopreparation No. 21. Patient Yu.F. No. 8658 tar. Erosion and erosion of the covering epithelium, swelling of the private layer and lymphoid infiltration. Staining: hematoxylin and eosin. X: ok. 10, ob. 40.

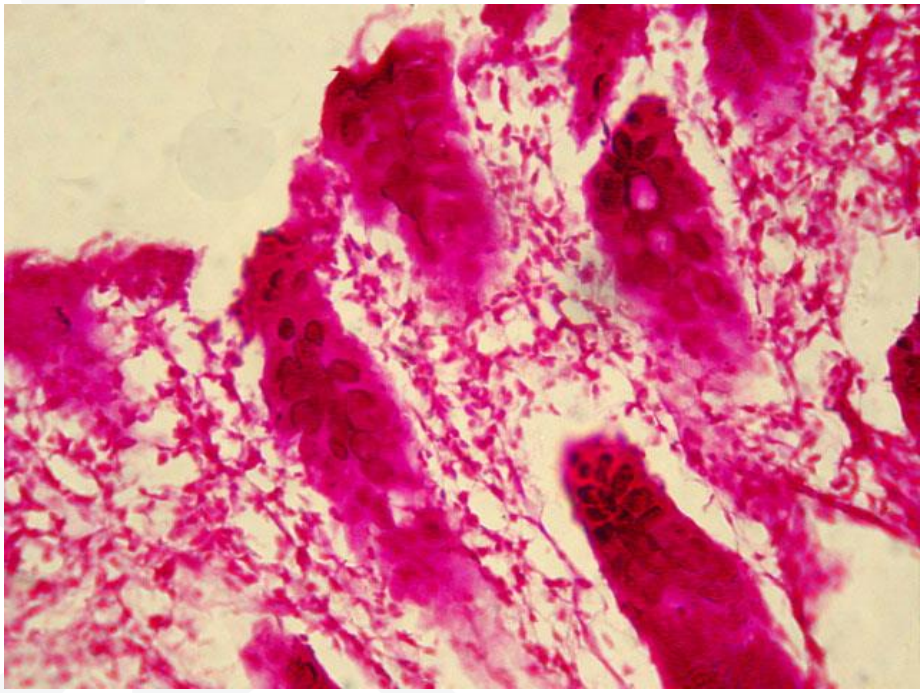

Figure 2. Photomicrograph of histopreparation No. 21. Patient Yu.F. No. 8658 tar. Abundance of glycogen in the apical part of the crypts. Staining: SHIK reaction. X: ok. 10, ob. 40.

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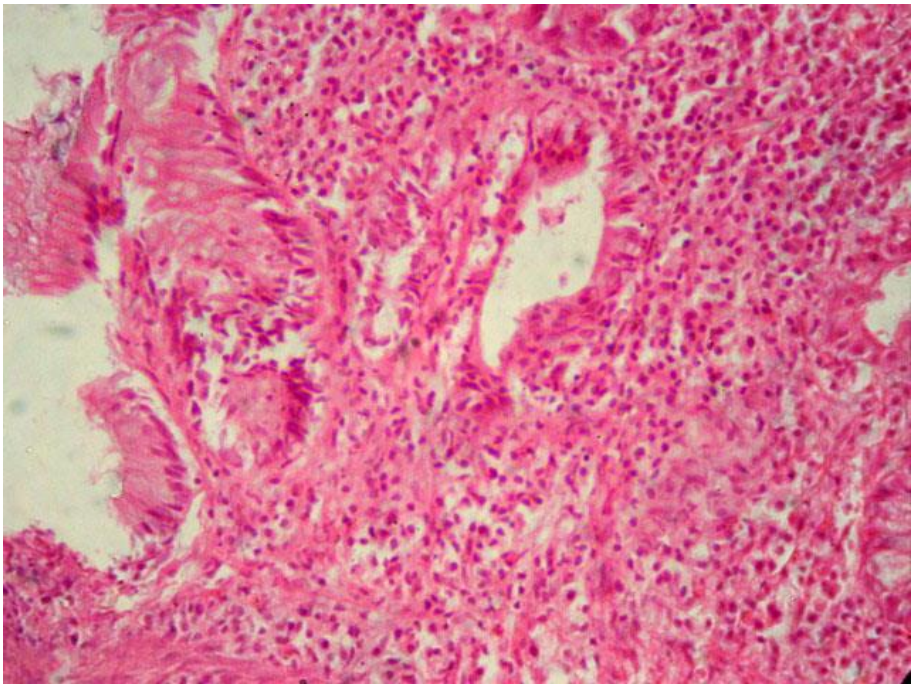

Figure 3. Photomicrograph of histopreparation No. 28. Patient T.S. No. 4494 tar. Clearly expressed lymphoid and plasmacytic infiltration in the wound area. Staining: hematoxylin and eosin. X: ok. 10, ob. 40.

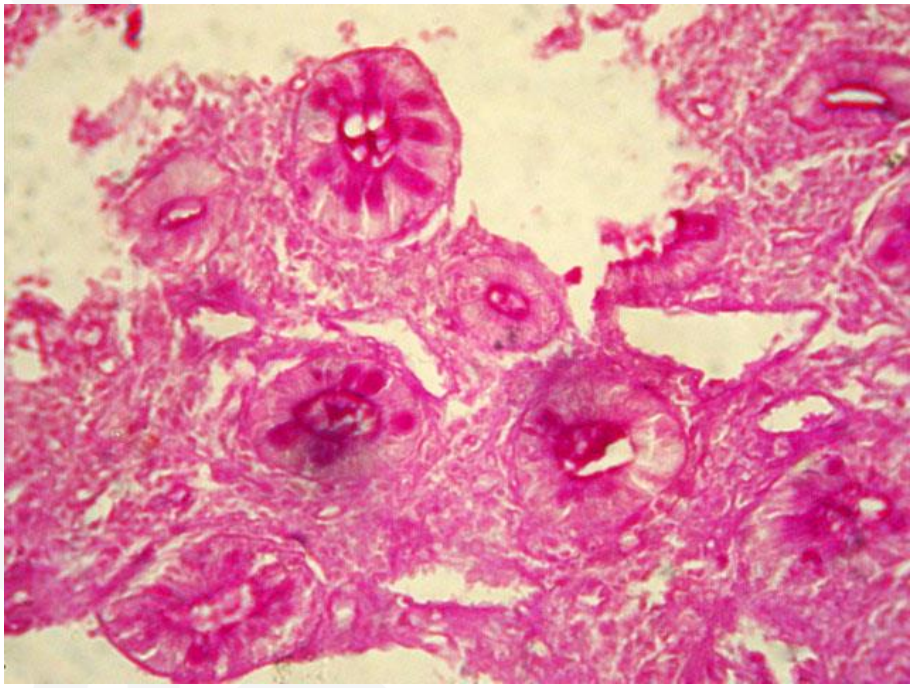

Figure 4. Photomicrograph of histopreparation No. 28. Patient T.S. No. 4494 tar. Decreased glycogen content in the crypt epithelium.
Staining: SHIK reaction. X: ok. 10, ob. 40.

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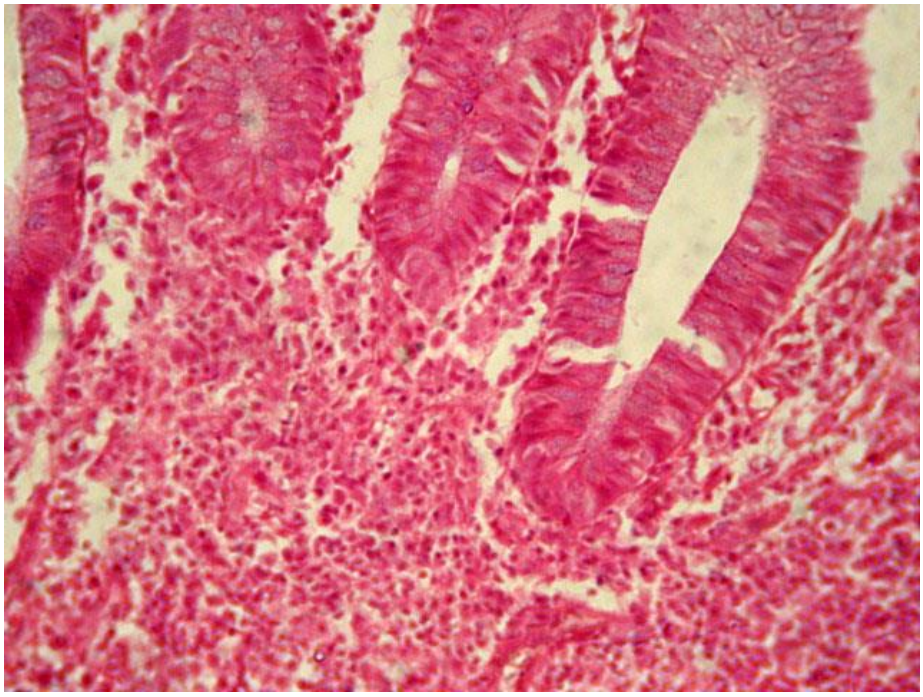

Figure 5. Photomicrograph of histopreparation No. 88. Patient I.A. #3026 ca. tar. Lymphoid and plasmacytic of the private layer of SB infiltration. Staining: hematoxylin and eosin. X: ok. 10, ob. 40.

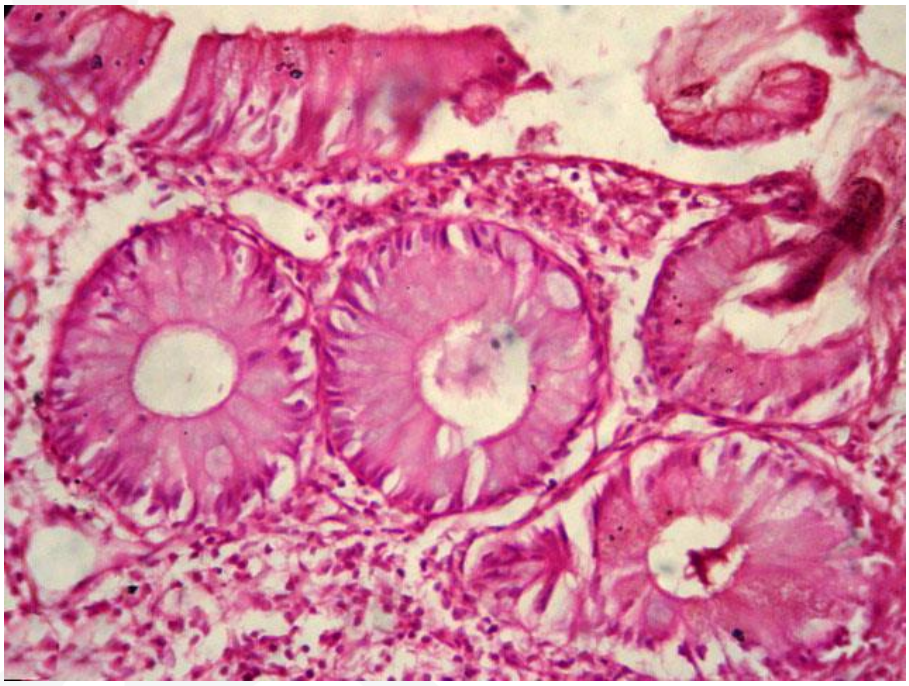

Figure 6. Photomicrograph of histopreparation No. 88. Patient I.A. No. 3026 tar. Decrease in the amount of positive substance of ШИК in the crypts. Staining: SHIK reaction. X: ok. 10, ob. 40.

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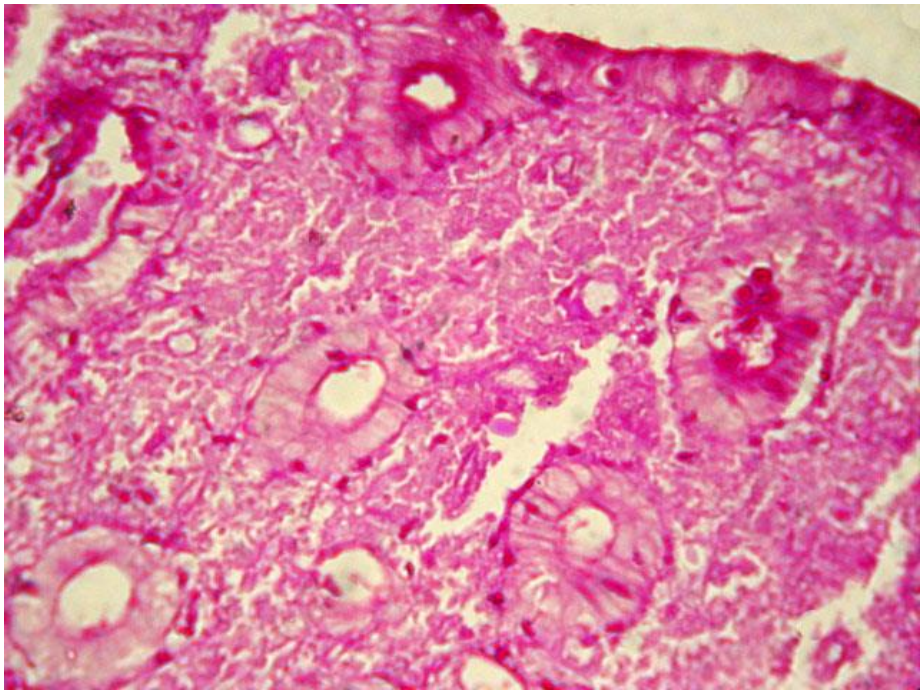

Figure 7. Photomicrograph of histopreparation No. 84. Patient A.Z. No. 3890 tar. Disappearance of the positive substance in the crypts, storage on the surface. Staining: SHIK reaction. X: ok. 10, ob. 40.

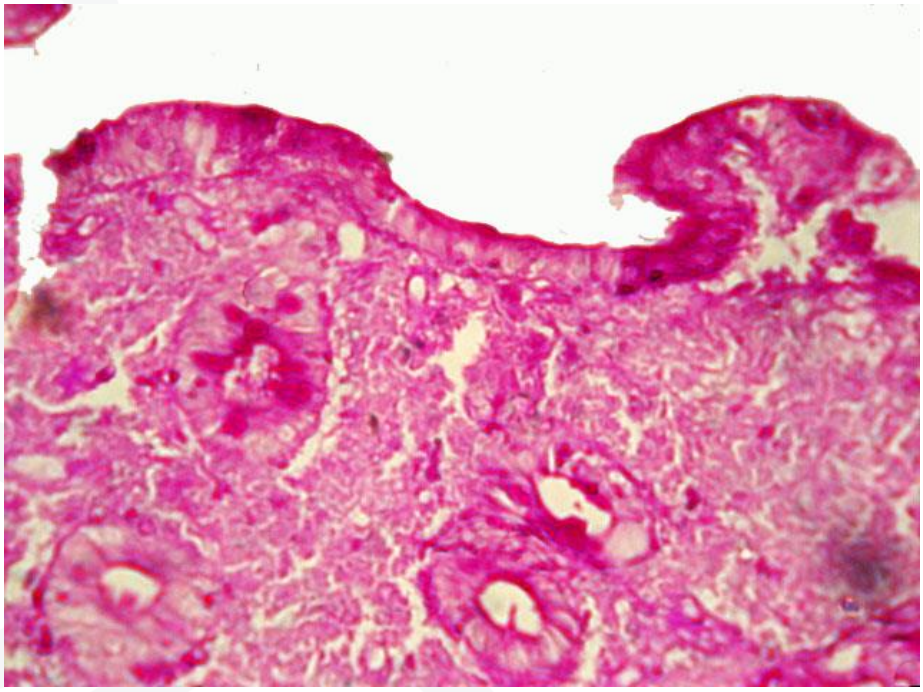

Figure 8. Photomicrograph of histopreparation #76. Patient K.A. No. 8202 tar. Irregularity of the amount of positive substance in the crypts. Staining: SHIK reaction. X: ok. 10, ob. 40.

Non-specific ulcerative colitis is characterized by erosive-ulcerative, granulosa-polyposis, catarrhal-purulent-bloody pathomorphological changes, which are based on dystrophic-necrobiotic, atrophic-hyperplastic changes on the part of the epithelium, and lymphoid-plasmacytic infiltration in the submucosa.

Thus, NUC is a polyetiological disease, in the pathogenesis, morphogenesis, and formation of pathomorphological signs, increased immune reactivity, autoimmunization, metabolic processes, nerve trophic disorders, and the development of secondary infection are important. Basically, dystrophic-necrobiotic, atrophic-hyperplastic by the epithelium, erosive wound with lymphoid-plasmocytic infiltration by the submucosal layer, granulosis-polyposis, catarrhal-purulent-hemorrhagic changes are typical pathomorphological changes for non-specific ulcerative colitis.

Адабиётлар

1. Белятко, Е.А. Инфликсимаб в лечении неспецифического язвенного колита: случаи клинического течения заболевания после родов / Е.А. Белятко, О.С. Шифрин // Доказательная гастроэнтерология.- 2012.-№2,-С. 83-88.
2. Caprioli, F. Disruption of inflammatory signals by cytokine-targeted therapies for inflammatory bowel diseases / F. Caprioli, R. Caruso, M. Sarra, F. Pallone, G. Monteleone // British Journal of Pharmacology.-№ 165 (2012), 4,- P. 820-828.
3. Измайлова, А.С. Диетотерапия при неспецифическом язвенном колите / А.С. Измайлова // Бюллетень медицинских интернет-конференций,- 2013.- Т 3,- № 3,- С. 711-711.
4. Ливзан, М.А. Неспецифический язвенный колит: генетика иммунного ответа / М.А. Ливзан, М.А. Макейкина // Клинические перспективы гастроэнтерологии, гепатологии,- 2012,- № 1.- С. 28-33.
5. Маркова, А.А. Современные методы диагностики и оценки тяжести течения неспецифического язвенного колита / А.А. Маркова, Е.И. Кашкина // Вестник Тамбовского университета. Серия: Естественные и технические науки.- 2012.- Т. 17.- № 3.- С. 915-919.
6. Симаненков, В.И. Язвенный колит: место пробиотической терапии / В.И. Симаненков, О.И. Соловьева, З.Р. Сундукова // Вестник Санкт-Петербургской академии последипломного образования,- 2012.- Т. 4,- № 3,- С. 29-36.
7. Сокуренко, С.И. Клиническое наблюдение пациента с преобладанием дерматологических проявлений при неспецифическом язвенном колите / С.И.

Сокуренко, Т.В. Борисова, М.Е. Семендяева, И.Ф. Бакулина // Клиническая практика,- 2012.- № 3.- С. 36-41.

8. Ruemelle F.M., et al. Consensus guidelines of ECCO/ESPGHAN on the medical management of pediatric Crohn's disease. – J. Crohn's Colitis, 2014, v. 8, 1179-1207. doi:10.1016/j.crohns.2014.04.005.

9. Тимербулатов, М.В. Возможности эндоскопической хирургии язвенного колита / М.В. Тимербулатов, А.А. Ибатуллин, Ф.М. Гайнутдинов, А.В. Куляпин, J1.Р. Аитова // Эндоскопическая хирургия.-2013,-№ l.-вып. №2.-С. 303.

10.Turner D, Levine A, Escher JC et al. Management of Pediatric Ulcerative Colitis: Joint ECCO and ESPGHAN Evidence-based Consensus Guidelines. J. Pediatr. Gastroenterol. Nutr. 2012; 55: 340–361.

11.Ткачев, А.В. Воспалительные заболевания кишечника: на перекрестке проблем / А.В. Ткачев, Л.С. Мкртчян, К.Е. Никитина, Е.И. Волынская // Практическая медицина,- 2012.- № 58.- С. 17-22.

12.Воспалительные заболевания кишечника. Клиническое руководство. / Штайн Д.Д., ред., ГЭОТАР-МЕД. 2017 – 256 с.

13.Levine A., de Bie C. L., Turner D. et al. Atypical disease phenotypes in paediatric ulcerative colitis: 5-year analyses of the EUROKIDS registry. Inflamm. Bowel Dis. 2013 Feb; 19(2):370-7.

14.Levine A., Koletzko S., Turner D., et al. ESPGHAN revised Porto criteria for the diagnosis of inflammatory bowel disease in children and adolescents – J.Pediatric Gastroenterol Nutr., 2014, v.58 (6), p.795-806.

15.Корниенко Е.А. Воспалительные заболевания кишечника у детей. – М., Прима Принт, 2014, 208 с. 62.

16. Кузнецова, О.В. Федорова, О.А. Тутина, О.В. Шумилова // Колопроктология.- 2012.- № 2.- С. 21-29.

17. Корниенко Е.А. Воспалительные заболевания кишечника у детей. – М., Прима Принт, 2014, 208 с. 62.