ISSN: 2692-5206
AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ON THE ISSUES OF DIAGNOSIS AND TREATMENT OF ULCERATIVE COLITIS

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Abstract. Non-specific ulcerative colitis (NUC) is an inflammatory disease of the colon, characterized by ulcerative-destructive changes in its mucous membrane, chronic course and relapses. The highest incidence rates of non-specific ulcerative colitis are observed in North America, Great Britain, Europe and Australia. At the same time, the number of patients with UC in different regions of the world varies from 28 to 117 patients per 100 thousand people. Epidemiological studies in the USA have shown that UC occurs 3-5 times more often in the white population than in African Americans, and 3.5 times more often in Jews than in non-Jewish people [1, 2, 3].

Key words: non-specific ulcerative colitis, diagnostics, treatment, invasiveness, radiation exposure, genetic factors, neuropsychiatric disorders, lifestyle.

INTRODUCTION.

One of the serious complications of UC is colorectal cancer (CRC). Epidemiological analysis has shown that the number of patients with this disease increases every year. Thus, if in 2012 there were 1.4 million patients with CRC registered in the world, then in 2020 1.9 million patients were identified, and the number of deaths from it was 700 thousand in 2012, in 2020 - 900 thousand cases [1, 2, 3].

Belousova E.A. notes that the problem of diagnostics and treatment of non-specific ulcerative colitis (UC) and Crohn's disease (CD) remains one of the most difficult in gastroenterological practice to date. Endoscopic, radiological and morphological research methods are used to diagnose these diseases. However, they have a number of disadvantages: invasiveness, radiation exposure, the need for the introduction of contrast agents, the presence of certain contraindications for use. Despite the research conducted in this area, the etiology and pathogenesis of this disease remain poorly understood. Significant factors in the genesis of UC are the use of non-steroidal anti-inflammatory drugs, oral contraceptives, high hygiene levels in childhood, lactose intolerance, consumption of large amounts of refined carbohydrates, the role of artificial feeding and psychological stress is discussed. Ordás I. and a number of other scientists agree that genetic, immunological and bacterial mechanisms are involved in the pathogenesis of UC.

Research conducted by I. L. Khalif showed that the highest mortality rates are observed during the first year of the disease, which is due to the extremely severe fulminant course of the disease. About 15% of patients with UC have a risk of a severe attack, and 10% of disease debuts initially occur in the form of fulminant colitis. The severity of UC disease can be assessed using the criteria of Truelove and Witts (1955), which were supplemented by M.H. Levitan (Table 1).

Signs	Mild	Average	Severe

ISSN: 2692-5206
AMERICAN ACADEMIC PUBLISHER
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Stool frequency	< 4 times a day	> 4 times a day	> 6 times a day
Rectal bleeding	Insignificant	Severe	Sharply expressed
Temperature	Normal	< 37.8°C	> 37.8°C for 2 days out of 4
Pulse rate	Normal	< 90 per min	> 90 per min
Hemoglobin, g/l	More than 111	105—111	Less than 105
ESR, mm/h	Less than 20	20—30	More than 30

The Mayo index is also used to determine the severity of UC. The Mayo index = stool frequency + presence of rectal bleeding + endoscopic examination data + general physician's opinion.

According to the Montreal classification, patients with UC are divided into:

- E1 or proctitis (a disease limited to the rectum);

- E2 or left-sided disease (distal to the splenic flexure);

- E3 or extensive colitis (the disease extends proximal to the splenic flexure).

The etiology and pathogenesis of UC have not been fully studied. According to various authors, autoimmune changes, dysbiotic shifts, genetic factors, neuropsychiatric disorders, lifestyle and diet are important in the pathogenesis of the disease.

Histological examination of the mucous membrane of the walls of various parts of the colon revealed the following pathological changes: hyperemia, edema, ulceration. The depth of ulcers may vary depending on the severity - from superficial erosions to deep ulcers that affect the entire thickness of the muscular layer of the intestinal wall. Microscopic changes are characterized by infiltration of the proper plate of the mucous membrane with plasma cells, eosinophils, lymphocytes, mast cells and neutrophils. The inflammatory process can spread to all layers of the intestinal wall, affecting the muscular layer and serous membrane, and in the chronic course transforming the intestine into a degenerative tube without taenia and haustra. With total acute and especially lightning-fast development of the disease, toxic damage to the muscular layers and nerve plexuses of the intestinal walls develops with still relatively small degenerative changes in the tissue of the deep layers of the intestinal wall. As a result, the colon loses tone, sharply expands, its walls become thinner, an extremely dangerous condition occurs - toxic dilation of the colon [5]. Clinically, these changes are expressed in the form of three syndromes associated with intestinal damage: stool disorders, hemorrhagic and pain syndromes.

Along with intestinal symptoms, extraintestinal manifestations are also observed, such as erythema nodosum and gangrenous pyoderma, aphthous stomatitis, eye damage (episcleritis, uveitis, conjunctivitis, keratitis, retrobulbar neuritis, choroiditis), inflammatory joint lesions (sacroiliitis, arthritis, ankylosing spondylitis), etc.

ISSN: 2692-5206
AMERICAN ACADEMIC PUBLISHER
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MATERIAL AND METHODS.

During the study, a study of domestic and international literature devoted to the study of the etiology, pathogenesis, diagnosis and treatment of nonspecific ulcerative colitis was conducted.

A comparative analysis of the effectiveness of various diagnostic methods, including instrumental, laboratory, histomorphological diagnostics of ulcerative colitis, taking into account the stage and characteristics of the course of this disease. The international experience of conservative and surgical treatment, their effectiveness, as well as modern methods of treatment and achievements in this field were studied.

RESULTS AND DISCUSSION.

Today, there are many methods for diagnosing nonspecific ulcerative colitis. The diagnosis of ulcerative colitis can be definitively established only on the basis of a general interpretation of clinical manifestations, laboratory tests, as well as endoscopic, histological and radiological data.

The main one is considered to be an endoscopic examination (colonoscopy) with biopsy, which allows confirming the diagnosis, assessing the degree of activity of the inflammatory process, establishing the extent of the process, monitoring the effectiveness of treatment. UC is characterized by the absence of a vascular pattern, granularity, hyperemia and edema of the mucous membrane, the presence of contact bleeding and / or erosions and ulcers. Histological examination of biopsy specimens is carried out to confirm the diagnosis: signs of nonspecific immune inflammation are revealed, which, however, are not pathognomonic for UC. However, in severe exacerbations, colonoscopy is not always possible due to the risk of complications [6].

Another diagnostic method is X-ray examination (irrigoscopy, irrigography), which allows to establish the extent of the process by characteristic signs: smoothing or absence of haustra (the "water pipe" symptom), shortening of the colon; it is possible to detect barium depots corresponding to ulcerative defects, pseudopolyps, strictures.

During an attack of UC, changes in hematological parameters are noted: leukocytosis, an increase in the content of neutrophils (especially band neutrophils), an increase in the erythrocyte sedimentation rate (ESR), anemia. The number of leukocytes increases in the acute phase of inflammation, but can change against the background of taking such drugs as azathioprine and glucocorticosteroids. ESR largely depends on the size and number of erythrocytes, as well as on other factors, including age, gender and pregnancy. Platelets also play an active role in the inflammatory response and are considered an important indicator of UC and Crohn's disease (CD) activity. The mean platelet volume has been proposed as a potential marker of clinical activity activity of the disease, it is inversely proportional to the level of C-reactive protein (CRP) and ESR [7].

Nonspecific ulcerative colitis has some features, one of which is that the infiltrate is mainly localized in the proper plate of the mucous membrane and rarely spreads to the submucosa. In this case, the epithelium of the crypts and their lumen are infiltrated with neutrophils so

ISSN: 2692-5206
AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

much that crypt abscesses are formed. After opening the crypt abscess, a small ulcer appears in the intestinal lumen. When several adjacent crypt abscesses merge, wide flat ulcers with undermined edges are formed.

However, in severe fulminant course of the disease, the infiltrate can be transmural, spreading to all layers of the intestinal wall, while the intestinal walls become sharply thinner, the mucous membrane disappears completely, the intestinal-muscular nerve plexus is destroyed. All this contributes to the development of toxic dilation, and then perforation of the colon.

In the case of a protracted, chronic course of ulcerative colitis, the characteristic histological signs are: atrophy of the mucous membrane with deformation of the crypts, reactive epithelial hyperplasia, an increase in the number of goblet cells, the appearance of an infiltrate consisting of plasma cells, lymphocytes and eosinophils, sclerosis of the muscular plate of the mucous membrane, inflammatory polyps and metaplasia of Paneth cells are more often found. During the period of remission of nonspecific ulcerative colitis, a large number of lymphoid follicles are noted in the basal parts of the mucous membrane, sclerosis and lipomatosis of the submucosa.

The traditional method of conservative treatment of nonspecific ulcerative colitis is treatment with 5-aminosalicylates, corticosteroids and immunosuppressants that cause and maintain remission. Since 2013, biologic therapy with anti-tumor necrosis factor alpha agent has been used to treat ulcerative colitis. Takeda Pharmaceutical Co. and its U.S. subsidiary (Takeda Pharmaceuticals) announced that the U.S. FDA has granted their application for registration of the biologic drug vedolizumab for the treatment of adults with moderate to severe active ulcerative colitis.

If conservative treatment is ineffective, surgical treatment is used, which includes removal of various parts of the colon or complete removal of the colon (colectomy).

CONCLUSION

In general, ulcerative colitis is a disease that occurs at the intersection of genetic predisposition, exposure to certain environmental factors, changes in intestinal microflora and shifts in the autoimmune system. These factors alone are not sufficient for the development of the disease. The prevalence of inflammatory bowel diseases is increasing in both economically developed and developing countries. Taking this into account, it is necessary to timely identify groups of people subject to in-depth instrumental and laboratory examination. As a result of the studies with a large number of patients with ulcerative colitis, it was possible to obtain information on the role of currently available biomarkers in determining the activity of the disease, in the implementation of differential diagnostics of intestinal pathology, predicting the therapeutic effect and early detection of relapse. The issue of including biomarkers in the examination protocols of patients with ulcerative colitis requires further study. The results of these studies will contribute to improving the early diagnosis of nonspecific ulcerative colitis and increasing the effectiveness of treatment of inflammatory bowel diseases.

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ISSN: 2692-5206
AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

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