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CLINICAL APPROACH TO THE FEATURES OF THE COURSE OF NONSPECIFIC ULCERATIVE COLITIS

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Abstract. Inflammatory bowel disease, which includes ulcerative colitis, remains one of the serious problems of the modern clinic of internal diseases. Inflammatory bowel disease is a systemic disease with an extremely diverse clinical picture of intestinal damage and a wide range of possible extraintestinal manifestations, which makes initial diagnosis extremely difficult. Often, intestinal symptoms of the disease may be absent or subclinical. This paper presents an overview of studies on extraintestinal complications. Extraintestinal lesions that occur in inflammatory bowel disease are very diverse. It is very important to remember that often extraintestinal lesions manifest earlier than intestinal symptoms. The correct interpretation of extraintestinal symptoms contributes to the diagnosis and the appointment of timely adequate therapy.

Keywords: ulcerative colitis, inflammatory bowel disease, extraintestinal manifestations, liver.

Currently, there is no clear information about the etiology and pathogenesis of nonspecific ulcerative colitis disease [NUC]. In the pathogenesis of the disease, the importance of changes in immunological reactivity, dysbiotic changes, allergic reactions, genetic factors, neuropsychiatric disorders is assumed. The variety of causative factors in their relationship can explain the variability of the clinical course and morphological manifestations of UC associated with impaired immunogenesis [12,13], death and regeneration of the epithelium [14], neuroendocrine cells [15], the prevalence of inflammation of the colon [16]. NUC is one of the diffuse chronic recurrent diseases of the colon, in which the mucous membrane of this hollow organ is predominantly affected. This disease occurs in all countries of the world [1]. From 3 to 15 new observations per 100,000 population are diagnosed per year, and the incidence reaches 50–80. Men and women get sick equally often. The first peak in

the detection of UC occurs at the age of 20 to 40 years, the second peak is 60–70 years [6]. The disease is most common in the Nordic countries, USA and Canada. In Russia, the incidence of ulcerative colitis is 2-3 cases per 100 thousand inhabitants, which is significantly lower than in other European countries and in the United States. In connection with the improvement of diagnostic capabilities, a significant increase in the frequency of registration of UC[2] has recently been noted.

The most frequently considered hereditary predisposition to the development of an autoimmune inflammatory process in the mucous membrane of the colon in response to the contamination of its surface with microorganisms and viruses, as well as the contact effects of food. This opinion is based on the frequent combination of UC with other autoimmune processes [7].

The longstanding view of ulcerative colitis as an autoimmune disease has recently been given a new boost by the evidence that the commensal microflora and its waste products serve as self-antigens, and that ulcerative colitis develops through a loss of tolerance to substances in the normal intestinal flora that are normally harmless. The detection of IgG antibodies to epithelial cells and p-ANCA in the colon mucosa only strengthened the position of supporters of this The performed studies to determine the ratio of T-lymphocytes in the mucosa of this intestine indicate a violation of immunoregulatory interactions of activated CD4- and CD8-lymphocytes. The synthesis of human monoclonal antibodies to the pANCA antigen has helped identify cross-reactive antigens, including: the cytoplasmic antigen of mast cells located in the colonic mucosa, ocular antigens of the ciliary body and retinal ganglion cells, and, more significantly, proteins of commensal bacteria such as Bacteroides sassae and E. coli [10]. hypothesis [9]. The performed studies to determine the ratio of T-lymphocytes in the mucosa of this intestine indicate a violation of immunoregulatory interactions of activated CD4- and CD8-lymphocytes. The long-held view of ulcerative colitis as an autoimmune disease has recently been given a new boost by the evidence that commensal microbiota and their waste products serve as self-antigens, and that ulcerative colitis develops through a loss of tolerance to substances in the normal intestinal flora that are normally harmless. The

detection of IgG antibodies to epithelial cells and p-ANCA in the colon mucosa only strengthened the position of supporters of this hypothesis [9].

There is a genetic predisposition to UC [familial cases of ulcerative colitis] and an association of UC with HLA histocompatibility complex antigens [3]. Among the closest relatives, UC occurs 15 times more often than in the general population. A family predisposition to UC has also been identified, while first-line relatives get sick much more often than the average in the population, and the use of oral contraceptives is clearly seen as risk factors, as well as same eating habits and psychosocial problems [6,8]

Morphologically, inflammation of various parts of the colon is determined. The mucous membrane is hyperemic, edematous, ulcerated; ulcers of a rounded shape, various sizes. Microscopic changes are characterized by infiltration of the lamina propria by plasma cells, eosinophils, lymphocytes, mast cells, and neutrophils.

Clinical picture. In the clinical picture, there are three leading syndromes associated with intestinal damage: stool disorders, hemorrhagic and pain syndromes [1,4]. The onset of the disease may be acute or gradual.

The main symptom is multiple [in severe cases up to 20 times a day] watery stools mixed with blood, pus and mucus in combination with tenesmus and false urge to defecate. Often, only bloody mucus is excreted when the urge to defecate. Diarrhea is most pronounced when the right half of the large intestine is affected, where water and electrolytes are absorbed. In the case of the spread of the inflammatory process in the proximal direction to a large part of the colon, the disease is accompanied by significant bleeding.

In the initial period of the disease, which occurs in the form of proctosigmoiditis, constipation may occur, mainly due to spasm of the sigmoid colon. During remission, diarrhea may completely stop.

• Pain in the abdomen - usually aching, less often - cramping. Localization of pain depends on the extent of the pathological process. Most often, this is the area of the sigmoid, colon and rectum, less often - the umbilical or right iliac region. Typically, pain increases before a bowel movement and eases after a bowel

movement. In many patients, the intensity of pain increases 30-90 minutes after eating. As the disease progresses

the connection between meals and abdominal pain is lost [i.e., the gastrocolytic reflex fades away, in which, after eating, increased intestinal motility occurs].

• Tenesmus - false urges with the release of blood, mucus and pus ["rectal spitting"] with little or no stool; are a sign of high activity of the inflammatory process in the rectum.

• Constipation [usually associated with tenesmus] due to spastic contraction of the intestinal segment above the lesion, characteristic of limited distal forms of UC.

• Later general symptoms join: anorexia, nausea and vomiting, weakness, weight loss, fever, anemia.

• The fulminant form is almost always characterized by a total lesion of the colon, the development of complications [toxic dilatation of the colon, perforation], in most cases it requires urgent surgical intervention. The disease begins acutely, within 1-2 days a pronounced clinical picture unfolds with a frequency of bloody stools more than 10 times a day, a decrease in hemoglobin level less than 60 g/l, an increase in ESR more than 30 mm/h.

Extraintestinal manifestations of IBD

Extraintestinal symptoms of UC are detected in 20% of patients. These include erythema nodosum, pyoderma gangrenosum, inflammatory eye diseases, arthritis, ankylosing spondylitis, respiratory system dysfunction, myositis, vasculitis, glomerulonephritis and other pathological processes outside the colon wall [6]:

• Erythema nodosum and pyoderma gangrenosum are due to the presence of circulating immune complexes, bacterial antigens and cryoproteins.

• Aphthous stomatitis is observed in 10% of patients with UC, aphthae disappear as the activity of the underlying disease decreases.

Eye damage - episcleritis, uveitis, conjunctivitis, keratitis, retrobulbar neuritis, choroiditis - occurs in 5-8% of cases.

• Inflammatory lesions of the joints [sacroiliitis, arthritis, ankylosing spondylitis] may be combined with colitis or occur before the onset of the main symptoms.

• Bone manifestations: osteoporosis, osteomalacia, ischemic and aseptic necrosis are complications of corticosteroid therapy.

NUC proceeds with the development of a number of extraintestinal lesions, among which a special place is given to changes in the liver and biliary tract. Extraintestinal lesions greatly complicate the course of the underlying disease, complicate differential diagnosis and therapy.

Extraintestinal manifestations in IBD occur in 5-25% of cases. Their largest share falls on total forms of UC [87.5%] and CD with involvement of the colon [29%] or colon and small intestine [58.1%] in the process.

General systemic complications of IBD according to the pathogenetic principle are divided into three groups:- arising from systemic hypersensitization - damage to the joints, eyes, skin, oral mucosa;- due to bacteremia and antigenemia in the portal system - damage to the liver and biliary tract;- developing secondarily with long-term disorders in the colon - anemia, electrolyte disorders.

Liver lesions were detected in 32.2% of patients, mainly with common forms, moderate and severe course, and high activity of UC [11].

Thus, the results of the literature data showed that parenchymal lesions of the liver were detected in 40 [22.2%] patients with UC, and changes in the biliary tract in 18 [10.0%] patients with UC. Non-alcoholic steatohepatitis [NASH], autoimmune hepatitis [AH], and primary biliary cirrhosis [PBC] of the liver were detected in 40 [69.0%] patients with UC, and lesions of extra- and intrahepatic bile ducts and gallbladder in 18 [31.0%] patients. NASH dominated among parenchymal liver lesions [53.5%]. AH was diagnosed in 8.6% of patients, PBC in 5.2% of patients with UC. All patients with UC with NASH, in addition to basic therapy, took the drug of essential phospholipids at a daily dose of 1800 mg [2 capsules 3 times a day] for three months. After the end of treatment, only 2 [6.5%] patients had minor complaints. Before treatment, all examined patients showed a significant increase in

AST and ALT by 2.3 and 2.2 times, respectively, in combination with an increase in GGT by 3.7 times compared with the norm, which may indicate a moderately pronounced cytolysis syndrome [11].A typical skin lesion in NUC is erythema nodosum, localized on the anterior surface of the legs, rarely on the face, torso and accompanied by fever, pain syndrome. Usually the rashes persist for several days, as they disappear, ecchymosis remains in their place, and then areas of discolored skin.

Chronic erythema nodosum is characterized by a persistent relapsing course, often with ulceration of the nodes. It occurs predominantly in the elderly. Specific treatment for erythema nodosum is not carried out, therapy should be directed to the underlying disease. Arthropathies are also considered to be the most common autoimmune extraintestinal manifestations in IBD. In CD with lesions of the colon, the frequency of arthropathies reaches 20-40%, in UC - much less often - 6%. It is believed that bacterial agents easily penetrate into the vascular bed through the damaged intestinal membrane, which causes a type III hypersensitivity reaction in the synovial membranes of the joints.

The range of articular lesions is very wide: the ankle, knee, interphalangeal joints are usually damaged. The intensity of pain and the degree of limitation of movements in the joints are usually small. With the onset of remission, articular changes completely disappear, deformity and dysfunction of the joints do not develop. Some patients develop transient spondylitis and sacroiliitis. The latter is noted more often and is more severe in case of more extensive and severe lesions of the large intestine, and is found on radiography in approximately 10% of patients with UC. Symptoms of sacroiliitis may precede the clinical manifestations of UC by many years. Thus, inflammatory bowel diseases, which include ulcerative colitis, remain one of the serious problems of the modern clinic of internal diseases. The versatility of the clinical picture, the complexity of diagnosis, the lack of alertness of general practitioners regarding these diseases leads to a large number of diagnostic errors and, consequently, to the loss of precious time to prescribe adequate treatment.

IBD is a systemic disease with an extremely diverse clinical picture of intestinal damage and a wide range of possible extraintestinal manifestations, which

makes primary diagnosis extremely difficult. Often, intestinal symptoms of the disease may be absent or subclinical. The correct interpretation of extraintestinal symptoms contributes to the establishment of a diagnosis and the appointment of timely adequate therapy for such an atypical variant of the disease, which, in turn, allows achieving a more stable remission, avoiding complications and preventing the formation of resistance to treatment.

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