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Effect of Ulceral Colitis on the Liver

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Abstract: Liver diseases can be a complication of inflammatory bowel disease (IBD), such as ulcerative colitis (UC) or Crohn's disease. The liver, which is the body's factory and sewer, produces proteins, breaks down toxins, and produces bile. It can become inflamed if inflammatory bowel disease is not treated properly. Unfortunately, some medications used to treat inflammatory bowel disease can also damage the liver.

Keywords: inflammatory bowel disease (IBD), ulcerative colitis (UC), liver.

Relevance. Liver disease can be a complication of inflammatory bowel disease (IBD), such as ulcerative colitis (UC) or Crohn's disease. The liver, which is the body's factory and sewer, produces proteins, breaks down toxins, and also produces bile. It can become inflamed if inflammatory bowel disease is not treated properly. Unfortunately, some medications used to treat inflammatory bowel disease can also damage the liver.

Liver lesions are one of the most common extraintestinal manifestations of UC. Of 180 patients with UC, liver lesions were detected in 58 (32.2%). In patients with UC, there were both lesions of the liver parenchyma - non-alcoholic steatohepatitis (NASH), autoimmune hepatitis, primary biliary cirrhosis (PBC), as well as changes in the extrahepatic bile ducts and gallbladder - primary sclerosing cholangitis (PSC), cholelithiasis (GSD). Parenchymal liver lesions were detected in 40 (22.2%) of 180 patients with UC, in 18 (10.0%). In UC, lesions of the liver parenchyma predominated; out of 58 patients with UC, NASH, autoimmune hepatitis and primary biliary cirrhosis were detected in 40 (69.0%) patients, and lesions of the extra- and intrahepatic bile ducts and gallbladder were in 18 (31.0%) sick. Non-alcoholic steatohepatitis develops mainly due to changes in the metabolism of the liver cell against the background of chronic endogenous intoxication, and cholelithiasis - due to disturbances in the metabolism and chemistry of bile, so these changes can be classified as metabolic. Metabolic lesions of the liver and biliary tract were detected in 43 (23.9%) patients, and in 74.2% of patients - lesions of the liver and biliary tract with UC (43 out of 58 patients) [Dorofeev A.E., Dorofeeva A.A. 2017].

Gut-activated T lymphocytes in UC patients may contribute to bile duct inflammation, as intestinal-liver endothelial adhesion molecule profiles (vascular address mucosal cell adhesion molecule 1 and vascular cell adhesion molecule 1 expression along with C-C chemokine motif ligand 25 secretion) are similar [Adams D. 2006; Lajsko E et al 2011].

The fact that the expression of Mad-CAM-1 in PSC liver depends on the role of vascular adhesion protein 1 may indicate that modulation of these proteins may influence the progression of PSC [Laylor P et al 2011].

Despite the presence of publications about the effects of experimental ulcerative colitis and its influence on the morphology of the liver, the morphometric changes occurring in the liver during ulcerative colitis are not sufficiently clarified and have not been studied. All this, of course, complicates the correct interpretation of the functional significance of the liver in normal conditions and in pathology.

The etiology of IBD, including 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, intestinal microflora and various environmental factors. Among the factors contributing to the development of UC, one should primarily mention hereditary predisposition. In first-degree

relatives of patients with UC, the risk of developing it is 10 times higher than in the general population. If both parents suffer from UC, the risk of its development in a child by the age of 20 increases to 52%. Genetic studies have shown that twin concordance (the incidence of the same nosological form in both twins) in UC is significantly lower than in Crohn's disease. A positive association has been identified between HLA DR2, as well as certain loci on chromosomes 2 and 6 (to a lesser extent 3, 7, 12 and 16) and the development of UC. The importance of nutrition in the etiology of UC is not as clearly defined as in Crohn's disease. When studying the dietary history, it was found that patients with UC consume less dietary fiber and more refined carbohydrates. There is a hypothesis about the important role of infectious agents, such as mycobacteria, measles virus, chlamydia, and Candida fungi, in the occurrence of UC. Immune impairments in the recognition of bacterial molecular markers (patterns) by dendritic cells, leading to hyperactivation of pro-inflammatory signaling pathways, are an important mechanism for the formation of UC.

Also, in UC, there is a decrease in the diversity of intestinal microflora due to a decrease in the proportion of anaerobic bacteria, mainly Bacteroidetes and Firmicutes. In the presence of these microbiological and immunological changes, UC develops under the influence of trigger factors, which include smoking, nervous stress, vitamin D deficiency, a diet with a low content of dietary fiber and a high content of animal protein, intestinal infections, especially C. difficile infection.

The result of the mutual influence of these risk factors is the activation of Th2 cells, overexpression of proinflammatory cytokines, primarily tumor necrosis factor alpha (TNF-α) and 10 cell adhesion molecules. The result of these reactions is lymphoplasmacytic infiltration of the colon mucosa with the development of characteristic macroscopic changes and symptoms of UC. The important role of the autoimmune reaction in the genesis of UC is indicated, in particular, by the typical chronically relapsing course of the disease, extraintestinal manifestations (primary sclerosing cholangitis, hemolytic anemia), detection of autoantibodies to colonocytes and perinuclear cytoplasmic antineutrophil antibodies (pANCA), and the effectiveness of immunosuppressive therapy. However, the autoantigens that would cause the formation of autoantibodies have not yet been clearly identified. One of the potential autoantigens may be the cytoskeletal microfilament protein, tropomyosin. The mechanism of damage to the intestinal mucosa that occurs in UC is complex. The damage involves T-lymphocytes, antibodies and complement, free oxygen radicals and proteases, and changes in apoptosis processes. Various cytokines also play an important role, such as epidermal growth factor, interleukins (IL) and interferon (IFN), in particular IL-1b, IL-2, IL-4, IL-15, IFN-g, as well as neuropeptides, adhesion molecules and intracellular signal. It should be noted that the dynamics of certain immunological parameters (changes in T cells, cytokines, characteristics of antibody formation) have so far been traced only in experimental studies performed on mice with severe combined immunodeficiency syndrome (SCID) and on animals with reproduced genetic changes. Naturally, this significantly complicates the analysis of the results obtained in relation to clinical conditions [I.V. Dolgalev 2021].

Complications. With UC, a variety of complications are observed, which can be divided into local and systemic. Local complications include colon perforation, acute toxic dilatation of the colon (or toxic megacolon), massive intestinal bleeding, and colon cancer. Systemic complications. Almost 60% of patients with UC have extraintestinal manifestations. 1. Autoimmune, associated with disease activity: Arthropathy – arthralgia, arthritis. Skin lesions – erythema nodosum, pyoderma gangrenosum. Damage to the mucous membranes – aphthous stomatitis. Eye damage – uveitis, iritis, iridocyclitis, episcleritis. 2. Autoimmune, not related to disease activity: Ankylosing spondylitis, sacroiliitis. Primary sclerosing cholangitis (PSC). Osteoporosis, osteomalacia. Psoriasis. Caused by prolonged inflammation and metabolic disorders: Cholelithiasis. Liver steatosis, steatohepatitis. Thrombosis of peripheral veins. Pulmonary embolism. Amyloidosis. [I.V. Dolgalev 2021].

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