A MODERN VIEW ON THE RELEVANCE OF THE PROBLEM OF NON-SPECIFIC ULCERATING COLITIS

Gulchehra Mardonalievna Akhmedova
2 REPUBLICAN COLLEGE OF PUBLIC HEALTH
NAMED AFTER ABU ALI IBN SINA
Umida Azizovna Asatullayeva
Mirzaakhmedova Samira Lenorovna
TASHKENT STATE MEDICAL UNIVERSITY
TASHKENT, UZBEKISTAN

Ulcerative colitis (UC) is one of the most serious and unresolved problems in gastroenterology. UC is a common chronic disease of the colon, characterized by ulcerative and structural changes in the colon mucosa. Globally, it has an incidence of 50–230 cases per 110,000 people [1]. The disease can worsen at absolutely any age, but men and women aged 18-45 are especially susceptible. Colitis is twice as rare in smokers as in non-smokers. Many factors influence the development of this disease, the most common of which are: Infectious, immunological, genetic, and, of course, environmental factors. The disease is characterized by a long, persistent course, a tendency toward seasonal exacerbations and severe complications, a high rate of disability, and primarily affects young and middle-aged individuals of working age. In recent years, increasing importance has been attached to the state of the immune system in the pathogenesis of UC, which largely determines the outcome of the disease [1,2]. UC was first described in medical literature many centuries ago. The first description of the morphological picture of UC belongs to the Viennese pathologist Karl Rokitansky, which he presented in 1842 in a report “On catarrhal inflammation of the intestine.” Having performed several thousand autopsies on patients who died from infectious colitis, he described "ulcer-like colitis." Circular No. 4 in 1865 already contained a detailed description of the histological picture of UC, based on the study of over 200 autopsies. The disease has been described in follicles and glandular structures of the intestinal epithelium. "Rupture of enlarged follicles resulted in confluent and spreading ulcerations" [2]. Ulcerative colitis (UC) is an unsolved problem in gastroenterology. The disease is characterized by a long, persistent course. In recent years, increasing importance in the pathogenesis of UC has been given to the state of the immune system, which largely determines the outcome of the disease [3]. UC is most widespread in Europe, North America, and Australia. Its incidence in these regions reaches 8–15 new cases per 100,000 population per year, and its prevalence is 80–120 people per 100,000 population [4]. The annual increase in the incidence of non-specific ulcerative colitis (UC) worldwide, the predominant affection of young, working-age individuals, and

complications leading to early disability, draws attention to this disease [5]. Epidemiological studies of recent decades indicate a significant increase in the incidence of ulcerative colitis, as well as high costs for treating patients. In the United States, where the prevalence of UC is 286 per 100,000 people, in Asian countries the prevalence of UC ranges from 5.3 to 63.6 cases per 100,000; the total annual cost of treating the disease ranges from $8.1 to $14.9 billion. The highest incidence of UC was observed in Northern European countries—up to 505 per 100,000 population. The cost of treating patients with UC ranges from 12 to 29.1 billion euros. The incidence rate in men and women is comparable, with the peak incidence occurring in the socially active age group of 20 to 40 years [6,7]. In recent decades, an increase in the incidence of UC has been observed in developing countries in Africa, Asia, and South America, including Brazil and Taiwan [8]. In the European population, the proportion of UC patients with distal lesions (proctitis) varies significantly from 27 to 60% depending on the country and time period [9]. Ulcerative colitis (UC) is a diffuse, chronic, relapsing disease of the colon that primarily affects the colon's mucosa. This condition occurs worldwide. Currently, there is no clear information about the etiology and pathogenesis of this disease. The etiology of inflammatory bowel diseases remains poorly understood [9,10]. It is believed that the development of the disease requires the interaction of a certain genetically predisposed diathesis, including those determining immune mechanisms, environmental factors, potential participants in pathogenesis - viruses (measles), bacteria and their metabolites, including intestinal microflora.

Most often, a hereditary predisposition to the development of an autoimmune inflammatory process in the colon mucosa in response to the seeding of its surface by microorganisms and viruses, as well as contact exposure to food products, is considered. This opinion is based on the frequent association of UC with other autoimmune processes [10]. Current data demonstrate the central role of dysregulation of the immune response against intestinal microflora in combination with disruption of anti-inflammatory or pro-inflammatory pathways in the pathogenesis of UC [2,3]. The causes of risk factors for ulcerative colitis include pathologies of the immune system, such as immunodeficiency states or hereditary mutations in the activity of immune cells, disturbances in the intestinal microflora - a decrease in the number of beneficial and an increase in the number of pathogenic microorganisms, long-term intestinal infections, poor diet, insufficient intake of vitamin D, prolonged stress and the resulting deterioration of digestion and disruption of the immune system [7,9,10]. Aberrant immunological reactions that occur in the intestine can affect the epithelial barrier, increasing its permeability to new antigens, leading to persistent chronic inflammation [11]. A family history of ulcerative colitis is a risk factor for the development of the disease, but, according to the generally accepted opinion of researchers, it is not a negative prognostic factor [12]. However,

there is some evidence that disease progression and genetic polymorphisms that determine susceptibility to UC are heterogeneous across different regions and populations, making the search for "universal" genes or triggers difficult. Despite the heterogeneity of risk factors for UC, some common trends have been identified [7,12]. The hypothesis of genetic susceptibility for non-specific ulcerative colitis is based on the proven link between the defect of the gene of the 16th chromosome NOD2/CARD15, which determines the synthesis of a protein that identifies bacterial metabolites and triggers immune reactions with the release of cytokines. An imbalance in the immune system, supported by genetic diathesis and exposure to various environmental factors, leads to an excessive inflammatory response to triggers. The Montreal classification of UC is also used to classify the severity of the pathological process [6,8]. This scale takes into account symptoms such as frequency of bowel movements per day, the presence of blood in the stool, heart rate, the presence of an increase in the patient's body temperature, a decrease in the hemoglobin level in the blood, and ESR. Remission (S0) on this scale is characterized by the absence of the listed symptoms. The active stage of UC is subdivided into S1, S2, and S3 sub-items characterizing the severity: S1 is characterized by a change in stool frequency – 4 or less times a day, the presence of blood in the stool and other symptoms may be absent, for S2 – stool > 4, blood in the stool, for S3 – stool ≥ 6 with blood [6,13]. There are several groups of symptoms characteristic of ulcerative colitis: intestinal and extraintestinal, toxic, and metabolic. This means that the disease can manifest itself in a variety of ways, from minor abdominal discomfort to a serious deterioration in general well-being. Intestinal, extraintestinal, toxic, and metabolic symptoms of ulcerative colitis. The first step in examining patients with suspected IBD is differential diagnosis with colitis of various etiologies, as well as between UC and CD. The various endoscopic, histological, clinical, and radiographic features of each IBD are described. Ulcerative colitis is characterized by damage to the colon only and retrograde ileitis with involvement of the ascending colon, diffuse damage to the segment, mandatory involvement of the rectum and spread of inflammation in the proximal direction, crypt abscesses and a decrease in goblet cells in biopsies.

In view of the increasing number of patients with inflammatory bowel diseases, the severity of the pathology, as well as early disability of patients, timely diagnosis of ulcerative colitis and Crohn's disease is an urgent task that requires a timely solution [8,14]. The unresolved issues of etiopathogenetic treatment make nonspecific ulcerative colitis relevant in relation to its treatment [15]. Bacteriological examination of feces is performed to exclude infectious colitis. Laboratory research methods are important for determining the severity of UC. In addition, with prolonged illness, diarrhea can lead to hyponatremia, hypochloremia, and hypoalbuminemia, as well as progressive weight loss; anemia is often observed. Severe forms of the disease are characterized by an elevated ESR and the presence of leukocytosis. Nonspecific

ulcerative colitis is differentiated primarily from infectious intestinal lesions, ischemic colitis, and Crohn's disease [16,17]. Ulcerative colitis is classified by a number of characteristics, including location, severity, development of complications, and other parameters. This classification helps doctors prescribe effective treatment and assess the likelihood of complications: by location, course of the disease, severity, and development of complications. Long-term inflammation and bleeding in the intestines can lead to a number of complications - from local ones that affect the condition of the gastrointestinal tract, to systemic ones that affect the entire body [8,16,17]. Surgical treatment is necessary in 10–30% of ulcerative colitis cases. Surgical treatment causes complications in 15–50% of cases. Ulcerative colitis can cause severe physical and psychological discomfort, and it cannot be completely cured—even with successful symptom relief, there is a risk of recurrence. Treatment for ulcerative colitis is long-term, lasting anywhere from several months to several years. If medications are discontinued after initial improvement, symptoms are highly likely to recur. Knowing the likely causes of ulcerative colitis can help prevent its development. Ulcerative colitis requires ongoing monitoring and treatment, even during periods of remission. With timely diagnosis and treatment, it's possible to achieve lasting remission and improve quality of life.

LIST OF REFERENCES:

1. Dusanov A.D., Yuldasheva D.A. Clinical immunological characteristics of non-specific ulcerative colitis. Journal of cardiorespiratory research. 2022, vol 3, issue 2, pp.

2. Knyazev O.V., Kagramanova A.V., Parfenov A.I. Ulcerative colitis. On the 180th anniversary of the description by Karl Rokitansky. Therapeutic archive. 2021;93(12):1564–1568. DOI: 10.26442/00403660.2021.12.201219.

3. Dusanov A.D.1, Mamurova N.N. Clinical and immunological characteristics of nonspecific ulcerative colitis. Therapeutic archive. 65-68.

4. Vatutin N.T., Shevelyok A.N., Karapysh V.A., Vasilenko I.V. Nonspecific ulcerative colitis. Archives of Internal Medicine. № 4(24). 2015. UDC. 616.345-002.44. 62-65.

5. Guido Adler. Crohn's disease and ulcerative colitis - M.: Geotar Med, 2001. - 527 p.

6. Baghaei A., Emami M.H., Adibi P., Tavakkoli H., Daghaghzadeh H., Tamizifar B. et al. Inflammatory Bowel Disease Registry and Monitoring: Feasibility Study and Application (Isfahan Inflammatory Bowel Disease Surveillance Project). Int J Prev Med. 2019;10:190. doi: 10.4103/ijpvm.IJPVM_316_17.

7. Bolotova E.V., Yumukyan K.A., Dudnikova A.V. Modern concepts of the mechanisms of development and predictors of the severity of ulcerative colitis. Doctor.Ru. 2022; 21(2): 34–39. DOI: 10.31550/1727-2378-2022-21-2-34-39.

8. Wei S.C., Sollano J., Hui Y.T. et al. Epidemiology, burden of disease, and unmet needs in the treatment of ulcerative colitis in Asia. Expert Rev. Gastroenterol. Hepatol. 2021; 15(3): 275–89. DOI: 10.1080/17474124.2021.1840976

9. Burisch J., Jess T., Martinato M., Lakatos P.L., Gastroenterol. 2005;19(SA):5A–36A. doi: 10.1155/2005/269076. 6. ECCO-EpiCom. The burden of inflammatory bowel disease in Europe. J Crohns Colitis. 2013;7(4): 322–337. doi: 10.1016/j.crohns.2013.01.010.

10. Shaposhnikov V.I., Storozhuk P.G., On the pathogenesis of nonspecific ulcerative colitis. Kuban Scientific Medical Bulletin. No. 7 (142) 2013. UDC. 616.345-002.3-022-089.168.1-06. 129-131.

11. [Tertychny A.S., Akhrieva Kh.M., Maev I.V. et al. Diagnostic problems of histological remission in patients with inflammatory bowel disease. Arkhiv Patologii. 2017; 79(3): 3–9. (in Russian)]. DOI: 10.17116/patol20177933-9

12. Orlanski-Meyer E., Aardoom M., Ricciuto A. et al. Predicting outcomes in pediatric ulcerative colitis for management optimization: systematic review and consensus statements from the pediatric inflammatory bowel disease-ahead program. Gastroenterology. 2021; 160(1): 378–402.e22. DOI: 10.1053/j.gastro.2020.07.066

13. Lugovkina A.A., Rudakova L.O., Kryukova N.A., Bessonov A.A., Skvortsov V.V. Features of diagnosis and treatment of nonspecific ulcerative colitis. Experimental and Clinical Gastroenterology.2019;164(4): 10–16. DOI: 10.31146/1682-8658-ecg-164-4-10-16.

14. A. A. Budzinskaya, E. A. Belousova, S. G. Tereshchenko, A. V. Levitskaya. Ulcerative colitis and Crohn's disease: problems of differential iagnostics.DOI: https://doi.org/10.56871/UTJ.2023.92.54.003

15. Egamov Yu.S., Khaidarov S.A. The primary importance of endomesenteric lymphotropic therapy in the complex treatment of nonspecific ulcerative colitis in the postoperative period.Re-Health Journal. RHJ-2(10)-2021. 260-266

16. Ulcerative colitis (adults): clinical guidelines / Russian Gastroenterological Association, All-Russian public organization "Association of Proctologists of Russia".2020.

17. Abdulhakov S.R., Abdulhakov R.A. Nonspecific ulcerative colitis: modern approaches to diagnosis and treatment. Bulletin of modern clinical medicine. 2009 Vol. 2, issue 1. UDC. 616.348-002.44-07-08. 32-41.