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КАФЕДРА ПОЛИКЛИНИЧЕСКОЙ ТЕРАПИИ

Е. А. Каразей, Е. С. Алексеева, Е. В. Рылатко

НЕИНФЕКЦИОННЫЕ ВОСПАЛИТЕЛЬНЫЕ ЗАБОЛЕВАНИЯ КИШЕЧНИКА

NON-INFECTIOUS INFLAMMATORY BOWEL DISEASES

Учебно-методическое пособие

Минск БГМУ 2026

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Р е ц е н з е н т ы: канд. мед. наук, доц. каф. внутренних болезней, кардиологии и ревматологии с курсом повышения квалификации и переподготовки Белорусского государственного медицинского университета О. А. Сиротко; каф. поликлинической терапии и общественной практики с курсом ФПКиП Гомельского государственного медицинского университета

Каразей, Е. А.

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Рассматриваются причины и механизм развития, этапы диагностического поиска и дифференциальной диагностики неинфекционных воспалительных заболеваний кишечника в амбулаторных условиях.

Предназначено для студентов 4-го курса медицинского факультета иностранных учащихся, обучающихся по специальности «Лечебное дело» на английском языке.

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ABBREVIATIONS

ANCA — anti-neutrophil cytoplasmic antibodies
ASCA — saccharomyces cerevisiae antibodies
BMI — body Mass Index
CBC — complete blood count
CDAI — Crohn’s disease activity index
CD — Crohn’s disease
CRC — colorectal cancer
CRP — C-reactive protein
CT — computed tomography
ESR — erythrocyte sedimentation rate
FC — fecal calprotectin
GCs — glucocorticosteroids
GGT — gamma-glutamyl transferase
GI — gastrointestinal
HR — heart rate
IBD — inflammatory bowel diseases
ICD-10 — the International Classification of Diseases, Tenth Revision
IL — interleukin
JAK — janus kinase
MR — magnetic resonance
NSAID — nonsteroidal anti-inflammatory drug
S1P — sphingosine-1-phosphate
TNF-α — tumor necrosis factor-alpha
TMS — total Mayo Score
UC — ulcerative colitis
5-ASA — 5-aminosalicylic acid

MOTIVATIONAL CHARACTERISTICS OF THE TOPIC

The academic manual "Non-infectious inflammatory bowel diseases" is intended for practical class on the topic "Non-infectious inflammatory bowel diseases, irritable bowel syndrome. Differential diagnosis of dyspepsia and abdominal pain syndrome."

Time: 6 hours.

This manual address current issues in the diagnosis, differential diagnosis, treatment, and rehabilitation of patients with non-infectious inflammatory bowel diseases in an outpatient setting.

Non-infectious inflammatory bowel diseases or inflammatory bowel diseases (IBD), primarily comprising Crohn's disease (CD) and ulcerative colitis (UC), represent chronic, idiopathic inflammatory disorders of the gastrointestinal tract (GI) with a complex and multifactorial etiology involving genetic predisposition, immunologic dysregulation, and environmental influences. Over the past several decades, the incidence and prevalence of both conditions have steadily increased throughout the world, establishing IBD as a growing global health burden that significantly impacts patient quality of life and healthcare systems. The diagnostic and therapeutic landscape for patients with CD and UC has also evolved considerably in recent years.

This educational manual is dedicated to the comprehensive consideration of current approaches to the diagnosis, differential diagnosis, treatment, and rehabilitation of patients with non-infectious inflammatory bowel diseases, with a particular focus on the outpatient setting. To ensure that this manual reflects the most current and scientifically valid approaches, it draws upon the established frameworks and evidence-based methodologies utilized in major clinical guidelines.

Aim of the practical class: to familiarize students with the concept and strategies for diagnosis, treatment, prevention of IBD.

Objectives: development of students' scientific knowledge regarding the provision of medical care, treatment strategies, indications for hospitalization, principles of primary and secondary prevention, and rehabilitation of patients with ulcerative colitis and Crohn's disease in outpatient settings. Mastering of practical skills in differential diagnosis, interpretation of examination data, formulation of clinical diagnosis and completion of medical documentation, and conducting temporary disability assessment.

Requirements for the initial level of knowledge. To fully master the topic, from the sections of the academic disciplines "Human Anatomy," "Normal Physiology," and "Pathological Physiology" the student should review the anatomy and functions of the gastrointestinal tract, as well as the pathophysiological mechanisms of inflammation development, the concept of malabsorption, maldigestion and malnutrition.

Control questions from related disciplines:

1. Macroscopic structure, topography of the digestive system organs.
2. Pathophysiological changes in IBD.
3. Laboratory diagnostic methods: blood, urine, and stool analysis in IBD.

Control questions on the topic of the lesson:

1. The concept of UC, risk factors for development, classification.
2. Clinical criteria, mechanisms of development, complications of UC.
3. Plan for examining patients UC.
4. Treatment tactics for the UC, indications for hospitalization, primary and secondary prevention, medical observation of patients with UC.
5. The concept of CD, risk factors for development, classification.
6. Clinical criteria, mechanisms of development, complications of CD.
7. Plan for examining patients CD.
8. Treatment tactics for the CD, indications for hospitalization, primary and secondary prevention, medical observation of patients with CD.

Assignments for student independent work: review the anatomy and physiology of the GI tract, the pathophysiology of inflammation development and the concept of malabsorption, maldigestion, and malnutrition. Familiarize with the clinical symptoms and diagnostic signs of UC and CD, as well as the examination methods used for these conditions. Study the mechanisms of action, indications, and contraindications of medications used in the treatment of IBD. Complete the test assignments available in the electronic educational complex.

DEFINITIONS

UC (K51, ICD-10) is a chronic inflammatory disease characterized by a relapsing and remitting course and continuous inflammation of the colonic mucosa without granulomas in biopsy specimens, with lesions of the rectum and varying extents of the colon.

CD (K50, ICD-10) is a chronic inflammatory process that can potentially affect any part of the intestinal tract from the mouth to the anus and is characterized by skip lesions, involvement of all layers of the intestinal wall (transmural inflammation), and the formation of epithelioid cell granulomas.

Unclassified colitis or **IBD unclassified** — may be established following analysis of medical history, results of endoscopic examination and histological evaluation of multiple colonic mucosal biopsy specimens, as well as radiological studies, when it is not possible to definitively diagnose UC, CD, or another form of colitis.

Indeterminate colitis — may be established following histological examination of tissue obtained during surgical intervention in cases where overlapping features of both ulcerative colitis and Crohn's disease are present.

Biologic Medicine (Biologics) — a group of medicinal products of biological origin, including monoclonal antibodies and recombinant proteins, obtained using

genetic engineering methods, specifically suppressing the immune-inflammatory process and slowing the progression of the disease.

Biosimilar is a biological medicinal product, which is similar but not identical to the biological drug already authorized. The biosimilar and its reference product are expected to display the same safety and efficacy profile and are generally used to treat the same conditions.

Bio-naïve patient — individual who has never received treatment with a specific class of medication, most commonly biologics or biosimilars.

ULCERATIVE COLITIS

UC is an immune-mediated, diffuse, chronic lesion of the colon. The inflammatory process is always localized in the mucosa (except in cases of severe attacks) and inevitably involves the rectum. Absence of rectal involvement has been noted in fewer than 5 % of adult patients. UC is characterized by pathology limited exclusively to the colon. The occurrence of involvement of the ileum or "backwash ileitis" is uncommon and is observed in approximately 10 % of patients.

Incidence rates of UC worldwide vary widely, from a low of 0.6 to a high of 24.3 per 100 000. The highest prevalence of UC (505 cases per 100 000 people) is registered in European countries. In the Asian region, incidence rates are lower, but a clear upward trend is observed. An analysis of demographic data shows that the peak of primary diagnosis occurs between the ages of 20 and 30, with a second peak observed at ages 60 and 70.

Men and women are affected equally, but race plays a role: the disease is more common among caucasians. A low incidence of mortality is a fortunate characteristic of UC, despite the significant morbidity it causes. The primary long-term risk associated with the disease is the development of dysplasia and colorectal cancer (CRC).

ETIOLOGY AND PATHOGENESIS

The etiology of UC remains unclear. It is suggested that the condition arises from a complex interaction of several **etiological factors**: hereditary predisposition, immune system dysfunction (both innate and adaptive), intestinal microbiota imbalance, and adverse environmental factors.

Hereditary predisposition and immune system dysfunction: approximately 100 genetic polymorphisms associated with the development of UC have been identified. The presence of certain genetic variants causes altered innate immune response and a distortion of adaptive immunity. The inability of dendritic cells to properly recognize bacterial molecular patterns is considered a critical factor, leading directly to the overstimulation of pro-inflammatory signals.

Intestinal microbiota imbalance: in UC decrease in the diversity of intestinal microflora is observed, mainly due to a decrease in the population of

anaerobic bacteria, which creates a favorable background for the implementation of a pathological immune response.

Adverse environmental factors: pathological immune response in UC can be initiated by various external influences, psychological stress, insufficient vitamin D levels, and dietary habits characterized by low fiber consumption and high animal protein intake. Additionally, gastrointestinal infections — particularly those caused by *Clostridioides difficile* and cytomegalovirus — serve as significant precipitating factors.

Genetic and environmental factors together induce a complex immune response with activation of diverse T-cell populations across different inflammatory phases. This stimulates hypersecretion of pro-inflammatory cytokines like tumor necrosis factor-alpha (TNF-α), interleukins (IL-1, IL-12, IL-23, IL-17), and adhesion molecules. The resulting pathological outcome is severe lymphoplasmacytic infiltration and colonic mucosal damage, which manifests as the hallmark macroscopic changes of UC.

CLASSIFICATION

By the extent of the colon lesion:

a) *distal lesion*: proctitis (rectum along is affected), proctosigmoiditis (the rectum and sigmoid colon are affected);

b) *left-sided colitis* (the lesion extends no further than the splenic flexure);

c) *extensive colitis* (the lesion extends proximally to the splenic flexure): subtotal colitis (the lesion extends to the hepatic flexure), total colitis (the entire colon is affected).

By the activity of the inflammatory process in the colon:

a) *active UC* — diagnosed when the patient has symptoms accompanied by an increase in measurable markers of inflammation (biochemical, endoscopic and/or microscopic signs of inflammation);

b) *exacerbation of UC* — is the onset of symptoms in a patient with a confirmed diagnosis of UC who has previously achieved clinical remission, either spontaneously or as a result of treatment. An early exacerbation develops within 3 months of achieving remission;

c) *remission of UC* — is defined as a stool frequency of 3 or fewer times per day, the absence of blood in the stool, and the absence of endoscopic signs of inflammation.

By the course of the inflammatory process in the colon:

a) *first attack of UC* — signs of active UC appear for the first time, their duration is no more than 6 months;

b) *relapsing course* — alternating exacerbations and remissions:

– with rare exacerbations (exacerbations once or less per year);

– with frequent exacerbations (exacerbations 2 or more times per year);

c) *continuous course* — persistence of symptoms for 6–8 months, despite adequate pharmacotherapy;

d) *acute severe UC* — bloody stool more than 6 times per day, systemic inflammation in combination with one or more of the following: body temperature > 37.8 °C; heart rate (HR) > 90 bpm; hemoglobin < 105 g/L or C-reactive protein (CRP) > 30 mg/L.

By severity:

a) Montreal classification of the severity of exacerbations of UC (clinical):

– *clinical remission* — asymptomatic;

– *mild exacerbation* — passage of 4 or fewer stools/day (with or without blood), absence of any systemic illness, and normal inflammatory markers (erythrocyte sedimentation rate (ESR) and CRP);

– *moderate exacerbation* — passage of more than 4 stools per day but with minimal signs of systemic toxicity;

– *severe exacerbation* — passage of at least 6 bloody stools daily, HR of at least 90 bpm, temperature of at least 37.5 °C, haemoglobin of less than 105 g/l, and ESR of at least 30 mm/h.

b) endoscopic criteria for the severity of exacerbation of UC:

– *mild exacerbation*: edema, hyperemia, decreased vascular pattern, mild friability;

– *moderate exacerbation*: marked erythema, lack of vascular pattern, friability, erosions, contact bleeding;

– *severe exacerbation*: spontaneous bleeding, large ulcerations.

The Total Mayo Score (TMS) for clinical and endoscopic assessment of the severity of UC exacerbation (Table 1).

Table 1

The total mayo score

Parameter	Clinical evaluation (single choice)	Score
Stool frequency (per day)	Normal number of stools	0
	1–2 more than normal	1
	3–4 more than normal	2
	≥ 5 more than normal	3
Rectal bleeding (indicate the most severe bleeding of the day)	None	0
	Streaks of blood with stool in in less than half of the cases	1
	Obvious blood with stools in most cases	2
	Blood alone passes	3
Endoscopic findings	Normal mucosa or inactive disease	0
	Mild activity (erythema, decreased vascular pattern, mild friability)	1
	Moderate activity (marked erythema, lack of vascular pattern, friability, erosions)	2
	Severe activity (spontaneous bleeding, large ulcerations)	3
Physician’s global assessment	Normal	0
	Mild disease	1
	Moderate disease	2
	Severe disease	3

Interpretation of results: remission < 2 points; mild activity 3–5 points; moderate activity 6–10 points; severe activity > 10 points.

By response to treatment:

– *responding to treatment UC*;

– *steroid-dependent disease* — within 3 months of treatment, a reduction in the dose of prednisolone below 10 mg per day or budesonide below 3 mg per day leads to an exacerbation of the disease or the development of an exacerbation within 3 months after discontinuing glucocorticosteroids (GCs);

– *steroid-refractory disease* — persistent UC despite using prednisolone at a dose of 0.75 mg/kg per day for 4 weeks or more. For patients with acute onset and severe UC, steroid-refractory disease means no response to treatment after 3 days of intravenous GCs administration;

– *refractory to immunomodulators UC* — persistent disease activity or development of an exacerbation despite using thiopurines for at least 3 months (azathioprine 2–2.5 mg/kg per day or mercaptopurine 0.75–1 mg/kg per day in the absence of leukopenia);

– *primary nonresponse* — lack of clinical improvement after completion of an induction course of biological therapy;

– *loss of response* — relapse during a maintenance course of biological therapy in a patient who had previously achieved clinical remission (may include patients in whom the dose of biological therapy used for maintenance treatment must be increased to maintain remission);

– *clinical response to treatment* — improvement in the patient's overall clinical condition, expressed as a reduction in the severity of symptoms (by at least 50 %);

– *endoscopic response to treatment* — improvement in the results of the endoscopic examination compared to the previous endoscopic examination (a decrease in the activity index according to the TMS for assessing UC activity by at least 1 point).

DIAGNOSTIC EVALUATION

The diagnostic process in UC involves a thorough clinical assessment alongside direct visualization of the colon, with histopathological examination of biopsy samples playing a key role.

Complaints and medical history. The following aspects should be emphasized during the interview:

– *stool characteristics* — frequency (increased), consistency (diarrhea, mainly at night), presence of pathological components such as blood, tenesmus (symptom characterized by the continuous, painful, or distressing urge to defecate, even when the bowels are already empty);

– *daily stool volume and duration of symptoms*;

– *location and nature of abdominal pain* — there may be moderate spastic abdominal pain, often before bowel movements;

– *recent travel to regions with a hot climate* — to rule out infectious causes;

– *use of medications* — especially antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), and antirheumatic agents;

– *family history* — presence of IBD or malignancy in relatives;

– *smoking* — it is known that UC occurs less frequently in smokers, in contrast to CD, where the association with smoking is reversed.

Physical examination. During physical examination of patients with UC, various manifestations of the disease are often detected: signs of inflammation and metabolic disorders, symptoms indicating surgical complications of UC (profuse colonic bleeding, toxic dilation of the colon, colonic perforation, CRC, as well as extraintestinal manifestations. Nutritional status assessment (e.g. by body mass index (BMI)) is performed in all patients with UC. As part of this examination, the perianal area is assessed and a digital rectal examination is performed.

Based on the assessment of complaints, medical history data, and physical examination findings, the following *clinical criteria for UC are determined*:

– *intestinal manifestations* — bloody diarrhea (hematochezia), tenesmus, colicky abdominal pain;

– *general signs of inflammation and metabolic disorders* — fever, tachycardia, increased ESR, leukocytosis, thrombocytosis, increased CRP, fibrinogen, anemia, weight loss, dehydration, hypoproteinemia, hypoalbuminemia, electrolyte disturbances;

– *extraintestinal manifestations* — eye lesions (episcleritis, anterior uveitis), skin lesions (pyoderma gangrenosum), arthritis, migratory monoarticular arthritis, ankylosing spondylitis, sacroiliitis, idiopathic thrombocytopenic purpura, osteoporosis, sclerosing cholangitis.

Instrumental diagnostic studies. *Colonoscopy* serves as a key tool for establishing the diagnosis of UC, assessing the degree of disease activity, and making a decision about the need for surgical intervention.

The most typical endoscopic manifestations include: signs of inflammation of the colon mucosa — swelling, hyperemia, decreased vascular pattern, granularity, contact or spontaneous bleeding, erosions, ulcers, fibrin deposits, pseudopolyps. Inflammation in UC is continuous (starting in the rectum and, as it spreads proximally, sequentially involves all anatomical segments of the colon) and diffuse (within one anatomical segment, the entire mucous membrane is affected).

If UC is suspected during the initial diagnostic workup, or in cases where the accuracy of a previously established diagnosis is in question, biopsy of the colonic mucosa to confirm the disease is performed.

Histological criteria for UC involve chronic, diffuse (inflammation is usually uniform across the biopsy) mucosal inflammation primarily affecting the mucosa (inflammation is generally confined to the mucosa and superficial submucosa, sparing deeper layers). Key markers include crypt architectural distortion (branching, shortening), diffuse basal lymphoplasmacytosis, mucin depletion. In the active phase of the disease and neutrophil-mediated epithelial injury (cryptitis/crypt abscesses) is revealed.

The recommended standard for biopsy when establishing a diagnosis is as follows: at least two mucosal samples from each examined segment of the intestine must be obtained; the specimens from different segments should be placed into separate containers; biopsy samples from any identified mass lesions (including solid ulcers) should be placed into a separate container for morphological verification.

If a persistent stricture of the intestinal lumen is detected in the setting of UC, it is mandatory to perform an evaluation to rule out colorectal cancer.

In cases of severe UC activity, a *rectosigmoidoscopy* (preferably up to the splenic flexure) with multiple biopsies (if there is no risk of perforation) is permitted for initial diagnosis.

Abdominal ultrasound is recommended for all patients with suspected UC during the initial diagnosis, when the correctness of a previously established diagnosis is in doubt, in cases of long-standing UC, when complications of UC are suspected, as well as to rule out pathology of other abdominal organs.

Intestinal ultrasound is recommended as the additional examination method for patients with UC to assess the activity of the inflammatory process.

In cases of severe UC activity *intestinal ultrasound*, *computed tomography colonography (CT colonography), or magnetic resonance colonography (MR colonography)* may be performed to evaluate the extent of colon involvement.

Capsule colonoscopy used in cases where performing a traditional endoscopic examination is difficult due to the patient's severe condition or is associated with a high risk of complications.

Optical coherence tomography may be used to differentiate between UC and CD.

Plain abdominal radiography is recommended for patients with severe activity of UC when complications need to be ruled out (to exclude perforation and toxic dilatation of the colon).

Laboratory diagnostic studies. *Complete blood count* (CBC) The results of the CBC may reveal: anemias (iron deficiency anemia, anemia of chronic inflammation, vitamin B_{12} deficiency anemia, or folate deficiency anemia); leukocytosis (caused either by chronic inflammation or by steroid therapy); thrombocytosis; an elevated ESR.

Blood biochemistry (bilirubin; total protein; AST; ALT; alkaline phosphatase; gamma-glutamyl transferase (GGT); CRP; urea; creatinine; glucose; cholesterol; iron; ferritin) used to assess general signs of inflammation and metabolic disorders.

Fecal calprotectin (FC) marker of inflammation and *faecal immunochemical test* (to detect hidden human blood in stool) are performed when UC is suspected.

It is also essential to rule out other conditions that present with similar symptoms, particularly infectious causes. The following tests are performed: *rectal swab for pathogenic microflora, stool analysis for helminth eggs and intestinal protozoa* (during the initial presentation or in cases of an atypical (severe) UC), *detection of Campylobacter and E. coli O157:H7 antigens in stool* (during the initial presentation or in cases of an atypical (severe) UC), *stool test for*

Clostridioides difficile toxins (during the initial presentation, in cases of an atypical (severe) UC, and/or if development of this infection is suspected), *detection of Cytomegalovirus infection* (in cases of an atypical (severe) UC and/or when deciding on the prescription of biologic therapy) using biopsy specimens obtained during esophagogastroduodenoscopy and ileocolonoscopy.

Anti-neutrophil cytoplasmic antibodies (ANCA) in serum performed if differentiating between UC and CD is challenging.

TREATMENT

UC is a chronic disease that necessitates treatment aimed both at achieving remission (clinical and endoscopic) and sustaining it without the use of GCs (meaning their withdrawal within 12 weeks of starting therapy).

The primary objective in UC is the complete resolution of clinical manifestations — absence of blood in the stool and normalization of stool frequency. Achieving endoscopic remission is considered mandatory. In cases of disease progression or the development of life-threatening conditions, the priority becomes surgical intervention.

Treatment strategies for UC should take into account three key factors: the ex-tent of colonic involvement, the severity of disease activity, and the expected long-term prognosis.

Induction of remission in mildly to moderately active UC. *Proctitis (proctosigmoiditis):* for inducing remission the first-line therapy is medications based on *5-aminosalicylic acid (5-ASA)* for topical use: rectal mesalamine (mesalazine) 1–2 g per day. The anti-inflammatory effect of mesalamine is mediated by inhibition of neutrophil lipoxygenase activity and the synthesis of prostaglandins and leukotrienes. It suppresses the migration, degranulation, and phagocytosis of neutrophils, as well as the secretion of immunoglobulins by lymphocytes.

If blood in the stool persists within 2 weeks from the start of treatment which means that therapy needs to be intensified one of the following medications is additionally prescribed: oral mesalamine 2–3 g per day; *topical GCs* — prednisolone microenemas 30 mg per day or rectal budesonide 2–4 mg per day.

For refractory proctitis, *tacrolimus* is administered rectally at a dose of 1–4 mg per day. The duration of the induction course is determined individually, and maintenance therapy is possible at a dose of 1–3 mg three times a week rectally. Tacrolimus is a potent immunosuppressant belonging to the calcineurin inhibitor class. Mechanism of action involves binding to the FKBP-12 protein, forming a complex that inhibits calcineurin phosphatase activity. This prevents T-cell activation and suppresses immune responses. In UC it is used off-label for refractory proctitis.

Left-sided UC: for induction of remission first-line therapy is rectal mesalamine 1–2 g per day **in combination** with oral mesalamine at 2–4 g per day. Oral mesalamine monotherapy at a dose of 2–4 g per day is acceptable if there is a response to treatment.

If therapy needs to be intensified (blood in the stool persists within 2 weeks from the start of treatment), oral GCs are prescribed: prednisolone in the morning (40 mg per day for 1 week, then 30 mg per day for 1 week, then 20 mg per day for 1 month, followed by a dose reduction of 5 mg per week until complete withdrawal (the duration of GCs therapy should be at least 2 months but no more than 4 months)). Alternatively, methylprednisolone may be prescribed (32 mg per day for 1 week, 24 mg per day for 1 week, 16 mg per day for 1 month, then tapered by 4 mg per week).

Extensive UC: for induction of remission first-line therapy is rectal mesalamine 1–2 g per day in combination with oral mesalamine 2–4 g per day. Oral mesalamine monotherapy at a dose of 2–4 g per day is acceptable if there is a response to treatment.

In case of an insufficient response to treatment or if a relapse occurs while on maintenance therapy with mesalamine at a dose exceeding 2 g per day, one of the following GCs is prescribed: prednisolone or methylprednisolone orally in the morning in accordance with the previously described schemes. For inflammation in the distal parts of the colon and when therapy needs to be intensified, budesonide is prescribed rectally at 2–4 mg per day.

Induction of remission in severely active UC of any extent or lack of response to previous treatment. One of the following medications or a combination thereof is prescribed: prednisolone 60–90 mg per day intravenously until a response to treatment develops, but for no less than 5 days, then oral prednisolone in accordance with the previously described scheme; or/and biologics ((IL 12/23p40 antibody — ustekinumab; IL23p19 inhibitors — guselkumab, mirikizumab, risankizumab; gut-selective α4β7 integrin antagonist — vedolizumab; anti-TNF-α therapy — infliximab, adalimumab, golimumab) or/and sphingosine-1-phosphate (S1P) receptor modulators (ozanimod and etrasimod) or/and janus kinase (JAK) inhibitor (tofacitinib).

In the absence of a response to intravenous prednisolone at 60–90 mg per day for 7–14 days, one of the biologics or S1P receptor modulators and JAK inhibitor or intravenous cyclosporine at 2 mg/kg per day for 1 week, followed by a switch to oral administration at 4–8 mg/kg per day is prescribed, or surgical intervention (proctocolectomy) is performed.

Induction of remission in cases of a continuous UC refractory to GCs. Surgical intervention (proctocolectomy), or one of the following medications: azathioprine orally at 2–2.5 mg/kg per day; mercaptopurine orally 1–1.5 mg/kg per day; biologics or S1P receptor modulators and JAK inhibitor (either alone or in combination with purine antagonists).

The purine antagonists mercaptopurine and azathioprine possess pronounced immunosuppressive activity. In the body they are converted into active thioguanine nucleotides (e.g., 6-thioguanine triphosphate). These metabolites are incorporated into DNA and RNA, thereby inhibiting nucleic acid synthesis. This process preferentially affects rapidly dividing lymphocytes, leading to reduced proliferation of T-cells and B-cells, decreased production of pro-inflammatory

cytokines (such as TNF-α, IL-6, and interferon -γ), and suppressed cytotoxic T-cell activity. In UC, this immunosuppressive effect helps reduce the chronic mucosal inflammation characteristic of the disease, induces and maintains remission, and decreases the need for corticosteroids.

Maintenance of remission. 5-ASA-based therapy is first-line treatment, the dose and route of administration is determined individually by the prior response to treatment: rectal mesalamine no less than 3 g per week (may be used as monotherapy in proctitis); oral mesalamine no less than 2 g per day (as monotherapy or in combination with topical mesalamine — the total dose will then be at least 2 g daily orally plus at least 3 g weekly rectally).

In case of intolerance to 5-ASA-based therapy, their ineffectiveness (frequent and early relapses), steroid dependence (or steroid refractoriness), as well as in situations where induction therapy involved the use of cyclosporine or other immunosuppressants, the patient is prescribed either azathioprine orally at 2–2.5 mg/kg per day or mercaptopurine orally at 1–1.5 mg/kg per day.

The decision to continue biologic therapy or S1P receptor modulators and JAK inhibitors for maintenance of remission (using the same medicinal product with which remission was achieved) is made on an individual basis.

The effectiveness of maintenance therapy is assessed based on clinical, laboratory, and endoscopic parameters. Clinical remission is diagnosed under the following conditions: the stool is firm in texture, there is no blood in the stool, and no clinical or laboratory signs of inflammation.

Repeated measurement of FC levels can be used as an approximate criterion for remission — normalization of previously elevated levels indicates remission. The final determination of remission is made based on endoscopic findings.

Indications for surgical intervention in UC: lack of response to pharmacotherapy; risk of malignancy; presence of a malignant neoplasm of the colon in the setting of UC; fibrosis of the colon.

Indications for emergency surgical intervention in UC: perforation of the colon; intestinal bleeding (confirmed by a loss of more than 100 mL of blood per day, with a progressive decrease in red blood cells despite therapy); toxic dilatation of the colon (characterized by dilation of the colonic lumen of at least 6 cm with signs of toxicity, a sudden decrease in stool frequency against a background of previously present diarrhea, abdominal distension, and reduction of abdominal pain syndrome while symptoms of general toxicity (hypotension, tachycardia) increase).

Regular outpatient follow-up for patients with UC is provided continuously by gastroenterologists and primary care physicians. With the exception of patients who have isolated proctitis, individuals with UC are classified as being at increased risk for CRC. Risk factors for CRC include: male gender, young age at diagnosis, family history of CRC, combination of UC and primary sclerosing cholangitis, and extensive UC. For patients with UC screening colonoscopy is performed for CRC prevention.

In outpatient management of UC successful treatment and monitoring are accessed by several key outcomes: keeping patient in remission without the need for GCs, preventing complications, identifying any precancerous lesions or early-stage colon cancer, ensuring adequate nutritional status, and minimizing the loss of ability to work.

CROHN'S DISEASE

CD is a chronic inflammatory condition capable of involving any segment of the GI tract, from the mouth to the anus, though it most frequently affects the small bowel, particularly the terminal ileum.

The incidence of CD in different regions of the world varies from 0.3 to 20.2 cases per 100 000 population, with the highest prevalence in Europe (322 per 100 000). In Asia, the rates are lower, but they are also on the rise. The peak incidence occurs at 20–30 years of age, and a second peak at 60–70 years has been described. The incidence is approximately equal in men and women.

ETIOLOGY AND PATHOGENESIS

As with UC the exact cause of CD remains unknown, the disease arises from a complex interplay of several factors: hereditary predisposition, immune system dysfunction (both innate and adaptive), intestinal microbiota imbalance, and adverse environmental factors.

The cascade of humoral and cellular reactions in CD results in transmural inflammation of the intestinal wall, with the formation of sarcoid-like granulomas composed of epithelioid histiocytes without necrosis, along with multinucleated giant cells. In CD, any part of the gastrointestinal tract may be affected.

CLASSIFICATION

Montreal classification for CD including age of onset, location and behavior:

1. ***A****ge of onset:*
   – A1 ≤ 16 years;
   – A2 17–40 years;
   – A3 > 40 years.
2. ***L****ocation:*
   – L1 terminal ileum;
   – L2 colon;
   – L3 ileocolon;
   – L4 upper gastrointestinal (L4 modifier can be added to L1, 2 and 3 when concomitant upper gastrointestinal disease is present).
3. ***B****ehavior:*
   – B1 non-stricturing, non-penetrating (inflammatory);

– B2 stricturing (narrowing of the bowel);

– B3 penetrating (fistulizing, forming abscesses or tunnels);

– *p* perianal (p modifier can be added to B1, 2 and 3 when concomitant perianal disease is present).

By the extent of the bowel lesion:

– *localized lesion* — less than 30 cm of bowel affected;

– *disseminated lesion* — more than 100 cm of bowel affected.

Clinical assessment of activity (severity of exacerbation) is carried out using the CD activity index (CDAI, Table 2).

Table 2

Crohn's disease activity index

Parameter	Input and score	Multiplier
Number of liquid or soft stools (evaluated within a week)	Indicate the total number of loose stools over the past 7 days	2
Daily abdominal pain (evaluated within a week)	0 — none 1 — mild 2 — moderate 3 — severe	5
Patient well-being (evaluated within a week)	0 — very well 1 — slightly below par 2 — poor 3 — very poor 4 — terrible	7
Complications	No (0 points) Yes* arthralgia or arthritis iritis or uveitis erythema nodosum, pyoderma gangrenosum or aphthous ulcer fissures, anal abscesses or fistulas other fistulas fever during the previous week	20
Use of loperamide or opiates as anti-diarrheal	0 — No 1 — Yes	30
Abdominal mass	0 — none 2 — dubious 5 — present	10
Hematocrit < 0.47 in men or < 0.42 in women	0 — No 1 — Yes	6
Body weight (% change vs. the standard weight)	Indicate a percentage**	1

* Multiple selection, each selected complication is counted with 1 point.

** The % change vs. the standard weight = 100 · ((Standard-Current) / Standard), Standard weight for men = = (height in m)^2 · 22.1. Standard weight for women = (height in m)^2 · 20.8.

Interpretation of results: remission < 150 points; mild activity 150–220 points; moderate activity 220–450 points; severe activity > 450 points.

By the activity (severity of exacerbation):

– *remission*: CDAI < 150; CRP within the reference range;

– *mild activity*: CDAI 150–220 (hospitalization not required; patient is able to eat and drink without assistance; weight loss < 10 %; no signs of obstruction, fever, dehydration, abdominal mass, or tenderness; CRP exceeds the upper limit of the reference range);

– *moderate activity*: CDAI 220–450 (intermittent vomiting or weight loss > 10 %; failure of treatment or tender abdominal mass; no clear signs of obstruction; CRP exceeds the upper limit of the reference range);

– *severe activity:* CDAI > 450 (malnutrition, BMI < 18 kg/m^2, or signs of obstruction, or abscess; symptoms persist despite intensive treatment; CRP exceeds the upper limit of the reference range).

By the course:

– *with rare exacerbations* — exacerbations once or less per year;

– *with frequent exacerbations* — exacerbations 2 or more times per year;

– *continuous course* — persistence of symptoms for 6–8 months, despite adequate pharmacotherapy.

By response to treatment:

– *responding to treatment CD*;

– *steroid-dependent disease* — within 3 months of treatment, a reduction in the dose of prednisolone below 10 mg per day or budesonide below 3 mg per day leads to an exacerbation of the disease or the development of an exacerbation within 3 months after discontinuing GCs;

– *steroid-refractory disease* — active CD remains despite using prednisolone at a dose of 0.75 mg/kg per day for 4 weeks or more;

– *refractory to immunomodulators CD* — persistent disease activity or development of an exacerbation despite using thiopurines for at least 3 months (azathioprine 2–2.5 mg/kg per day or mercaptopurine 0.75–1 mg/kg per day in the absence of leukopenia) or biologics.

DIAGNOSTIC EVALUATION

The diagnostic process in CD as in UC involves clinical assessment, direct visualization of the colon, histopathological examination of biopsy samples.

The following aspects considering **complaints and medical history** should be emphasized during the interview: stool characteristics, daily stool volume and duration of symptoms, location and nature of abdominal pain, recent travel to regions with a hot climate use of medications, family history, smoking.

Physical examination. The physical examination algorithm for patients with CD does not differ from that for UC.

Based on the assessment of complaints, medical history data, and physical examination findings, the *clinical criteria for CD are*:

– *intestinal manifestations* — chronic bloody or non-bloody diarrhea and/or constipation; crampy abdominal pain (usually right lower quadrant, acute

manifestation of the disease can clinically resemble appendicitis), nausea, and vomiting;

– *general signs of inflammation and metabolic disorders* — malabsorption and weight loss, dehydration, hypoproteinemia, hypoalbuminemia, electrolyte disturbances fever, tachycardia, increased ESR, leukocytosis, thrombocytosis, increased CRP, fibrinogen, anemia;

– *extraintestinal manifestations* — eye lesions (episcleritis, anterior uveitis), skin lesions (pyoderma gangrenosum), arthritis, migratory monoarticular arthritis, ankylosing spondylitis, sacroiliitis, idiopathic thrombocytopenic purpura, osteoporosis, gallstones;

– *perianal lesions* — fistulas; abscesses; anorectal strictures;

– *CD complications* — bowel obstruction, intestinal bleeding, toxic dilation of the colon, intestinal perforation, abdominal and/or retroperitoneal abscess, intestinal fistulas, CRC.

Instrumental diagnostic studies. *Colonoscopy* — the most typical endoscopic manifestations in CD include: discontinious inflammation of the intestinal mucosa (aphthous lesions, ulcers, cobblestone appearance, luminal narrowing and deformation, strictures, pseudopolyps, fistula openings); focal involvement (within a single anatomical segment, the mucosa is not affected throughout); involvement of the ileum with sparing of the rectum.

Histological criteria for CD include chronic, transmural, discontinious (affected intestinal segments alternate with unchanged ones), focal (present not in all fragments taken from one anatomical segment of the intestine, unevenly distributed within one biopsy fragment) mucosal inflammation; epithelioid cell granulomas without multinucleated giant cells and necrosis, located in the lamina propria of the mucous membrane; decreasing gradient of inflammatory changes from the right to the left; lesion of the ileum.

Upper endoscopy reveals upper gastrointestinal tract involvement.

Abdominal ultrasound rules out pathology of other abdominal organs.

Intestinal ultrasound is recommended as the additional examination method for patients with CD to assess the extent of the lesion when colonoscopy is not possible or for a preliminary assessment of small bowel involvement.

Transrectal ultrasound is recommended as the additional examination method for perianal lesions.

CT enterography, or MR enterography are recommended to assess the location, extent, inflammatory activity, abdominal infiltrates, fistulas, perforations, and strictures.

MRI of the pelvis is recommended to assess perianal fistulas.

Capsule enteroscopy is recommended to assess small bowel involvement.

Optical coherence tomography may be used to differentiate between UC and CD.

Plain abdominal radiography is recommended for patients with severe activity when complications need to be ruled out.

Laboratory diagnostic studies. *CBC* may reveal: anemias (iron deficiency anemia, anemia of chronic inflammation, vitamin B12 deficiency anemia, or folate deficiency anemia); leukocytosis (caused either by chronic inflammation or by steroid therapy); thrombocytosis; an elevated ESR.

Blood biochemistry (bilirubin; total protein; AST; ALT; alkaline phosphatase; GGT; CRP; urea; creatinine; glucose; cholesterol; iron; ferritin) used to assess general signs of inflammation and metabolic disorders.

FC marker of inflammation and *faecal immunochemical test* (to detect hidden human blood in stool).

To rule out infectious causes the following tests are performed: *rectal swab for pathogenic microflora, stool analysis for helminth eggs and intestinal protozoa* (during the initial presentation or in cases of an atypical (severe) CD), *detection of Campylobacter and E. coli O157:H7 antigens in stool* (during the initial presentation or in cases of an atypical (severe) CD), *detection of Yersinia enterocolitica antigens in feces and/or a blood test for antibodies to Yersinia enterocolitica* (at the initial consultation in case of small bowel damage), *stool test for Clostridioides difficile toxins* (during the initial presentation, in cases of an atypical (severe) CD, and/or if development of this infection is suspected), *detection of Cytomegalovirus infection* (in cases of an atypical (severe) CD and/or when deciding on the prescription of biologic therapy) using biopsy specimens obtained during esophagogastroduodenoscopy and ileocolonoscopy.

Saccharomyces cerevisiae antibodies (ASCA) in serum performed if differentiating between UC and CD is challenging.

TREATMENT

Treatment of CD pursues the following goals: achieving and maintaining corticosteroid-free remission (clinical and endoscopic), prevention of CRC, correction of concomitant disorders and complications.

The treatment strategy for patients with CD depends on the following: the activity of the disease (severity of exacerbation), the extent and localization of the inflammatory process in the GI tract, the presence of extraintestinal manifestations and intestinal complications, the risk of the developing of complications, the presence of risk factors for an unfavorable prognosis at the onset of CD (indicating a disabling course of the disease within the next 5 years).

Induction of remission in CD. Lifestyle and dietary modifications (preliminary assessment of nutritional status and screening for malnutrition are required) during the period of induction of remission in all patients with CD include: smoking cessation, restriction of dietary fiber, daily protein intake of 1–1.5 g/kg body weight. In patients at high nutritional risk or with high disease activity enteral nutrition (500–1000 kcal/day) is prescribed additionally. In cases of high disease activity, a switch to exclusive enteral nutrition is undertaken.

Mild ileocecal and colonic CD: controlled-ileal release budesonide (orally 9 mg per day for 8 weeks) used to provide induction of remission in patients with mild to moderate CD limited to the terminal ileum and right colon.

Moderate ileocecal and colonic CD: oral GCs are prescribed: prednisolone in the morning (40 mg per day for 1 week, then 30 mg per day for 1 week, then 20 mg per day for 1 month, followed by a dose reduction of 5 mg per week until complete withdrawal (the duration of GCs therapy should be at least 2 months but no more than 4 months)). Alternatively, methylprednisolone may be prescribed (32 mg per day for 1 week, 24 mg per day for 1 week, 16 mg per day for 1 month, then tapered by 4 mg per week).

Severe ileocecal and colonic CD: oral or parenteral corticosteroids or biologics (IL12/23p40 antibody — ustekinumab; IL23p19 inhibitors — guselkumab, mirikizumab, risankizumab; gut-selective α4β7 integrin antagonist — vedolizumab; anti- TNF-α therapy — infliximab, adalimumab, certolizumab pegol) or JAK inhibitor (upadacitinib) prescribed in combination with purine antagonists or methotrexate for short-term remission induction. Also surgical intervention performed in case of ileocecal or colonic lesions with high activity or in the absence of a response to previous treatment.

Small-bowel CD: surgical intervention or oral/parenteral corticosteroids or biologics or JAK inhibitor in combination with purine antagonists or methotrexate for short-term remission induction. Nutritional support using enteral nutrition formulas, and if necessary, parenteral nutrition is provided.

Perianal/fistulizing CD: for patients with simple perianal fistulas as a primary therapy antibiotics are prescribed. Imidazoles (metronidazole orally 500 mg 2–3 times daily), or ciprofloxacin (orally 500 mg twice daily) for 6–8 weeks. If ineffective, one of the following drugs or their combination is prescribed: azathioprine orally 2–2.5 mg/kg daily, or mercaptopurine orally 1–1.5 mg/kg daily, for at least 3 months; biologics or JAK inhibitor.

If such signs of an unfavorable prognosis at the onset of CD as perianal fistulas, penetrating CD, extensive small bowel disease are identified the treatment strategy assumes early initiation of immunomodulators or biologics.

Maintenance of remission. Maintenance therapy in CD includes continuing the same biologics or JAK inhibitor on which remission was achieved, in combination with one of the immunosuppressants (azathioprine, mercaptopurine, or methotrexate at appropriate doses). If the effectiveness of the biologics or JAK inhibitor decreases, dose escalation, switching to another biologic agent, or surgery is undertaken.

The effectiveness of maintenance therapy is assessed based on CDAI (< 150) and endoscopic healing of the mucosa.

Indications for surgical intervention in CD: acute complications (uncontrollable intestinal bleeding, perforation, toxic dilation of the colon, abscesses); chronic complications (strictures, abdominal infiltrate, intestinal fistulas, neoplasms); as well as resistance to drug therapy.

Regular outpatient follow-up for patients with Crohn's disease is provided continuously by gastroenterologists and primary care physicians. Individuals with CD are classified as being at increased risk for CRC. For patients with CD screening colonoscopy is performed for CRC prevention. Screening colonoscopy is performed during remission and is accompanied by biopsy or chromoendoscopy with biopsy from suspicious areas.

As in patients with UC in CD successful treatment and monitoring are accessed by keeping patient in corticosteroid-free remission, preventing complications, identifying any precancerous lesions or early-stage colon cancer, ensuring adequate nutritional status, and minimizing the loss of ability to work.

SELF-MONITORING OF TOPIC ACQUISITION

1. Which of the following is a characteristic feature of US:

a) transmural inflammation with granulomas;
b) skip lesions alternating with normal mucosa;
c) continuous inflammation starting in the rectum;
d) perianal fistulas as a common finding?

2. What is the first-line therapy for inducing remission in mild to moderate proctitis:

a) oral prednisolone 40 mg/day;
b) rectal mesalamine 1–2 g per day;
c) intravenous cyclosporine;
d) oral budesonide 9 mg/day?

3. According to the TMC, which score range indicates moderate disease activity:

a) < 2 points;
b) 3–5 points;
c) 6–10 points;
d) > 10 points?

4. Which of the following is a histological criterion for UC:

a) epithelioid cell granulomas without necrosis;
b) transmural lymphoid aggregates;
c) crypt architectural distortion (branching, shortening);
d) skip lesions in the biopsy specimen?

5. In UC, steroid-refractory disease is defined as:

a) relapse within 3 months after stopping GCs;
b) need for prednisolone >10 mg/day to maintain remission;
c) persistent disease despite prednisolone 0.75 mg/kg/day for ≥ 4 weeks;
d) no response to intravenous GCs after 1 day.

6. For left-sided UC, if blood in the stool persists after 2 weeks of mesalamine, what is added:

a) oral glucocorticosteroids (e.g., prednisolone);
b) infliximab 5 mg/kg;
c) methotrexate 15 mg/week;
d) topical tacrolimus?

7. Which medication is recommended for maintenance of remission in UC:

a) prednisolone;
b) mesalamine;
c) metronidazole;
d) ciprofloxacin?

8. Which of the following is an extraintestinal manifestation of UC:

a) gallstones;
b) sclerosing cholangitis;
c) perianal fistulas;
d) epithelioid granulomas?

9. An emergency surgical indication in UC is:

a) chronic stricture without symptoms;
b) toxic dilation of the colon;
c) internal fistulas;
d) perianal abscess.

10. Which factor is identified as a risk factor for colorectal cancer in patients with UC:

a) isolated proctitis;
b) female gender;
c) extensive colitis;
d) age at diagnosis > 60 years?

11. Which part of the gastrointestinal tract is most frequently affected in CD:

a) stomach;
b) duodenum;
c) terminal ileum;
d) rectum alone?

12. In the Montreal classification for CD, B3 (behaviour) stands for:

a) non-stricturing, non-penetrating;
b) stricturing;
c) penetrating;
d) perianal disease.

13. According to the CDAI, remission in CD is defined as a score of:

a) < 150 points; c) 220–450 points;
b) 150–220 points; d) > 450 points.

14. Which histological finding is characteristic of CD:

a) increasing gradient of inflammatory changes from the right to the left;
b) no lesion of the ileum;
c) epithelioid cell granulomas without necrosis;
d) mucin depletion only?

15. For mild ileocecal CD limited to the terminal ileum and right colon, which agent is recommended for induction of remission:

a) oral prednisolone 40 mg/day;
b) controlled-ileal release budesonide 9 mg/day;
c) infliximab 5 mg/kg;
d) metronidazole monotherapy?

16. Which antibiotic is first-line for simple perianal fistulas inCD:

a) vancomycin;
b) metronidazole;
c) clarithromycin;
d) doxycycline?

17. Which of the following is a sign of unfavorable prognosis at the onset of CD, prompting early immunomodulator/biologic therapy:

a) isolated colonic disease;
b) age > 40 years;
c) perianal fistulas;
d) inflammatory (non-stricturing, non-penetrating) behavior?

18. What is a stated goal of treatment for CD:

a) lifelong low-dose corticosteroid therapy;
b) achieving and maintaining corticosteroid-free remission;
c) complete surgical removal of all affected bowel;
d) exclusive enteral nutrition for all patients?

19. Which imaging study is recommended to assess perianal fistulas in CD:

a) CT enterography;
b) capsule enteroscopy;
c) MRI of the pelvis;
d) plain abdominal radiography?

20. An indication for surgical intervention in CD is:

a) mild activity with CDAI = 160;
b) uncontrollable intestinal bleeding;
c) isolated aphthous ulcers in the terminal ileum;
d) successful response to infliximab.

Answer key: 1 — c; **2** — b; **3** — c; **4** — c; **5** — c; **6** — a; **7** — b; **8** — b; **9** — b; **10** — c; **11** — c; **12** — c; **13** — a; **14** — c; **15** — b; **16** — b; **17** — c; **18** — b; **19** — c; **20** — b.

REFERENCES

1. *Клинические* рекомендации: болезнь Крона // Ассоциация колопроктологов России, Российская гастроэнтерологическая ассоциация. – URL: https://congress-med.ru/assets/files/%D0%BE%D0%B1%D0%BD%D0%BE%D0%B2%D0%B0/1/2024-klinicheskie-rekomendaczii-mz-rf-po-diagnostike-i-lecheniyu-bolezni-krona.pdf (дата обращения: 15.03.2026).

2. *Клинические* рекомендации: язвенный колит // Ассоциация колопроктологов России, Российская гастроэнтерологическая ассоциация. – URL: https://congress-med.ru/assets/files/%D0%9F%D0%BE%D0%BB%D0%B5%D0%B7%D0%BD%D0%B0%D1%8F%20%D0%B8%D0%BD%D1%84%D0%BE%D1%80%D0%BC%D0%B0%D1%86%D0%B8%D1%8F/2024/10.08.24/2024-klinicheskie-rekomendaczii-mz-rf-po-diagnostike-i-lecheniyu-yazvennogo-kolita.pdf (дата обращения: 15.03.2026).

3. *Об утверждении* клинического протокола «Диагностика и лечение пациентов (взрослое население) с заболеваниями кишечника» : постановление М-ва здравоохранения Респ. Беларусь от 11 нояб. 2025 г. № 185 // Министерство здравоохранения Республики Беларусь. – URL: https://minzdrav.gov.by/upload/dadvfiles/CProtokol/КП_Диагностика_лечение_пациентов_вз_нас_заболеваниями_кишечника_пост_МЗ_2025_185.pdf (дата обращения: 01.03.2026).

4. *Поликлиническая* терапия : учеб. / М. В. Зюзенков [и др.]. – 3-е изд., испр. – Минск : Выш. шк., 2022. – 623 с.

5. *ACG* Clinical Guideline: Management of Crohn’s Disease in Adults / G. R. Lichtenstein [et al.] // Am. J. Gastroenterol. – 2025. – Vol. 120. – P. 1225–1264.

6. *ACG* Clinical Guideline Update: Ulcerative Colitis in Adults / D. T. Rubin [et al.] // Am. J. Gastroenterol. – 2025. – Vol. 120. – P. 1187–1224.

CONTENTS

Abbreviations 3
Motivational characteristics of the topic 4
Definitions 5
Ulcerative colitis 6
    Etiology and pathogenesis 6
    Classification 7
    Diagnostic evaluation 9
    Treatment 12
Crohn’s disease 15
    Etiology and pathogenesis 15
    Classification 15
    Diagnostic evaluation 17
    Treatment 19
Self-monitoring of topic acquisition 21
References 24

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Каразей Елена Александровна
Алексеева Елена Сергеевна
Рылатко Елена Викторовна

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