Ulcerative colitis and Crohn's disease: modern views (part 2). Diagnostics and differential therapy

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Diagnostics. Timely diagnosis of ulcerative colitis (UC) and Crohn's disease (CD) is difficult. On average, these diseases are diagnosed through a 4,6±0,8 years after their start, due to nonspecific symptoms and the presence of diseases, in varying degrees, mimic UC and CD.

Methods of diagnosis and treatment of UC and CD were discussed at conferences European Crohn's and Colitis Organisation (ECCO) in 2006 and 2008 [9, 37, 38]. This organization, created in 2000, brings together experts studying UC and CD.

In the diagnosis of UC and CD, in addition to clinical symptoms, which has been described previously, using a variety of instrumental and laboratory methods, including morphological study of biopsy material.

The most important value in the diagnosis of UC belongs to endoscopic methods: sigmoidoscopy and ileocolonofibroscopy with biopsy.

In the words of visual endoscopic changes proposed to distinguish four degrees of UC.

I. **(Minimum) degree:** available inflammatory edema mucous shell congestion vessels (spotted hyperemia), punctulated hemorrhage, light contact bleeding.

II. **(Moderate) degree:** in addition to edema and hyperemia, marked expressed contact bleeding, defined grainy erosion, drain hemorrhage, fibrinous raid.

III. **(Expression) degree:** there are multiple merging erosion and flat ulcers on background described above changes mucous membranes, at lumen guts blood and pus.

IV. **(Sharply severe) degree:** those the changes bleeding ulcers, inflammatory pseudopolyps and saccular pseudodiverticulum; fibrinous — purulent plaque after remove whom uncovered diffusely bleeding grained surface multiple ulcers round and stellate forms not penetrating, however, deeper own Records mucous shell and her submucosal layers, combined from lots of erosions, creating impression pitted moth surface.

The remission UC stored grain and folds thickened mucosa.

X-ray examination (barium enema) noted the intestinal wall rigidity, lack of smoothness haustrum and relief; presence of inflammatory edema, ulceration, pseudopolyps; rearrangement mucosa with the presence of coarse longitudinal and transverse folds.

Survey abdominal radiography can detect such severe complications UC as toxic megacolon, flowing from the increase in the diameter of the colon to 10-14 cm or more, as well as intestinal obstruction and perforation of the colon.

UC is characterized by the continuous failure of the colonic mucosa and in 100% of cases lesion of the rectum. The anal region is affected in 25% of cases, and serosa remains intact; possible shortening of the colon.

Additional diagnostic data can be obtained using ultrasound, including Endoscopic ultrasound, computed tomography and magnetic resonance.

For CD is characterized by intermittent (segmental) defeat the intestines; the rectum is involved in the pathological process in 50% of cases, and the anal region — 75%; struck by the small intestine in 30%. Observed deep slit-like ulcers, giving the mucosa a cobblestone street views. Typically, affected bowel serosa (serositis, interintestinal adhesions), observed obstruction (stenosis) of the intestinal lumen, the presence of fistulas.

In the diagnosis of CD is also used ileo- and colonofibroscopy with biopsy and the histological study of biopsies, barium enema, radiopaque study of the small intestine. Recently, in the diagnosis of CD small intestine and used videocapsular balloon endoscopy [35, 36].

Diagnosis infiltrates, abscesses and fistulas is performed using magnetic resonance imaging, fistulography, capsule endoscopy and balloon.

In CD in the inflammatory process involved submucous nerve terminal, which is the main cause of abdominal pain [1, 5, 6, 7, 13, 27, 28, 51].

A morphological (histological) study of biopsies reveal accumulation of histiocytes and lymphocytes, forming microgranuloma. True granulomas are found in 50-80% of cases, localized in the submucosa and lamina propria [41]. They are clusters of epithelioid and giant cell histiocytic (Pirogov-Langhans), surrounded by a rim of lymphocytes without fibrosis without necrosis in the center [1].

In a laboratory study can detect anemia (iron, B_{12} or folic acid deficiency), acute phase proteins of inflammation (C-reactive protein, α1-antitrypsin, etc.).

Some importance in the laboratory diagnosis of UC and CD belongs immunoassay (ELISA) which detects the presence in the serum of perinuclear antineutrophil cytoplasmic antibodies (rANSA) and anti-Saccharomyces cerevisiae (ASSA); in the absence and presence of ASSA rANSA diagnose CD, and in the presence and absence of rANSA ASSA — UC [13].

Valuable data obtained with scatological and microbiological studies of feces. A significant increase in the concentration of calprotectin in feces observed in the course of active UC and CD, as well as intestinal polyps, and adenocarcinomas [27, 28, 44].

However, it should be recognized that the UC and CD are no pathognomonic diagnostic features. The difference between UC and CD in most cases is not so much qualitative as quantitative. In this regard, it was paradoxical sentence: to recognize the fundamental identity of UC and CD and treat them as variants of a single disease [20], however, agree with this point of view it is difficult, and now we consider it premature and ill-founded. Still, these are two different, though related diseases.

Differential diagnosis of UC and CD performed with numerous diseases of the colon and small bowel bacterial, viral, radiation, drug, and other nature

(infectious enterocolitis, pseudomembranous colitis, ischemic colitis, microscopic colitis — collagen, lymphocytic, diverticulitis, amebiasis, and others.).

Treatment. The goal of treatment — a resistant and long-term achievement of clinical remission, prevention of complications, improve the quality of life of patients. An important role is played by creating a favorable psychological climate, the psychological support of patients by a doctor.

Clinical nutrition is important in CD, flowing with the defeat of the small intestine, and malabsorption and maldigestion syndromes. It aims to provide a functional bowel rest and reducing the load on the affected functional its departments, elimination of food antigens from the intestinal lumen and recovery trophological status of patients and suppression of inflammation and reducing the permeability of the intestinal wall.

Basic principles of clinical nutrition at CD: in the diet should contain complete proteins, fats and carbohydrates; necessary to eliminate rough roughage and dairy products; should consider the individual patient intolerance of certain foods and dishes [1].

Recognition received a diet low molecular weight oligopeptide having low osmolarity, good taste, containing vitamins E, K, B_{12} and folic acid, and zinc, magnesium and iron.

In severe CD flowing syndromes maldigestion and malabsorption, and a sharp decrease in body weight (15%), the advisability of appointing short term parenteral nutrition.

At UC recommended the exclusion from the diet of dairy products. Upon constipation it is advisable to use dietary fiber. Useful fluid and electrolyte solutions. The need for parenteral nutrition in UC is rare [1, 5, 27, 28].

Pharmacotherapy. Conventionally distinguish basic therapy UC and CD: drugs 5-amino salicylic acid (5-ASA); corticosteroids (CS) systemic and local action; nonsteroidal immunosuppressants; Monoclonal antibodies to tumor necrosis factor α (TNF); Additional therapeutic agents: antibacterial (metronidazole, ciprofloxacin, rifaximin), pro- and synbiotics, tryptase inhibitors,

etc.; symptomatic agents: myotropic antispasmodics, sucralfate, clonidine, Calcium-D $_3$Nycomed et al. [37, 38].

When selecting medical means it is necessary to consider the presence of steroid and steroid resistance.

It is also important to know the effectiveness of previously used drugs, and their tolerance of the patient (side effects).

In mild and moderate forms of UC and CD is recommended to use a step diagram starting treatment with less active drugs, causing the minimum of side effects, while their inefficiency should be given more active agents, the use of which, however, is fraught with various undesirable (adverse) effects [13] .

The 5-ASA derivatives. The first drug of this group used in the treatment of UC and CD was sulfasalazine (25% of 5-ASA + 60-80% sulfapyridine), acting only in the colon and has anti-inflammatory and antibacterial properties. Because of the numerous (in 10-45% of patients) side effects its use is now restricted.

Mesalazine (salofalk, mesakol, pentasil, et al.) contains only 5-ASA. Each drug mesalazine has a different protective coating that releases the active ingredient at different pH values in certain parts of the gastrointestinal tract, it is important to know when the appointment of its various representatives. Mesalazine has a pronounced anti-inflammatory effect by inhibiting liposomes and cyclooxygenase pathway of arachidonic acid inhibits the formation of superoxide radicals by activated neutrophils possess immunomodulatory activity, blocks the synthesis of inflammatory mediators (cytokines, prostaglandins, leukotrienes, and others.), Inhibits migration, neutrophil degranulation and phagocytosis and production of immunoglobulins (Ig) lymphocytes et al. [44].

Indications: mild and moderate UC and (partly) CD, especially in the presence of proctitis and proctosigmoiditis, when mesalazine is taken orally and is used as a therapeutic micro-enemas and suppositories.

Doses: mesalazine drugs produce a dose-dependent effect and apply to the UC in a dose of 3 g/day, while CD — 4.0-4.5 g/day for 8 weeks, followed by maintenance therapy at a dose of 1.5-2 g/day for 2 years at UC and 3-4 years at

CD. Recurrences after discontinuation of 5-ASA throughout the year reaches 80%. Side effects are detected in no more than 10% of cases. Mesalazine can be combined with CS (prednisone, budesonide), modulators of intestinal motility (trimedat), vitamins B, C and PP. The effect of mesalazine appears in relieving acute UC and 1F and the achievement of clinical and endoscopic remission in 65.4% of patients; prevention of early relapse [2, 5, 8, 11, 17]. When widespread, severe and complicated forms of UC and CD mesalazine monotherapy is ineffective [1].

Salofalk tablets have an enteric coating. Dose: 250 and 500 mg 3-4 times a day before meals. Released in the ileum and the mid transverse colon. Commonly used in CD with said localization process. When UC is preferable to use in the granules according salofalk 0,5-1,0-1,5 g (at a dose of 3 g/d), which are uniformly deallocated operate only in the colon, taken one time a day in the morning regardless of the food. When left-sided colitis salofalk used as micro enema (based on 4 g product per 60 ml or 2 g product per 30 ml) acting to splenic angle colon, or in the form of suppositories (0.25 and 0.5 g) appointed when proctitis; efficiency — 79.4%.

Pentasil is mesalazine microgranules coated with ethylcellulose shell slow release of the active substance, which starts to be released at pH 1.0 in the stomach and duodenum to act on the colon. The ileum is retained only 60-70% active drug, and colon — 50%. In CD pentasil administered in a dose of 4-6 g/day regardless of the presence of diarrhea and intestinal dysbiosis, which occurs in 38-84% of patients [2, 8, 11, 17, 48]. Pentasil microclyster may be administered (at a dose of 1 g in 100 ml).

Asacol (mesakol) tablets of 0.4 g and 0.8 g is only released in the large intestine at pH>7.0. Diarrhea, when the pH is less than 7.0, the effectiveness of the drug is greatly reduced.

In recent years there has been new formulations of mesalazine, of which the most promising mezavant. It is characterized by slow-release mesalazine with multimatrichnoy delivery system over the entire length of the colon, including

direct, eliminating the purpose of medical micro-enemas or suppositories. The drug has a core which is surrounded by an envelope consisting of methacrylic acid copolymer A and B and releasing mesalazine in the ileocecal region of the intestine, forming a gel mass at a slow diffusion of mesalazine in the lumen; dose of 3.6 or 4.8 g (3-4 tablets) 1 times a day for 8 weeks. After remission supporting therapy at a dose of 2.4 g of 1 times a day for one year or more. Clinical and endoscopic remission in mild to moderate forms of UC was achieved in 41.2% of patients and maintained throughout the year at 67.8% [4, 47].

Corticosteroids. Distinguish system CS (prednisone or methylprednisolone — metipred) and local CS (budesonide Budenofalk).

CS have a powerful anti-inflammatory and immunosuppressive effects. By binding to the receptors of the cytoplasm of cells, stimulate the synthesis of lipocortin CS — a protein which inhibits the enzyme phospholipase $_{A}$ 2 and thus inhibits the formation of arachidonic acid and its metabolites. The latter are active mediators histamine release from mast cells and basophils; inhibit the phagocytic activity of macrophages; inhibit formation of proinflammatory cytokines — interleukin (IL) 1, 2, 6, 8, and TNF production adhesin molecules [1, 5, 21, 27, 34, 43].

Action prednisone (tablets 5 mg), taken by mouth, continued for 36 hours. The drug is prescribed for UC and CD moderate to severe dose of 1-1.5 mg/kg per day inside, and to achieve a rapid effect is administered intravenously (at a dose of 240-300 mg/day). When the distal lesions of the bowel can be used as a CS microenema and suppository. After achieving clinical remission (usually 3-4 weeks of treatment), the dose is gradually reduced CS (2.5-5 mg every 5-7 days) with gradual withdrawal of the drug. The duration of treatment is typically 8-10 weeks.

Metipred (tablets of 4 mg) has mineralocorticoid properties inherent prednisolone, and more active (48 mg metipred corresponds to 60 mg prednisolone).

The main side effects of systemic CS (Cushing's syndrome, osteoporosis, cataracts, ulcers of the stomach and intestines, the activation of latently occurring infections including tuberculosis, breach of tolerance to sugars, high blood pressure, etc.) are developed independently of the dose. Known withdrawal system CS flowing in some cases with the development of acute adrenal insufficiency, fever, anorexia, arthralgia, and others.

Budesonide tablets (Budenofalk capsules) administered 3 mg 3 times a day, usually at moderate and moderate forms of UC and CD, often with terminal ileotiflite and with the defeat of the ascending colon. It is believed that corresponds to 9 mg of budesonide to 40 mg prednisolone. Taken internally, budesonide has a pronounced anti-inflammatory and immunosuppressive effects. It is well absorbed in the intestine, easily penetrating the intestinal wall (up to 88%), which binds to the receptors (its affinity for steroid receptors is 20 times higher than metipred). After picking up 90% of budesonide is metabolised in the liver, turning into inactive metabolites. It does not suppress the adrenal function, and therefore it has a 2-fold fewer side effects than prednisolone. As CS topical budesonide provides the therapeutic effect only in the segment of the intestine, which he achieved. Therefore, it is inappropriate to prescribe in advanced lesions of the intestine and the presence of extra-intestinal lesions; therapeutic dose of budesonide 9 mg/day (up to 10 weeks). The drug gives a dose-dependent effect. Increasing the dose of budesonide in severe CD to 18 mg/day increases the effectiveness of therapy in 1.5-3 times. Clinical remission was achieved in 51-60% of patients, which is not inferior to the effect of prednisolone ($p>0.5$).

If it affects the distal colon budesonide prescribe medicinal microclyster the rate of 2-3 mg per 100 ml [32, 49]. Efficiency of 2 mg budesonide microclyster 2 times higher than that of prednisolone 30-25 mg. For the prevention of UC and CD using the CS.

In connection with CS therapy UC and CD were two clinical concepts: steroid resistance and steroid dependence.

Steroid resistance — is the lack of effect in the treatment of adequate doses of the CS for 7-21 days. It occurs in 20-50% of patients.

Steroid dependence is an inability to reduce the dose of the CS (up to 10 mg/day of prednisone and 3 mg/day budesonide) without reactivation of the inflammatory process in the intestine with the development or recurrence of UC CD for 3 months after treatment of the CS [13, 37, 38].

Non-steroidal immunosuppressants. This group of drugs include azathioprine and 6-mercaptopurine (6-MP), methotrexate and cyclosporin A. They are used to treat active UC and CD forms to induce clinical remission and maintain it, and to overcome steroidorezistentnosti and steroid [12, 21]. Non-steroidal immunosuppressive drugs are sometimes called reserve.

Azathioprine is cytostatic (antimetabolite), a precursor 6-MP: 88% of it is converted to 6-MP under the influence of the sulfhydryl compounds. The mechanism of therapeutic action is not exactly set. In conjunction with the CS, they are responsible for the achievement of clinical and morphologic remission in 56-65% of patients with UC and CD, including the refractory forms of the disease, and the overcoming of steroid 38%.

The therapeutic dose of azathioprine 2-2.5 mg/kg per day and 6-MP — 1-1.5 mg/kg per day; course duration 15-18 weeks (6 months), and then move on to a maintenance dose of azathioprine 1-1.5 mg/kg per day (up to 4 years) [31].

Methotrexate — analogue dehydrocholic acid is a cofactor in the synthesis of pyrimidine and purine bases, and formation of methionine. Its therapeutic effect is characterized by an immunosuppressive effect on neutrophil function, formation of pro-inflammatory cytokines (IL-2, IL-8, TNF) and IL-1 by binding to the target cells. Methotrexate enhances anti-inflammatory cytokine IL-10.

The recommended dose is 10-25 mg per week intravenously or intramuscularly; course up to 16 weeks. The drug is not effective when taken orally. Applicable only in CD. In 39.4% of cases achieved clinical and morphologic remission is possible to "get away" from the steroid and improve the quality of life of patients.

Side effects: diarrhea, stomatitis, infectious complications, degeneration of hematopoiesis [33].

Cyclosporin A is a powerful immunosuppressant that inhibits immune responses that mediate inflammation and T-lymphocyte immune response. Inhibits the formation of pro-inflammatory cytokines (IL-2, interferon γ, etc.), And factors which activate the T-helper cells. Cyclosporine A may be used in UC and CD in treatment failure CS within 5-7 days in a dose of 2-4 mg/kg per day (intravenous injection of 50 mg/100 ml); Good effect is observed in 42% of cases. When administered in capsules, the dose is 5-15 mg/kg per day. When steroid drug is effective in 50-80% of patients; 50% eliminates the need of surgery. It has mainly systemic effect, local application inefficient. The course of treatment varies from 4-5 days to 3 months. For maintenance therapy, the drug is not used.

Adverse events observed in 12% of patients: paraesthesia, hypertrichosis, tremor, anorexia, nausea, vomiting, hepato- and nephrotoxicity [1, 5, 6, 21, 27, 51].

Tacrolimus by the action similar to cyclosporine A, but unlike the latter is well absorbed in the gastrointestinal tract. The drug can be used in UC and CD rate of 0.1-0.2 mg/kg per day orally or 0.01-0.02 mg/kg per day intravenously. Its effect is superior to cyclosporine A many times. The drug is safe, but can cause increases in serum creatinine.

Biological therapy. The treatment of UC and CD in recent years using fundamentally new biological drugs: chimeric IgG_1-monoclonal antibodies that inhibit the molecular mechanisms of inflammation in the intestines. They bind (neutralized) soluble and fixed on the cell membrane TNF — a key mediator of inflammation, lyse macrophages and induce apoptosis of activated T-lymphocytes by increasing the synthesis of anti-apoptotic molecule Bcl-2 and inhibit the production of proinflammatory cytokines (IL-2 and interferon-γ), eliminating the antibody-dependent cytotoxicity.

From this group of drugs most studied infliximab (Remicade). Infliximab is used in the early stages of the disease, mostly in moderate and severe forms of CD and UC, with complications occurring and extraintestinal lesions refractory to

immunosuppressive therapy, as well as steroid. The effect increases as the combination of infliximab with azathioprine: 2.5 mg/kg daily for 6-12 months. At relapse of the disease, treatment can be repeated at intervals of not more than 16 weeks (otherwise increase the risk of developing allergic reactions). If there is no effect after 12 weeks of treatment, the drug should be discontinued.

Infliximab is administered (by means of special equipment) intravenously at a dose of 5 mg/kg per day; course of treatment — 3 injections: 1st, 2 and 6 weeks. after the 1st administration. In a subsequent infusion of the drug produced every 8 weeks. for 6-12 months.

Clinical and morphological remission was achieved in 18-67% of patients; 29% of patients with steroid-dependent can't cancel the CS, and 90% — to reduce their dose.

When CD Best Activity Index is reduced by 70-150 points, and quality of life (SF-36 questionnaire) — 60 points or more. In patients with fistula form CD marked the elimination of external and internal fistulas (after 2 weeks. — 31% after 6 weeks. — 43% after 14 weeks. — 48%), and systemic manifestations of CD and UC will be able to eliminate at 30-40% of patients. In some cases treatment by infliximab allows to avoid hospitalization and surgery [42, 45].

Adverse effects: heart failure, delayed allergic reactions, exacerbation of latent infection (including TB), the increased risk of developing lymphoma [1, 5, 11, 14, 18, 22, 44].

Additional therapeutic agents — antibiotics (metronidazole, ciprofloxacin, rifaximin) used in UC because of the development in patients with colonic dysbiosis and loss of immunological tolerance to intestinal microflora, which affects the clinical course of UC. Metronidazole is used more often in CD. A successful is a combination of metronidazole with ciprofloxacin. Metronidazole 500-750 mg is prescribed 2 times a day, ciprofloxacin — 300 mg 2 times per day (rates — on the testimony, sometimes long). Gain recognition and is not absorbed antibiotic rifaximin and intestines (200-400 mg 2-3 times a day). Antibiotics have no effect on the outcome of UC and CD, but their adequate application makes it

easier to achieve clinical remission and reducing the number of exacerbations [10, 13].

Indications: ileocolitis light and moderate flow; complications state after resection of the terminal ileum, toxic megacolon, purulent complications. With long-term antibiotic therapy may develop side effects. Reaction to antibiotics is observed in 40-50% of patients [1, 11, 15, 46, 50].

Probiotics are involved in the protection of intestinal epithelium are antagonists opportunistic bacteria secretor IgA increase the production, reduce the permeability of the intestinal walls, restoring its barrier function. Bifiform, Linex and other symbiotics containing bifidobacteria and lactobacilli, take 2 capsules 2 times daily rates for a long time. They contribute to the induction and prolongation of remission in UC and CD [12, 23, 40].

In the publications of domestic and foreign authors as additional and symptomatic treatment are recommended tryptase inhibitor (drug JEM-2059) [44], somatostatin (octreotide) — reduces the permeability of the intestinal wall, the index of activity in UC and CD). Somatostatin and its analogues (octreotide) increase the likelihood of closing the intestinal fistulas and reduce the time of liquidation, although no effect on total mortality; debridat (trimebutine) — an antagonist of opiate receptors, which normalizes enkephalinergic system of regulation of motor function of the gastrointestinal tract irrespective of the initial state of the motor; other (smectite, de-nol — bismuth tridicitratobismuthate); in UC can be further applied tsileygon — lipoxygenase inhibitor, which allows to reduce the dose of the CS; eykozopentanovuyu acid increases the synthesis of leukotriene B_5 and reduces the level of leukotriene B_4, which is used together with 5-ASA and the CS; short chain fatty acids (oleic acid) — butyrate enema and zakofalk (250 mg of calcium butyrate + 250 mg of inulin prebiotic) 2-4 tablets a day before meals, with liquid (at least 4 weeks). In severe UC hemosorbtion and plasmapheresis are effective [24, 26].

Recently, as an adjuvant (supporting) therapy of UC and CD recommend the use of melatonin at a dose of 3 mg, which is taken in the late evening hours,

combined with basic therapy for 3-4 weeks. Melatonin regenerates and synchronizes biological rhythms (daily periodization) provides anti-stress effect, reduces immune hyperactivity has a marked antioxidant effect and possesses anti-inflammatory activity, inhibiting the production of proinflammatory cytokines, reduces the side effects of other pharmacological agents [3, 19, 30, 39].

Tactics of treatment for various forms of UC and CD is presented in the recommendations of the European Organization for the diagnosis and treatment of inflammatory bowel disease (ECCO) [9, 25, 37, 38].

Recommendations for treatment of CD [22, 37]:

1. When the terminal ileite and typhlitis mild drug of choice is budesonide (Budenofalk) at a dose of 9 mg/day orally.
2. In the treatment of moderate CD recommended budesonide at the same dose or prednisolone (1 mg/kg per day); with the risk of infection treatment supplement antibacterial agents (metronidazole, ciprofloxacin, rifaximin, etc.).
3. Severe CD requires use (orally or intravenously) prednisolone in combination with non-steroidal immunosuppressants (azathioprine or 6-MP, while their ineffectiveness or intolerance — to metotre KSAT) have a steroid saving effect. In the absence of a transition effect to the infusion of infliximab, which often allows to avoid surgery.
4. In CD, the colon is recommended mesalazine (salofalk granules, mezavant) at a dose of 4 g/day, while no effect — prednisone 1 mg/kg per day.
5. In early relapse of CD it is recommended to supplement the treatment with azathioprine or 6-MP, and in their intolerance — methotrexate; a successful combination of infliximab + azathioprine; some researchers feel justified supplementation metronidazole (the rate of 10 — 20 mg/kg at day).
6. With widespread lesions of the small intestine are the system of choice in conjunction with CS nonsteroidal immunosuppressants (azathioprine, methotrexate, and others.).

7. If you have extra-intestinal manifestations of CS appoint, azathioprine, infliximab. In primary sclerosing cholangitis ursodeoxycholic acid is prescribed (at the rate of 20 mg/kg per day).

8. Upon steroid resistance forms and CD azathioprine and/or infliximab are used (5 mg/kg); upon their inefficiency metotreksat is prescribed [9, 25, 37, 38].

9. Supportive CD therapy by means of azathioprine (the rate of 2-2.5 mg/kg per day).

Upon ileitis 5-ASA drugs are used (tablets of salofalk).

Recommendations for the treatment of UC [38]:

1. Upon UC of mild and moderate severity with the expression of distal colon (proctitis, proctosigmoiditis) recommended the appointment of 5-ASA preparations (mesalazine, salofalk) in the form of suppositories and medical mikroklizm (2-4 g/day).

2. Upon common forms of UC a combination of mesalazine is prescribed (including its area of action) for oral and per rectum as microclysters (3-4 g/day).

3. In the absence of effect within 10-14 days and under heavy current UC prednisolone is appointed at the rate of 1 mg/kg per day orally or intravenously (240-300 mg/day).

4. If you have steroid resistance and/or erased steroid dependence it is recommended to appoint additional azathioprine or 6-MP, and in the case of inefficiency — infliximab.

5. For maintenance of remission in distal forms of UC (proctitis, proctosigmoiditis) mesalazine is used as suppository and microclyster (dose of 1.5-2 g/day) for1 year or more, in advanced lesions of the colon — mesalazine acting in the colon (salofalk in the granules at a dose of 1.5-2 g/day or mezavant at a dose of 2.4 g/d) for 1 year or more.

6. With early treatment of recurrent UC complement nonsteroidal immunosuppressants (azathioprine rate of 2.5 mg/kg per day) [25, 38].

The indications for surgical treatment of UC and CD are ineffective complex pharmacotherapy; complications: massive bleeding; toxic megacolon, not amenable to medical and endoscopic correction; perforation, abdominal abscesses, peritonitis, intestinal obstruction, fistulas, and the presence of signs of chronic obstructive; malignancy process.

UC patients within the first year need for surgical treatment in 10% of cases, after 10 years — 23%, and after 25 — 32%. At UC as the operation of choice recommended subtotal resection of the colon with the formation and ileo-sigmoidostoma. Subsequently, addressed the issue of the possibility of reconstructive surgery or proctectomy [16]. In patients with UC if possible surgical removal of the entire affected area there are no relapses.

70-80% of patients with CD require surgery. Operations performed in the CD, are diverse and depend on the location of the lesion, the nature and severity of complications [1]. CD cure through surgery is not possible, since 40% of patients within 6 months. If you cancel after the operation maintenance therapy develop recurrences. When conducting an adequate anti-treatment CD marked decrease in the incidence of postoperative complications from 34 to 13%, and recurrence of the disease — from 55 to 24%.

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Ulcerative colitis and Crohn's disease: modern views (part 2). Diagnostics and differential therapy

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Key words: ulcerative colitis, Crohn's disease, etiology and pathogenesis, classification, diagnostics, treatment

Definitions of ulcerative colitis (UC) and Crohn's disease (CD) are given, related terminological problems are discussed, prevalence of UC and CD in the population is considered along with their etiology, pathogenesis, clinical symptoms, complications and extraintestinal (systemic) lesions. Classification and diagnostics of UC and CD are discussed with special reference to current international recommendations on their diagnostics and differential treatment, rejuvenation and extraintestinal lesions.