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Кафедра Внутрішньої медицини №3
Факультет VI по підготовці іноземних студентів

ЗАТВЕРДЖЕНО

на засіданні кафедри внутрішньої медицини №3

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Зав. кафедри _____ д.мед.н., професор Л.В. Журавльова

**МЕТОДИЧНІ ВКАЗІВКИ
для студентів**

з дисципліни «Внутрішня медицина (в тому числі з ендокринологією)
студенти 4 курсу I, II, III медичних факультетів, V та VI факультетів по підготовці
іноземних студентів

Запальні захворювання кишки. Синдром подразненої кишки

Харків 2016

**KHARKOV NATIONAL MEDICAL UNIVERSITY
DEPARTMENT OF INTERNAL MEDICINE N3**

METHODOLOGICAL RECOMMENDATIONS FOR STUDENTS

**"Chronic diseases of large intestine: ulcerative colitis, Crohn's disease, irritable
bowel syndrome"**

Kharkiv 2016

Practical class "Chronic diseases of large intestine", 4 hours

Topicality:

The inflammatory bowel disease (IBD) – a term that defines a group of chronic diseases, which are characterized by the destructive nonspecific immune inflammation of the wall of the gut. Their etiology is unknown. Inflammatory diseases of intestines include ulcerative colitis (UC) and the Crohn's disease (CD).

The prevalence of UC varies from 28 to 117, CD - from 34 to 146 patients per 100000 of population and depends on ethnicity and a geographical zone. In southern countries the morbidity is low. European population shows higher prevalence of IBD than African one. The prevalence of UC in the big cities is 1,5-4 times higher than in rural areas. The age peak of UC morbidity is 20-40 years, as regards CD – the peak is between 20-29 years. The isolated lesion of large intestine in case of UC is more frequently observed in patients older than 70 years, the combined lesion of the large and small intestine or the isolated lesion of the small intestine are more frequently found in the young persons. Persons of both sexes get sick with the identical frequency, but there is a tendency to the relatively more frequent CD morbidity in women, and more frequent UC in men. For UC and CD the genetic predisposition is typical - about 10% of patients with UC and 20% of patients with CD have relatives of the first degree, which are sick with the same illnesses.

Irritable Bowel Syndrome (IBS) - one of the most common diseases in gastroenterological practice. IBS possesses 2nd place after colds among the causes for inability to work in the U.S. IBS significantly affects the quality of life of patients and forces them to go to the doctor at an average of 3 times a year, 19% of them - more than 5 times a year.

The prevalence of IBS varies from 14-22 to 30-48%, the average is 20%. The variability in evaluation of IBS incidence is caused by different number of symptoms that are taken into consideration at making diagnosis. Another important fact is that about 2/3 of the patients don't go to the doctor and treat themselves independently. IBS occurs in women 2-4 times more often than in men. Typically disease occurs at a young age - 30-40 years. Among men older than 50 years IBS occurs as often as in women. When symptoms of IBS occur in patients older than 60 years, the diagnosis is doubtful and other diseases of intestines should be considered .

The educational purposes:

- To teach students to recognize the basic symptoms and syndromes of IBS and IBD;
- To provide students with physical methods of examination of IBS and IBD;
- To provide students with methods of research which are applied to diagnosis of IBD and IBS taking into consideration indications and contraindications for their performance; techniques of their application; the diagnostic value of each of them;
- To teach students to interpret results of the main research methods independently;
- To teach students to recognize and diagnose complications of IBS and IBD;
- To teach students to prescribe treatment at of IBS and IBD

What should the student know?

- Incidence of IBS and IBD;
- Etiological factors of IBS and IBD;
- Pathogenesis of IBS and IBD;
- The basic clinical syndromes of IBS and IBD;
- The general and alarm symptoms of IBS and IBD;
- Clinical signs of IBS and IBD;
- Diagnostics of IBS and IBD;

- Morphological studies of intestines in case of IBS and IBD;
- Instrumental methods of diagnostics of IBS and IBD;
- Differential diagnostics of IBS and IBD;
- Classification of IBS and IBD;
- Complications of IBS and IBD;
- Treatment of IBS and IBD (change of life style, a balanced diet, drug therapy).

What should the student be able to do?

- to define main clinical syndromes of IBS and IBD;
- to interpret the results of biochemical and immunoenzyme assays;
- to interpret the data of bowel biopsy;
- to interpret the data of instrumental methods of diagnostics;
- to assess the correspondence of specific patient to the criteria of successful therapy;
- to assess the differential diagnosis;
- to prescribe regimen for patients with IBS and IBD.

The list of practical skills which the student should possess:

- Examination of abdomen;
- Superficial palpation of abdomen;
- Deep methodical sliding palpation of abdomen,
- Examination of a skin and mucous membranes;
- Physical examination of liver.

Topics content:

INFLAMMATORY BOWEL DESEASE

Inflammatory bowel disease (IBD) is an idiopathic disease caused by a dysregulated immune response to host intestinal microflora. The two major types of inflammatory bowel disease are ulcerative colitis (UC), which is limited to the colon, and Crohn disease (CD), which can affect any segment of the gastrointestinal tract from the mouth to the anus, involves "skip lesions," and is transmural. There is a genetic predisposition for IBD, and patients with this condition are more prone to the development of malignancy.

UC and CD share many extraintestinal manifestations, although some of these tend to occur more commonly with either condition. Although both UC and CD have distinct pathologic findings, approximately 10-15% of patients cannot be classified definitively into either type; in such patients, the disease is labeled as indeterminate colitis.

Epidemiology

The highest rates of IBD are assumed to be in developed countries, and the lowest are considered to be in developing regions; colder-climate regions and urban areas have a greater rate of IBD than those of warmer climates and rural areas. Internationally, the incidence of IBD is approximately 0.5-24.5 cases per 100,000 person-years for ulcerative colitis and 0.1-16 cases per 100,000 person-years for Crohn disease. Overall, the prevalence for IBD is 396 cases per 100,000 persons annually.

Peak incidence of UC and CD is in the second to fourth decades, with 78% of CD studies and 51% of UC studies reporting the highest incidence among those age 20–29 years old. A second modest rise in incidence occurs between the seventh and ninth decades of life. The female-to-male ratio ranges from 0.51 to 1.58 for UC studies and 0.34 to 1.65 for CD studies, suggesting that the diagnosis of IBD is not gender specific. The greatest incidence of IBD is among white and Jewish people, but the incidence of

IBD in Hispanic and Asian people is increasing, as noted above. Urban areas have a higher prevalence of IBD than rural areas, and high socioeconomic classes have a higher prevalence than lower socioeconomic classes.

Etiology

Three characteristics define the etiology of inflammatory bowel disease (IBD): (1) genetic predisposition; (2) an altered, dysregulated immune response; and (3) an altered response to gut microorganisms. However, the triggering event for the activation of the immune response in IBD has yet to be identified. Possible factors related to this event include a pathogenic organism (as yet unidentified) or an inappropriate response (ie, failure to downgrade the inflammatory response to an antigen, such as an alteration in barrier function). During the course of infections or other environmental stimuli in the normal host, full activation of the gut-associated lymphoid tissues occurs but is rapidly superseded by dampening of the immune response and tissue repair. In IBD such processes may not be regulated normally.

Genetics

The genetic underpinning of IBD is known from its occurrence in the context of several genetic syndromes and the development of severe, refractory IBD in early life in the setting of single gene defects that affect the immune system (Turner's syndrome, Hermansky-Pudlak, Wiskott-Aldrich syndrome, glycogen storage disease, immune dysregulation polyendocrinopathy, enteropathy X-linked). In addition, IBD has a familial origin in at least 10% of afflicted individuals. First-degree relatives have a 5- to 20-fold increased risk of developing IBD, as compared with persons from unaffected families. The child of a parent with IBD has a 5% risk of developing IBD. Twin studies show a concordance of approximately 70% in identical twins, versus 5-10% in nonidentical twins.

Commensal microbiota and IBD

The endogenous commensal microbiota within the intestines plays a central role in the pathogenesis of IBD. Humans are born sterile and acquire their commensal microbiota initially from the mother during egress through the birth canal and subsequently from environmental sources. The commensal microbiota in patients with both UC and CD is demonstrably different from nonafflicted individuals, a state of dysbiosis, suggesting the presence of microorganisms that drive disease (e.g., Proteobacteria such as enteroinvasive and adherent *Escherichia coli*) and to which the immune response is directed and/or the loss of microorganisms that hinder inflammation (e.g., Firmicutes such as *Faecalibacterium prausnitzii*). Many of the changes in the commensal microbiota occur as a consequence of the inflammation. In addition, agents that alter the intestinal microbiota such as metronidazole, ciprofloxacin, and elemental diets, may improve CD.

Defective immune regulation in IBD

The mucosal immune system is normally unreactive to luminal contents due to oral (mucosal) tolerance. Oral tolerance may be responsible for the lack of immune responsiveness to dietary antigens and the commensal microbiota in the intestinal lumen. In IBD this suppression of inflammation is altered, leading to uncontrolled inflammation. The mechanisms of this regulated immune suppression are incompletely known. In both UC and CD, an inflammatory pathway thus likely emerges from the genetic predisposition that is associated with inappropriate innate immune and epithelial sensing and reactivity to commensal bacteria that secrete inflammatory mediators together with inadequate regulatory pathways that lead to activated CD4+ and CD8+ T cells within the epithelium and lamina propria that altogether secrete excessive quantities of inflammatory cytokines relative to anti-inflammatory cytokines.

Smoking

The risk of developing UC is higher in nonsmokers and former smokers than in current smokers. The onset of UC occasionally appears to coincide with smoking cessation; however, this does not imply that smoking would improve the symptoms of UC. There has been limited success with the use of nicotine patches. CD patients have a higher incidence of smoking than the general population, and smoking appears to lessen the response to medical therapy.

Pathophysiology

The common end pathway of ulcerative colitis is inflammation of the mucosa of the intestinal tract, causing ulceration, edema, bleeding, and fluid and electrolyte loss. In several studies, genetic factors appeared to influence the risk of IBD by causing a disruption of epithelial barrier integrity, deficits in autophagy, deficiencies in innate pattern recognition receptors, and problems with lymphocyte differentiation, especially in CD.

Inflammatory mediators have been identified in IBD, and considerable evidence suggests that these mediators play an important role in the pathologic and clinical characteristics of these disorders. Cytokines, which are released by macrophages in response to various antigenic stimuli, bind to different receptors and produce autocrine, paracrine, and endocrine effects. Cytokines differentiate lymphocytes into different types of T cells. Helper T cells, type 1 (Th-1), are associated principally with CD, whereas Th-2 cells are associated principally with ulcerative colitis. The immune response disrupts the intestinal mucosa and leads to a chronic inflammatory process.

Ulcerative colitis

In UC, the inflammation begins in the rectum and extends proximally in an uninterrupted fashion to the proximal colon and could eventually involve the entire length of the large intestine. The rectum is always involved in UC; and unlike in CD, there are no "skip areas" (ie, normal areas of the bowel interspersed with diseased areas), unless pretreated with topical rectal therapy (ie, a steroid or 5-aminosalicylic acid [5-ASA] enema).

The disease remains confined to the rectum in approximately 25% of cases, and in the remainder of cases, UC spreads proximally and contiguously. Pancolitis occurs in 10% of patients. The distal terminal ileum may become inflamed in a superficial manner, referred to as backwash ileitis. Even with less than total colonic involvement, the disease is strikingly and uniformly continuous. As UC becomes chronic, the colon becomes a rigid foreshortened tube that lacks its usual haustral markings, leading to the lead-pipe appearance observed on barium enema.

Crohn disease

CD can affect any portion of the gastrointestinal tract, from the mouth to the anus, and causes 3 patterns of involvement: inflammatory disease, strictures, and fistulas. This disease consists of segmental involvement by a nonspecific granulomatous inflammatory process. The most important pathologic feature of CD is that it is transmural, involving all layers of the bowel, not just the mucosa and the submucosa, which is characteristic of UC. Furthermore, CD is discontinuous, with skip areas interspersed between 2 or more involved areas.

Late in the disease, the mucosa develops a cobblestone appearance, which results from deep, longitudinal ulcerations interlaced with intervening normal mucosa (see the images below). In 35% of cases, CD occurs in the ileum and colon; in 32%, solely in the colon; in 28%, in the small bowel; and in 5%, in the gastroduodenal region. Diarrhea, cramping, and abdominal pain are common symptoms of CD in all of the above locations, except for the gastroduodenal region, in which anorexia, nausea, and vomiting are more common.

Rectal sparing is a typical but not constant feature of CD. However, anorectal complications (eg, fistulas, abscesses) are common. Much less commonly, CD involves the more proximal parts of the GI tract, including the mouth, tongue, esophagus, stomach, and duodenum.

The incidence of gallstones and kidney stones is increased in CD because of malabsorption of fat and bile salts. Gallstones are formed because of increased cholesterol concentration in the bile, which is caused by a reduced bile salt pool.

Patients who have CD with ileal disease or resection are also likely to form calcium oxalate kidney stones. With the fat malabsorption, unabsorbed long-chain fatty acids bind calcium in the lumen. Oxalate in the lumen is normally bound to calcium. Calcium oxalate is poorly soluble and poorly absorbed; however, if calcium is bound to malabsorbed fatty acids, oxalate combines with sodium to form sodium oxalate, which is soluble and is absorbed in the colon (enteric hyperoxaluria). The development of calcium oxalate stones in CD requires an intact colon to absorb oxalate. Patients with ileostomies generally do not develop calcium oxalate stones, but they may develop uric acid or mixed stones.

Clinical presentation

Ulcerative colitis

Patients with UC predominantly complain of the following:

- Rectal bleeding
- Frequent stools
- Mucous discharge from the rectum
- Tenesmus (occasionally)
- Lower abdominal pain and severe dehydration from purulent rectal discharge (in severe cases, especially in the elderly)

In some cases, UC has a fulminant course marked by the following:

- Severe diarrhea and cramps
- Fever
- Leukocytosis
- Abdominal distention

UC is associated with various extracolonic manifestations, as follows:

- Uveitis
- Pyoderma gangrenosum
- Pleuritis
- Erythema nodosum
- Ankylosing spondylitis
- Spondyloarthropathies

Other conditions associated with UC include the following:

- primary sclerosing cholangitis
- Recurrent subcutaneous abscesses unrelated to pyoderma gangrenosum
- Multiple sclerosis
- Immunobullous disease of the skin

Physical findings are typically normal in mild disease, except for mild tenderness in the lower left abdominal quadrant. In severe disease, the following may be observed:

- Fever
- Tachycardia
- Significant abdominal tenderness

- Weight loss

The **severity** of UC can be graded as follows:

- Mild: Bleeding per rectum, fewer than 4 bowel motions per day
- Moderate: Bleeding per rectum, more than 4 bowel motions per day
- Severe: Bleeding per rectum, more than 4 bowel motions per day, and a systemic illness with hypoalbuminemia (< 30 g/L)

Crohn disease

The characteristic presentation in CD is abdominal pain and diarrhea, which may be complicated by intestinal fistulization or obstruction. Unpredictable flares and remissions characterize the long-term course.

Other signs and symptoms of CD may include the following:

- Rectal bleeding
- Fever
- Weight loss, anorexia
- Nausea, vomiting
- Malnutrition, vitamin deficiencies
- Generalized fatigability
- Bone loss
- Psychosocial issues (eg, depression, anxiety, and coping difficulty); pediatric patients may also experience psychological issues regarding quality of life and body image
- Growth failure in pediatric patients: may precede gastrointestinal symptoms by years

Examination for Crohn disease includes the following:

- Vital signs: normal, but possible presence of tachycardia in anemic or dehydrated patients; possible chronic intermittent fever
- Gastrointestinal: may vary from normal to those of an acute abdomen; assess for rectal sphincter tone, gross rectal mucosal abnormalities, presence of hematochezia
- Genitourinary: may include presence of skin tags, fistulae, ulcers, abscesses, and scarring in the perianal region; nephrolithiasis, hydronephrosis, and enterovesical fistulae
- Musculoskeletal: possible arthritis and arthralgia, particularly of the large joints
- Dermatologic: may show pallor or jaundice, mucocutaneous or aphthous ulcers, erythema nodosum, and pyoderma gangrenosum
- Ophthalmologic: may reveal episcleritis; possible uveitis
- Growth delay: decreased growth velocity (eg, height), pubertal delay
- Hematologic: hypercoagulable state

Low-grade fever, prolonged diarrhea with abdominal pain, weight loss, and generalized fatigability are usually reported. Crampy or steady right lower quadrant or periumbilical pain may develop; the pain precedes and may be partially relieved by defecation. Diarrhea is usually not grossly bloody and is often intermittent. If the colon is involved, patients may report diffuse abdominal pain accompanied by mucus, blood, and pus in the stool. It is important to note that colonic CD may be clinically indistinguishable from UC, with symptoms of bloody mucopurulent diarrhea, cramping abdominal pain, and urgency to defecate.

CD of the small intestine usually presents with evidence of malabsorption, including diarrhea, abdominal pain, weight loss, and anorexia. Initially, these symptoms may be quite subtle. Patients with gastroduodenal involvement more commonly have anorexia, nausea, and vomiting. Those with perianal disease may have debilitating perirectal pain, malodorous discharge from the fistula, and disfiguring scars from active disease or previous surgery. Patients may also present with complaints suggestive of

intestinal obstruction.

The **physical examination** should focus on temperature, weight, nutritional status, the presence of abdominal tenderness or a mass, perianal and rectal examination findings, and extraintestinal manifestations. Vital signs are usually normal in patients with CD, though tachycardia may be present in anemic or dehydrated patients. Chronic intermittent fever is a common presenting sign.

Abdominal findings may vary from normal to those of an acute abdomen. Diffuse abdominal tenderness or localized pain may be present.

In addition to local complications, various extraintestinal manifestations may be associated with CD, usually involving the skin, joints, mouth, eyes, liver, or bile ducts. The most common extraintestinal manifestations are arthritis and arthralgia. The large joints (eg, hips, knees, ankles) are typically involved.

Disease Classification and Activity Scoring Systems

A system to homogenize the classification of IBD on the basis of disease location and behavior was first established in 1998 in Vienna, Austria. Further developments in the field were reviewed, and the Montreal revision of the Vienna classification was proposed (see below), with special attention to predominant parameters of age at diagnosis, location, and behavior of disease.

Montreal classification system

The Montreal revision of the Vienna system is based on the following 3 variables:

- Age at diagnosis
- Disease distribution/location
- Disease behavior

Age at diagnosis (A) has 3 categories, as follows:

- A1 – ≤ 16 years
- A2 – 17-40 years
- A3 – > 40 years

Disease distribution/location (L) has the following 4 categories, 1 of which is a modifier for upper GI involvement:

- L1 – Ileal
- L2 – Colonic
- L3 – Ileocolonic
- L4 – Isolated upper GI disease; L4 is a modifier that can be added to L1-L3 when there is concomitant upper GI involvement

Disease behavior (B) has 1 interim category (B1) and 2 specified categories, with an additional modifier for perianal diseases (p), as follows:

- B1 – Nonstricturing, nonpenetrating; B1p: nonstricturing, nonpenetrating with perianal involvement
- B2 – Stricturing; B2p: stricturing with perianal involvement
- B3 – Penetrating; B3p: penetrating with perianal involvement

Diagnosis

Ulcerative colitis

Endoscopy and biopsy

The diagnosis of UC is best made with endoscopy. Endoscopically, ulcerative colitis is characterized by abnormal erythematous mucosa, with or without ulcerations, extending from the rectum to part or the entire colon. The inflammation is uniform, without intervening areas of normal mucosa, while skip lesions tend to characterize CD. Contact bleeding may also be observed, with mucus identified in the lumen of the bowel. Biopsy of the mucosa is recommended to identify the extent of the disease with

respect to the thickness of the bowel wall. Pathologic features that are typically seen include intense infiltration of the mucosa and submucosa with neutrophils and crypt abscesses, lamina propria with lymphoid aggregates, plasma cells, mast cells and eosinophils, and shortening and branching of the crypts.

The **extent of disease** is defined by the following:

- Extensive disease - Evidence of ulcerative colitis proximal to the splenic flexure
- Left-sided disease - Ulcerative colitis present in the descending colon up to, but not proximal to, the splenic flexure
- Proctosigmoiditis - Disease limited to the rectum with or without sigmoid involvement

Laboratory Studies

Serologic markers

Antineutrophil cytoplasmic antibodies (ANCA) assay results are positive in 60-80% of patients with ulcerative colitis. The finding of ANCA is roughly 50% sensitive, is 94% specific, and has a 76% positive predictive value for ulcerative colitis.

Complete blood count (CBC)

Findings on CBC count may include the following:

- Anemia (ie, hemoglobin < 14 g/dL in males and < 12 g/dL in females)
- Thrombocytosis (ie, platelet count >350,000/ μ L)

Comprehensive metabolic panel

Findings on the comprehensive metabolic panel may include the following:

- Hypoalbuminemia (ie, albumin < 3.5 g/dL)
- Hypokalemia (ie, potassium < 3.5 mEq/L)
- Hypomagnesemia (ie, magnesium < 1.5 mg/dL)
- Elevated alkaline phosphatase: More than 125 U/L suggests primary sclerosing cholangitis (usually >3 times the upper limit of the reference range).

Inflammatory markers

Elevation of the erythrocyte sedimentation rate (variable reference ranges, usually 0-33 mm/h) and C-reactive protein level (ie, >100 mg/L) correlates with disease activity.

Stool assays

Stool studies are used to exclude other causes. These include evaluation of fecal leukocytes, ova and parasite studies, culture for bacterial pathogens, and *Clostridium difficile* titer.

Radiologic Assessment of Ulcerative Colitis

The earliest radiologic change of UC seen on single-contrast barium enema is a fine mucosal granularity. With increasing severity, the mucosa becomes thickened, and superficial ulcers are seen. Deep ulcerations can appear as “collar-button” ulcers, which indicate that the ulceration has penetrated the mucosa. Haustral folds may be normal in mild disease, but as activity progresses they become edematous and thickened. Loss of haustration can occur, especially in patients with long-standing disease. In addition, the colon becomes shortened and narrowed. Polyps in the colon may be postinflammatory polyps or pseudopolyps, adenomatous polyps, or carcinoma.

Computed tomography (CT) scanning or magnetic resonance imaging (MRI) is not as helpful as endoscopy in making the diagnosis of UC, but typical findings include mild mural thickening (<1.5 cm), inhomogeneous wall density, absence of small bowel thickening, increased perirectal and presacral fat, target appearance of the rectum, and adenopathy.

Crohn disease

CD is initially diagnosed on the basis of a combination of clinical, laboratory, histologic, and radiologic findings. Laboratory study results are generally nonspecific but may be helpful in supporting the

diagnosis and managing the disease. Serologic studies are sometimes used to facilitate differentiation of CD from ulcerative colitis or IBD of undetermined type.

Endoscopy and biopsy

Endoscopic features of CD include rectal sparing, aphthous ulcerations, fistulas, and skip lesions. The entire intestinal wall is involved with inflammation. Biopsy specimens may demonstrate granulomas (approximately 50% of the time). Colonoscopy allows examination and biopsy of mass lesions or strictures and biopsy of the terminal ileum. Upper endoscopy is useful in diagnosing gastroduodenal involvement in patients with upper tract symptoms. Ileal or colonic strictures may be dilated with balloons introduced through the colonoscope. Strictures ≤ 4 cm and those at anastomotic sites respond better to endoscopic dilation. The perforation rate is as high as 10%. Most endoscopists dilate only fibrotic strictures and not those associated with active inflammation. Wireless capsule endoscopy allows direct visualization of the entire small-bowel mucosa.

Routine **laboratory studies** include the following:

- CBC count (anemia, leukocytosis)
- Chemistry panel (electrolyte analysis, hypoalbuminemia, deficiencies in iron and micronutrients)
- Liver function tests - results may be elevated, either transiently (because of inflammation) or chronically (because of sclerosing cholangitis)
- Inflammatory markers (C-reactive protein level or erythrocyte sedimentation rate)
- Stool studies (white blood cells, occult blood, routine pathogens, ova, parasites, and *Clostridium difficile* toxin, fecal calprotectin)

Serologic tests. There are 2 serologic tests that are currently used in efforts to differentiate UC from CD. Antibodies to the yeast *Saccharomyces cerevisiae* (ie, anti-*S cerevisiae* antibodies [ASCA]) are found more commonly in CD than in UC, whereas perinuclear antineutrophil cytoplasmic antibody (p-ANCA), a myeloperoxidase antigen, is found more commonly in UC than in CD.

Plain abdominal radiography. Findings may include mural thickening and dilatation, small bowel and colonic mucosal abnormalities, and abnormal fecal distribution with areas of colonic involvement without fecal material, presence of bowel obstruction, perforation (free air), or toxic colon distention.

Barium Contrast Studies. Mucosal fissures, bowel fistulae, strictures, and obstructions can be visualized.

Computed Tomography. CT is helpful in—and considered the imaging technique of choice for—the assessment of extramural complications as well as hepatobiliary and renal complications in adults and children. It may show bowel wall thickening, bowel obstruction, mesenteric edema, abscesses, or fistulae.

Magnetic Resonance Imaging. MRI has been shown to yield a higher sensitivity and specificity than ileocolonoscopy (the criterion standard) both for diagnosing CD and for determining its severity. It is especially useful for evaluating pelvic and perianal disease when one is investigating for evidence of perianal fistulae and abscesses.

Distinguishing Ulcerative Colitis from Crohn Disease

Ulcerative Colitis	Crohn Disease
Only colon involved	Panintestinal
Continuous inflammation extending proximally from rectum	Skip-lesions with intervening normal mucosa
Inflammation in mucosa and submucosa only	Transmural inflammation
No granulomas	Noncaseating granulomas
Perinuclear ANCA (pANCA) positive	ASCA positive
Bleeding (common)	Bleeding (uncommon)
Fistulae (rare)	Fistulae (common)

Different clinical, endoscopic and radiographic features

	Ulcerative Colitis	Crohn's Disease
Clinical		
Gross blood in stool	Yes	Occasionally
Mucus	Yes	Occasionally
Systemic symptoms	Occasionally	Frequently
Pain	Occasionally	Frequently
Abdominal mass	Rarely	Yes
Significant perineal disease	No	Frequently
Fistulas	No	Yes
Small intestinal obstruction	No	Frequently
Colonic obstruction	Rarely	Frequently
Response to antibiotics	No	Yes
Recurrence after surgery	No	Yes
Endoscopic		
Rectal sparing	Rarely	Frequently
Continuous disease	Yes	Occasionally
“Cobblestoning”	No	Yes
Granuloma on biopsy	No	Occasionally
Radiographic		
Small bowel significantly abnormal	No	Yes
Abnormal terminal ileum	No	Yes
Segmental colitis	No	Yes
Asymmetric colitis	No	Yes
Stricture	Occasionally	Frequently

Differential diagnosis

- Celiac disease
- Microscopic colitis
- Irritable bowel syndrome
- Lactose intolerance
- Functional diarrhea
- Gastrointestinal infections
- AIDS
- Colorectal malignancy
- Ischemic colitis
- Radiation-induced colitis
- Nonsteroidal anti-inflammatory drug (NSAID) enteropathy
- Intestinal tuberculosis

Complications of IBD disease

Intestinal complications

IBD can be associated with several gastrointestinal complications, including risk of hemorrhage, perforation, strictures, and fistulas—as well as perianal disease and related complications, such as perianal or pelvic abscesses, toxic megacolon (complicating acute severe colitis), and malignancy (colorectal cancer, cholangiocarcinoma complicating primary sclerosing cholangitis).

Extraintestinal complications

Extraintestinal complications occur in approximately 20-25% of patients with IBD. In some cases, they may be more symptomatic than the bowel disease itself. These include osteoporosis (usually a consequence of prolonged corticosteroid use), hypercoagulability resulting in venous thromboembolism, anemia, gallstones, primary sclerosing cholangitis, aphthous ulcers, arthritis, iritis (uveitis) and episcleritis, and skin complications (pyoderma gangrenosum, erythema nodosum).

Treatment

The 2 goals of therapy are the achievement of remission (induction) and the prevention of disease flares (maintenance). The care of a patient with IBD can be either medical or surgical in nature or, in many patients, a combination of both. The management algorithm is also dependent on whether the diagnosis is CD or UC. The medical approach for patients with IBD is both symptomatic care (ie, relief of symptoms) and mucosal healing following a stepwise approach to medication, with escalation of the medical regimen until a response is achieved.

A **stepwise approach** (now generally referred to as the **step-up approach**) may be taken in mild to moderate IBD. The first step in medication therapy for IBD is usually aminosalicylates. These agents appear to have greater efficacy for the treatment of UC than for CD, for which efficacy data are limited. For CD, metronidazole or ciprofloxacin is occasionally used, particularly for perianal disease or an inflammatory mass. If the patient's condition fails to respond to an adequate dose of aminosalicylates, the second step is often corticosteroids, which tend to provide rapid relief of symptoms and a significant decrease in inflammation. The most common range for moderate flares of IBD is oral prednisone at 10-40 mg/day; for more severe flares, the higher end of the range is used (occasionally doses up to 60 mg/day). Once a clinical response is seen, the dose is tapered. Most patients who use oral corticosteroids can tolerate a relatively rapid taper after a response is achieved; occasionally, a very prolonged steroid taper is necessary to prevent relapse in patients who have had prolonged exposure to steroids in the past. The immune-modifying agents are step III drugs and are used if corticosteroids fail or are required for

prolonged periods. Anti-TNF monoclonal antibody therapies are also step III drugs that are effective in both CD and UC; some studies have demonstrated that they have a greater efficacy than azathioprine. Traditionally, anti-TNF agents have been administered when CD has been unresponsive to steroids and immunosuppressants; however, the early introduction of these agents in conjunction with immunosuppressants in those with an increased risk of a complicated, severe, or possibly aggressive IBD has the potential to modify the disease course. In general, one major goal is to wean the patient off steroids as soon as possible to prevent long-term adverse effects from these agents.

In addition to treatment of the underlying inflammation, patients with IBD may require symptomatic therapy, particularly when their symptoms are not related to active inflammation. Treatment with antidiarrheal agents such as loperamine or diphenoxylate/atropine should generally be avoided in patients with active inflammation, as these drugs can precipitate toxic megacolon in individuals with significant colonic inflammation.

Patients are candidates for immunomodulators (azathioprine, 6-mercaptopurine, methotrexate) or anti-TNF agents (infliximab, adalimumab, certolizumab pegol) and biologic agents if flares are frequent (>1-2 times), if the duration of steroid use is prolonged (more than a few weeks per year), if reduction of the steroid dose causes recurrence of symptoms (steroid dependent), or if steroids do not appear to be working (steroid refractory).

Osteoporosis is a very serious complication, involving 40% of patients with IBD, and increases the risk for fractures. All patients who have been using steroids for longer than 3 months, as well as postmenopausal women, should undergo testing with bone-density studies; treatment with bisphosphonates and calcium supplements can be initiated in patients with significantly low bone density.

Ulcerative colitis

Medical treatment of mild UC includes the following:

- Mild disease confined to the rectum: Topical mesalazine via suppository (preferred) or budesonide rectal foam
- Left-side colonic disease: Mesalazine suppository and oral aminosalicylate (oral mesalazine is preferred to oral sulfasalazine)
- Systemic steroids, when disease does not quickly respond to aminosalicylates
- Oral budesonide
- After remission, long-term maintenance therapy (eg, once-daily mesalazine)

Medical treatment of acute, severe UC may include the following:

- Hospitalization
- Intravenous high-dose corticosteroids
- Alternative induction medications: Cyclosporine, tacrolimus, infliximab, adalimumab, golimumab

Indications for urgent surgery include the following:

- Toxic megacolon refractory to medical management
- Fulminant attack refractory to medical management
- Uncontrolled colonic bleeding

Indications for elective surgery include the following:

- Long-term steroid dependence
- Dysplasia or adenocarcinoma found on screening biopsy
- Disease present 7-10 years

Surgical options include the following:

- Total colectomy (panproctocolectomy) and ileostomy
- Total colectomy
- Ileoanal pouch reconstruction or ileorectal anastomosis
- In an emergency, subtotal colectomy with end-ileostomy

Crohn disease

Pharmacotherapy

Medications used in the treatment of Crohn disease include the following:

- 5-Aminosalicylic acid derivative agents (eg, mesalamine rectal, mesalamine, sulfasalazine, balsalazide)
- Corticosteroids (eg, prednisone, methylprednisolone, budesonide, hydrocortisone, prednisolone)
- Immunosuppressive agents (eg, mercaptopurine, methotrexate, tacrolimus)
- Monoclonal antibodies (eg, infliximab, adalimumab, certolizumab pegol, natalizumab, vedolizumab)
- Antibiotics (eg, metronidazole, ciprofloxacin)
- Antidiarrheal agents (eg, loperamide, diphenoxylate-atropine)
- Bile acid sequestrants (eg, cholestyramine, colestipol)
- Anticholinergic agents (eg, dicyclomine, hyoscyamine, propantheline)

Surgery

Unlike ulcerative colitis, Crohn disease has no surgical cure. Most patients with Crohn disease require surgical intervention during their lifetime.

Surgical management of the terminal ileum, ileocolon, and/or upper gastrointestinal tract may include the following:

- Resection of the affected bowel
- Ileocolostomy or proximal loop ileostomy
- Drainage of any septic foci with later definitive resection
- Strictureplasty
- Bypass
- Endoscopic dilatation of symptomatic, accessible strictures

Surgical management of the colon may include the following:

- Subtotal or total colectomy with end ileostomy (laparoscopic or open approach)
- Segmental or total colectomy with or without primary anastomosis
- Total proctocolectomy or proctectomy with stoma creation

IRRITABLE BOWEL SYNDROME

Irritable bowel syndrome (IBS) is a functional GI disorder characterized by abdominal pain and altered bowel habits in the absence of a specific and unique organic pathology, although microscopic inflammation has been documented in some patients. Population-based studies estimate the prevalence of irritable bowel syndrome at 10-20% and the incidence of irritable bowel syndrome at 1-2% per year.

Etiology and pathogenesis

The cause of IBS is unknown. No anatomic cause can be found on laboratory tests, x-rays, and biopsies. Emotional factors, diet, drugs, or hormones may precipitate or aggravate GI symptoms. Historically, the disorder was often considered as purely psychosomatic. Although psychosocial factors are involved, IBS is better understood as a combination of psychosocial and physiologic factors.

Psychosocial factors. Psychological distress is common among patients with IBS, especially in those who seek medical care. Some patients have anxiety disorders, depression, or a somatization disorder. Sleep disturbances also coexist. However, stress and emotional conflict do not always coincide with symptom onset and recurrence. Some patients with IBS seem to have a learned aberrant illness behavior (ie, they express emotional conflict as a GI complaint, usually abdominal pain). The physician evaluating patients with IBS, particularly those with refractory symptoms, should investigate for unresolved

psychologic issues, including the possibility of sexual or physical abuse. Psychosocial factors also affect the outcome in IBS.

Physiologic factors. A variety of physiologic factors seem to be involved in IBS symptoms. These factors include altered motility, visceral hyperalgesia, and various genetic and environmental factors.

Visceral hyperalgesia refers to hypersensitivity to normal amounts of intraluminal distention and heightened perception of pain in the presence of normal quantities of intestinal gas; it may result from remodeling of neural pathways in the brain-gut axis. Some patients (perhaps 1 in 7) have reported their IBS symptoms began after an episode of acute gastroenteritis (termed postinfectious IBS). A subset of patients with IBS has autonomic dysfunctions. However, many patients have no demonstrable physiologic abnormalities, and even in those that do, the abnormalities may not correlate with symptoms. Constipation may be explained by slower colonic transit, and diarrhea may be explained by faster colonic transit. Some patients with constipation have fewer colonic high amplitude-propagated contractions, which propel colonic contents over several segments. Conversely, excess sigmoid motor activity may retard transit in functional constipation.

Postprandial abdominal discomfort may be attributed to an exaggerated gastro-colonic reflex (the colonic contractile response to a meal), the presence of colonic high amplitude-propagated contractions, increased intestinal sensitivity (visceral hyperalgesia), or a combination of these. Fat ingestion may exaggerate hypersensitivity.

Signs and symptoms

Manifestations of IBS are as follows:

- Altered bowel habits
- Abdominal pain
- Abdominal distention

Altered bowel habits in IBS may have the following characteristics:

- Constipation variably results in complaints of hard stools of narrow caliber, painful or infrequent defecation, and intractability to laxatives
- Diarrhea usually is described as small volumes of loose stool, with evacuation preceded by urgency or frequent defecation
- Postprandial urgency is common, as is alternation between constipation and diarrhea
- Characteristically, one feature predominates in a single patient, but significant variability exists among patients

Abdominal pain in IBS is protean, but may have the following characteristics:

- Pain frequently is diffuse without radiation
- Common sites of pain include the lower abdomen, specifically the left lower quadrant
- Acute episodes of sharp pain are often superimposed on a more constant dull ache
- Meals may precipitate pain
- Defecation commonly improves pain but may not fully relieve it
- Pain from presumed gas pockets in the splenic flexure may masquerade as anterior chest pain or left upper quadrant abdominal pain

Additional symptoms consistent with irritable bowel syndrome are as follows:

- Clear or white mucorrhea of a noninflammatory etiology
- Dyspepsia, heartburn
- Nausea, vomiting
- Sexual dysfunction (including dyspareunia and poor libido)
- Urinary frequency and urgency have been noted
- Worsening of symptoms in the perimenstrual period
- Comorbid fibromyalgia

- Stressor-related symptoms

Symptoms not consistent with irritable bowel syndrome should alert the clinician to the possibility of an organic pathology. Inconsistent symptoms include the following:

- Onset in middle age or older
- Acute symptoms (irritable bowel syndrome is defined by chronicity)
- Progressive symptoms
- Nocturnal symptoms
- Anorexia or weight loss
- Fever
- Rectal bleeding
- Painless diarrhea
- Steatorrhea
- Gluten intolerance

Diagnosis

The Rome III criteria for the diagnosis of irritable bowel syndrome require that patients have had recurrent abdominal pain or discomfort at least 3 days per month during the previous 3 months that is associated with 2 or more of the following:

- Relieved by defecation
- Onset associated with a change in stool frequency
- Onset associated with a change in stool form or appearance
- Supporting symptoms include the following:
 - Altered stool frequency
 - Altered stool form
 - Altered stool passage (straining and/or urgency)
 - Mucorrhea
 - Abdominal bloating or subjective distention

Four bowel patterns may be seen with irritable bowel syndrome. These patterns include the following:

- IBS-D (diarrhea predominant)
- IBS-C (constipation predominant)
- IBS-M (mixed diarrhea and constipation)
- IBS-A (alternating diarrhea and constipation)

Notably, within 1 year, 75% of patients change subtypes, and 29% switch between constipation-predominant IBS and diarrhea-predominant IBS.

A comprehensive history, a physical examination, and tailored laboratory and radiographic studies can establish a diagnosis of IBS in most patients. The American College of Gastroenterologists does not recommend laboratory testing or diagnostic imaging in patients younger than 50 years with typical IBS symptoms and without the following “alarm features”:

- Weight loss
- Iron deficiency anemia
- Family history of certain organic GI illnesses (eg, inflammatory bowel disease, celiac sprue, colorectal cancer)

Screening studies to rule out disorders other than IBS include the following:

- Complete blood count with differential to screen for anemia, inflammation, and infection
- A comprehensive metabolic panel to evaluate for metabolic disorders and to rule out dehydration/electrolyte abnormalities in patients with diarrhea
- Stool examinations for ova and parasites, enteric pathogens, leukocytes, *Clostridium difficile* toxin, and possibly *Giardia* antigen

History-specific studies include the following:

- Hydrogen breath testing to exclude bacterial overgrowth in patients with diarrhea to screen for lactose and/or fructose intolerance
- Tissue transglutaminase antibody testing and small bowel biopsy in IBS-D to diagnose celiac disease.
- Thyroid function tests
- Serum calcium testing to screen for hyperparathyroidism
- Erythrocyte sedimentation rate and C-reactive protein measurement are nonspecific screening tests for inflammation

Differential diagnosis:

1. Mild form of UC
2. Crohn's Disease
3. Celiac disease
4. Colorectal cancer
5. Intestinal polyposis
6. Parasitic infections and intestinal infections
7. Gynecological disorders (endometriosis)
8. Endocrine diseases (diabetic enteropathy, hyperthyroidism).

Management

Management of IBS consists primarily of providing psychological support and recommending dietary measures. Pharmacologic treatment is adjunctive and should be directed at symptoms.

Dietary measures may include the following:

- Fiber supplementation may improve symptoms of constipation and diarrhea
- Polycarbophil compounds may produce less flatulence than psyllium compounds
- Judicious water intake is recommended in patients who predominantly experience constipation
- Caffeine avoidance may limit anxiety and symptom exacerbation
- Legume avoidance may decrease abdominal bloating
- Lactose, fructose, and/or FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) should be limited or avoided in patients with these contributing disorders
- Probiotics are being studied for their use in decreasing IBS symptoms

Psychological interventions, cognitive-behavioral therapy, dynamic psychotherapy, and hypnotherapy are more effective than placebo.

Pharmacologic agents used for management of symptoms in IBS include the following:

- Anticholinergics (eg, dicyclomine, hyoscyamine)
- Antidiarrheals (eg, diphenoxylate, loperamide)
- Tricyclic antidepressants (eg, imipramine, amitriptyline)
- Prokinetics
- Bulk-forming laxatives
- Serotonin receptor antagonists (eg, alosetron)
- Chloride channel activators (eg, lubiprostone)
- Guanylate cyclase C agonists (eg, linaclotide)
- Antispasmodics (eg, peppermint oil, pinaverium, trimebutine, cimetropium/dicyclomine)

The control of an initial level of knowledge:

1. What pathogenetic mechanisms cause disorders of defecation?

- A. Intestinal hypersecretion, acceleration of a chymus passage, decrease of absorption of bile acids
 - B. Flatulence, enzymopathy
 - C. Spastic contraction of intestines
 - D. Decrease of contents of electrolytes and vitamins
 - E. Hypovitaminosis
2. What gut microflora is considered to be obligate?
- A. Bifidobacteria, lactobacilli, Escherichia coli
 - B. Proteus, micrococci, enterococci
 - S. Escherichia coli, Proteus, Staphylococcus
 - D. Anaerobes, lactobacilli
 - E. Micrococci, enterococci, bacteroides
3. Which of the listed factors are **not** caused by microflora of intestines?
- A. Creation of sour ph-environment in intestines
 - B. Synthesis of vitamins and biologically active substances
 - C. Increase of intestinal peristalsis
 - D. Synthesis of amylase, lipase, tripsin.
 - E. Increase immunoreactivity of an organism
4. What method can assist in diagnostics of disorders of bowel motility?
- A. Irrigoscopy
 - B. Colonoscopy
 - C. Passage of barium through intestines under control after 24, 48 and 72 hours
 - D. Endoscopy
 - E. None of listed above.
5. The main reason for pain syndrome in IBS is:
- A. Dynamic intestinal obstruction
 - B. Increase of mucus production
 - C. Trombosis of arteries and veins of mesentery
 - D. Lesion of intramural nerve plexus
 - E. Increased perception of pain impulses
6. What departments of GIT can get involved in pathologic process in case of CD?
- A. Ileum
 - B. Entire digestive tract
 - C. Large intestine
 - D. Small intestine
7. Ulcerative colitis is:
- A. Inflammation of entire GIT
 - B. Necrotizing inflammation of mucosa of small intestine
 - C. Necrotizing inflammation of mucosa of large intestine
 - D. Disease which begins in oral cavity
 - E. Disease related to stomach ulcer
8. Extraintestinal signs of ulcerative colitis include:
- A. Weight loss
 - B. Fever
 - C. Arthralgia

- D. Erythema nodosum
- E. Everything listed above

9. The basic method of diagnostics of CD is:

- A. Radiology of GIT
- B. Irrigoscopy
- C. Rektosigmoidoscopy
- D. Endoscopy with biopsy
- E. Ultrasonography

10. Which one of the listed below symptoms doesn't belong to IBS?

- A. Non-painful diarrhea
- B. Progression of symptoms
- C. Steatorrhea
- D. Morbid depression
- E. Gluten intolerance

Correct answers:

- 1. A
- 2. A
- 3. D
- 4. C
- 5. E
- 6. B
- 7. C
- 8. E
- 9. D
- 10. B

The control of a final level of knowledge:

1. What are the earliest permanent signs of CD:

- A. Diarrhea
- B. Abdominal pain
- C. Fever
- D. Bloody stool
- E. Pus in stool

2. The mechanism, which has no value in the development of IBS:

- A. Motoric dysfunction
- B. Visceral hyperalgesia
- C. Presence of psychopathy
- D. Cholestasis
- E. Intestinal dysbiosis

3. Which one of the listed below symptoms contradict the diagnosis of IBS:

- A. Abdominal pain which relieves after defecation
 - B. Lactose intolerance
 - C. Discharge of mucus with feces
 - D. Abdominal swelling
 - E. Disbacteriosis
4. What is not typical for IBS:
- A. Vasospastic reaction
 - B. Feeling of lump in the throat
 - C. Urinary disorders
 - D. Absence of appetite and loss of weight
 - E. Sexual dysfunction
5. The treatment of UC includes:
- A. Antibiotics
 - B. Preparations of 5-ASA
 - C. Antacids
 - D. Preparations of bismuth
 - E. Metronidazol
6. The treatment of CD includes :
- A. Hepatoprotectors,
 - B. Spasmolytics
 - C. Glucocorticoids, cytostatics
 - D. Antiviral medications, blockers of H₂- histamine receptors
 - E. Antacids and vitamins
7. What belongs to Intestinal complications of UC?
- A. Toxic megacolon
 - B. Perforation
 - C. Bleeding
 - D. Carcinoma
 - E. Everything listed above
8. What helps to perform differential diagnosis of UC and dysentery:
- A. Irrigoscopy
 - B. Colonoscopy
 - C. Bacteriological test of feces
 - D. Rectosigmoidoscopy
 - E. Clinical analysis of feces
9. What is the indication for surgical treatment of UC?
- A. Erythema nodosum
 - B. Toxic dilation of large intestine
 - C. Combination with stomach ulcer
 - D. Presence of sclerosing cholangitis
 - E. Everything listed above

The control of a final level of knowledge, correct answers:

1. **A**
2. **D**
3. **B**
4. **D**
5. **B**
6. **C**
7. **E**
8. **C**
9. **B**

Case-based questions.

1. The man of 38 years old complains on cramp-like pains in left iliac area and frequent liquid excrements 6-10 times a day with blood and pus, general weakness, loss of weight. He has been sick for more than 5 years. During the palpation the abdomen is painful in left iliac area. Liver +2 cm.; CBC - Hb - 80 g/L, er.- $3,5 \times 10^{12}/L$, ESR - 34 mm/h. What disease is the most probable cause for development of anemic syndrome in this patient?

- A. Cancer of intestines
- B. Chronic enteritis
- C. Cron's disease
- D. Polyposis of intestines
- E. Ulcerative colitis

2. The man of 48 years old complains on dull pain in lateral parts of belly which relieve after defecation and passage of gases; alternation of diarrhea and constipation. He had dysentery 2 years ago. On abdominal palpation – tenderness, alternation of spastic and atonic sections of large intestine, rumbling. What method of examination is the most informative one for making of the diagnosis:

- A. Coprocytogram in dynamics
- B. Manual examination of rectum
- C. Sigmoidoscopy
- D. Colonoscopy
- E. Ultrasonography

3. The patient of 32 years old took massive antibacterial therapy. Complains on pains in abdomen, frequent liquid excrements (4-6 times a day), and general weakness. On palpation abdomen is soft, tender in the lower parts, liver and spleen are not enlarged. Prescription of what preparation is reasonable in this case?

- A. Imodium
- B. Panzynorm
- C. Essentiale
- D. Motilium
- E. Linex

4. Patient of 45 years old has diarrhea 6-8 times a day, feces contain mucus, blood and pus. Body temperature is 37,6; abdominal pains during palpation. He suffers from formation of internal and external fistulas from time to time. The diagnosis is Crohn's disease. What sign differentiates this disease from ulcerative colitis?

- A. Abdominal pain during palpation
- B. Diarrhea
- C. Fistulas
- D. Blood in feces
- E. Fever

5. Man of 26 years old, complains on cramp-like pains in abdomen, frequent defecations with liquid feces with mucous and blood. He has been sick for 3 years, his weight decreased by 14 kg. T- 37,6 C; abdomen is soft, tender when palpated along the large intestine, especially in the left side. Irrigoscopy shows the signs of narrowing of large intestine, loss of haustration, the contours are uneven. What is the most probable diagnosis?

- A. Ulcerative colitis
- B. Tuberculosis of intestines
- C. Intestinal amebiasis
- D. Crohn's disease
- E. Irritated bowel syndrome

6. The patient of 55 years old, complains on swelling and rumbling in abdomen; increased passage of gases; frequent defecations of foamy character, with a sour smell, which appear after intake of dairy products. How is this syndrome named?

- A. Syndrome of fermentative dyspepsia
- B. Syndrome of putrefactive dyspepsia
- C. Syndrome of fatty dyspepsia
- D. Syndrome of a dyskinesia
- E. Syndrome of malabsorption

7. The Patient of 41 years complains on frequent defecations (10-12 times a day) with mucus and blood; pain in the lower abdomen, loss of weight. He has been sick for 2 years. The diagnoses of acute infectious diseases were excluded. Abdomen is soft, but there is tenderness during palpation of sigmoid colon. Colonoscopy - in the certain part of sigmoid colon the mucosa is pale, with pseudopolyps and flat superficial ulcers. What is the preliminary diagnosis?

- A. **Polyposis of intestines**
- B. Chronic colitis
- C. Chronic pancreatitis
- D. Crohn's disease
- E. Ulcerative colitis

8. The patient of 19 years old, complains on cramp-like pain in abdomen, frequent defecations 6-8 times a day with mucus and blood. He has been sick for 2 years, has lost 12 kg of weight. Abdomen is soft, tender when palpated along the large intestine, especially at the left side, sigmoid colon is spastic. Irrigoscopy - the large intestine is narrowed, haustras are absent, contours are uneven, the symptom of "drain pipe" is present. What diagnosis is the most probable one?

- A. Chronic enterocolitis
- B. Ulcerative colitis
- C. Intestinal amebiasis
- D. Tuberculosis of intestines
- E. Crohn's disease

9. The patient of 48 years old, complains on spastic pains in the lower abdomen, which worsen after psycho-emotional stress. Defecations are intermittent: 2-3 defecations after awakening alter

with constipation during next 1-2 days. There is moderate pain during palpation of sigmoid colon. Proctosigmoidoscopy is painful because of spastic condition of intestines; the mucosa of intestines is not changed; there is a lot of mucus in the lumen. What disease is the most probable one?

- A. Ulcerative colitis
- B. Crohn's disease
- C. Irritated bowel syndrome
- D. Acute ischemia of intestine
- E. Syndrome of malabsorption

10. The patient of 51 years old, complains on frequent liquid defecations with mucus and blood streaks, diffuse pain in inferior and lateral parts of abdomen, weight loss of 6 kg during 1 month, low grade fever. The abdomen is soft, sigmoid colon is tender, spastic, rumbling. What is the most possible disease in this patient?

- A. Intestinal enzymopathy
- B. Bacillary dysentery
- C. Sprue
- D. Ulcerative colitis
- E. Helminthiasis

CORRECT ANSWERS

- 1. E
- 2. D
- 3. E
- 4. C
- 5. A
- 6. A
- 7. E
- 8. E
- 9. C
- 10. D

Control questions.

1. Give definition of UC, Crohn's disease, IBS;
2. Basic clinical syndromes of IBD;
3. Data of physical examination of IBD patients;
4. Clinical presentatiton of UC;
5. Clinical presentatiton of Crohn's disease;
6. Clinical presentatiton of IBS;
7. Methods of IBD diagnosis;
8. Complication of IBD;
9. Approach of treatment of UC and Crohn's disease.
10. Approach of treatment of IBS.
11. Lifestyle and diet therapy at IBS.
12. Medical therapy of IBD depending on severity and activity of disease
13. Preventive measures for IBD and IBS.

Practical tasks.

1. To make examination of patients with IBD and IBS.
2. To provide interpretation for the data of laboratory test.
3. To provide interpretation for the data of instrumental tests.
4. To make differential diagnosis between IBD and IBS.

5. To list complications of IBD.
6. To write recipes concerning the therapy of IBD and IBS.

The report of clinical supervision of the patient (the uniform form)

Name, Surname _____

Age _____ Profession _____

Complaints _____

Anamnesis morbi

Last aggravation _____

Anamnesis morbi

Results of physical examination of the patient:

The preliminary diagnosis:

The examination plan:

Results of additional studies:

Substantiation of the clinical diagnosis:

The clinical diagnosis:

Main disease: _____

Accompanying diseases:

Complication

Treatment:

Regimen _____

Diet _____

Materials for self-preparation:

1. Davidson's "Principles and Practice of Medicine" 21st edition, Alimentary tract and pancreatic disease, p. 835-919.
2. Current Medical Diagnosis and Treatment, Gastrointestinal disorders, 2014, p. 564-662
3. Harrison's, Principles of Internal Medicine, 19th edition, Gastroenterology and Hepatology, p.257-398
4. V.G.Perederiy, S.M.Tkach. Clinical lectures on internal diseases in 2 volumes. Kiev, Manuscript, 1998.
5. V.N.Khvorostinka, N.I. Yabluchanskiy, S.N. Panchuk, L.M. Pasiashvili. Therapeutic gastroenterology, Kharkov, Osнова, 1999, 368 p.
6. I.I. Degtyareva, Diseases of digestive tract.- K.: Demos, 2000,- 412 p.
7. S. M. Gandzha, V.M. Kovalenko, N.M. Shuba, etc. Internal diseases. K.: Zdorovya, 2002. - 992 P-
8. N.I. Shvets, A.V. Pidaev, T.M. Bentsa etc. Standards of practical skills in therapeutics. Kiev, Glavmeddruk, 2005, 540 p.
9. N.I. Shvets, A.V. Pidaev, T.M. Bentsa etc. Urgent conditions in clinics of internal medicine. Kiev, 2006. - 752 p.
10. V.G. Ostapenko, F.M. Zhmudikov. Acute diseases of abdomen.- M: Belarus, 1993. - 432 p.
11. R. Konden, R. Nayhus. Clinical surgery, - M., Practica, 1998. - 716 p.
12. L.T. Malaya, V.N.Khvorostinka, Therapy: guidance for students and interns.- H.: Fact, 2001.- 1032 p.
13. V.N.Khvorostinka., T.A Moiseenko, L.V. Zhuravleva, Faculty therapy: Textbook -H.: Fact, 2000,-888 p.
14. Diseases of digestive apparatus: Guidance in schemes and tables /Under redaction of V.N.Khvorostinka.- H.: Fact, 2001.-239 p.
15. L.T. Malaya, V.N.Khvorostinka, Therapy. Diseases of respiratory system. Diseases of digestive tract. Kidneys disease. Immunology. Allergic diseases. Systemic disease of connective tissue. Blood system diseases: guidance for students and interns, 2nd edition -Kharkov: Folio, 2005.-879 p.

Інформаційні ресурси

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Методична вказівка складена: асистентом А.К. Журавльовою

Методична вказівка переглянута і затверджена на засіданні кафедри:

З доповненнями (змінами) _____

Завідувач кафедри

Л.В. Журавльова

