

Post-infectious irritable bowel syndrome

E. Y. Plotnikova

Kemerovo State Medical University, Kemerovo, Russia

Key words: post-infectious irritable bowel syndrome, diarrhea, probiotics, rifaximin, diagnostics

Irritable bowel syndrome (IBS) is a chronic relapsing and remitting functional disorders of the gastrointestinal tract (GI tract). In the past, IBS was considered a diagnosis of exclusion. In the past 10 -20 years IBS has become a cause of considerable scientific interest. Adoption of the biopsychosocial model [10], the use of symptoms based on diagnostic criteria (for example, III Roman diagnostic criteria), the growth of biological and behavioral research technologies [11, 12] allowed to create an effective territory for new SRC studies with the prospect for more effective treatment.

It is now clear that IBS is not one disease, but a clearly identifiable symptom complex that combines the symptoms of various underlying physiological determinants "from the intestine to the brain and back." Thus, a "magic wand" as a treatment for this disorder is hardly applicable: the treatment should be based on mono — or polinapravlenom pharmacologic effects, for the correction target physiological determinants, individual for each person [13].

IBS — suffering, having polyethological nature, characterized by recurrent abdominal pain or discomfort along with frequency or stool-type disorders [21].

IBS has a certain symptomatology — "ABCD" of irritable bowel syndrome:

A — abdominal pain or discomfort, usually in the lower abdomen, but can be without a clear localization throughout the abdomen (**A**bdominal pain or discomfort).

B — bloating of the abdomen or feeling of fullness (**B**loating or visible distention).

C — constipation: difficulty evacuation during defecation or rare defecation (**C**onstipation: hard, difficult-to-evacuate, or infrequent stools).

D — diarrhea: mushy, watery or frequent stools (**D**iarrhea: loose, watery, or frequent stools).

Extra-intestinal symptoms such as fatigue, headache, back pain, muscle pain, sleep disorders (Extra-bowel symptoms such as fatigue, headache, backache, muscle pain, and sleep disturbance).

➤ Based on the symptoms:

- ❖ IBS with predominance of intestinal dysfunction
- ❖ IBS with prevalence of pain syndrome
- ❖ IBS with predominance of flatulence

➤ Based on the presence of aggravating factors:

- ❖ Postinfectious IBS (PI-SRK)
- ❖ IBS associated with certain foods
- ❖ IBS associated with stress

In this article, we want to make out more detail the type of IBS, which is found in almost one in three patients who had bacterial, viral or parasitic acute gastro-intestinal infections (AII) — post-infectious IBS (PI-IBS). For the first time PI-SRK described NA Chaudhary and SC. Truelove [5]. Fter studying 130 cases of 'syndrome and irritable bowel ', they revealed 26% of patients in whom IBS was the result of a previous dysentery.

Most people who develop acute bacterial diarrhea she spontaneously ie disappearance of symptoms occurs within <5 days, but some patients develop non-specific intestinal symptoms that mo st be manifested over the years [32]. PI- IBS occurs in 4 — 32% of patients after suffering a bacterial gastroenteritis and th [43] as a response to nonspecific infections caused by various intestinal pathogens, such as, for example, *Campylobacter*, *Salmonella*, *diarrheal strains Escherichia coli*, *Shigella*, *Entamoeba histolytica*, *Yersinia*, *Cryptosporidia*, *Legionella* [26, 23, 45, 49]. K. Rex Douglas in his study found that PI-IBS develops after a lambliaisis in 46% of cases, in the control group in 14% [37].

PI-IBS is diagnosed if there are symptoms consistent with the III Roman diagnostic criteria of IBS — the presence of recurrent abdominal pain or discomfort for at least 3 days per month for the last 3 months with the onset of symptoms of at least 6 months that should be combined with two or more of the following signs: improvement after defecation; the beginning is associated with a change in stool frequency; The beginning is associated with a change in the shape of the stool [21].

After acute infectious gastroenteritis episode must have two of the following symptoms: fever, vomiting, diarrhea and detection AII markers in biological fluids of the patient [17]. As a rule, acute infectious symptoms of vomiting and fever are stopped after a few days with the permission of eating infection, however, abdominal discomfort, bloating, and diarrhea persist. A number of factors, such as duration and severity of OCI, may increase the risk of developing PI-IBS [25, 31].

A key factor in the development of IBS is abnormalities in the brain's intestinal system. A number of neurotransmitters, such as cholecystokinin, vasoactive intestinal peptide, serotonin, etc. To date, more than 60 genes have been examined for their effect on the development of IBS.

PI-IBP occurs as a result of a sharp response of the immune system to an infection, with a subsequent slow weakening of the immune response. In this case, patients have mild inflammation of the colon and increased intestinal permeability. Important regulators of the immune response are cytokines. In a number of independent studies, polymorphism has been linked in the IL-4, IL-6, IL-10, and TNF- α genes encoding pro — inflammatory interleukins, with the development of post-infection IBS. Gwee et al. [27] reported an increase in IL-1b expression in rectal biopsy in patients with PI-IBS, compared with those who had infectious enteritis without the subsequent PI-SRK. Wang et al. [28] also observed an increase in IL-1b patients with IBS-PI after shigellosis infection compared with patients without IBS [49]. A recent study by Villani A, et al. [48] identified three genes — TLR9, CDH1 and IL6, which were associated with the development of PI-IBP. The TLR9 gene encodes a transmembrane receptor, which is an important component of the body's immune response. Also, a single nucleotide substitution link was found in the promoter of the CDH1 gene, which encodes a transmembrane glycoprotein regulating intercellular adhesion, with the development of post-infective CPK [46, 51].

In patients with PI-IBS, a number of studies have shown an increase in serotonin levels in enterochromaffin cells (EC) of the intestine compared to healthy people. The results of biopsy in all patients were within the normal range using the usual criteria, however, a quantitative study of n had an increase in EC cells ($p = 0.017$) in comparison with the control group ($p = 0.02$). Own plate was also increased T lymphocytes ($p = 0.026$) and mast cell number ($p = 0.054$) compared to the control group [16]. An increase in the number of EC cells by 25% in the rectal zone may lead to an increase in the level of serotonin and to subsequent diarrhea. In patients with PI-IBS, an increase in postprandial serotonin in plasma compared with patients with IBS-3 (constipation) and healthy volunteers [2].

In ospalenie mucosa with variable OCI yaet visceral th September ku with ori, and a top portion precedes the symptoms in patients with IBS PI. E ven easy asymptomatic acute colitis can cause long-term th visceral hyperalgesia in the presence of additional stimuli [41]. This "swelling" caused a painful reaction in patients with IBS significantly faster than in the control group [38, 39].

Risk of development PI IBS armature p eliruet with the severity and AII povysh aetsya at least twice if diarrhea lasts for more than 1 week spruce, and three times if the diarrhea lasts for more than 3 weeks [24, 31]. Abdominal cramps and pain, weight loss, bloody stools increasing the risk of th t the occurrence of PI-IBS by a factor of four [24, 31]. Pathogens like *Campylobacter* still is, and of *Shigella*, can cause more serious damage to the mucous membrane and longer disease than *Salmonella* [7]. In a study of 231 patients who were observed for 3 months, more often (4.2% of 119 patients) developed PI-IBP after *Campylobacter* infection than

after infection with Salmonella (2.6% of 38 patients), but this difference is not was significant [25].

M. A. Sykes et al. [33] determined that people with psychiatric premorbid diagnoses, in particular anxiety disorders, are also at increased risk of developing PI-IBP after OCD. In addition, depression, neuroticism, somatization disorders, stress and negative perception of the disease are also associated with PI-IBS [9, 36, 42]. In a recent study, patients who developed PI-IBS had a significantly higher level of perception of tension, anxiety, somatization, and a negative attitude toward the disease during OCI than patients who did not develop PI-IBP [42]. In addition, Gwee et al. [40] found that patients with PI-IBS had higher rates of hypochondria. These observations suggest a psycho somatic interactions that s mo gut cause symptoms of PI-IBS and keeping the five long-term mental disorders in these patients[42]. This paradigm provides support for cognitive-behavioral therapy for the treatment of PI-IBS. For an objective assessment of the psychological picture, the Hospital Anxiety and Depression Scale (HADS) can help [1]. We in our practice regularly use this simple questionnaire of 14 items, designed to assess the level of anxiety and depression.

Despite the fact that there is no gender th t b d differences in the development of acute intestinal and immune response to th e risk of PI-IBS is higher among women than men [8, 42]. Women's susceptibility to the PI-IBS may be caused by a more pronounced reaction to the psychological stress s. In a study by Gwee et al. [40], the female sex is not was a significant risk factor when psychological variables are controlled in a multivariate analysis. In two studies, the risk of developing PI-IBP decreases with an increase in age over 6 0 years [31]. Dunlop et al. [15] showed that older people have fewer lymphocytes and mast cells in the rectal mucosa, which can ease inflammatory responses in the lumen of antigens and help reduce the risk of PI-IBS.

The likelihood of developing PI-IBS increases sixfold after acute gastrointestinal infection in the presence of the following risk factors and the development of PI-IBS: younger age, female gender,diarrhea> 7 days, the presence of blood in the stool, abdominal pain, weight loss, at least ten pounds (about 4.5 kg), anxiety or depressive disorder, hypochondriasis, previous adverse life events [44].

The diagnosis of PI-IBS is a diagnosis that can be delivered after a thorough examination of the patient, allowing the patient to exclude organic disease is, I am the gastrointestinal tract. The decisive role in the examination of patients with PI- IBS has data Irrigoscopy, computer colonoscopy, duodenoscopy, sigmoidoscopy or endoscopic colonoscopy for the exclusion of inflammatory bowel diseases,celiac disease, parasitosis and other bowel diseases (Table 1). Fecal examination was performed to determine the number of leukocytes, latent blood, parasites. The detection of diverticulosis of the intestine does not contradict the diagnosis of PI- IBS. Some patients with celiac disease and sprue also have PI- IBS symptoms. In patients with PI- IBP with HLA-DQ2 and intestinal antibodies to gliadin and other edible proteins, after deficit of gluten, the frequency of defecations and the level of intestinal Ig decrease.

Table 1
Differential diagnosis with PI-IBS [44]

Diagnosis	Main features
Colon cancer	L IC and ≥ 40 years of age and the patient's family history with colon cancer
Diverticular disease	P e revied the patient's
Celiac disease	Retroduodenal biopsy ; increased titers of various antibodies:antigliodine (to L-gliadin); to the endomyosin of smooth muscle cells, tissue transglutaminase
Crohn's disease	Anemia, increased ESR, nocturnal diarrhea and pain
Ulcerative colitis	Colonoscopy and biopsy
Drug-induced diarrhea	Reception of antibiotics, magnesium-containing antacids, proton pump inhibitors, angiotensin-converting enzyme or statins

Microscopic colitis	Recto-manoscopy and biopsy
Syndrome of excessive bacterial growth	Respiratory hydrogen test with lactulose
Lactase insufficiency	Respiratory hydrogen test with lactose

Treatment of PI-IBS, as a rule, is symptomatically directed and includes dietary recommendations, psychotherapy and funds intended for the treatment of IBS with diarrhea (IBS-D). Empathy and doctor support are crucial for patient. The physician should emphasize that PI-IBS is not a life-threatening disorder, and should soothe the patient, explain that his symptoms are not imaginary and are associated with various factors. Dietary recommendations are aimed at excluding products provoking diarrhea and provoking flatulence, the food should be chemically and thermally sparing with good culinary processing. Psychotherapeutic measures are aimed at eliminating tension, anxiety, depression, hypochondria, and includes various techniques and / or psychotropic correction. It is reasonable to assign psychotropic drugs that have a side effect of constipation and in this situation working t anxiolytics or antidepressants, while reducing or eliminating diarrhea. These drugs include : sulpiride,amitriptyline, antidepressants with a 5-HT₁ application point or 5-HT₃ receptors, antihistamines and GABAergic Anxi about litiki.

Antidiarrheal agents, such as loperamide, can be effective in reducing the frequency of diarrhea. Loperamide inhibits motility and fluid secretion, which leads to an increase in the transit of timgastrointestinal tract, improves uptake and e fluids and electrolytes in the gastrointestinal tract. However, loperamide does not reduce pain in the abdomen associated with PI-IBS [3]. Common adverse reactions to loperamide include cramps and nausea. It is also necessary to exclude the syndrome of excessive bacterial growth in the small intestine, in which the reception of loperamide can increase intoxication.

Quite often PI-IBS is associated with SIBR, especially after courses of specific therapy for OCD. In such patients, malabsorption syndromes, maldigestions are even more pronounced, disconjugation of bile acids is disturbed, flatulence, diarrhea and abdominal pain disturb. And Exclude it or Confirm dit diagnosis ARIS helps hydrogen breath test with lactulose. When combined with PI-IBP and SIBR,antibiotics [47], prebiotics and probiotics are indicated [1]. The drug of choice for decontamination of the small intestine in many studies and recommendations was rifaximin, which showed good results in the treatment of IBS. In a randomized a placebo-controlled study of 124 patients conducted a 10-day course of rifaximin 400 mg 3 times daily or placebo, rifaximin statistically significant l Reduce andbloating flotation [1]. These results were confirmed in a double-blind, placebo-controlled study involving 87 patients with IBS in 2006. After completion and treatment, most patients in the rifaximin groupWe noted a significant decrease in global IBS symptom s (37,7% vs 23,4%) (p <0,05) and stable clinical response s (37,2% vs 15,9%) (p <0,05) [18].

The leading symptom of PI-IBS is pain caused by spasm, which is based on an involuntary contraction of the smooth muscles of the intestines, not accompanied by their immediate relaxation. The main mechanisms of development of abdominal pain are caused by a violation of intestinal motility and visceral hypersensitivity. In the national practical guidelines (Guideline IBS) of different countries, the main antispasmodics of various groups are recommended : otilonium, trimebutine, cimetropium, hyoscine, pinaverium, alverine, mebeverine, including peppermint (peppermint oil). All of them showed a significant advantage in comparison with placebo [29]. Depending on the main mechanism of influence on the stages of muscle fiber reduction, several groups of muscle relaxants are isolated.

Anticholinergics (atropine, platifilin, hyoscine butyl bromide) reduce the concentration of intracellular calcium ions, which leads to muscle relaxation. It is important to note that the degree of relaxation is directly dependent on the previous tone of the parasympathetic nervous system.

Blockers phosphodiesterase — myotropic antispasmodics (papaverine, drotaverin) contribute to the accumulation of cAMP in the cell and a decrease in the concentration of calcium ions, which inhibits the combination of actin with myosin. These drugs are used for a short time (from a single dose to a week) to stop the spasm, but not for a course of treatment aimed at stopping and preventing the recurrence of the disease.

A definite value in the regulation of the motor function of the gastrointestinal tract is given to endogenous opiates. Currently, the agonist is used in the treatment of IBS patients opiate receptors -trimebutin — regulator of gastrointestinal motility. In a randomized placebo-kontrolliruemmyh studies of IBS A. O. Quartero et al. (2005) and A. The C. Ford et al. (2008), trimebutin showed no significant benefit and was not statistically significantly different from placebo [4, 19].

Mebeverin (Duspatalin, ABBOTT PRODUCTS Ltd.) is an antispasmodic that has been successfully used in the treatment of IBS for many years. It has an antispasmodic effect, normalizing the motor function of the intestine [50]. In therapeutic doses, mebeverine has a direct blocking effect on sodium channels, which limits the influx of Na⁺ and prevents the sequence of events leading to muscle spasms. During ingestion of a dose of 135 — 270 mg three times a day, it does not cause typical x anticholinergic side effects, such as with uhost mouth, blurred vision, and urinary disorders. The incidence of side effects caused by mebeverine was not higher than placebo. This drug is now sold in about 56 countries, and its efficacy and tolerability have been demonstrated in 10 controlled and in many open clinical trials [30, 50]. Assign Dyuspatalin with IBS for 1 capsule (200 mg) 2 times a day, take 20 minutes before meals.

Ditsetel ® (pr-in Abbott Products Ltd), the main active substance is the pinaverium bromide is an antispasmodic myotropic action — a calcium antagonist for the treatment of intestinal motility disorders [22]. It selectively blocks the potential calcium-dependent channels of the smooth muscles of the intestine and prevents excessive intake of calcium inside the cell. Unlike other calcium antagonists, Dicitel ® exhibits the same affinity to the channels in an accessible and inactivated state, which makes it highly effective and not addictive [20]. The balanced work of the smooth muscle apparatus of the gastrointestinal tract depends on the concentration of calcium in the cytoplasm of the myocyte and its movements through the cell membrane. Calcium ions play a role not only in the "excitation — contraction" bundle, but also in the "excitement — relaxation" bundle. Calcium channels of L-type smooth muscle cells of the gastrointestinal tract are activated in 2 ways:

➤ The first, most fully studied, is characterized by depolarization of the cell membrane by the action of a nerve impulse, followed by contraction of the smooth muscle cell.

➤ The second method involves the activation of the calcium channel by digestive hormones and mediators, such as cholecystokinin, gastrin or P substance. By binding to specific receptors, they activate receptor-controlled Ca²⁺ channels, which causes depolarization of the cell membrane and leads to the discovery of potential-dependent calcium channels. In the event that the calcium channels are blocked by the molecules of pinaverium bromide, the action of the above digestive hormones and mediators can not be realized.

Thus, pinaverium bromide inhibits not only the intestinal hypermotoria, but also the pathway involved in visceral hypersensitivity, the second sign of irritable colon syndrome. In addition, calcium channels, sensitive to mechanical influences, have recently been discovered in the membranes of smooth muscle cells of the human intestine. The existence of this type of calcium channel can change the concept of gastrointestinal smooth muscle as a "purely motor organ," which has both a motor and sensory function. The effectiveness of Dicitel ® in patients with IBS of all types is evidenced by a large number of studies, both in our country and abroad [6, 35]. Most studies Nij show good and very good results for complete relief and reduce the intensity of pain in IBS in patients receiving Ditsetela ® as monotherapy in standard dosage (at 60%).

A review of 22 randomized controlled trials (n = 1778) compared 12 different antispasmodics (including pinaverium, trimebutine and dicyclomine) showed a statistically significant effect of therapy. Decrease in pain occurred in 39% of treated patients, and in 56% of patients receiving placebo. In this case, pinaverium showed a statistically significant effect of therapy unlike trimebutin. The pain syndrome persisted in 28% of patients receiving treatment and in 61% of patients receiving placebo [6, 35].

Dicetel ® in the acute period (3-6 days) is prescribed for 100 mg 2-3 times daily with meals. After decrease exacerbation maintenance dosage pinaveriuma bromide is standard dose — 50 mg 3-4 times a day, that is assigned to the course of 2 to 6 weeks or more. Acting selectively on the intestine, Dicetel ® has no side effects anticholinergic, so it can safely be prescribed to patients with glaucoma and prostatic hypertrophy.

In our clinical practice, we meet quite often with IBS, including PI-IBS. Peculiarities of PI-IBS flow consist in more pronounced and prolonged diarrhea and persistent pain syndrome.

Here are some interesting clinical cases from our practice.

Patient M., 35 years old. In December 2009, suffered a severe stress (death of his mother from cancer), after a few days had diarrhea up to 5 — 7 times a day, which has progressed, independent attempts were in vain to stop the symptoms. In January 2010, he turned to an infectious disease specialist, he was diagnosed with antibodies to yersiniosis and a course of antibiotic treatment was conducted. Diarrhea persisted. In February 2010, the re-testing the titer of antibodies to Yersinia about memory remained at the same level, a course of antibiotic therapy was repeated. After the second course of treatment di and Reynaud the syndrome persisted. During the treatment weight loss more than 10 kilograms, decreased ability to work, pronounced weakness, anhedonia, carcinophobia, insomnia. From March and by April 2010 had a meeting with several gastroenterologists examination — clinical and biochemical blood tests, barium enema, colonoscopy, koproovoskopiya — without pathology, the conventional treatment of diarrhea (loperamide, smectite) was ineffective. In May 2010, a course of treatment is scheduled, taking into account the main symptoms of PI-IBS: Dietz e tel 200 mg in the morning and 100 mg in the evening for a month, Prosulfin 50 mg 3 times a day for a month. A respiratory hydrogen test with lactulose was prescribed, which resulted in high bacterial contamination of the small intestine. By treatment added default Normiks 400 mg 3 times daily — the course 6 days, then Lineks 2 capsules 3 times daily — the course of 14 days and Dufalac 5 ml to 3 months. After a month of treatment, a significant improvement is noted, the stool is not more often 1 to 2 times and in the morning after breakfast, there remains a moderate anxious-depressive symptomatology, a fear of regaining diarrhea. Dicetel Recommended for admission on demand for abdominal pains, instead of prosulfin, Trazodone is prescribed 150 mg per day up to 6 months. In December 2010 — the next visit to the gastroenterologist — no complaints, weight recovered, sports, returned to normal professional activities.

Patient P., 86 years old. Directed infectious disease specialist in April 2012 with a strong abdominal pain, diarrhea, fear of th meal, which increases the pain in the abdomen. The history month ago suffered OCI combined with giardiasis, was treated with antibiotics, nitrofurans, I lost 7 — 8 kg. Examinations — clinical and biochemical blood tests, ultrasound of the abdominal cavity, FGD, colonoscopy — within the age limit. In the hydrogen respiratory test with lactulose, SIBR was detected. Treatment is prescribed : Dicetel 200 mg in the morning and 100 mg in the evening for a month, Alfa-Normix 400 mg 2 times a day for 6 days, then Riaflora Balance 2 capsules 2 times a day for 14 weeks. A month later at the reception of the gastroenterologist — a significant improvement in the state of health, abdominal pain occurs much less often, the chair is not more often 2 times a day, there is a fear of eating, disturbed sleep. Recommended intake Ditsetela 50 — 100 mg with abdominal pain on demand, fluvoxamine 50 mg at bedtime for six months. In June 2012, a significant improvement in well-being, weight gain, stool normalized, widened the diet in consumed products, continues to receive Fluvoxamine.

Conclusion. Treatment of PI-IBS should have clear and realistic goals, to which one should strive, both to the doctor and the patient. Coordinated treatment tactics should include

measures to reduce symptoms (or elimination) and improve the quality of life of patients. Most patients need a correction of SIBR by decontamination of the small intestine with the help of modern enteroseptics. Parts of patients with the course of PI-IBS of moderate severity and severity, rational psychotherapy is often necessary, the administration of drugs of different pharmacological groups — antispasmodics, probiotics, prebiotics, antibiotics, anti- drugs, antidepressants and some others. It is reasonable to use different schemes of these drugs, both for treating the symptoms of exacerbation, and with a preventive goal for lengthening remission and improving the quality of life of patients with PI-IBS.

References:

1. Практические рекомендации Всемирной гастроэнтерологической организации. Синдром раздраженного кишечника: глобальные перспективы / E. Quigley, M. Fried, K. A. Gwee [et al.] — 20 апреля 2009. — Available at: <http://www.worldgastroenterology>
2. Abnormalities of 5-hydroxytryptamine metabolism in irritable bowel syndrome / S. P. Dunlop, N. S. Coleman, E. Blackshaw [et al.] // *Clin. Gastroenterol. Hepatol.* — 2005. — Vol. 3. — P. 349–357.
3. Baker D. E. Loperamide : a pharmacological review / D. E. Baker // *Rev. Gastroenterol. Disord.* — 2007. — Vol. 7, Suppl. 3. — S. 11–18.
4. Bulking agents, antispasmodic and antidepressant medication for the treatment of irritable bowel syndrome / A. O. Quartero, V. Meineche-Schmidt, J. Muris [et al.] // *Cochrane Database of Systematic Reviews.* — 2005. — Vol. 2 — CD003460.
5. Chaudhary N. A. The irritable colon syndrome : a study of the clinical features, predisposing causes, and prognosis in 130 cases / N. A. Chaudhary, S. C. Truelove // *Q. J. Med.* — 1962. — Vol. 31. — P. 307–322.
6. Christen M. O. Action of pinaverium bromide, a calcium-antagonist, on gastrointestinal motility disorders / M. O. Christen // *General Pharmacology: The Vascular System.* — 1990. — Vol. 21, No 6. — P. 821–825.
7. Connor B. A. Sequelae of traveler's diarrhea: focus on postinfectious irritable bowel syndrome / B. A. Connor // *Clin. Infect. Dis.* — 2005. — Vol. 41, Suppl. 8. — S. 577–586.
8. Development of functional diarrhea, constipation, irritable bowel syndrome, and dyspepsia during and after traveling outside the USA / A. K. Tuteja, N. Talley, S. S. Gelman [et al.] // *Dig. Dis. Sci.* — 2008. — Vol. 53. — P. 271–276.
9. Does bacterial gastroenteritis predispose people to functional gastrointestinal disorders? A prospective, community-based, case-control study / S. D. Parry, R. Stansfield, D. Jelley [et al.] // *Am. J. Gastroenterol.* — 2003. — Vol. 98. — P. 1970–1975.
10. Drossman D. A. Presidential address: gastrointestinal illness and the biopsychosocial model / D. A. Drossman // *Psychosom Med.* — 1998. — Vol. 60. — P. 258–267.
11. Drossman D. A. The functional gastrointestinal disorders and the Rome III process / D. A. Drossman // *Rome III: the functional gastrointestinal disorders* / D. A. Drossman, E. Corazzari, M. Delvaux [et al.]. — 3 rd ed. — VA : Degnon Associates, 2006. — P. 1–30.
12. Drossman D. A. The functional gastrointestinal disorders and the Rome III process / D. A. Drossman // *Gastroenterology.* — 2006. — Vol. 130. — P. 1377–1390.
13. Drossman D. A. Treatment for bacterial overgrowth in the irritable bowel syndrome / D. A. Drossman // *Ann Intern Med.* — 2006. — Vol. 145, No 8. — P. 557–563.
14. Du Pont H. L. Postinfectious irritable bowel syndrome: clinical aspects, pathophysiology, and treatment / H. L. du Pont // *Practical Gastroenterology.* — 2007. — Volume XXXI, No 9. — P. 18–24.
15. Dunlop S. P. Age-related decline in rectal mucosal lymphocytes and mast cells / S. P. Dunlop, D. Jenkins, R. C. Spiller // *Eur. J. Gastroenterol. Hepatol.* — 2004. — Vol. 16. — P. 1011–1015.

16. Dunlop S. P. Distinctive clinical, psychological, and histological features of postinfective irritable bowel syndrome / S. P. Dunlop, D. Jenkins, R. C. Spiller // *Am. J. Gastroenterol.* — 2003. — Vol. 98. — P. 1578–1583.
17. Dunlop S. P. Distinctive histological patterns of chronic inflammatory cells in rectal biopsies of patients with different clinical subtypes of IBS / S. P. Dunlop, D. Jenkins, R. C. Spiller // *Gastroenterology.* — 2002. — Vol. 122, Suppl. 1. — P. 60.
18. The effect of a nonabsorbed oral antibiotic (rifaximin) on the symptoms of the irritable bowel syndrome : a randomized trial / M. Pimentel, S. Park, J. Mirocha [et al.] // *Ann. Intern. Med.* — 2006. — Vol. 145. — P. 557–563.
19. Effect of fibre, antispasmodics, and peppermint oil in the treatment of irritable bowel syndrome : systematic review and meta-analysis / A. C. Ford, N. J. Talley, M. R. Spiegel [et al.] // *BMJ.* — 2008. — Vol. 337. — P. 2313.
20. Effectiveness of pinaverium bromide therapy on colonic motility disorders in irritable bowel syndrome / T. Wittmann, A. Feher, A. Rosztoczy, J. Janosi // *Orv. Hetil.* — 1999. — Vol. 140, No 9. — P. 469–473.
21. Functional bowel disorders / G. F. Longstreth, W. G. Thompson, W. D. Chey [et al.] // *Gastroenterology.* — 2006. — Vol. 130. — P. 1480–1491.
22. Guslandi M. The clinical pharmacological profile of pinaverium bromide / M. Guslandi // *Minerva Med.* — 1994. — Vol. 85, No 4. — P. 179–185.
23. Gwee K. A. Increased rectal mucosal expression of interleukin 1(5 in recently acquired postinfectious irritable bowel syndrome / K. A. Gwee, S. M. Collins, N. W. Read // *Gut.* — 2003. — Vol. 52. — P. 523–526.
24. Incidence and epidemiology of irritable bowel syndrome after a large waterborne outbreak of bacterial dysentery / J. K. Marshall, M. Thabane, A. X. Garg [et al.] // *Gastroenterology.* — 2006. — Vol. 131. — P. 445–450.
25. The incidence of irritable bowel syndrome among community subjects with previous acute enteric infection / M. R. Borgaonkar, D. C. Ford, J. K. Marshall [et al.] // *Dig. Dis. Sci.* — 2006. — Vol. 51. — P. 1026–1032.
26. Increased rectal mucosal enteroendocrine cells, T lymphocytes and increased gut permeability following acute *Campylobacter* enteritis and in post-dysenteric irritable bowel syndrome / R. C. Spiller, D. Jenkins, J. P. Thornley [et al.] // *Gut.* — 2000. — Vol. 47. — P. 804–811.
27. Increased rectal mucosal expression of interleukin 1 beta in recently acquired post-infectious irritable bowel syndrome / K. A. Gwee, S. M. Collins, N. W. Read [et al.] // *Gut.* — 2003. — Vol. 52. — P. 523–526.
28. Intestinal permeability in patients with irritable bowel syndrome after a waterborne outbreak of acute gastroenteritis in Walkerton, Ontario / J. K. Marshall, M. Thabane, A. X. Garg [et al.] // *Aliment. Pharmacol. Ther.* — 2004. — Vol. 20. — P. 1317–1322.
29. Irritable bowel syndrome in adults. Diagnosis and management of irritable bowel syndrome in primary care / National Collaborating Centre for Nursing and Supportive Care. — No. 61. — London : National Institute for Health and Clinical Excellence, 2008. — 881 p.
30. McCallum R. W. The role of calcium and calcium antagonism in motility disorders of the gastrointestinal tract / R. W. McCallum // *Calcium antagonism & Gastrointestinal motility // Experta Medica.* — 1989. — P. 28–31.
31. Neal K. R. Prevalence of gastrointestinal symptoms six months after bacterial gastroenteritis and risk factors for development of the irritable bowel syndrome : postal survey of patients / K. R. Neal, J. Hebden, R. Spiller // *BMJ.* — 1997. — Vol. 314. — P. 779–782.
32. Neal K. R. Prognosis in post- infective irritable bowel syndrome : a six year follow up study / K. R. Neal, L. Barker, R. C. Spiller // *Gut.* — 2002. — Vol. 51. — P. 410–413.

33. Psychopathology in irritable bowel syndrome: support for a psychophysiological model / M. A. Sykes, E. B. Blanchard, J. Lackner [et al.] // *J. Behav. Med.* — 2003. — Vol. 26. — P. 361–372.
34. A randomized double-blind placebo-controlled trial of rifaximin in patients with abdominal bloating and flatulence / A. I. Sharara, E. Aoun, H. Abdul-Baki [et al.] // *Am. J. Gastroenterol.* — 2006. — Vol. 101. — P. 326–333.
35. Reduction of post-prandial motility by pinaverium bromide a calcium channel blocker acting selectively on the gastrointestinal tract in patients with irritable bowel syndrome / R. A. Awad, V. H. Cordova, M. Dibildox [et al.] // *Acta Gastroenterol. Latinoam.* — 1997. — Vol. 27, No 4. — P. 247–251.
36. Relative importance of enterochromaffin cell hyperplasia, anxiety, and depression in postinfectious IBS / S. P. Dunlop, D. Jenkins, K. R. Neal, R. C. Spiller // *Gastroenterology.* — 2003. — Vol. 125. — P. 1651–1659.
37. Rex D. K. Add parasites to the causes of postinfectious irritable bowel syndrome / D. K. Rex // *Gut.* — 2012. — Vol. 61. — P. 214.
38. Ritchie J. Pain from distension of the pelvic colon by inflating a balloon in the irritable colon syndrome / J. Ritchie // *Gut.* — 1973. — Vol. 14. — P. 125–132.
39. Rogers J. Increased segmental activity and intraluminal pressures in the sigmoid colon of patients with the irritable bowel syndrome / J. Rogers, M. M. Henry, J. J. Misiewicz // *Gut.* — 1989. — Vol. 30. — P. 634–641.
40. The role of psychological and biological factors in postinfective gut dysfunction / K. A. Gwee, Y. L. Leong, C. Graham [et al.] // *Gut.* — 1999. — Vol. 44. — P. 400–406.
41. Severity of mucosal inflammation as a predictor for alterations of visceral sensory function in a rat model / A. B. T. Liebrechts, J. M. Gschossmann [et al.] // *Pain.* — 2006. — Vol. 123. — P. 179–186.
42. Spence M. J. The cognitive behavioural model of irritable bowel syndrome : a prospective investigation of patients with gastroenteritis / M. J. Spence, R. Moss-Morris // *Gut.* — 2007. — Vol. 56. — P. 1066–1071.
43. Spiller R. C. Post-infectious irritable bowel syndrome / R. C. Spiller, E. Campbell // *Curr. Opin. Gastroenterol.* — 2006. — Vol. 22. — P. 13–17.
44. Thabane M. Systematic review and meta-analysis : the incidence and prognosis of post-infectious irritable bowel syndrome / M. Thabane, D. T. Kottachchi, J. K. Marshall // *Aliment. Pharmacol. Ther.* — 2007. — Vol. 15. — P. 535–544.
45. Triantafillidis J. K. Postinfectious Irritable bowel syndrome / J. K. Triantafillidis, G. Peros // *Annals of Gastroenterology.* — 2007. — Vol. 20, No 4. — P. 243–245.
46. Van der Veen P. P. Role of tumor necrosis factor-alpha and interleukin-10 gene polymorphisms in irritable bowel syndrome / P. P. van der Veen, M. van den Berg, Y. E. de Kroon // *Am. J. Gastroenterol.* — 2005. — Vol. 100. — P. 2510–2516.
47. Viera J. A. Management of irritable bowel syndrome / J. A. Viera, S. Hoag, J. Shaugnessy // *Am. Fam. Physician.* — 2002. — Vol. 66, No 10. — P. 1867–1875.
48. Villani A. Genetic risk factors for post-infectious IBS in the E. coli 0157: H7 outbreak in Walkerton (Canada) in 2000 / A. Villani, M. Lemire, M. Thabane // *Gastroenterology.* — 2008. — Vol. 134. — A122.
49. Wang L. H. Bacillary dysentery as a causative factor of irritable bowel syndrome and its pathogenesis / L. H. Wang, X. C. Fang, G. Z. Pan // *Gut.* — 2004. — Vol. 53. — P. 1096–1101.
50. Wesdorp I. The central role of Ca⁺⁺ as mediator of gastrointestinal motility / I. Wesdorp // *Calcium antagonism & Gastrointestinal motility // Experta Medica.* — 1989. — P. 20–27.
51. Zanini B. Norovirus gastroenteritis may lead to post-infectious IBS / B. Zanini // *Am. J. Gastroenterol.* — 2012. — Vol. 10. — P. 1038.

Post-infectious irritable bowel syndrome

E. Y. Plotnikova

Kemerovo State Medical University, Kemerovo, Russia

Key words: post-infectious irritable bowel syndrome, diarrhea, probiotics, rifaximin, diagnostics

Data on prevalence and etiopathogenesis of development of a post-infectious irritable bowel syndrome are provided in the article, the main manifestations of this disease and methods of their diagnostics are described. Treatment tactics is considered in details, the application of probiotics both for prevention, and for therapy of the described disease is being emphasized. Different studies on the related subject are presented.