PIĄTKOWSKA, Natalia, KAJZAR, Magdalena, RODAK, Małgorzata, NITKA, Kamila, IWAN, Magdalena, SZYMCZUK, Błażej, SMOROŃSKA-RYPEL, Joanna, TROJANOWSKI, Dominik, MILCZAREK, Jakub and ADAMIEC, Ksawery. Effect of physical activity on the course of inflammatory bowel disease. Quality in Sport. 2024;15:52425. eISSN 2450-3118.
https://dx.doi.org/10.12775/QS.2024.15.52425
https://apcz.umk.pl/QS/article/view/52425

The journal has had 20 points in Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 11.06.2024. Revised: 20.06.2024. Accepted: 01.07.2024. Published: 08.07.2024.

Effect of physical activity on the course of inflammatory bowel disease

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Abstract

The inflammatory bowel disease (IBD) group includes ulcerative colitis (UC), Crohn's disease (CD) and microscopic enteritis. In UC, the lesions involve only the large intestine and do not cross the mucosa, while CD can involve any part of the gastrointestinal tract, from the mouth to the anus. Inflammatory bowel disease usually occurs between the ages of 20-30, with another peak incidence in the seventh decade of life. The etiology of inflammatory bowel disease is not fully understood. Diagnosis is made on the basis of clinical evaluation, endoscopic examination with histopathological evaluation and imaging studies. Treatment involves the introduction of an appropriate diet, and pharmacological, biological and surgical treatments are used. The prognosis is inauspicious, with inflammatory lesions often recurring, even after years of remission of the disease. The purpose of this paper is to describe in detail the two most common diseases in the inflammatory bowel disease group, highlighting the differences and similarities between them at the level of lesion localization, symptoms, diagnosis and treatment. Patients with inflammatory bowel disease, due to the accompanying symptoms that make daily activities difficult, are less active than healthy people. There is ample evidence of the beneficial effects of physical activity on quality and length of life. It is recommended to conduct moderate intensity of both endurance and resistance exercises.

Keywords: inflammatory bowel disease, Crohn disease, ulcerative colitis.

Epidemiology

It has been observed that the incidence of IBD increased significantly in the early 20th century. Most patients are diagnosed in childhood. In the paediatric group, the highest incidence of IBD is found in Europe and North America [1]. The highest number of new cases is reported in Canada, the United States and Western European countries [2].

The incidence also increases at the beginning of the second decade of life. In addition, Western lifestyles and the increasing industrialization of countries predispose to the behaviour [1]. CD is more common in Caucasian people, with a particularly high incidence observed in Ashkenazi Jews. The incidence in Europe is more common in the north than in the south, and lowest in Africa and Asia. The peak incidence occurs between the ages of 15 and 35, and is equally common in both sexes [3]. New cases reported in North America ranged from 6.3 to 23.8 per 100,000 population/year, and in Europe from 0 to 23.1 cases/100,000 [2]. The prevalence of UC is also highest among the Caucasian race. An incidence of about 10/100,000 of the population has been reported in Europe. The disease affects all age groups, but is most common between 20-40 years of age. The incidence affects women and men with similar frequency [4].

Pathogenesis

The pathogenesis of IBD is multifactorial, taking into account the contribution of genetic, environmental, immunological, allergic, bacterial and viral infections [5]. Individuals with a genetic predisposition have an increased risk of IBD due to an abnormal inflammatory response to environmental factors. Intestinal inflammation occurs through cellular response mechanisms and through the release of inflammatory mediators [6]. Activation of the immune system against bacterial antigens and against the colonic mucosa occurs through antigenic cross-reactivity. The inflammatory process is also directed against extraintestinal organs possessing antigens that show similarity to colon antigens [7]. Antigens and toxins from the gastrointestinal tract enter the bloodstream and further into various extraintestinal tissues in the form of antigen-antibody complexes [8]. Autoantibodies against the colonic mucosa cross-react with the biliary epithelium, leading to the development of primary sclerosing cholangitis. In addition, it has been observed that colonic epithelial protein (CEP) and human tropomyosin 5 (hTM5) isoforms present in the colon, as well as in the biliary tract, skin, eyes, and joints, are attacked by autoimmunity [7]. Individuals who carry variants of the Muc2 and FUT2 gene are more susceptible to developing IBD. Muc2 gene variants are responsible for reduced mucus production in the gastrointestinal tract, while FUT2 gene variants reduce ABO antigen secretion [9]. UC is familial in 6-7% of cases. The risk of the disease in first-degree relatives is about 5% [4]. Among the genes predisposing to the development of UC are: NOD2 (nucleotide-binding oligomerization domain containing 2), IL-12B (interleukin 12 beta subunit), JAK2 (Janus kinase 2), STAT3 (signal transducer and activator of transcription 3), CCR3 (C-C chemokine receptor type 3) and TNFSF15 (Tumour Necrosis Factor superfamily member 15) [2].

Among environmental factors, the influence of smoking on the course of inflammatory bowel diseases has been observed. UC is more common in non-smokers, while CD is more common in smokers [6]. The risk of developing UC increases 8-10 times with Salmonella or Campylobacter infection. A history of appendectomy protects against the development of UC [4]. Patients with IBD have been reported to have a higher prevalence of Enterobacteriaceae, Veillonellaceae, Neisseriaceae, Gemellaceae and Fusobacteriaceae, while adherent-invasive strains of Escherichia coli have been observed in the ileum [2]. It has been shown that the incidence of IBD is higher in people who consume highly processed foods while consuming a reduced amount of dietary fiber. The intestinal microbiome is then adversely affected and this predisposes to the development of IBD. The disruption of the intestinal barrier is influenced by the increased use of antibiotics in childhood, the use of non-steroidal anti-inflammatory drugs (NSAIDs) and oral contraceptives, which increases the risk of IBD in the future. Perception of psychological stress disrupts the brain-gut axis, which in turn disrupts intestinal homeostasis and IBD symptoms may recur or worsen [10].

Symptoms

The characteristic symptoms of UC include rectal bleeding and bloody or mucous diarrhoea. When inflammatory lesions involve the rectum there is rectal bleeding, mucosal prolapse and painful constipation. Bloody diarrhoea with mucus and frequent crampy abdominal pain indicate left-sided colitis [6]. The Truelove-Witts scale assesses the severity of UC based on six parameters, such as the number of daily stools, the presence of blood in the stool, body temperature, heart rate, blood haemoglobin level, and erythrocyte sedimentation rate (ESR). Disease remission is defined when there are one to two stools per day without blood, no fever, tachycardia, normal haemoglobin levels, ESR and weight gain. Based on the above criteria, three degrees of disease severity can be distinguished [11]. Fifteen percent of patients have severe flare-ups, which may be the first manifestation of the disease or occur after a prolonged course of the disease [12]. They manifest as passing equal or more than 6 bloody stools per day, weakness, high body temperature, weight loss, tachycardia and hypotension [13]. There is also anaemia and a markedly elevated ESR of more than 30 mm/h [11]. Severe UC can lead to the development of life-threatening complications. The most dangerous form is fulminant UC, which manifests with profuse diarrhoea, severe abdominal pain, which can lead to shock. The lesions involve the entire colon [14]. In mild cast, no abnormalities are found on physical examination; patients report diarrhoea and bleeding [13]. Patients report four or fewer stools per day with little blood. In addition, there is no fever or tachycardia, anaemia is not severe, and ESR does not exceed 30 mm/h [11]. At moderate severity, which affects about one-third of patients, there are 5-6 bloody stools per day, abdominal pain, weakness and elevated body temperature [13].

CD is manifested by abdominal pain, diarrhoea and weight loss. Diarrheal is usually watery, without blood or mucus. Weight loss can result from malabsorption, and this leads to protein, fat and vitamin deficiencies. Features include the absence of rectal lesions and the presence of perianal lesions [6]. On physical examination, a tumour may be found in the right iliac fossa [15]. In addition to gastrointestinal symptoms, systemic symptoms may occur, including fever, weakness and weight loss. Patients with stricture disease may develop intestinal obstruction, most commonly in the small intestine. In the penetrating form, fistulas or abscesses often develop. Depending on the location of the fistula, there are other symptoms. Intestinal-intestinal fistula leads to diarrhoea, intestinal-bladder

fistula leads to urinary tract infection, and vaginal excretion of stool as a result of intestinal-vaginal fistula formation [16,17].

Extraintestinal manifestation (EIM)

Extraintestinal symptoms affect up to 40% of IBD patients. These include diseases that may run parallel to the intestinal inflammatory process, such as peripheral arthritis, episcleritis, erythema nodosum, aphthous stomatitis, and Sweet's syndrome. Independently of enteritis, ankylosing spondylitis, uveitis, orbital myositis, and gastrocnemius pain syndrome occur. However, diseases that may or may not occur in IBD include primary sclerosing cholangitis and pyoderma gangrenosum [18]. Risk factors for EIM include age under 40, female gender, use of biological therapy, family history of IBD, extensive lesions in the gastrointestinal tract, and the presence of any other EIM [19]. It has been observed that extraintestinal manifestations occur more often in patients with CD than with UC. The exceptions are ocular symptoms and primary sclerosing cholangitis, which occur with similar frequency in both types of IBD [20].

Musculoskeletal Manifestations

Muscle involvement is one of the most common extraintestinal manifestations of IBD, especially in CD. Chronic inflammation, nutritional deficiencies, and chronic use of steroids adversely affect muscles and bones. IBD patients are at increased risk of developing secondary osteoporosis. Lower bone mass and reduced bone functionality are more often observed in adults and children compared to healthy people [21]. On the part of the musculoskeletal system, pain and arthritis predominate, and these are frequent extraintestinal manifestations in the pediatric group. It has been observed that joint pain occurs more often than arthritis in the course of UC, affecting 32% of patients with UC, compared to 22% in CD. However, arthritis occurs in 15.5% of children with CD and in 9% of children with UC [22]. Articular lesions can be divided into axial and peripheral arthritis. Among peripheral arthritis, type 1 is distinguished, which is characterized by the involvement of large joints, the inflammatory process is limited to joints and runs parallel to IBD. Type 2, on the other hand, is polyarthritis affecting small joints, occurring independently of IBD [20].

Dermatological Manifestations

Cutaneous symptoms may occur in approximately 10% of IBD patients. The most common cutaneous and mucosal lesions are erythema nodosum, pyoderma gangrenosum and aphthous stomatitis.

As a result of the spread of the inflammatory process from the intestines to the skin and/or mucous membranes, specific changes occur, showing the same histological features as the basic intestinal disease. Ulceration around the anus, facial lesions and non-caseous granulomas in places distant from the gastrointestinal tract, typical of CD, develop.

Mucocutaneous disorders associated with IBD include aphthous stomatitis, psoriasis, erythema nodosum, and epidermolysis bullosa. It has been observed that they occur more often in people expressing human leukocyte antigen (HLA) genes, such as HLA-DR2 and HLA-B27.

Aphthous stomatitis affects approximately 10% of IBD patients. Lesions occur in the form of round, painful ulcers with erythematous edges, which are located on the mucous membrane of the cheeks or lips.

Erythema nodosum occurs in approximately 4-15% of patients with CD and in 3-10% of UC cases. It more often affects women and people with HLA-B27. The characteristic feature is the sudden appearance of symmetrical, erythematous, painful and non-ulcerative nodules, 1-5 cm in size. They can occur in any part of the body, but most often on the extensors of the lower limbs.

Reactive cutaneous and mucosal lesions show different histopathological results than in IBD, but are characterized by a similar pathogenetic mechanism. These include pyoderma gangrenosum (PG), pyodermatitis vegetative (PDPSV), Sweet's syndrome (SS), enteric-associated dermatosis and arthritis syndrome (BADAS), and aseptic abscess syndrome.

Pyoderma gangrenosum is a severe cutaneous manifestation of IBD, affecting 1-2% of patients. It is more common in women, black people, patients with UC and a history of UC. It most often affects the lower limbs and the skin around the stoma.

Initially, papules, pustules or nodules appear, which quickly develop into painful ulcers with typical purple raised edges [23].

Ophthalmic manifestations

The incidence of ocular involvement in UC is 12-35%, and in CD it is 25-70%. Moreover, 6-40% of patients have at least two EIMs [19]. Ocular involvement occurs more frequently in the course of CD and active IBD, especially in the presence of other EIMs. The most common ocular symptoms associated with IBD are episcleritis (2%-5%) and uveitis (0.5%-3.5%). Ocular symptoms are usually non-specific and may be asymptomatic [8]. Eye disorders may be primary or secondary. Primary complications are associated with periods of acute exacerbations and disappear after the initiation of immunological treatment of inflammatory bowel disease. These include: episcleritis, uveitis, scleritis and corneal diseases [19].

The most common ocular manifestation of IBD is episcleritis. It is often characterized by a burning sensation, irritation and redness of one or both eyes.

In patients with IBD, uveitis usually occurs in the anterior chamber of the eye, it affects the iris and ciliary body. Typical clinical symptoms are photophobia, pain, redness, tearing and periodic deterioration of visual acuity.

Scleritis refers to the inflammatory process within the deep blood vessels of the sclera and occurs with a frequency of less than 1%. It manifests itself as redness, irritation and severe pain in the eyes, and may lead to permanent loss of vision or even retinal detachment and optic neuritis.

Secondary complications occur as an undesirable effect of immunological treatment and are the result of primary complications. They are also caused by glucocorticoids, immunosuppressive and anticholinergic drugs. There are usually little or no ocular symptoms. However, long-term use of systemic steroids may lead to posterior subcapsular cataracts. With local use of glucocorticoids, the risk of open-angle glaucoma is increased. Among immunosuppressive drugs, methotrexate has a particularly adverse effect on the visual organ, as it may lead to irritation of the eyelids, cornea and conjunctiva and, similarly to cyclosporine, may lead to optic neuritis, ophthalmoplegia and nystagmus [19].

Hepatobiliary Manifestations

In patients with IBD, hepatobiliary manifestations may be mild or, in more severe cases, progress to liver failure. The most common is primary sclerosing cholangitis, mainly in the course of UC. It is a chronic, cholestatic disease that gradually leads to inflammation and fibrosis of the intra- and/or extrahepatic bile ducts. In approximately 50% of cases, it is asymptomatic, and only after biochemical liver tests are abnormalities detected. Diagnosis is possible based on imaging tests such as endoscopic retrograde cholangiopancreatography, magnetic resonance cholangiopancreatography or percutaneous transhepatic cholangiography.

Gallstones are found in 13-24% of CD patients. The location of inflammatory changes in the intestine is associated with the risk of gallstones. It is smaller when only the ileum is involved compared to the involvement of the cecum-

ileal region. Risk factors for the formation of biliary deposits include enterohepatic circulation disorders resulting from intestinal damage or resection, reduced gallbladder motility, long-term fasting or parenteral nutrition, and previous intestinal resections [24].

Diagnosis

To diagnose IBD, an endoscopic examination of the gastrointestinal tract is performed. It allows you to visualize typical disease lesions. During endoscopy, samples are taken for histopathological examination. UC is indicated by the presence of lesions extending from the cecum proximally [25,26]. The lesions are limited to the mucosa of the large intestine with various degrees of infiltration by lymphocytes, plasma cells and granulocytes. There are distortions of the crypt architecture, their atrophy, abscesses and branching. The presence of Paneth cell metaplasia also indicates a chronic inflammatory process in the intestine [27].

In CD, there are segmental lesions, most often located in the ileocecal region [26]. The macroscopic feature is the presence of fragments of completely normal mucosa separated by inflammatory changes, the appearance of a "cobbled" mucosa. Histological examination reveals a focal, chronic inflammatory infiltrate with lymphocytes and plasma cells, focal irregularity of the intestinal crypts and the presence of granulomas [3]. In 40-50% of patients, the lesions are located in the final part of the ileum. At the same time, the small and large intestine are affected in 30-40% of cases, while only the large intestine in 20% [15]. If there are diagnostic doubts, anemia or gastrointestinal bleeding of a cause not determined by gastroscopy or colonoscopy, capsule endoscopy or double-balloon enteroscopy is performed. Capsule endoscopy is used after excluding intestinal strictures. Double-balloon enteroscopy enables macroscopic assessment along with taking samples for histopathological examination and performing procedures such as dilation of strictures, stopping bleeding and removal of trapped endoscopic capsules. This test is rarely used due to its high price and lack of significant diagnostic advantage over radiological tests or capsule endoscopy [2].

An ultrasound examination of the abdominal cavity is also a useful examination, as it allows to visualize changes in the structure of the intestinal walls and to detect extraintestinal changes [26].

Radiological examination is performed if it is not possible to visualize the final section of the small intestine during colonoscopy [26]. By choice, passage of the small intestine is performed, and less frequently, computed tomography or magnetic resonance imaging with enteroclysis is performed. They make it possible to visualize not only changes in the intestine, but also in other structures of the abdominal cavity. Rectal enema is indicated when colonoscopy is unsuccessful or when strictures occur. It should be remembered that this test should not be performed in severe cases of UC, as it may lead to the development of megacolon toxicum [3].

In laboratory diagnostics, it is useful to determine morphology, inflammatory parameters, and assess the presence of ASCA and ANCA antibodies. CD is characterized by the presence of ASCA antibodies and higher values of inflammatory markers. The presence of ANCA antibodies indicates UC, and anemia is typical of the active form [26]. Calprotectin, lactoferrin, lysozyme, elastase and myeloperoxidase can be determined in feces [3]. During an exacerbation of CD, the blood count shows anemia, thrombocytosis, and leukocytosis. Malnutrition and ongoing inflammation in the body are indicated by a deficiency of total protein and albumin. Inflammatory markers, including CRP, are within the reference range in the remission phase, while in the active phase they increase [2]. Normal inflammatory markers do not exclude the disease [27].

The concentration of calprotectin increases in the active phase of IBD, therefore its measurement is used to monitor the activity of the disease. In patients in remission, its concentration is also measured to assess the risk of disease exacerbation. A concentration above 240 μg/g means a high probability of disease exacerbation within 12 months [2]. Despite numerous diagnostic tests, none of the tests is specific for IBD, because elevated laboratory test results may be elevated in the course of intestinal inflammation or infection [27]. The differential diagnosis of UC should include infectious diarrhea, food allergies, colon polyps, malabsorption syndrome, CD, and irritable bowel syndrome. When CD is suspected, UC, ileocecal lymphoma, infectious diseases, and irritable bowel syndrome should be excluded [25].

Treatment

Medical treatment depends on various factors, such as the severity of the disease, the location of the lesions and the severity of the disease outbreak. Treatment involves inducing inflammation or is aimed at achieving and maintaining remission [25, 28].

A common consequence of IBD is malnutrition, which affects 65-75% of patients with CD and 18-62% of patients with UC. It develops as a result of reduced intestinal absorption, changes in the intestinal microbiota and as a result of nausea, vomiting and loss of appetite. In malnourished patients with CD, in addition to oral nutrition, enteral nutrition (EN) and parenteral nutrition (PN) are recommended as supportive nutrition and before gastrointestinal surgery. Enteral nutrition involves the use of liquid foods, excluding solid foods. It is used for 6-8 weeks during a relapse to achieve remission. Parenteral nutrition involves delivering nutrients through a catheter in a central vein. Indications for its use include: acute inflammation in malnourished patients, difficulties in oral feeding of patients or use of EN for at least 7 days, intestinal obstruction, intestinal ischemia, massive hemorrhage [29].

IBD patients often follow elimination diets. A low-carbohydrate diet (SCD) is used supportively. It excludes all cereals, simple sugars, processed foods and milk. Excluding milk and simple sugars from the diet is intended to reduce the occurrence of gases and fermentation processes. It is recommended to eat most fruits and vegetables, nuts, cottage cheese, cheese, meat, eggs, butter and oils. Additional supplementation with probiotics is recommended to enrich the intestinal flora [14]. Limiting the consumption of carbohydrates is important because they promote the development of bacteria and yeast in the intestine, thus disturbing the balance of gastrointestinal microorganisms. Bacteria and yeast produce toxins and acids that damage the intestinal mucosa and impair the action of digestive enzymes, which promotes the development of colitis [30]. It is also possible to follow a diet with a low content of fermentable oligo-, di- and monosaccharides and polyols (FODMAP), which involves the exclusion of fermentable sugars such as fructo-oligosaccharides, lactose, fructose, galactose and polyols [14]. Patients should limit the consumption of honey, onions, garlic, beans, lentils, legumes and fruits such as dates, watermelon and apples. There is no need to limit sucrose. It has been observed that a semi-vegetarian diet effectively prevents the recurrence of CD. It involves limiting the consumption of meat and fish, but does not eliminate them completely. Recommended products are cereals, milk, yogurts, fruits and vegetables [29]. Patients with IBD should have an individually developed nutritional plan based on the diagnosis of food allergies and intolerances. The diet should provide a full supply of nutrients to prevent deficiencies [14].

Pharmacological treatment uses derivatives of 5-aminosalicylic acid, glucocorticoids, antibiotics and immunosuppressive drugs [5].

In CD, the first-line drugs are oral steroids (prednisolone or budesonide). Antibiotics such as metronidazole or ciprofloxacin may be administered as an adjunct to the treatment of fistulae. Immunosuppressive drugs are used in steroid-resistant and steroid-dependent patients. If there is resistance to glucocorticoids or immunosuppressive drugs, anti-TNF drugs are used–α.

In the course of UC, in the case of low and medium disease activity, 5-ASA is used for induction and maintenance of remission. In moderate and severe disease, when salicylates are ineffective, glucocorticoids are used. Infliximab and Adalimumab are used in steroid-resistant and steroid-dependent patients. In cases that are resistant to pharmacological treatment, in fulminant cases and in conditions that threaten the patient's life, such as massive bleeding, perforation, megacolon toxicum, surgical treatment in the form of colectomy is used.

GCS is used for induction, but should not be used to maintain disease remission. Their use is associated with the risk of numerous side effects, including irreversible ones. Moreover, patients treated chronically with glucocorticoids may develop steroid resistance. Thiopurines (azathioprine, 6-mercaptopurine) are effective in maintaining remission achieved after using glucocorticoids, but the full therapeutic effect appears after 12-16 weeks. From the group of immunosuppressive drugs in CD, methotrexate is used to induce and maintain remission, and in severe flares of UC, cyclosporine is used to postpone colectomy [28].

Biological therapy uses anti-TNF-α antibodies to treat moderate and severe CD in induction and to maintain remission. Among them, the most frequently used are Infiliximab, Adalimumab and Certolizumab [9]. They are used in the case of existing fistulas and when satisfactory therapeutic effects have not been achieved after the use of 5-ASA, glucocorticoids or thiopurines. The use of biological drugs at an early stage of treatment allows for a higher remission rate, reduces the number of hospitalizations and the need for surgery, and reduces the need for glucocorticoids [3].

When conservative treatment is ineffective, the strictures are dilated endoscopically. This applies to short and isolated strictures that are within the scope of the colonoscope. Performing endoscopic dilations allows to postpone or avoid surgery [31].

Surgical treatment varies depending on the type of IBD. In UC, total colectomy is performed, in CD, intestinal-sparing resections and strictureplasty are performed [5]. Approximately ¼ of patients with UC require colectomy because the progressive inflammatory process cannot be controlled [28].

If pharmacological and endoscopic treatment does not provide therapeutic effects, surgical resection of the diseased intestine is performed. Resection should be as limited as possible to prevent short bowel syndrome. Plastic surgery of the narrowed area of the intestine without the need to shorten it is also commonly used. Depending on the size of the stenosis, a different technique is used. For stenosis below 10 cm, a method similar to the Heineke-Mikulicz technique is used, for stenosis above 10 cm and below 25 cm, indirect techniques, including the Finney procedure, and for stenosis above 25 cm, enteroenterostomy [31].

Currently, many IBD patients remain in moderate remission because they are not achieving the desired effect of treatment. This may lead to the development of chronic inflammation and complications [28].

Physical activity

Studies conducted on the effects of physical activity in IBD patients have shown improved physical performance, increased bone mineral density, reduced stress and anxiety. It is recommended that patients with mild to moderate IBD engage in physical activity for at least 30 minutes a day three times a week [32].

It has been observed that physical activity has a protective effect against the onset of IBD, and this association is more relevant to CD than UC. Patients with inflammatory bowel disease are stressed, are more prone to fatigue, and have an increased risk of developing osteoporosis or ankylosing spondylitis. Physical therapy improves flexibility and mobility in the joints. Based on the study by Robinson et al, it can be concluded that exercise not only increases bone mineral density in patients with CD, but may also reduce the risk of osteoporotic fractures. Exercise may also help reduce fatigue. Stress affecting gastrointestinal function can lead to a worsening of IBD symptoms, and physical activity can help improve the mood and general condition of patients [33]. Although there is ample evidence of the positive effects of physical activity on the body, more research is needed to establish specific recommendations for patients with IBD [32].

Conclusion

Inflammatory bowel diseases are chronic inflammatory diseases of the digestive tract. They are characterized by a relapsing course, with periods of exacerbations and remissions. Their etiopathogenesis is not fully known. The influence of environmental, genetic and immunological factors on their occurrence has been observed.They are characterized by an unfavorable prognosis and it is rarely possible to cure the patient completely.

CD occurs throughout the entire gastrointestinal tract, inflammatory changes are localized and most often affect the lower part of the gastrointestinal tract. The dominant symptoms are postprandial abdominal pain, water-mucous diarrhea, flatulence, colic, perianal lesions and aphthous stomatitis. The basic diagnostic test is ileocolonoscopy with a thorough assessment of the terminal ileum. Supportive nutritional treatment is used in the active phase of the disease. Drugs used to induce remission include steroids, immunosuppressants, biological drugs and antibiotics. Thiopurines, methotrexate or anti-TNF drugs should be the treatment of choice. Surgical treatment depends on the extent of the disease. When inflammatory changes affect only the small intestine, segmental resections or strictureplasty are used. In case of extensive lesions in the colon, colectomy with ileorectal anastomosis or proctocolectomy with creation of a permanent ileostomy is performed. Relapses after surgery are up to 70%.

UC is a chronic inflammatory process of the mucosa of the large intestine, most often located in the colon and rectum. The dominant symptoms of the disease depend on the location of the inflammatory lesions. Usually there is diarrhea with blood and mucus, a feeling of urgency to defecate and abdominal pain. It usually involves the lower abdomen and left iliac fossa. Endoscopic examination of the lower gastrointestinal tract with histological evaluation of specimens is of primary importance in the diagnosis. The treatment of choice for remission induction is aminosalicylates, followed by glucocorticoids if symptoms persist, immunosuppressive and biological drugs. In a severe attack of the disease, hospitalization of the patient is necessary, intravenous correction of water, electrolyte and albumin deficiencies and intravenous administration of glucocorticoids. In order to maintain remission, 5-ASA derivatives are administered in the first line, and if the therapeutic effect is not satisfactory, thiopurine is administered. The surgical treatment of choice is proctocolectomy with the creation of a pouch from the terminal section of the ileum and its anastomosis to the anal canal.

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Receiving funding: no funding was received.

All authors have read and agreed with the published version of the manuscript.

Disclosures: No disclosures.

Financial support: No financial support was received.

Conflict of interest: The authors declare no conflict of interest.

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