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SMALL INTESTINAL BACTERIAL OVERGROWTH - SMALL INTESTINE, BIG STRUGGLE

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SUMMARY

The gut microbiota plays an important role in the proper functioning of the human body. The microbes present in the intestines are important for the correct functioning of the digestive tract as well as other, often distant, organs of the human body. Unfavorable conditions may lead to an imbalance of intestinal microorganisms, leading to unfavorable consequences.

Under physiological conditions, the large intestine is inhabited by a significant amount of bacteria, while the small intestine contains only a small amount of them. The appearance of pathological conditions in the gastrointestinal tract may lead to the colonization of the small intestine by an excessive amount of bacteria, which, in combination with gastrointestinal symptoms, is called small intestinal bacterial overgrowth. The most common symptoms associated with this disease are abdominal pain, diarrhea, and gas.

It is difficult to diagnose this disease due to the lack of specific symptoms and imperfect diagnostic methods. The highest quality test is the microbiological test, however, due to the invasiveness of the method and the difficulties with precise collection of the material, less invasive tests are used, primarily the hydrogen breath test.

The mainstay of treatment of bacterial overgrowth is antibiotic therapy, which aims to reduce the amount of bacteria to the extent that the patient does not feel the disease. It is also

important to eat a diet rich in fiber and low in fermenting oligo-, di- and monosaccharides and polyols.

Patients with the small intestinal bacterial overgrowth rarely develop symptoms of a severe course of the disease, however, due to the unpleasant symptoms and relatively easy treatment methods, the disease should be included in the differential diagnosis in people with risk factors and present symptoms.

Key words: small intestinal bacterial overgrowth, dysbiosis, bacterial overgrowth, intestines, digestive disorders

1. INTRODUCTION AND METHODOLOGY

The intestinal microbiota is an important factor for the proper functioning of the intestines and the entire body. In the past, it was believed that the digestive tract's task is only to digest and absorb food. Now it is now known that microorganisms present in the intestines (bacteria, fungi, viruses) modulate physiological processes, such as gastrointestinal motility and secretion, or maintaining the integrity of the epithelial barrier. They also regulate the homeostasis of the whole organism by influencing metabolism, immunity and production of various chemical compounds, e.g. some neurotransmitters [1,2].

The state of the correct qualitative and quantitative structure of the intestinal microflora is called eubiosis [2]. The digestive tract is the largest reservoir of microorganisms in the human body [3]. It is estimated that even 10^{14} microorganisms inhabit it. The largest amount of them, more than 70% of their total weight, is found in the large intestine, while the small intestine, under physiological conditions, contains small amounts of bacteria [4]. Under the influence of unfavorable conditions, excessive colonization of the small intestine by bacteria may occur, which with the concomitant gastrointestinal symptoms (e.g. abdominal pain, diarrhea, flatulence) leads to the development of small intestinal bacterial overgrowth (SIBO). The bacterial balance in the small intestine is disturbed, which is referred to as dysbiosis [1,5]. Depending on the severity of bacterial growth, various clinical symptoms are manifested, often non-specific [5]. Due to the limited availability of the small intestine for examination, among the several available methods for diagnosing SIBO, there is no single diagnostic test that would allow for unambiguous confirmation of this diagnosis. Therefore, often only a good response to empirical treatment turns out to be the final confirmation of the diagnosis of SIBO [5,6,11].

The aim of this study is to present the current state of knowledge on SIBO, including epidemiology, diagnostic methods and treatment methods for this disease entity. The guidelines of gastroenterological societies and publications from 2012-2022 available in the PubMed, Google Scholar and MedRxiv scientific databases were analyzed, using keywords compliant with MeSH.

2. DESCRIPTION OF THE STATE OF KNOWLEDGE

2.1. DEFINITION AND ETIOLOGY

SIBO is a heterogeneous disorder in which excessive bacterial growth in the small intestine coexists with gastrointestinal symptoms [3,5]. The normal number of bacteria in the small intestine is usually $<10^3$ colony forming units per milliliter (CFU/ml). These are mainly bacteria from the *Lactobacillus* group, *Enterococcus*, Gram-positive aerobic bacteria and relative anaerobes [7,8]. SIBO is usually caused by colonization of the small intestine by colon-derived bacteria [8].

Due to the type of bacteria inhabiting the small intestine, two types of bacterial growth in SIBO can be distinguished:

- 1) the upper gastrointestinal tract (UAT), which is dominated by orally derived gram-negative bacteria such as *Streptococcus viridans* and *Prevotella spp.* The factors that predispose to the occurrence of UAT SIBO include slow bowel function caused by drugs, hypochlorhydria as a result of chronic use of proton pump inhibitors (PPI) or atrophic gastritis typical of the elderly.
- 2) the lower digestive tract, *coliform*, in which the dominant types of bacteria are *Escherichia coli*, *Klebsiella pneumoniae*, *Enterococcus spp.*, *Proteus mirabilis* and *Clostridium spp.* Coliform SIBO is predisposed to intestinal motility disorders and anatomical abnormalities, e.g. colonic diverticula [5].

There are mechanisms in the human body to prevent bacteria from over colonizing the small intestine. These include: low pH of gastric juice, bile, pancreatic proteolytic enzymes, secretory IgA, normal intestinal peristalsis and the ileocecal valve. As a result of the efficient operation of these mechanisms, bacteria are destroyed and effectively removed thanks to the progressive movement of the intestines. Any disturbance of the above-mentioned mechanisms, both local and systemic, may lead to the development of SIBO [5,9,10]. The etiology of the disease is usually complex [10]. It is possible to divide the factors contributing to the development of SIBO based on their type, as shown in Table 1.

Table 1. SIBO etiological factors [10,12].

Type of disorder	Example
Disorders of antimicrobial protective mechanisms	achlorhydria, pancreatic exocrine insufficiency, immunodeficiency syndromes
Anatomical abnormalities	obstruction of the small intestine, diverticula, fistulas, surgical blind loop, previous ileocecal resections
Motor disorders	irritable bowel syndrome (IBS), systemic sclerosis, diabetic autonomic neuropathy, radiation enteropathy, pseudo-obstruction of the small intestine

It is estimated that about 90% of SIBO cases are caused by intestinal motility disorders and chronic pancreatitis. In some patients, more than one factor contributes to the development of the disease, while in the elderly, SIBO may develop despite the lack of visible pathology and be the result of impaired motility of the small intestine and reduced secretion of hydrochloric acid in the stomach [7,10]. In some cases, a vicious circle may arise when SIBO develops on the basis of the underlying disease, followed by SIBO directly or indirectly worsening the course of the underlying disease [10]. Therefore, it is extremely important to correctly determine the causes of SIBO, as effective therapy of the underlying disease is important to reduce the risk of SIBO recurrence [7].

PATHOPHYSIOLOGY. Ailments caused by SIBO arise on the basis of various mechanisms. As a result of bacterial fermentation of carbohydrates, excessive amounts of gas and water are produced and the pH of the intestinal contents is reduced [5,6,11]. Bile acid deconjugation by bacteria leads to poorer absorption of fat-soluble vitamins. The bacteria compete with the host for macro- and micronutrients - as a result of their consumption by the bacteria, the host has less nutrients left to absorb. The blunting of the villi also leads to lower carbohydrate absorption. In addition, attention is drawn to the reduced production of short chain fatty acids as well as bacterial enteritis and systemic inflammation. Increased intestinal permeability is also indicated as a result of excessive bacterial colonization, however, some publications contradict this phenomenon [11].

SYMPTOMS. The dominant symptom in SIBO is abdominal distension, which is produced by excess gases produced by bacteria. Other common symptoms are abdominal pain, a feeling of fullness in the abdomen and diarrhea (fatty or watery) [6,7]. Fatty diarrhea is the result of bile salt deconjugation and thus impaired digestion of fats [7,13]. Free bile acids are irritating and stimulate the secretion of water into the lumen of the large intestine - watery diarrhea [13]. This leads to impaired absorption of fat-soluble vitamins, however, deficiency symptoms (especially vitamins A and D) appear only in severe cases of SIBO, usually with anatomical abnormalities. Moreover, also in severe form, symptoms of vitamin B12 deficiency may occur as a result of competitive metabolism of the vitamin by bacteria [6,7]. The concentration of folate may be increased due to bacterial production [7]. If methane-producing bacteria (archaea) grow in the gut, constipation develops. Increased absorption of bacterial antigens into the bloodstream and the formation of immune complexes that circulate in the body may also cause symptoms beyond the gastrointestinal tract, such as erythema nodosum, glomerulonephritis and arthritis [6,12]. Other symptoms that may occur in advanced form are: weight loss and malnutrition, hypoproteinemia and edema (intestinal protein loss syndrome), maculopapular exanthema, and inflammatory changes in the lumen of the small intestine [6,7].

The physical examination usually does not show any abnormalities. Sometimes an enlarged circumference of the abdomen and palpable distended intestinal loops are observed. In more advanced cases, symptoms of hypoproteinemia may be visible [6,7]. In patients with hepatic insufficiency, excessive decomposition of proteins and urea with the production of ammonia promotes the development of encephalopathy [6]. Laboratory tests also show abnormalities in more severe cases. They include megaloblastic anemia, deficiency of fat-

soluble vitamins and vitamin B12, and hypoalbuminemia [6,7]. In the majority of patients with SIBO, endoscopic examination and histological evaluation of small intestine specimens show no pathological changes [7].

However, none of the above-mentioned symptoms is specific for SIBO, which delays the correct diagnosis, therefore it is extremely important to take into account the risk factors of SIBO and previous attempts to treat comorbidities manifested by similar symptoms [7]. The examination should also exclude alarm symptoms, such as gastrointestinal bleeding, weight loss, low-grade fever or weakness [5].

2.2. EPIDEMIOLOGY

Due to diagnostic difficulties and non-specific symptoms, the incidence of SIBO is not exactly known. It is estimated that in the adult population it may be 2.5-22%. Little data provides information on the prevalence of SIBO in children [12]. In the publication of Avelar Rodriguez et al, a noteworthy compilation has been made, which presents the results of studies of the incidence of SIBO in children with different clinical contexts and different risk factors. The authors pay attention to the limitations of the described data due to the small number of available studies, the lack of appropriate controls in some studies, as well as the varied research methodology and the diagnostic threshold values used [11]. Table 2 lists the studies with the largest group of subjects (selected range: min. 99 subjects).

Table 2. Study characteristics and reported SIBO prevalence in children with a wide variety of clinical contexts and risk factors [11].

References	Year	Study population	Study design	Sample size (n)	Diagnostic tests and criteria for positivity	Reported SIBO prevalence*
Pereira et al.	1991	Children under the age of 5 years living in a rural village in Myanmar.	Cross-sectional	Cases: 340	LHBT Positivity was defined as “a transient breath hydrogen peak at the 20, 40, or 60 min breath samples following the lactulose test meal, and distinguishable from the later colonic peak”.	27.2%
Lewindon et al.	1998	Children with cystic fibrosis and non-cystic	Cross-sectional	Cases: 19 Controls:	LHBT Positivity was not specified.	Cases: 32% Controls: 7%

		fibrosis children (controls).		508		P: <0.003
Dos Reis et al.	2007	Children living in a slum and age and sex-matched controls aged 5 to 11 years.	Cross-sectional	Cases: 50 Controls: 50	Glucose and lactulose H ₂ breath tests. Positivity was defined as an increase in H ₂ of ≥ 20 ppm over baseline in the initial 60 min.	Lactulose: Cases: 37.5% Controls: 2.1% P: <0.001
Scarpellini et al.	2009	Children with IBS (Rome II criteria) and healthy age- and sex-matched controls.	Cross-sectional	Cases: 43 Controls: 56	Lactulose H ₂ /CH ₄ breath test. Positivity was defined as an early rise in H ₂ or CH ₄ excretion of >20 ppm within the first 90 min.	Cases: 65% Controls: 7% P: <0.00001
Lisowska et al.	2009	Children with cystic fibrosis and controls with gastrointestinal symptoms aged 5 to 17 years.	Cross-sectional	Cases: 62 Controls: 390	Glucose H ₂ /CH ₄ breath test. Positivity was defined as a fasting H ₂ or CH ₄ level of ≥ 20 ppm and ≥ 10 ppm, respectively; or an increase in H ₂ or CH ₄ over baseline during the test of ≥ 12 pm and ≥ 6 , respectively.	Cases: 37.1% Controls: 13.3% P: <0.00001
Collins et al.	2010	Children with CAP (Rome II criteria) aged 8 to 18 years and healthy controls.	Cross-sectional	Cases: 75 Controls: 40	LHBT. Positivity was defined as a rise in H ₂ >20 ppm before the first 90 min	Cases: 91% Controls: 35% P: <0.0001
Jones et al.	2011	Children with chronic diarrhoea and/or abdominal pain and/or bloating	Cross-sectional	Cases: 287	CO ₂ -corrected H ₂ and CH ₄ levels. Positivity was defined as an increase in H ₂ >10 ppm over	87%

		and/or irritability younger than 15 years of age.			baseline in the initial 45 min of the test. Patients were classified as H ₂ or CH ₄ producers if they produced >10 ppm of these gases at any time point.	
Mello et al.	2012	Children of poor socioeconomic conditions residing in a slum and children of socioeconomically advantaged families aged 6 to 10 years.	Cross-sectional	Cases: 85 Controls: 43	Lactulose H ₂ /CH ₄ breath test. Positivity was defined as an increase in H ₂ of ≥ 20 ppm or CH ₄ of ≥ 10 ppm with respect to the fasting value within the first 60 min after the ingestion of lactulose? Subjects were considered CH ₄ -producers when the concentration of CH ₄ was ≥ 3 .	Cases: 30.9% Controls: 2.4% P: 0.0007
Kortnerink et al.	2014	Children with abdominal pain-related functional gastrointestinal disorders (AP-FGID; Rome III criteria) aged 6 to 18 years.	Prospective	Cases: 161	GHBT. Positivity was defined as fasting breath H ₂ concentration ≥ 20 ppm or increase in H ₂ ≥ 12 ppm over baseline value.	14.3%
Wang et al.	2017	Children with autism spectrum disorder and age- and sex-matched healthy controls (age was not specified)	Cross-sectional	Cases: 310 Controls: 1240	Glucose H ₂ /CH ₄ breath test. Positivity was defined as an increase in H ₂ of ≥ 20 ppm or CH ₄ of ≥ 10 ppm over baseline before the first 60 min.	Cases: 31.0% Controls: 9.3% P: < 0.0001

Mello et al.	2017	Children of low socio-economic status living in an urban slum aged 5 to 11 years	Cross-sectional	Cases: 100	Lactulose H ₂ /CH ₄ breath test. Positivity was defined as an increase in H ₂ of ≥ 20 ppm or CH ₄ of ≥ 10 ppm over baseline before the first 60 min. Real-time polymerase chain reaction (results are described in aetiology section)	61.0%
Belei et al.	2017	Children with overweight or obesity aged 10 to 18 years and age- and sex-matched controls	Cross-sectional	Cases: 125 Controls: 120	GHBT Positivity was defined as an increase in H ₂ in two consecutive measurements of at least 15 ppm over baseline.	Cases: 37.6% Controls: 3.3%
Gaffar et al.	2018	Children living in a disadvantaged urban community aged 12 to 18 months.	Prospective	Cases: 194	GHBT Positivity was defined as an increase in H ₂ of ≥ 12 ppm over baseline measurement on any single post-glucose reading.	14.9%

*A few studies also evaluated the incidence of SIBO in children (indicated in parenthesis). GHBT, glucose hydrogen breath test; LHBT, lactulose hydrogen breath test; H₂, hydrogen; CH₄, methane; CO₂, carbon dioxide; ppm, parts per million.

2.3. DIAGNOSIS

The "gold standard" in the diagnosis of SIBO is the culture of duodenal aspirate during upper gastrointestinal endoscopy, however, due to the invasiveness of the examination and difficulties in aseptic sampling from the small intestine, hydrogen breath testing (HBT) with lactulose, glucose or lactose is used as an alternative method, as well as others, e.g. the test with p-aminobenzene [12].

MICROBIOLOGICAL TESTING. Quantitative and qualitative bacteriological examination of the content from the proximal jejunum or duodenum is performed. During endoscopy from the distal part of the duodenum, 3-5 ml of the content is aspirated and placed on a culture medium under aerobic and anaerobic conditions [6,7]. This is done using a catheter with side openings, which is inserted into the suction canal of the endoscope [7]. According to the American College of Gastroenterology (ACG) 2020 guidelines, the presence of $\geq 10^3$ CFU/ml of aspirate supports the diagnosis of SIBO [7]. Most often these are bacteria of the genera *Escherichia*, *Shigella*, *Aeromonas* and *Pseudomonas* [6].

The limitations of the method include the lack of standardized aseptic sampling techniques, its time consumption, cost and invasiveness, as well as the possibility of sample contamination [7,11].

BREATH TESTS. Breath tests are a relatively inexpensive, non-invasive, easy and widely available examination [13]. They are based on the principle that human cells do not produce hydrogen or methane. Therefore, the presence of these gases in the breath indicates the metabolism of undigested carbohydrates by intestinal microorganisms, whose fermentation products end up in the blood and are then excreted with the exhaled air [5,7,13].

Glucose and lactulose are the most commonly used substrates in breath testing. The patient is given 75 g of glucose or 10 g of lactulose dissolved in 250 ml of water. The concentrations of both hydrogen and methane are then measured in the exhaust air. To recognize SIBO, a hydrogen concentration increase of ≥ 20 ppm over baseline over 90 minutes is required. In the case of methane, a concentration of 10 ppm at any time point in the test indicates colonization with methanogens (methane-producing microorganisms). Methanogens are not the bacteria to which the name SIBO refers, but are archaea. Therefore, ACG experts suggest using the term "intestinal methanogen overgrowth" (IMO) instead of SIBO [5,7,13]. IMO may concern only the small intestine, only the large intestine and both intestines simultaneously [5,13].

Lactulose breath tests have two measuring peaks:

- 1) initial - associated with the growth of bacteria in the small intestine - necessary for the diagnosis of SIBO (must occur up to 90 minutes from the administration of the substrate),
- 2) resulting from bacterial fermentation in the colon - it is not necessary for the diagnosis of SIBO [7,13].

In order to avoid false-negative results, proper preparation of the patient is essential [5,7]. Antibiotics should not be taken 4 weeks before the examination, and prokinetic and laxative drugs should not be taken at least 1 week before the examination (according to Jabłkowski et al., Prokinetic drugs should be discontinued 4 weeks before the examination [5]) [7, 13]. In the case of long-term use of probiotics or an episode of infectious diarrhea, one should wait 2 weeks [5]. Proton pump inhibitors should be discontinued one week prior to the test. On the eve of the test, one should not eat fermentable foods (e.g. complex carbohydrates), maintain an 8-12 hour fast, avoid smoking and limit physical exertion [7,13].

The sensitivity of these tests is 31-68% for lactulose and 20-93% for glucose, and the specificity is 44-100% and 30-86% respectively. The high percentage of false-positive results for the lactulose test is most often due to accelerated passage and fermentation in the large intestine. The glucose breath test has low sensitivity in detecting bacterial growth in the distal

small intestine due to the absorption of glucose in the proximal part of the duodenum. In diabetics, lactulose or fructose should be used as a substrate, as glucose loading may cause hyperglycemia and intestinal motility disorders, which affects the test result [5,7,13].

The diagnostic compatibility of small intestine aspirates with respiratory tests is about 65%. This means that the use of one test method may not be sufficient for the definitive diagnosis of SIBO and that additional tests may be necessary, especially in patients with persistent symptoms and a high probability of SIBO [5,13].

X-RAY OF GASTROINTESTINAL TRACT. Examination with the use of a contrast agent and assessment of passage may reveal a passage impairment or an anatomical defect, e.g. diverticulum, blind loop or intestinal stricture [6].

ENDOSCOPY. In most patients, the results of endoscopic and histological examinations of small intestine specimens are normal [6].

LABORATORY TESTS. The abnormalities are usually found in the advanced form of the disease. They include: macrocytic anemia, vitamin B12 deficiency, hypoalbuminemia, and increased serum folic acid concentration [6].

There are known methods that are currently not standardly used in the diagnosis of SIBO. These are:

- 1) Test with p-aminobenzoic acid, which is based on the deconjugation of bile acids by bacteria. The p-aminobenzoic acid-cholic acid complex is cleaved in the intestine by the bacteria, then the p-aminobenzoic acid is absorbed and excreted in the urine, and its concentration can be measured. The disadvantage of the test is its low sensitivity and specificity [12].
- 2) Tests with C13 or C14-labeled substances, which are based on the phenomenon of decomposition of these substances by bacteria and the formation of carbon dioxide, the concentration of which is then measured in the exhaled air. The disadvantages of the test are carbon radioactivity and the high cost of the test [12]. This technique is currently not being used due to concerns about the safety of radiolabelled substrates [13].
- 3) A recent study assessed the role of H₂S in patients undergoing SIBO treatment, but the cut-off value for the diagnosis of SIBO using H₂S gas requires validation and determination of its usefulness [5,13].

2.4. DIFFERENTIAL DIAGNOSIS

The differential diagnosis should take into account, first of all, other possible causes of chronic diarrhea [6,9]. Diseases such as irritable bowel syndrome (IBS), celiac disease and inflammatory bowel disease (IBD) overlap to a large extent. IBS has recurrent abdominal pain associated with bowel movements that is associated with a change in the frequency or appearance of the stools. Celiac disease and SIBO have similar clinical symptoms, but celiac

disease is characterized by a positive serological test and a negative respiratory test. Both in Crohn's disease and SIBO, patchy mucositis may occur, however, in Crohn's disease, transmural inflammation, biopsy granulomas and rectal involvement may also be observed [9].

2.5. TREATMENT

The main goal of SIBO treatment is to reduce the number of bacteria in the small intestine, which alleviates symptoms [6]. Moreover, it is aimed at maintaining remission, correcting vitamin deficiencies and other nutritional deficiencies [5]. The mainstay of treatment, both in adults and in children, are antibiotics [13,14].

ANTIBIOTICS. The antibiotic is usually selected empirically [7,13,14]. The optimal choice are antibiotics that selectively modify only the microbiota of the gastrointestinal tract. The preparations with a broad spectrum of activity are used, such as rifaximin, co-trimoxazole (trimethoprim/sulfamethoxazole), metronidazole, amoxicillin with clavulanic acid, gentamicin, neomycin and ciprofloxacin [14]. Currently, the most preferred is rifaximin, non-absorbable from the gastrointestinal tract, which is also used in treat and see therapy - symptom relief confirms the diagnosis of SIBO [5]. The goal of antibiotic therapy is not to completely eradicate microorganisms, but to reduce their population to the point where the reported symptoms disappear. Due to the frequently observed relapse of SIBO after antibiotic therapy, another treatment with an antibiotic from a different group is commonly used [7].

DIET. The main dietary recommendation is to limit the consumption of fermentable ingredients and low-fiber foods, as well as polyalcohols and fermentable sweeteners such as sucralose. It is also recommended to avoid prebiotics such as inulin [7,13]. Some studies show a beneficial effect of the FODMAP diet - a diet low in fermenting oligo-, di- and monosaccharides and polyols [6,7,13]. The recommended duration of the FODMAP diet is about 4-6 weeks [5,7]. Longer use of the above-mentioned diet (> 8 weeks) is associated with the risk of changing the composition of the microbiota to an even more unfavorable one [15].

PROBIOTICS. There are no studies that would clearly show the benefits of using probiotics in SIBO therapy. While some of them demonstrated the prokinetic effects of probiotics and a reduction in hydrogen production, others point to probiotics as the cause of SIBO and lactic acidosis. Therefore, ACG experts do not recommend the routine use of probiotics in the treatment of SIBO [5,7].

GUT MICROBIOTA TRANSPLANTATION. There is no basis for transplanting the gut microbiota in SIBO therapy, so this method is not recommended. Data on its effectiveness are sparse and casuistic [7].

It is important to strive for the elimination of factors contributing to SIBO and treatment of the underlying disease, e.g. surgical correction of anatomical abnormalities, re-evaluation of

indications for the use of drugs that reduce the production of hydrochloric acid or slow down intestinal peristalsis [6].

3. CONCLUSIONS

SIBO is a disease entity that doctors of various specialties encounter on a daily basis. Despite the relatively wide dissemination in the society and the increasing amount of information about the disease itself, diagnostic problems make it difficult to effectively diagnose SIBO in patients and to precisely estimate the scale of the disease in society. In the presence of symptoms such as chronic diarrhea, abdominal pain and flatulence, SIBO should be included in the differential diagnosis, especially in patients with risk factors. New, non-invasive methods of diagnosis and improved treatment recommendations allow for more and more effective treatment of the disease and improvement of patients' quality of life.

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