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Small Intestinal Bacterial Overgrowth (SIBO): A Review of Diagnostics and Therapeutics

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ABSTRACT

Introduction and purpose. Small intestinal bacterial overgrowth (SIBO) is defined as an abnormal presence of colon-type bacteria in the small intestine ($\geq 10^5$ CFU/ml). This condition causes excessive gas, bloating, and potentially malabsorption or vitamin deficiencies. The aim of this review is to summarize the current knowledge regarding SIBO's clinical manifestation, diagnostics, and treatment.

A brief description of the state of knowledge. SIBO is associated with motor disorders, anatomical defects, and systemic diseases. Its symptoms are non-specific and overlap with irritable bowel syndrome. While jejunal aspiration with quantitative culture is the diagnostic gold standard, non-invasive breath tests measuring hydrogen, methane, and hydrogen sulfide are currently utilized to distinguish specific SIBO subtypes. Treatment relies on antibiotics for bacterial eradication, with rifaximin being the preferred antibiotic due to its efficacy and tolerability in patients. Such treatment is supplemented by short-term (4-6 weeks) dietary interventions, such as the low-FODMAP diet, to restore intestinal eubiosis by eliminating fermentable nutrients. Furthermore, probiotics have shown potential in supporting therapy as well as fecal microbiota transplantation, though scientific evidence remains limited.

Summary. Managing SIBO is a complex challenge requiring objective diagnosis and targeted antibiotic eradication. Crucially, preventing recurrence strictly depends on treating the underlying predisposing disease. While temporary dietary restrictions assist in restoring eubiosis, as well as the usage of probiotic and FMT the current quality of scientific evidence is insufficient.

Keywords: SIBO; treatment; low-FODMAP;

1. Introduction

The bacterial flora of the human digestive tract differs between its sections, in particular, these differences between the small and large intestines are visible. Due to the ease of material collection, most studies focus on the analysis of the fecal microbiota reflecting the bacterial population of the large intestine. This is mainly due to the rapid flow in the intestinal lumen, pH, oxygen availability and the secretion of bactericidal substances such as bile acids [1]. Under physiological conditions, the stomach and small intestine exhibit sparse bacterial colonization. When present, isolated cultures consist of Gram-positive aerobic or facultative anaerobic bacteria (primarily lactobacilli and enterococci), with a maximum concentration of 10^3 cells per milliliter of intestinal contents. The distal ileum serves as a transitional area between the microbiome of the duodenum and jejunum and the flora of the large intestine. In contrast to the large intestine, which is dominated by strictly anaerobic flora, aerobic species still predominate in this region. In the immediate vicinity of the ileocecal valve bacterial density increases dramatically, reaching concentrations ranging from 10^5 to 10^9 organisms/mL, with a prominent presence of strict anaerobes and Enterobacteriaceae [2].

SIBO, or small intestinal bacterial overgrowth, is a condition in which there are changes in the standard ratio of the small intestine microbiota and the number of colon-type bacteria in the titer ≥ 10^5 CFU/ml is determined [3]. Bacterial overgrowth leads to excessive gas production, resulting in bloating and abdominal pain in the umbilical cord area [4]. An excessive amount of bacteria in the small intestine can cause disorders that go beyond somatic symptoms, such as malabsorption disorders, deficiencies of fat-soluble vitamins and the presence of fatty diarrhea. These disorders result from potentially harmful interactions between bacterial metabolites and nutrients found in food [5].

2. Epidemiology and symptoms

The gastrointestinal tract of a healthy person is equipped with various defense mechanisms to prevent expansive bacterial colonization. Secretion of biliary and gastric juices regulates the growth of ingested bacteria by ensuring proper absorption of food and preventing fermentation by intestinal bacteria. Proper intestinal motility, reduces intestinal stasis therefore preventing bacterial growth. Maintaining the proper function of the ileocecal valve prevents the migration of colonic bacteria into the small intestine and the development of SIBO [6].

In most studies, SIBO was detected in 0-20% of healthy control individuals, while the prevalence of this disease among the general population is unknown. It is most often associated with motor disorders as well as anatomical defects of the small intestine, which are the most common risk factors for excessive bacterial overgrowth [7]. Other possible risk factors or co-occurring pathologies in SIBO include the following causes: postoperative - blind loop, partial gastrectomy, cholecystectomy, systemic diseases - e.g. diabetes, systemic sclerosis, malabsorption syndromes/diseases of the digestive system - irritable bowel syndrome, inflammatory bowel disease, pancreatic failure, cirrhosis, iatrogenic or as a result of immune deficiencies arising during HIV or COVID infection [1]. The most predominant causes of SIBO accounting for 80-90% of cases are: Irritable bowel syndrome, intestinal motility disorders, and chronic pancreatitis [8]. A higher incidence of SIBO has been observed among the elderly. Probably due to reduced motor skills, increased incidence of comorbidities, changes in diet and reduced immunity of the body [9].

Bacterial overgrowth of the small intestine manifests itself non-specifically, with symptoms overlapping with other gastrointestinal disorders, or it may remain clinically asymptomatic, making it difficult to correctly diagnose the patient. Symptoms associated with hypertrophy include symptoms resembling irritable bowel syndrome with abdominal pain, bloating, and diarrhea or

constipation. SIBO can lead to rare, severe manifestations including weight loss, anemia, nutritional deficiencies including and malabsorption syndromes. vitamin B12 deficiencies as a result of its direct consumption by bacteria. However, due to the synthesis of vitamin K and folic acid by bacteria, their levels may oscillate in the upper or above normal limits [9, 10].

3. Diagnosis

The mere presence of clinical symptoms is not sufficient to make a certain diagnosis and implement antibiotic therapy to eradicate excess bacteria. To this end, it is necessary to deepen diagnostics in order to exclude diseases presenting a similar clinical picture. Physical examination usually does not reveal significant abnormalities, in some patients palpation may reveal segmentally narrowed or distended intestinal loops. Similarly, endoscopic examination of the upper and lower gastrointestinal tract does not reveal pathological changes. Due to the similar manifestation of SIBO and celiac disease and the established correct architecture of intestinal villi occurring in the so-called potential celiac disease, serological tests of specific celiac antibodies should be performed [11]. The gold standard of SIBO diagnostics includes aspiration of the jejunum and quantitative culture to directly measure the number of bacteria in the small intestine. The test result is positive when $\geq 10^5$ CFU bacteria/ml of aspiration proximal part of the jejunum is detected, compared to the normal value of $\leq 10^3$ CFU/ml [12]. Despite being considered the gold standard, culture can give false positive results due to contamination of the sample with the oral microbiota, as well as possible false negative results due to the lack of detection of anaerobic bacteria after air penetration during sample collection [13]. Additionally due to difficulties in performing this test is not available in most hospitals and primary care settings [6].

Recently, in the case of suspected SIBO, sensitive, non-invasive methods have been introduced to measure gases that human cells are not able to produce on their own - hydrogen, hydrogen sulfide and methane in the exhaled air. Based on the dominance of one type of gas, 3 subtypes of SIBO can be distinguished. With the predominance of hydrogen production caused by bacteria such as Klebsiella or E.coli associated with diarrhea. Less commonly diagnosed subtype with hydrogen sulfide production presents with bloating and diarrhea caused by the growth of Vibrio and Desulfovibrio bacteria. With an increased concentration of methane, constipation presents itself as a dominant symptom, most often caused by the presence of Methanobrevibacter smithii. There are 3 types of breath tests available for diagnostics: the hydrogen breath test (HBT), which includes the

measurement of hydrogen and carbon dioxide after prior oral administration of 75 g of glucose or 10 g of lactose, the hydrogen-methane breath test (HMBT), which measures hydrogen, methane and carbon dioxide, and the TRIO SMART® test, which measures hydrogen, methane, hydrogen sulfide, and carbon dioxide. In the case of HBT or HMBT, the test result is considered positive if one of the following criteria is met: positive hydrogen: fasting hydrogen concentration ≥20 ppm or hydrogen concentration increases by ≥20 ppm within 90 minutes, methane positive: methane concentration ≥10 ppm at any time of the test [12, 14, 15]. According to a review by Romagnuolo J et al., the specificity of the glucose breath test oscillates between 45-86% whereas sensitivity of the glucose breath test is 20-93%. The lactose breath test has 17-68% sensitivity and 44-86 % specificity [8]. In order to perform the test correctly, the impact of medications and lifestyle factors on the test results should be minimized accordingly. The day before the test, it is recommended to avoid foods rich in fermenting carbohydrates and dietary fiber, while on the day of the test, chewing gum, eating sweets and smoking is prohibited. A breath test should be performed preferably in the morning after at least 8 hours of fasting. It is recommended to discontinue any medications that may affect the time of intestinal transit, prebiotics and probiotics at least 24 hours before the test. In the case of antibiotics being used by the subject, due to significant changes in the intestinal microbiota and hydrogen production, it is recommended to perform the test 4 weeks after the end of therapy [16]. Due to its nonspecific symptoms in patients without specific risk factors for SIBO both upper and lower endoscopies are performed in order to rule out atrophic gastritis and Crohn disease. If endoscopies show no pathology, other imaging studies are done to rule out causes like partial obstruction, diverticula, fistula, or other inflammatory findings. Usage of magnetic enterography increases the sensitivity of diagnosing small intestinal bowel strictures but is costly and therefore not used in routine diagnostics [8].

4. Treatment

4.1 Antibiotics

The basic line of treatment, the so-called gold standard in the case of SIBO, includes the use of antibiotics to eradicate bacterial overgrowth. In order to reduce the risk of recurrence of SIBO, one of the pillars of management is the treatment of the underlying disease predisposing to excessive bacterial overgrowth and the compensation of possible nutrient deficiencies. The choice of antibiotic used depends on the subtype SIBO associated with methane production associated with anaerobic conditions or hydrogen associated with excessive growth of Gram-

positive bacteria [17]. To the methane producers causing M-SIBO in the small intestine belong single-celled organisms of the Kingdom Archaea, predominantly Methaninobrevibacter smithii and Methanospaera stadmagnae. Bacteria found in H-SIBO include microaerophilic bacteria, such as Streptococcus and Escherichia coli, along with anaerobic bacteria, such as Lactobacillus and Bacteroides [18]. Due to the diagnostic limitations of the type of pathogen, empirical antibiotic therapy is most often used in patients with suspected SIBO. However, one should be mindful about possible complications associated with intensive antibiotic therapy - the development of antibiotic-resistant microorganisms and infections.

Antibiotics used to treat SIBO have traditionally included fluoroquinolones, co-trimoxazole and tetracyclines. Recently, the use of rifaximin has been preferred as it works against aerobic and anaerobic Gram-positive and Gram-negative bacteria. The effectiveness of rifaximin for controlling and mitigating the underlying disease remains controversial, with an overall eradication rate of around 60% for SIBO. The effect of rifaximin treatment is dose-dependent, the highest possible eradication rate can be obtained by using it at a dose of 1600 mg/24 hours for a week. Different treatment durations do not have a significant effect on the efficacy of rifaximin in eradicating SIBO. Rifaximin is generally well tolerated, and a limited number of mild side effects have been reported [19, 20]. Aside from the rifaximin line of treatment for H-SIBO include doxycycline and amoxicillin. M-SIBO in addition to rifaximin following drugs are used: neomycin, metronidazole, amoxicillin/clavulanate or ciprofloxacin and metronidazole [18].

4.2 Dietary changes

The treatment is complemented by a non-pharmacological intervention involving the use of an appropriate diet as well as probiotics and prebiotics. Due to a similar cross-section of clinical symptoms, a diet with a low FODMAP content - Fermentable Oligosaccharides, Disaccharides, Monosaccharides and Polyols is used, or a less restrictive diet with low fermentation [21]. The purpose of using these diets is to achieve re-eubiosis of the intestine and maintain remission in the patient. This is possible by eliminating foods that are a nutrient for bacteria, thus preventing the proliferation of bacteria living in the intestine [22]. Attention is paid to the appropriate duration of using a low-FODMAP diet of 4-6 weeks, because despite its beneficial short-term effect, its use for a longer period of time may cause depletion of the normal intestinal microbiota [23]. Following a low FODMAPs diet for at least a month shows a possibility to reduce SIBO

and optimize intestinal microbiota, allowing increasing efficacy of the pharmacological therapy that the patient was already being subjected to [24].

4.3 Use of probiotics

The results of the available meta-analysis indicate that probiotic therapy using species such as Bifidobacterium sp., Lactobacillus sp., as well as Saccharomyces boulardii and Bacillus clausii, carried out in the time interval from 5 days to 6 months, may be effective in adjuvant therapy in SIBO, resulting in remission of symptoms and normalization of hydrogen breath test results. Consequently, in accordance with the paradigm of evidence-based medicine (EBM), due to the insufficient quality of scientific evidence, there are currently no grounds for formulating official clinical recommendations recommending the routine use of probiotics in the treatment or prevention of SIBO [25].

4.4 Fecal microbiota transplantation

Fecal microbiota transplantation (FMT) is a promising form of treatment for gut dysbiosis related diseases as it can help to rebuild the composition and function of the microbiota by transferring fecal preparations from healthy donors. Each gram of human feces contains approximately 10^{11} bacterial cells, 10^{8} -10^{9} virus-like particles, 10^{7} colonocytes, 10^{8} archaea, 10^{6} fungi, protists, and metabolites [26]. Fecal microbiota transplantation may be utilized as a treatment of patients with constipation presenting SIBO. It causes no additional adverse effects for patients during the process. Before treatment, SIBO patients naturally harbor abnormally high levels of Streptococcus and Veillonella and lower amounts of Escherichia-Shigella in their small intestinal fluid compared to non-SIBO patients. FMT remodels the small intestine's microbiome, drastically reducing the Streptococcus and Veillonella populations while boosting Escherichia-Shigella. Without causing drastic changes to the colonic microbiota [27].

Conclusions

Due to its nonspecific clinical manifestations, small intestinal bacterial overgrowth (SIBO) presents a significant challenge both in diagnosis and treatment. Proper diagnosis is based on objective demonstration of excessive bacterial proliferation in the small intestine. Although invasive aspiration of intestinal contents combined with quantitative culture remains the gold

standard, non-invasive breath tests have been introduced in clinical practice, which additionally allow for the isolation of disease subtypes based on the assessment of the concentration of gases produced. The main focus of medical intervention remains antibiotic therapy, aimed at eradicating the excess bacteria. Due to its high safety profile, rifaximin is usually the first-line drug of choice. However, it should be emphasized that long-term therapeutic success and prevention of relapses depend almost entirely on the identification and optimal treatment of the underlying disease entity. Pharmacotherapy is often supplemented with non-pharmacological interventions, primarily the temporary implementation of a low-FODMAP diet, which is intended to promote the restoration of intestinal eubiosis.The role of probiotics, however, remains ambiguous, although some studies suggest their potential in alleviating symptoms and supporting treatment, the lack of strong scientific evidence within the evidence-based medicine prevents official recommendations for their routine use. Furthermore, in a specific group of patients with SIBO associated with constipation, the potential benefits of fecal microbiota transplantation are being considered.

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