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Small Intestinal Bacterial Overgrowth Syndrome: New Clinical Insights for Multimorbid and High-Risk Patients

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Abstract

Introduction

Small Intestinal Bacterial Overgrowth (SIBO) syndrome has emerged as a significant clinical issue associated with various gastrointestinal and systemic conditions. The syndrome is characterized by an excessive proliferation of bacteria in the small intestine, leading to symptoms such as bloating, diarrhoea, constipation, abdominal pain, and malabsorption. These symptoms contribute to a decline in patients' quality of life, malnutrition, and nutritional deficiencies. Despite its clinical relevance, SIBO remains underdiagnosed in routine practice.

Purpose of Research

This study aims to review the current understanding of the pathophysiology, clinical presentation, diagnostic approaches, and treatment options for SIBO, with a focus on improving clinical recognition and management.

Materials and Methods

A comprehensive literature review was conducted, analyzing 31 articles sourced from PubMed, Scopus, Web of Science, and ScienceDirect. The search was based on the keywords: *SIBO*, *small intestinal bacterial overgrowth syndrome*, *dysbiosis*, *diagnosis of SIBO*, and *treatments of SIBO*. Articles published between 2011 and 2024 were included, with at least 80% of the sources published after 2015 to ensure up-to-date information.

Basic Results

SIBO is associated with diverse etiological factors, including gastrointestinal motility disorders, anatomical abnormalities, and immune deficiencies. Diagnosis primarily relies on clinical history and breath tests, though these have limitations in accuracy. Antimicrobial therapy, alongside management of nutritional deficiencies and the underlying condition, are the cornerstone of treatment.

Conclusions

SIBO presents with nonspecific symptoms commonly seen in clinical practice, especially in elderly and high-risk patients. Early diagnosis and appropriate treatment can significantly improve patients' quality of life.

Keywords: SIBO; small intestinal bacterial overgrowth syndrome; dysbiosis; diagnosis of SIBO; treatments of SIBO

Introduction

Small intestinal bacterial overgrowth (SIBO) is a gastrointestinal disorder that has gained increasing attention in the pathology of gastrointestinal tract dysfunction in recent years. SIBO is characterized by the colonization of the small intestinal mucosa by bacterial species typically found in the large intestine, resulting in the development of gastrointestinal symptoms. According to a recently adopted international definition, SIBO is identified by the presence of commensal bacteria characteristic of the large intestine in the small intestine at a concentration equal to or greater than 10^5 colony-forming units per milliliter (CFU/mL). Some authors have suggested that this definition should be revised to a threshold of 10^3 CFU/mL, which may better correlate with the general population [1].

In SIBO, the colonization of the small intestine occurs by both gram-positive and gram-negative aerobic and anaerobic bacteria commonly found in the large intestine, such as *Streptococcus*, *Staphylococcus*, *Bacteroides*, and *Lactobacillus*. Among pathogens from the *Enterobacteriaceae* family, an increased prevalence of bacteria from the genera *Escherichia*, *Klebsiella*, and *Proteus* is observed [2]. Colonization of the jejunal and ileal mucosa by these bacterial species leads to the development of symptoms associated with malabsorption syndrome and bacterial fermentation, such as bloating, constipation, diarrhea, and abdominal pain. Additionally, the pathogenesis of SIBO symptoms is believed to involve local intestinal inflammation and the impact of bacterial metabolites on the intestinal mucosa [3].

Certain patient groups, particularly those with diabetes and, notably, geriatric patients, are at an increased risk of developing SIBO [4]. The role of the geriatrician should involve actively identifying symptoms suggestive of SIBO in high-risk populations and considering this condition in the differential diagnosis of gastrointestinal complaints. Early diagnosis and treatment can improve patient quality of life and prevent complications such as nutritional deficiencies.

Prevalence of Small Intestinal Bacterial Overgrowth

The prevalence of SIBO in the general population and its comorbidity with other diseases remain largely unknown. The lack of epidemiological data can be attributed to several factors. Some patients may not seek medical advice from their primary care physician, ignoring their symptoms. SIBO may also be asymptomatic or present with nonspecific symptoms. Additionally, the symptoms of SIBO can be misattributed to an underlying disease that itself contributes to the development of SIBO. Lastly, the underestimation of SIBO prevalence is often due to its omission by physicians in the differential diagnosis of gastrointestinal symptoms [5].

In the absence of robust epidemiological data from the general population, most of our knowledge about SIBO prevalence is derived from retrospective clinical studies in populations with symptoms suggestive of SIBO or in high-risk groups. The reported prevalence varies depending on the diagnostic method and study methodology used [3].

In certain patient populations, SIBO is identified with particularly high frequency. These populations include individuals with gastrointestinal disorders such as irritable bowel syndrome (IBS), short bowel syndrome, adult lactose intolerance, celiac disease, inflammatory bowel disease, functional dyspepsia, cystic fibrosis, and acute or chronic pancreatitis. An increased prevalence of SIBO has also been documented in various liver diseases, including cirrhosis, non-alcoholic fatty liver disease (NAFLD), and primary biliary cholangitis. Patients undergoing abdominal surgeries, such as colectomy, cholecystectomy, gastrectomy, or bariatric surgery, have also been reported to experience symptomatic SIBO more frequently.

Other factors associated with a higher prevalence of SIBO include chronic use of proton pump inhibitors (PPIs), a low-fiber diet, regular alcohol consumption, smoking, and *Helicobacter pylori* infection. Notably, there are reports of significantly higher SIBO prevalence in diabetic patients with type 1 and type 2 diabetes, as well as those with hypothyroidism. Evidence also suggests an increased prevalence of SIBO in several neurological, rheumatological, and cardiovascular diseases, as well as in patients with chronic kidney disease, depression, obesity, and hyperlipidemia [4,6,7,8]. Further research is needed to elucidate the role of SIBO in these conditions.

It is also important to emphasize the higher prevalence of SIBO in bedridden patients and those in severe clinical condition, where impaired intestinal motility and blood flow contribute to bacterial colonization of the small intestine [9]. The aging process may further contribute to the

increased occurrence of SIBO due to slowed intestinal motility and alterations in the composition of the gut microbiota [10].

Risk Factors for Small Intestinal Bacterial Overgrowth

The majority of risk factors for SIBO facilitate the translocation of bacteria from the colon to the small intestine and their subsequent colonization of the mucosal surface. These factors primarily include intestinal motility disorders, hypochlorhydria, immune deficiencies, abdominal surgeries, chronic inflammatory diseases of the gastrointestinal tract (mainly inflammatory bowel diseases), exocrine pancreatic insufficiency, and combinations of these factors [3,5].

Normal intestinal motility, particularly the migrating motor complex (MMC), prevents the stagnation of intestinal contents, thereby limiting the colonization of the upper gastrointestinal tract by commensal bacteria from the colon. Various diseases can disrupt or inhibit normal intestinal motility. One of the best examples is autonomic neuropathy in patients with type 1 or type 2 diabetes or systemic sclerosis. Patients with long-standing diabetes are particularly susceptible, and SIBO should be considered a potential cause of gastrointestinal symptoms reported during primary care visits [11]. Other examples of motility disorders include small intestinal diverticulosis and mechanical or paralytic bowel obstruction. Table 1 summarizes the major pathophysiological mechanisms contributing to the development of SIBO in various gastrointestinal conditions.

Table 1. Pathophysiological Mechanisms Contributing to SIBO Development in Various Gastrointestinal Conditions. Based on: [3,5,11,12,13,14,15,16]

Mechanism	Description	Impact on SIBO Development
Intestinal Motility Disorders	Disruptions in the normal peristaltic movement of the intestines, particularly the migrating motor complex (MMC).	Allows stagnation of intestinal contents, leading to bacterial overgrowth.
Autonomic Neuropathy	Affects intestinal motility, common in long-standing diabetes and systemic sclerosis.	Increases risk of bacterial translocation to the small intestine.
Small Intestinal Diverticulosis	Outpouchings of the small intestine.	Provides a niche for bacterial growth.
Mechanical or Paralytic Bowel Obstruction	Obstruction that impairs intestinal flow.	Leads to bacterial accumulation.

Hypochlorhydria	Reduced gastric acid production.	Reduces the bactericidal effect of gastric acid, increasing bacterial colonization.
Proton Pump Inhibitor (PPI) Use	Prolonged use of PPIs, especially over one year.	Increases risk of SIBO due to decreased gastric acidity.
Chronic Helicobacter pylori Infection	Reduces gastric acid secretion.	Facilitates bacterial overgrowth in the small intestine.
Immune Deficiencies	Conditions like hypogammaglobulinemia and AIDS.	Impair local immune response, allowing bacterial translocation and colonization.
Gastrointestinal Surgeries	Surgeries altering abdominal anatomy, such as colectomy, cholecystectomy, and gastrectomy.	Lead to changes in motility, bile secretion, and intestinal structure, predisposing to SIBO.
Chronic Inflammatory Diseases	Conditions like Crohn's disease and ulcerative colitis.	Inflammation alters the intestinal environment, increasing the risk of SIBO.
Chronic Pancreatitis	Pancreatic enzyme insufficiency and chronic inflammation.	Creates a multifactorial environment conducive to SIBO.
Irritable Bowel Syndrome (IBS)	Altered bowel function and motility disorders.	Exacerbates gastrointestinal symptoms due to bacterial overgrowth.
Celiac Disease	Involves intestinal motility disorders and impaired mucosal defense.	Linked to refractory celiac disease and contributes to SIBO development.

Abbreviations:

SIBO - Small Intestinal Bacterial Overgrowth; MMC - Migrating Motor Complex; PPI - Proton Pump Inhibitor; AIDS - Acquired Immunodeficiency Syndrome; IBS - Irritable Bowel Syndrome

Hypochlorhydria and prolonged use of proton pump inhibitors are associated with an increased risk of gastric and small intestinal bacterial colonization, leading to the development of SIBO. The risk is especially elevated in patients who have been on PPI therapy for more than one year [12,13]. Chronic *Helicobacter pylori* infection, which also reduces gastric acid secretion, constitutes another risk factor for SIBO development [14].

Immune deficiencies, particularly hypogammaglobulinemia and deficits in both cellular and humoral immunity, have been identified as risk factors for SIBO. The most common secondary immunodeficiency in adults leading to SIBO is AIDS. In children, less common primary immunodeficiencies, such as isolated IgA deficiency or common variable immunodeficiency, may predispose to SIBO [5]. These immune deficiencies impair the local immune response in the intestinal mucosa, which under normal conditions prevents bacterial translocation to the small intestine, inhibits bacterial adhesion to the intestinal epithelium, and limits the permeability of the intestinal barrier to bacterial metabolites and cells.

Gastrointestinal surgeries that alter the anatomy of the abdominal organs have long been recognized as predisposing factors for postoperative SIBO. Postoperative patients represent a significant high-risk group for SIBO development. Examples of surgeries that increase this risk include colectomy, cholecystectomy, and gastrectomy. In fact, many of the earliest studies on SIBO were conducted in post-gastrectomy patients [14]. The etiology of SIBO in these patients is multifactorial and includes paralytic bowel obstruction, pancreatic and intestinal secretory disorders, impaired bile secretion and circulation, intestinal ischemia, necrotic changes, strictures, and fistulas [3]. Gastrointestinal symptoms reported by patients during primary care visits following these surgeries should raise suspicion of developing SIBO.

Inflammatory changes in the gastrointestinal tract, particularly in the small and large intestines, predispose individuals to SIBO. This is a key factor in the increased prevalence of SIBO in patients with inflammatory bowel diseases, such as Crohn's disease and ulcerative colitis. The prevalence of SIBO is significantly higher in Crohn's disease compared to other inflammatory bowel diseases [15].

It is important to note that the development of SIBO often involves a combination of these risk factors. For instance, in chronic pancreatitis, SIBO risk factors include pancreatic enzyme insufficiency, intestinal motility disorders, chronic inflammation, and the effects of medications. A similar multifactorial process contributes to SIBO development in patients with irritable bowel syndrome. SIBO in these patients likely exacerbates some gastrointestinal symptoms, and the association between SIBO and IBS is one of the most well-documented causal relationships between SIBO and other diseases [16].

In recent years, SIBO has also been linked to celiac disease [3]. Several studies associate SIBO with refractory celiac disease. As in previous cases, the multifactorial influence of celiac disease on SIBO development is proposed, involving intestinal motility disorders, exocrine pancreatic insufficiency, and impaired mucosal antibacterial defenses.

Clinical Manifestations

The clinical presentation of Small Intestinal Bacterial Overgrowth can vary significantly depending on the duration and severity of the condition, as well as the underlying disease. In less advanced cases, SIBO may be asymptomatic in some patients, while in others it may manifest as nonspecific gastrointestinal symptoms or deficiency-related syndromes. The symptoms may be masked by or misattributed to the primary disease. The most commonly reported gastrointestinal symptoms of SIBO include bloating, abdominal pain, chronic diarrhea,

constipation, and a general sense of abdominal discomfort [7,17]. These symptoms are primarily driven by bacterial fermentation and metabolism within the small intestine, the irritative effect on the intestinal wall, and the development of localized inflammation [18].

Recurrent or prolonged SIBO often leads to symptoms associated with nutrient deficiencies and the development of deficiency syndromes. These manifestations can be diverse, affecting multiple organs and systems, overlapping with one another, and often confusing clinicians regarding their correct etiology. Common symptoms include weight loss, steatorrhea, anemia, tetany, edema, polyneuropathy, and sensory disturbances.

Anemia in SIBO has a multifactorial etiology and may present as: macrocytic anemia due to vitamin B12 deficiency, microcytic anemia resulting from iron deficiency, or normocytic anemia due to chronic inflammation of the small intestine. Steatorrhea results from disrupted enterohepatic circulation of bile acids, impaired fat absorption in the intestine, and bacterial conversion of fats into secondary metabolites. Peripheral edema has a complex etiology, often resulting from overlapping factors such as anemia, hypoproteinemia, and deficiencies in vitamin B12 and other nutrients [18].

Deficiencies in fat-soluble vitamins and calcium may lead to conditions such as osteomalacia, osteoporosis, visual disturbances, epithelial maturation defects, and neuropathies. It is important to note, however, that serum levels of vitamin K and folic acid are often within normal limits in SIBO patients due to their synthesis by intestinal bacteria. Iron deficiency, exacerbated by bacterial consumption, leads to chronic anemia and reduced physical exercise tolerance [5]. Furthermore, bacterial metabolites absorbed into the portal circulation undergo intensive detoxification in the liver, which over time may result in hepatic injury [19].

The symptoms described above represent only a subset of the diverse clinical manifestations observed in patients with chronic SIBO. Each patient's clinical presentation may differ depending on the duration of the disease, its severity, nutrient deficiencies, and various factors that modify its course.

Diagnosis and Diagnostic Tools

Several diagnostic tools assist clinicians in identifying Small Intestinal Bacterial Overgrowth. Table 2 presents an array of diagnostic tools currently employed in SIBO diagnosis.

Table 2. Currently employed diagnostic Tools for SIBO. Based on: [1,20,21,22,23,24,25]

Diagnostic Tool	Description	Impact on SIBO Diagnosis
Direct Quantitative Bacterial Assessment	Invasive method of bacterial assessment from a	Gold standard but rarely used due to its complexity and cost.

	small intestinal aspirate with a threshold of >10^5 CFU/mL.	
Hydrogen Breath Test	Non-invasive test measuring hydrogen or methane in exhaled air after ingestion of a sugar (lactose, glucose, sucrose, or lactulose).	Common in clinical practice but with variable sensitivity (60%-90%) and specificity (~85%).
Breath Test Interpretation Criteria	Increased hydrogen concentration by >20 ppm and double peak on hydrogen curve.	Positive result indicating SIBO, but interpretation criteria are not universally validated.
Stool Microscopy for Fat Droplets	Microscopic examination of stool for fat droplets to diagnose steatorrhea.	Useful for diagnosing malabsorption associated with SIBO.
Complete Blood Count with Differential	Blood test measuring cell counts and iron metabolism parameters.	Helps diagnose malabsorption, as megaloblastic anemia is common in SIBO.
Iron Metabolism Parameters	Test measuring iron and related parameters.	Essential for detecting anemia due to malabsorption in SIBO.
Serum Albumin and Vitamin B12 Levels	Tests measuring albumin and vitamin B12 in serum.	Used to detect malabsorption and megaloblastic anemia in SIBO.

Abbreviations:

SIBO - Small Intestinal Bacterial Overgrowth; CFU - Colony-Forming Units; ppm - Parts Per Million

However, it shall be noted that the diagnostic process typically begins with the clinical suspicion of SIBO based on the patient's symptoms and the presence of risk factors. Despite years of research, no internationally validated diagnostic tool for SIBO has been established to date [1].

In clinical studies, the gold standard for diagnosing SIBO has been the invasive method of direct quantitative bacterial assessment from a small intestinal aspirate, with a diagnostic threshold of more than 10^5 CFU/mL (colony-forming units per milliliter) of aspirate [20]. However, non-invasive breath tests, particularly the hydrogen breath test, are commonly used in clinical practice [21]. These tests measure hydrogen or a combination of hydrogen and methane in exhaled air after the oral administration of a readily absorbable sugar, typically lactose, glucose, sucrose, or lactulose. The sensitivity of breath tests ranges from 60% to 90%, with a specificity of approximately 85% [22].

The principle underlying breath tests is that bacterial fermentation of carbohydrates, primarily by anaerobic bacteria in the colon, is the sole source of hydrogen in the human body. When colonic bacteria colonize the small intestine, as in SIBO, carbohydrate fermentation in the small

intestine leads to the early and excessive production of gas. Hydrogen and other gases diffuse into the bloodstream and are exhaled through the lungs.

Interpretation criteria for breath tests are not universally validated and can sometimes be problematic [23]. Nonetheless, the most commonly accepted criterion for a positive result is an increase in hydrogen concentration in exhaled air by more than 20 parts per million (ppm) and the identification of a double peak on the hydrogen concentration curve. The first peak represents premature gas production by bacteria in the small intestine, while the second peak corresponds to the activity of colonic bacteria. Most researchers agree that the initial hydrogen rise should occur within 90 minutes of sugar ingestion [1].

The primary limitations of breath tests include the lack of international standardization and the potential for false results. Factors such as recent antibiotic use, salicylates, alcohol consumption, or smoking can affect test outcomes [23]. Additionally, conditions like delayed gastric emptying may lead to false-negative results, while rapid intestinal transit can cause false positives due to early substrate entry into the colon. It is also important to note that certain commensal bacteria associated with SIBO may not ferment the sugars used in breath tests or may not produce hydrogen, leading to inaccurate results. Absolute contraindications for these tests include hereditary fructose intolerance and reactive postprandial hypoglycemia [24].

The direct quantitative assessment of bacterial colonies from intestinal aspirates, though valuable in clinical research, is rarely used in routine practice due to its time-consuming, complex, and costly nature. Another useful diagnostic tool is stool microscopy for fat droplets, which aids in diagnosing steatorrhea.

A diagnostic panel for suspected SIBO should include a complete blood count with differential, iron metabolism parameters, and measurements of serum albumin and vitamin B12 levels [25]. These markers are crucial because megaloblastic anemia and hypoalbuminemia are among the most common manifestations of malabsorption in SIBO.

Treatment

The management of Small Intestinal Bacterial Overgrowth should be comprehensive and individualized. It is based on three key principles [20]:

- Treatment of the underlying condition leading to SIBO.
- Management of symptomatic bacterial overgrowth in the small intestine.
- Correction of associated nutritional deficiencies.

Treatment of the Underlying Condition

The primary goal of causal treatment is to eliminate the disease or disorder responsible for the development of SIBO. In cases of anatomical abnormalities, such as strictures, fistulas, or intestinal obstructions, surgical correction of the underlying defect is the most effective approach to prevent recurrence and enhance treatment outcomes.

In patients with chronic use of medications that inhibit gastric acid secretion, intestinal motility, or pancreatic secretion, it is essential, where possible, to modify the pharmacotherapy [3]. For patients with reduced small intestinal motility, prokinetic agents may offer potential benefits in preventing SIBO and alleviating its symptoms [25]. Identifying the underlying cause of SIBO is a critical step in every case. However, in routine clinical practice, determining the cause may be challenging and often requires referral to specialized clinics and further diagnostic investigations.

Management of Symptomatic Bacterial Overgrowth

In addition to addressing the underlying cause, most patients require symptomatic treatment, with antibiotic therapy being the cornerstone. The goal of antibiotic treatment is to selectively target the bacterial species responsible for SIBO, aiming to reduce, rather than eradicate, the intestinal microbiota, thereby alleviating symptoms.

Optimal antibiotic selection is possible only after obtaining small intestinal microbiota samples and performing antibiotic susceptibility testing. However, due to the complexity and costs involved, this approach is not routinely implemented in clinical practice.

Empirical antibiotic therapy is commonly used instead, with broad-spectrum antibiotics targeting both aerobic and anaerobic intestinal bacteria. There are no universally accepted guidelines regarding the choice, dosage, or duration of antibiotic treatment. The most frequently used antibiotics include ciprofloxacin, norfloxacin, metronidazole combined with cephalexin or trimethoprim-sulfamethoxazole, amoxicillin with clavulanic acid, and, more recently, rifaximin [1].

Rifaximin is considered by many authors as the first-line antibiotic for SIBO treatment. It is a non-absorbable antibiotic that acts locally on the gastrointestinal mucosa, has minimal side effects, and is associated with a lower risk of antibiotic resistance compared to other antibiotics mentioned. Monitoring the effectiveness of treatment with breath tests is recommended [26]. Since there are no controlled clinical trials defining the optimal duration of antibiotic therapy for SIBO, the decision should be made by the attending physician.

In symptomatic treatment, dietary management as an adjunct to antibiotic therapy may benefit certain patients. For those with coexisting IBS, a low-FODMAP diet—low in fermentable

oligosaccharides, disaccharides, monosaccharides, and polyols—is recommended. Short-term adherence to this diet can effectively complement traditional antibiotic therapy, but prolonged use may reduce beneficial colonic bacteria [27]. Therefore, it is advised to limit its application to a six-week period. It should be noted that dietary management is recommended primarily for patients with concurrent IBS, as there is insufficient evidence regarding the efficacy of a low-FODMAP diet in treating SIBO in patients without IBS [28,29].

The benefits of probiotics in SIBO management have not been conclusively demonstrated. Studies on their use often yield conflicting results, depending on study methodology and the bacterial strains used in probiotic formulations [28,30].

Fecal Microbiota Transplantation (FMT) has shown promising results in clinical trials for SIBO patients and may emerge as a potential alternative to antibiotic therapy in the future [31].

Correction of Nutritional Deficiencies

Every patient with SIBO should be evaluated for symptoms of malabsorption and nutritional deficiencies. Particular attention should be given to correcting protein, vitamin, and mineral deficiencies [20]. For patients with suspected deficiency-related symptoms, appropriate supplementation should be initiated for a sufficient duration, with regular monitoring to ensure correction of nutritional imbalances.

Conclusions

Small Intestinal Bacterial Overgrowth presents with a range of nonspecific symptoms frequently encountered by geriatricians and physicians of various specialties in daily clinical practice. This condition should be considered in the differential diagnosis, particularly in elderly patients and high-risk populations. With appropriate diagnosis and treatment, the quality of life for patients with SIBO can be significantly improved within a relatively short period.

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