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Small Intestinal Bacterial Overgrowth: A Primary Care Review

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Abstract

Gastrointestinal symptoms are commonly seen in the primary care setting.¹ These patient presentations can be nonspecific, leading to a broad differential diagnosis. Small intestinal bacterial overgrowth is a clinical entity that can present with many of these nonspecific gastrointestinal symptoms. The recent interest in the microbiome by those in the medical and lay communities has made this syndrome all the more relevant. This review gives the primary care provider an up-to-date understanding of the etiology, risk factors and predisposing factors, presentation, diagnostic testing, and management of small intestinal bacterial overgrowth.

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BACKGROUND

Gastrointestinal (GI) symptoms are commonly seen in the primary care setting, accounting for 15.9 million visits per year in the United States by recent estimation.¹ This statistic highlights the importance of understanding the presentation,

etiologies, and management of common GI syndromes. One of these syndromes, small intestinal bacterial overgrowth (SIBO), is a diagnosis often entertained in the primary care and gastroenterology settings. This dysbiosis syndrome is most often referred to as *SIBO* but is less frequently referred to as

blind loop or *stagnant loop syndrome*.² The syndrome was first described by Faber in 1897 when he described a case of “blind loop syndrome” in a patient with underlying intestinal strictures.³ Although the prevalence of SIBO has been difficult to determine, estimates range from 0% to 15.6% in healthy individuals, with increasing prevalence with age and medical comorbidities.⁴

Small intestinal bacterial overgrowth is often considered in the differential diagnosis owing to its nonspecific presentation. A consensus on the exact definition of SIBO has been difficult to establish, but SIBO can be broadly defined as excessive bacteria in the small intestine. More recently, the definition has been widely accepted as an increase in the number of bacteria in the small bowel to greater than 10^5 CFU/mL, with some arguing for a threshold of 10^3 CFU/mL.⁵ The clinical implications, and even the diagnostic criteria themselves, have been debated recently. This review focuses on current understanding of predisposing risk factors, clinical manifestations, diagnostic options, and, finally, clinical management in the primary care setting.

ETIOLOGY

As with many conditions, there does not seem to be a single unifying underlying etiology for SIBO. Abnormalities in anatomy, motility, pH, and immunity are all contributors to the development of dysbiosis. These allow for local proliferation of coliform bacteria or penetration of oral-type bacteria.⁶ This dysbiosis is characterized by colonic-type bacteria that ferment carbohydrates, leading to gas production.⁷ Anatomical risk factors can be intrinsic, traumatic, or iatrogenic. Intrinsic anatomical risk factors of the small intestine include obstruction, diverticula, and fistulas.⁸ Individuals with a history of abdominal surgical intervention can be at increased risk due to either intentional alteration in existing anatomy (ie, Roux-en-Y) or postoperative complications, including strictures and adhesions.⁹ These anatomical alterations can lead to dysmotility, which can independently increase the risk of SIBO.¹⁰

Primary dysmotility can be seen, but secondary dysmotility is much more common. Secondary dysmotility can be a consequence of systemic disease, irradiation, or medication

use. Underlying systemic diseases known to alter motility and associated with SIBO include Parkinson disease, systemic sclerosis, hypothyroidism, and diabetes mellitus.^{11,12} The increasing incidence of SIBO with age is also likely secondary to changes in intestinal motility. Medications, as always, are an important consideration, and narcotics are infamous for their effects on GI motility. Another class of medications that has been implicated recently is the proton pump inhibitors due to their effect on the gastric pH barrier between the upper and lower GI tracts. There has been some controversy as to their contribution, but recent evidence suggests that there is a strong association.¹³ The incidence of hypochlorhydria is also known to increase with age. This, along with changes in motility and, inevitably, polypharmacy, helps explain the increased risk of SIBO with aging.^{6,14} Outside of these classic risk factors, studies have shown a higher prevalence of SIBO in patients with cirrhosis, celiac disease, morbid obesity, pancreatitis, and, somewhat controversially, irritable bowel syndrome (IBS).^{4,15} This IBS controversy has implications in the primary care setting because functional GI disorders are quite common.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Often, SIBO is entertained in the differential diagnosis due to the variety of people at risk and its nonspecific presentation. The classic presentation of SIBO is that of steatorrhea, abdominal bloating, and weight loss, but this is an infrequent presentation. More commonly, patients with SIBO report bloating, flatulence, abdominal pain, and diarrhea. In more severe cases, patients can experience malabsorption leading to weight loss and malnutrition.⁶ Patients with severe symptoms are at risk for a variety of deficiencies, most notably vitamins A, D, E, B₁₂, and iron. These deficiencies, in turn, can lead to either macrocytic or microcytic anemia, polyneuropathy, and metabolic bone disease.^{16,17} Of note, vitamin K is usually unaffected because it is a by-product of bacterial metabolism.

The nonspecific presentation makes for a broad differential diagnosis and difficulty in making a clinical diagnosis with a high degree of pretest confidence. In fact, recent studies

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