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The Clinical Relevance Of Small Intestinal Bacterial Overgrowth (SIBO) In Functional Gastrointestinal Disorders: A Narrative Review

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Abstract
Small intestinal bacterial overgrowth (SIBO) is increasingly recognized as a contributing factor in functional gastrointestinal disorders (FGIDs), particularly irritable bowel syndrome (IBS). However, its clinical significance remains controversial due to limitations in diagnostic methods and heterogeneity across studies. This narrative review summarizes current evidence regarding the relationship between SIBO and FGIDs, discusses pathophysiological mechanisms, and explores therapeutic strategies targeting small bowel dysbiosis. No original data were collected for this article.

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I. Introduction

FGIDs such as IBS and functional dyspepsia (FD) are common disorders traditionally defined by chronic gastrointestinal symptoms occurring without detectable structural abnormalities. However, recent advances suggest that disruptions in gut microbial ecosystems may contribute to symptom generation. SIBO, defined as an abnormal proliferation or altered composition of bacteria within the small intestine, has emerged as a potential mechanistic factor influencing bloating, abdominal pain, diarrhea, and visceral hypersensitivity [1,2].

Several studies have demonstrated higher rates of positive breath tests in IBS patients compared with healthy controls, suggesting a possible association between SIBO and FGID symptomatology [9,10]. The extent to which SIBO represents a causal factor versus a downstream consequence of altered gut motility remains an active area of debate.

II. Methods

This narrative review included studies published between 2014 and 2024, identified through PubMed, Scopus, and Google Scholar. Key terms included “SIBO”, “small bowel dysbiosis”, “IBS”, “breath testing”, and “rifaximin”. Peer-reviewed clinical trials, observational studies, systematic reviews, and expert consensus guidelines were included [1–5].

Pathophysiology of SIBO

The small intestine normally maintains a relatively low bacterial load due to mechanisms including gastric acidity, coordinated small bowel motility, intact mucosal immunity, and normal anatomy. When these systems fail—because of hypochlorhydria, impaired migrating motor complex (MMC), strictures, diverticula, or mucosal damage—microbial overgrowth can ensue [1,7,19].

Once present, SIBO influences intestinal physiology through multiple pathways. Excess bacteria ferment dietary carbohydrates extensively, producing hydrogen, methane, and hydrogen sulfide, which increase luminal distension and contribute to bloating, pain, and altered motility [8]. Methane production in particular has been shown to slow intestinal transit and is strongly associated with constipation-predominant IBS [12,18].

Microbial byproducts such as lipopolysaccharides activate mucosal immune pathways, generating low-grade inflammation and increasing intestinal permeability [2,15]. This inflammatory environment can alter visceral sensitivity and contribute to persistent FGID symptoms.

Relationship Between SIBO and IBS

Multiple clinical studies have reported increased prevalence of SIBO among patients with IBS, particularly in diarrhea-predominant subtypes [9]. Symptomatic improvement following treatment with rifaximin further supports a possible mechanistic link [4,20]. Nonetheless, the association is not universally accepted. Variability in breath test accuracy and the absence of standardized diagnostic criteria complicate interpretation.

Moreover, IBS itself can impair motility, potentially predisposing patients to secondary SIBO rather than vice versa [16].

Despite these controversies, many researchers now consider SIBO to be a contributing factor in a subset of IBS patients, particularly those with bloating, excessive gas, and post-infectious onset [3].

Diagnostic Challenges

The diagnosis of SIBO remains problematic. Although jejunal aspirate culture is considered the gold standard, it is invasive, costly, and susceptible to contamination [1]. Breath tests such as lactulose and glucose breath testing are more widely available, yet both suffer from limited sensitivity and specificity [14].

Lactulose breath tests are prone to false positives due to rapid colonic transit, while glucose breath tests may miss distal SIBO. Furthermore, cutoff values and testing protocols vary widely across centers [14]. These limitations underscore the need for more robust diagnostic biomarkers, including molecular sequencing and metabolomic signatures [13]

Therapeutic Strategies Targeting SIBO

Antibiotics remain the primary treatment for SIBO, and rifaximin is the most widely studied agent. Clinical trials have demonstrated significant improvements in bloating, diarrhea, and breath test profiles, although recurrence rates remain high, often requiring repeated treatment cycles [4,17,20].

Probiotics represent a complementary approach aimed at restoring microbial diversity and reducing inflammation. Strains such as *Lactobacillus plantarum* and *Saccharomyces boulardii* have shown the most encouraging results, though evidence remains mixed and requires further validation [6,22].

Dietary interventions, particularly the low-FODMAP diet, reduce the availability of fermentable substrates for bacterial fermentation. Several studies have shown reductions in luminal hydrogen production and symptom burden, even in patients without confirmed SIBO [21].

In patients with underlying motility disorders, prokinetics such as prucalopride or low-dose erythromycin may reduce SIBO recurrence by enhancing MMC activity [19].

III. Discussion

SIBO likely plays a role in a subset of FGID patients, particularly those with prominent gas-related symptoms, post-infectious onset, and motility abnormalities. However, substantial uncertainties remain due to the absence of standardized diagnostic tools and the heterogeneous nature of FGIDs. The development of microbial, metabolomic, and inflammatory biomarkers may help identify patients most likely to benefit from targeted therapy. Future research should also address long-term outcomes and personalized treatment algorithms [15].

IV. Conclusion

SIBO represents an important but incompletely understood contributor to FGID pathophysiology. Although antibiotic therapy, probiotics, dietary approaches, and prokinetics show promise, diagnosis remains challenging. Advances in biomarker discovery and individualized therapeutic strategies may help clarify the true role of SIBO and improve patient outcomes.

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