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Microbiome Modulation After Bariatric Surgery: A Potential Link to Diverticulitis

Ali Mahmood, MD, FACS, FASCRS
Underwood Digestive Disease Center, Houston Methodist Sugar Land Hospital Department of Surgery, Clinical Assistant Professor, Weill – Cornell College of Medicine, 16605 Southwest Freeway Suite 430, Sugar Land, TX 77479, USA

Michael Yaakovian, MD, FACS
Houston Methodist Sugar Land Hospital, Institute of Academic Medicine Department Chief, Department of Surgery 16605 Southwest Freeway Suite 450 Sugar Land, TX 77479, USA

Aman Barkat Ali, MD, FACS
Houston Methodist Sugar Land Hospital Division of Bariatric Surgery, Department of Surgery, Houston Methodist Hospital, 16651 Southwest Freeway Suite 450, Sugar Land, TX 77479, USA

Svetang Vijay Desai
Houston Methodist Sugar Land Hospital, Division Chief, Division of Gastroenterology, Department of Medicine, 16537 Southwest Freeway, Suite 110, Sugar Land, TX 77479, USA

Aiva Mariam Mahmood
Houston Methodist Sugar Land Hospital, Department of Surgery, 16605 Southwest Freeway, Suite 430, Sugar Land, TX 77479, USA

Emaan Anya Mahmood
Houston Methodist Sugar Land Hospital, Department of Surgery, 16605 Southwest Freeway, Suite 430, Sugar Land, TX, 77479, USA

ABSTRACT

Background: Diverticulitis is an increasingly prevalent gastrointestinal condition in the United States, with rising incidence, recurrent disease burden, and significant morbidity. Obesity is a well-established risk factor for both the development and complications of diverticulitis, and emerging evidence highlights the gastrointestinal microbiome as a key mediator of intestinal inflammation. Bariatric surgery induces profound alterations in gastrointestinal anatomy, physiology, and microbial composition. However, the relationship between post-bariatric microbiome changes and diverticular disease remains poorly understood. Objective: To evaluate the impact of bariatric surgery—particularly the microbial and physiologic changes following gastric bypass—on the development, recurrence, and severity of diverticulitis. Methods: We conducted a retrospective review of all patients undergoing bariatric surgery between January 1, 2024, and January 1,

2025. Patient records were examined for prior history of diverticulitis, postoperative incidence of diverticulitis, disease severity, and need for medical or surgical intervention. Microbiome-related data were contextualized using current literature on microbial patterns associated with obesity, post-bariatric physiology, and diverticular inflammation. Results: Bariatric surgery is known to induce substantial shifts in gut microbial ecology, including increased microbial diversity, changes in bile-acid–metabolizing species, and reduced abundance of pro-inflammatory taxa. These shifts may theoretically mitigate obesity-associated inflammatory risk, yet they may also influence mucosal vulnerability within existing diverticula. Over the study period, the bariatric surgery and colorectal surgery services jointly managed 16 mutual patients. Among these, 4 patients required sigmoid resection for diverticulitis. Compared to the prior calendar year—during which only 1 mutual patient underwent sigmoid resection—this represents a four-fold increase. Conclusions: The interplay between bariatric surgery, the gastrointestinal microbiome, and diverticulitis is multifactorial and incompletely defined. While weight loss and improved metabolic health may reduce overall inflammatory risk, surgery-induced microbial shifts could modulate susceptibility to diverticular inflammation in unpredictable ways. Further prospective, microbiome-focused studies are needed to clarify these mechanisms and inform personalized risk stratification and postoperative management for bariatric patients with known or potential diverticular disease.

INTRODUCTION

Diverticulitis has emerged as an increasingly prevalent gastrointestinal disorder in the United States and now represents a significant clinical and public health burden. Recent estimates indicate a prevalence of approximately 180 cases per 100,000 individuals, translating to 500,000–600,000 cases annually and more than 2.7 million outpatient visits each year. [1] Epidemiologic trends demonstrate a steady rise in both incidence and healthcare utilization over recent decades. From the late 1990s through the first decade of the 2000s, diverticulitis incidence increased by 55%. [2] A first episode of diverticulitis also confers an elevated risk of recurrence, further contributing to long-term disease burden. Notably, the disease is increasingly diagnosed in younger adults, suggesting evolving risk factors beyond age-related structural changes. [3]

Mortality associated with complicated diverticulitis remains clinically significant. Emergent surgery for diverticular disease carries an estimated mortality rate of 7%, and the social and emotional impact on patients and families is substantial. [4] Despite advances in cross-sectional imaging, medical therapy, and minimally invasive surgery, management strategies remain variable, and recurrent or complicated disease continues to contribute to morbidity and rising healthcare expenditures. Given current trends, diverticulitis is increasingly regarded as an epidemic within Western societies, underscoring the need for a deeper understanding of its epidemiology, pathophysiology, and modifiable risk factors.

Morbid obesity (BMI ≥40 kg/m^2) has emerged as an independent risk factor not only for the development of diverticular disease but also for complications such as abscess, perforation, recurrence, and the need for surgical intervention. Simultaneously, the gastrointestinal microbiome is now recognized as a key determinant of intestinal inflammation and mucosal

integrity, with dysbiosis implicated in the progression from diverticulosis to diverticulitis. (Schieffer)

The purpose of our study was to evaluate the relationship between bariatric surgery—specifically post-gastric bypass physiology—and diverticulitis. Given the profound changes in gastrointestinal anatomy and microbial composition that follow bariatric procedures, understanding their impact on diverticular disease is increasingly important.

METHODS AND STUDY DESIGN

We conducted a retrospective review of all patients who underwent weight-reduction surgery between January 1, 2024, and January 1, 2025. These patients were subsequently followed to assess the development or progression of diverticular disease. We recorded the incidence of diverticulitis and its management, including conservative therapy and surgical intervention. Patients with a prior history of diverticulitis were included regardless of whether their episodes occurred before or after bariatric surgery.

RESULTS

Over the study period, the bariatric surgery and colorectal surgery services jointly managed 16 mutual patients. Among these, 4 patients required sigmoid resection for diverticulitis. Compared to the prior calendar year—during which only 1 mutual patient underwent sigmoid resection—this represents a four-fold increase. This rise suggests either increased recognition of complicated diverticular disease within the post-bariatric population, a true increase in disease burden, or improved cross-service coordination leading to earlier surgical referral. A visual representation of this trend is shown in the figure below.

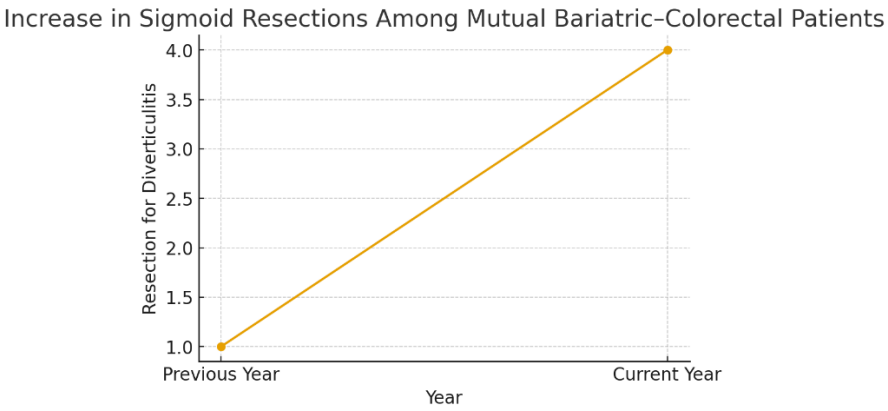

Figure 1: The graph shows the direct increasing trend of sigmoid resection following bariatric surgery.

DISCUSSION

The pathogenesis of diverticulitis is multifactorial and remains only partially understood. Proposed mechanisms include alterations in the gut microbiota, low-grade mucosal inflammation, visceral hypersensitivity, genetic susceptibility, and environmental influences such as low dietary fiber intake, obesity, and physical inactivity. The rise of diverticulitis in parallel with Western dietary patterns highlights the role of modifiable lifestyle factors in colonic health.

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Growing evidence suggests that the gastrointestinal microbiome plays a critical role in diverticular disease. Dysbiosis—characterized by altered microbial diversity and function—may contribute to chronic inflammation and mucosal vulnerability. Studies suggest that immune dysregulation may facilitate diverticulum formation and progression to diverticulitis. [5] A 2023 genomic analysis demonstrated that patients with perforated diverticulitis exhibited increased levels of sulfur-reducing and sulfur-oxidizing bacteria compared with non-diseased tissue, a pattern associated with elevated hydrogen sulfide production and heightened inflammatory responses. [6] However, other studies have failed to replicate these associations, indicating that the microbiome–diverticulitis connection remains incompletely defined. [7]

Obesity further complicates this interplay. Visceral adiposity promotes systemic low-grade inflammation, immune dysregulation, altered cytokine signaling, impaired motility, increased intraluminal pressure, and microbiome changes—all of which may predispose to diverticulitis. Obese patients often experience more complicated disease courses, including higher rates of abscess formation and perforation. Surgical management is more challenging due to technical complexity, anesthesia risks, and increased rates of postoperative complications such as wound infection and hernia. While modern strategies increasingly favor primary anastomosis with diverting stoma over Hartmann's procedure—reducing permanent stoma rates and mortality—morbidity remains substantial. [8] Large cohort studies report anastomotic leak rates of approximately 4–5%. [9]

Non-operative strategies have also evolved. Percutaneous drainage and colonic lavage have reduced the need for interval colectomy in select cases. However, the offending segment of colon remains in place, and recurrence risk persists. [10]

MICROBIOME CONSIDERATIONS

Dysbiosis may influence diverticular inflammation through several mechanisms. Overrepresentation of pro-inflammatory species such as *Escherichia coli* and *Bacteroides fragilis* can promote epithelial injury via increased reactive oxygen species, disruption of tight junctions, and activation of inflammatory signaling pathways. [11] E. Coli has been particularly isolated and identified with a marked increased risk of severe septic peritonitis. [Brown and Vashsat] Conversely, reductions in beneficial species—particularly *Faecalibacterium prausnitzii* and *Roseburia* spp.—lead to decreased short-chain fatty acid (SCFA) production, compromising epithelial barrier integrity and impairing immune regulation. [12]

Together, these microbial shifts may predispose susceptible individuals to localized inflammation, infection, and microperforation. Understanding the microbiome's role in diverticulitis offers opportunities for novel diagnostic and therapeutic strategies, including microbial profiling, dietary modulation, probiotics, and microbiota-directed therapies. Whether bariatric surgery alters this relationship remains uncertain. While weight loss improves metabolic health and may reduce diverticular risk, the profound shifts in microbial composition after surgery could theoretically influence inflammation within existing diverticula. This dynamic warrants further investigation.

CONCLUSION

As the prevalence of both obesity and diverticulitis continues to rise, understanding the mechanistic interplay between excess adiposity, the gastrointestinal microbiome, and colonic

inflammation has never been more critical. The emerging role of the gut microbiome offers a promising lens through which to reinterpret diverticulitis pathogenesis and identify new avenues for prevention and treatment. Given the significant alterations to the gastrointestinal environment following bariatric surgery, further research is needed to elucidate how postoperative microbial changes may influence diverticular disease. Improved understanding in this area may enable more personalized, proactive, and effective management strategies for this growing patient population.

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