

Esophageal candidiasis in an immunocompetent patient after laparoscopic sleeve gastrectomy: A case report

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Abstract

Esophageal candidiasis is an opportunistic infection typically observed in immunocompromised individuals, whereas laparoscopic sleeve gastrectomy is a widely performed bariatric procedure with relatively low rates of infectious complications. We report the case of a 51-year-old immunocompetent woman who developed esophageal candidiasis one year after laparoscopic sleeve gastrectomy and presented with a postprandial burning sensation and epigastric discomfort. Upper gastrointestinal endoscopy revealed diffuse white plaques along the esophageal mucosa, and histopathological examination confirmed *Candida* hyphae. The patient was treated with oral fluconazole, resulting in rapid clinical and endoscopic improvement. This case highlights the importance of considering esophageal candidiasis in the differential diagnosis of upper gastrointestinal symptoms after bariatric surgery, even in immunocompetent patients.

Keywords: esophageal candidiasis, bariatric surgery, candida infection, laparoscopic sleeve gastrectomy, postoperative complications, immunocompetent patient

Introduction

Laparoscopic sleeve gastrectomy (LSG) has emerged as one of the most commonly performed bariatric procedures worldwide because of its proven efficacy and relatively low complication rates [1]. In contrast, esophageal candidiasis is an uncommon condition that is typically observed in immunocompromised individuals [2]. Esophageal candidiasis in immunocompetent patients after bariatric surgery is rare and has been infrequently reported. We present a case of an immunocompetent patient with morbid obesity who underwent elective LSG and was subsequently diagnosed with esophageal candidiasis. This case highlights the need to consider esophageal candidiasis in patients presenting with unexplained upper gastrointestinal symptoms after bariatric surgery and contributes to the limited literature on atypical infectious complications after LSG.

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Informed Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. Patient anonymity has been preserved.

Conflict of Interest

No conflict of interest was declared by the authors.

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Case presentation

A 51-year-old woman with a history of type 2 diabetes mellitus presented with morbid obesity and a body mass index (BMI) of 35.5 kg/m². She was receiving metformin therapy and had no history of smoking. Preoperative cardiovascular, pulmonary, gastrointestinal, metabolic, and psychological assessments were unremarkable. The patient underwent elective laparoscopic sleeve gastrectomy and was discharged two days after surgery with vitamin supplementation and proton pump inhibitor therapy for 15 days.

Twelve months after surgery, she reported postprandial epigastric pain and a burning sensation. Routine laboratory investigations were otherwise within normal limits; however, inflammatory markers were elevated, with a C-reactive protein (CRP) level of 25 mg/L and an erythrocyte sedimentation rate (ESR) of 56 mm/h. After cardiac and pulmonary causes were excluded, upper gastrointestinal endoscopy was performed and revealed multiple adherent plaques consistent with esophageal candidiasis (Figure 1). Biopsy specimens were obtained from the esophageal lesions, and histopathological examination confirmed *Candida* hyphae with associated inflammatory infiltration (Figure 2). No clinical evidence of immunosuppression or endoscopic evidence of esophageal obstruction was identified, and serological testing for human immunodeficiency virus was negative. The patient was treated with oral fluconazole at 200 mg/day, resulting in marked symptomatic improvement within three days. Follow-up upper gastrointestinal endoscopy performed one week later demonstrated marked regression of the lesions after antifungal therapy (Figure 3). After completing a two-week course of antifungal therapy, the patient achieved complete clinical recovery.

Figure 1. Upper gastrointestinal endoscopy showing diffuse white plaques along the esophageal mucosa consistent with esophageal candidiasis.

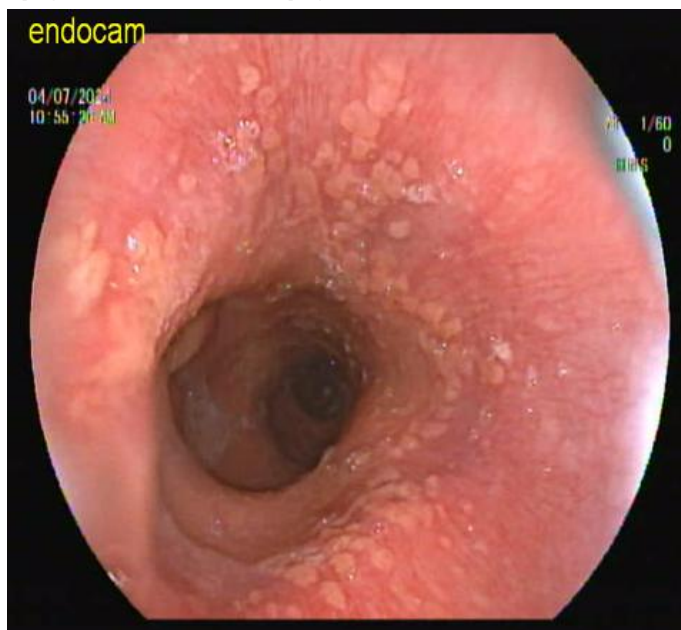


Figure 2. Histopathological examination of the esophageal biopsy showing *Candida* hyphae and inflammatory infiltration. Periodic acid–Schiff (PAS) staining, original magnification $\times 400$.

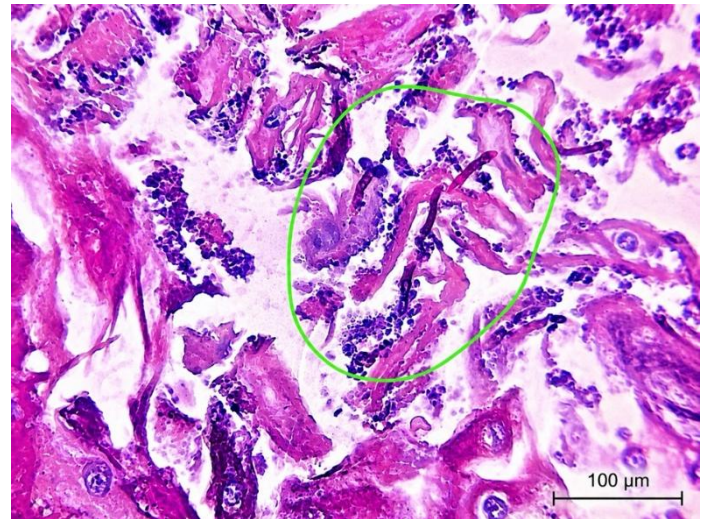


Figure 3. Follow-up upper gastrointestinal endoscopy showing marked regression of the esophageal lesions after antifungal therapy.



Discussion

Candida esophagitis is most commonly associated with immunocompromised states; however, it may also occur in immunocompetent individuals under certain predisposing conditions [2]. In the present case, the absence of classical immunosuppression highlights the importance of considering alternative risk factors, particularly in patients with a history of bariatric surgery. The pathogenesis of *Candida* esophagitis generally involves impaired host defense mechanisms or conditions that promote fungal overgrowth within the esophagus. These conditions may include esophageal stasis resulting from motility disorders or structural abnormalities, as well as alterations in the local microbial environment [3]. In this patient, no evidence of mechanical obstruction or a primary motility disorder was identified, suggesting that other contributing factors may have played a role.

Laparoscopic sleeve gastrectomy is associated with several postoperative physiological changes that may predispose patients to esophageal pathology. These include alterations in gastric anatomy, increased intragastric pressure, and a higher incidence of gastroesophageal reflux [4, 8, 9]. Changes in esophageal motility after LSG have also been reported and may contribute to impaired clearance of esophageal contents. Together,

these factors may disrupt the normal esophageal environment and facilitate fungal colonization.

Esophageal candidiasis after LSG has been reported in the literature but remains a rare postoperative complication [5]. In an outpatient series, prior gastric surgery and diabetes mellitus were among the reported predisposing factors for *Candida* esophagitis [6]. The patient's underlying type 2 diabetes mellitus may therefore have contributed to susceptibility to *Candida* infection. Diabetes has also been associated with increased gastrointestinal *Candida* colonization [7].

The present case is notable because of its delayed onset and occurrence in the absence of overt immunosuppression. The development of esophageal candidiasis one year after LSG suggests that long-term physiological changes may contribute to fungal overgrowth. The combination of postoperative anatomical and functional alterations, together with metabolic factors such as diabetes, may create an environment favorable to *Candida* colonization. This case underscores the importance of maintaining a high index of suspicion for atypical infectious etiologies in patients with persistent upper gastrointestinal symptoms after bariatric surgery. Early recognition and appropriate antifungal treatment can result in rapid clinical and endoscopic improvement, as demonstrated in this patient.

Conclusion

Esophageal candidiasis should be considered in patients who have undergone laparoscopic sleeve gastrectomy and present with unexplained upper gastrointestinal symptoms or postprandial burning sensations. Further studies are needed to clarify the pathophysiological mechanisms that may contribute to this complication.

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