Barrett’s Esophagus in a Patient with Bulimia Nervosa: A Case Report

Barrett’s Esophagus and Bulimia Nervosa

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Abstract

BACKGROUND
Barrett’s esophagus is a known complication of long-standing Gastroesophageal reflux disease, and it is a potential risk factor of developing esophageal adenocarcinoma.

CASE SUMMARY
Here, we present a case of 47 years old male patient referred to the Gastroenterology clinic for upper endoscopy because he has a long-standing history of heartburn and vomiting after meals. On examination, he has characteristic findings of self-induced vomiting as abrasions and callosities on the dorsum of the right hand and dental erosions. A detailed history revealed that he has 17 years of binge eating with self-induced vomiting. His upper endoscopy showed Gastroesophageal reflux grade D with salmon-red mucosal projections, and the biopsy revealed intestinal mucosal metaplasia.

CONCLUSION
This case emphasizes the importance of considering upper endoscopy screening for Barrett’s Esophagus in patients with eating disorders, especially those with self-induced vomiting, as in bulimia nervosa.

Key Words: Barrett’s Esophagus; Bulimia nervosa; GERD


Core Tip: Barrett’s esophagus is a known complication of long-standing gastroesophageal reflux disease. Here, we present a case of 47 years old male patient with a long-standing history of heartburn and vomiting after meals. Upper endoscopy showed gastroesophageal reflux grade D with intestinal mucosal metaplasia. This emphasizes the importance of considering upper endoscopy screening for Barrett’s
esophagus in patients with eating disorders, especially those with self-induced vomiting, as in bulimia nervosa.

**INTRODUCTION**

Barrett's esophagus is the condition in which metaplastic columnar epithelial cells with gastric and intestinal features replace the stratified squamous epithelium that normally lines the distal esophagus. The condition develops due to chronic gastroesophageal reflux disease (GERD) and is a significant risk factor for adenocarcinoma of the esophagus [1]. Bulimia nervosa is characterized by recurrent episodes of binge eating followed by inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting, misuse of medications such as laxatives, diuretics, insulin, or thyroid hormone [2].

Binge eating disorder represents a real health problem. Low treatment rates highlight the importance of questioning patients about eating problems even when not mentioned in their presenting complaints [3]. The complications that occur with bulimia nervosa can affect many organ systems and depend upon the method and frequency of purging (i.e., self-induced vomiting or misuse of laxatives, diuretics, or enemas) [4]. Gastrointestinal complications of bulimia nervosa can include GERD and Barrett’s Esophagus [5].

**CASE PRESENTATION**

*Chief complaints*

We present a case of 47 years old male patient referred for Upper endoscopy for having heartburn and vomiting after meals.

*History of present illness*

The patient denied any history of eating or psychological disorders.

*History of past illness*
Upon intense history taking, and after several attempts, the patient reported a 17-year history of having frequent heavy meals and drinking large amounts of carbonated drinks up to 10 cans every day, followed by self-induced vomiting using the index finger of the right hand. This condition confirms the diagnosis of bulimia nervosa. The patient also reported heavy smoking of Shisha.

**Personal and family history**

His body mass index was maintained throughout this period, with no significant medical history.

**Physical examination**

Upon physical examination, abrasions and callosities were noticed on the dorsum of the right hand; (Russell’s sign of self-induced vomiting, picture 1) and teeth erosions (picture 2).

**Laboratory examinations**

Routine laboratory investigations were within the accepted ranges.

**Imaging examinations**

Upper endoscopic examination showed incompetent dilated cardia. With GERD grade D (Los Angeles classification), lesions start 25 centimeters from the incisors. Salmon-red mucosal projections into the esophageal lumen and mucosal Islands. Multiple biopsies were taken, which later showed metaplastic columnar epithelium typical for Barrett’s esophagus without dysplasia (Pictures 3 and 4).

**FINAL DIAGNOSIS**

The patient was diagnosed as a case of bulimia nervosa and Barrett’s esophagus.

**TREATMENT**
Long term acid suppression was decided as a treatment for Barrett’s esophagus, in addition to the scheduling of an endoscopic surveillance program.

OUTCOME AND FOLLOW-UP

Patient was referred for psychiatric consultation.

DISCUSSION

GERD symptoms in patients with eating disorders such as bulimia nervosa are usually linked to repeated, self-induced vomiting, but the relationship is still unclear [6]. Acid exposure is not limited to purging patients; binge eating itself, which is commonly associated with various esophageal disorders, could be a risk factor for GERD [7]. Repeated acid exposure can be associated with the development of Barrett’s esophagus, whereby the esophageal squamous epithelium is replaced by metaplastic columnar epithelium, being more susceptible to malignancy [8]. Theoretically speaking, prolonged standing self-induced vomiting may be associated with the development of Barrett’s esophagus, but a lack of data makes in getting a definitive conclusion [9]. Barrett’s esophagus is associated with a 30-fold increased risk of developing esophageal adenocarcinoma over the general population [10]. Moreover, there are few case reports for bulimia nervosa presenting with worsening epigastric pain and reflux, who were finally diagnosed with esophageal adenocarcinoma [11].

In our case, there was a history of upper gastrointestinal problems, the chief presenting complaint. On the other hand, a more profound history revealed the riddle of the bulimia nervosa diagnosis that lies beneath this presenting ailment. The patient had been suffering from bulimia nervosa for 17 years without a diagnosis because of his unwillingness to consult a therapist or because of the stigma possibly associated with the disease in his imagination. The patient had a history of binge eating episodes, including increased calorie intake and compensatory purging to eliminate the extra food intake. This led to the diagnosis of bulimia nervosa induced Barrett’s esophagus in our case, which is a rare occurrence. Many cohort studies reported that patients with
Barrett's esophagus who get maintenance therapy with proton pump inhibitors had a lower probability of developing neoplastic Barrett's esophagus than those who do not receive maintenance therapy \[12\]. Diagnosing Barrett's esophagus in such cases should make a difference, considering the possibility of prescribing long-term proton pump inhibitors.

**CONCLUSION**

A thorough understanding of the risk factors for Barrett's esophagus is required to combat the rising incidence of this pre-cancerous lesion worldwide. The emerging risk factors for GERD and Barrett's esophagus must be updated considering the rising incidence of psychological eating disorders in today's world. Additionally, providers should consider endoscopic evaluation of patients with eating disorders who have persistent symptoms of dyspepsia or vomiting, given the potential risk of esophageal pre-cancerous and cancerous disorders.


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