CASE REPORT

Esophageal Bezoar in a Patient with Achalasia: Case Report and Literature Review

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Esophageal bezoars are rare, but are recognized as a distinct clinical entity. They are known to occur in patients with esophageal structural and functional abnormalities, but only a few cases of the development of esophageal bezoars in patients with esophageal motility disorders have only been described. We report a rare case of an esophageal bezoar that developed in a patient with achalasia, and review the literature concerning esophageal bezoars associated with esophageal motility disorders. (*Gut Liver* 2010;4:106-109)

Key Words: Bezoar; Esophagus; Esophageal achalasia; Esophageal motility disorders

INTRODUCTION

Bezoars may occur as complications in clinical conditions that cause stasis within the gastrointestinal tract, such as motility disorders and mechanical obstruction.¹ Esophageal bezoars are a rare but distinct clinical entity.² These bezoars are known to occur in patients with structural and functional abnormalities of the esophagus,³ but only a few patients with esophageal motility disorders have been reported to develop esophageal bezoars.⁴⁻⁸ Here, we report a rare case of esophageal bezoar in a patient with achalasia and review the literature concerning esophageal bezoars associated with esophageal motility disorders.

CASE REPORT

A 74-year-old woman was admitted to our emergency department with complaints of dysphagia and odynophagia. Odynophagia had developed 3 days ago, but she had

experienced dysphagia for the past 6 months. She reported no other gastrointestinal or systemic symptoms. She had not taken any medication. The results of physical examination and laboratory tests performed on admission were unremarkable. Esophageal endoscopy revealed a round hard yellow-whitish mass that measured approximately 4 cm and was covered with numerous roe-like particles. This mass was composed of rice fragments and obstructed the lumen of the lower esophagus. Repeated attempts were made to disrupt the bezoar by using a biopsy forceps and by washing. Eventually, the center of the mass was exposed and was found to contain linear whitish vegetable fibers (Fig. 1). The fragments produced during the disruption process were introduced into the stomach using an endoscope. After the removal of the bezoar, mucosal edema, erosions, and hyperemia were observed in the lower esophagus; these findings were attributed to the pressure effect of the mass (Fig. 1). Multiple biopsy specimens were collected from this site. The examination of these specimens revealed nonspecific findings. Therefore, we excluded inflammatory and infiltrative disorders such as eosinophilic esophagitis and esophageal malignancy.

On barium swallow esophagography, bird's beak appearance of the lower esophageal sphincter (LES) was noted, and the contrast material was found to move up and down in the esophageal body by nonpropulsive, tertiary contractions (Fig. 2). A manometric study revealed that sequentially propagated waves, which normally traverse the distal esophageal body, were absent, and that contraction waves were of low amplitude and simultaneous in onset. The lower esophageal sphincter pressure (LESP) was not elevated, but inadequate LES relaxation

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Fig. 1. Endoscopic findings. (A) An approximately 4-cm-wide round, hard, yellow-whitish mass covered with numerous roe-like particles is seen blocking the lumen of the lower esophagus. (B) Mechanical disruption of the bezoar is attempted with a biopsy forceps. (C) Long, whitish vegetable fibers are seen in the center of the mass after fragmentation of the bezoar. (D) The bezoar has been removed, and edema, erosions, and hyperemia of the lower esophageal mucosa are visible.

Fig. 2. A barium esophagogram showing a bird's beak-like narrowing at the lower esophageal sphincter.

was observed in response to wet swallows, confirming the diagnosis of achalasia (Fig. 3). The patient has been treated with medications, including calcium channel blockers and nitrates, for 9 months. She has not had any complaints.

DISCUSSION

Esophageal bezoars may occur in clinical situations that are associated with peristaltic abnormalities or mechanical obstruction of the esophagus. Thus far, 8 cases, including our case, have been reported of esophageal bezoars associated with esophageal motility disorders, namely, achalasia,^{2,4} diffuse esophageal spasm (DES),⁵ Guillain-Barre syndrome,^{6,7} myasthenia gravis,⁸ and progressive systemic sclerosis.⁹ Of these 8 cases, the patients in 7 were older than 60 years. The presenting symptoms in the above patients were variable. All 8 had an underlying peristaltic abnormality; additional abnormalities were LES incompetence⁶⁻⁸ or obstructive phenomena attributable to abnormal LES relaxation or lower esophageal stricture.^{2,5,9}

Esophageal bezoars are typically medication bezoars or phytobezoars; the present patient had a phytobezoar. Most medication bezoars tend to occur in critically ill patients receiving ventilator therapy or in bedridden patients receiving a casein-containing enteral formula or sucralfate via a nasogastric tube.² These materials easily aggregate in an acidic environment and thereby form bezoars.³ In patients with gastroesophageal reflux, food materials and acid are simultaneously introduced into the esophagus.⁸

Fig. 3. Manometric findings. (A) The lower esophageal sphincter (LES) does not sufficiently relax after a wet swallow, and the LES pressure is not reduced to the intragastric pressure (black arrow). (B) The broad pressure response following a wet swallow does not progress through the esophageal body. The contraction waves are of low amplitude and have a simultaneous onset.

Table 1. Clinical Manifestations in Patients with Esophageal Bezoars Associated with Esophageal Motility Disorders

Case	Age/ Sex	Underlying disease (duration)	Status	Feeding tube/ Medication	Bezoar type	Structural abnormality	Reference
1	79/M	GB syndrome (N.D.)	ICU Ventilator Bedridden	Yes/Sucralfate, opiate antacid	Medication bezoar	No	7
2	61/M	GB syndrome (N.D.)	ICU Ventilator Bedridden	Yes/Sucralfate, opiate	Medication bezoar	No	6
3	53/M	PSS (18 yrs)	Outpatient	No/No	Phytobezoar	Yes	9
4	68/M	MG (N.D.)	Bedridden	Yes/Pyridostigmine	Medication bezoar	No	8
5	89/F	DES (diagnosed at adm.)	Outpatient	No/No	Phytobezoar	No	5
6	89/F	Achalasia (20 yrs)	Outpatient	No/No	Phytobezoar	No	1
7	70/M	Achalasia (2 yrs)	Bedridden	No/Sucralfate	Medication/Phytobezoar	No	4
8	74/F	Achalasia (diagnosed at adm.)	Outpatient	No/No	Phytobezoar	No	Present case

N.D., not described; GB syndrome, Guillain-Barre syndrome; PSS, progressive systemic sclerosis; MG, myasthenia gravis; DES, diffuse esophageal spasm; adm, admission.

Gastroesophageal reflux frequently develops in patients with LES incompetence. Four cases of medication bezoars have been reported in patients who have esophageal motility disorders with underlying LES incompetence, such as Guillain-Barre syndrome, myasthenia gravis, and balloon-treated achalasia.

Phytobezoars develop in patients with conditions causing esophageal obstruction, such as LES dysfunction, or a lower esophageal abnormality, including achalasia, DES, and progressive systemic sclerosis with a distal esophageal stricture.^{2,5,9}

Most bezoars develop in patients with various causative factors and a longstanding esophageal motility disorder or a terminal condition and associated factors such as sucralfate administration, bedridden state, and extreme old age (Table 1). Our patient was physically well and had no associated factors, and therefore, achalasia was considered to be the only causative factor.

Endoscopy is regarded as the mainstay for the diagnosis and treatment of esophageal bezoars³ and is used to fragment and evacuate bezoars using biopsy forceps or polypectomy snares. We selected endoscopic therapy as the first-line therapeutic modality because of its safety and efficacy (Table 2).

After the removal of an esophageal bezoar, the mass obtained should be examined to determine the underlying cause. Esophageal manometry and barium esophagography are useful diagnostic modalities for evaluating underlying motility disorders.

In conclusion, careful history-taking and examination of underlying esophageal disorders should be performed in patients with esophageal bezoars. To prevent bezoar formation in patients with esophageal motility disorders, the consumption of sufficient amounts of water, the ingestion of appropriate medications, and frequent changes in position may be beneficial.

Table 2. Symptoms, Treatment Modalities, and Mechanisms of Esophageal Involvement Related to Esophageal Bezoar Formation in Patients with Esophageal Motility Disorders

Case	Symptoms	Mechanisms of esophageal involvement	Treatment
1	Difficulty passing a feeding tube	a. Abnormality of esophageal motility, LESP and gastric emptying b. GERD	Endoscopic therapy
2	Difficulty removing a feeding tube	a. Abnormality of esophageal motility, LESP and gastric emptying b. GERD	Endoscopic therapy
3	Epigastric fullness, postprandial vomiting	a. Hypoperistalsis in the esophagus b. Incompetence of the LES c. GERD	Endoscopic therapy
4	Difficulty removing a feeding tube	Peristaltic dysfunction	Vigorous lavage, endoscopic therapy: failed
5	Severe dysphagia	Simultaneous contraction in the distal third of the esophagus	Endoscopic therapy: failed
6	Nocturnal regurgitation	a. Aperistalsis of esophageal body b. LES relaxation abnormality	Endoscopic therapy
7	Sudden dysphagia	a. Aperistalsis of esophageal body b. LES relaxation abnormality	Endoscopic therapy
Present case	Dysphagia Odynophagia	a. Aperistalsis of esophageal body b. LES relaxation abnormality	Endoscopic therapy

LESP, lower esophageal sphincter pressure; GERD, gastroesophageal reflux disease; LES, lower esophageal sphincter.

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