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*Review Article*

# Upper Gastrointestinal Manifestation of Bezoars and the Etiological Factors: A Literature Review

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A gastric bezoar is a compact mass of indigestible foreign materials that accumulate and consolidate in the stomach; however, it can be found in other sites of the gastrointestinal tract. The causative manner of this condition is complex and multifactorial. The main purpose of the review was to raise awareness among clinicians, particularly gastroenterologists, that patients with certain risk factors or comorbid conditions are predisposed to gastric bezoar formation. Early diagnosis and prompt intervention are crucial to avoid bezoar-induced complications. Upper gastrointestinal endoscopy is the standard diagnostic and therapeutic method for gastric bezoars. However, for large size bezoars, surgical intervention is needed.

## 1. Introduction

Bezoars are congregations or compact masses that formed by the accumulation of matter, especially nonedible materials, including high-fiber vegetable diet, hair, and certain pharmaceutical agents. They are found more frequently in the stomach in patients with normal or abnormal gastric function or in patients with poor gastric peristalsis resulting in delayed gastric draining and other associated disorders [1, 2].

The majority of gastric bezoars are found to be present in adolescents and young ladies with a history of pica, predominantly psychiatric disorders. In contrast to adults, the majority of gastric bezoars are associated with gastroparesis, anatomical abnormalities, and former gastric surgeries that reduced gastric motility and ultimately resulting in delayed stomach emptying [1].

The most common clinical presenting symptoms in patients with gastric bezoars include nausea and vomiting, epigastric pain, dyspepsia, and weight loss [1, 3]. They can also be discovered accidentally in asymptomatic patients who undergo upper gastrointestinal (GI) endoscopic evaluation for other indications.

*1.1. Etiological Factors and Classifications.* Bezoars occur most commonly in people with certain risk factors (Table 1) [4–14] or in patients with coexisting medical disorders (Table 2) [2, 12, 14–44].

Bezoars are categorized according to the following materials that form them.

(1) Phytobezoars or diospyrobezoar: composed of indigestible fruit or vegetable content

(2) Trichobezoars: composed of hair

(3) Lactobezoars: composed of milk products

(4) Pharmacobezoars: composed of tablets and medications

Table 1: Most common risk factors associated with gastric bezoars.

| | Fibers rich diet | Milk products | Medications | Pica consumption | Mastication disorders | Insufficient fluid intake | Honeycomb consumption |
|---|---|---|---|---|---|---|---|
| Risk factors | Vegetarians | Synthetic milk | Overdose medicines | Nonnutritive constituents | Dental status | Elder people | Patients with large quantity of honeycomb ingestion for multiple health benefits |
| | Fiber-rich fruits | Feeding method | Medication for suicidal attempt | Pregnant women and small children | Abnormal mastication | Labors in hot climate | |
| | Patient with partial gastrectomy on a high-fiber diet | Premature birth | Bulk-forming agents | Patients with autism | Denture wearers | Inadequate fluid ingestion | |
| | | Failure to thrive | Extended-release medications | Patients with bariatric surgery | | Kidney disease | |
| | | Anemic children | | | | | |

Table 2: Most common comorbid conditions associated with gastric bezoars.

| | Medical disorders | Anatomic abnormalities | Gastric motility disorders |
|---|---|---|---|
| Comorbid conditions | Rapunzel syndrome<br>Anorexia nervosa & bulimia nervosa<br>Sickle cell & gastrointestinal amyloidosis<br>Diabetes mellitus & cystic fibrosis<br>Guillain–Barre syndrome & Bouveret's syndrome<br>Hypothyroidism & renal failure<br>Scleroderma & myotonic dystrophy<br>Ménétrier's disease<br>Hypochlorhydria or achlorhydria | Gastric diverticula<br>Gastric outlet obstruction<br>Pyloric stenosis<br>Cholecystogastric fistula<br>Cholecystoduodenal fistula | Gastroparesis<br>Diabetic gastroparesis<br>Idiopathic gastroparesis<br>Postsurgical gastroparesis<br>Previous gastric surgeries |

Hypothetically, the partially digested and undigested materials accompanied by gastric mucus can be a source of gastric bezoar.

## 2. Risk Factors

*2.1. High Fiber Diet.* Diets with high-fiber content (vegetables and fruits, i.e., cellulose) are more common in regions where cultures/beliefs play a key role in consumption [4]. A high-fiber diet has many benefits and is being suggested by health care institutions. Though this suggestion is appropriate for wider consumers and especially the aged population [45], the people with previous gastric surgeries should avoid high-fiber intake because they are more likely to form gastric phytobezoars. These fibers are found in fruits and vegetables including celery, pumpkin, green beans, prunes, raisins, leeks, beets, and sunflower seed shells that are merged into a mass and most often contribute to the development of gastric bezoar [3]. A specific kind of phytobezoar named a diospyrobezoar is made from unripened persimmons, coconuts, and jujubes [1, 5]. A gastric bezoar has also been reported in a patient taking vegetable-derived oil touted to contain lecithin for health purposes in lowering cholesterol levels and improving memory [46].

*2.2. Undigested Milk Products.* A gastric lactobezoar is a mass composed of a specific form of inspissated milk and mucus components [6]. This type of bezoar is commonly discovered in premature kids receiving formula diets [8]. The pathogenesis is usually complex, involving both exogenous and endogenous risk factors (i.e., synthetic milk, feeding methods, dehydration, premature birth, low birth weight, and insufficient activity and capacity of the GI tract) [6, 7]. Rarely, gastric bezoars may develop in pediatric patients with failure to thrive and iron deficiency anemia due to malnutrition [43]. Moreover, recent advances in artificial milk conformation, mother's education, and improvements in premature newborn management dramatically affected the incidence of gastric lactobezoar.

*2.3. Pharmaceutical Agents.* Pharmacobezoars are characterized by aggregations of medicines that do not properly liquefy in the GI tract and can be found in patients taking a pharmaceutical agent, tablets or somewhat liquid masses of drugs; they are usually found following an overdose of medications or in a suicidal attempt [9]. The most frequently involved medication in this entity is bulk-forming hygroscopic laxatives, e.g., perdiem and psyllium preparations, guar gum [6]. Because of the advancement of technology and time delivery-facilitated drug tablets/capsules to be slowly dissolved and gradually release active ingredients of the medication, extended-release medicines, e.g., nifedipine and verapamil, are coated with cellulose acetate; cellulose acetate may amass and lead to the progression of gastric bezoar [6]. Moreover, aluminum hydroxide gel, enteric-coated aspirin, sucralfate, cholestyramine, enteral feeding formulas, mesalamine pills, and meprobamate appear to contribute to the development of pharmacobezoars [47, 48]. Furthermore, a case by Croitoru et al. [10] reported a sodium polystyrene sulfonate gastric bezoar in a patient who mechanically ventilated after cardiopulmonary resuscitation secondary to pericarditis, primary lung cancer, and kidney failure with concomitant hyperkalemia.

*2.4. Pica Ingestion.* Pica consumption is closely linked to buildup gastric mass characterized by mainly nonnutritious materials, such as ice, pagophagia; paper, papyrophagia; drywall or paint; metal, metallophagia; stones, lithophagia; soil, geophagia; glass, hyalophagia; feces, coprophagia; and chalk. Pica consumption is most frequently found in pregnant women, small children, and those with developmental abnormalities, such as autism [11]. Children ingesting painted plaster may suffer brain damage and learning disabilities from lead poisoning. Furthermore, there is a high risk of GI obstruction or tearing in the stomach. Pica has recently been reported in patients with postbariatric surgery, who presented with pagophagia [49].

*2.5. Impaired Mastication.* Mastication is a multifactorial semiautonomic sensory motor pathway by which food content is converted into a bolus throughout the course of intraoral manipulation. Influencing factors involve dental status, active adaptation in conducting mastication during bolus formation and properties amalgamation of a bolus which may increase the possibility of GI diseases and reduce gut absorption. Mastication efficacy in denture wearers and dentate subjects is vastly different. In denture wearers, the mastication is known to be highly impaired during bolus formation. In addition to abnormal chewing behaviors and gastric motility, delay gastric emptying occurs due to large fragmented gastric bolus and consequently multiple gastric anomalies [12, 13].

*2.6. Inadequate Fluid Intake.* Fluids play a critical role in the regularity and the avoidance of GI disorders. Dietary fluid intake and renal excretion regulate total body sodium content. Inadequate fluid intake causes low blood pressure, constipation, kidney disease, electrolyte imbalance, mental changes, and dry stomach. Adequate fluids provide the source for the production of mucus in the GI tract and keep things lubricated and moistened, and thereby, the food bolus and stool can easily move through the GI tract and thus prevented GI disorders [14]. In addition, aged people and the people who work in hot climates are susceptible to dehydration and malnourishment due to age factors, economic status, and environmental factors.

*2.7. Honeycomb Ingestion.* Recently, honeycomb consumptions are widely used for various health purposes such as heart diseases, liver diseases, and metabolic disorder. However, ingesting a huge quantity of honeycomb may cause GI obstruction and life-threatening consequences. Moreover, Katsinelos et al. [14] reported a patient with irritable bowel syndrome who consumed a large quantity of honeycomb for relieving the symptoms and eventually developed a giant gastric bezoar.

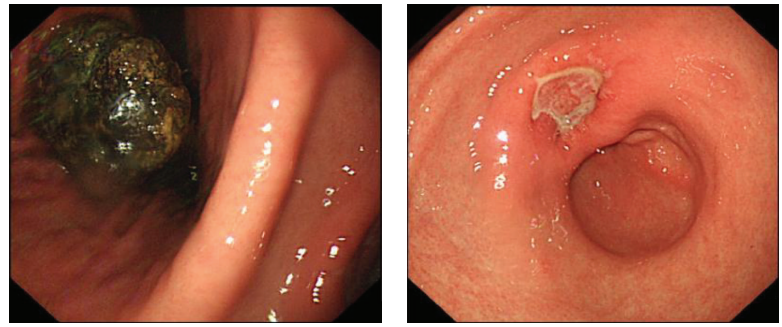
## 3. Comorbid Conditions

*3.1. Coexisting Medical Disorders*

*3.1.1. Psychiatric Disorders.* Trichobezoar commonly appears in patients with a history of Rapunzel syndrome. In this condition, patients have significant psychological or behavioral abnormalities most commonly found in females and can be associated with trichotillomania and trichotillophagia (urge to pullout one's own hair) combined with trichophagia [2, 17]. Rarely, recurrent trichobezoar may link with animals' feet stew with skin and hair intact [15]. Gastric bezoars with anorexia nervosa, bulimia nervosa [16–18], and sickle cell disease [19] have also been reported in this entity.

*3.1.2. Gastrointestinal Amyloidosis.* Amyloidosis is a condition caused by deposition of unsolvable abnormal (misfolded protein) amyloid fibrils that modify the normal function of organs and tissues [20]. The small bowel is the most common site for amyloid deposits [21]. Numerous endoscopic features of gastric amyloidosis are nonspecific. Findings include erosions, ulcerations, thickened gastric folds, friability, edema, and submucosal hematoma [50]. The delay of gastric emptying can be the result of several causes. However, amyloid light-chain amyloidosis and amyloid A amyloidosis subtype [21] can cause abnormal GI peristalsis that consequently delayed emptying of food from the stomach and leads to the formation of bezoar [20].

Certain comorbid conditions [11] such as diabetes mellitus, cystic fibrosis, Guillain–Barre syndrome, Bouveret's syndrome, hypothyroidism, renal failure, scleroderma, myotonic dystrophy, Ménétrier's disease, multiple myeloma, and hypochlorhydria or achlorhydria have been associated with a higher risk of bezoar formation. (1) Diabetes mellitus is a disorder that causes gastroparesis as a specific complication of diabetes which does not seem to raise the mortality rate. The series of gastric motor irregularities among diabetic patients like irregular distribution of gastric food, a decreased incidence of the antral element that induces antral hypomotility, antral dilatation, fasting, postprandial hypomotility, electrical dysrhythmias, reduced fundic tone, and hyperglycemia can delay gastric emptying [44]. (2) Cystic fibrosis is a hereditary condition that causes intense damage to the lungs, gastrointestinal system (malabsorption), and other organs in the body. Cystic fibrosis potentially dysfunction exocrine gland cells, including mucus-producing cells, sweat, and cells of digestive enzymes. According to Ong et al. [22], these secreted fluids of exocrine glands are generally thin and greasy. But in people with cystic fibrosis, a faulty gene cystic fibrosis transmembrane conductance regulator protein causes the secretions to become sticky, thick, and block lumens. (3) Guillain–Barre syndrome is however rarely associated with a gastric mass and characterized by an acute inflammatory demyelinating polyneuropathy, affecting the peripheral nervous system which leads to weakness and loss of tendon reflexes, dysphagia, difficulty in chewing, and loss of sphincter functions [23]. (4) Bouveret's syndrome is a very rare form of gallstone ileus caused by the passage and impaction of a large gallstone which passes into the duodenal bulb through a cholecystogastric or cholecystoduodenal fistula and ultimately blocks gastric outflow [24, 25]. Gastric-outlet-obstruction can be due to bacterial infection or gastric wall abscess after cholecystitis [26]. (5) Hypothyroidism, myxoedema or underactive thyroid, is mostly seen in women and is believed to cause gastric bezoar. It is a condition causing slowdown metabolism, GI upset, constipation, etc. [27]. (6) Renal failure is one of the leading causes of delayed gastric emptying and gastric stasis, especially in uremic patients and uremic neuropathy that are so common in these patients [28, 29]. (7) Scleroderma is a prolonged autoimmune disease that is usually associated with abnormal GI motility more commonly in patients with diffuse or limited scleroderma which causes malabsorption, weight loss, severe malnutrition, and delayed gastric emptying in the absence of a mechanical obstruction [30, 31]. (8) Myotonic dystrophy or muscular dystrophy is known to cause GI motility disorder such as edema, atrophy, and fibrosis of smooth muscles of the GI tract. The most common is the Duchenne muscular dystrophy. It is a long-term genetic disorder that affects the function muscles characterized by progressive destruction of striated muscular fibers that may often contract and/or unable to relax [32, 33]. (9-10) Rarely, intragastric bezoar may be associated with multiple myeloma [51] and Ménétrier's disease [34]. Ménétrier's disease is a rare condition characterized by gyriform or nodular enlargement of gastric mucosal folds and protein-losing hypertrophic gastroenteropathy. (11) Hypochlorhydria [14] or achlorhydria is a condition of a mild or complete absence of hydrochloric acid in gastric secretions of the stomach and other digestive organs due to dietary factors or medical interventions, respectively. This results in impaired digestion and numerous other effects on the GI tract. Moreover, hypomotility and hyposecretion are the two most significant factors in gastric bezoar formation.



(a) (b)

Figure 1: (a) Upper GI endoscopy showing a giant 7 × 5 cm diameter gastric diospyrobezoar. (b) Upper GI endoscopy showing a necrotic pressure ulcer of size 0.8 × 0.5 (white coated).

### 3.2. Anatomic Abnormalities

*3.2.1. Gastric Diverticula.* A gastric diverticulum is a rare cause of gastric bezoar when a bulk of undigested food remnant expelled from the diverticula of size (1-10 cm). It can be categorized into congenital type and acquired type. The congenital type being more common and less involved in gastric mass formation compared to acquired type is mostly found in the posterior wall of the fundus and account for about 70%. The false diverticula are usually located in the gastric antrum and greater curvature with a contextual history of chronic GI diseases, such as peptic ulcer, pancreatitis, malignancy [52], surgical management with amputation, and gastric segmental resection [35, 36].

*3.2.2. Pyloric Stenosis.* Pyloric stenosis is a tightening of the pyloric canal most frequently found in infants with a cesarean section or preterm birth [53]. The etiology of pyloric stenosis is complex, with some genetic and some environmental factors. Adults with pyloric stenosis may be due to the idiopathic hypertrophic pylorus [37] or related to underlying gastric pathology such as recurrent peptic ulcers, malignancy, and hypertrophic gastritis that weakens gastric emptying into the duodenum; as a result, all consumed foodstuff stuck in the stomach due to the pyloric obstruction and developed gastric mass [48]. Pyloric obstruction can also be a result of Bouveret's syndrome [24] and bacterial infection of the gastric wall or gastric wall abscess after cholecystitis [26]. Endoscopic submucosal dissection of the pyloric ring has also been found to be a risk factor for pyloric stenosis [38].

Rarely, gastric bezoars formed when gallstone migrated to the stomach through a cholecystogastric fistula [39] or cholecystoduodenal fistula after endoscopic retrograde cholangiopancreatography [12]. In most cases, the gallstone enters the duodenum through a cholecystoduodenal fistula followed by retrograde migration to the stomach. Small stones are generally eliminated via the stools, and stones measuring more than 2.5 cm are likely to cause obstruction [54]. The most common clinical manifestation is an acute obstruction, either at the duodenum bulb, causing pyloric obstruction, or at the ileum, causing gallstone ileus. Diabetic diathesis might be the major risk factor accountable for producing the pathologic derangement of the gallbladder and stomach and earlier history of gastroparesis, which led to the formation of bezoar and severe complications [39].

### 3.3. Gastric Dysmotility

*3.3.1. Gastroparesis.* Gastroparesis or gastric stasis is a disorder that affects gastric muscle activity, and consequently, foodstuff rests in the stomach for a prolonged time [41]. The causative factor of gastric stasis is usually unknown. However, the gastric motor defect may result from autonomic neuropathy, enteric neuropathy; defective interstitial cells of Cajal, diabetes mellitus, develop gastroparesis or idiopathic gastroparesis [40]. Moreover, postoperative gastroparesis is often caused by damage to the vagus nerve.

*3.3.2. Previous Gastric Surgeries.* The majority of gastric bezoars develop in patients with previous gastric surgeries such as Laparoscopic adjustable gastric banding [42, 43] and Roux-en-Y gastric bypass [55, 56]. Bezoars can develop months to years postoperatively. People, who undergo surgical procedures for bariatric surgery, and particularly partial gastrectomy for gastric cancer are prone to form gastric bezoars due to reduced gastric motility, loss of antral-pyloric function, hypoacidity, and rarely vagotomy that are the major causes of gastric stasis [14, 57].

## 4. Diagnostic Workup

Gastric bezoars are usually asymptomatic. They are rarely suspected by referring clinicians except in psychiatric patients. They often cause ulceration due to pressure necrosis, pyloric obstruction, peritonitis, and rarely perforation [2, 3, 58] (Figures 1(a) and 1(b)). Therefore, prompt diagnosis and early management of gastric bezoars are essential. A summary of case studies regarding gastric bezoars is presented in Table 3.

An abdominal examination has limited the efficacy in identifying gastric masses; though, sometimes on abdominal palpation intragastric mass or halitosis from the putrefying items can be found. However, these observations are not definitive and much harder to differentiate.

Upper GI series is the first step in diagnosis gastric bezoar if suspected. Appearance on CT is a mass-like filling defect with various composition-dependent characteristics.

Table 3: A summary table with case studies regarding gastric bezoars.

| Case no. A/G [ref no.] | History/previous operation | Symptoms | Clinical findings | Locations of bezoar in the stomach | Size of bezoar (cm) | Associated gastric lesions | Composition of the bezoar | Management | Complications |
|---|---|---|---|---|---|---|---|---|---|
| (1) 49/M [1] | Habitual jujubes ingestion | Epigastric pain<br>Nausea and vomiting<br>Gastric reflux | Anemic<br>Abdominal tenderness | Body | 8 × 5 cm | Necrotic ulcer | Jujubes (diospyrobezoar) | Coca-Cola Lithotripsy | None |
| (2) 18/F [2] | Trichophagia (Rapunzel syndrome) | Acute abdominal pain<br>Vomiting | Weight loss | Full-length | 120 cm | Ulcer | Hair (trichobezoar) | Laparotomy | Gastric perforation |
| (3) 47/M [3] | 6-month | Epigastric pain | Weight loss | Body | 9 × 4 cm | None | Phloem fibers<br>Raw stinging nettle (phytobezoar) | Laparotomy | None |
| (4) 76/M [4] | Arterial hypertension | Dyspepsia<br>Epigastric pain | None | Body | 10 cm | Ulcer | Vegetable fibers (phytobezoar) | Endoscopic (polypectomy snare) | None |
| (5) M [46] | None | Abdominal pain<br>Early satiety | Weight loss | Body | N/A | None | Fatty acids and lecithin (phytobezoar) | Surgical removal | None |
| (6) 96 cases [7] | Prematurity<br>Low birth weight | Abdominal distension<br>Vomiting<br>Diarrhea | Palpable abdominal mass | N/A | N/A | None | High casein content 54.2%, medium chain triglycerides 54.2%<br>Caloric density 65.6% (lactobezoars) | Cessation of oral feedings administration of intravenous fluids<br>Gastric lavage surgery | Perforations (7 patients) |
| (7) 44/F [9] | Anxiety disorder | Semiconscious<br>Fast breathing | Potassium overdose (hyperkalemia)<br>Bp-89/59 mmHg<br>Pulse 82/min, resp. 20/min | Gastric fundus | N/A | None | Extended-release potassium chloride (pharmacobezoar) | Whole bowel irrigation using polyethylene glycol (NG tube)<br>Upper GI endoscopic removal of pharmacobezoar | None |
| (8) 60/F [47] | Open cholecystectomy and choledicholithotomy | Epigastric pain<br>Vomiting | Mildly anemic<br>Dehydrated<br>Tachycardia<br>Epigastric tenderness | Pyloric canal | N/A | None | Aluminum hydroxide tablets (pharmacobezoar) | Endoscopic removal using biopsy forceps and Dormia basket | None |

Table 3: Continued.

| Case no. A/G [ref no.] | History/previous operation | Symptoms | Clinical findings | Locations of bezoar in the stomach | Size of bezoar (cm) | Associated gastric lesions | Composition of the bezoar | Management | Complications |
|---|---|---|---|---|---|---|---|---|---|
| (9) 58/M [48] | 3-month<br>Suspected Crohn's disease | Abdominal pain<br>Vomiting | Circumferential wall thickening of pylorus | Pylorus | N/A | Gastritis noncaseating epithelioid<br>Multiple hyperplastic polyps | Mesalamine pills (pharmacobezoar) | Laparoscopic gastrojejunostomy | Gastric outlet obstruction |
| (10) 54/M [10] | Primary lung cancer (metastatic)<br>Mechanically ventilated | Hyperkalemia | Constrictive effusive metastatic pericarditis<br>kidney failure | Body | $13 \times 6 \times 7$ cm | None | Sodium polystyrene sulfonate (pharmacobezoar) | Postmortem | Expired |
| (11) 7/M [11] | Pica | Abdominal pain<br>Vomiting | Abdominal tenderness<br>guarding | Full-length gastric bezoar | $13 \times 11$ cm | None | Wooden bezoar | Laparoscopic<br>Psychiatric evaluation | Gastric perforation |
| (12) 53/F [49] | Pica (anxiety, depression)<br>Roux-en-Y gastric bypass | Severe personality disorders<br>Vomiting<br>Constipation | Mild abdominal distention | N/A | $2.5 \times 1 \times 0.8$ cm | None | Cardboard and newspaper | Endoscopic removal<br>Psychiatric evaluation | None |
| (13) 69.4 ± 5.7 M/F [13] | Denture wearers<br>Impaired mastication<br>(8 male/6 female) | Not mentioned | Muscle bursts were longer = lower muscle work<br>Muscle burst decreased significantly for denture wearers<br>Longer chewing duration<br>Food boli were less disorganized | N/A | N/A | N/A | Chewing of paraffin and meat | N/A | Impaired chewing in complete denture wearers modifies the dynamics of meat bezoar formation due to large fragmented bolus |
| (14) 44/F [14] | Irritable bowel syndrome<br>Consumption of large quantities of honeycomb for health benefits | Epigastric pain<br>Nausea | None | Body | N/A | N/A | Honeycomb | Endoscopic removal<br>100 ml of hydrogen peroxide<br>Modified and conventional needle-knife<br>Snares and baskets | None |

Table 3: Continued.

| Case no. A/G [ref no.] | History/previous operation | Symptoms | Clinical findings | Locations of bezoar in the stomach | Size of bezoar (cm) | Associated gastric lesions | Composition of the bezoar | Management | Complications |
|---|---|---|---|---|---|---|---|---|---|
| (15) 69/F [12] | Cholelithiasis Choledocholithiasis | Right-sided upper abdominal pain Nausea and vomiting | Multiple biliary stones in the common bile duct | Pylorus and duodenal bulb | N/A | N/A | Gallstones and indigestible material | Proton pump inhibitor and cola drink | None |
| (16) 14/F [17] | Anorexia nervosa Thalassemia trait and growth hormone replacement. Trichotillomania | Nausea and vomiting | Weight loss Nontender, large, firm, left upper quadrant mass | Full-length (entire stomach and duodenum) | N/A | None | Hair (trichobezoar) | Laparotomy | None |
| (17) 45/F [15] | Habitual consumption of cows' feet stew with hair and skin intact. Previous history of gastric bezoar via laparotomy | Dysphagia Abdominal distension Abdominal pain Shortness of breath Generalized weakness | Microcytic anemia Malnourished CT = large gastric bezoar | Lesser curvature | 2.42 kg | Ulcer at the lesser curvature | Mass of hair Leathery skin and altered food (trichobezoar) | Laparotomy Gastrotomy | None |
| (18) 19/F [16] | Anorexia nervosa Binge-purge Hematemesis | Nausea and vomiting Constipation | Weight loss Parotid hypertrophy bilaterally | Vomited a cylindrical bezoar from the stomach | 4 cm | Possible erosions or ulcer | Debris and birefringent Foreign material Vegetable matter | Conservative treatment | N/A |
| (19) 21/F [18] | Bulimia nervosa Binge eating episodes | Abdominal pain Nausea Retching | Afebrile, normotensive with mild tachycardia Distended abdomen Weight loss | Greater curvature overlying the pylorus | 30.9 × 16.1 cm | None | Food matter | Coca-Cola Metoclopramide Endoscopic Psychotherapy | None |
| (20) 3/F [19] | Sickle cell disease | Upper abdominal pain Nonbilious emesis Anorexia | Large intra-abdominal mass epigastric tenderness Hemoglobin 9.6 g/dL Leukocyte 20.4 × $10^3/\mu$L Polymorphonuclear leukocyte 69% Platelet 254,000/$\mu$L | Stomach extended to the duodenum | 12 × 6 × 4 cm | N/A | Trichobezoar | Laparotomy Gastrotomy | None |

Table 3: Continued.

| Case no. A/G [ref no.] | History/previous operation | Symptoms | Clinical findings | Locations of bezoar in the stomach | Size of bezoar (cm) | Associated gastric lesions | Composition of the bezoar | Management | Complications |
|---|---|---|---|---|---|---|---|---|---|
| (21) 62/F [51] | Multiple myeloma | Epigastric pain<br>Vomiting<br>Weight loss<br>Fatigue | Elevated IgG of 49.2 g/L<br>Low IgM and IgA levels<br>IgG<br>Lambda paraprotein 35 g/L<br>Lambda Bence-Jones protein in the urine, elevated $\beta_2$-microglobulin 5.50 mg/mL | Body extended pylorus | N/A | Mild focal intestinal metaplasia and glandular atrophy | Phytobezoar | Coca-Cola pancreatic enzyme supplementation | Expired in 1 month |
| (22) 42/F [39] | Hypertension<br>Type 2 diabetes mellitus<br>Peripheral neuropathy<br>Gastroparesis | Nausea and vomiting<br>Abdominal pain<br>Fever | Obese, epigastric tenderness<br>Significant distress<br>Abdominal distension<br>Hypoactive bowel sounds | Antrum | 5.5 × 3.5 cm | N/A | Cholesterol gallstone induced bezoar | Laparotomy<br>Gastrotomy | None |
| (23) 34/F [42] | Laparoscopic adjustable gastric banding | Epigastric fullness<br>Nausea and vomiting | Obese<br>BMI 37 kg/m2 | In eccentric pouch dilatation | N/A | N/A | Bezoar | Liquid diet<br>Laparoscopy | Anterolateral slippage of the band |
| (24) 48/M [43] | Laparoscopic adjustable gastric banding | Dysphagia | N/A | Body | N/A | Erosions | Phytobezoar | Papain (1 week) | None |
| (25) 70/M [54] | Cholecystogastric fistula | Painful lump in the right hypochondriac region with fever and anorexia | CT revealed fistula between the gallbladder and gastric antrum. | Antrum | 9 × 5 × 5 cm | Fistulous opening in the prepyloric region | Gallstone bezoar (cholesterol and calcium oxalate) | Laparotomy | None |
| (26) 63/F [56] | Roux-en-Y gastric bypass | Abdominal distention<br>Nausea and vomiting | Morbid obese (body mass index 49.5 kg/m$^2$)<br>14 months postsurgery BMI 28 kg/m$^2$ | Gastric pouch | 5 cm | None | Persimmon Vegetables | Endoscopic Biopsy snare | None |
| (27) 65/M [58] | Chestnuts consumption | Abdominal pain | Abdominal CT indicated gastric perforation | Lesser curvature | N/A | Ulcer | Tannin Chestnut bezoars | Surgery<br>Coca-Cola | Gastric perforation |

Table 3: Continued.

| Case no. A/G [ref no.] | History/previous operation | Symptoms | Clinical findings | Locations of bezoar in the stomach | Size of bezoar (cm) | Associated gastric lesions | Composition of the bezoar | Management | Complications |
|---|---|---|---|---|---|---|---|---|---|
| (28) 73M/58F [59] | (2 cases) (1) Billroth I partial gastrectomy for gastric cancer. (2) Laparoscopic adjustable gastric banding | N/A | Cancer Obesity | Proximal gastric pouch | 10 cm 8 cm | N/A | Phytobezoar | 200 micron laser fiber and 550 micron laser fiber (Ho:YAG laser) | None |
| (29) 62/F [61] | Acute gastritis and gallstones | Epigastric pain Nausea and vomiting Hiccups Heartburn Dark loose stools | Abdominal tenderness Positive Murphy sign Hyperactive bowel sounds Pale tongue Occult blood in the vomit | Body | N/A | Gastric angle with multiple lesions Bleeding Gastric ulcers Venous aneurysm | Bezoar | Chinese medicine purgative combined with pantoprazole sodium intravenous infusion, 40 mg each time, twice a day for 5 days | None |

A/G: age/gender; M: male; F: female; NA: not available; cm: centimeter.

Trichobezoars often have a lamellated appearance. The gold standard for imaging is direct visualization with upper GI endoscopy for both diagnostic and therapeutic purposes [1, 14].

## 5. Management

Gastric bezoars can be managed either medicinally, endoscopic, or surgically. Bezoars with small size may pass via the GI tract freely on their own. In the management of gastric bezoars, there are three most common approaches which mostly focus on dissolution or eliminating bezoars. (1) Enzymatic treatment (Coca-Cola irrigations, gastroprokinetic agents, and enzymes cellulose) [4, 5, 18]. (2) Endoscopic management as the mainstream treatment includes (biopsy and alligator forceps, lithotripters, needle cutter, snares of polypectomy, and lithotripsy with Nd:YAG laser-ignited mini-explosive procedure) [4, 59]. (3) However, surgical management is the best technique for bigger ones. Recently, a laparoscopic procedure with Alexis wound retractor was effectively used in the management of bezoars [2, 4, 60]. More recently, holmium:YAG (Ho:YAG) laser lithotripsy for giant bezoar and a laparoscopic technique with endobag in the stomach to prevent bezoar spillage have shown promising results [59]. Traditional Chinese medicine purgative has also shown effectiveness in the dissolution of giant gastric bezoar and associated gastric lesions [61]. Furthermore, psychiatric treatment and dietetic instruction are suggested.

## 6. Conclusions

Gastric bezoars most frequently occur in patients with certain risk factors including psychiatric conditions, anatomic anomalies, and weakened gastric motility or in patients with coexisting medical conditions. Early diagnosis and appropriate treatment strategy are essential to prevent bezoar-induced complications. Upper GI endoscopy is a safe and effective procedure for diagnostic and therapeutic purposes of gastric bezoars. Besides, careful endoscopic surveillance should be carried out if the bezoars recur repeatedly, especially in patients with anatomical abnormalities or previous gastric surgeries. There could be a number of other contributing factors that can lead to gastric bezoar but have not yet been known to the clinicians. However, further studies are required to address this issue.

## Abbreviations

GI: Gastrointestinal
Ho:YAG: Holmium:YAG.

## Conflicts of Interest

The authors report no conflicts of interest.

## Authors' Contributions

All the names of the persons who have made substantial contributions to the work reported in the manuscript are declared in the author list. SK contributed to the paper in writing, data collection, data analysis, and manuscript preparation. KJ and LZ contributed in literature search and in the definition of intellectual content. IAK, KU, and SK contributed to the final review. XC and BMW contributed to the study concept, design, manuscript editing, and manuscript review. All authors read and approved the final manuscript.

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