

Case Report

# Intestinal Obstruction Caused by Persimmon Bezoar: A Case Report

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Owing to their rare occurrence, persimmon bezoars are often overlooked as a cause of small bowel obstruction. We herein report a small bowel obstruction in a 67-year-old Japanese female who regularly consumed persimmons in autumn. The patient presented to our hospital with typical complaints of abdominal distension with pain for 2 days. Based on the patient's history of a cesarean section 34 years ago, we initially diagnosed her with small bowel obstruction resulting from adhesions and placed an ileus tube. At first, the patient rejected the operation in spite of our recommendation. After 10 days, because the ileus tube was unable to relieve the obstruction, finally surgery was scheduled. Upon releasing the obstruction by partial resection of the small bowel, we found an impacted bezoar without any evidence of adhesions. After stone analysis, we first realized her regular persimmon intake. This case serves as an important reminder to obtain dietary history in order to investigate all possible causes of small bowel obstruction is suspected.

Key words: Persimmon – Bezoar – Intestinal obstruction

B ezoars are concretion-like solid masses formed by the compaction of stomach contents in the gastrointestinal tract. Fortunately, gastrointestinal bezoars rarely occur and have an incidence of less than 1% in the general population.<sup>1</sup> Moreover, relatively few cases have been published. In the

present case, we could not reach the correct diagnosis preoperatively because we assumed adhesions were the cause of the obstruction. Therefore, in the future, we should consider bezoar as a differential diagnosis of small bowel obstruction.

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Table 1	Laboratory	data	on	arrival
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$\begin{array}{llllllllllllllllllllllllllllllllllll$		
Hb 15.1 g/dL   Ht 43.2%   Plt 207×10 <sup>3</sup> /µL   PT 90%   PT-INR 1.05   AST 13 IU/L   ALT 9 IU/L   LDH 188 IU/L   T-Bil <sup>a</sup> 2.2 mg/dL   CK 33 U/L   Na 138 mmol/L   Cl 100 mmol/L   CRP <sup>a</sup> 2.6 mg/dL   BUN 17 mg/dL   Cr 0.50 mg/dL   TP 6.4 g/dL	WBC <sup>a</sup>	<u>9500/μL</u>
Ht $43.2\%$ Plt $207 \times 10^3 / \mu L$ PT $90\%$ PT-INR $1.05$ AST $13$ IU/LALT $9$ IU/LLDH $188$ IU/LT-Bil <sup>a</sup> $2.2$ mg/dLCK $33$ U/LNa $138$ mmol/LK <sup>a</sup> $3.3$ mmol/LCl $100$ mmol/LCRP <sup>a</sup> $2.6$ mg/dLBUN $17$ mg/dLCr $0.50$ mg/dLTP $6.4$ g/dL	RBC	4.91×10 <sup>6</sup> /μL
$\begin{array}{llllllllllllllllllllllllllllllllllll$	Hb	15.1 g/dL
PT 90%   PT-INR 1.05   AST 13 IU/L   ALT 9 IU/L   LDH 188 IU/L   T-Bil <sup>a</sup> 2.2 mg/dL   CK 33 U/L   Na 138 mmol/L   K <sup>a</sup> 3.3 mmol/L   Cl 100 mmol/L   CRP <sup>a</sup> 2.6 mg/dL   BUN 17 mg/dL   Cr 0.50 mg/dL   TP 6.4 g/dL	Ht	43.2%
PT-INR 1.05   AST 13 IU/L   ALT 9 IU/L   LDH 188 IU/L   T-Bil <sup>a</sup> 2.2 mg/dL   CK 33 U/L   Na 138 mmol/L   K <sup>a</sup> 3.3 mmol/L   Cl 100 mmol/L   CRP <sup>a</sup> 2.6 mg/dL   BUN 17 mg/dL   Cr 0.50 mg/dL   TP 6.4 g/dL	Plt	$207 \times 10^{3} / \mu L$
AST 13 IU/L   ALT 9 IU/L   LDH 188 IU/L   T-Bil <sup>a</sup> 2.2 mg/dL   CK 33 U/L   Na 138 mmol/L   K <sup>a</sup> 3.3 mmol/L   Cl 100 mmol/L   CRP <sup>a</sup> 2.6 mg/dL   BUN 17 mg/dL   Cr 0.50 mg/dL   TP 6.4 g/dL	PT	90%
ALT 9 IU/L   LDH 188 IU/L   T-Bil <sup>a</sup> 2.2 mg/dL   CK 33 U/L   Na 138 mmol/L   K <sup>a</sup> 3.3 mmol/L   Cl 100 mmol/L   CRP <sup>a</sup> 2.6 mg/dL   BUN 17 mg/dL   Cr 0.50 mg/dL   TP 6.4 g/dL	PT-INR	1.05
LDH 188 IU/L   T-Bil <sup>a</sup> 2.2 mg/dL   CK 33 U/L   Na 138 mmol/L   K <sup>a</sup> 3.3 mmol/L   Cl 100 mmol/L   CRP <sup>a</sup> 2.6 mg/dL   BUN 17 mg/dL   Cr 0.50 mg/dL   TP 6.4 g/dL	AST	13 IU/L
T-Bil <sup>a</sup> 2.2 mg/dL   CK 33 U/L   Na 138 mmol/L   K <sup>a</sup> 3.3 mmol/L   Cl 100 mmol/L   CRP <sup>a</sup> 2.6 mg/dL   BUN 17 mg/dL   Cr 0.50 mg/dL   TP 6.4 g/dL	ALT	9 IU/L
CK   33 U/L     Na   138 mmol/L     K <sup>a</sup> 3.3 mmol/L     Cl   100 mmol/L     CRP <sup>a</sup> 2.6 mg/dL     BUN   17 mg/dL     Cr   0.50 mg/dL     TP   6.4 g/dL	LDH	188 IU/L
Na   138 mmol/L     K <sup>a</sup> 3.3 mmol/L     Cl   100 mmol/L     CRP <sup>a</sup> 2.6 mg/dL     BUN   17 mg/dL     Cr   0.50 mg/dL     TP   6.4 g/dL	T-Bil <sup>a</sup>	2.2 mg/dL
K <sup>a</sup> 3.3 mmol/L     Cl   100 mmol/L     CRP <sup>a</sup> 2.6 mg/dL     BUN   17 mg/dL     Cr   0.50 mg/dL     TP   6.4 g/dL	СК	33 U/L
Cl   100 mmol/L     CRP <sup>a</sup> 2.6 mg/dL     BUN   17 mg/dL     Cr   0.50 mg/dL     TP   6.4 g/dL	Na	138 mmol/L
CRPa   2.6 mg/dL     BUN   17 mg/dL     Cr   0.50 mg/dL     TP   6.4 g/dL	K <sup>a</sup>	3.3 mmol/L
BUN   17 mg/dL     Cr   0.50 mg/dL     TP   6.4 g/dL	Cl	100 mmol/L
Cr   0.50 mg/dL     TP   6.4 g/dL	CRP <sup>a</sup>	2.6 mg/dL
TP 6.4 g/dL	BUN	17 mg/dL
8,	Cr	0.50 mg/dL
Alb 3.8 g/dL	TP	6.4 g/dL
	Alb	3.8 g/dL

WBC, white blood cells; RBC, red blood cells; Hb, hemoglobin; Ht, hematocrit; Plt, platelet; PT, prothrombin time; PT-INR, prothrombin time-international normalized ratio; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; T-Bil, total bilirubin; CK, creatine kinase; Na, sodium; K, potassium; Cl, chloride; CRP, C-reactive protein; BUN, blood urea nitrogen; Cr, creatinine; TP, total protein; Alb, albumin.

<sup>a</sup>Abnormal value.

## Case Report

A 67-year-old female patient was referred to our hospital with a 2-day history of abdominal distension, pain, and vomiting. She had a positive history of cesarean section as well as surgery for an esophageal cyst that occurred 33 and 15 years previously, respectively. On admission, her vital signs were as follows: blood pressure, 160/120 mmHg; heart rate, 72 bpm; and body temperature, 36.8°C. Physical examination by direct palpation revealed an abdominal pain without rebound tenderness. A routine blood test did not show any abnormalities except for elevated total bilirubin and C-reactive protein (Table 1). As expected, an abdominal X-ray was positive for multiple air-fluid levels of the small intestine (Fig. 1), while a plain computed tomography (CT) showed distension of the small intestine and stomach without ascites (Fig. 2). Based on the patient's history and our findings, we initially diagnosed a small bowel obstruction caused by adhesions secondary to cesarean section. At that time, an ileus tube was inserted for 10 days. However, the small bowel obstruction persisted, she subsequently underwent a surgical procedure with





**Fig. 1** The abdominal X-ray showed dilated loops of small intestine with multiple air-fluid levels.

lower median laparotomy to relieve obstruction. During surgery, a hard and immovable mass was palpable at 60 cm oral side from the terminal ileum, but there was no evidence that suggested malignancy (Fig. 3). After resection, the abdominal wall was closed by a 1-layer and continuous technique using nonabsorbable sutures. In the resected specimen, a stone measuring  $7.0 \times 4.5 \times 4.0$  cm was discovered (Fig. 4), and composition analysis of the stone revealed 98% tannins. Therefore, we confirmed her dietary history. She has the habit of eating 1 persimmon every day in autumn. As a result, our final diagnosis was small bowel obstruction caused by a persimmon bezoar. The postoperative course was uneventful; however, the patient was discharged on postoperative day 20 owing to hoping of extended hospital stay for anxiety.

### Discussion

Bezoars are defined as masses of indigestible hard materials formed in the gastrointestinal tract. Be-



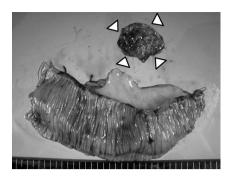
**Fig. 2** CT showed distended small intestine that extended down to the terminal ileum.



**Fig. 3** The intraoperative photograph demonstrated small bowel obstruction caused by a hard mass. The patient underwent segmental resection of the ileum.

zoars are classified into 4 primary types and further subdivided based on their constituents.<sup>2</sup> The 4 primary types of bezoars are phytobezoars, trichobezoars, lactobezoars, and pharmacobezoars.<sup>2</sup> In particular, diospyrobezoars are a subtype of phytobezoars formed after excessive persimmon intake. Persimmons are a rich source of tannins, which are polyphenolic compounds found in many other organic materials including plants, seeds, and wood.<sup>3</sup> When large enough concentrations are achieved, tannins polymerize in the acidic environment of the stomach, leading to obstruction.<sup>3</sup> Bezoars have a propensity to develop in both the stomach and small intestine, and account for 0.4% to 4% of all gastrointestinal obstructions.<sup>4</sup>

The word "bezoar" is derived from the Persian Farsi language and was coined in 1854 by a pathologist named Quain when he found an intragastric mass during autopsy.<sup>5,6</sup> The mechanisms of phytobezoar formation may be associated with shibuol (a tannin found in persimmons), gastric acid concentration, gastric motility, and delayed gastric emptying. In addition, a variety of other factors including anatomic, dietary, and alimentary factors may also affect the development of intestinal phytobezoars.<sup>7,8</sup> More specifically, gastrectomy has been identified as a major predisposing factor of bezoar formation, increasing the frequency of formation from 5% to 7%.9-12 Additionally, Kement et al<sup>13</sup> found that gastric surgery was common amongst 42.8% of patients in their study who had complications related to bezoar formation.<sup>13</sup> It is thought that other predisposing factors to bezoar formation include eating habits, such as excessive persimmon consumption, diabetic gastropathy, cerebral infarction, and medications that reduce gastrointestinal motility.<sup>14</sup> Kement et al also demonstrated that 50% of patients with bezoar



**Fig. 4** The bezoar, which was later identified to be composed of tannin (persimmons), was hard, elastic, and measured  $7.0 \times 4.5 \times 4.0$  cm in size.

had several overlapping predisposing factors, while 5.9% to 14.3% of their patients had no predisposing risk factors at all.<sup>13,14</sup> In 1986, Krausz *et al*<sup>9</sup> showed that 91.2% of their patients with phytobezoars had consumed persimmons. Likewise, Erzurumlu *et al*<sup>14</sup> reported that 17.6% of patients with bezoar had a history of persimmon consumption. In the current case, the patient's positive history of cesarean section and persimmon consumption (1 persimmon per day for a month) is thought to have contributed to bezoar formation. As expected, intestinal obstruction is the most common complication of bezoar formation.<sup>15</sup>

The rarity of obstruction by bezoars often leads to its lack of consideration in a differential, but several publications have revealed a diagnostic triad of physical examination, plain X-ray, and small-bowel studies to increase the likelihood of diagnosing one. Unfortunately, this triad has only been useful in accurately diagnosing 10% of preoperative cases.<sup>16,17</sup> On the other hand, Kement et al<sup>13</sup> have reported that abdominal CT was useful for accurate preoperative diagnosis in 77.7% of cases. Furthermore, another report emphasized that CT should be performed in all patients with bowel obstruction to avoid a misdiagnosis and to reduce the use of more invasive treatments.<sup>17</sup> If bezoar formation does occur, the majority has been removed by endoscopic or surgical procedure.<sup>18–20</sup> Recently, Ladas *et al*<sup>21</sup> showed that Coca-Cola (The Coca-Cola Company, Atlanta, Georgia) consumption alone has been effective in dissolving the bezoars without the need for more invasive procedures in 50% of all gastric phytobezoars. Moreover, Coca-Cola consumption combined with endoscopic methods has been successful in resolving more than 90% of all cases.<sup>21</sup> Several reports also showed that the dissolution of diospyrobezoars with Coca-Cola was clinically effective and had no apparent side effects.<sup>10,22</sup> The mechanism by which Coca-Cola causes dissolution is still unknown. Kement *et al*<sup>13</sup> have reported that endoscopic fragmentation has successfully treated 71.5% of bezoar obstructions but claim that limitations do exist. Kement's group states that specific equipment is needed for fragmentation, and that bleeding, perforation, and the possibility of further obstruction caused by the passage of fragmented stones exist.<sup>13</sup> If conservative treatment fails, surgical procedures are required.

Robles et al<sup>23</sup> have reported recurrent bezoar in approximately 14% patients after initial treatment. Therefore, patients who undergo treatment should also be instructed to avoid high fiber foods, persimmons, and certain medications to avoid recurrence. Similarly, Rumley et al<sup>24</sup> found that partial enzymatic dissolution of gastric bezoars leads to subsequent small bowel obstructions. In support of Rumley's findings, Nomura et al<sup>25</sup> report on patients who experienced small bowel obstruction after undergoing enzymatic treatment for gastric diospyrobezoars. In many instances, gastric and intestinal bezoars are often coexistent<sup>13</sup> and have been found concomitantly in 17% to 22.2% of patients.<sup>13,23,26</sup> In the present case, postoperative gastric endoscopy ruled out the presence of a concurrent gastric bezoar.

In summary, we reported a case of small bowel obstruction caused by a persimmon bezoar and emphasized the importance of reviewing dietary history so that diospyrobezoars can be differentiated. Moreover, if a bezoar obstruction is diagnosed, further studies may be required to determine if gastric and intestinal bezoars coexist.

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