A 98-year-old woman presented with several weeks of nausea, vomiting, abdominal pain and anorexia, and was found to have a persistent small bowel obstruction (SBO). A 4 × 3 cm calcified mass was discovered in the small bowel on computed tomography causing suspicion for gallstone ileus. The diagnosis of a calcified enterolith was made after exploratory laparoscopy given the absence of findings for gallstone ileus and the composition of the large amorphous stone.

Small bowel obstruction resulting from stone impaction is very rare, with most cases caused by a gallstone ileus or by an enterolith formed in a small bowel diverticulum. We report the second case of proximal SBO caused by a calcified enterolith, but our case is unique due to the absence of small bowel diverticula.
INTRODUCTION
Primary enteroliths are formed in the small bowel and secondary enteroliths (gallstones) are formed in the gallbladder (1). In the differential diagnosis of a small bowel obstruction (SBO) occurring in the elderly, one should always consider gallstone ileus, which occurs when a gallstone is passed through a biliary-enteric fistula. Gallstone ileus is well described, but there are few reported cases of primary enteroliths causing SBO. Herein, we report an exceedingly rare case of a calcified enterolith causing jejunal obstruction without associated small bowel diverticula. This is only the second reported case of a calcified primary enterolith causing proximal SBO (2).

CASE REPORT
A 98-year-old woman presented with several weeks of nausea, vomiting, abdominal pain and anorexia. This was her third admission in the past month for these complaints. She had a history of diverticulosis coli, dementia, arthritis, hypertension, ischemic stroke and congestive heart failure. Her medications included ciprofloxacin, diltiazem, pantoprazole and supplemental nutritional drinks. Her past surgeries included an abdominal hernia repair and hysterectomy.

Due to advanced age, previous stroke and dementia, she was mostly bedridden and cared for by her family at home.

On examination the patient was afebrile and had mild tachycardia. The abdomen was distended with increased bowel sounds and mild tenderness. There were no hernias. White blood cell count was 13,200 cells/cumm and hemoglobin 11.2 g/dL. Electrolytes, amylase, lipase and liver tests were normal. Computed tomography (CT) revealed a 4 × 3 cm calcified mass in the small bowel, located in the pelvis, with dilated proximal loops (Figures 1–2). There was no pneumobilia and the gallbladder was normal. On review of prior radiographs, the calcified mass was not definitely discernable on the plain films, but it was visible, though overlooked, on a prior CT exam.

The patient was initially treated conservatively with IV fluids, no oral intake and nasogastric tube suction. After several days, however, she developed increased abdominal pain and absent bowel sounds. Exploratory laparoscopy was performed. Intra-operatively, a hard non-mobile mass was palpated in the distal jejunum with dilated bowel proximally. The entire small bowel was carefully inspected and no diverticula were identified. There were no hernias and the gallbladder and

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common bile duct were normal. The mass could not be “milked” in either direction, so approximately 2–3 cm of jejunum was resected on either side of the mass. Pathology results of the resected small bowel revealed acute mucosal necrosis in the area where the stone was lying, but the surrounding mucosa was normal and there was no underlying stricture or other small bowel pathology. The stone measured $4.5 \times 3 \times 3$ cm and sectioning revealed multiple layers (Figures 3–4). Stone analysis showed miscellaneous material with an amorphous component containing bilious byproducts.

After surgery, her symptoms resolved and she was able to tolerate oral intake. However, due to comorbidities, advanced age and deconditioning, she had a lengthy hospital stay and was not discharged until 2 weeks later.

**DISCUSSION**

Enteroliths are divided into two groups: false enteroliths and true enteroliths (3). False enteroliths (i.e. fecoliths, varnish stones, almond pits, fruit skins, oat stones, phytobezoars or trichobezoars, and foreign bodies) are formed by clumping together and inspissation of intestinal content (3–5). True enteroliths result from precipitation and deposition of substances from alimentary chime (3). Proximal small bowel enteroliths are usually composed of bile acids, while those in the

**Fig. 3.** Intact stone measuring $4.5 \times 3 \times 3$ cm.

**Fig. 4.** Sectioned stone revealing multiple layers.
distal small bowel are mainly composed of calcium salts (1,3). Among true enteroliths with bilious composition, primary enteroliths are formed in the small bowel and secondary enteroliths (gallstones) are formed in the gallbladder (1).

Enterolith formation is thought to be secondary to hypomotility or stasis, although many conditions have been implicated (3,6,7). Small bowel diverticulosis is a well-established pre-disposing condition, where stones form de novo or around a central nidus such as a fruit stone or undigested vegetable matter (bezoar) (2). Since there were no small bowel diverticula in our case, we hypothesize that hypomotility or stasis—due to advanced age, immobility, and possibly diltiazem (8–10)—led to the formation of the enterolith. Radiological diagnosis of a primary enterolith is uncommon unless it is calcified, which usually only occurs in the more alkaline ileum (4). Conceivably, age-related hypochlorhydria and proton pump inhibition (pantoprazole) could have created an alkaline milieu in the proximal small bowel resulting in calcium deposition in the jejunum.

Gallstone ileus, or SBO due to a gallstone (secondary enterolith), occurs in about 1 in 200 patients with cholelithiasis (11). The average age at presentation is 70, and it accounts for 25% of nonstrangulated SBO’s in those over 65 years of age (11,12). On the other hand, there are fewer than 100 reported cases of primary enteroliths causing SBO (6,13). While the stone analysis in our case revealed an amorphous component containing bilious byproducts, the gallbladder and common bile duct were normal by CT and at laparoscopy, which strengthens the evidence that our case was a primary enterolith. Therefore, given the absence of findings for gallstone ileus and the radiodensity and composition of the large amorphous stone, our case represents a calcified primary enterolith causing proximal SBO. Only one other case of proximal SBO caused by a calcified enterolith has been reported (2), but our case is unique due to the absence of small bowel diverticula.

CONCLUSION

In the clinical setting of a SBO due to a mass, enteroliths must be considered in the differential diagnosis when there is no evidence of gallstone ileus or malignancy. Definitive treatment for enterolith-induced SBO is surgical with most patients requiring enterotomy or occasionally resection. Prognosis is good if timely therapy is rendered, so the desire to establish a diagnosis must not delay treatment because patients with an unresolved SBO need surgery (laparotomy or laparoscopy) rather than a diagnosis (14). ■

References