CASE REPORT

'BALL VALVE' OBSTRUCTION OF ILEUM, CAUSED BY A CALCIFIED PRIMARY ENTEROLITH

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ABSTRACT: 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 a rare case of distal small bowel obstruction caused by a calcified enterolith, but our case is unique due to its large size causing "ball valve" obstruction of ileum, at the site of stricture

KEYWORDS: Enterolith, Intestinal Obstruction.

INTRODUCTION: In the differential diagnosis of a small bowel obstruction (SBO). One should always consider gall stone 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 enterolith causing SBO. Herein, we report an exceedingly rare case of a primary calcified enterolith causing distal small intestinal obstruction associated with small bowel strictures.

CASE REPORT: A 42 years old woman presented with several years of nausea, vomiting, abdominal pain and anorexia. This was her third admission in the past few years for these complaints. Her past surgeries included two caesarean sections. On examination the patient was afebrile and had mild tachycardia. The abdomen was distended with increased bowel sounds and mild tenderness. There was no hernia, but there was a transverse scar over the lower abdomen. White blood cell count was 13,200 cells/cumm and hemoglobin was 11.2g/dL. Digital X-ray abdomen showed two radio-opaque shadows with dilated loops of small bowel (Fig. 1).

Computed tomography (CT) revealed 4X3 cm and 3X2.5 cm two calcified masses in the small bowel, located within dilated proximal loops (Fig. 2). There was no pneumobilia and the gallbladder was normal.

The patient was initially treated conservatively with intra venous fluids, no oral intake and nasogastric tube suction. Intra-operatively, two hard non-mobile masses were palpated in the distal ileum with dilated bowel proximally. The entire small bowel was carefully inspected for diverticula. There were no internal hernias and the gall bladder and common bile duct were normal. Terminal

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ileum showed five strictures, 20 cms proximal to ileocaecal junction. The distal mass could not be “milked” in either direction, so approximately two feet of ileum was resected on either side of the masses including strictures and end to end anastomosis was done (Fig 3).

Pathology results to the resected small bowel revealed acute mucosal necrosis in the area where the stone was lying but the surrounding mucosa was normal and there were five underlying tubercular strictures in the small bowel. The larger stone measured 4X3 cm and sectioning revealed multiple layers (Fig.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 tubercular strictures in the small bowel she was put on anti tubercular treatment.

DISCUSSION: Enteroliths are divided into two groups: false enterolith and true enterolith. Among true enterolith with bilious composition, primary enteroliths are formed in the small bowel and secondary enteroliths (gallstones) are formed in the gallbladder¹. False enterolith (i.e faecoliths, varnish stone, almond pits, fruit skins, oat stone, phytobezoars or trichobezoars, and foreign bodies) are formed by clumping together and inspissations of intestinal content. True enteroliths result from precipitation and deposition of substances from alimentary chime. Proximal small bowel enteroliths are usually composed of bile acids, while those in the distal small bowel are mainly composed of calcium salts. 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 (bezor)^{2,3}.

Enterolith formation is thought to be secondary to hypomotility or stasis, although many conditions have been implicated^{3,5,6}. Since there were multiple strictures in the ileum in our case we hypothesize that hypomotility and stasis 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 ileum⁴.

Gallstone ileus, or SBO due to a gallstone (secondary enterolith), occurs in about 1 in 200 patients with cholelithiasis. The average age at presentation is 70 years, and it accounts for 25% of non-strangulated SBO's in those over 65 years of age. On the other hand, there are fewer than 100 reported cases of primary enterolith causing SBO^{6,7}. 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 laparotomy, which strengthens the evidence that our case was a primary enterolith. Therefore, given the absence of finding for gallstone ileus and the radio-density and composition of the large amorphous stone our case represents a calcified primary enteroliths causing distal SBO. Our case is unique due to large enterolith causing complete ball valve like obstruction at the distal small intestine stricture.

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.

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(Figure 1) Digital X- Ray. Arrows showing enteroliths.

(Figure 2) CT scan. Arrow showing enterolith.

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(Figure 3) Resected specimen of ileum, arrows showing strictures.

(Figure 4) Complete enterolith [A] and section of enterolith [B] showing lamellar pattern.