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Enterolith: A Rare Differential for Small Bowel Obstruction

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Abstract

One surgical complication that is frequently seen in hospitals is small intestinal obstruction. It is uncommon and sometimes challenging to diagnose small intestinal obstruction caused by an enterolith. Due to a variety of illnesses, intestinal stasis can lead to the development of gastrointestinal concretions, or enterolithiasis, which can manifest clinically in a variety of ways. A 48-year-old male came to the Surgery OPD complaining of intermittent spasmodic pain in the epigastric region and right upper abdomen for ten days. He had a distended abdomen. He had a history of previous operations for Meckel's Diverticulum 39 years back in 1981. Initial conservative management was given, but with no relief, the patient was planned for laparoscopic adhesiolysis. During the procedure, an enterolith was detected and removed via enterotomy.

INTRODUCTION

Enteroliths are calculi that develop inside the intestinal lumen. In everyday life, it is uncommon. It is speculated that the underlying cause of this illness is either gut stasis or hypomotility. Primary enteroliths form inside the intestine, but secondary enteroliths, like gallstones and urine stones, form outside the intestine and migrate into it through a fistula. Primary stones can be real (made when the chyme's contents precipitate) or false (produced when insoluble foreign materials, as bezoar). Usually, intestinal diverticula or the area immediately surrounding a stenosed lesion cause stasis in the digestive tract, which results in enterolithiasis.

Many conditions, including Crohn's disease, TB and strictures following surgery, like the one in our case, might generate the stenotic lesions linked to enterolithiasis. Although symptoms are frequently vague, they can include "tumbling" stomach pain, vomiting and nausea from bowel obstruction.

Case Presentation: A 48-year-old male came to the OPD with complaints of intermittent right upper abdominal pain and intermittent spasmodic epigastric pain for ten days. The pain was dull and aching and increased with food intake. The patient gave no history of fever. There was no history of trauma and the patient did not have any urinary complaints. The patient has a history of previous operation for Meckel's diverticulum 30 years back.

The patient is nondiabetic and non-hypertensive and does not have any other comorbidities. Upon examination, the patient's abdomen was slightly distended, soft and non-tender. No palpable swelling is present. Bowel sounds present. Blood investigation reports were usual. An erect abdominal X-ray exhibited a radiopaque mass in RIF with a dilated portion of the small bowel, seen on the X-ray (Fig. 1). The USG of the patient showed evidence of dilated bowel loops (Maximum diameter-3.3cm) in bilateral lumbar, hypogastric and left iliac fossa regions. A CECT abdomen showed well defined hyper dense (Average HU: + 800 to +1000) oval-shaped lesion of approximate size 4.5 × 4.5 × 4.0cm in the lumen of the distal ileum causing upstream dilatation of proximal ileal (Maximum Diameter: 5.3cm) and distal jejunal (Maximum Diameter: 3.3cm) loops. A redundant sigmoid colon was noted. The remaining small and large bowel loops are partially opacified with contrast and appear normal. CECT abdomen and pelvis reveal features suggestive of small bowel obstruction likely secondary to enterolith in the distal ileum. On Diagnostic laparoscopy, multiple adhesions were noted in the distal ileum. The bowel was traced and an enterolith was present around 30cm proximal to the IC

junction. The bowel proximal to the enterolith was found to be dilated and slightly oedematous. Adhesions between the small bowels were observed. The rest of the bowel was traced and found to be normal. Adhesiolysis was done with a harmonic scalpel near the obstruction site to mobilize the bowel freely. The abdomen was opened by a mini-laparotomy incision of 6cm on the right side of the lower abdomen over the previous scar, dissection was done and the peritoneal cavity opened. Enterolith obstructed bowel was removed (Fig.2) and enterolith was removed via enterotomy. (Fig.3) Primary closure of enterotomy done in two layers with vicryl 3-0 and silk 2-0). The enterolith retrieved measured 5x5cm. Patient was given IV antibiotics and analgesic. Post operative recovery was uneventful.

RESULTS AND DISCUSSIONS

Enteroliths, or enterogenous foreign bodies, are very prevalent in animals like horses but uncommon in humans as clinical and radiological entities. Enteroliths can be categorized as primary or secondary. Primary enteroliths originate in the large or small intestine, whereas secondary enteroliths form in organs that are related to the intestines, such as the urine or gall bladder. Primary enterolithiasis is typically associated with clinical diseases that cause stasis and hypomotility (Singhal^[1]). Enteroliths can develop in any part of the intestinal tract. Stomach (bezoar), appendix (coprolith) and colon (faecolith) have been reported to have the highest frequency (Kia and Dragstedt^[2]). The "composition of true primary enteroliths varies depending on the location and is generated from chemical substances already present in the intestine in physically compromised sites of stasis. Patients with cholecysto-enteric fistula may experience an obstruction such as a gallstone due to secondary enteroliths, which originate outside the gastrointestinal tract proper and migrate into the intestines (Sharma^[3]). Primary as well as secondary enterolithiasis occurrence ranges significantly from 0.3-10% (Gurvits and Lan^[4]).

Under typical feeding circumstances, materials present in chyme form true primary enteroliths. They are further separated into enteroliths, which are calcium salts and choleic acid (Atwell and Pollock^[5]). Choleic acid enteroliths are usually located in the proximal small bowel and require an acidic pH. However, calcium salts' primary enteroliths are most frequently generated in the terminal ileum because they need a higher pH to precipitate (Gurvits and Lan^[4]). As a result of ingesting exogenous particles like bezoars, insoluble foreign compounds in the colon might produce false primary enteroliths. External calcification of false enteroliths in the distal small



Fig. 1: X-ray abdomen showing Enterolith in RIF



Fig. 2: Enterolith causing obstruction in distal ileum



Fig. 3: Enterolith removed via longitudinal incision at distal ileum

bowel might lead to mixed concretions (Atwell and Pollock^[5]). An obstruction results from the migration of secondary enteroliths, which originate outside the alimentary system and move into the colon. These consist of renal or gallstones, ileus (Gurvits and Lan^[4]). Enterolithiasis's clinical presentation varies depending on the cause, age, location, chemical makeup and, lastly, the size of the stone. A younger patient with underlying TB (developing countries) or inflammatory bowel illness (developed countries), or an older patient undergoing intestinal surgery, such as our patient, should be suspected of having primary enterolithiasis.

Clinical signs of enterolithiasis include nausea, vomiting, distention and abdominal discomfort that can be occasionally rapid but frequently fluctuate subacutely. This condition is caused by the enterolith falling into the bowel lumen (Gupta^[6], Mendes Ribeiro and Nolan^[7], Tewari^[8]). Traditionally, the first method for finding enteroliths has been abdominal

radiography. In as many as one-third of the cases, it can identify stones (Athey^[9]). The calcium concentration affects the enterolith's visibility. When compared to choleic acid enteroliths, calcium salts enteroliths are more radiopaque (Paige^[10], Tewari^[8], Yuan^[11]).

In follow-up exams, they appear round, rectangular, or oval with a pale core, dense rim and mobility (Singhal^[1]). A CT scan could be useful in determining the location and number of the enteroliths causing the problem. To rule out secondary enterolithiasis, the bile duct and gallbladder must also be evaluated (Gurvits and Lan^[4]). An enterolith is categorized as idiopathic if all other possible causes have been ruled out. An idiopathic enterolith is often solitary, greater than 25mm in diameter, primarily made of calcium salts and positioned in the terminal ileum (Athey^[9]). Acute small intestinal obstruction is infrequently caused by enterolithiasis (Klingler^[12]). Enteroliths can result in intermittent, partial, acute, subacute, or chronic intestinal obstruction (Jones and McWhirter^[13]). The propagation of the endoluminal contents would normally clear enteroliths with a diameter of less than 20mm. In the absence of an underlying luminal impairment, enteroliths greater than 25mm in diameter may produce intestinal obstruction (Nakao^[14]).

The goal of treating enterolithiasis is to remove the enterolith and treat the underlying condition to stop more enteroliths from forming in the future (Gurvits and Lan^[4]). Surgery is the primary treatment for small bowel obstruction produced by an enterolith. In approximately 50% of instances, the enterolith can be digitally fragmented and then manually milked into the colon in smaller pieces. This causes the obstruction to pass. As in our instance, an alternative procedure that may be used is proximal enterotomy with enterolith removal (Leow and Lau^[15], Steenvoorde^[16]).

In the absence of underlying luminal impairment, expectant therapy "with serial abdominal assessments, hydration, nasogastric tube suctioning and electrolyte correction may be explored for acute bowel obstruction along with enteroliths smaller than 20 mm in diameter (Ihara^[17]). The death rate for uncomplicated primary enterolithiasis is" extremely low, although it can rise to 3 percent in patients who have severe obstruction, are in poor health, or whose diagnosis was delayed. (Steenvoorde^[16]).

CONCLUSIONS

It can be difficult to make a definitive diagnosis of enterolithiasis-related bowel obstruction during the preoperative period. Suspicion for Enterolithiasis is needed to prevent treatment delay and misdiagnosis.

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