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Case Report

An unusual case of small bowel obstruction due to multiple primary enteroliths: a case report

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ABSTRACT

Enteroliths are caused by intestinal stasis or gut hypomotility. It can be primary enteroliths which is produced within the intestine or secondary where stones are migrated from outside gastrointestinal tract like gall stone ileus. A 57-year-old lady presented with complaints of pain in abdomen with constipation. On abdomen X-ray there was 3 large laminated calcified bodies in pelvis. On ultrasound there were gall stones present and hyperechoic bodies present in bowel loops, urinary bladder was empty. On CECT 3 enteroliths present in ileum causing small bowel obstruction. Patient was planned for diagnostic laparoscopy where focal bowel dilatation present at mid ileum. There was no cholecystoenteric fistula present. Laparoscopy cholecystectomy along with laparoscopy assisted extracorporeal resection and anastomosis of involved bowel with removal of 3 enteroliths of size 4×4 cm done. Small enteroliths can pass spontaneously but large and impacted enteroliths needs surgical intervention.

Keywords: Primary enteroliths, Small bowel obstruction, Resection and anastomosis

INTRODUCTION

The stones that develop inside the intestinal lumen are referred to as "enteroliths." These are rare in humans, despite being frequent in the equine population.

This syndrome is believed to be caused by either gut stasis or hypomotility.[1] The condition is known as enterolithiasis, or the presence of stones in the digestive system. Enteroliths can be classified as primary which is produced within the intestine or secondary (like gallstones and urine stones, which form outside and migrate into the digestive tract through a fistula). Primary stones can be false which are made from insoluble foreign compounds such as bezoar or true which is produced by the precipitation of chyme's contents.[2]

Enterolithiasis typically results from intestinal tract stasis, which can occur in case of intestinal diverticula or in the proximity of a stenosed lesion.[3]

CASE REPORT

A 57-year-old lady presented with complains of pain in central abdomen which was colicky nature since associated with constipation and bloating. Patient had no medical comorbidities and no history of tuberculosis or inflammatory bowel disease. Patient had no previous surgical history. On per abdomen examination abdomen was soft, non-tender with no guarding or rigidity. On Auscultation bowel sounds were present and were normal. Digital rectal examination was unremarkable. All the blood parameters were within normal limits.

On abdomen X-ray there were three large laminated calcified bodies in pelvic region. At first urinary bladder calculi was suspected due its location. On ultrasonography bladder was empty there was calcified body with suspected location in ileum. On USG gall stones were also present.

Due to diagnostic dilemma patient was advised for CECT abdomen. On CECT abdomen there was 3 large hyperdense lesions of average size 4.3×3.5 cm intraluminally within small bowel (ileum) causing focal bowel dilatation of 5.4 cm and gross dilatation of bowel loops proximal to enteroliths suggestive of small bowel obstruction due to small bowel enteroliths.

Patient was planned for diagnostic laparoscopy and proceed. On laparoscopy findings there was gross dilatation of bowel 200 cm proximal to Ileocecal junction forming transition point proximal to which bowel loops were dilated and distally bowel loops were collapsed. No lymphadenopathy or signs of inflammatory bowel disease present. Gall bladder was seen distended with bile and stones. No cholecystoenteric fistula was present. Bowel loops were traced and three enteroliths were present in small bowel causing gross dilatation of bowel and wall edema with signs of impending ulceration of bowel. Laparoscopy cholecystectomy along with laparoscopy assisted extracorporeal resection and anastomosis was done via infraumbilical midline incision and 5 cm of bowel segment with enteroliths were resected and side to side isoperistaltic stapled anastomosis done.

Postoperative period was uneventful. No definitive cause of enteroliths were found either on CT scan or intraoperative.

Histopathology report-section from intestines shows focal mucosal ulceration and inflammatory infiltrates, section from gall bladder shows chronic cholecystitis.

Enteroliths were sent for stone analysis and were composed of calcium oxalate monohydrate (60%), calcium oxalate dihydrate (30%) and carbonic apatite (10%).

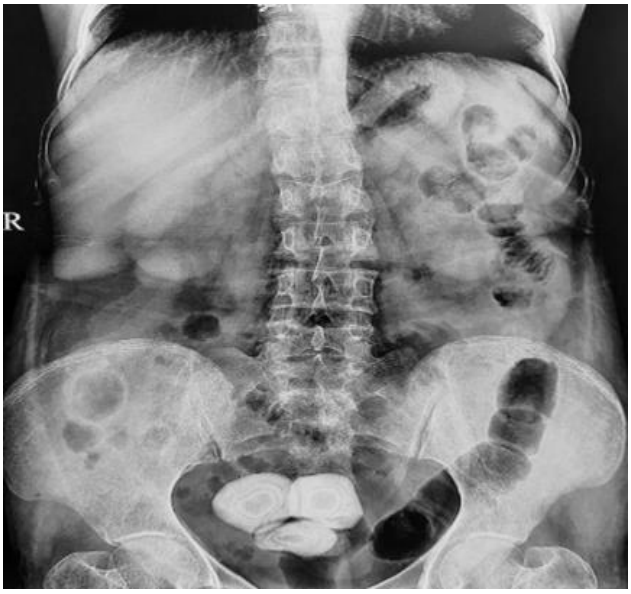

Figure 1: X-ray of 3 calcified laminated radiopaque bodies in pelvis.

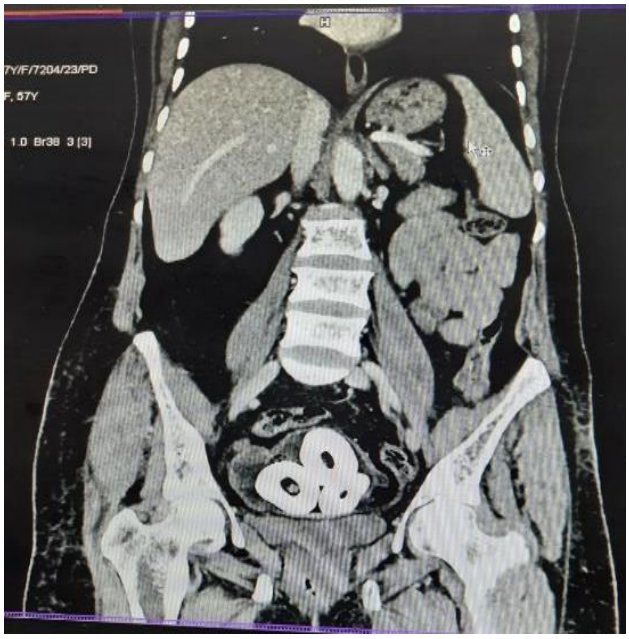

Figure 2: CECT abdomen sagittal section showing calcified bodies in bowel.

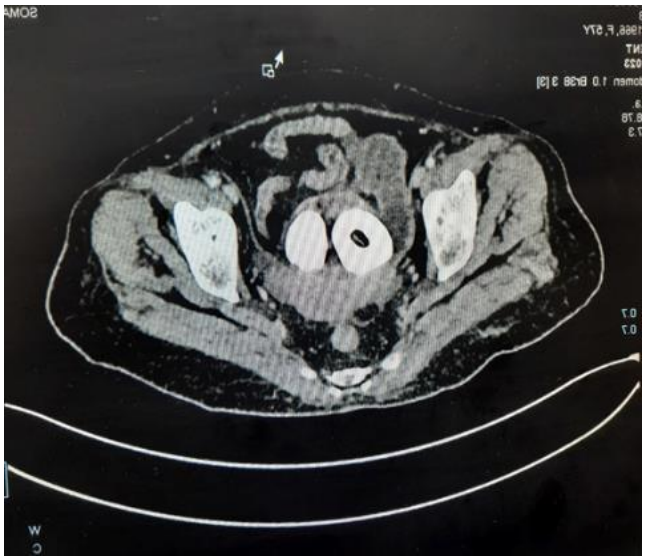

Figure 3: CECT abdomen axial section showing enteroliths in bowel.

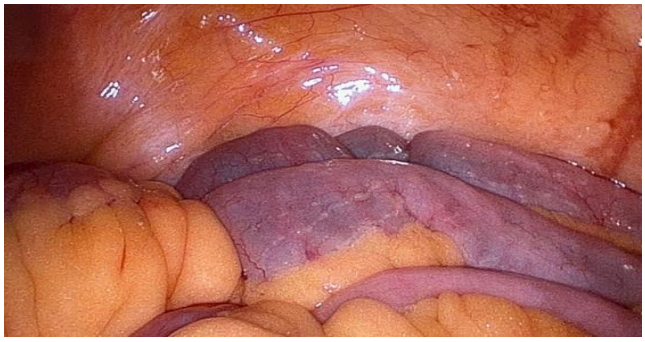

Figure 4: Laparoscopic finding of grossly dilated bowel loops (blue arrow).

Figure 5: Dilated bowel loops present 200 cm proximal to ileocecal junction (yellow arrow).

Figure 6: Cut section of bowel showing 3 large enteroliths.

Figure 7: Resected bowel segment showing mucosal ulcers and strictures.

Figure 8: Three large enteroliths.

DISCUSSION

Enterolith are rare clinical and radiological entities in humans, although they are very common in animals such as horses.[4]

Primary enteroliths develop in conditions characterised by intestinal stasis, including diverticular disease, surgical enteroanastomosis, blind pouches, intestinal stenosis, and strictures associated with inflammatory or infectious bowel disorders. Enteroliths are a rare cause of small intestinal blockage and are usually found accidentally during imaging.[4]

True primary enteroliths are formed from substances found in chyme under normal alimentary conditions. They are subdivided into the choleic acid and calcium salts enteroliths.[4] Choleic acid enteroliths require lower pH and are typically found in the proximal small bowel. On the other hand, calcium salts primary enteroliths require an alkaline pH to precipitate and thus are most often formed in the terminal ileum.[3] 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 bowel might lead to mixed concretions.[4]

Secondary enteroliths are formed outside the gastrointestinal tract and then migrate into the bowel, causing an obstruction like gall stone ileus due cholecysto-entric fistula.[3]

Primary enterolithiasis can arise from a variety of causes, including congenital and acquired diverticular diseases, surgeries, afferent loops, blind pouches, TB and Crohn's disease, radiation or eosinophilic enteritis, mucosal diaphragmatic disease, intestinal duplication, fistula, malignancy, intra-abdominal adhesions, external

compressions, incarcerated hernias, intestinal aganglionosis, intestinal amoebiasis, and ischemic enteritis.[3] Here we report a case of small bowel obstruction due to multiple primary enteroliths in a 57 year old lady which was diagnosed on abdominal radiographs and CECT abdomen. Patient was managed with surgery with removal of enteroliths and resection and anastomosis of involved bowel segment.

Abdominal radiography is typically the first step used in diagnostic investigations to find enteroliths. It can identify stones in one third of cases. The visibility on the abdomen x-ray is determined by the calcium content. Compared to choleic acid enteroliths, calcium salts enteroliths exhibit greater radiopacity. Enteroliths typically appear oval or spherical on radiographic investigations, with dense rims and a pale centre. They are typically movable during serial examinations.[5] A number of differential diagnoses, such as calcified lymph nodes, enteroliths, fat necrosis, biliary and urinary calculi, mesenteric teratoma, and calcified fibroids, can be made in relation to calcifications shown on abdominal radiography.[6]

Due to their varied sites, enteroliths might be misdiagnosed as bladder, renal, or ureteral calculi, which can lead to diagnostic dilemma. For diagnostic confirmation CECT abdomen can be done.[7]

The removal of enteroliths and the correction of underlying pathology to stop the creation of new enteroliths in the future constitute the optimal management of enterolithiasis.[6]

Generally, most of the smaller stones are likely to pass down spontaneously. However, large and impacted stones like our patient may require surgical intervention. The operative management includes enterolith fragmentation, milking into the proximal colon, and removal through an enterotomy, or segmental resection and anastomosis. Surgical management is the base of therapy in case of small bowel obstruction secondary to enterolithiasis.[6]

Expectant management with serial abdominal examinations, hydration, electrolyte correction, and nasogastric tube suctioning may be taken into consideration for acute small intestinal obstruction with enteroliths smaller than 20 mm in diameter if lumen is not compromised.[8]

Although the mortality rate for uncomplicated primary enterolithiasis is extremely low, it can rise to 3% in patients who have severe obstruction, are in poor condition, or whose diagnosis was delayed.[9]

CONCLUSION

Enteroliths are rare cause of small bowel obstruction. It can be diagnosed on abdomen x-rays in one third of cases but it can be misdiagnosed as bladder calculi due its site as in our case and can cause diagnostic dilemma, in such cases CECT abdomen is helpful. Small sized enteroliths can pass spontaneously but surgery is required in cases of large or impacted enteroliths. A high index of suspicion is required to prevent delayed or misdiagnosis.

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