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Case Report

A diagnostic dilemma: enterolith formation secondary to stricture and subsequent bowel obstruction: a case report

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ABSTRACT

We report a case of a 67 years male patient who presented with complaints of pain in abdomen, loose motions since 3 months and distension of abdomen since 1 day. At the time of admission patient was vitally stable, with no history of vomiting and constipation. Plain x-ray abdomen erects showed evidence of approximately 4-5 radio opaque foreign bodies in the abdomen. Contrast Enhanced computed tomography (CECT) of abdomen revealed multiple ileal strictures with closed loop obstruction with large monoliths. Exploratory laparotomy confirmed 7 ileal strictures each 10 cm apart with proximal dilatation. Resection of ileal stricture was performed and ileoileal anastomosis done. The resected segment contained seven hard, black enteroliths.

Keywords: Enterolithiasis, Crohn's disease, Subacute intestinal obstruction, Strictures

INTRODUCTION

Subacute intestinal obstruction is a frequently encountered surgical emergency. Its primary causes include strictures, polyps, and tumors, with gallstones or foreign bodies being fewer common culprits. Enteroliths, although uncommon, can also lead to intestinal obstruction.[1] They typically form near strictures, within diverticula, or in blind loops, often due to stasis.[2] The phenomenon of enteroliths was initially documented by Pfahler and Stamm in 1915.[3] In this context, we present a case involving subacute intestinal obstruction with multiple strictures with multiple enterolith located in the distal ileum. The patient underwent a procedure involving segmental ileal resection and ileo-ileal anastomoses. On histopathological examination of strictures, it turned out to be Crohn's disease (CD).

CASE REPORT

A 67 years male patient referred to us with complaints of pain in abdomen, loose motions since 3 months and distension of abdomen since 1 day. At the time of admission patient was vitally stable, with no history of vomiting and constipation, his past medical history was not suggestive of any associated medical condition and no history of any ingestion of herbal medications. On repeated asking patient does not give any history of exposure to tuberculosis (TB) or TB contact. A plain radiograph abdomen erects showed evidence of approximately 4-5 radio opaque densities in the abdomen. Baseline blood investigations, chest X-ray and ECG were normal. Ultrasound of the abdomen revealed distended bowel loops in right iliac fossa and right lumbar with maximum diameter of 3.1 cm and no evidence of cholelithiasis or cholecystitis. A CECT of abdomen revealed multifocal areas of circumferential symmetric stricturous narrowing seen in ileum causing significant luminal narrowing with resultant proximal bowel dilatation, with closed loop obstruction and evidence of multiple large fecoliths with central lucency seen in dilated ileal loops largest of size 3×1.6×1.6 cm. The patient underwent exploratory laprotomy with midline incision. During the procedure 7 strictures were identified

starting from 60 cm from the duodenojejunal flexure, each 10cms apart with palpable enterolith in between the strictures. Segmental ileal resection with end-to-end hand sewen ileo-ileal anastomosis done. There was no fistula between the gall bladder and the duodenum. Rest of the bowel was healthy. An abdominal drain kept in the pelvis and abdomen closed in layers. From the resected ileal loop, enterolith retrieved measuring 3×1.5×1.5 cm and bowel sent for histopathological examination. Postoperative period was uneventful and patient discharged on 10th day histopathological examination suggestive of marked inflammation consisting of neutrophils, lymphocytes, plasma cells, and eosinophils in lamina propria seen, surrounding mucosa shows inflammation in lamina and at places transmural inflammation seen. Occasional epithelial granuloma can be seen. Transmural lymphoid aggregates also noted suggestive of CD. Stone analysis was suggestive of calcium oxalate stones.

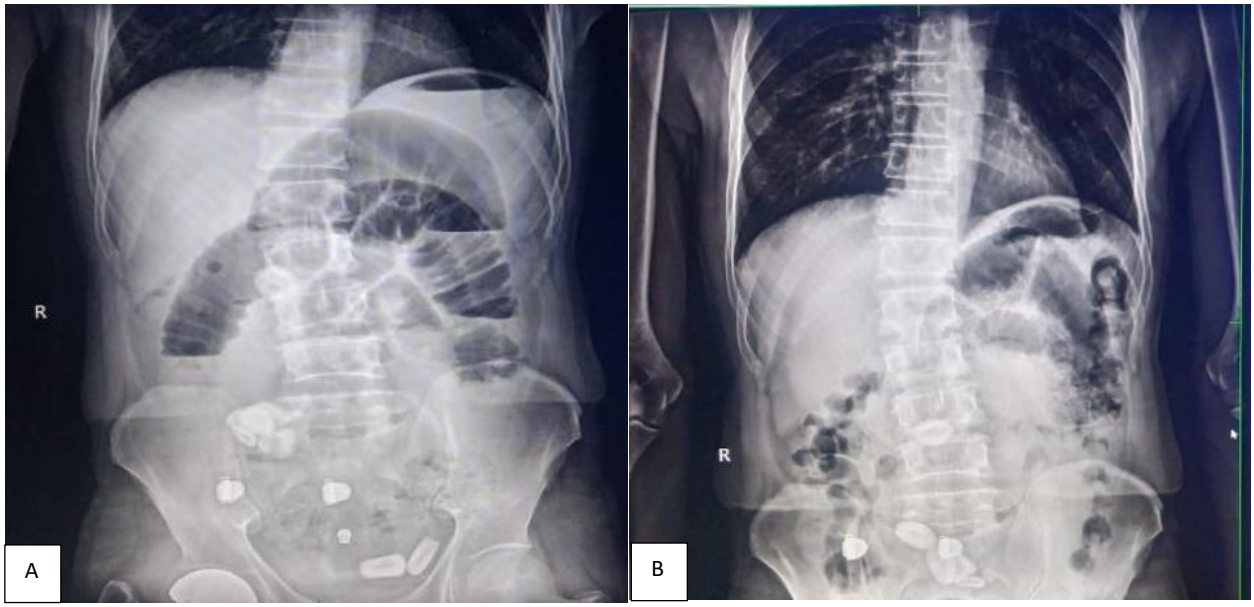

Figure 1 (A and B): X-ray abdomen erect showing radio-opaque densities in right iliac fossa and pelvis. Follow up x-ray abdomen erect with change of position of the radio-opaque densities.

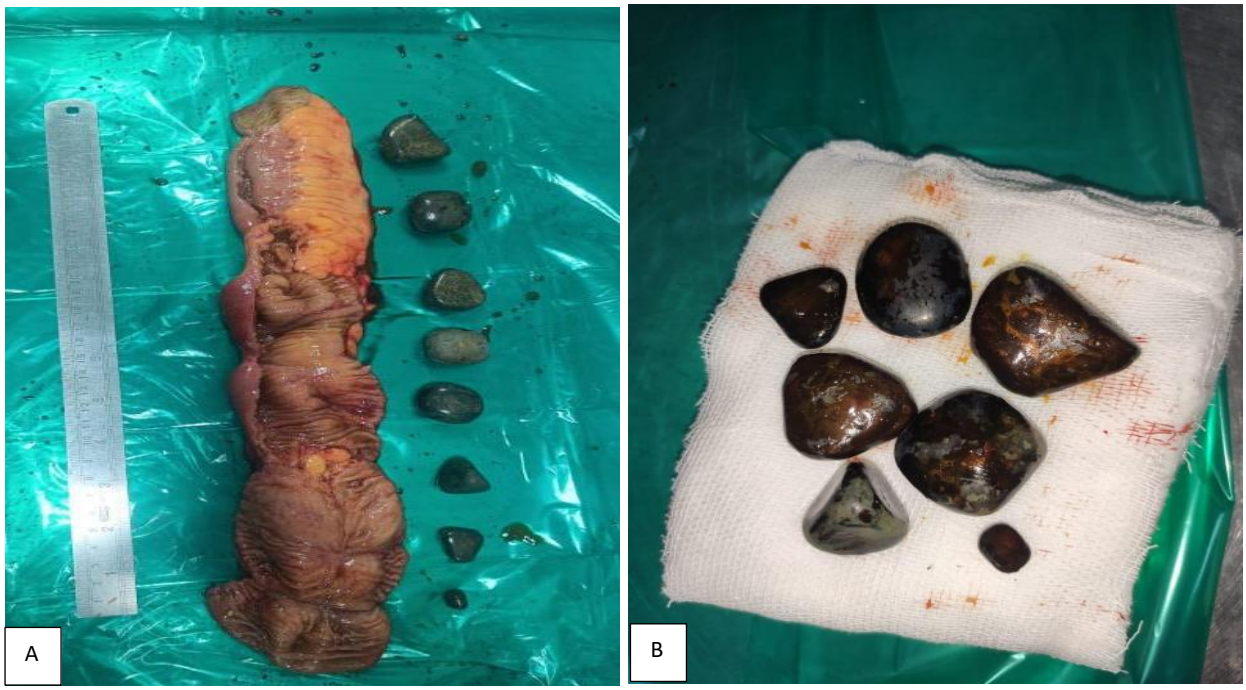

Figure 2 (A and B): Specimen of resected ileal segment. Enteroliths max measuring 3×2×1.5 cm.

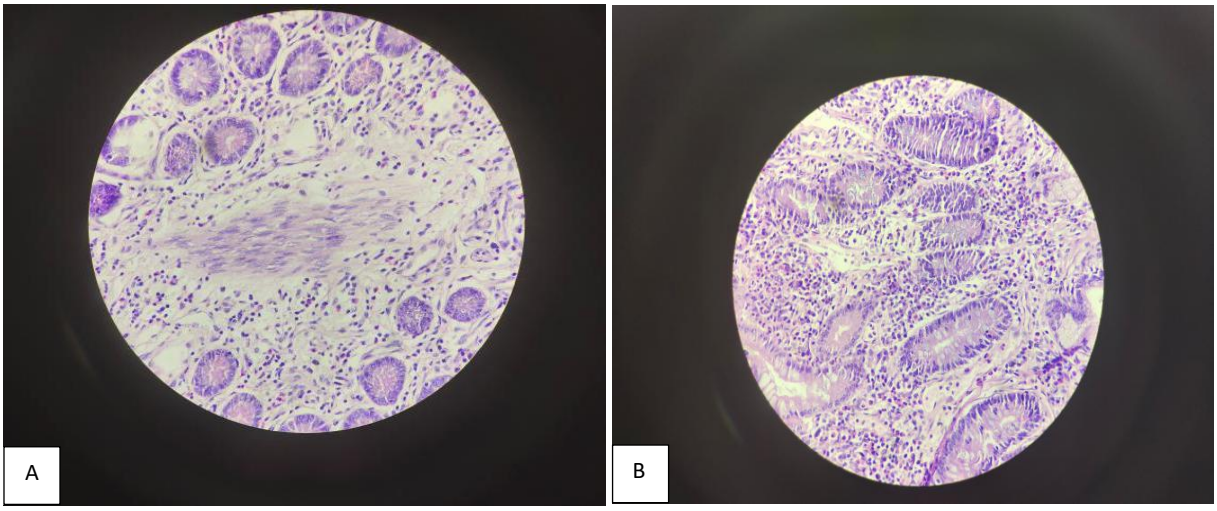

Figure 3 (A and B): Histopathology show in epithelioid granuloma. Inflammatory infiltrate in submucosa including eosinophils.

DISCUSSION

The formation of enteroliths in the small intestine can be classified into primary enteroliths, originating within the small bowel, and secondary enteroliths, formed in the gallbladder as gallstones.[2] True enteroliths in the small intestine can be categorized into three types based on their composition: those primarily made of bile acids, phosphates, or calcium oxalate.[3,4] It's worth noting that false endogenous enteroliths are more prevalent than true enteroliths.[5]

The chemical composition of enteroliths is influenced by the site and pH of the intestinal lumen. The relatively high acidity in the proximal duodenum and jejunum facilitates the precipitation of bile acids, notably cholic acid, which are radiolucent. On the other hand, the alkaline pH in the lower distal parts of the small intestine promotes the precipitation of calcium, leading to the formation of radio-opaque enteroliths.[6]

Stasis plays a crucial role in enterolith formation, whether it's due to stricture formation associated with conditions like intestinal TB (ITB), CD, carcinoid tumors, post-traumatic or post-surgical strictures, radiation enteritis, etc. or due to diverticulae.[6] These conditions create an environment where the ingredients for enteroliths can accumulate and precipitate, contributing to their development within the small intestine. In CD, it's relatively common to encounter multiple areas of small bowel stenosis, although cases where stenosis is complicated by enteroliths are rare. Geoghegan et al documented a case where small bowel obstruction resulted from a large enterolith in a patient with CD. While most enteroliths remain asymptomatic and cause no issues, they can sometimes lead to complications such as intestinal obstruction, ileus, or perforation.

Differentiating CD from ITB remains a significant challenge for clinicians in developing countries where ITB is endemic and the incidence of inflammatory bowel disease is rising. While there are specific clinical differences-such as diarrhea, hematochezia, and perianal disease being more common in CD, and fever and night sweats being more typical in ITB-other features also help distinguish the two. Endoscopically, Crohn's often presents with longitudinal and aphthous ulcers, whereas ITB shows transverse ulcers and a patulous ileocecal valve. Histologically, ITB is characterized by caseating, confluent, and large granulomas, while CD typically has microgranulomas. Microbiologically, ITB can be identified by a positive stain or culture for acid-fast bacilli (AFB). Radiologically, CD usually involves long segments, the comb sign, and skip lesions, while ITB features necrotic lymph nodes and contiguous ileocecal involvement. Despite these differences, the definitive diagnostic features for ITB are caseation necrosis on biopsy, a positive AFB smear and/or culture, and necrotic lymph nodes on cross-sectional imaging, underscoring the need for a comprehensive diagnostic approach.[8]

CONCLUSION

In India, the differential diagnosis for acute or subacute intestinal obstruction must consider the regional prevalence of certain diseases, particularly ITB, which is more common than CD. However, the clinical presentation of foreign body intestine causing intestinal obstruction should also raise a high suspicion for CD. Post-surgical management of CD is multifaceted, focusing on preventing recurrence, managing symptoms, and improving quality of life. This includes medical therapy with maintenance medications such as amino-salicylates for mild cases, immunomodulators to reduce steroid dependency, and biologic therapies like anti-TNF agents, integrin inhibitors, and IL-12/23 inhibitors for moderate to severe cases. Regular follow-ups are essential, involving clinical assessments, blood tests for inflammation markers, periodic imaging like MR enterography or CT enterography, and endoscopies to monitor disease activity and detect complications early.

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