



A rare case of primary true enterolithiasis presenting with large bowel obstruction in a patient with prior bowel resection and newly confirmed Crohn's disease

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BACKGROUND

Enterolithiasis refers to hard, dense masses within the bowel that may lead to obstruction. Enteroliths are classified as primary or secondary. Secondary enteroliths originate outside the gastrointestinal tract and migrate into the bowel, most commonly as gallstones. Primary enteroliths are subdivided into true and false types. True primary enteroliths form within the bowel from luminal substances in areas of anatomical alteration and stasis (1). Their composition varies by location: proximal small intestine stones are typically composed of cholic acid, while distal small intestine stones often contain calcium phosphate, calcium oxalate, or calcium carbonate. Enteroliths are most frequently observed in post-surgical patients, particularly at side-to-side or end-to-side anastomoses. Stenosing Crohn's disease is a recognized risk factor, with stones commonly forming in aneurysmal, saccular, or dilated segments. Rarely, enteroliths can cause acute bowel obstruction (2, 3).

CASE PRESENTATION

A 58-year-old female was admitted to our ward with subacute diarrhea and signs of bowel obstruction. Her history included a partial large bowel resection

with a caeco-sigmoid anastomosis due to acute bowel obstruction caused by multiple intramucosal lipomas. Five years prior, she underwent balloon dilatation of the anastomosis for symptomatic stenosis. On admission, she reported diarrhea lasting nearly four weeks, associated with nausea, and on the day of admission she experienced fever (38.5°C) and chills. Physical examination revealed a febrile patient with a distended, diffusely tender abdomen and hyperactive bowel sounds. A plain abdominal X-ray demonstrated a 6 cm-wide colonic loop in the right lower quadrant, borderline dilated small bowel loops, and a ~40 mm calcified mass in the pelvic cavity. Subsequent abdominal CT revealed obstruction of the cecum and an additional 20 mm calcified lesion in the pelvis, with no signs of perforation (figure 1). Given a presumed diagnosis of anastomotic stenosis, a colonoscopy with possible balloon dilatation was planned. Endoscopic examination revealed a narrowed anastomosis with a lumen of approximately 8 mm; dilatation was performed using a TTS balloon up to 12 mm. Upon passing the anastomosis with a therapeutic gastroscope, a large enterolith was visualized. Ulcerations were observed at the Bauchin valve and in the small bowel, from which biopsies were taken. The large enterolith was considered the likely cause of obstruction, and lithotripsy was attempted. The stone was extremely

hard, and each attempt at partial fragmentation, although successful, resulted in damage to the lithotripter (figure 2). The second calcified lesion was not identified during the procedure. Post-procedure, the patient's symptoms rapidly resolved, and she was discharged four days later. Stone analysis confirmed a calcium phosphate composition, and histology of the ileum revealed features consistent with Crohn's disease. Three weeks later, the patient remained asymptomatic, and a second colonoscopy was performed to fragment the remaining stone.



Figure 1: Enterolith visible in right lower abdominal quadrant on CT scan

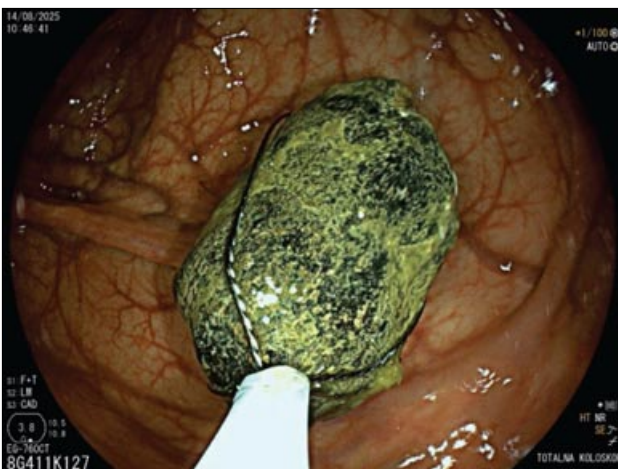


Figure 2: endoscopic lithotripsy of the enterolith

CONCLUSION

Enterolithiasis is a rare but important cause of bowel obstruction, particularly in patients with prior bowel surgery or anatomical alterations. In our case, the obstruction was successfully managed endoscopically, avoiding the need for surgery. However, due to the patient's underlying Crohn's disease and anatomical predisposition, she remains at risk for recurrence. Careful follow-up and monitoring are therefore essential to promptly identify and manage any future episodes. This case highlights that with timely endoscopic intervention, even large and hard enteroliths can be treated non-surgically.

References:

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