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Bowel ischemia and gangrene—primary true enterolith

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ABSTRACT

Enterolithiasis, or presence of stone concretions in the gastrointestinal tract, is an important but relatively uncommon clinical condition that has recently gained significant attention with advances in the gastrointestinal field.

Here, we present a case of an old male having features of bowel Ischemia and gangrene formation. Patient underwent exploratory laparotomy and there was a presence of inflamed Gut with a perforation just one and half feet away from IC junction and an obstruction was also identified by following the dilated bowel loops distally, palpation revealed a hard stone and enterotomy incision at the site delivered a stone. Following it, resection of thickened mesentery was done and loop ileostomy was made.

The most important prognostic factor in such cases relies on time interval between onset of symptoms and definitive treatment. By improving the awareness and early recognition of mesenteric ischemia will lead to improved survival in the condition.

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1. Introduction

Bowel ischemia can affect a small or large intestine and can occur by any cause, which leads to intestinal blood flow reduction [1]. This is an uncommon medical condition, but it has a high mortality rate [2]. Intestinal ischemia occurs when at least a 75% reduction in intestinal blood flow for more than 12 h [3]. However, after a prolongation of low perfusion or hypoxemia, progressive vasoconstriction leads to reducing collateral flow and subsequently full-thickness necrosis of the intestinal wall and perforation [4]. Bowel ischemia can be classified as small intestine ischemia, which is commonly known as mesenteric ischemia [5] and large intestine ischemia, which generally referred to as colonic ischemia [6]. Mesenteric ischemia (MI) accounts for 0.1 % of all hospital admissions, despite this being a rare medical condition. It has high mortality rates ranging from 24 % to 94 % [7].

Incidentally, detected enterolith (the enterogenous foreign bodies) are relatively rare causes of small bowel obstruction, none the less, if detected, they must be followed up for the development of bowel obstruction, direct pressure injury to the intestinal mucosa, intestinal gangrene, intussusceptions, afferent loop syndrome, diverticulitis, iron deficiency anemia, gastrointestinal hemorrhage, and perforation [8]. Basically enteroliths are stones primarily formed in gastrointestinal tract and classified in two groups: Primary enterolith in the small intestine, while secondary enteroliths

in the associated organs like gall bladder. Mortality of primary enterolithiasis may reach 3% and secondary enterolithiasis 8% [9].

Here we present a case report of patient having small bowel ischemia and gangrene with primary enterolith.

2. Case presentation

A 56-years-old male who is a known case of DM Type-2 (on regular medications), has presented here with a complaint of abdominal pain which is severe and colicky in nature followed by progressively increasing distension of the abdomen, vomiting (non-projectile and non-bilious) and constipation for 3–4 days. On clinical evaluation of the abdomen, there was gross distension along with rebound tenderness over umbilical region. Patient was then investigated as per protocol and USG whole abdomen was done which showed fatty liver with right hepatic cysts and dilated bowel loops with fluid. X-ray abdomen showed mildly distended loops of small gut in upper abdomen with small air fluid levels. X-ray chest showed right hemi-diaphragm slightly higher in position. Routine blood investigations were carried out which showed deranged hepatic and renal function. Urine routine analysis showed proteinuria. CECT whole abdomen revealed abnormal wall of loop of small bowel likely mid jejunal bowel loop with intramural air and multiple air pockets in its mesentery which is extending to the right side and all along the mesenteric root and 2nd part of duodenum with obvious mesenteric fat plane stranding, thin rim of fluid in the pelvis with dilated proximal small bowel loops—S/O Bowel Ischemia and gangrene formation (Fig. 1). Following it, planned for surgical intervention. After pre-operative investigations, cardiac and anaesthesiology clearance was obtained. On priority, exploratory laparotomy was done

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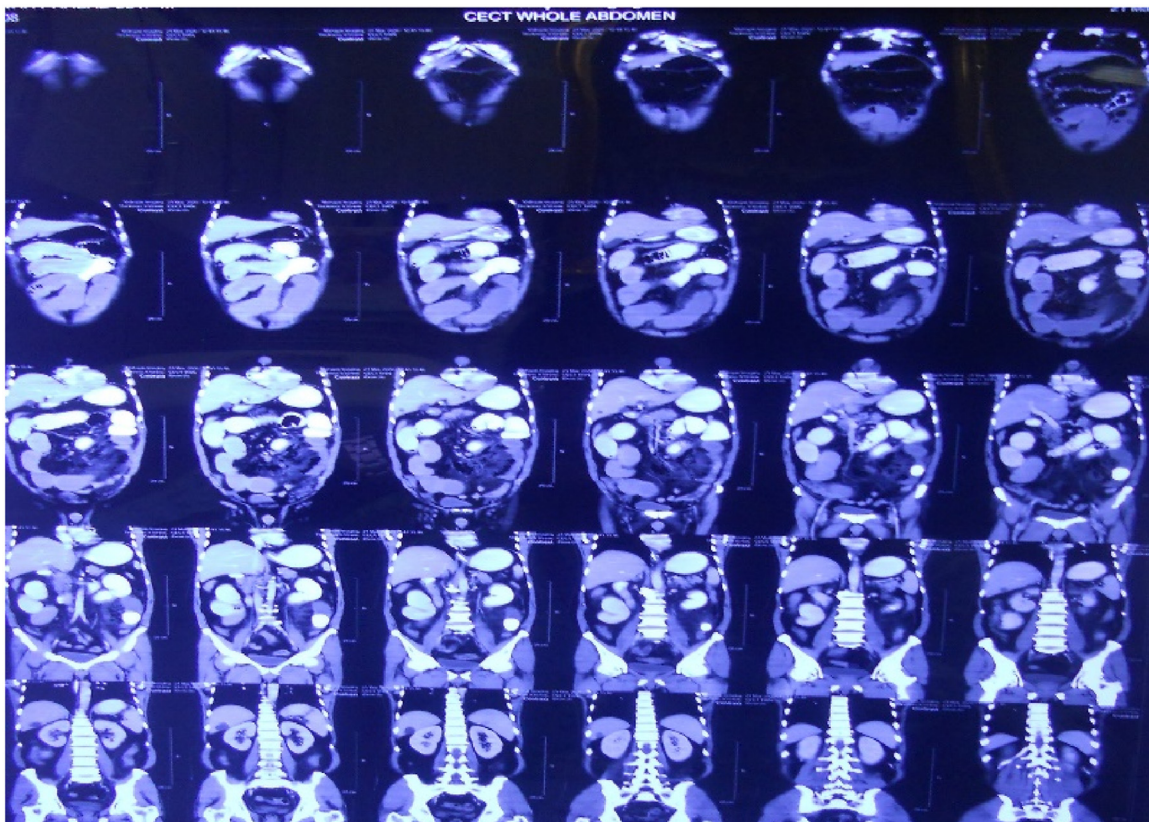


Fig. 1. Pre-operative CT Findings.

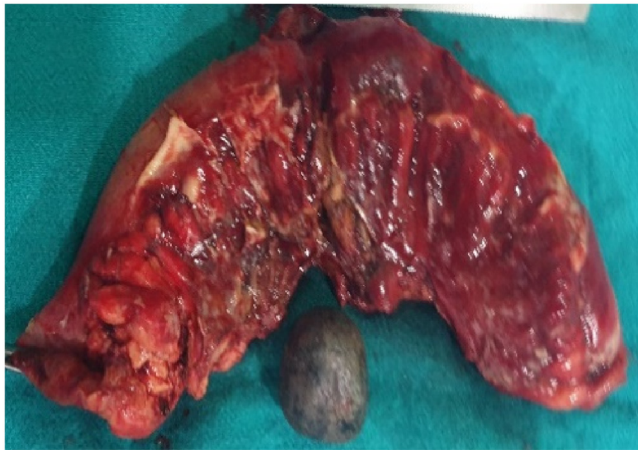


Fig. 2. Resected bowel and enterolith.

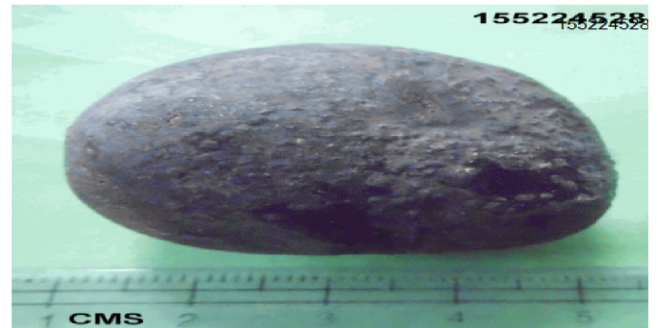


Fig. 3. Enterolith.

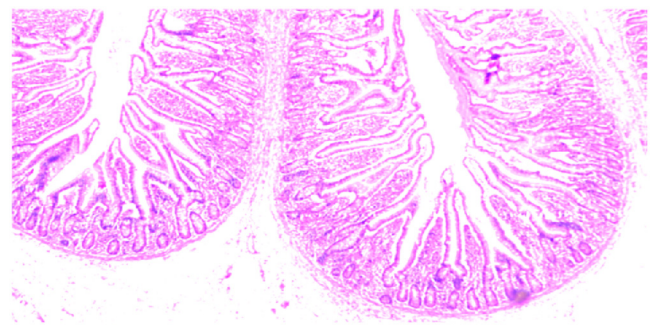


Fig. 4. Histopathological findings of resected bowel.

under general anaesthesia which revealed inflamed Gut and a perforation in small gut just one and half feet away from IC junction. An obstruction was identified by following the dilated bowel loops distally, palpation revealed a hard stone and enterotomy incision at the site delivered a stone (weighing approx. 40.696 gms) (Fig. 3). Thickened mesentery resection was done (Fig. 2) and then both the proximal and distal end of ileostomy brought out and closure done in layers after peritoneal lavage and drainage. Immediate post-operative period was uneventful. Histopathology (Fig. 4) revealed a segment of small intestine measuring 25.0 cm in length and 3.0 cm in width and attached fat measuring 2.5 cm, outer surface of specimen showed haemorrhage, congested covered with exudate and cut surface shows ulceration covered with exudate (1.5 cm from proximal end and 11.0 cm away from distal cut end). Biochemi-

cal analysis of stones revealed a combination of calcium oxalate monohydrate (70 %) and calcium oxalate dehydrate (30 %) which is suggestive of True primary enterolith in the Gut.

At the end of 4 months, patient was readmitted here for ileostomy closure which was done under general anaesthesia by giving encircling incision, loop was dissected and after being separated ends are cut and refashioned and then ileo-ileal anastomosis done. Post-operative recovery was uneventful and the patient was discharged on 7th post-operative day.

3. Discussion

Primary enteroliths which are formed in small bowel usually found within an abnormal pouch such as a diverticulum [10]. Primary enteroliths are subdivided into true and false subtypes: True enteroliths are made of substances found in chyme under normal alimentary conditions and false enteroliths are formed by insoluble foreign substances.

Enterolith formation is thought to be due to bowel hypomotility or stasis and anatomy of the intestinal tract also plays an integral role in enterolith formation and proposed hypothesis for the formation of enterolith is luminal pH and microenvironment specific to each segment of the gut, coupled with development of diverticular disease, altered endoluminal propagation and peristaltic functionality are important factors in developing conditions necessary for stone formation. Under physiologic conditions, established small intestinal pH variation is region specific, starting at about pH of 6 in the duodenum and progressively increasing to about pH 7.4 in the terminal ileum [9]. Choleic acid enteroliths that require lower pH are typically found in the proximal small intestine, largely affected by significant diverticular disease, strictures or stenosis. On the other hand, calcium phosphate, calcium oxalate, and calcium carbonate primary enteroliths are found in the distal small bowel. These salts are soluble in water and acidic environments, requiring an alkaline pH to precipitate and thus are most often formed in the terminal ileum [11–13].

The small bowel is able to tolerate a significant reduction in blood flow. However, when the ischemia is prolonged, it leads to disruption of the intestinal mucosa. Necrosis of the mucosal villi is the initial change, which usually happens within 3–4 h after intestinal ischemia. Following that, transmural, mural, or mucosa infarction can happen. Splenic flexure and rectosigmoid junction are the common susceptible area to hypoperfusion and hypoxia. Initially, in response to the injury, the intestinal wall becomes congested. It then appears edematous, friable, and hemorrhagic. Without intervention, bowel hemorrhage can occur within 1–4 days. Subsequently, enteric bacteria leads to intestinal gangrene and perforation [14,15].

Diagnostic laparoscopy is not widely accepted because it may miss areas of nonviable bowel. After initial resuscitation and stabilization of the patient, surgery is required for all patients who have evidence of threatened bowel and surgeon proceed with revascularization before resecting any intestine unless faced with an area of frank necrosis or perforation or peritoneal soilage. In such cases resection of the affected bowel without reanastomosis and containment of the spillage should be rapidly achieved before revascularization [16]. The surgical approach includes adhesiolysis and resection of non-viable intestine and the extension of intestinal resection depends on the purple or black discoloration of ischemic or necrotic bowel.

In this case, CECT whole abdomen revealed bowel ischemia with gangrene hence exploratory laparotomy with ileostomy is the preferred choice of surgery and during surgery found to have an enterolith in the gut and the biochemical analysis of the stone was done which was found to be composed of calcium oxalate monohydrate and calcium oxalate dehydrate.

4. Conclusion

- The most important prognostic factor in such cases relies on time interval between onset of symptoms and definitive treatment. By improving the awareness and early recognition of mesenteric ischemia will lead to improved survival in the condition.
- Enteroliths may occur in the absence of any bowel pathology.

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Ethical approval

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Consent

A written informed consent was obtained from the patient for publication of this case report and accompanying images.

Author contribution

Drafting of manuscript, proofreading. All authors read and approved the final manuscript.

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Declaration of Competing Interest

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References

- [1] O. Corcos, A. Nuzzo, Gastro-intestinal vascular emergencies, *Best Pract. Res. Clin. Gastroenterol.* 27 (October(5)) (2013) 709–725 [PubMed].
- [2] A. Roussel, Y. Castier, A. Nuzzo, Q. Pellenc, A. Sibert, Y. Panis, Y. Bouhnik, O. Corcos, Revascularization of acute mesenteric ischemia after creation of a dedicated multidisciplinary center, *J. Vasc. Surg.* 62 (November(5)) (2015) 1251–1256 [PubMed].
- [3] C. Ha, S. Magowan, N.A. Accortt, J. Chen, C.D. Stone, Risk of arterial thrombotic events in inflammatory bowel disease, *Am. J. Gastroenterol.* 104 (June (6)) (2009) 1445–1451 [PubMed].
- [4] J.F. Reinus, L.J. Brandt, S.J. Boley, Ischemic diseases of the bowel, *Gastroenterol. Clin. North Am.* 19 (June(2)) (1990) 319–343 [PubMed].
- [5] A. Patel, R.N. Kaleya, R.J. Sammartano, Pathophysiology of mesenteric ischemia, *Surg. Clin. North Am.* 72 (February(1)) (1992) 31–41 [PubMed].
- [6] J.D. Rosenblum, C.M. Boyle, L.B. Schwartz, The mesenteric circulation. *Anatomy and physiology, Surg. Clin. North Am.* 77 (April(2)) (1997) 289–306 [PubMed].
- [7] J.F. McKinsey, B.L. Gewertz, Acute mesenteric ischemia, *Surg. Clin. North Am.* 77 (April(2)) (1997) 307–318 [PubMed].
- [8] S. Sudharsanan, T.P. Elamurugan, C. Vijayakumar, K. Rajnish, S. Jagdish, An unusual cause of small bowel obstruction: a case report, *Cureus* 9 (3) (2017) e1116.
- [9] G.E. Gurvits, G. Lan, Enterolithiasis, *World J. Gastroenterol.* 20 (47) (2014) 17819–17829.
- [10] P.J. Klingler, M.H. Seelig, N.R. Floch, S.A. Branton, P.P. Metzger, Small-intestinal enteroliths—unusual cause of small-intestinal obstruction, *Dis. Colon Rectum* 42 (5) (1999) 676–679.
- [11] S. Acosta, Surgical management of peritonitis secondary to acute superior mesenteric artery occlusion, *World J. Gastroenterol.* 20 (August(29)) (2014) 9936–9941 [PubMed].

- [12] J. Fallingborg, Intraluminal pH of the human gastrointestinal tract, *Dan. Med. Bull.* 46 (1999) 183–196 [PubMed].
- [13] A. Tewari, J. Weiden, J.O. Johnson, Small-bowel obstruction associated with Crohn's enterolith, *Emerg. Radiol.* 20 (2013) 341–344 [PubMed].
- [14] E. Perez Nolan, R. Phillips Cherie, D. Webber John, C.B. Guzman, F.A. Saksouk, M.N. Ehrinpreis, Jejunal obstruction caused by a calcified enterolith, *Pract. Gastroenterol.* 30 (2006) 49–53.
- [15] F. Paterno, W.E. Longo, The etiology and pathogenesis of vascular disorders of the intestine, *Radiol. Clin. North Am.* 46 (September(5)) (2008) 877–885 [PubMed].
- [16] Carlo Vallicelli, Federico Coccolini, Fausto Catena, Luca Ansaloni, Giulia Montori, Salomone Di Saverio, Antonio D. Pinna, Small bowel emergency surgery: literature's review, *World J. Emerg. Surg.* 6 (2021), Article number: 1.

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