Case Report

A case of ileal enterolith secondary to eosinophilic enteritic stricture causing small bowel obstruction: a rare case report

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Received: 08 April 2024
Accepted: 13 May 2024

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ABSTRACT

Eosinophilic enteritis is an underdiagnosed and a rare inflammatory disease, that selectively affects the gastrointestinal tract with eosinophil rich inflammation in the absence of any known causes for eosinophilia. Enteroliths refer to stones formed in bowel due to severe stool stasis. They form around a nidus in layers which may contain calcification visible on radiographs. Here we present a case of a 65-year-old male with previous history of tuberculosis, who presented with transient small bowel obstruction with a large enterolith in distal ileum. He underwent exploratory laparotomy and was found to have an ileal stricture, secondary to eosinophilic enteritis. The post operative period was uneventful, and the patient was discharged on post operative day 8. Eosinophilic enteritis involves all layers of bowel wall and hence present with a wide range of symptoms from dyspepsia to bowel perforation. Stricture formation causing small bowel obstruction secondary to eosinophilic enteritis is rare. In this case report, stricture leads to stasis and formation of a fecolith, leading to small bowel obstruction. Eosinophilic enteritis needs to be considered as an important differential diagnosis for small bowel obstruction and small bowel strictures. In developing countries, as incidence of tuberculosis is high, it is common to wrongly diagnose a case of eosinophilic enteritis as tuberculosis. Thus, a strong clinical suspicion and awareness of this clinical entity are essential among surgical fraternity.

Keywords: Eosinophilic enteritis, Enterolith, Ileal stricture, Fecolith, Small bowel obstruction

INTRODUCTION

Eosinophilic enteritis is an uncommon disease of unknown etiology, which can affect any area of the gastrointestinal tract, from the oesophagus to the rectum, although the stomach and small bowel are most commonly involved.1 It usually presents as colicky abdominal pain, and rarely as an acute intestinal obstruction or perforation, making the initial diagnosis very difficult.2,3 The presentation depends upon the predominant layer involving eosinophilic infiltration, and hence classified as mucosal, muscularis, and subserosal types. Involvement of mucosa and muscularis layer may result in stricture causing bowel obstruction.4 Enteroliths are also a cause of intestinal obstruction but are rare. They occur proximal to stricture or in a diverticulum or a blind loop. Stasis is an important factor in their production.5 Enteroliths were first described by Pfahler and Stamm in 1915, however its association with eosinophilic enteritis has been reported infrequently as enterolithiasis in itself is rare.6 Here we present a case of a 65-year-old male with previous history of tuberculosis, who presented with transient small bowel obstruction with a large enterolith in distal ileum secondary to...
eosinophilic enteritic stricture, in a tertiary care hospital in Western Maharashtra.

**CASE REPORT**

A 63-year-old male, farmer by occupation, was brought to casualty with complaints of sudden onset generalised abdominal pain since the last 4 days associated with one episode of fever and 3 episodes of non-bilious non-blood-stained vomiting for the last 4 days. This was associated with one episode of loose stools 2 days ago followed by constipation and obstipation associated with abdomen distension. There was no history of trauma to abdomen, no dysuria, no cough, breathlessness, or chest pain. He had no history of similar complaints in the past. There was history of tuberculosis of abdomen 10 years ago with no available documentation currently but has taken antitubercular therapy for a duration of 6 months. He had no history of diabetes, hypertension, ischaemic heart disease or allergies. He had no prior surgeries. He denied history of taking any drugs. He had no history of drug allergy, asthma, or allergic rhinitis. There was no history of allergy or atopy in family members. He is a known tobacco consumer for the last 20 years.

On physical examination, patient was vitally stable. His abdomen was slightly distended with mild generalised tenderness but no guarding or rigidity. This was associated with exaggerated bowel sounds. There was no organomegaly. Other systemic examination was normal. Laboratory investigations showed Hb-12.4 gm/L and total leucocyte count 12.6×10⁹/L. Differential count revealed polymorphs-86%, lymphocytes-12% and eosinophils-2%. Absolute eosinophil count was 252. Serum calcium, electrolytes and uric acid were within normal limits. Stool and urine examination were within normal limit.

Chest X-ray was within limits. Abdominal X-ray showed a 3×3 cm opaque circular object suggestive of foreign body in large bowel. The patient however denied any history of ingestion of any foreign body. Colonoscopy done the following day was normal.

**Figure 1 (A and B): Abdominal X-ray showing dilated small bowel loops with enteroliths.**

USG (Abdomen and pelvis) showed dilated small bowel loops with a maximum diameter of 3.8 cm. Distal bowel loops appeared collapsed and showed to and fro peristalsis suggestive of small bowel obstruction. Diffuse circumferential wall thickening of ascending, transverse, and descending colon was present.

The patient passed flatus and stools the following day and abdominal pain subsided. Repeat USG (Abdomen and pelvis) revealed diffuse circumferential wall thickening of small bowel loops with minimal inter bowel free fluid. Small bowel showed sluggish peristalsis. No dilated bowel loops or foreign body was noted in distal ileum.

**Figure 2: USG abdomen and pelvis.**

Repeat x-ray erect abdomen did not show any dilated bowel loops or features of obstruction. However, the foreign body remained. The patient was clinically asymptomatic, tolerating orally and passing stools normally. However, since the foreign body was noted in situ, a CECT was done.

CECT report of abdomen and pelvis showed a 4.3×3.1 cm well defined hyperdense laminated solid lesion with central hypodensity with average HU 900 noted in ileal loop. It was abutting left lateral wall of bladder. There was no evidence of dilated bowel loops, pneumoperitoneum, fat stranding, nor enhancement on post contrast study. Findings were suggestive of foreign body in terminal ileum. Rest of small and large bowel loops were normal. There was no evidence of ascites, lymphadenopathy, pleural effusion, or pericardial effusion. However, post CT scan, the patient developed one episode of blood in stools while having no features of obstruction.
Figure 3 (A and B): CECT of abdomen and pelvis showing dilated small bowel loop with enteroliths.

Patient underwent exploratory laparotomy by lower midline vertical incision and bowel inspected. A stricture of 2 cm noted around 10 cm from ileocolic junction with proximal hard mass with dilated bowel loops and distal collapse of small bowel. An enterotomy incision taken over antimesenteric border over foreign body and mass was expelled out, only to find that it was an enterolith.

Finger couldn’t be passed through distal strictures which showed hypertrophy of wall and congestion of mucosa. Hence resection of stricture segment of around 4 cm done and sent for histopathological examination. Anastomosis was done. Multiple edematous patches were noted along bowel wall in the ileum.

Gross examination revealed an ileal segment with normal serosa but thickened mucosa with focal dark brown areas. Microscopic exam revealed infiltration of eosinophils (60/hpf), few lymphocytes and plasma cells diffusely in lamina propria, submucosa and muscular mucosa. Serosa was covered with fibrous tissue, blood vessels and similar infiltrate, suggestive of organizing peritonitis. Stone analysis revealed ammonium urate and uric acid as its composition. Postop period was uneventful, and the patient was discharged on post operative day 8. Patient was followed up after 6 months and was asymptomatic.

Figure 4 (A-E): Clinical pictures.

Figure 5 (A-C): Enterolith size comparison to a scale.

Figure 6: On breaking the enterolith.

Figure 7 (A-D): Histopathology of eosinophilic infiltrates in ileal mucosa and lamina propria.

DISCUSSION

Eosinophilic gastrointestinal (EGE) disease was first described by Kaijser in 1937. In India, Venkataraman et
al have reported seven cases of EGE over a ten-year period.8

Eosinophilic enteritis can be asymptomatic or symptomatic. The clinical presentations of eosinophilic enteritis depend on the level of involvement. The mucosal form is the most common form and presents with vomiting, diarrhea, abdominal pain, anemia, and protein loosing enteropathy. Involvement of the muscularis is characterized by eosinophilic infiltration mainly in the muscular layer, leading to thickening of the bowel wall, resulting in obstructive symptoms. The serosal involvement occurs only in a minority of the patients and is characterized by exudative ascites with higher peripheral eosinophilic counts as compared to other forms.5,6 However, in our case, there was predominant muscular involvement and no peripheral eosinophilia.

CT scan may show nodular and irregular thickening of the foregut which may mimic other conditions like Crohn’s disease, tuberculosis or lymphoma. Since either layer of the GI tract can be involved, endoscopic biopsy can be normal in patients with the muscularis subtype, serosal subtype, or both. Despite all the clinical features, diagnostic criteria can be made based on presence of increased eosinophils in biopsy specimens from the GI tract wall, the infiltration of eosinophils within intestinal crypts and gastric glands, the lack of involvement of other organs, and the exclusion of other causes of eosinophilia. The histological characteristic of eosinophilia is edema and an inflammatory cell infiltrate composed of eosinophils, which may appear in clumps.9

Diagnostic criteria suggest an eosinophil load of ≥56 eos/hpf in ileum, ≥52 eos/hpf in duodenum to diagnose eosinophilic enteritis and accordingly fulfils the criteria in our case.10 All other causes of tissue eosinophilia were also ruled out.

Enteroliths refer to stones formed in bowel due to severe stool stasis. It may be primary or secondary in nature. Primary enteroliths are formed in the small bowel and secondary enteroliths are formed in gallbladder as gall stones.5 True enteroliths of the small intestine are of three main types: (1) those consisting mainly of bile acids; (2) those consisting mainly of calcium oxalate; and (3) those consisting mainly of phosphate. Bile acid enteroliths are made up mainly of choleic acid.6 In general, enteroliths rarely form within the GI tract, except in certain pathologic conditions like Crohn's disease or blind loop syndrome.

Wide-spread use of acid suppression, improving surgical techniques, conservative management of chronic intestinal conditions and dietary consumption of calcium products may alter conventional norms of traditional enterolith formation; therefore, the true incidence and prevalence of primary enteroliths remains to be determined.1 In humans, enteroliths are rare and may be difficult to distinguish from gall stones. Most enteroliths are inapparent and cause no complications. However, any complications that do occur are likely to be severe. Of these, bowel obstruction is most common, followed by ileus and perforation.11

Gamblin et al and Jones and McWhirter reported cases of enterolith formation in Meckles diverticulum.12,13 Sureka et al reported an interesting case of a radiopaque shadow in the pelvic region which was thought to be a vesical calculus but on further investigation was diagnosed as an enterolith in the ileal loop.14 In Crohn’s disease, multiple areas of small bowel stenosis are relatively common, but there are only few reported cases with stenosis complicated by enterolith. Geoghegan et al reported a case of small bowel obstruction secondary to a giant enterolith in a patient of Crohn’s disease.15 Svanes and Halvorsen reported cases of enteroliths in jejunal diverticula.16 However, most enteroliths are in apparent and cause no complications but sometimes may present with complications like intestinal obstruction, ileus, or perforation. Klinger et al in their article suggested that first therapeutic approach should be nonsurgical, and surgery should be considered only if obstruction persists. Surgical management commonly involves enterotomy or occasionally resection.17

As stated, enterolith usually forms in a diverticulum of small bowel causing small bowel obstruction.13 Decreased peristalsis is an important factor pre-disposing to the development of an enterolith.12 Stasis in the distal small bowel may occur in a variety of clinical conditions, including structuring Crohn’s disease, post-surgical anatomical alterations, post-radiation enteritis, gastrointestinal tuberculosis, congenital or acquired ileo-jejunal diverticular disease while our patient had a history of hypothyroidism with ongoing treatment. Miscellaneous causes of small bowel obstruction account for about 2-3% of all cases and must be considered among the differentials. Since there was no small bowel diverticulum, small bowel tumour, Crohn’s disease, intestinal tuberculosis, or history of previous surgeries in our case, we hypothesise, that the enterolith is due to hypo-motility or stasis.5

Although known to cause intestinal obstruction, association of enteroliths and ileal stricture with eosinophilic enteritis has only been reported once so far in the literature, to the best of our knowledge. Eosinophilic enteritis is a diagnostic dilemma, and the clinical presentation and investigations are only contributory. Surgeons must be aware of this rare cause of acute abdomen.

CONCLUSION

Eosinophilic enteritis even though a rare disorder has a varied spectrum of presentation which is easily misinterpreted as neoplasm, tuberculosis, and inflammatory bowel disease. Hence, eosinophilic enteritis needs to be considered as an important differential
diagnosis for small bowel obstruction and small bowel strictures. Since it can be easily treated medically and surgically it has to be diagnosed with certainty. In developing countries, as incidence of Tuberculosis is high it is common to wrongly diagnose case of eosinophilic enteritis as tuberculosis. Thus, a strong clinical suspicion and awareness of this clinical entity are essential among surgical fraternity.

**Funding:** No funding sources  
**Conflict of interest:** None declared  
**Ethical approval:** Not required

**REFERENCES**


Cite this article as: Winford ME, Bhosle D, Momin YA. A case of ileal enterolith secondary to eosinophilic enteritic stricture causing small bowel obstruction-a rare case report. Int Surg J 2024;11:1011-5.