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Case Report

Isolated ileal tuberculosis presenting as massive lower gastrointestinal bleeding: rare case report

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ABSTRACT

Intestinal tuberculosis (TB) is a common form of extrapulmonary (EPTB) in developing countries. It usually presents with obstruction, perforation, or malabsorption. Presentation as massive lower gastrointestinal (LGI) bleeding is extremely rare. A 65-year-old male, presented with sudden onset of massive bleeding per rectum associated with haemorrhagic shock. He required multiple Blood products transfusion. Contrast-enhanced CT abdomen showed distal ileal wall thickening with intraluminal contrast extravasation, suggestive of a vascular malformation. Laparotomy showed multiple areas of congestion and ulceration noted in distal ileum with active bleed intraluminally, associated enlarged mesenteric lymph nodes present. Limited ileocecal resection was performed. Histopathological examination revealed granulomatous lesion-Koch's etiology. Isolated lleal TB, rarely presents as massive lower GI bleeding. Early surgical intervention plays a vital role in both diagnosis and management, especially when the patient presents with hemodynamic instability

Keywords: Ileal tuberculosis, Massive lower GI bleed, Haemorrhagic shock, Ileocecal resection

INTRODUCTION

Tuberculosis (TB) remains a major global health problem, particularly in developing nations such as India.[1] Of the total TB cases, 15-20% are EPTB, and among EPTB, abdominal TB accounts for approximately 10-15%, Within abdominal TB, the ileocecal region is most frequently involved (35-50%), owing to its rich lymphoid tissue and the relative stasis of intestinal contents that favor Mycobacterium TB infection. Clinical manifestations of intestinal TB vary and include abdominal pain, altered bowel habits, intestinal obstruction, perforation, and constitutional symptoms.[2,3] Hematochezia, which is a rare presentation of GIT TB, only seen in 7-20% of cases.[2,3] The cause of bleeding is postulated to be due to endarteritis-granulomatous inflammation caused by the bacilli but is usually mild or occult. Presentation as massive LGI haemorrhage is exceedingly rare, with fewer than 12 cases reported in literature.[4-7] We report a case of ileal TB presenting as massive lower GI bleed requiring emergency surgery, highlighting the diagnostic dilemma and management considerations.

CASE REPORT

A 65-year-old male was admitted with sudden onset of copious bleeding per rectum for three days. There was no associated abdominal pain, diarrhea, vomiting, or tenesmus. He was a known case of type 2 diabetes mellitus on insulin for 2 years and had a history of chronic alcohol consumption for more than 30 years. On examination, the patient was drowsy, pale, cold and clammy extremities and dehydrated. His heart rate was 143/min, blood pressure 60/40 mmHg, and SpO_2 is not recordable. The abdomen was soft and non-tender with normal bowel sounds. Per rectal examination revealed fresh blood with clots. The patient was intubated due to his drowsy state and poor respiratory effort. He was hemodynamically unstable with a hemoglobin level of 3

g/dL, and hence, massive transfusion protocol was initiated. The patient received 6 units of packed red blood cells, 4 units of fresh frozen plasma, and 4 units of platelets during resuscitation.

Given the massive LGI bleed, initial differential diagnoses included colonic angiodysplasia and diverticular bleeding, considering the patient's age and presentation. After initial stabilization, CECT abdomen and pelvis (Figure 1 A and B) was performed, which revealed distal ileal wall thickening with intraluminal contrast extravasation noted in arterial phase with progressive increase in venous phase, suggestive of a possible vascular malformation. There is no evidence of any thickening noted in the rectum and other large bowel loops. No obvious evidence of abdominal TB was noted on imaging.

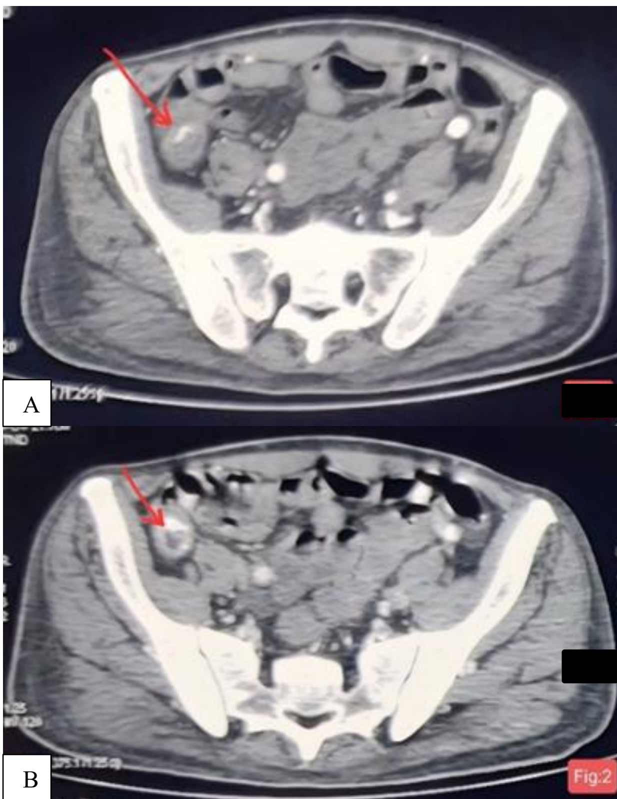

Figure 1 (A and B): The arrow indicates a focally thickened segment of the distal ileum with active contrast extravasation, consistent with ongoing haemorrhage.

As part of the planned workup, colonoscopy and angiographic embolization were considered. Owing to persistent bleeding and hemodynamic instability, angiographic embolization and colonoscopy could not be performed, and the patient was taken up for emergency exploratory laparotomy.

Intraoperatively, there were multiple areas of congestion (Figure 2) and ulceration (Figure 3) noted in distal ileum with active bleed and clots intraluminally, the mesenteric lymph nodes were also enlarged. A limited ileocecal resection with end ileostomy was performed.

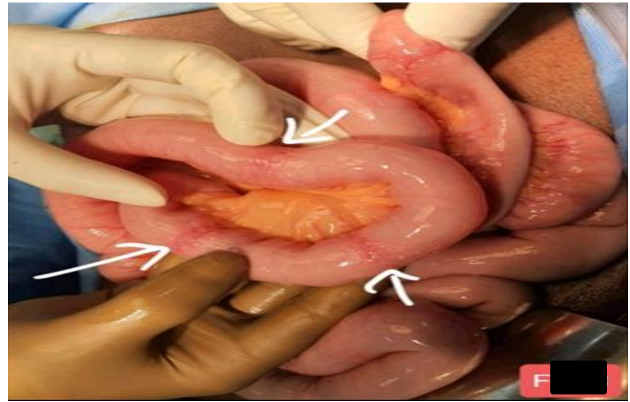

Figure 2: The arrows highlight multiple congested segments of the small bowel, suggestive of localized vascular engorgement.

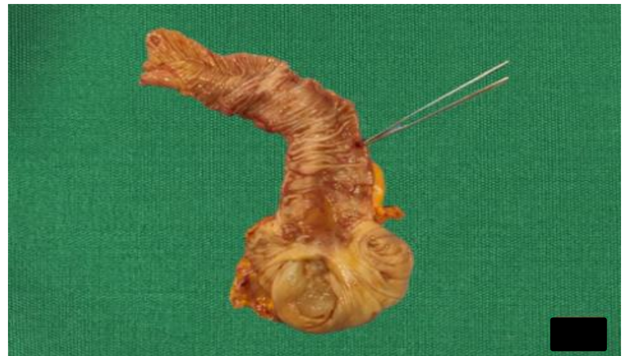

Figure 3: The indicated area demonstrates a mucosal ulcer, identified as the causative source of the massive gastrointestinal haemorrhage.

Histopathological examination of the resected specimen revealed ileal mucosa (Figure 4) with ulceration, lamina propria and submucosa shows numerous granulomas. Serosa shows congestion, confirming intestinal TB as the cause of bleeding.

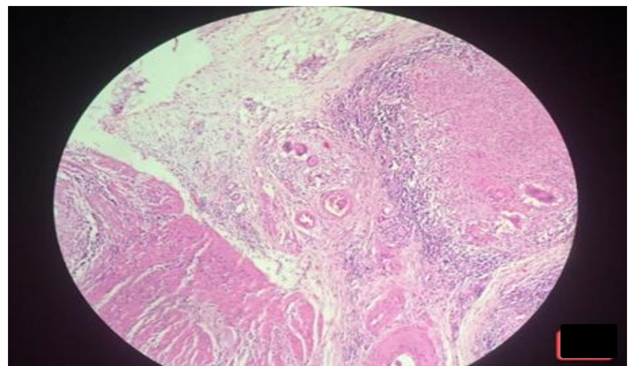

Figure 4: Section shows ulcerated ileal mucosa.

*The underlying lamina propria and submucosa reveal multiple granulomas consisting of epithelioid histiocytes, lymphocytes and characteristic Langhans-type multinucleated gaint cells.

The resected node (Figure 5) showed evidence of granulomatous lymphadenitis. Chest medicine consultation was obtained, and was advised to initiate anti-tubercular therapy (ATT) once the patient got stabilized.

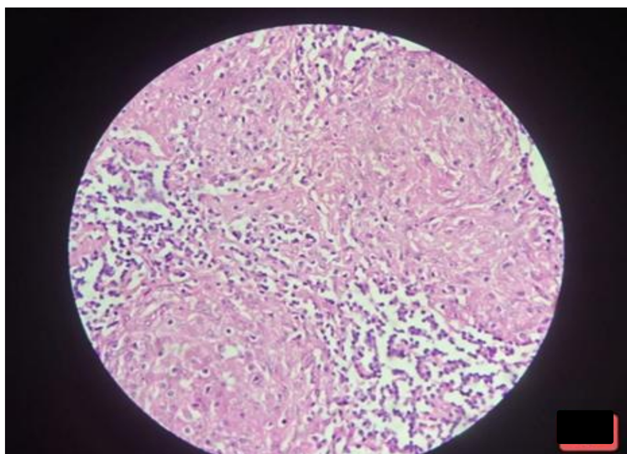

Figure 5: Section shows normal nodal architecture is entirely effaced, with replacement by caseous necrosis and granulomatous inflammation.

Postoperative course

Postoperatively, the patient had no further episodes of bleeding, and his vitals remained stable initially. However, he continued to have poor respiratory efforts and remained intubated. Over the next few days, his condition gradually deteriorated, and hypoxic-ischemic injury secondary to severe anemia and shock was suspected. Despite continued intensive care and supportive management, the patient expired on postoperative day seven due to multi-organ dysfunction likely secondary to hypoxic-ischemic encephalopathy.

DISCUSSION

Massive LGI bleeding is an uncommon but potentially life-threatening surgical emergency. The most frequent causes in the elderly include diverticulosis, colonic angiodysplasia and malignancy, whereas infective etiologies such as TB are exceedingly rare. Intestinal TB most commonly involves the ileocecal region, but its presentation as acute massive bleeding is unusual, posing a diagnostic and therapeutic challenge.[2,3] Bleeding as a presentation occurs in 7-20% of cases, and massive bleeding requiring surgical intervention is much rarer.[2,3]

The pathology attributed with bleed is due to ulceration and erosion of mesenteric vessels secondary to endarteritis-granulomatous inflammation. Radiological diagnosis in such cases is often difficult. However, in this case, CECT abdomen and pelvis revealed ileal wall thickening with contrast extravasation, suggestive of a vascular lesion, which led to an initial misdiagnosis. This underscores the diagnostic dilemma, as intestinal TB can mimic vascular malformations.

Colonoscopic evaluation and angiography are valuable diagnostic and therapeutic tools for LGI bleed; however, in this case, these could not be performed due to hemodynamic instability and ongoing active bleeding. Emergency laparotomy remains a lifesaving option. Despite prompt resuscitation and surgical management, the patient's postoperative course was complicated by hypoxic-ischemic encephalopathy secondary to profound anemia and shock, leading to mortality on postoperative day seven. This highlights the importance of early recognition, aggressive resuscitation, and timely surgical intervention in managing such catastrophic bleeds.

A review of published cases shows fewer than 12 reports of ileal TB presenting as massive lower GI bleed.[4-7] In all reports, the final diagnosis was made postoperatively by histopathology. Early recognition and surgical management can prevent mortality in such presentations. The role of ATT remains crucial in postoperative management. Early initiation of ATT after stabilization is essential to prevent recurrence and systemic progression. In this case, ATT was planned but the patient succumbed before therapy could be commenced.

CONCLUSION

Massive LGI bleeding secondary to intestinal tuberculosis is rare but potentially fatal. It should be considered in the differential diagnosis of obscure or recurrent LGI bleeds, especially in tuberculosis-endemic regions. Radiological findings may be non-specific and can mimic other vascular or inflammatory conditions. When conservative and interventional approaches fail or are not feasible due to instability, timely surgical exploration is critical for diagnosis and control of bleeding.

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