Middle East J Dig Dis. July 2023;15(3):207-209

doi 10.34172/mejdd.2023.346 http://mejdd.org

Middle East Journal of Digestive Diseases MEJDD

Case Report

A Case of Massive Gastrointestinal Bleeding Due to Abdominal Tuberculosis

Sai Krishna Reddy Bana[1], Suja Lakshmanan[1], Vaasanthi Rajendran[1*], Senthil N[1], Archa Anna Anil[1], Nanthakumar L[1]

[1]Department of General Medicine, Sri Ramachandra Medical College and Research Institute, Chennai, India

Abstract

This case is about a complication of abdominal tuberculosis in the form of a massive lower gastrointestinal (GI) bleed, which was timely intervened by angioembolization. A young man in his mid-20s on empirical anti-tubercular therapy (ATT) for abdominal tuberculosis, presented with severe abdominal pain. The patient then developed frank per rectal bleeding, leading to a significant drop in hemoglobin level, requiring multiple blood transfusions. Upper GI endoscopy and colonoscopy findings were inconclusive. Contrast-enhanced computed tomography (CECT) of the abdomen was performed, which revealed a contrast extravasation into the jejunum due to a leak in the jejunal branch of the superior mesenteric artery (SMA), followed by selective SMA angiography (digital subtraction angiography), which was arrested by angioembolization. The patient had multiple abdominal lymphadenopathies with omental nodules. Histopathological examination of the omental nodules revealed epithelioid granuloma with Langerhans-type cells. The patient is currently receiving ATT and is doing well.

Keywords: GI bleed, Tuberculosis, Angioembolization

Cite this article as: Reddy Bana SK, Lakshmanan S, Rajendran V, Senthil N, Anna Anil A, Nanthakumar L. A case of massive gastrointestinal bleeding due to abdominal tuberculosis. *Middle East J Dig Dis* 2023;15(3):207-209. doi: 10.34172/mejdd.2023.346.

Received: March 31, 2023, **Accepted:** June 22, 2023, **ePublished:** July 30, 2023

Case Report

A young man in his mid-20s, who was recently diagnosed as having abdominal tuberculosis based on clinicoradiological findings and was started on empirical anti-tubercular therapy (ATT) 2 weeks prior to this current admission at our hospital, was referred to us. He had no other known comorbidities. The patient presented to the emergency department with abdominal pain, nausea, vomiting, and giddiness for 2 days. On examination, the patient was conscious, oriented, and pale. Pulse rate was 136/min, blood pressure (BP) was 90/60 mm Hg, and abdominal examination showed tenderness over the epigastrium and right hypochondrium. Complete blood count revealed Hb 9 g/dL, total count-3600, and platelets 1.7 lakh. Subsequently after admission, he had an episode of frank hematochezia with a hemoglobin drop to 6 g/dL along with increasing serum lactate levels. Two units of packed red blood cells (RBCs) were transfused, and pantoprazole and somatostatin infusions were started. Esophagogastroduodenoscopy (OGD) and a colonoscopy were done. OGD showed gastric erosions. Colonoscopy was attempted but could not be done as the vision of the scope was obscured by oncoming blood and blood products. Bleeding per rectum continued, and serial hemoglobin drop was noted. Hence further packed RBC transfusions were given. Contrast-enhanced computed tomography (CECT) of the abdomen showed contrast extravasation in the distal jejunal loop from the Superior mesenteric artery with hyper-dense blood products in the distal jejunum, ileum, and large bowel (Figure 1). Omental caking was present with smooth thickening of the peritoneum and mesentery. Mild fluid in the perianal compartment and posterior compartment with perisplenic, peripancreatic, paraaortic, aortocaval, and external iliac lymphadenopathy was seen. Selective superior mesenteric artery (SMA) angiogram established contrast extravasation from the second jejunal branch of SMA at the vasa recta. Angioembolization was done using 33% glue. Hemoglobin was monitored post-procedure, and the patient did not have further bleeding. The patient was shifted to the medical ward from intensive care. A repeat colonoscopy was performed, which did not show any mucosal lesions. For confirming the TB diagnosis, a USG-guided omental biopsy was done, which revealed epithelioid granuloma (Figure 2) with Langerhans type of giant cells (Figure 3) surrounded by lymphocytes with granulomatous inflammation suggestive of Koch's etiology. However, HIV-1 and 2 were negative.

The patient was restarted on ATT and was discharged. He is currently on regular follow-up without any further complications.

Discussion

This young man was diagnosed as having abdominal tuberculosis at a private hospital based on clinico-radiological findings. He had a 6-month history of weight loss, loss of appetite, and night sweats, for which he was evaluated further. He underwent chest radiography and

*Corresponding Author: Vaasanthi Rajendran, Email: vaasanthi_r@yahoo.co.in

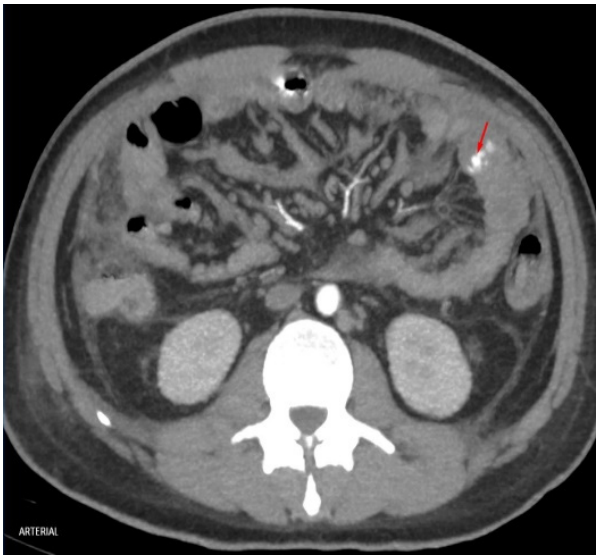

Figure 1. Showing active contrast leak [red arrow] from SMA branch into the distal jejunum

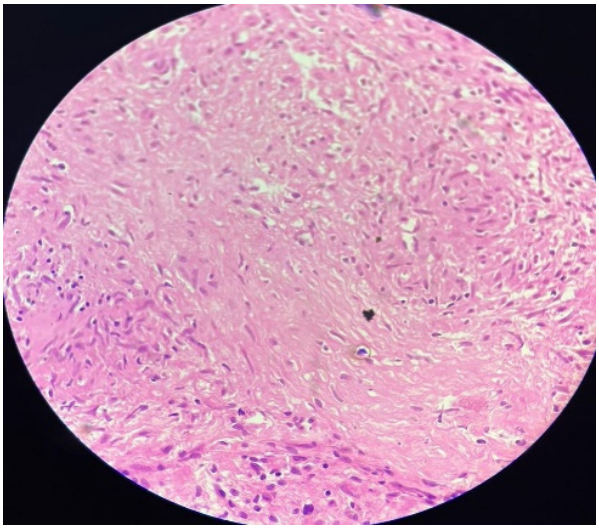

Figure 2. Showing epithelioid granuloma from the omental biopsy

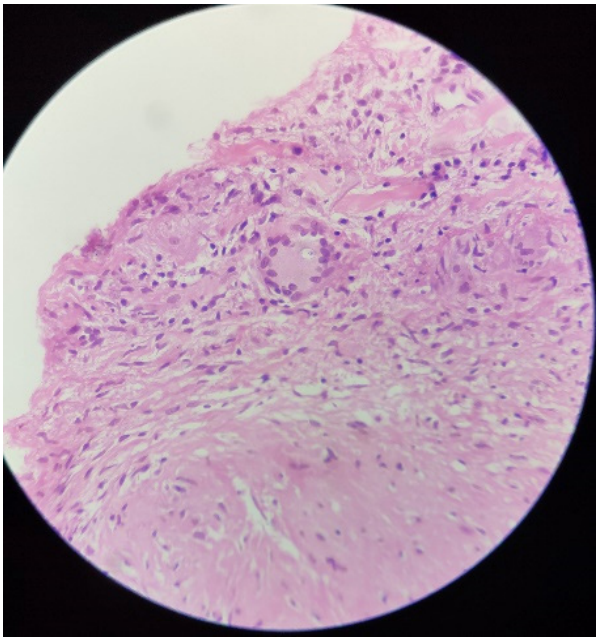

Figure 3. Showing Langerhans type of giant cell from the omental biopsy

CECT of the abdomen. The chest radiograph did not show any abnormality. CECT of the abdomen showed rectal and jejunal thickening with mesenteric lymphadenopathy. Hence he was empirically started on ATT with rifampicin (R), isoniazid (H), pyrazinamide (Z), and ethambutol (E). He continued ATT for 2 weeks. But the dose at which he was started was sub-optimal.

Intestinal TB accounts for 1% to 3% of TB around the globe. Among extrapulmonary TB, intestinal involvement is less common than the involvement of other sites. HIV infection, prior glucocorticoid therapy or anti-TNF agents use, and development of lymphoma are the risk factors for developing intestinal TB. Both *Mycobacterium tuberculosis* and *Mycobacterium bovis* are responsible for causing tuberculous enteritis. Route of disease causation is direct penetration of the gut mucosa by the ingested organisms. Alternatively direct spread from adjacent organs and miliary spread can also occur. TB can affect any site or multiple sites of the gastrointestinal (GI) tract, although the ileum and cecum are the most common sites of GI involvement (around 75%). In TB enteritis, usually, both sides of the ileocecal valve are affected, leading to the incompetence of the valve, a finding which helps distinguish TB from Crohn's disease. The gross appearance of intestinal TB is divided into three categories[1]: ulcerative lesions (more common),[2] hypertrophic lesions that can mimic carcinoma[3]; and ulcero-hypertrophic lesions where mucosal ulcerations are seen along with healing and scar formation. Healing of ulcers results in fibrosis, causing the formation of strictures. Histologically, the characteristic lesion is the formation of granuloma, with or without caseation. TB bacilli are identified with an acid-fast stain or by polymerase chain reaction (PCR). PCR is more sensitive, around 65%, and can be performed on biopsied tissue or on stool. Complications include intestinal hemorrhage, obstruction, fistula formation, perforation, and malabsorption.

In the review of the literature, only very few cases of abdominal tuberculosis presented with massive lower GI bleed, and those were most commonly from the ileocecal region.[1,2] Few cases had bleeding from the transverse[3,4] and descending colon.[5] In our patient, there was a massive lower GI bleed. Upper and lower GI endoscopic evaluation was normal. CECT of the abdomen showed active bleeding from a small bowel which was confirmed by digital subtraction angiography and managed by timely angioembolization. According to a study done by Lv and Gu, selective arterial embolization has been shown to arrest bleeding successfully in all 26 patients who underwent this procedure.[6] With this approach of selective angioembolization, the significant risk of bowel ischemia can be averted, and the need for major exploratory laparotomy may be brought down.

Intestinal TB is treated similarly to pulmonary TB but for a longer duration. The regimen we follow for extrapulmonary TB is based on WHO recommendations. The regimen includes two months intensive phase and

four months continuation phase, which can be extended as per the treating clinician's discretion (2HRZE + 4HRE). Usually, clinical response to ATT often occurs in two weeks, although the ileocecal masses, strictures, and stenosis may respond more slowly. In this patient, suboptimal dosing might be the reason for developing complications despite therapy. A surgical approach occasionally is still required for obstructive disease or when intestinal perforation or hemorrhage occur.

Conclusion

The complication of a tubercular abdomen with massive GI bleed is rare and highly fatal if not addressed by early intervention.

Competing Interests

The authors declare no conflict of interest related to this work.

Ethical Approval

Written informed consent was obtained from the patient.

References

1. Goudarzi HA, Mason LB. Fatal rectal bleeding due to tuberculosis of the cecum. *JAMA* 1982;247(5):667-8. doi: 10.1001/jama.1982.03320300071031
2. Monkemuller KE, Lewis JB Jr. Massive rectal bleeding from colonic tuberculosis. *Am J Gastroenterol* 1996;91(7):1439-41.
3. Verma P, Kapur BM. Massive rectal bleeding due to intestinal tuberculosis. *Am J Gastroenterol* 1979;71(2):217-9.
4. Tishler JM. Tuberculosis of the transverse colon. *AJR Am J Roentgenol* 1979;133(2):229-32. doi: 10.2214/ajr.133.2.229
5. al Karawi MA, Mohamed AE, Yasawy MI, Graham DY, Shariq S, Ahmed AM, et al. Protean manifestation of gastrointestinal tuberculosis: report on 130 patients. *J Clin Gastroenterol* 1995;20(3):225-32. doi: 10.1097/00004836-199504000-00013
6. Lv LS, Gu JT. Super-selective arterial embolization in the control of acute lower gastrointestinal hemorrhage. *World J Clin Cases* 2019;7(22):3728-33. doi: 10.12998/wjcc.v7.i22.3728