

Case Report

A rare complication of the great mimicker: free tubercular ileal perforation

S. P. Gayathre, Kudiyarasu, R. Kannan, N. Shajini*

Institute of General Surgery, Madras Medical College, Rajiv Gandhi Government General Hospital, Chennai, Tamil Nadu, India

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*Correspondence:

Dr. N. Shajini,

E-mail: shajini222@gmail.com

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ABSTRACT

Primary intestinal tuberculosis is an uncommon form of extra-pulmonary tuberculosis. With non-specific manifestations, unusual presentations and no proven superior diagnostic modality, differentiation and diagnosis of abdominal tuberculosis is a difficult scenario. Undiagnosed, untreated cases may present with complications. Free perforation of small bowel due to tuberculosis is a rare complication (1-2%) because of adhesion formation and reactive peritoneal thickening; however, when it occurs is most feared for its association with high mortality. We report a case of an elderly male who presented with features of intestinal perforation, treated with emergency surgery and diversion ostomy for small bowel perforation which was diagnosed post-operatively to be of tubercular etiology. Acute care surgeons may be trapped in diagnosing such cases with its non-specific imaging findings, and hence must maintain a low threshold for surgical intervention in the emergency setting.

Keywords: Abdominal tuberculosis, Ghon's complex, Ileocaecal region, Perforation, Mesenteric vasculitis

INTRODUCTION

Tuberculosis (TB), is a disease of global concern that has haunted mankind for centuries. It is still a nightmare to the medical community, more so with the emergence of multidrug resistant strains. 20% cases with pulmonary tuberculosis develop extra-pulmonary disease, of which abdominal involvement is seen in 10%.¹ While the abdomen is not spared from the clutches of this dreaded disease; with its protean manifestations, abdominal tuberculosis (AbT) is a great GI masquerader. Perforation is a rare complication of intestinal tuberculosis, seen in 4.9% cases.² Free tubercular perforation however is seen in only 1-2%.² With the diagnostic difficulties and the high mortality associated with the condition, we report a case of an elderly male who presented with free tubercular ileal perforation.

CASE REPORT

A 50-year-old male, a daily wage labourer, who is a smoker, presented with the chief complaint of abdominal pain for 1 day. Pain was sudden in onset, initially periumbilical and later diffuse, aggravated with movement. There was no history of fever, vomiting or obstipation. On examination, he was poorly built and nourished. His vital signs were stable. His abdomen was distended, with umbilicus flushed to surface, tense with diffuse tenderness and guarding without rigidity. There was obliteration of liver dullness and bowel sounds were absent. Digital rectal examination showed watery, yellowish fecal staining. Laboratory investigation revealed anaemia (hemoglobin - 8.2 g/dl), leucocytosis of 18,600 cells/cu.mm with predominant neutrophils (95.9%), hypoalbuminemia (2.2 g/dl). Non-contrast Computed

tomography of abdomen revealed pneumoperitoneum. Computed tomography of chest (for COVID screening) revealed features suggestive of active tubercular infection. With a provisional diagnosis of hollow viscus perforation, after adequate resuscitation, patient was taken up for emergency exploratory celiotomy. Intraoperatively, approximately 300 ml of seropurulent fluid was evacuated. There was extensive inflammatory flakes and bowel oedema. Two perforations were noted on the anti-mesenteric border of ileum, 70 cm from Ileo-caecal junction, each measuring 1×1 cm, 5 cm apart. There were no tubercles nor a distal stricturous segment. Approximately 7 cm of ileal segment was resected and double barrel ileostomy was fashioned in left iliac fossa. Post-operatively, on further evaluation, sputum Acid Fast Bacilli (AFB) staining was negative. However, sputum CBNAAT detected rifampicin sensitive *Mycobacterium tuberculosis* (MTB) and peritoneal fluid analysis was also AFB positive; HIV screening was negative. Histopathology of resected specimen showed ileal mucosa with transmural ulceration. Lamina propria showed epithelioid granulomas with Langhan's giant cells, epithelioid histiocytes and lymphocytes; serosa showed necro-inflammatory exudate, suggestive of granulomatous lesion of tuberculous etiology. He was started on anti-tuberculous drugs.

DISCUSSION

AbT (10%) is the sixth most common type of extra-pulmonary TB next only to lymphatic (58.7%), genitourinary, bone & joint, miliary and meningeal.³ In India, the disease burden amounts to 1.3 per 100 admitted patients.⁴ Abdominal TB with co-existent pulmonary TB is found in 20–33.95% patients.⁵ The average age of occurrence is 21–45 years, with a slight male predominance (1.4:1). AbT is a great mimicker with protean manifestations – abdominal pain (61%), anorexia (98%), fever (88%), weight loss, ascites.^{3,4} The various postulated modes of spread resulting in AbT are: hematogenous spread from active pulmonary tuberculous foci, ingestion of infected sputum, lymphatic route from involved lymph nodes, direct spread as from contiguous site and tubercular salpingitis (in females).⁶ With the theory that states the spread resulting from *Mycobacterium Bovis* due to raw milk consumption no longer accepted, the following route of involvement has been put forward in cases of AbT without active lung lesion: 1) aerosol inhalation, 2) pulmonary Ghon's complex (Ghon's focus with regional hilar lymphadenopathy favourable host status, 3) healed lesion (harbours dormant bacilli), 4) reactivation of latent infection (waning of host immunity) and 5) hematogenous/lymphatic spread of dormant bacilli abdominal tuberculosis.⁷

AbT can be peritoneal (most common), intestinal, tubercular lymphadenopathy or visceral. The site of predilection of intestinal TB is ileocaecal region, often manifesting as strictures. This is due to zone of physiologic stasis, minimal digestive activity, increased peyer's patches, higher absorption of fluid and electrolytes.⁸ There

are 3 forms of intestinal TB: (1) Ulcerative: here multiple, superficial ulcers restricted to the epithelial surface are seen; (2) Hypertrophic: Characterised by submucosal and subserosal fibroblast proliferation which thickens bowel wall, eventually resulting in scarring & fibrosis; (3) Ulcerohypertrophic.⁹

The most common complication observed in cases of intestinal TB is bowel obstruction (31.7%) secondary to strictures or adhesions. Intestinal perforation (proximal to a stricture segment from pressure necrosis due to distension) is seen in 4.9%. The others are enterocutaneous fistulas (2.4%) and small bowel volvulus (2.4%) due to mesenteric lymphadenitis.²

However, free tubercular intestinal perforation without distal obstruction is even rare observed in only 1-2%. It is often a complication of severe, untreated disease. It still can occur during anti-tuberculous therapy. It is common in the terminal ileum and is often solitary (90%) and multiple perforations as in our patient is seen in 10-40%.¹⁰ It is associated with high mortality rates nearing 30%.¹¹ The basic pathophysiology behind is that the hematogenous spread of bacilli incites a granulomatous inflammation of mesenteric vasculature (medium & small vessels)⁷ which results in vasculitis and eventually submucosal endarteritis.¹² This causes ischemia and thus tissue breakdown and perforation.

The significance of this rare complication lies in the diagnostic difficulties associated with it. Neither is a specific diagnostic investigation available nor does a single investigation carry high diagnostic accuracy. The common investigative parameters often looked for in a case of acute abdomen proves to be diagnostic fallacy with leucocytosis noted in 29%, free air under diaphragm in x-ray in 25%.⁷ Computed tomography of abdomen has a better sensitivity of 81% in picking up tubercular perforation.¹³ Demonstration of typical histopathology of caseating necrosis, MTB in lesion or culture is the definitive way of establishing diagnosis.⁸ Thus, in a patient with intestinal TB presenting with generalized peritonitis, a high index of suspicion must be maintained with a low threshold for exploratory laparotomy.

The dilemma does not end there and treatment decisions are based on: patient's general condition, timing of surgery since occurrence, intra-peritoneal contamination, number of perforations and bowel status, surgeon's experience. Primary closure being associated with high incidence of anastomotic leak and fistula formation is not recommended. Resection of involved bowel segment with primary anastomosis or exteriorization is better accepted.¹⁴

The backbone of management post-operatively is the administration of anti-tuberculous therapy as directly observed therapy based on isoniazid, rifampicin, ethambutol, pyrazinamide daily or thrice weekly for a duration of six months.¹⁵ After completion of therapy, a follow-up period of 12-39 months is essential.

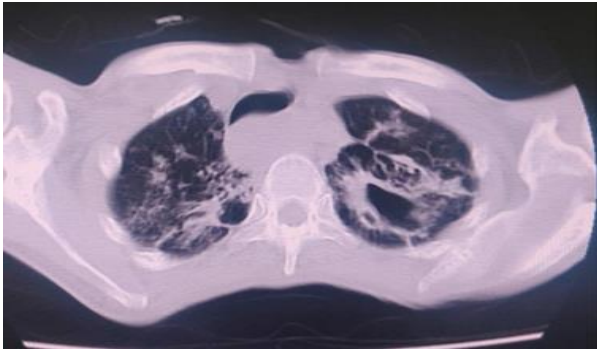


Figure 1: CT chest showing tractional bronchiectasis in bilateral upper lobes.

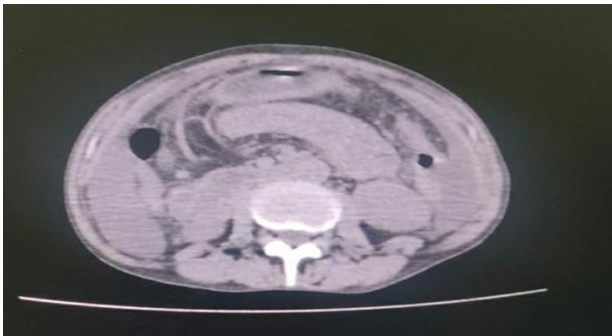


Figure 2: CT abdomen showing few air pockets in peri-splenic and left paracolic gutter with free fluid.

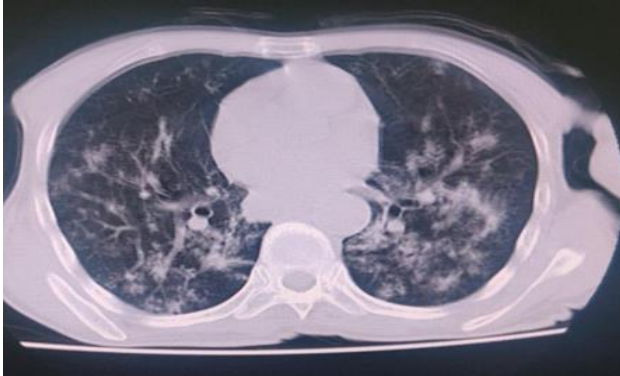


Figure 3: CT chest showing diffuse peribronchovascular ground glass opacities.

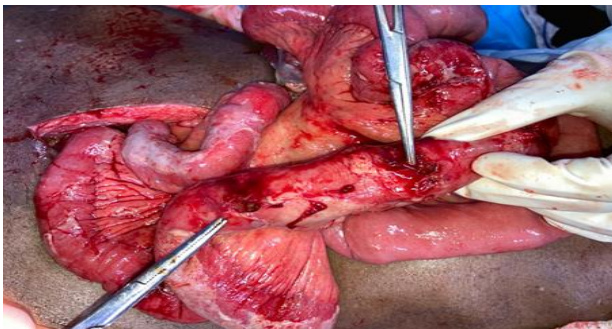


Figure 4: Intraoperative picture showing multiple ileal perforation.



Figure 5: Postoperative picture showing the fashioned double barrel ileostomy after resection of perforated ileal segment.

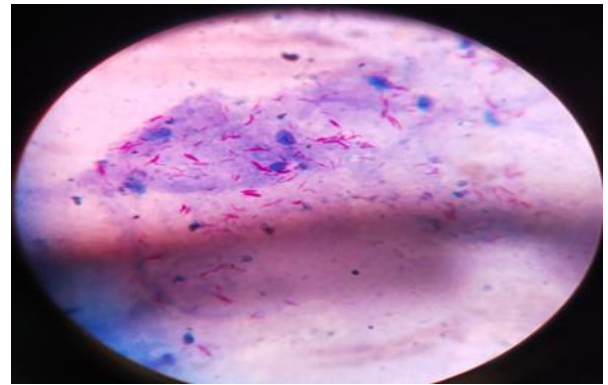


Figure 6: Peritoneal fluid AFB staining positivity.

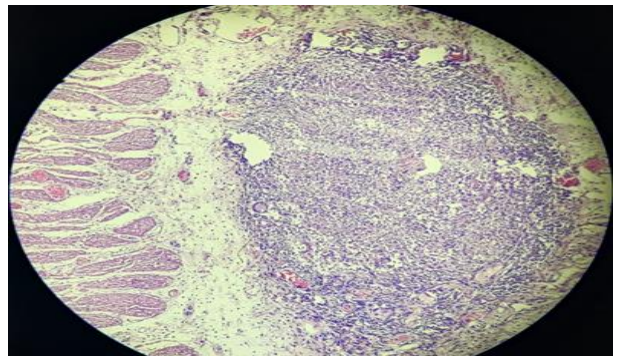


Figure 7: Histopathology showing ileal submucosal epithelioid granulomas with Langhans giant cells.

CONCLUSION

Tuberculosis though a disease rampant in developing nations, is of surge even in western world due to increase in immunocompromised states and emergence of resistant strains. Though free bowel perforation due to tuberculosis is rare; with a resurgence of tuberculosis it is essential that surgeons are aware of this complication in acute abdomen cases as a possible differential.

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Ethical approval: Not required

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