

## Case Report

# A rare cause of faecal peritonitis: jejunal perforation in a patient undergoing treatment for pulmonary tuberculosis

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Abdominal tuberculosis (TB) is a rare but well-documented cause of faecal peritonitis, occasionally occurring in cases where the diagnosis has been delayed, thus resulting in progressive disease. Frequently occurring in the ileo-caecal region, it requires commencement of anti-tubercular regimen and can necessitate surgical intervention. We present a rare case of faecal peritonitis in a young immuno-competent patient with a perforated jejunal stricture, despite triple therapy for known pulmonary TB.

## INTRODUCTION

The incidence of tuberculosis (TB) has risen in developed countries, owing mainly to immune-compromisation from acquired immunodeficiency syndrome (AIDS), multidrug-resistant organism strains and increased immigration from high-risk areas such as the Indian subcontinent, where rates of TB are 50 times higher than in Europe.

*Mycobacterium tuberculosis*, the most common pathogenic organism, can manifest itself within the abdomen via haematogenous spread from a pulmonary primary, transmural invasion of the bowel following ingestion, reactivation of dormant disease or tuberculous salpingitis. *Mycobacterium bovis* is a less common cause in the western world, found in dairy products.

Symptoms are insidious, including fever, malaise, nausea, anorexia and weight loss. Clinical history and evidence of pulmonary involvement AIDS diagnosis. However, abdominal TB often presents itself in its late stage, resulting in complications of inflammatory masses or strictures. Abdominal distension may be a secondary to ascites, bowel obstruction or perforation. The latter results in peritonitis and signs of sepsis. Ziehl–Nelson testing and analysis of peritoneal fluid are commonly unhelpful, lending peritoneal biopsy or tissue analysis greater importance in diagnosis. TB peritonitis requires urgent surgical intervention and commencement of anti-tuberculous therapy. Complications of TB peritonitis include adhesions, fistula formation and, in the case of significant bowel resection, nutritional deficiencies.

## CASE REPORT

A 26-year-old Indian man presented with generalized abdominal pain, vomiting and absolute constipation. Symptoms had developed over several months, with night sweats, weight loss, malaise and reduced appetite, shortly after returning from a trip to India.

He had been started on triple therapy for presumed pulmonary TB, based on a suspicious chest radiograph 6 months prior, showing bilateral ill-defined shadowing in the upper-lung zones with signs of paratracheal lymph node enlargement.

On assessment he was found to be septic with tachycardia, hypotension and peritonitis on examination. He underwent an emergency laparotomy, revealing two tight jejunal strictures, one of which had perforated. Two small bowel resections of lengths 7 and 10 cm were performed, with primary anastomosis.

One week post-operatively, he developed pyrexia and right iliac fossa pain. An abdominal ultrasound scan revealed a 1 × 6 × 8 cm collection in the right iliac fossa. Although not amenable to drainage, a diagnostic aspiration was performed and the specimen returned negative for acid-fast bacilli. A repeat scan 10 days later after antibiotics detected a tiny volume of free fluid, with no identifiable collection.

Histology revealed fibrinous exudates over the serosal surface of both resected jejunal specimens, with lymphoid aggregates and occasional granulomas. This, in context with the presentation, was in keeping with abdominal TB.

Following recovery, he was discharged and completed the remaining duration of anti-tuberculous therapy. He made satisfactory progress on follow-up and required long-term supplements for Vitamin D deficiency following extensive jejunal resection.

## DISCUSSION

Evidence has shown that the incidence of extra-pulmonary disease has risen recently [1], accounting for 17% of notified TB cases in Europe in 2010 [2]. The abdomen is the sixth commonest site of extra-pulmonary TB [3], involving the Gastrointestinal tract, pancreas, adrenals, omentum, liver, spleen and female genital tract. The ileum and caecum are involved in 75% of cases. Intestinal TB, generally a complication of pulmonary TB from ingestion of infected sputum, virtually always involves the small bowel. Primary intestinal TB is a rare occurrence in the western world since introducing milk pasteurization. Concurrent active pulmonary TB or radiologically evident lesions are found in half of the patients with abdominal disease [4]; however, pulmonary involvement with cavitation and positive sputum smears are reported to increase the risk of developing intestinal disease.

Non-specific symptoms precede the diagnosis of abdominal TB by more than 4 months in over 70% of patients [5] making diagnosis challenging. Advanced disease resulting in perforation may be the first presentation. Classic signs of peritonitis include a rigid abdomen, distension, muscle guarding and absent or hypoactive bowel sounds. In the deteriorating patient fever, tachycardia and hypotension mark sepsis and need for urgent intervention.

Initial management involves treatment of electrolyte abnormalities secondary to fluid shifts and sepsis with fluid resuscitation and broad-spectrum antimicrobial therapy. Emergency laparotomy follows, to identify and control the source of infection. Macroscopically, the bowel appears thickened, often surrounded by an inflammatory mass. Faecal contamination results in a mixture of organisms on peritoneal aspiration.

Testing for acid-fast organisms in ascitic fluid is frequently negative in patients with abdominal TB, with 3% being positive in TB peritonitis [6]. Analysis of ascitic fluid suggests inflammation, with high protein and leucocytes, and low glucose concentration. High ascitic lymphocytic counts warrant cytological investigation, leaving fluid culture and percutaneous peritoneal biopsy as the superior methods of TB diagnosis [7, 8].

Secondary disease causes ulcerated lesions of caseating foci within the mucosa and submucosa. Progression of the results in circumferential extension of the bowel along the lymphatics and healing of the insult by fibrosis results in scarring. Classically, infiltration of the ileo-caecal valve has

varying degrees of extension into the ileum and caecum. The main processes are of macrophage accumulation into granulomas and reactive fibrous connective tissue formation, giving rise to the offending stricture. Macroscopically, there is an inflammatory mass, with thickened bowel. Appearance can be difficult to distinguish from Crohn's disease. However, tuberculous granulomas are generally confluent, with a caseous centre and peripheral lymphocytic ring; these features, along with pale serosal tubercles serve as distinguishing diagnostic features of TB. In addition, ileo-caecal valve damage and involvement of fewer than four parts of the bowel should lend greater suspicion to a diagnosis of TB [9].

Treatment is with 6-month anti-tuberculous regimen of Isoniazid and Rifampicin, with additional Pyrazinamide and Ethambutol for the first 2 months. A retrospective review of patients with abdominal ( $n = 34$ ) and pulmonary ( $n = 163$ ) TB treated with anti-tuberculous treatment revealed only four patients with overlapping disease, with no difference in treatment success between them [10]. Drug-resistant disease presents a problem with recurrence and spread of disease, requiring close follow-up even after completion of treatment.

TB should be considered a differential cause of peritonitis, especially in immigrant patients or those who have been in close or frequent contact with the disease, despite an often misleading status of immuno-competency as seen in this case.

## REFERENCES

1. Raviglione MC, Snider DE Jr, Kochi A. Global epidemiology of tuberculosis. Morbidity and mortality of a worldwide epidemic. *JAMA* 1995;273:220–6.
2. European Centre for Disease Prevention and Control/WHO Regional Office for Europe. Tuberculosis surveillance and monitoring in Europe. Report. Stockholm: European Centre for Disease Prevention and Control 2012.
3. Singal R, Gupta S, Gupta S. Primary abdominal tuberculosis presenting as peritonitis in a young child—managed surgically. *Asian Pac J Trop Med* 2012;5:413–5.
4. Winter HS, Gervais DA, Branda JA. Case records of the Massachusetts General Hospital. Case 27—2011. A 17-year-old boy with abdominal pain and weight loss. *N Engl J Med* 2011;365:940–50.
5. Yamada T, Alpers AH, Kaplowitz N, Laine L, Owyang O, Powell DW eds. *Textbook of Gastroenterology*. 4th edn. Philadelphia: Lippincott Williams & Wilkins, 2003: 2552–3.
6. Sanai FM, Bzeizi KI. Systematic review: tuberculous peritonitis—presenting features, diagnostic strategies and treatment. *Aliment Pharmacol Ther* 2005;22:685–700.
7. Levine H. Needle biopsy of peritoneum in exudative ascites. *Arch Intern Med* 1967;120:542–5.
8. Levine H. Needle biopsy in diagnosis of Tuberculous peritonitis. *Am Rev Respir Dis* 1968;98:519.
9. Lee YJ, Yang SK, Byeon JS, Myung SJ, Chang HS, Hong SS, et al. Analysis of colonoscopic findings in the differential diagnosis between intestinal tuberculosis and Crohn's disease. *Endoscopy* 2006;38:592–7.
10. Chong VH. Differences in patient profiles of abdominal and pulmonary tuberculosis: a comparative study. *Med J Malaysia* 2011;66:318–21.