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A 14-year-old girl presenting with tuberculous intestinal perforation while in a temporary shelter after the 2015 earthquake in Nepal

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ABSTRACT

A 14-year-old Nepalese girl presented with fever, abdominal pain and vomiting. She was living with her family in a temporary settlement camp following the earthquake in Nepal in 2015. She had had abdominal pain for 2 months and fever for 1 month. Abdominal examination suggested acute peritonitis. At laparotomy, three ileal perforations were detected and histopathology demonstrated caseous granulomas. Her father had sputum-positive pulmonary tuberculosis. She was diagnosed with abdominal tuberculosis and responded well to anti-tuberculosis chemotherapy. Intestinal perforation is a rare complication of tuberculosis in children.

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Introduction

Acute abdominal perforation is a recognised but rare complication of tuberculosis in children and adolescents. Abdominal tuberculosis accounts for up to 5% of tuberculosis, of which only 8% present with acute abdomen with intestinal perforation [1–3]. An adolescent with intestinal perforation owing to tuberculosis is described.

Case Report

A 14-year-old girl presented to a temporary health centre in a settlement camp in Bhakatpur City for victims of the 7.8-magnitude earthquake in 2015 which had hit the Kathmandu valley two weeks previously. She had had fever, chills and rigors for 2 days and then severe abdominal pain and vomiting for one day. On examination, her temperature was 38.9°C; there was localised peri-umbilical tenderness and absent bowel sounds but no signs of ascites. Peritonitis was suspected and she was immediately referred to Siddhi Memorial Hospital, a charity-funded 50-bed children's hospital in Bhaktapur. Her parents reported that she had had abdominal pain (dull and non-colicky) on and off for 2 months and fever for 1 month. On examination, she looked ill, cachectic and was in distress. Her temperature was 38.9°C, pulse 120/min and weak, respiratory rate 30/min, blood pressure 90/60 mm Hg and SpO_2 was 94% in room air. Her weight was 32 kg (weight for age < 3rd centile). She had tenderness in the peri-umbilical area with guarding, rigidity and rebound tenderness, and absent bowel sounds.

Investigations

Haemoglobin was 9.9 g/dL, white blood cell count (WBC) 9.7×10^9/L (neutrophils 70%, lymphocytes 27%, monocytes 1%, eosinophils 2%) and platelet count 669×10^9/L. CRP was 457 nmol/L (0.76–28.5), serum sodium 136 mmol/L, serum potassium 3.3 mmol/L, random blood glucose 4.4 mmol/L (3.9–6.1), serum urea 7.1 mmol/L (2.9–8.2) and creatinine 53.04 µmol/L (53–106). The Widal test titre was 1:160 for O and H antigens (significant titre > 1:80). A chest radiograph and plain abdominal radiograph (erect) demonstrated free gas under both diaphragms (Figure 1). A full chest radiograph on admission was normal.

The clinical diagnosis was acute intestinal perforation secondary to acute appendicitis, typhoid fever or intestinal tuberculosis. She was stablised with intravenous (IV) fluid and IV ceftriaxone (1 g), and underwent exploratory laparotomy. Three perforations were detected in the anti-mesenteric border of the mid and distal ileum (Figure 2). There were hypertrophied and congested Payer's patches in the area of the ileum. Biopsies were taken from the edge of the ulcers for histopathological examination, and the perforations were oversewn. She was continued on IV ceftriaxone 12-hourly for 7 days.

In the post-operative period, she was further investigated for tuberculosis and typhoid fever. Sputum microscopy for acid-fast bacilli was negative on three samples (mycobacterial culture was not available). Mantoux test was negative. ESR was 53 mm in the first hour. Blood culture was negative. Ten days after surgery,

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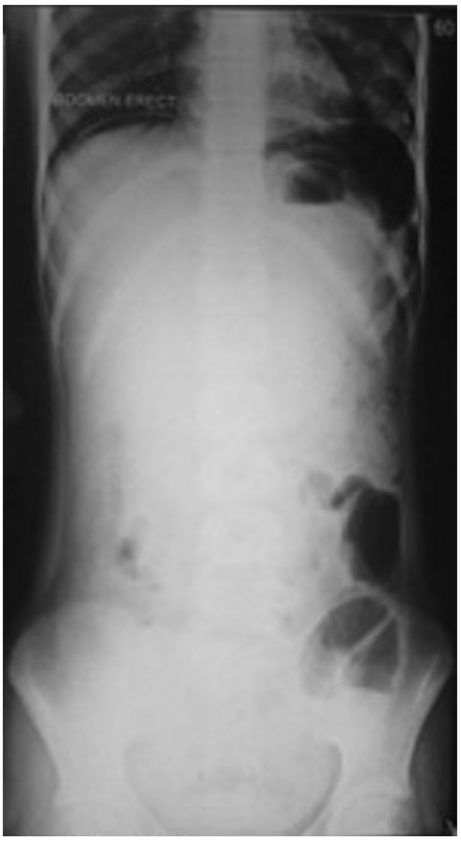

Figure 1. Day 1: abdominal radiograph showing free gas under both domes of the diaphragm.

there was still fever and her general condition had not improved. The incision site was still tender and there was mild abdominal distension. Chest radiograph was repeated and showed right-sided consolidation with a pleural effusion (Figure 3). Analysis of pleural fluid showed total WBC 2.1×10^6/L (neutrophils 5%, lymphocytes 95%), glucose 1.78 mmol/L, protein 88 g/L and lactic dehydrogenase 897 U/L (<200). Histopathological examination of the perforated intestine showed an infiltration of chronic inflammatory cells, areas of caseous necrosis (Figure 4(A)) and multinucleated Langerhans giant cells (Figure 4(B)). The possibility of a family history of tuberculosis was investigated and a chest radiograph of her father demonstrated a cavitating lesion in the right upper zone, supportive of pulmonary tuberculosis. His sputum smear was acid-fast bacteria (AFB)-positive.

The ceftriaxone was stopped and she was commenced on antituberculous therapy (ATT). After 5 days of ATT, the fever subsided and she was discharged home with a diagnosis of abdominal tuberculosis with intestinal perforation with probable pleural involvement. At a follow-up visit after 6 months of ATT, she was well and had no abdominal symptoms. A repeat chest radiograph showed a small right pleural effusion with residual fibrosis and pleural adhesions. ATT was stopped after a total of 8 months of chemotherapy. Two months after cessation of ATT, a chest radiograph demonstrated a small amount of pleural thickening only.

Discussion

The final diagnosis was abdominal tuberculous complicated by intestinal perforation, and probably pulmonary tuberculosis also. There was no evidence of disseminated disease. The primary focus of the disease is not clear but, in view of the pulmonary involvement and the close contact, it is likely to be in the lungs and the abdominal disease owing to swallowed sputum. The appearance of consolidation and pleural effusion despite parenteral ceftriaxone and the slow resolution is more in favour of tuberculosis than an acute bacterial pneumonia. The hospital did not have capacity for bacteriological confirmation of the diagnosis. In Kathmandu valley, the usual differential diagnosis for an acute abdomen in a child is either a perforated appendix or typhoid ulcer [4]. The features of intestinal perforation owing to typhoid and tuberculosis are compared in Table 1.

In children, the diagnosis of abdominal tuberculosis is extremely difficult as the symptoms are non-specific

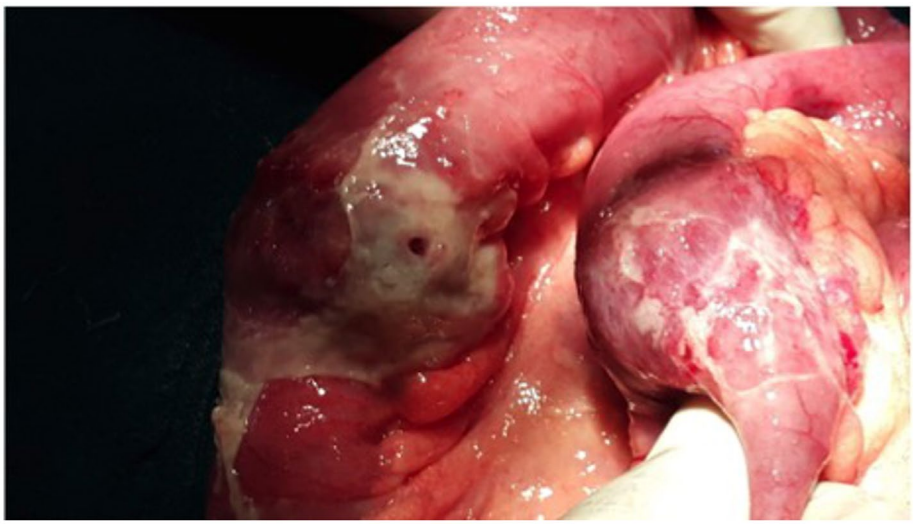

Figure 2. Perforations of the anti-mesenteric border of the ileum.

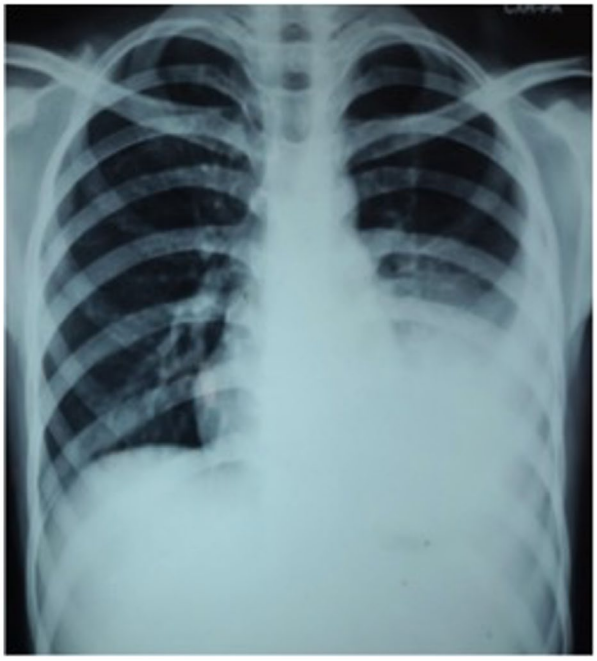

Figure 3. Day 10: chest radiograph demonstrating left-sided consolidation with pleural effusion after laparotomy and before the anti-tuberculosis treatment was commenced.

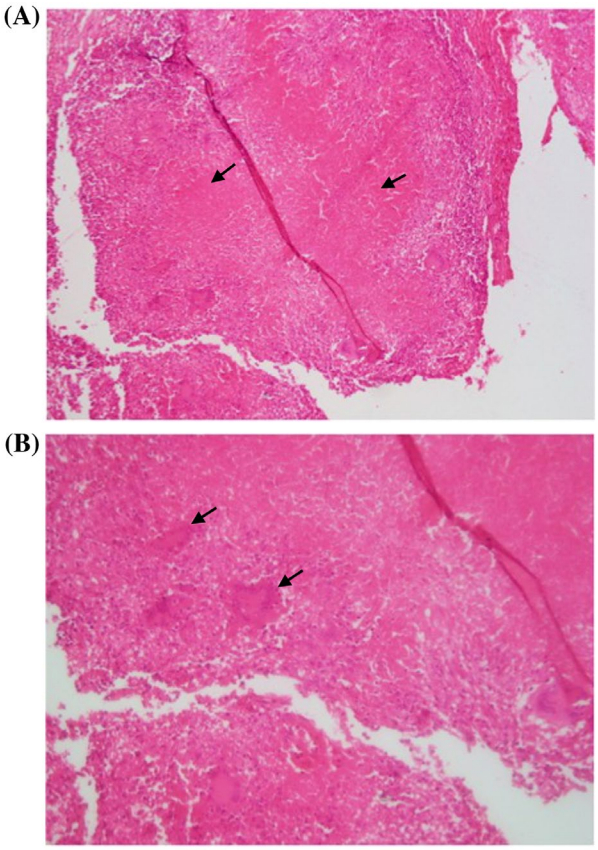

Figure 4. Histology of the ileal tissue demonstrating (A) caseous necrosis (arrows) and (B) Langerhans giant cells (arrows).

and sensitive tests are lacking. It can be confirmed by examination of abdominal tissue (biopsy of lymph nodes, peritoneum or omentum by laparoscopy, fine-needle aspiration cytology and laparotomy, and

Table 1. Differences between typhoidal and tuberculous intestinal perforations [5].

Typhoid fever perforation	Tuberculosis perforation
Single or multiple ulcers. Shallow irregular-oval ulcers generally occur longitudinally on the anti-mesenteric border of the ileum	Usually transverse ulcers in the anti-mesenteric border of the ileum
Histological examination: lymphocytic infiltration with red blood cells ingested by the macrophages (typhoid cells)	Histological examination: lymphocytic infiltration with caseous necrosis and Langerhans giant cells
Usually occurs in second to third week after onset of fever	Usually occurs after weeks or months of gradual onset of fever
There may be a history of contact with a recent typhoid patient	There is often a history of tuberculosis contact
Blood culture may grow *Salmonella enterica* serovar Typhi or Paratyphi A	Sputum microscopy may be AFB smear-positive in older children

by the detection of intestinal ulcers by colonoscopy) for a positive AFB smear or mycobacterial culture and histopathology. As these are invasive procedures, there can be a danger of surgical complications such as peritonitis, septicaemia and delayed wound healing. Abdominal ultrasonography or computerised tomography scan of the abdomen may demonstrate features of tuberculosis including ascites, mesenteric lymphadenopathy, mesenteric thickening, hepatomegaly, splenomegaly, thickened bowel loops, adhesions in the caecal area or an abdominal mass [1]. Surgical repair of intestinal perforation and prompt commencement of ATT is the optimal therapy for abdominal tuberculosis.

Abdominal tuberculosis accounts for only 1–5% of cases of tuberculosis in children [2] and is the sixth most common form of extra-pulmonary tuberculosis [3]. It is more common in school children and adolescents than in children under 5 years [1,6,7]. It develops following ingestion of tubercle bacilli, either from swallowed sputum or direct ingestion of contaminated food products including animal milk. The microfold, or M-cells, found in the epithelium of Peyer's patches are thought to provide the route of entry for the bacillus. The most common site of infection is the ileo-caecal region because of the presence of abundant lymphoid tissue, but any part of the gastro-intestinal tract can be infected by tuberculosis. Intestinal tuberculosis can present in three forms: ulcerative, hypertrophic or ulcero-hypertrophic [8]. The ulcerative form is the most common type and accounts for 60% of abdominal tuberculosis. Bowel perforation is a serious but very rare presentation of abdominal tuberculosis [6,7,9].

In a retrospective 10-year study of 334 Indian children <12 years of age who required an exploratory laparotomy because of an acute abdomen, only 4.2% showed evidence of abdominal tuberculosis and only one case had an ileal perforation [10]. Other case series of intestinal perforation in children and adults in the Indian sub-continent report abdominal tuberculosis as a cause in 4–10% of cases [11,12]. An acute abdomen

with ileal perforation and peritonitis is therefore a rare presentation of intestinal tuberculosis and the case highlights the contribution to the diagnosis of a family history of active tuberculosis in areas where the capacity for laboratory confirmation of tuberculosis by culture is lacking.

Ethics approval

The patient and her parents agreed to publication.

Disclosure statement

No potential conflict of interest was reported by the authors.

Contributors

DS, KA, CMP, BGD designed the study; MKS performed the surgery, prepared the histopathological specimen, and took intra-operation picture; DS, GBR, AB, BGD collected the clinical data; KH did the histopathological examination and prepared the laboratory findings. DS, KA, CMP, BGD prepared the first draft; all authors revised and finalised the manuscript.

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