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Multiple Intestinal Strictures with Perforation in a Patient under Antitubercular Treatment for Abdominal Tuberculosis

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ABSTRACT

Abdominal tuberculosis (TB) constitutes about 12% of the extrapulmonary TB. Abdominal TB can present with varying signs and symptoms like vague abdominal pain, abdominal mass, ascites and intestinal obstruction. Intestinal perforation is a relatively uncommon but a serious complication of abdominal TB. Antitubercular drugs are considered extremely effective but their role in the presence of a stenotic lesion of the bowel is controversial. Once symptoms of bowel obstruction or peritonitis appear, antitubercular drugs are of little use and surgery is inevitable.

We report a case of abdominal TB in a patient under antitubercular treatment who later developed multiple intestinal strictures and a perforation.

Keywords: perforation; stricture; tuberculosis.

INTRODUCTION

The World Health Organization (WHO) estimates 9.27 million new cases of tuberculosis (TB) in 2007. Tuberculosis is endemic in many parts of the developing countries and its incidence is increasing worldwide due to the emergence of multidrug resistant TB and HIV infection.[1] Abdominal TB constitutes about 12% of the extrapulmonary TB and about 1-3% of total.[2] The reason for its relative rarity is attributed to bactericidal property of gastric acid, scarcity of lymphoid tissue in gastric wall and intact gastric mucosa of the stomach.[3] Abdominal TB can involve the gastrointestinal tract, peritoneum, lymph nodes and solid viscera including the liver, spleen and the pancreas.[4] We report a case of abdominal TB presenting with multiple strictures and a perforation.

CASE

A 21-year-old male presented to the emergency department of tertiary level center with abdominal pain, vomiting and constipation for 3 days. Abdominal pain was insidious in onset, gradually progressive, intermittent and pricking without any aggravating and relieving factors and radiated towards loins and back bilaterally. He had 2 episodes of non-projectile, non-bilious vomiting that contained food particles, not mixed with blood. Patient had no history of cough, fever, and chest pain. In his past medical history, patient was under antitubercular treatment for abdominal TB since two months.

Cardiorespiratory examination was unremarkable. Abdominal examination revealed abdominal distention and ascites. There was generalized abdominal tenderness but no organomegaly with sluggish bowel sounds. Laboratory examinations revealed a total count of 23,100/mm^3 with a differential count of neutrophils84%, lymphocytes 15%, eosinophils 1% and an elevated CRP. Abdominal X-ray supine showed dilated bowel loops. Abdominal USG showed dilated bowel loops with minimal interloop fluid and mesenteric lymphadenopathy. Ascitic fluid analysis showed cells > 1000/cu mm predominantly lymphocytes, negative for acid-fast bacilli and ADA 56 U/l. The patient was admitted with aprovisional diagnosis of sub-acute intestinal obstruction and few days later developed symptoms consistent with intestinal perforation. Chest x-ray (Posteroanterior view) showed

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gas under the diaphragm suggestive of hollow viscus perforation for which patient underwent an exploratory laparotomy which demonstrated multiple strictures at 35 cm, 45 cm, 60 cm from ileocecal junction, and 15cm, 20cm, 25 cm from the duodenojejunal junction. A perforation was noted at 60 cm from the ileocecal junction with adhesions and multiple bands. Primary closure of the perforation was done and stricturoplasty was performed at the sites of strictures.

Histopathology of the specimen showed necrotic tissue with neutrophilic exudation and round cells infiltration. The adherent omental tissue showed microscopic foci of round cells infiltration. The findings were consistent with benign ulcerative lesion.

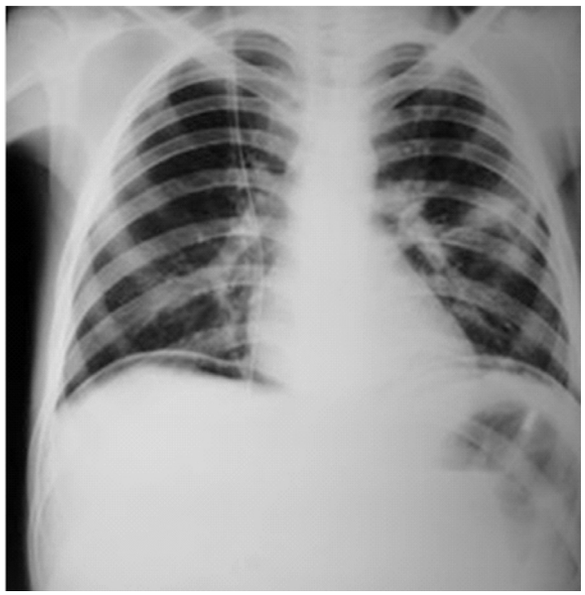

Figure 1. Air under the diaphragm due to intestinal perforation.

Figure 2. Perforation 60 cm from the ileocecal junction.

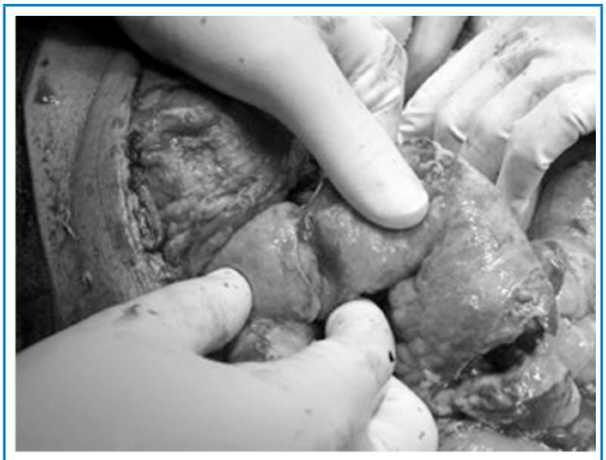

Figure 3. Stricture.

In the postoperative period, the patient returned to the ward after spending 5 days on the intensive care unit, and had an uneventful recovery. He was then discharged and was asked to follow up regularly for his antitubercular treatment.

DISCUSSION

The etiopathogenesis of abdominal TB is usually linked to hematogeneous spread from a pulmonary focus acquired during a primary infection in childhood. Another possible mechanism could be due to ingestion of the bacilli, which pass through the Peyer's patches of the intestinal mucosa and are transported by the macrophages to the mesenteric lymph nodes via the lymphatics.[4] Conditions such as malnutrition, alcoholism, diabetes, chronic renal failure, immunosupression and AIDS increase the risk of reactivation.[5] The symptoms and signs of abdominal involvement vary with the site of involvement, the type of lesion and the mode of presentation. Patients can present with fever, weight loss, abdominal mass, ascites, diarrhea, malabsorption, features of intestinal obstruction and peritonitis.[6]

Three types of intestinal lesions are commonly seen- ulcerative, stricturous, and hypertrophic. Cicatricial healing of the ulcerative lesions results in strictures and can cause acute or subacute intestinal obstruction.[7] Intestinal perforation is a relatively uncommon but a serious complication of abdominal tuberculosis occurring in 1-15% of patients,[8-10] and usually occurs due to reactivation of a dormant focus. Tuberculous perforations are predominantly solitary and located immediately proximal to the site of stricture; although there are reports of multiple perforations they are usually uncommon.[11] In a study the terminal ileum was the site of perforation in 54.54% of the cases and the clinical features of perforation were consistent with those of generalized peritonitis in 72.72% of the cases.

Ascitic fluid adenosine deaminase (ADA) activity has been proposed as a useful diagnostic test for abdominal TB especially in countries with a high incidence of TB and in high risk patients.[12] Ultrasonography (US) of the abdomen is a useful initial investigation,[13] with characteristic findings that include low attenuation lymphadenopathy, omental or ileocaecal inflammatory mass, peritoneal thickening and ascites.[14] US guided FNAC of lymph nodes or a solid organ lesion is a useful diagnostic tool due to its safety and rapid definitive diagnosis of tuberculous granulomas. CT scan can be carried out in case of inconclusive US examination which can show high density ascites and caseous necrosis of lymph nodes.[13]

Crohn's disease should be differentiated from abdominal TB due to the harm associated with steroids and immunomodulatory agents. The combination of these agents would have adverse consequences in cases of abdominal TB misdiagnosed as Crohn's disease.[15]

Antitubercular drugs are extremely effective but their role in the presence of a stenotic lesion of the bowel is controversial[16] since they may precipitate intestinal obstruction due to healing by fibrosis and cicatrisation, or even result in intestinal perforation.[16-18] It is usually recommended that once symptoms of bowel obstruction, peritonitis or perforation appear, antitubercular drugs are of little use and surgery is unavoidable.[19-21] Strictureplasty is preferred for small intestinal strictures, but resection is performed in patients with tight fibrotic strictures that almost totally obliterate the lumen.[6]

CONCLUSIONS

Patients with abdominal TB may present with features of intestinal obstruction accompanied by perforation. Radiology and histopathological examination are useful modalities for diagnosis. Antitubercular drugs are widely accepted as the mainstay of treatment of abdominal TB but complications (e.g. stricture, perforation, peritonitis) necessitate surgical intervention.

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