CASE REPORT

SHOCK AND TUBERCULOUS ENTERITIS IN A NON-HIV INFECTED HEALTHY MAN

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Abstract. We reported a case of shock, diarrhea and tuberculous enteritis in a previously healthy 32-year-old non-HIV infected man. This case presented initially with acute profuse watery diarrhea. The bacterial and virus cultures were negative. Tuberculous enteritis was diagnosed by detecting *Mycobacterium tuberculosis* on stool culture 52 days after presentation. The symptoms resolved after treatment with intravenous fluids and corticosteroids but was treated with anti- tuberculous medication. Tuberculous enteritis should be included in the differential diagnosis of patients with enteritis and shock.

Keywords: *Mycobacterium tuberculosis*, enteritis

INTRODUCTION

The diagnosis of abdominal tuberculosis is difficult to make even in the tuberculous endemic areas. Patients often present with non-specific symptoms such as abdominal pain, fever, weight loss and diarrhea. We present a 32 year old man with acute, profuse diarrhea, and compromised hemodynamics needing intensive care.

CASE REPORT

A previously healthy 32 year old man without a history of serious or chronic illness, not taking any immunosuppressive drugs or having close contacts with patients with *Mycobacterium tuberculosis* infection presented to our hospital with a 3-day history of fever, watery diarrhea and epigastric cramping pain. He had watery stools 20 times per day that were yellow colored. The volume of stool passed each time was about 150 ml. There was no blood or mucus seen in the stool. He denied any previous history of watery diarrhea prior to this episode. On presentation his systolic blood pressure was 85 mmHg and diastolic blood pressure was 50 mmHg. His temperature was 37.5°C, his pulse rate was 120/minute and his respiratory rate was 28/minute. Physical examination revealed epigastric tenderness, with diffuse rebound tenderness but no guarding. The digital rectal examination revealed yellowish stool with no blood. The Computerized Tomogra-

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phy (CT) scan of the abdomen showed reactive mesenteric lymph nodes along the small intestine, dilated loops of small bowel and focal wall thickening of the jejunum. Laboratory examination showed a peripheral leukocyte count of $3.08x10^3$ cells/mm^3 with 16% band forms and 59% segmented neutrophils. Liver function testing showed glutamic oxaloacetic transaminase and glutamic pyruvic transaminase levels of 58 IU/l (normal range 5-35 IU/l) and 87 IU/l(normal range 0-40 IU/l), respectively. His serum creatinine was 2 mg/dl (normal range 0.7-1.5 mg/dl) and his plasma lactate dehydrogenase was 27.1 mg/dl (normal range 6.3-18.9 mg/dl). His HIV ELISA test was negative. Stool analysis revealed the presence of occult blood. The blood culture was negative but the stool culture yielded *M. tuberculosis* 52 days later.

The patient initially received ceftriaxone, metronidazole, minocycline and hydrocortisone for tentative diagnoses of intra-abdominal infection or scrub typhus. He was resuscitated with 11 liters of intravenous fluid and a vasopressor since he did not respond to intravenous fluid alone; he gradually improved. A small intestine X-ray series was performed as an out patient one month later and it was normal. After the culture results were obtained 52 days later, he was treated with isoniazid, rifampicin, ethambutol and pyrazinamide for 6 months successfully.

DISCUSSION

In this case, tuberculosis was diagnosed by positive stool culture for tuberculosis and abdominal CT scan findings. Anti-tuberculous medication was not prescribed initially, but he responded well to rehydration, inotropic agents and antibiotic therapy. Septic shock is normally associated with gram-negative bacteria, toxin production from gram-positive bacteria, or fungal infection. In mycobacterial disease it is thought tumor necrosis factor (TNF) production stimulated by lipoarabinomannan from *M. tuberculosis* stimulates release of TNF from human monocytes and activated peritoneal macrophages (Vadillo *et al*, 1994). Therefore, improvement in the clinical status of the patient before anti-tuberculosis treatment could be due to the steroid and hemodynamic management.

The etiology of tuberculous enteritis has been attributed to swallowing infected sputum, hematogenous spread of active pulmonary tuberculosis, ingestion of contaminated milk or food or contiguous spread from adjacent organs (Marshall, 1993; Horvath and Whelan, 1998; Kapoor, 1998). It is likely this patient was infected by swallowing infected sputum or ingesting contaminated milk or food, since he had normal chest radiography and the abdominal CT scan did not support the presence of tuberculosis in adjacent organs. In patients who present with diarrhea and shock, the possibility of tuberculous enteritis should be considered.

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