

HIV infection presenting with duodenal tuberculosis

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ABSTRACT

Extrapulmonary tuberculosis is a protean and often difficult to recognise infection. Gastrointestinal tuberculosis is a rare condition that mainly occurs in immunodeficient people. We report a case of duodenal tuberculosis, which presented with gastrointestinal symptoms, anaemia and hyponatraemia, in a patient with previously undiagnosed HIV infection.

INTRODUCTION

Tuberculosis, one of the oldest infections known to affect humans, is today still one of the leading causes of death globally. The disease occurs worldwide, although it is found primarily in developing countries, where poor sanitation contributes to its spread.^{1,2} Pulmonary manifestations predominate in most tuberculosis cases, yet in up to one-third of patients other organs are affected. Gastrointestinal involvement may be present as a part of the multiorgan disease process or, less commonly, as primary gastrointestinal tuberculosis. We report a patient, with previously undiagnosed HIV infection, who presented with anaemia and duodenal tuberculosis.

CASE REPORT

A 23-year-old Nigerian male presented with a four-week history of fever, malaise, nausea, vomiting, anorexia and a 15 kg weight loss. His past medical record was unremarkable except for malaria. He was cachectic but not acutely

ill, temperature was 38.3°C, and lymph nodes (Ø 2 cm) were felt in both sides of his neck, while the rest of the examination was normal. Blood analysis showed albumin 1.8 g/dl, lactic dehydrogenase 728 IU/l, gamma-glutamyl transpeptidase 100 IU/l, alkaline phosphatase 799 IU/l, C-reactive protein 126 mg/dl, IgG 3604 mg/dl, IgM 770 mg/dl, iron 23 µg/dl, total iron binding capacity 495 µg/dl, transferrin saturation 9%, ferritin 1317 ng/ml, haemoglobin 8.4 g/dl, mean corpuscular volume 76 fl, erythrocyte sedimentation rate 120 mm in the first hour, D-dimer 622 ng/ml, sodium 120 mEq/l, and osmolality 280 mOsm/kg, while all other results, including haemoglobin electrophoresis, haptoglobin, reticulocyte count, vitamin B12, folic acid, and thyroid hormones, were normal. Urine analysis disclosed abundant red blood cells, sodium 31 mEq/l, and osmolality 369 mOsm/kg, while all other results were normal.

The electrolyte disorder was classified as the syndrome of inappropriate ADH and the patient was treated with water restriction. Cultures of blood and urine, acid-fast bacilli of sputum, and serology of multiple infections were negative, except for anti-HIV, ELISA and *Western blot*, which were positive. CD4 cell count was 159 per mm³, and HIV RNA was 100,000 copies/ml. An electrocardiogram showed no abnormalities. Chest radiographs revealed a tenuous left lower lobe infiltrate. An upper gastrointestinal endoscopy demonstrated severe inflammation and ulceration of the mucosa situated just distal to the duodenal bulb, with the presence of a mucosal bridge (*figure 1*); a biopsy of the affected area revealed a granulomatous and necrotic inflammatory infiltrate, as well as the presence of acid-fast bacilli. Abdominal ultrasound and computed tomography exam-



Figure 1
Inflammation and ulceration of the mucosa distal to the duodenal bulb, resembling Crohn's disease, with the presence of a mucosal bridge (arrow in A)

inations showed multiple lymphadenopathies. A lymph node biopsy revealed a granulomatous and necrotic infiltrate. Suspecting tuberculosis, treatment was instituted with isoniazid, rifampin, pyrazinamide and ethambutol, and the patient's condition slowly improved over the next few weeks. By the second week of therapy *Mycobacterium tuberculosis* grew in the sputum. When the patient had completed eight weeks of antituberculous treatment, antiretroviral therapy was initiated, isoniazid and rifampin were continued, and pyrazinamide and ethambutol were withdrawn. At that time he was completely asymptomatic. He received no corticosteroid therapy.

DISCUSSION

In this paper we report the duodenal manifestations of tuberculosis in an HIV-positive African patient. Although fever and wasting in a patient of African descent always points in the direction of tuberculosis, a duodenal manifestation was not suspected clinically.

Tuberculosis usually affects the lungs, although extrapulmonary tuberculosis is also prevalent, especially among people with HIV infection.³ The extrapulmonary sites most commonly involved are the lymph nodes, pleura, genitourinary tract, bones and joints, meninges, and peritoneum. However, virtually all organ systems may be implicated.

The gastrointestinal tract is only rarely affected. Prior to the HIV epidemic, it was seen most commonly in immunocompetent persons with untreated advanced pulmonary disease.⁴ Today, it is most commonly observed in association with immunosuppression and, in one series, more than 40% of patients with gastrointestinal tuberculosis had AIDS.⁵ Swallowing of sputum with direct seeding and haematogenous spread are the main pathogenetic mechanisms. Any portion of the gastrointestinal tract may be involved, although the terminal ileum and caecum are the most commonly affected sites. Abdominal pain, diarrhoea, obstruction, haematochezia and a palpable mass are common findings at presentation. Fever, weight loss and night sweats are also frequent.^{6,7}

Duodenal tuberculosis generally presents with obstructive symptoms. Other nonspecific upper gastrointestinal complaints are also common. The differential diagnosis includes cancer, other infections, and chronic inflammatory conditions. Crohn's disease and intestinal tuberculosis may closely resemble each other, not only macroscopically, but also microscopically. Because the management of both conditions is so different, it is critical to distinguish them, generally with a biopsy of the affected mucosa.⁸⁻¹⁰

Our case of duodenal tuberculosis is another example of close similarity of such infection with inflammatory bowel

disease. Probably, as a result of the inflammatory process, our patient developed a mucosal bridge, a finding that has also been described in Crohn's disease.¹¹

Also remarkable in the reported patient is the presentation with severe hyponatraemia, probably resulting from inappropriate secretion of antidiuretic hormone (SIADH), a rare initial presentation of tuberculosis.^{12,13} The described case serves as an illustration of the protean and difficult to recognise presentations that tuberculosis may adopt.

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