

Parasternum pleural-cutaneous fistula in a severely immunosuppressed HIV-positive patient

ABSTRACT

Pleural tuberculosis occurs in 30% of patients with tuberculosis, and the percentage of patients with tuberculosis pleural effusions is comparable to human immunodeficiency virus HIV-positive and HIV-negative individuals, although pleural tuberculosis is rare in HIV-positive patients with CD4+ counts < 200 cells/mm³. Pleural tuberculosis in HIV-positive patients is likely to happen in young patients, and is more frequent in intravenous drug abusers, with more acid-fast bacilli identifiable in pleural tissue. We report a rare case of pleural tuberculosis in a severely immunosuppressed HIV-positive patient, presented as two parasternum pleural-cutaneous fistula.

Keywords: pleural tuberculosis, parasternum pleural-cutaneous fistula, severely immunosuppressed HIV-positive patient.

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INTRODUCTION

Pleural tuberculosis occurs in 30% of patients with tuberculosis, and the percentage of patients with tuberculosis pleural effusions is comparable to human immunodeficiency virus HIV-positive and HIV-negative individuals, although pleural tuberculosis is rare in HIV-positive patients with CD4+ counts < 200 cells/mm³.¹

HIV infection changes the clinical presentation of tuberculosis infection, favoring atypical radiographs and extrapulmonary involvement. In a previous study, pleural tuberculosis in HIV-positive patients was more common in younger patients, who were intravenous drug abusers, had significantly more acid-fast bacilli identifiable in pleural tissue and in sputum cultures.²

We examined a rare case of parasternum pleural-cutaneous fistula from pleural tuberculosis in a HIV-positive patient with a CD4+ count of 11 cells/mm³.

CASE REPORT

A 33-year-old male patient, from the city of São Paulo, was sent to the Clinical Emergency Department of the Conjunto Hospitalar do Mandaqui due to weight loss of 19 kilograms,

asthenia, weakness, cough, daily vespertine fever, night sweating, abdominal pain, and a progressively worsening dyspnea in the previous three weeks. The patient was HIV-positive for about 14 years, was an intravenous drug user, had started the antiretroviral therapy five years before this admission, and had abandoned the antiretroviral therapy. His medical history included a pulmonary tuberculosis infection treated appropriately in 2004; he was a chronic hepatitis B virus carrier and had chronic asymptomatic hepatitis C virus.

At admission, the patient was thin and had a consumptive status, oral lesions compatible with *Candida albicans* infection, non-specific abdominal pain and absent pulmonary murmurs at the inferior two-thirds of the left hemithorax. Diagnostic hypothesis were AIDS, bacterial pneumonia, pulmonary tuberculosis, or *Pneumocystis jiroveci* pneumonia with pleural effusion. Initial exams included hemogram, electrolytes, hepatic and canalicular enzymes, coagulation tests, serum albumin, sputum, and blood cultures, which resulted negative for infection.

His HIV viral load had increased by approximately one log, and his CD4+ count had decreased substantially in the last two

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years before this admission, corresponding to transitory non-adherence to antiretroviral therapy (Table 1).

As the initial clinical symptoms were suggestive of pulmonary tuberculosis in a severely immunosuppressed HIV-positive patient, sputum samples have been collected, searching for acid-fast bacilli related to pulmonary infection, but in all samples, no bacteria or acid-fast bacilli has been identified.

A month after his admission, the patient presented a non tender mass of elastic consistency adhered to deep planes in the left parasternum region, below healthy skin, with no point of fluctuation or local warmth. Ultrasonographic evaluation revealed heterogeneous density containing liquid and dense material with septa, without clear cleavage plane in relation to the intercostal muscles, measuring 48 x 42 mm. Thoracic surgeons have performed puncture and aspiration of the mass, and the aspirate analysis revealed acid-fast bacilli in great amount (+++), with no other microbiologic elements suggestive of infection, except *Mycobacterium tuberculo-*

sis. Patient received the following prescription: rifampicin, isoniazid, pyrazinamide, and ethambutol. Seven days after the first evaluation of the parasternum mass, another two thoracic masses had arisen in the intercostal spaces of left parasternum region, with irregular borders, liquid material, debris, and septations, measuring 40 x 24 mm and 25 x 13 mm, also aspirated. Both pleural aspirates revealed lymphocytic predominance, high levels of DHL (1,798 and 960 mg/dL), presence of high parasternum quantity of acid-fast bacilli (more than five bacilli per field) and negative bacterial cultures.

A thoracic CT scan has been done for a detailed analysis of the masses and their anatomic relation with the lungs, pleura, and subcutaneous thoracic tissue and revealed a clear dissemination of the masses from the left pleura to the muscular thoracic layers and to the subcutaneous tissue (Figures 1 and 2). We diagnosed pleural tuberculosis in a severely immunosuppressed HIV-positive patient.

Table 1. Viral load, CD4 counting, CD8 counting, and CD4/CD8 ratio

Date	VL (copies/mm ³)	Log	CD4 (cells/mm ³)	CD8 (cells/mm ³)	CD4/CD8
April 11 2006	-----	-----	11	93	0.12
February 21 2006	-----	-----	11	120	0.09
June 20 2005	15,100	4,179	121	926	0.13
March 23 2005	1,140	3,057	220	902	0.24
September 14 2004	-----	-----	35	218	0.16
August 27 2004	-----	-----	8	75	0.11
November 27 2002	-----	-----	301	794	0.38
August 05 2002	-----	-----	142	419	0.34

Figure 1: Thoracic CT scan of parasternum masses, showing left pleural effusion and evident continuity between the pleural space and the thoracic wall.

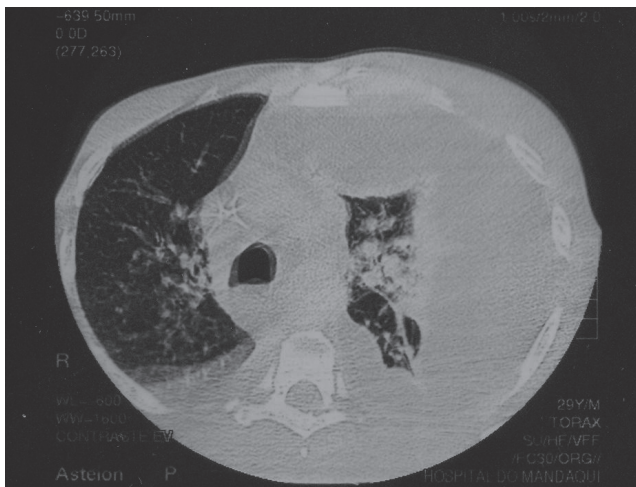


Figure 2: Thoracic scan showing tracheal deviation to the right, and predominance of left pleural effusion.



DISCUSSION

Pleural tuberculosis and HIV infection are strongly associated in HIV-infected patients. The pathogenesis of HIV-associated pleural tuberculosis involves direct bacterial invasion of pleural space, and the loss of immunity due to tuberculosis in HIV infection result from CD4+ Tcell depletion and reduction of antigen-specific cytokines responses, resulting in an uncontrolled mycobacterium replication, despite of the elevated levels of interferon-gamma and tumor necrosis factor-alpha.³

In this case report, among all clinical clues of extra pulmonary tuberculosis,⁴ the patient had an exudative pleural effusion, with lymphocytic predominance, negative bacterial cultures, and HIV infection. Moreover, the caseous-purulent aspect of the fistulized material, associated with the heterogenic density at the ultrasonografic evaluation, as reported elsewhere in the literature⁵ are typical evidence of pleural tuberculosis.

This patient presented four of the independent risk factors for pleural tuberculosis:⁶ history of liver disease (hepatitis B and C infection), *Mycobacterium tuberculosis* culture negative in sputum, chest pain, and presence of symptoms for less than 60 days. Additional suggestive symptoms were dyspnea and abdominal pain, more frequent in HIV-infected patients with pleural tuberculosis than with pulmonary tuberculosis.

Antituberculous therapy minimizes the morbimortality associated with tuberculosis infection and must be initiated empirically in most cases, once negative smear form acid-fast bacillus, lack of granulomas in HIV-infected patients, and negative cultures to *Mycobacterium tuberculosis* are not rare in severely immunosuppressed HIV-infected patients with pleural tuberculosis.

Even with the empirical introduction of antituberculous therapy, the mortality rate in HIV-patients remains elevated.

Treatment with rifampicin, isoniazid, pyrazinamide, and ethambutol had been continued, and, after two weeks of treatment, one of the masses fistulized to the skin, exteriorizing a yellow, caseous-purulent material, rich in acid-fast bacilli. Despite introduction of the adequate treatment for pleural tuberculosis, the patient died after 32 days of treatment.

CONCLUSION

Severely immunosuppressed HIV-positive patients usually present pleural tuberculosis at a younger age than HIV-negative patients. Once their immune response is impaired, when CD4+ count is < 200 cells/mm³, they do not present granulomas and have higher quantity of acid-fast bacilli identified in both pleural effusion and pleural tissue. In this subgroup of patients, pleural tuberculosis indicates a severe immunosuppression caused by HIV infection and, despite of the prompt introduction of correct treatment against tuberculosis, the response may be too late and the patient may not survive.

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