

Case Report of Lymph Nodal, Hepatic and Splenic Tuberculosis in an HIV-Positive Patient

Bianca Barone¹, Patrícia Lago Kreuzig¹,
 Patricia Medeiros Gusmão¹, Frederico de C. Escalera¹,
 Daniel Chamié¹, Sabrina Bezerra¹, Pedro Pinheiro¹,
 Pedro Coscarelli¹, Daurita Paiva¹, Leila Fonseca²,
 Anna Marsico³, André Cirigliano¹ and Mário Perez¹

¹Hospital Universitário Pedro Ernesto University Hospital (HUPE); ²Microbiology Institute, ³Thoracic Disease Institute, Federal University of Rio de Janeiro; Rio de Janeiro, RJ, Brazil

We describe a case of a male patient, 38 years old, HIV-positive (most recent CD₄ count about 259/mm³), with abdominal pain, nausea, vomiting, anorexia, weight loss, and vespertine high fever with chills. His hemogram showed normocytic and normochromic anemia, with a high erythrocyte sedimentation rate (ESR) and gross granulations in the neutrophils. Transaminases were normal. Bone marrow biopsy evidenced a chronic disease anemia pattern and a lack of infectious agents. Abdominal ultrasound examination showed a normal-size spleen, which exhibited heterogeneous parenchyma and multiple small hypoechoic images, together with small ascites, peripancreatic and para-aortic lymphadenopathy. These findings were confirmed by abdominal CT. The liver was normal in size, but had a hyperechoic image, which was not visualized on CT. Histopathological analysis of one of the multiple abdominal lymph nodes obtained by laparoscopic biopsy exhibited a chronic granulomatous inflammatory process, with caseous necrosis. Tissue sections were positive for BAAR (acid-alcohol-resistant bacillus), and the cultures were positive for *Mycobacterium tuberculosis*. Anti-tuberculosis treatment was begun, and the patient evolved with improvement of his general state, fever remission and weight gain. Splenic tuberculosis is a rare disease, occurring predominantly in patients in late stages of AIDS and/or disseminated tuberculosis. It is a difficult diagnosis, since there are no specific findings. Hence, complementary examinations, such as abdominal ultrasound/ CT, or fine needle aspiration, are usually necessary for investigation and differential diagnosis. Often, lesion regression after anti-tuberculosis regimens can be seen, and splenectomy is restricted to complicated or refractory disease.

Key Words: Tuberculosis, HIV, treatment.

The extra-pulmonary presentations of tuberculosis are more frequent in patients with AIDS than in those without HIV infection. [1-7]. Projections show that up to 70% of patients co-infected with HIV and *Mycobacterium tuberculosis* will develop at least one extra-pulmonary form of this mycobacteriosis [8-10]. Under these circumstances, the lymph nodal presentation is the most common, mostly involving peripheral lymph nodes. In abdominal forms, peritoneal [6,11-13] and lymph nodal [8] tuberculosis predominate, and they sometimes are associated with involvement of other solid organs, such as the liver. Splenic tuberculosis is very rare [1,8,14-17]; it occurs predominantly in patients with CD₄ counts less than 100/mm³ [1,18,19].

We report a case of an HIV-positive patient with hepatosplenic and lymph nodal forms of tuberculosis.

Case Report

A 38 year-old man, HIV positive with a most recent CD₄ count of 259/mm³ and a viral load of 9,300 copies/mm³ (nine months before the beginning of the symptoms) was being

Received on 13 December 2005; revised 17 March 2006.

Address for correspondence: Dr. Bianca Barone, M.D., bbarone@terra.com.br - Av. 28 de Setembro, 87/3o. andar, enfermaria 17/18 de Clínica Médica – Vila Isabel, Rio de Janeiro/ RJ, Brazil.

The Brazilian Journal of Infectious Diseases 2006;10(2):149-153.
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treated with anti-retroviral therapy (stavudine, lamivudine and efavirenz), presenting abdominal pain in the epigastric region that worsened when he ate. He was initially treated with 300 mg ranitidine daily, with transient remission of the pain, which returned after one week; this pain was then associated with nausea, vomiting, anorexia and two isolated episodes of diarrhea without infectious characteristics. After a week, he had begun to present vespertine high fever (about 39°C), with chills, weight loss (about 5 kg), eructation and heartburn.

This patient had previously been afflicted with lymph nodal tuberculosis seven years earlier, at the time that he was diagnosed to have HIV infection. At that time, he had been treated with rifampicin, isoniazid and pyrazinamide for six months, which provoked drug-related hepatitis. Despite this drug-related complication, the lymph nodal enlargement had complete resolution. Since then, he had been asymptomatic. The patient was homosexual, with a stable partner, and he denied intravenous drug use.

On clinical examination, the patient was lucid, oriented in time and space, cooperative, wasted, febrile, pale +/4+, hypotensive, tachycardic, acyanotic, anicteric and normopneic, without peripheral lymphadenopathy or oral candidiasis. Cardiovascular and respiratory examinations were unremarkable. He had abdominal tenderness in the epigastric region, without hepatosplenomegaly.

He brought exams from the day before admission, including a CD₄ count of 85/mm³ and a viral load of 1,300 copies/ mm³.

Pertinent laboratory findings on admission included: hemoglobin 7.23 g/dL, hematocrit 21.4%, ESR 72 mm/h, leucocytes 8,120/mL (immature neutrophils 18%, neutrophils 61.5%, lymphocytes 7.4%, monocytes 12%), and platelet count 248,000/mL. A peripheral blood smear showed macrocytosis (+/4+), with gross granulations inside the neutrophils. Some squizocytes and a few "hat" red cells were seen. The chest x-ray was normal. Bacteriological cultures of blood, urine and stools were negative. Serum albumin, iron, TIBC and ferritin were, respectively: 2.6 g/dL, 10 mg/dL, 150 mmol/L and 992.62 mg/dL. Serum sodium, total protein, transaminases, lactic dehydrogenase, amylase, glutamic-transferase and alkaline phosphatase were normal. Other relevant results were: serum potassium 5.8 mmol/L, urea 95 mg/dL and creatinine 2.7 mg/dL, which returned to normal range values after parenteral hydration.

Biopsy showed a normocellular bone marrow, with evident maturation of erythroid and myeloid series, and megaloblastosis. There were no infectious agents, nor was there malignancy. Prussian blue staining of the bone marrow showed probable chronic anemia, without mobilizations of iron body stores. Cultures for bacteria, fungi and mycobacteria were negative. Ziehl-Neelsen staining was negative.

Upper gastrointestinal digestive endoscopy showed mild active chronic atrophic gastritis. The specimen was negative for *Helicobacter pylori* and/or malignancies. On abdominal ultrasonography, there were small ascites, without hepatomegaly, but with an hyperechogenic image

on the upper region of the left lobe, of about 1.8 cm. The spleen was also normal in size, but with heterogeneous parenchyma and multiple small hypoechogenic images, with peripancreatic and para-aortic lymphadenopathy. Abdominal CT revealed multiple retroperitoneal lymph nodes, with peripheral enhancement and low-attenuation spots at the centers of the nodes (Figure 1). We could not see any hepatic lesions. Laparoscopy showed liver and spleen with multiple plain lesions on the surface, about 2-3 mm in size. There was lymphadenopathy in the hepatic hilum and para-aortic chains. The patient was submitted to a laparoscopic biopsy of one of the multiple abdominal lymph nodes. Hystopathological analysis was characterized by a chronic granulomatous inflammatory process, with giant cells, caseous necrosis and cells containing acid-fast bacilli (Figure 2). Culture of the tissue specimen was positive for *Mycobacterium tuberculosis*. The bacterial strain was susceptible to all anti-tuberculosis drugs tested (rifampicin, isoniazid, ethambutol, streptomycin and ethionamid). We began anti-tuberculous medication (rifampicin, isoniazid and pyrazinamide), and the patient evolved with improvement of general state, fever remission and weight gain, without complicating hepatitis. The patient was treated for nine months; follow-up ultrasonography was then performed, showing complete resolution of splenic abscesses, with calcifications still visible.

Figure 1. Abdominal CT showing multiple hypodense images in splenic parenchyma (two indicated by yellow arrows), associated with retroperitoneal enlarged lymph nodes, with peripheral enhancement and low-attenuation spots at the centers of the nodes (red arrows).

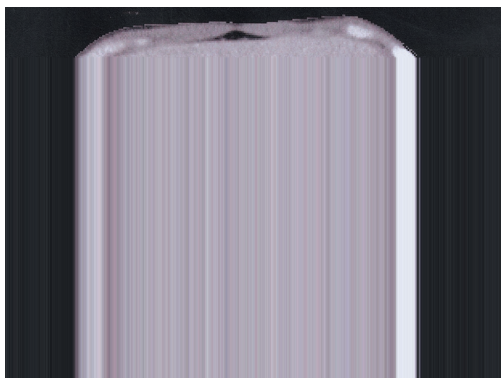
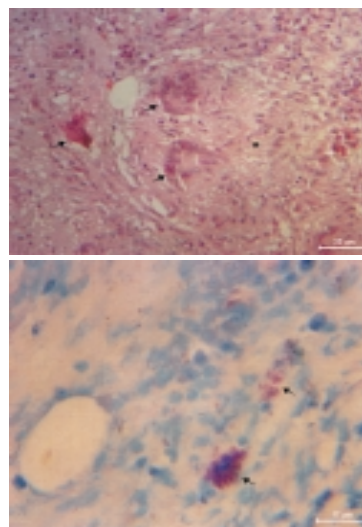


Figure 2. (2a) Paraffin section of an abdominal lymph node, showing a granulomatous reaction with many giant cells (arrows) and caseous necrosis (asterisk) (HE). (2b) Some cells contained acid-fast bacilli (arrows).



Discussion

In recent years, splenic tuberculosis case reports have come from areas with a high incidence of both HIV and tuberculosis, such as South Africa. However, reports of this presentation of the disease have also been made in Central Europe [20,21] and in other regions [22].

In Brazil, among AIDS reports notified by the Health Ministry, tuberculosis is the third commonest co-infection (24%), only surpassed by candidiasis (54%) and by *Pneumocystis jiruveci* pneumonia [23]. Tuberculosis may act as a co-factor that accelerates the clinical course of the HIV infection, which reduces survival more in HIV-positive than in seronegative patients [24].

Some studies have shown that splenic tuberculosis in HIV-positive patients is associated with intravenous drug use [1], which was not true in our patient. Splenic involvement is common in the milliary or disseminated forms of this mycobacteriosis [25], especially in HIV-positive patients with extra-pulmonary tuberculosis [1,26-28], but this isolated form of presentation is very rare [1,8,14].

Splenic tuberculosis is, generally, a difficult diagnosis, since there are no specific findings. Splenomegaly, pyrexia of unknown origin, with chills, weight loss, anorexia, diarrhea, abdominal pain, ascites, cough and lymphadenopathy, are some of the clinical findings of splenic tuberculosis in HIV-positive patients [1,8,26,29-40]. There are few reports of spontaneous splenic rupture due to this mycobacterial infection, causing acute abdominal pain (both in HIV-positive patients and in otherwise normal individuals) [14]; there are also few reports of hypersplenism with bleeding tendencies (petechiae, purpura, ecchymoses) [15-17,41,42]. Since clinical findings are unspecific, complementary exams are needed for clinical investigation and differential diagnosis in relation to other splenic lesions that can be seen in patients who have AIDS, such as other opportunistic infections [1,8,26,27,29,30-40], lymphomas and splenic Kaposi's sarcoma [1].

Among the complementary examinations that help diagnose splenic tuberculosis, abdominal ultrasonography and CT are the most informative. On abdominal ultrasound examination, the most suggestive findings are multiple small (< 1 cm), round hypoechogenic images, which are frequently hypodense on abdominal CT [6,8,11,21,26,43-50]. Splenomegaly [6,27,51-54] is also seen, and it is considered to be the most common feature of splenic involvement [55]. However, some reports point out the possibility of presentation with uniloculated pseudotumoral macronodules [5,6,11,22,46,56,57]. A hyperechogenic nodule, with or without calcification, has also been described [6,58,59]. Relative leucocytosis is another unspecific finding in splenic tuberculosis [21]. In some cases, it is possible to attain a final diagnosis by isolating *Mycobacterium tuberculosis* in blood cultures [8]. Unfortunately, when the spleen is the only organ affected, the final diagnosis is generally made by histopathological examination, using specimens obtained

with laparotomy or percutaneous fine needle aspiration [26,45,60-63].

The fact that HIV patients with splenic tuberculosis have lower CD₄ counts at the moment of diagnosis does not usually imply a poor prognosis. On the contrary, regression of the splenic lesions occurs only with specific drug treatment (triple drug regimen: rifampicin, isoniazid and pyrazinamide), and the time of treatment is not different from that proposed for the pulmonary forms (RFP, plus INH and PZA: two months; RFP plus INH: four months). Total resolution of splenic lesions may occur with this regimen. Some authors suggest that anti-tuberculosis treatment should be extended for 12 months, since patients often have the generalized form of this mycobacteriosis, even if initially presenting isolated splenic involvement [64]. As with other forms of tuberculosis, the time of treatment of the hepatosplenic form is similar to that proposed for non-HIV patients. However, when the acid-fast sputum smear takes a long time to become negative (i.e.: after two to three months of anti-tuberculous treatment), treatment should be maintained for nine months [65,66].

Some antiretroviral drugs have important interactions with anti-tuberculosis drugs, especially protease inhibitors and non-nucleoside reverse-transcriptase inhibitors, such as rifampicin and rifabutin. Treating tuberculosis should have priority, sometimes requiring changes in antiretroviral regimens. Patients already on antiretroviral therapy should have their regimen adapted to a compatible one during tuberculosis treatment. As soon as anti-tuberculosis drugs can be stopped, the drugs used before the diagnosis of tuberculosis can be reintroduced, depending on the patient's health status. In the case of recently-diagnosed HIV infection, there are two possibilities. The first one is to delay the introduction of antiretroviral therapy until the end of tuberculosis treatment; this strategy is easier, as it can reduce toxicity and eventual interactions between antiretroviral and anti-tuberculous drugs. The disadvantage of this option is the possibility of appearance of other HIV-related diseases during tuberculosis treatment, especially in patients with advanced stages of immunodeficiency, since tuberculosis causes an increase of HIV-viral load and/or a decrease in CD₄ count. The second option is to introduce the antiretroviral therapy a few months after the start of anti-tuberculosis drugs, since the side effects of these anti-tuberculosis drugs are more intense during the first months of use. This is the best option in the case of patients with a CD₄ count under 200/mm³ or with disseminated forms of tuberculosis. However, if the CD₄ count is very low, or there are manifestations of advanced immunodeficiency, the best choice is to start the antiretroviral regimen as soon as the tolerance to anti-tuberculous drugs has been established. The main antiretroviral regimens recommended during the treatment of tuberculosis are the association of two nucleoside reverse-transcriptase inhibitors with efavirenz or with the combination saquinavir/ ritonavir. During pregnancy, efavirenz is

contraindicated; nevirapine should replace this drug in such circumstances [65,66].

Follow-up ultrasonography 20 days - 22 months after the splenic tuberculosis diagnosis should be made, with the intention to verify complete resolution of splenic abscesses [1,19,21,27]. In refractory cases, splenectomy is proposed [1,21,26,27,32,35,37,39,40]. Few studies report the use of corticoid adjuvant treatment in slow-responding cases, with the intention to prevent an eventual need for therapeutic splenectomy [8,19].

Splenic rupture is another absolute indication for splenectomy in primary treatment of splenic tuberculosis, but anti-tuberculous drugs must be used as complementary treatment. In these cases, conservative surgeries should not be proposed, due to the fragility of splenic tissue and the high risk of recurrence [14].

Acknowledgements

The authors are indebted to Dr. Marcelo Pelajo-Machado (Department of Pathology – Instituto Oswaldo Cruz – FIOCRUZ, Rio de Janeiro/ RJ, Brazil) for constructive comments and for technical support.

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