



A case of isolated splenic tuberculosis



Johanna V. Basa*, Lilly Singh, Wassim Abi Jaoude, Gainosuke Sugiyama

SUNY Downstate Medical Center, 450 Clarkson Avenue, Box 40, Brooklyn, NY 11203, United States

ARTICLE INFO

Article history:

Received 1 November 2013
Received in revised form 26 August 2014
Accepted 10 October 2014
Available online 23 December 2014

Keywords:

Extrapulmonary
Spleen
Tuberculosis

ABSTRACT

There are few cases of isolated splenic tuberculosis reported in the literature internationally, and nearly none from western medical centers. The incidence of tuberculosis has declined in the United States since the 1950s, with 11,585 reported cases in 2009, 21% of which were exclusively extrapulmonary. Splenic tuberculosis occurs mostly as part of miliary tuberculosis in immunocompromised patients. Isolated splenic tuberculosis is extremely rare, particularly in the immunocompetent patient. Patients susceptible to acquiring splenic tuberculosis usually have one of the following risk factors: immunosuppression, preceding pyogenic infections, splenic abnormalities, prior trauma to the spleen, sickle cell disease and other hemopathies, and in the immunocompetent patient another body site infected by *M. tuberculosis*. In this report we present the case of a young immunocompetent male with no other significant past medical history with isolated splenic tuberculosis.

Published by Elsevier Ltd. on behalf of Surgical Associates Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/3.0/>).

1. Introduction

There are few cases of isolated splenic tuberculosis reported in the literature internationally, and nearly none from western medical centers. The incidence of tuberculosis has declined in the United States since the 1950s, with 11,585 reported cases in 2009, 21% of which were exclusively extrapulmonary.

Splenic tuberculosis occurs mostly as part of miliary tuberculosis in immunocompromised patients. Isolated splenic tuberculosis is extremely rare, particularly in the immunocompetent patient. Patients susceptible to acquiring splenic tuberculosis usually have one of the following risk factors: immunosuppression, preceding pyogenic infections, splenic abnormalities, prior trauma to the spleen, sickle cell disease and other hemopathies, and in the immunocompetent patient another body site infected by *M. tuberculosis*. In this report we present the case of a young immunocompetent male with no other significant past medical history with isolated splenic tuberculosis.

2. Presentation of case

A 27 year old African American male presented to the emergency department with epistaxis for three days. He had associated generalized weakness, anorexia, unintentional 50 lb weight loss, and night sweats. His history was significant for 10 years incarceration; he was released five months prior to this admission and his health subsequently deteriorated with regards to weight loss and fatigue.

Physical exam revealed a cachectic, afebrile man with a tender abdomen and hepatosplenomegaly. His blood work was remarkable for platelet count of 8000, AST 59, alkaline phosphatase 606 and LDH 388. HIV ELISA was negative. He was admitted with a working diagnosis of idiopathic thrombocytopenic purpura.

On his first hospital day, PPD was placed and was negative. He was started on IVIg at 0.5 mg/kg. Computed tomography (CT) imaging of abdomen and pelvis showed splenomegaly with diffuse heterogenous enhancement and innumerable lesions, as well as diffuse lymphadenopathy in the porta hepatis, splenic hilum, retroperitoneum and pelvis. Homogenous hepatomegaly without focal hypodensities was also noted (Fig. 1). CT of the chest showed 1.9 cm left lung lower lobe nodule with irregular margins, bilateral hilar lymphadenopathy, and multiple enlarged mediastinal lymph nodes were present. Whole body PET scan was performed to evaluate for lymphoma and metastatic disease and showed splenomegaly with hypermetabolic FDG activity, with hypermetabolic lymphadenopathy in mediastinum, retroperitoneum, mesentery and pelvis.

Bone marrow biopsy was performed and revealed megakaryocytic hyperplasia. On day three of IVIg treatment he showed no major improvement and therapy with decadron initiated. On fifth hospital day, his platelet count was less than 5000 and he was subsequently given platelet transfusion and evaluated by surgery for splenectomy. On seventh hospital day, a repeat CT of the abdomen showed significant gastric varices and portal hypertension. Splenectomy was performed due to failure of medical management. The splenectomy was approached via laparotomy due to the preoperative evidence of gastric varices. Operative findings were significant for large hepatosplenomegaly and highly-vascularized splenic ligaments. The patient's postoperative course

* Corresponding author. Tel.: +1 718 270 2683; fax: +1 718 270 4676.
E-mail address: Johanna.basa@gmail.com (J.V. Basa).



Fig. 1. CT abdomen and pelvis showing splenomegaly with diffuse lymphadenopathy in the porta hepatis, splenic hilum and retroperitoneum.



Fig. 2. Gross appearance of the spleen shows irregular nodularity.

was complicated by fungemia; he was treated and discharged home.

The weight of the spleen was 1430 grams and it measured $33 \times 18 \times 8$ cm. On gross appearance, the surface of the spleen was irregularly nodular and brown (Fig. 2). The entire spleen was replaced by caseating granulomas and necrotizing-type granulomatous inflammation (Figs. 3 and 4). Mycobacterium tuberculosis bacteria were seen by AFB stain. Splenic hilar lymph nodes with non-caseating granulomas were also visualized.

3. Discussion

Tuberculosis of the spleen was first described in literature in 1846 by Coley referring to enlarged spleen secondary to tuberculosis with absent or limited involvement of other organs.⁸ Approximately 15–20% of all cases of TB are extrapulmonary,⁴ and of these, 3–11% are abdominal.^{4,10} As a corollary to the lack of isolated splenic tuberculosis in the United States, it is necessary to turn to international literature for guidelines for diagnosis and treatment of these patients.

Clinically, the most common symptoms that patients present with are fever (82.3%), fatigue and weight loss (44.12%), and splenomegaly (13.2–100%).^{7,11} Rarely, patients can be asymptomatic and their diagnosis is delayed. Pain is an uncommon

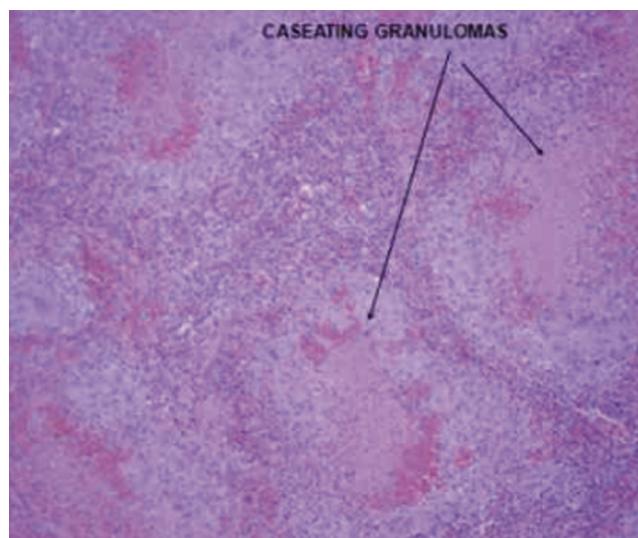


Fig. 3. Microscopic view of the spleen showing caseating granulomas.

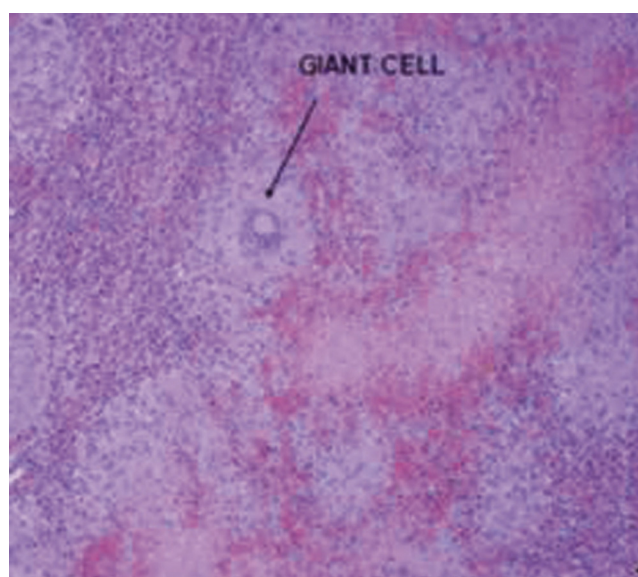


Fig. 4. Microscopic view of the spleen showing granulomatous inflammation.

symptom.⁶ Other presentations include splenic rupture,⁴ hypersplenism, portal hypertension with and without gastrointestinal bleed,¹¹ and fulminant form involving rapid progression of fever, cachexia, hemorrhage and sepsis.¹¹ Splenic tuberculosis patients can also present with hematologic abnormalities. Usually they are cytopenic, but cases of polycythemia have also been reported.¹

Diagnosis. PPD is often positive in these patients. However, this is unreliable in endemic countries, immunocompromised patients, and foreign-born patients who received BCG vaccine.¹ PCR has much higher sensitivity and specificity, and can identify organism to the species.^{1,6} Sonogram can help differentiate miliary tuberculosis, nodular, tuberculous abscess, calcific tuberculosis, and mixed type.¹³ Tuberculomas can also be identified as multiple hypochoic lesions, well-demarcated with posterior enhancement. When these are visualized, lymphoma, acute leukemia, angiomas, metastases, and fungal infections should also be ruled out. On computed tomography, tuberculomas appear as non-enhancing homogenous hypodensities and homogenous splenomegaly can also be appreciated.^{11,12} Fine needle aspiration cytology is a valuable

too, with sensitivity of 88% and specificity of up to 100%.^{10,11} It should be considered whenever splenomegaly presents with fever of unknown origin. FNAC can also reveal caseating granulomas.^{11,13} Laparoscopy may be indicated when less invasive diagnostic methods have failed.^{9,11} Splenectomy should be a last resort for diagnosis.

Treatment. Antituberculous medications are the first line of treatment.^{1,4,5,7,11,13} Triple or quadruple therapy for at least 12 months is indicated.^{4,5,13} Splenectomy is usually unnecessary as a mode of treatment, except for under particular conditions.^{4,11} These indications include failure of medical treatment, cytopenia or polycythemia, tuberculous splenomegaly with gastrointestinal bleeding secondary to portal hypertension, failure of percutaneous abscess drainage, and multiple splenic abscesses. Antituberculous

therapy should also be initiated in patients status post splenectomy. Imaging studies such as ultrasound or computed tomography can be used to gauge the success of treatment.¹¹ MRI and PET scans are useful in determining the amount of activity in splenic lesions and differentiating active vs. fibrotic scars.¹²

4. Conclusion

Isolated splenic tuberculosis is rare and is usually associated with immunosuppression, though not exclusively. Usually there is another focus of infection. Splenectomy should be reserved for diagnostic uncertainty, cases refractory to medical treatment, and in specific conditions as mentioned above.

Key learning points

- Isolated splenic tuberculosis is such a rare entity, that only small case reports such as ours are found in the literature.
- We hope that our case highlights one of the many ways splenic tuberculosis in a patient that is immunocompetent may present.
- If one is found to have a patient with similar laboratory findings, signs and symptoms they will be able to place isolated splenic tuberculosis in their diagnostic differential.

Conflicts of interest

There are no conflicts of interest to disclose.

Ethical approval

None.

Authors contributions

Dr. Basa is the first author. Dr. Singh was responsible for data collection and literature review and Drs. Gainosuke and Abe Jauode also contributed equally to the article. Drs. Gainosuke and Basa accept full responsibility for the work.

References

1. Berady S, Rahbi M, Bahrouch L, Sair K, Benzaine H, Benkirane A, et al. La tuberculose splénique pseudotumorale. *La Revue de Médecine Interne* 2005;**26**: 588–91.
2. Hamizah R, Rohana AG, Anwar SA, Ong TZ, Hamazaini AH, Zuikarnaen AN. Splenic tuberculosis presenting as pyrexia of unknown origin. *Med J Malaysia* 2007;**62**(1):70–1.
3. Ho PL, Chim CS, Yuen KY. Isolated splenic tuberculosis presenting with pyrexia of unknown origin. *Scand J Infect Dis* 2000;**32**:700–1.
4. Imani Fooladi AA, Hosseini MJ, Azizi T. Splenic tuberculosis: a case report. *Int J Inf Dis* 2009;**13**:e273–5.
5. Mazloom W, Marion A, Ferron C, Lucht F, Mosnier JF. Tuberculose splénique: a partir d'un cas et revue de la littérature. *Medicine et Maladies Infectieuses* 2002;**32**:444–6.
6. Meredith HC, Early JQ, Becker W. Tuberculous splenomegaly with the hyper-splenism syndrome. *Blood* 1949;**4**:1367–73.
7. Meshikhes AN, Al-Momen SAM. Laparoscopic diagnosis of splenic tuberculosis. *Surg Laparosc Endosc Percutan Tech* 2006;**16**(5):355–6.
8. Pottakkat B, Kumar A, Rastogi A, Krishnani N, Kapoor VK, Saxena R. Tuberculosis of the spleen as a cause of fever of unknown origin and splenomegaly. *Gut Liver* 2010;**1**(4):94–7.
9. Rhazal F, Lahlou MK, Benamer S, Daghri JM, Essadel E, Mohammadine E, et al. Splénomégalie et pseudotumeur splénique d'origine tuberculeuse: six nouvelles observations. *Ann Chir* 2004;**129**:410–4.
10. Sharma SK, Smith-Rohrberg D, Tahir M, Mohan A, Seith A. Radiological manifestations of splenic tuberculosis: a 23-patient case series from India. *Indian J Med Res* 2007;**125**:669–78.
11. Zhan F, Wang CJ, Lin JZ, Zhong PJ, Qiu WZ, Lin HH, et al. Isolated splenic tuberculosis: a case report. *World J Gastrointest Pathophysiol* 2010;**3**(1):109–11.

Open Access

This article is published Open Access at scimedirect.com. It is distributed under the [IJSCR Supplemental terms and conditions](#), which permits unrestricted non commercial use, distribution, and reproduction in any medium, provided the original authors and source are credited.