Case Report

# Hepatosplenic tuberculosis, a rare presentation

*Javvid Muzamil, Naveed Mohsin[1], Firdousa Nabi[2], Shabir Ahmed Rather[1]*

Departments of Medical Oncology and [1]Internal Medicine, SKIMS, Soura, [2]Department of Prosthodonsia, Government Dental College, Srinagar, Jammu and Kashmir, India

## ABSTRACT

Tuberculosis (TB) is one of the known endemic diseases in this part of world; the most common organ involvement is lung. Among extrapulmonary involvements, tubercular lymphadenitis is the most common. Gastrointestinal TB is very uncommon, making up to 3.5% of extrapulmonary TB. Hepatosplenic TB with abdominal tubercular lymphadenitis is very rare form of TB, accounting <1%. We are describing a patient who had lymphoma-like presentation and at the end proved to be a rare form of TB.

**Key words**: Acid fast *Bacillus*, Epstein–Barr virus, polymerase chain reaction, tuberculosis

## INTRODUCTION

Tuberculosis (TB) is one of the oldest infectious diseases known to affect humans and is a major cause of death worldwide. The disease is caused by *Mycobacterium tuberculosis* complex and usually affects the lungs, although extrapulmonary involvement occurs up to one-third of cases. Most common extrapulmonary TB is lymphadenitis followed by plural, genitourinary, bones and joints, meninges, peritoneum, pericardium, gastrointestinal, and hepatosplenic TB.

TB is known to involve the hepatosplenic axis in different ways. Miliary TB is the most common and is said to occur in 50-80% of all patients dying of pulmonary TB.[1] Although isolated hepatobiliary TB is less common and constitutes <1% of all cases.[2] Hepatic TB is classified into three categories viz., miliary, granulomatous, and localized hepatic form.[3] The pathophysiology of localized hepatic TB is similar to other forms of extrapulmonary TB, which results from hematogenous dissemination of the infection through the hepatic artery, the portal vein, or lymphatics.[4-6]

Histopathological examination is needed for the exact diagnosis of hepatosplenic TB.[7] Hepatic granulomas may also be observed in other infectious diseases such as brucellosis, infectious mononucleosis, chronic hepatitis, and fungal infections, or noninfectious diseases such as early-stage primary biliary cirrhosis, sarcoidosis, Hodgkin's disease, Crohn's disease, drug hypersensitivity, and extrahepatic biliary obstruction.[2,8] Epithelioid granuloma can be demonstrated in liver TBC in 80-100% of cases, caseation necrosis in 30-83%, and acid fast *Bacillus* (AFB) on smear examination in 0-59% of the cases.[4] In our case, we could not detect AFB on direct examination. Detection of mycobacterial DNA on tissue specimen by polymerase chain reaction (PCR) has 88% sensitivity and 100% specificity. In one study, PCR was positive in 57% of hepatic granulomas.[2] In our case, PCR test was also negative on the tissue specimen.

**Address for correspondence**:
Dr. Javvid Muzamil, Married Hostel, Room No. F16,
SKIMS, Soura, Srinagar - 190 011, Jammu and Kashmir, India.
E-mail: javvidmd@gmail.com

| Access this article online | |
|---|---|
| Quick Response Code: | Website: www.caijournal.com |
| | DOI: 10.4103/2225-6482.166083 |



**How to cite this article:** Muzamil J, Mohsin N, Nabi F, Rather SA. Hepatosplenic tuberculosis, a rare presentation. Community Acquir Infect 2015;2:103-6.

Imaging is nonspecific in hepatosplenic TB and actual diagnosis is established by histopathology. Treatment of abdominal TB is same as other extrapulmonary TB.

## CASE REPORT

A 60-year-old male, nonsmoker presented with fever, more during evenings, with diffuse noncolicky pain abdomen for a period of 1-month. It was associated with loss of appetite and easy fatigability. There was no history of night sweats, cough, hemoptysis or apparent bleeding, weight loss, and altered bowel habits. There was no significant past and family history. Examination was unremarkable except pallor and splenomegaly (3 cm below left costal margin).

On evaluation, complete hemogram revealed pancytopenia with Hb 8 g/dL, total lymphocyte count 2 × $10^3$/μL, platelets of 80 × $10^3$/μL with peripheral blood film revealing neutrophils 55%, lymphocytes 35%, monocytes of 5%, and no atypical cell. Other lab investigations like alkaline phosphatase were 320 IU/L ($n$ = 150-250), and lactate dehydrogenase was 643 ($n$ ≤ 300). Rests of investigations like kidney function test, liver function test, arterial blood gas electrolytes, uric acid, and coagulogram were normal. Serology for hepatitis B, C, HIV, and Epstein–Barr virus was negative. Brucella serology was nonreactive. Blood and urine cultures were sterile.

Both chest X-ray and electrocardiogram were normal. Ultrasound abdomen revealed peripancreatic, periportal and aortocaval lymphadenopathy, splenomegaly with a multiple hypoehoic lesion in the spleen, and cholelithiasis with mild ascites. Contrast computed tomographic (CT) abdomen with chest revealed gastric wall thickening, cholelithiasis, perigastric and retroperitoneal nodes, and massive splenomegaly with multiple hypodense lesions with prominent portosplenic axis [Figures 1 and 2]. Likely provisional diagnosis was lymphoma and patient was further investigated on those lines.

Bone marrow aspiration revealed cellular marrow with megaloblastic erythroid hyperplasia and adequate iron stores. Bone marrow biopsy revealed megaloblastic erythropoiesis, normal myelopoiesis, increased eosinophilic precursors, and adequate megakaryocytes. Cytogenetic analysis (karyotyping) was normal.

Both endoscopy and colonoscopy were normal. Diagnosis still remained obscured and patient underwent laparotomy with splenectomy, cholecystectomy, and liver biopsy. Intraoperatively liver, spleen, mesentery, and peritoneum were studded with miliary deposits with retroperitoneal lymphadenopathy. Histopathological examination of spleen revealed multiple epithelioid granulomas exhibiting caseation necrosis [Figure 3]. Histopathological examination of liver revealed distorted hepatic architecture with presence of parenchymal caseation epithelioid granulomas along with Langerhans type giant cells [Figure 4]. Hilar and hepatoduodenal lymph nodes also showed presence of caseation granulomas. AFB stains and PCR came negative.

Our initial diagnosis was a lymphoproliferative disorder which got ruled out after histopathology. So the final diagnosis was hepatosplenic TB. This is a rare form of abdominal TB, and it mimics lymphoma presentation. Even though no tubercle *Bacillus* was seen in biopsy specimen but caseation necrosis is pathognomic of TB.

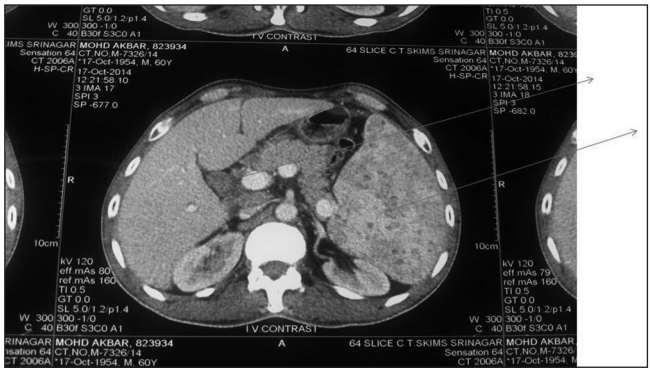


**Figure 1: Contrast-enhanced computed tomography abdomen showing multiple miliary shadows of spleen with abdominal lymphnodes**

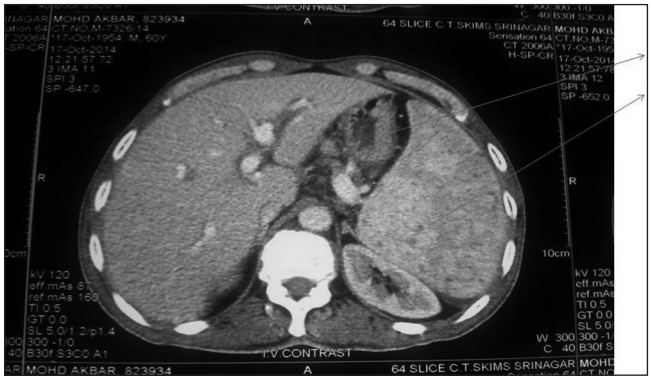


**Figure 2: Contrast-enhanced computed tomography abdomen showing miliary splenic shadows with thick gastric wall**

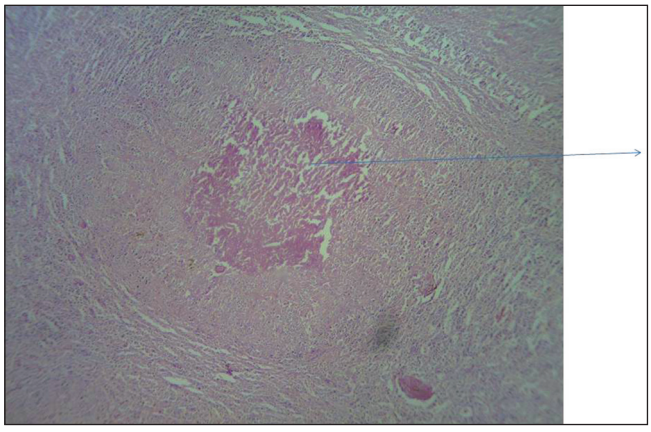

**Figure 3: Histopathology of spleen showing caseation granuloma**

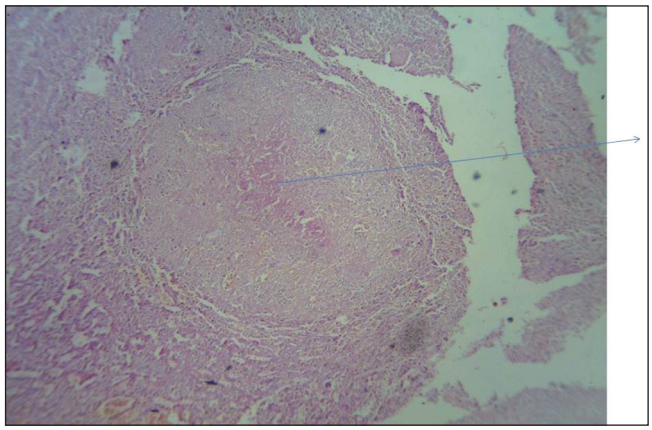

**Figure 4: Liver biopsy histopathology showing granuloma and caseation necrosis**

Patient was put on antitubercular drugs (four drug regimen) and he showed remarkable response in clinical and laboratory parameters and repeat scans after 9 months were absolutely normal. ATT was continued for a total 9 months. Patient is on follow-up and is doing well without any treatment.

## DISCUSSION

TB is known to involve the liver in different ways. Miliary TB of the liver is the most common and is said to occur in 50-80% of all patients dying of pulmonary TB.[1] Leader, in an extensive review of the world literature in 1952, documented only 80 cases of hepatic TB with large abscesses and nodules or tuberculomas and classified TB of the liver into miliary, which is part of generalized miliary TB and local, which he further divided into focal or nodular TB (to include tuberculous hepatic abscess and tuberculomas) and into the tubular form (involving intrahepatic ducts).[9]

Hepatobiliary TB has a 2:1 male preponderance with the majority falling within the 11-50-year-old age group[10] with a peak age incidence in the second decade of life in both sexes. The majority of localized hepatic TB reported in the literature occurs in the 30-50-year-old age group.[11] Our patient was also male and was in sixth decade of life. More than half of the 130 cases reported by the author in 1983[10] were symptomatic for more than 1-year prior to admission. Abdominal pain appeared to be the most important symptom of hepatobiliary TB in several series. Abdominal pain was present in 45% of the patients in the jaundiced group, often associated with fever and chills. In the nonjaundiced group, abdominal pain was present in 39% of cases. Hersch, in a study of 200 black South African patients reported abdominal pain in approximately half of the patients in his series although localized hepatic TB was present only in 14% of the cases.[12] Fever was present in more than 60% in four large series.[10,12-14] Our patient also presented with pain abdomen and fever. Hepatomegaly was the most common finding present in 96% of the cases reported by the author.[11] The enlarged liver was nodular in 55% of cases simulating cancer of the liver and it was tender in 36% of cases simulating liver abscess.[11] Hersch reported hepatomegaly in 95% of his patients with localized hepatic TB, half of them with hepatic tenderness[12] while Essop *et al*.[13] reported it in 80% and Maharaj *et al*.[14] in 95% of their patients. Splenomegaly was present in 25-57% of cases.[10,12-14] Our patient had only splenomegaly without jaundice. Jaundice was seen in 35% of the cases reported by the author.[10] It was obstructive in nature, simulating other conditions that exhibit extrahepatic biliary obstruction. Jaundice occurred in a minority of patients in the other reports. Essop *et al*. reported elevated alanine transaminase and aspartate transaminase in 70% of their cases.

Chest X-ray showed abnormalities in 65% of the cases reported by the author[10] demonstrating pulmonary TB but were negative in 35% of cases. Maharaj *et al*. reported normal chest X-ray in 22% of cases.[14] Our patient also had normal chest X-ray. Ultrasound of the liver showed hypoechoic lesions and complex masses, particularly in those reports with tuberculous liver abscess, and could not be differentiated from carcinoma.[11,15-17] CT scans of the liver can show solitary or multiple focal masses due to a large tuberculoma or tuberculous liver abscess, which can be difficult to differentiate from malignancy.[11,15-17]

The most common findings on CT that are highly suggestive of abdominal TB are high density ascites, lymphadenopathy, bowel wall thickening, and irregular soft tissue densities in the omental area.[18-21] Involvement of the liver and spleen in miliary TB may appear on CT as tiny low density foci widely scattered throughout the organ as was evident in our case too. The macro nodular form of hepatosplenic TB may be seen as multiple low attenuation (15-50 HU), 1-3 cm round lesions, or simple tumor like masses. The lesions may show peripheral enhancement after intravenous contrast administration.[22]

Laparotomy is definitely indicated where malignancy cannot be ruled out with certainty. Our patient also had diagnostic dilemma that is why he underwent laparotomy. In many patients, it may not be possible to rule out malignancy even at laparotomy. A frozen section examination may help in such cases. A mesenteric lymph node should always be removed in such cases as caseation and granulomas are much more likely to be present in lymph nodes than intestinal lesions.[23,24]

Histopathology is the gold standard for detecting TB, as was evident in our case. Treatment of hepatosplenic TB is same as other form of extrapulmonary TB. Finally, our patient was also treated with four antitubercular drugs and all symptoms and signs abated within 3 months, and treatment was continued for total of 9 months.

## CONCLUSION

Every patient of lymphoma-like presentation is not lymphoma, a thorough work up is needed to rule out same and infectious diseases in endemic area must be looked for.

### Financial support and sponsorship
Nil.

### Conflicts of interest
There are no conflicts of interest.

## REFERENCES

1. Morris E. Tuberculosis of the liver. Am Rev Tuberc 1930;22:585-92.
2. Arora R, Sharma A, Bhowate P, Bansal VK, Guleria S, Dinda AK. Hepatic tuberculosis mimicking Klatskin tumor: A diagnostic dilemma. Indian J Pathol Microbiol 2008;51:382-5.
3. Lewis JH. Granuloma of the liver. In: Schiff ER, Sorrell MF, Maddrey WC, editors. Schiff's Diseases of the Liver. 10th ed., Vol. 2. Philadelphia: Lippincott Williams & Wilkins; 2007. p. 2204-6.
4. Puri AS, Nayyar AK, Vij JC. Hepatic tuberculosis. Indian J Tuberc 1994;41:131-4.
5. Drebber U, Kasper HU, Ratering J, Wedemeyer I, Schirmacher P, Dienes HP, *et al.* Hepatic granulomas: Histological and molecular pathological approach to differential diagnosis — A study of 442 cases. Liver Int 2008;28:828-34.
6. Saluja SS, Ray S, Pal S, Kukeraja M, Srivastava DN, Sahni P, *et al.* Hepatobiliary and pancreatic tuberculosis: A two decade experience. BMC Surg 2007;7:10.
7. Parsak CK, Hanta I, Aslan A, Alabaz O. Isolated hepatic tuberculosis presenting as cystic-like and tumour-like mass lesions. Case Rep Gastroenterol 2008;2:18-21.
8. Desai CS, Josh AG, Abraham P, Desai DC, Deshpande RB, Bhaduri A, *et al.* Hepatic tuberculosis in absence of disseminated abdominal tuberculosis. Ann Hepatol 2006;5:41-3.
9. Leader SA. Tuberculosis of the liver and gall-bladder with abscess formation: A review and case report. Ann Intern Med 1952;37:594-606.
10. Alvarez SZ, Carpio R. Hepatobiliary tuberculosis. Dig Dis Sci 1983;28:193-200.
11. Mustard RA, Mackenzie RL, Gray RG. Percutaneous drainage of a tuberculous liver abscess. Can J Surg 1986;29:449-50.
12. Hersch C. Tuberculosis of the liver. A study of 200 cases. S Afr Med J 1964;38:857-63.
13. Essop AR, Posen JA, Hodkinson JH, Segal I. Tuberculosis hepatitis: A clinical review of 96 cases. Q J Med 1984;53:465-77.
14. Maharaj B, Leary WP, Pudifin DJ. A prospective study of hepatic tuberculosis in 41 black patients. Q J Med 1987;63:517-22.
15. Blangy S, Cornud F, Sibert A, Vissuzaine C, Saraux JL, Benacerraf R. Hepatitis tuberculosis presenting as tumoral disease on ultrasonography. Gastrointest Radiol 1988;13:52-4.
16. Brauner M, Buffard MD, Jeantils V, Legrand I, Gotheil C. Sonography and computed tomography of macroscopic tuberculosis of the liver. J Clin Ultrasound 1989;17:563-8.
17. Epstein BM, Leibowitz CB. Ultrasonographic and computed tomographic appearance of focal tuberculosis of the liver. A case report. S Afr Med J 1987;71:461-2.
18. Epstein BM, Mann JH. CT of abdominal tuberculosis. AJR Am J Roentgenol 1982;139:861-6.
19. Hulnick DH, Megibow AJ, Naidich DP, Hilton S, Cho KC, Balthazar EJ. Abdominal tuberculosis: CT evaluation. Radiology 1985;157:199-204.
20. Bankier AA, Fleischmann D, Wiesmayr MN, Putz D, Kontrus M, Hübsch P, *et al.* Update: Abdominal tuberculosis — Unusual findings on CT. Clin Radiol 1995;50:223-8.
21. Suri S, Gupta S, Suri R. Computed tomography in abdominal tuberculosis — Pictorial review. Br J Radiol 1999;72:92-8.
22. Buxi TB, Vohra RB, Sujatha Y, Chawla D, Byotra SP, Gupta PS, *et al.* CT appearances in macronodular hepatosplenic tuberculosis: A review with five additional new cases. Comput Med Imaging Graph 1992;16:381-7.
23. Bhansali SK. Abdominal tuberculosis. Experiences with 300 cases. Am J Gastroenterol 1977;67:324-37.
24. Tandon HD, Prakash A. Pathology of intestinal tuberculosis and its distinction from Crohn's disease. Gut 1972;13:260-9.