Case report: tuberculosis liver abscess in male alcoholism patient

W P Siahaan* and F Ginting

1 Faculty of Medicine, Universitas Sumatera Utara, Medan, Indonesia
2 Division of Infectious and Tropical Diseases, Department of Internal Medicine, Haji Adam Malik Hospital, Universitas Sumatera Utara, Medan, Indonesia
*Corresponding author: Widyaprawirani@rockemail.com

Abstract. A liver abscess often occurs in low-middle income countries such as Indonesia. Two most common liver abscesses are amoebic and pyogenic liver abscess. Data that reported tuberculosis liver abscess (TLA) is extremely rare. A diagnostic criterion for tuberculosis liver abscess is rare and remains unclear. A 52-year-old man developed a TLA which was not associated with any pulmonary or gastrointestinal tract foci of tuberculosis. An ultrasonogram and abdominal scan showed an abscess in the right lobe. We performed paracentesis, and the pus from the lesion was positive tubercular bacilli on acid-fast bacilli staining. The patient was started on systemic antitubercular therapy to which he responded favorably. This report emphasizes the fact that, although a TLA is a very rare entity, it should be included in the differential diagnosis of liver abscess especially in Indonesia where the prevalence of tuberculosis is extremely high.

1. Introduction
Tuberculosis is a very common disease in the developing country especially in Indonesia, based on WHO report, Indonesia is on the second rank worldwide with the total incidence of tuberculosis is 1,020,000 in 2015.[1] TLA is a rare form of extra pulmonary tuberculosis, and only a few cases have been reported in the literature worldwide. Symptoms of the disease are non-specific and commonly confused with pyogenic amoebic abscess or hepatoma and can cause misdiagnosed and a wrong given therapy.[2] The definitive diagnosis can only be confirmed by histological and bacteriological investigations.[3] TLA is very rare in Indonesia whereas the number of tuberculosis cases is very high. This should be a warning sign for clinicians that whenever a diagnosis of liver abscess is found in a patient, the diagnosis of tuberculosis has to be raised as a differential diagnosis as well. Here, we report a case of TLA in alcoholism male patient and review the related literature.

2. Case report
A 52 years old man, alcoholism, was admitted to Adam Malik hospital with no radiating pain in the right hypochondria and epigastrum associated with vomiting, intermittent fever with chills, and anorexia for 12 days. There was no previous history of a cough, night sweats, hemoptyisis, body weight loss or direct contact with any tuberculosis patient. He had been immunocompetent and had no history of diabetes mellitus and hypertension.
On vital sign examination, his blood pressure was 120/80 mm Hg, pulse rate 78/min, respiratory rate 20/min, and temperature 37.8°C. Physical examination revealed mild right hypochondriaknocking pain. Laboratory test showed normal complete blood count. Liver function was normal except for globulin 1.8 g/dL (2.6 – 3.6 g/dL). Chest X-ray showed no lesson suggestive of TB (figure 1), therefore we performed ultrasonography and abdominal scan, and this showed large hypoechoic lesion 61.1 mm x 53.7 mm on the right lobe of the liver and the lesion also appeared in the above diaphragm suggestive of liver abscess (figure 2). A contrast-enhanced CT – Scan showed a large 6.4 x 6.4 x 5 cm hypodense lesion, with discrete and hyperdense border involving segment 8 right quadrant of the hear lobes suggestive of liver abscess (Figure 3).

**Figure 1.** A normal chest x-ray.

**Figure 2.** Ultrasonogram of the liver showed a hypoechoic lesion on the right lobe.

**Figure 3.** A contrast enhanced CT – Scan showed hypodense lesion, with discrete and hyperdense border involving segment 8 right qudran of the hepar lobes.
Paracentesis was carried out, and pus cultures were performed to detect the presence of bacteria or fungus (Figure 4). We found 100 cc yellowish like caseous necrosis and granulomatous pus and the aspirate was then sent for microbiological investigation (figure 5).

![Figure 4. Paracentesis.](image1.png) ![Figure 5. Seropurulent coloured pus aspirated.](image2.png)

We performed aerobic and anaerobic cultures, eosin and Lugol staining to find amoebic and both showed a negative result, but on the Acid-fast bacilli (AFB) staining we found AFB-positive tubercular bacilli and confirmed the diagnosis of tuberculosis. The patient was treated with oral tuberculosis fixed-dose combination. The fever and pain of right upper quadrant disappeared after 5 days of treatment.

3. Discussion

Hepatic tuberculosis is a rare form of extrapulmonary tuberculosis even in Indonesia where the incidence of tuberculosis is high. Reed et al. described three morphologic types of hepatic tuberculosis: (1) miliary tuberculosis of the liver associated with generalized miliary tuberculosis, (2) primary miliary tuberculosis of the liver without the involvement of other organs, and (3) primary tuberculosis granuloma or abscesses of the liver. The TLA is extremely rare.[4] Most of hepatic tuberculosis usually occur simultaneously with miliary lung tuberculosis.[5] In our patient, there was no evidence of any pulmonary or gastrointestinal tuberculosis which is a rare form of hepatic tuberculosis called isolated hepatic tuberculosis liver abscess. Isolated tuberculosis liver abscess rarely occurs probably because there is low oxygen level in the liver which is an unfavorable place for bacilli to grow. It usually has a primary focus in the lung or gastrointestinal tract (GIT), and then spreads to the liver via hepatic artery, and less commonly via the portal vein.[6]

The sign and symptoms of a hepatic abscess are non-specific which often lead to a misdiagnosis and diagnostic delay. Due to the non-specific sign and symptoms, the diagnosis is usually made at biopsy or during laparotomy.[2] The symptoms usually present with fever, anorexia, weight loss, abdominal pain, hepatomegaly, and splenomegaly.[7] Similar to our case, the symptoms that were found included fever, abdominal pain, anorexia and weight loss. Jaundice is a rare sign of TLA and may be caused by extra or intrahepatic obstruction.[2,8]

The laboratory finding of TLA is non-specific. The complete blood count of TLA usually within normal limit, unlike amoebic or pyogenic liver abscess which usually presents with elevated leukocytes.[9] Another test that is commonly used to diagnose TLA is liver function test, although it is said that the test is non-specific. There are many different opinions regarding serum-alkaline phosphatase (SAP) as one of the markers to diagnose TLA, some authors said that elevated SAP is indicated as hepatic TB, even though the normal level of SAP is usually found in most cases like our case.[10] There are several standard markers that can be used in diagnosing TLA such as serum albumin, serum globulin, albumin/globulin ratio, and gamma-glutamyl transpeptidase but all of these...
are categorized as non-specific tests. Moreover, we can not rely completely on this tests in diagnosing hepatic TB.[10] There are several imaging tools can be used in diagnosing TLA, such as ultrasonography and abdominal CT-scan. On abdominal scanning, TLA showed a low-attenuation lesion with or without ring enhancement, and septated (i.e., honeycomb-like) abscesses.[11] On ultrasonography findings, TLA showed hypoechoic lesion, but some previous investigators also stated that in some cases, they find hypechoic lesion instead.[2,11] Therefore, the definitive diagnosis of TLA is based on the findings of AFB in pus, aspirate or biopsy specimen or the necrotic tissue.

WHO recommends the main treatment of the extrapolmonary TB using rifampin, isoniazid, ethambutol, and pyrazinamide for two months, followed by 4 months of rifampin and isoniazid.[8] some previous cases showed TLA had been successfully treated by percutaneous drainage combined with a transcatheater infusion of antitubercular drugs.[12] The treatment has been applied and showed a good outcome. After applying the main treatment to the patients, the symptoms gradually improved such as improvement of the appetite, weight gain, resolution of fever, reduced jaundice, and decreased hepatosplenomegaly within the first two to three months.[13] In our case, the patient responded well to systemic Anti tubercular treatment (ATT) and was improving after 5 days of medication.

4. Conclusions

Although the cases are quite rare, the possibility of TLA should always be kept in mind as one of the differential diagnosis of liver abscess itself, especially in a high TB prevalence area like Indonesia. It is also always a possibility to make the diagnosis in a case of a noresolving liver abscess in order to manage and treat the patient as soon as possible.

References

[1] WHO 2016 Global tuberculosis report (WHO Press) p 37
[2] Baveja C P, Gumma V, Chaudhary M and Jha H 2009 Primary tubercular liver abscess in an immunocompetent adult: a case report J. Med. Case Rep. 3 1–4
[3] Patel R, Choksi D, Poddar P, Shah K, Ingle M and Sawant P 2016 Primary tubercular liver abscess complicated by tubercular meningitis in portal cavernomacholangiopathy ACG Case Rep. J. 3(4) 1–4
[4] Reed D H, Nash A F and Valabhji P 1990 Radiological diagnosis and management of a solitary tuberculous hepatic abscess Br. J. Radiol. 63 902–4
[5] Bhattacharya K P, Roy A, Jamil M, Talukdar K K, Khonglah Y and Sarma K 2015 Isolated tubercular subcapsular liver abscess: a rare manifestation of extrapulmonary tuberculosis J. Hepatitis 1 1-6
[6] Singh R, Kumar N, Sundriyal D and Trisal D 2013 Mixed pyogenic and tuberculous liver abscess: clinical suspicion is what matters BMJ Case Rep. 1-3
[7] Islam M R, Maksud M, Baral P, Hossain M and Kabir A 2015 Case report of hepatic tuberculosis Bangl. J. Med. 26(1) 43–5
[8] World Health Organization 2010 Treatment of tuberculosis: guideline 4th edition (WHO Press) p 2
[9] Pandya J S, Kandekar R V, Tiwari A R, Kadam R and Adhikari D R 2016 Primary liver abscess with anterior abdominal wall extension caused by Mycobacterium tuberculosis complex J. Clin. Diagn. Res. 10(9) 8–9
[10] Dey J, Gautam H, Venugopal S, Porwal C, Mirdha B R, Gupta N and Singh U B 2016 Tuberculosis as an etiological factor in aliver abscess in adults Tuberc. Res. Treatment 1–4
[11] Chen T C, Chou L T, Huang C C, Lai A B and Wang J H 2016 Isolated tuberculous liver abscess in an immunocompetent adult patient: A case report and literature review J. Microbiol. Immunol. Infect. 49 455–8
[12] Hassani K I M, Ousadden A, Ankouz A, Mazaz K and Taleb K A 2010 Isolated liver tuberculosis abscess in a patient without immunodeficiency: A case report *World J. Hepatol.* **2**(9) 354-7

[13] Hickey A J, Gounder L, Moosa M Y S and Drain P K 2015 A systematic review of hepatic tuberculosis with considerations in human immunodeficiency virus co-infection *BMC Infect. Dis.* **15** 1-11