CASE REPORT

Mixed pyogenic and tuberculous liver abscess: clinical suspicion is what matters

Rajnish Singh, Naveen Kumar, Deepak Sundriyal, Deepshikha Trisal

Department of Medicine,
PGIMER & Dr Ram Manohar
Lohia Hospital, New Delhi,
India

Correspondence to

Dr Naveen Kumar,
docnaveen2605@yahoo.co.in

SUMMARY

Tuberculosis (TB) is a rare cause of liver abscess, even in country like India where it is a very common infection. Moreover, tubercular liver abscess (TLA) is the most unusual pattern of hepatic tuberculosis. We report an unusual case of liver abscess in an immune-competent patient presenting only with weight loss. On investigation, initially it appeared pyogenic, but later turn out to be a mixed infection with tuberculosis. He responded well to antibiotic and antitubercular drugs. A mixed pyogenic and TLA is very uncommon. We conclude that, tuberculosis should be suspected in liver abscess, especially in the absence of typical features and failure to respond to antibiotics.

raised alkaline phosphatase (480 IU/L). Rest of biochemistry and urinalysis were normal. In further workup, erythrocyte sedimentation rate was raised to 60 mm in first hour (Westergren); Mantoux test was positive (15×14 mm); sputum was negative for acid-fast bacilli (AFB); hepatitis B surface antigen, antihepatitis C virus and ELISA for HIV were negative. Chest X-ray showed right pleural effusion; however, lung parenchyma was normal. Contrast-enhanced CT scan of the chest and abdomen was carried out, which had a 5.2×4.7 cm² size liver abscess located in segment IV of the liver with right sided pleural effusion (figure 1). Amoebic serology was negative. Pleural fluid aspirated was straw colored, the analysis of which showed 200 cells/mL

BACKGROUND

Liver abscess is common in tropical countries like India. Most of them are pyogenic or amoebic abscesses. Uncommonly, it can be a manifestation of hepatic tuberculosis (TB). We report a patient with liver abscess initially thought to be due to *Proteus mirabilis*. As the clinical presentation of the patient was more consistent with a chronic infection like TB and since the patient did not respond to antibiotics, further workup was carried out which ultimately proved the suspicion. The case has been reported for its unusual presentation and rarity, and to emphasise the fact that clinical suspicion and repeated workup can clinch the diagnosis.

CASE PRESENTATION

A 60-year-old man presented with weight loss of 8 kg (from 78 to 70 kg) and decreased appetite for 2 months. There was no history of fever, night sweats, cough, haemoptysis, breathlessness, abdominal pain, vomiting or altered bowel habit. He was a smoker (15 pack-years), but not an alcoholic, diabetic or hypertensive. There was no history of TB or high-risk sexual behaviour. On examination, he was a cooperative patient with average build (body mass index 22.3 kg/m²). Blood pressure was 124/74 mm Hg, pulse rate 88/min, respiratory rate 16/min and temperature 97.4 F. General physical examination was within normal limits. In systemic examination, there were diminished breath sounds in right infrascapular region with stony dullness, and tender moderate (3 cm below costal margin) hepatomegaly.

INVESTIGATIONS

On investigation, haemoglobin was 13.2 g% and total lymphocyte count was 8700 cells/mL (P78, L20, E2). Liver function test was normal except for

Figure 1 Contrast-enhanced CT of the chest and abdomen showing hepatomegaly with abscess in segment 4 of the liver and moderate right-sided effusion.

To cite: Singh R, Kumar N, Sundriyal D, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-008768

(90% lymphocyte) and 5.4 g% protein (Light's ratio of protein 0.66). Adenosine deaminase level was not raised (22.7 U/L; <24 U/L) and it was negative for Gram's stain, Ziehl-Neelsen (Z-N) stain, malignant cell and PCR for TB. Liver abscess was aspirated, which had thick foul smelling brownish pus; it was negative on Gram stain and Z-N stain, but grew *P mirabilis* on culture. According to sensitivity, the patient was started on injection piperacillin-tazobactam. Over the next 2 weeks, there was some improvement in general well-being and no further loss of weight, however, appetite did not improve. Moreover, pleural effusion increased, reaching half of the hemithorax. A repeat pleural tap was carried out and 300 mL of fluid was sent for analysis. Cytology and biochemistry were similar as previously reported, but the centrifuged sample was positive for AFB and PCR for TB. Liver abscess was aspirated again, which also showed AFB on Z-N staining (figure 2).

TREATMENT

The patient was started on antitubercular treatment (ATT) regimen with four drugs.

OUTCOME AND FOLLOW-UP

At the end of 2 months of ATT, weight had increased by 2 kg, appetite had improved and pleural effusion reduced to minimal (figure 3).

DISCUSSION

Hepatic involvement is common in disseminated TB. However, isolated hepatic TB is uncommon, probably because low oxygen level in liver is unfavourable for bacilli. It usually has a primary focus in lung or gastrointestinal tract (GIT). Later, it spreads to the liver as a part of haematogenous spread from lung via hepatic artery, and less commonly via portal vein from gastrointestinal focus.^{1 2} Reed *et al*³ described three morphological pattern of hepatic TB: diffuse involvement of liver with miliary or pulmonary TB; diffuse parenchymal involvement without any evidence of existing TB anywhere (primary miliary TB of liver); and focal lesion in the liver, multiple or solitary, presenting as nodule or abscess. Out of these patterns, isolated tubercular liver abscess (TLA) is the rarest; Essop *et al*⁴ found TLA in only 0.34% of patients with hepatic TB.

Symptoms of the disease are non-specific: fever, abdominal pain, abdominal mass, anorexia, weight loss, lethargy, weakness and malaise. Hepatomegaly is a common physical finding. Jaundice is an uncommon manifestation. The main differential diagnoses of TLA are hepatoma, pyogenic liver abscess and

Figure 2 Acid-fast bacilli (AFB) staining of liver abscess aspirate showing AFB-positive tubercular bacilli.

Figure 3 Follow-up chest X-ray at the end of 2 months showing only blunting of right costophrenic angle, with resolution of the effusion.

amoebic liver abscess. The radiological findings of TLA have a low specificity.⁵ It can present as mass on ultrasonography (hypoechoic lesions with hyperechoic rims and complex masses) and CT scan (hypodense lesions). The definitive diagnosis can only be made on histological and bacteriological workup. It is difficult to demonstrate AFB in TLA; pus is positive in only 0–45% of cases.⁶ Cultures can be positive in only 10% of patients, which can increase up to 60% in miliary cases.⁶ PCRs coming as a useful diagnostic tool for hepatic TB. In a study by Diaz *et al*,⁷ as compare to conventional diagnostic techniques, PCR was positive in 57% of tubercular hepatic granulomas. Another advantage is that it can distinguish *Mycobacterium tuberculosis* from other *Mycobacterium*.

Our case had some important peculiarities. Our patient, unlike other reported cases, did not have fever, any symptoms pertaining to lung and GIT or leukocytosis. Similar to our case, Lee *et al*⁸ reported a case of TLA in a patient who presented only with 3 months history of intermittent chills. Moreover, the spread of infection to liver from lung was unlikely, as there was no parenchymal lesion. It was most probably a case of primary TLA. Also, there was no predisposing factor like diabetes mellitus, HIV, alcoholism and diseases of gut or cancer. A primary localised TLA in an immune-competent patient is quite rare.

Mixed pyogenic and tuberculous infection in liver abscess are rare, but has been reported in the literature. Silverberg *et al*⁹ reported liver abscess which was co-infected with *Escherichia coli*, *P mirabilis*, *P morgani*, *Bacteroides fragilis* and *M tuberculosis*. In another case of a patient with carcinoma colon, Rafailidis *et al*¹⁰ reported a liver abscess co-infected with *Klebsiella pneumoniae*, *Candida albicans* and *M tuberculosis*. In our case, initially we attributed *P mirabilis* as the cause of abscess. However, clinical picture was more consistent with tuberculous process and repeated investigation confirmed it. The diagnosis of TB could have been easily missed, had not the index of suspicion for TB been high. Similar observation was made by Singh *et al*,¹¹ where they picked up TLA when the patient did not respond to antibiotic and amoebicides. The infection with *P mirabilis* could be a secondary process, due to the spread from the gastrointestinal tract. It could have contributed to abscess formation leading to immediate deterioration before admission.

Management of tuberculous liver abscess is still a subject of discussion. Standard four drug ATT is recommended for 1 year. But, according to Gracey,¹² thick fibrous tissue around the abscesses and large size may prevent antibiotics from reaching the target. Hence, percutaneous drainage, combined with ATT has been used in difficult cases.¹³ Adding further modification, cases has been managed by percutaneous drainage followed with transcatheter infusion of antitubercular drugs.^{14 15} Surgery is indicated if percutaneous aspiration is not successful, or difficult because of site, and multiseptate abscess.¹⁶

Learning points

- ▶ Tuberculosis should be kept in differential diagnosis of liver abscess, especially in the absence of typical features and failure to respond to antibiotics, as also concluded by Lee *et al*⁸ and Singh *et al*.¹¹
- ▶ Apart from investigation, clinical impression should always be used as the guiding factor of decision-making in patient management.
- ▶ Primary tubercular liver abscess in an immune-competent patient is quite uncommon and a co-infection with pyogenic organism is very unusual.

Contributors RS and NK were involved in the diagnosis and management of patient, conception and design of the manuscript, acquisition of data, drafting and revising the article. DS was involved in conception and design of the manuscript, drafting and revising the article. DT participated in the diagnosis and management of patient, conception and design of the manuscript and acquisition of data.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Terry RB, Gunnar RM. Primary miliary tuberculosis of the liver. *JAMA* 1957;164:150–7.
- 2 Hersch C. Tuberculosis of the liver. A study of 200 cases. *S Afr Med J* 1964;38:857–63.
- 3 Reed DH, Nash AF, Valabhji P. Radiological diagnosis and management of a solitary tuberculous hepatic abscess. *Br J Radiol* 1990;63:902–4.
- 4 Essop AR, Segal I, Posen J, *et al*. Tuberculous abscess of the liver. *S Afr Med J* 1983;63:825–6.
- 5 Polat KY, Aydinli B, Yilmaz O, *et al*. Intestinal tuberculosis and secondary liver abscess. *Mt Sinai J Med* 2006;73:887–90.
- 6 Levine C. Primary macronodular hepatic tuberculosis: US and CT appearances. *Gastrointest Radiol* 1990;15:307–9.
- 7 Diaz ML, Herrera T, Lopez-Vidal Y, *et al*. Polymerase chain reaction for the detection of *Mycobacterium tuberculosis* DNA in tissue and assessment of its utility in the diagnosis of hepatic granulomas. *J Lab Clin Med* 1996;127:359–63.
- 8 Lee SW, Lien HC, Chang CS. Tuberculous liver abscess in a case without lung involvement. *Kaohsiung J Med Sci* 2010;26:99–104.
- 9 Silverberg LF, Johnston C, Haber K. Tuberculous involvement of a polymicrobial liver abscess. *West J Med* 1984;141:682–4.
- 10 Rafailidis P, Kapaskelis A, Christodoulou C, *et al*. Tuberculosis, *Klebsiella pneumoniae* and *Candida albicans* infection in liver metastasis of bowel carcinoma. *Eur J Clin Microbiol Infect Dis* 2008;27:753–5.
- 11 Singh B, Saxena PD, Kumar V, *et al*. Tubercular liver abscess in immunocompetent patients. *J Assoc Physicians India* 2011;59:523–4.
- 12 Gracey L. Tuberculous abscess of liver. *Br J Surg* 1965;52:442–3.
- 13 Chen HC, Chao YC, Shyu RY, *et al*. Isolated tuberculous liver abscesses with multiple hyperechoic masses on ultrasound: a case report and review of literature. *Liver Int* 2003;23:346–50.
- 14 Mustard RA, Mackenzie RL, Gray RG. Percutaneous drainage of a tuberculous liver abscess. *Can J Surg* 1986;29:449–50.
- 15 Kubota H, Ageta M, Kubo H, *et al*. Tuberculous liver abscess treated by percutaneous infusion of antituberculous agents. *Intern Med* 1994;33:351–6.
- 16 Chaudhary A, Wakhlu A. Isolated tubercular liver abscess in pediatric age group. *Int J Trop Med* 2005;2:1–5.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow