

Tubercular granuloma mimicking pyogenic liver abscess

Mohit Garg¹, Yasmeen Khan², Monika Pathania¹

¹Department of Medicine, AIIMS Rishikesh, Uttarakhand, ²Department of Medicine, AIIMS Bhopal, Madhya Pradesh, India

ABSTRACT

Hepatic Tuberculosis (TB) is extremely rare without miliary involvement in immunocompetent patients. Even in countries like India where TB is a major public health problem only few cases have been diagnosed and treated. We report a case of an immunocompetent patient who presented with undiagnosed pyrexia of 11 days, was initially diagnosed as pyogenic liver abscess, he did not responded to treatment and on liver biopsy was diagnosed as hepatic tuberculoma. Antitubercular treatment (ATT) was started and the patient responded well. We concluded that though hepatic TB is rare in immunocompetent patient, it is important to keep it as a differential diagnosis in patients of liver abscesses who are not responding to treatment in order to avoid needless investigations.

Keywords: Hepatic abscess, hepatic granuloma, hepatic tuberculosis

Introduction

Tuberculosis (TB) remains a major health problem in developing world; it is the major cause of morbidity and mortality due to infectious diseases in developing countries. According to WHO, there were 8.6 million new TB cases and 1.3 million TB deaths worldwide in 2012.^[1] Hepatic TB compromises less than 1% of all the cases of TB.^[2] Granuloma is aggregation of epithelioid histiocytes with or without multinucleated giant cells that are usually surrounded by lymphocytes.^[3] Only in rare cases, granulomas possess certain distinct histological features like caseous necrosis containing acid fast bacilli (TB) or ova of *Schistosoma mansoni* (schistosomiasis).^[4]

Case History

Patient X, a 19-year-old male Hindu, and a 2nd year graduate student, presented to AIIMS Bhopal OPD, with chief complaints of fever and pain in the abdomen since 11 days. Fever was high grade, documented to be 103°F with evening rise of temperature. No history suggestive of any chronic cough, weight loss, burning micturition, loose stools, earache

or runny nose. No history of fever or repeated hospital admissions in past. Patient was admitted and baseline investigations were done [Table 1].

Ultrasound (USG) suggestive of hepatomegaly 17 cm with hypoechoic lesion (40 cc) in right lobe [Figure 1]. A probable diagnosis of liver abscess was made. Patient was started on I.V. metronidazole and ceftriaxone. Patient symptoms begin to improve; fever was not documented for last 2 days and was discharged after 7 days of injectable antibiotics on oral cefexime and metronidazole. Patient again came with complaints of fever after 7 days, which was high grade, documented 105°F at home. An urgent USG abdomen was suggestive of resolving abscess. Patient was again admitted in hospital and investigations were sent in view of high grade fever [Table 2].

Patient was again started on I.V. ceftriaxone and metronidazole. This time, fever persisted despite I.V. antibiotics and continued to high grade, usually in evening and documented to be 104-105°F. Repeat USG suggestive of hepatomegaly 16 cm with focal, heterogeneous hypoechoic lesion (30 cc) in the right lobe of liver (segment V), with no liquefaction and no cystic change. Patient was continued on I.V. antibiotics, but fever persisted and was high grade. CT abdomen was

Address for correspondence: Dr. Monika Pathania, Associate Professor, Department of Medicine, AIIMS Rishikesh, Uttarakhand, India. E-mail: anshupathania27@gmail.com

Received: 07-08-2019

Revised: 02-12-2019

Accepted: 13-12-2019

Published: 28-01-2020

Access this article online

Quick Response Code:



Website:
www.jfmipc.com

DOI:
10.4103/jfmipc.jfmipc_630_19

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Garg M, Khan Y, Pathania M. Tubercular granuloma mimicking pyogenic liver abscess. J Family Med Prim Care 2020;9:424-7.

planned which shows an ill-defined non-enhancing hypodense lesion (5.8 × 5.4 × 4.8 cm) in segment V/IV b of liver ?infective etiology [Figure 2].

In view of some atypical features, MRI abdomen was provisionally done which was suggestive of T2 hypo intense lesion with bright ADC in segment V of liver infective inflammatory [Figure 3].

A USG-guided liver biopsy was planned on basis of above findings. Two biopsy samples were taken, one in normal saline send for culture and sensitivity, and other in formalin for histopathology.

Provisional liver biopsy report was suggestive of granuloma in liver, [Figure 4] AFB stain was positive so patient was started on ATT, based on the long standing fever with evening rise, ESR 92 and 62 and non-resolving fever. Patient’s fever responded on 3rd day and was discharged after 3 days of afebrile period.

Discussion

Isolated primary tuberculosis is a rare disease entity, although hepatic tuberculomas with miliary tuberculosis can be seen.

Table 1: Basic investigation of patient at first admission

Date	18/08/2017
Hemoglobin	12.2
TLC	10180
DLC	N73L15M10
Platelets	464
ESR	96
B.Urea	9
Cretinine	0.54
SGOT	16
SGPT	18
Total protein	7.02
Albumin	3.30
Urine C/S	Sterile
Chest Xray	NAD



Figure 1: Ultrasound image of the patient

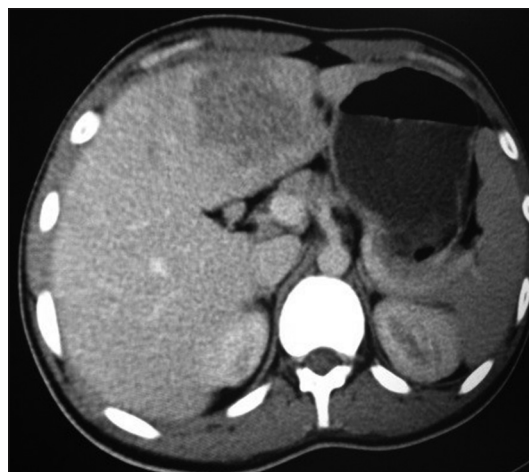


Figure 2: CT abdomen of the patient



Figure 3: Showing MRI Abdomen of the patient

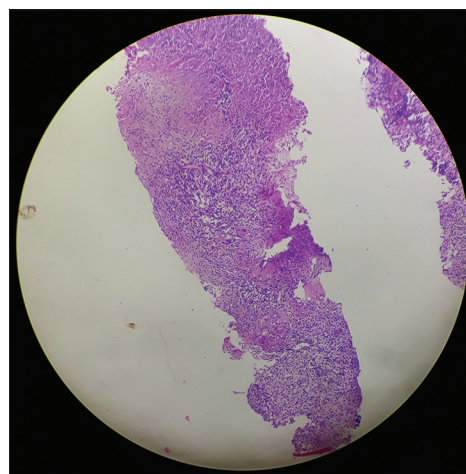


Figure 4: Biopsy of the patient suggestive of caseation necrosis, granuloma formation

Table 2: Basic investigations of patient at second admission

Date	06/09/2017	14/09/2017
Hemoglobin	11.7	10.0
TLC	12,490	15,180
DLC	N75L15M09	N81L12M6
Platelets	445	420
ESR	62	
SGOT	17	
SGPT	19	
HIV	NON-REACTIVE	IgM Dengue- negative
MALARIA -ICT	NEAGTIVE	IgM Entamoeba- negative
HEPATITIS B AND C	NON-REACTIVE	

The most common clinical presentation is of PUO, which is often misdiagnosed or undiagnosed and could be fatal if not treated properly. Liver histology in an individual in tropical country differs from those who live in temperate climates.^[5] The granuloma formation in liver occurs due to its rich blood supply and reticuloendothelial cell network, but since there is only mild derangement of liver function so most of patients remain mildly symptomatic or asymptomatic.^[6]

We have reviewed the literature and look into various causes of granuloma of liver. In developed countries, autoimmune and non-infective causes are much prevalent whereas in developing countries, infective etiology was the major cause of hepatic granuloma. In a study of United Kingdom (2003), Hepatic granulomas were found in 63. Of those identified, 47 were female, with a mean age of 42 years. Underlying aetiologies were as follows: primary biliary cirrhosis (PBC; 23.8%), sarcoidosis (11.1%), idiopathic (11.1%), drug induced (9.5%), HCV (9.5%), PBC/autoimmune hepatitis overlap (6.3%), Hodgkin lymphoma (6.3%), AIH (4.8%), tuberculosis (4.8%), resolving biliary obstruction (3.2%).^[7] One study of MAMC (India) 2003 described prevalence of tuberculosis of liver to be 10-53% under various aetiologies of granulomatous hepatitis.^[8]

Hepatic TB is extremely rare form of extra pulmonary TB which usually presents in association with military TB.^[9] The first reported case was in 1958 by Bristow. Primary hepatic TB is commonly confused with other more common diagnostic entities such as hepatoma, pyogenic liver abscess, and liver metastasis of other primary cancers. Because of the nonspecific clinical presentation, the diagnosis of primary hepatic TB is usually made at autopsy or occasionally after laparotomy has been performed.^[10] Reported incidence of primary hepatic TB is less than 1% of all tubercular infections.^[11]

Diagnosis of tubercular liver abscess in our case is clinical, radiological and histopathological. Histopathological diagnosis is not possible in many centres in India. Even the histological sensitivity of culture is 8.3–83% (depending on amount cultured) and AFB smear is 3.2%.^[12] We were lucky in a way that we find AFB stained bacilli in the biopsy specimen.

As a clinician, it needs strong clinical suspicion in a patient of non-resolving liver abscess. Thin built patient with history of fever, weight loss and non-resolving abscess should be suspected to be tubercular even when the chest/systemic involvement is not there in a high prevalence country like India.^[12] Our country has one-fourth of global TB cases as reported by RNTCP 2016 review.^[13] A simple plain ultrasound report cannot differentiate between bacterial and tubercular liver abscess. So high end investigations like CT scan and MRI abdomen may be required in such cases. Even MRI findings are non-specific in our case so a liver biopsy is done.

Our case is unique in several aspects as the patient first presented with non-specific symptoms such as fever, diffuse pain in abdomen, chest x- ray was normal. Patient fever responded on initial treatment with metronidazole which recurs after 7 days. USG initial reported pyogenic liver abscess, so was managed on lines of pyogenic abscess. Sonographic features differentiating from typical liver abscess were no internal or peripheral vascularity and no cystic/liquefied component. Still after extensive radiological investigations, final diagnosis was made on liver biopsy which showed granuloma with AFB stain positive. This case reminds us of the fact that though hepatic TB is a rare phenomenon, we always keep it as differential while dealing with cases of PUO and liver abscesses not responding to treatment.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Self.

Conflicts of interest

There is no conflicts of interest.

References

1. WHO. Tuberculosis (TB). Global Tuberculosis Report 2013. Geneva: World Health Organization, 2013. Available from: http://www.who.int/tb/publications/global_report/gtbr12_main.pdf. [Last accessed on 2014 March 18].
2. Mert A, Ozaras R, Tabak F, Ozturk R, Bilir M. Localized hepatic tuberculosis. *Eur J Intern Med* 2003;14:511-2.
3. Dourakis SP, Saramadou R, Alexopoulou A, Kafiri G, Deutsh M, Koskinas J, et al. Hepatic granuloma: A 6-year experience in a single center in Greece. *Europ J Gastroenterol Hepatol* 2007;19:101-4.
4. Klatskin G. Hepatic granulomata: Problems in interpretation.

- Ann N Y Acad Sci 1976;278:427-31.
5. Reynolds TB, Campra JL, Peters RL. Granulomatous liver disease. In: Zakim D, Boyer TD, editors. *Hepatology: A Textbook of Liver Diseases*. 3rd ed. Philadelphia: WB Saunders; 1996. p. 1472-89.
 6. Singh S, Jain P, Aggarwal G, Dhiman P, Singh S, Sen R. Primary hepatic tuberculosis: A rare but fatal clinical entity if undiagnosed. *Asian Pac J Trop Med* 2012;5:498-9.
 7. Gaya DR, Thorburn D, Oien KA, Morris AJ, Stanley AJ. Hepatic granulomas: A 10 year single centre experience. *J Clin Pathol* 2003;56:850-3.
 8. Rajan A, Kar P. Hepatic Granulomatosis. *JAPI*; 2003.
 9. Bangaroo AK, Malhotra AS. Isolated hepatic tuberculosis. *J Ind Assoc Paediatr Surg* 2005;10:105-7.
 10. Balsarkar D, Joshi MA. Isolated tuberculous hepatic abscess in a non-immunocompromised patient. *J Postgrad Med* 2000;46:108-9.
 11. Kumar P, Taneja S, Gupta K, Duseja A, Dhiman RK, Chawla YK. Unusual presentation of tubercular liver abscess in an immune-competent adult. *J Clin Exp Hepatol* 2017;7:77-9.
 12. Dey J, Gautam H, Venugopal S, Porwal C, Mirdha BR, Gupta N, *et al.* Tuberculosis as an etiological factor in liver abscess in adults. *Tuberc Res Treat* 2016;2016:8479456.
 13. TB. Revised National Tuberculosis Control Programme Overview 2016. New Delhi, India: Central TB Division, Directorate General of Health Services, Ministry of Health and Family Welfare; 2016.