

Hepato-pulmonary amebiasis: a case report

Authors

Vishnu Prasad Shenoy,
PhD¹
Shashidhar Vishwanath,
MD²
Bairy Indira, MD³
Rodrigues G, MS⁴

¹Selection grade lecturer
at the Microbiology
Department, Kasturba
Medical College, Manipal,
India.

²Assistant Professor at the
Microbiology Department,
Kasturba Medical College,
Manipal, India.

³Professor and Head at the
Microbiology Department,
Kasturba Medical College,
Manipal, India.

⁴Professor at the Dept. of
Surgery, Kasturba Medical
College, Manipal, India.

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Correspondence to:

Dr. Vishnu Prasad Shenoy
Selection Grade Lecturer,
Department of
Microbiology,
Kasturba Medical College,
Manipal – Karnataka –
India
576104
Phone: +91-820-2922322
E-mail: vishnuprasad.
shenoy@rediffmail.com

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ABSTRACT

Infections with *Entamoeba histolytica* are seen worldwide and are more prevalent in the tropics. About 90% of infections are asymptomatic, and the remaining 10% produce a spectrum of clinical syndromes, ranging from dysentery to abscesses of the liver or other organs. Extra-intestinal infection by *E. histolytica* most often involves liver. Pleuro-pulmonary involvement, seen as the second most common extra-intestinal pattern of infection, is frequently associated with amebic liver abscess. Pulmonary amebiasis occurs in about 2-3% of patients with invasive amebiasis. We report herein the case of a 45-year-old male presenting with hepato-pulmonary amebiasis. The diagnosis was established from direct examination of sputum, in which trophozoites of *E. histolytica* were detected, and by serology. Following treatment with metronidazole and chloroquine, the clinical evolution improved significantly. On regular follow-up visits, the patient was asymptomatic. This case report reiterates the need for collaboration between clinicians and microbiologists for timely diagnosis of such infections.

Keywords: *Entamoeba histolytica*, liver abscess, microscopy, pulmonary involvement.

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INTRODUCTION

Among parasitic diseases, amebiasis caused by *Entamoeba histolytica*, is the third most frequent cause of mortality after malaria and schistosomiasis.¹ Developing countries are the most affected by this disease.² The largest burden of *E. histolytica* infection is seen in Central and South America, Africa, and the Indian sub-continent.³

Entamoeba histolytica, an amoebic protozoan parasite, is the most invasive of the *Entamoeba* group. The life cycle of the protozoan includes an infective cyst and an invasive trophozoite form, and infection occurs due to fecal-oral mechanism through water or food contaminated with feces.³ Clinically, the disease presentation in amebiasis ranges from asymptomatic colonization to colitis and/or liver abscess.¹

Pulmonary amebiasis, the second most common extra-intestinal pattern of infection, is frequently associated with amebic liver abscesses. It occurs in 2-3% of patients with invasive amebiasis.^{1,2} Lung disease without liver involvement is exceptional and it is

believed that infection of the lung is a result of haematogenous spread from a primary site, usually colon.²

For diagnosis of amebiasis, microscopy is a very useful method, especially in developing countries, although worldwide other more advanced methods such as antigen detection, polymerase chain reaction or serology are available.³

The present clinical report aims to present a patient with amebic liver abscess with secondary rupture into the right lung at a tertiary care centre in Manipal. Retrospective analysis of medical records of the patient was done to prepare the case.

CASE REPORT

A 45-year-old previously healthy male from Ankola, a town in Coastal Karnataka, India, presented with complaints of high grade fever with chills of 15 days duration and abdominal pain in the right hypochondrium of 7 days duration. He was admitted to a local hospital for the above complaints and was referred to our hospital on 11/06/2006 in view of the worsen-

ing pain in abdomen and pain on taking deep breaths. Past history revealed a dysentery episode one month previous to the present illness. He was a known smoker and alcoholic.

On physical examination, there was bradycardia, bilateral pitting pedal edema and signs of dehydration. Abdomen was tense with tenderness in right hypochondrium and epigastric region. Hepatomegaly was seen. On auscultation, breath sounds were decreased in the right lower lung field along with presence of pleural rub.

His laboratory test results at admission were as follows: Hemoglobin – 13.4 g/dL; Hematocrit – 40.8%; Total leukocyte count – 33,500 cells/mm³; ESR – 76 mm/hr; Total bilirubin: 5.3 mg/dL; Direct bilirubin: 3.4 mg/dL; Serum aspartate amino transferase (SGOT) – 596 U/L; Serum alanine amino transferase (SGPT) – 296 U/L and Alkaline phosphatase: 626 IU/L.

Chest X-ray showed right sided pleural effusion and left lung pneumonia. Abdomen ultrasonography revealed hepatomegaly (Liver span: 20.5 cm) with abscess in the right lobe of liver, thickened gall bladder wall, fluid in Morrison's pouch, and minimal right sided pleural effusion. CT abdomen showed right liver abscess with multiple septations.

A provisional diagnoses of liver abscess was made. The differential diagnoses with a bacterial abscess, tuberculous abscess, and neoplastic disease were considered, and ultrasound-guided percutaneous drainage of the abscess was performed. The patient was put on intravenous antibiotics, ceftriaxone and metronidazole, with which he improved symptomatically. Alpha-fetoprotein level was 33 ng/mL and Beta HCG – 0.1 MU/mL. Gram stain of the aspirated pus showed numerous pus cells and no bacteria. Aerobic bacterial culture was sterile after 48 hours of incubation. Amebic liver abscess was suspected. Later, the patient developed increased cough with coffee-ground sputum and decreased oxygen saturation, for which he was shifted to intensive care unit. Amebic etiology of the abscess was confirmed by the serological test – Indirect hemagglutination (Fumouze diagnostics, France) – which showed a significant antibody titre of 1:320. Rupture of amebic liver abscess into right lung was suspected and sputum was sent for examination. Sputum was liver coloured; saline mount performed on two consecutive spu-

tum samples revealed *Entamoeba histolytica* trophozoites. However, an earlier stool microscopy at admission did not reveal any *Entamoeba histolytica* cysts or trophozoites. Repeat chest X-ray showed raised right hemi-diaphragm. Also moderate growth of *Escherichia coli* was seen in two sputum samples. The final diagnosis was Hepato-Pulmonary amebiasis.

The patient was treated with metronidazole for five weeks and chloroquine for three weeks and he responded well to treatment. He was also treated with piperacillin-tazobactam, clindamycin, amikacin and diloxanide furoate. At discharge, the patient had improved symptomatically. On regular follow up visits, the patient was asymptomatic and USG abdomen showed hyperechoic area in right lobe of liver consistent with focal scarring.

DISCUSSION

This patient had presented a month earlier, in May 2006, with intestinal symptomatology (dysentery) and was not diagnosed at that time. The evolution into liver abscess continued for another few days, ending with the invasion of the lung. Pulmonary complications are usually secondary to a liver abscess as also seen in the present case, with secondary pulmonary involvement following rupture of liver abscess. A routine microscopic examination of the sputum sample along with positive serology for *Entamoeba histolytica* confirmed the diagnosis. This case report stresses the importance of collaboration between clinicians and microbiologists and also highlights the importance of establishing the correct diagnosis of pulmonary amebiasis by sputum microscopy and confirmation by serological techniques.

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