



ENTAMOEBA HISTOLYTICA PLEUROPULMONARY INFECTION CASE REPORT

Asfia Sultan^{*1}, Adil Raza¹, Haris M Khan¹, Saifullah Khalid², Anees Akhtar¹,
Mohd Shameem³

¹Department of Microbiology, ²Department of Radiodiagnosis, ³Department of TB and Chest,
Jawaharlal Nehru Medical College and Hospital, AMU, Aligarh, U.P, India.

Corresponding Author: - Asfia Sultan

E-mail: drasfia@gmail.com

<p>Article Info Received 15/12/2014 Revised 27/12/2014 Accepted 12/01/2015</p> <p>Key words: Invasive amoebiasis, Pulmonary involvement, Microscopy.</p>	<p>ABSTRACT Pleuropulmonary amoebiasis is the 2nd most common extraintestinal manifestation after liver abscess. It occurs in 2%-3% of patients with invasive amoebiasis. This clinical report presents a case with pleuropulmonary amoebiasis with chest pain and pleural effusion. The diagnosis was established by direct examination of sputum, in which trophozoites of <i>E.histolytica</i> were detected, and by serology. Following treatment with satronidazole and chloroquine, the clinical condition improved significantly. Pleuropulmonary amoebiasis should be considered in patients with bile tinged sputum, culture negative, and failure of antibacterial therapy. This case report reiterates the need for collaboration between clinicians and microbiologists for timely diagnosis of such infections.</p>
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INTRODUCTION

E.histolytica, an amoebic protozoan parasite, is the most invasive of the *Entamoeba* group. This parasite is endemic in most tropical and subtropical areas of the world, where it causes millions of cases of dysentery. [1] Developing countries like India are more commonly affected by this disease. According to the World Health Organization (WHO) more than 100,000 deaths are annually reported. [2] Clinically, disease presentation ranges from asymptomatic colonization (about 90% of cases) to colitis and/or liver abscess (40-50 million cases). Microscopy is a very useful diagnostic method, especially in developing countries.

Pulmonary amoebiasis, the second most common extraintestinal pattern of infection, is frequently associated with amoebic liver abscesses. It occurs in 2%-3% of patients with invasive amoebiasis. [3] The main factors contributing to the development of pulmonary amoebiasis are poor socioeconomic conditions, malnutrition, and chronic alcoholism. In India, the prevalence of *E.histolytica* on routine stool examination varies from 2-

67% depending upon the population being screened. In Northern India studies showed 2-10% prevalence. [4] The last Indian case of amoebic hepatic abscess with pulmonary complications in adults was reported in 2008. [5]

The present clinical report aims to present a patient with amoebic liver abscess with secondary rupture into the left lung at a tertiary care centre in Aligarh region of northern India. Retrospective analysis of medical records of the patient was done to prepare the case.

CASE REPORT

A 26 yr old male patient from rural area of Rampur district of Uttar Pradesh was brought to emergency room with pain in right hypochondrial region and chest, shortness of breath for past 20 days. There was also history of cough with greenish yellow sputum production. He also complained of previous episodes of diarrhoea. No history of fever, abdominal pain, dysentery, any previous hospital stay, alcoholism, smoking, jaundice



and household contact. Patient belongs to low socio-economic class. Source of water was hand pump and defecation was in open fields.

On physical examination, pulse rate was 96/min, BP was 100/60 mm of Hg, but patient was slightly dyspnoeic. Abdomen was tense with tenderness in right hypochondrium and epigastric region. On auscultation, breath sounds were decreased in the right lower lung field along with presence of pleural rub. There was no pedal edema and icterus. Hepatosplenomegaly was present.

His laboratory test results at the time of admission were as follows: Hemoglobin - 10 g/dL; Hematocrit - 31%; Total leukocyte count - $15.8 \times 10^3/\mu\text{L}$; ESR - 115 mm/hr; Total bilirubin: 1.2 mg/dL; Serum aspartate amino transferase (SGOT) - 31.8 IU/L; Serum alanine amino transferase (SGPT) - 22.9 IU/L and Alkaline phosphatase: 150 IU/L. HIV status of patient was negative but he was HbsAg positive.

Chest X-ray showed right sided pleural effusion. On Contrast enhanced computed tomography, a peripherally enhancing lesion with air-fluid level involving segment 7 and 8 of liver with indistinct peripheral wall was seen (suggesting Ruptured Liver abscess, A). There was diaphragmatic perforation on right side with communication with pleural cavity forming multiple loculated air-fluid levels (B) and formation of lung abscess in apical segment of right lower lobe (C) with communicating, dilated segmental bronchus (arrow in D).

The pleural space was communicating with the lung abscess (C & D) confirming the imaging diagnosis of ruptured liver abscess with diaphragmatic perforation and hepato-pleurobronchial fistula formation. A provisional diagnosis of lung abscess was made.

Sputum examination

The sputum was greenish yellow in colour. On direct microscopy, Gram stain of the sputum showed numerous pus cells and no bacteria. Acid fast bacilli were not seen in Ziehl Neelsen staining. Aerobic bacterial culture was sterile after 48 hours of incubation. Amoebic liver abscess was suspected. Amoebic etiology of the abscess was confirmed by; saline and iodine mount performed on two consecutive sputum samples which revealed *E.histolytica* trophozoites. ELISA for antigen detection was also performed on patient's serum for *E.histolytica* which was suggestive of *E.histolytica* infection. The final diagnosis of Hepato-Pulmonary amebiasis was made. Stool microscopy was negative for *E. histolytica* cysts or trophozoites.

The patient was treated with sataronidazole for 4 weeks and chloroquine for 3 weeks and he responded well to treatment. No invasive procedure was performed for drainage of abscess. At discharge, the patient had improved symptomatically. On regular follow up visits, the patient was asymptomatic and repeat CT showed evidences of scarring and healing of liver abscess.

Figure 1. Contrast enhanced computed tomography of the patient shows a peripherally enhancing lesion suggesting ruptured Liver abscess, (A). Diaphragmatic perforation on right side forming multiple loculated air-fluid levels (B). (C) Shows formation of lung abscess in apical segment of right lower lobe. Communication between the lung abscess and pleural space (C & D) confirms the diagnosis of ruptured liver abscess with diaphragmatic perforation and hepato-pleurobronchial fistula formation

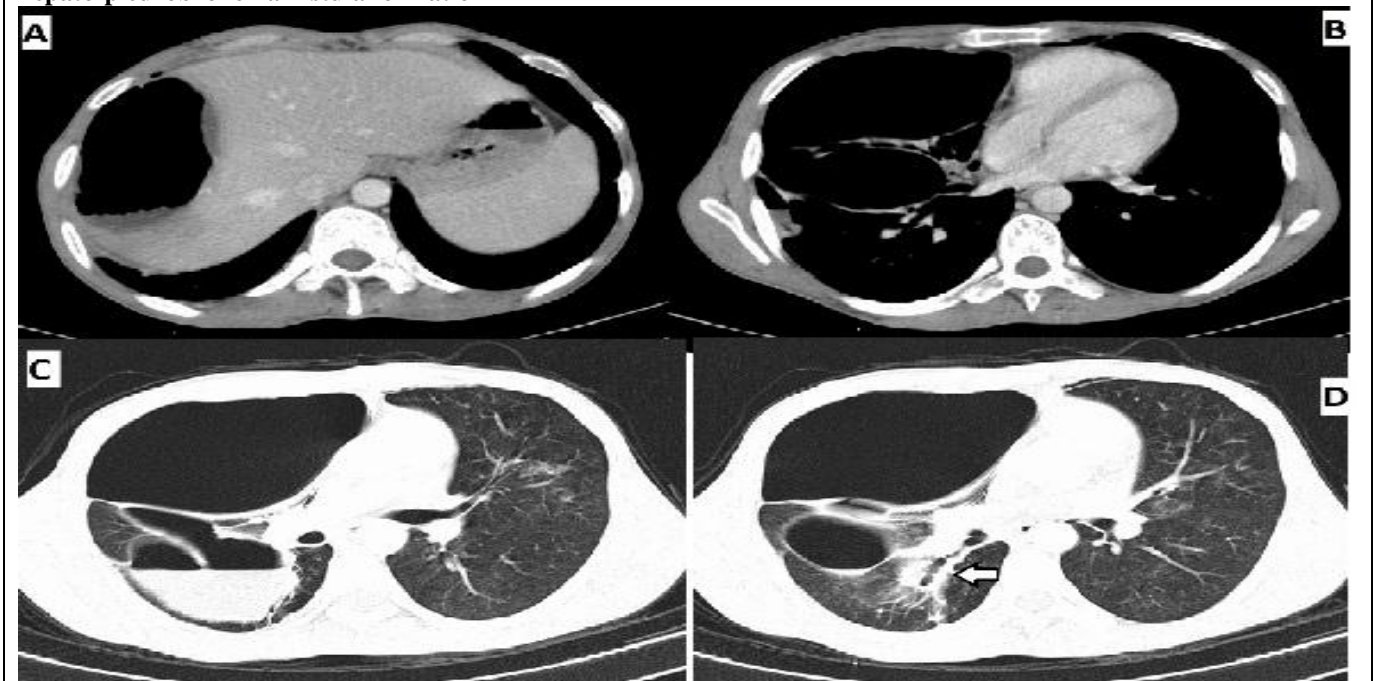


Table 1. Patient's laboratory test results

Indicators	The patient's values	Normal
White blood count ($\times 1000/\mu\text{L}$)	15.8	4.0–10.0
Red blood count ($\times 10^6/\mu\text{L}$)	3.62	4.0-5.5
Eosinophil (%)	7.0	1–10
Hemoglobin concentration (g/dL)	10	11.5–18
ESR (mm/hour)	115	0-10
Na (mEq/L)	132	135–145
K (mEq/L)	3.38	3.9–5.5
Ca (mg/dL)	8.86	8.8-10.4
P (mg/dL)	3.76	2.7-4.5
Serum total protein (g/dL)	4.6	6–8.5
S.creatinine (mg/dl)	1.1	0.7-1.4
Total bilirubin (mg/dl)	1.2	0-1
S.uric acid (mg/dl)	7.9	3.4-7
SGOT (IU/L)	31.8	37
SGPT (IU/L)	22.9	37
Alkaline phosphatase (IU/L)	150	117

DISCUSSION

Amoebiasis is defined by the World Health Organization (WHO) as infection with *E.histolytica*, regardless of symptomatology. [2] It infects people of both sexes and all ages; but males are ten times more affected by amoebiasis than females. Spread of the infection occurs due to consumption of food and water that is contaminated with the cyst. Our 26-year-old male patient was of younger age group as the patient described in another recent Indian case report with invasive amoebiasis. [5] History of travel is not important in this case as India is endemic for such infection.

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. It is estimated that amoebic lung disease without liver involvement occurs in 14.3% of all cases with lung involvement by amoeba. [3] There was no history of associated diarrhea in the clinical presentation. The differential diagnosis with a bacterial abscess, tuberculous abscess, and neoplastic disease were

considered. In this case, routine microscopic sputum examination clarified the diagnosis, pointing out the importance of collaboration between the infectious disease clinicians and parasitologists.

CONCLUSION

In conclusion an early precise diagnosis is of prime importance because appropriate treatment in addition to supportive care can be life-saving for such patients. 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.

CONFLICT OF INTEREST

There is no conflict of interest. There is no source of funding. The research article is neither published nor under consideration anywhere. All the authors have approved the final article.

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