



Presentation of Amoebic Liver Abscess as Massive Pleural Effusion and Reactive Pericarditis

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Abstract

Amoebic liver abscess is caused by Entamoeba histolytica, which is commonly prevalent in tropical countries like India. Although fever and right sided pleural effusion are commonly reported complications of amoebic liver abscess; massive pleural effusion with entirely opaque right hemithorax due to hepatopleural fistula and pericarditis are less commonly reported as presenting feature of the disease. Here we report a case of amoebic liver abscess presenting with fever, massive pleural effusion and reactive pericarditis. Patients was managed on antimicrobial treatment with intercostals tuber drainage and patient responded well to the treatment.

INTRODUCTION

Liver abscesses are caused by bacterial, parasitic, tubercular or fungal organisms. Amoebic liver abscesses are more common in developing countries as compared to developed countries where liver abscesses are mostly pyogenic.¹ Pyogenic abscesses of the liver occur secondary to biliary or intestinal tract infections, hematogenous spread or extension of local infection, and carry a high mortality rate even with appropriate management. On the other hand, amoebic abscesses respond well to drug therapy and rarely require drainage. Amoebic liver abscess can produce pleural effusion either by diaphragmatic irritation leading to sympathetic pleural effusion in a manner analogous to that seen with pyogenic liver abscess or ruptures through the diaphragm in to the pleural space.^{2,3} In our patient; right sided chest pain with acute pericarditis was the

presenting feature which is not so common presentation of amoebic liver abscess. Hence we describe this case of right lobe liver abscess with first presentation as massive right sided pleural effusion and reactive pericarditis.

CASE REPORT

68 years non alcoholic male patient presented in our emergency department with off and on low grade fever since last two weeks, right sided chest pain and breathlessness since last 2 days. On general examination patient was febrile (temp. - 39⁰c), pulse 113/min, BP 126/76 mmHg, RR 26/min, SpO2 94% at room air. On chest examination; air entry was reduced over right hemi thorax with stony dull note on percussion. On abdominal examination; slight right hypochondrial tenderness was present but no hepatomegaly was appreciable. Investigations

revealed Hb 12 gm/dl, TLC 18200/cumm, DLC P90%, L8%, B1, E1, platelets 4 lac/cu.mm, total bilirubin 0.6 mg/dl, SGOT/SGPT 27/31 U/L, alkaline phosphatase 225 U/L, blood urea 33 mg/dl and creatinine 0.8 mg/dl. HIV, HBsAg and anti HCV by ELISA were negative. ECG showed sinus tachycardia, diffuse PR segment depression with upwardly concave apparent ST elevation; except lead aVR which showed PR segment elevation with apparent ST depression, diffuse TP segment sloping (Spodick sign) suggestive of acute pericarditis (Fig.1). Chest x ray done 4 days back showed elevation of right hemidiaphragm suggestive of subdiaphragmatic abscess (Fig.2). Recent chest x-ray showed three radiopaque shadows in right hemithorax suggestive of loculated collections in pleural cavity (Fig.3). USG abdomen revealed a hypoechoic lesion seen in right lobe of liver with large collection in right pleural cavity. CECT chest confirmed the findings with a well defined peripherally enhancing hypodense lesion measuring 4.5×3.2×6cm in segment 8 of right lobe liver with a rent measuring 1 cm in right hemidiaphragm through which abscess was seen communicating with right pleural cavity [Fig. 4(a),4(b),4(c)]. Pleural fluid aspirated was pus, sterile on bacterial culture and gen expert negative for Mycobacterium tuberculosis. IgM for E. histolytica was positive. Intercostal tube placed in right 6th intercostal space and about one litre fluid drained. Patient put on injectable metronidazole, ciprofloxacin and amoxiclav. After 3 days of treatment patient was completely afebrile and TLC settled to 8000/mm³. Chest tube was removed on 12th day when drainage was < 50 ml/day.

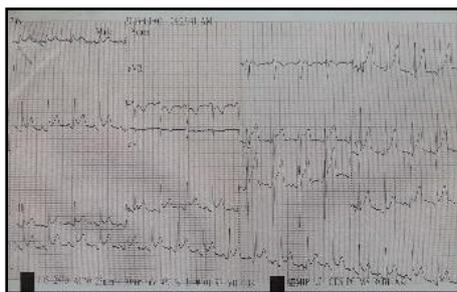


Fig.1

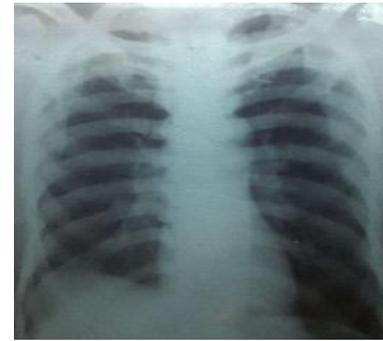


Fig.2



Fig.3

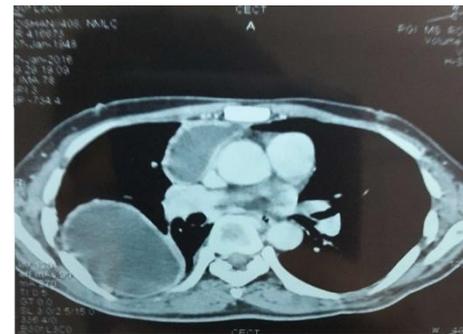


Fig.4 (a)



Fig. 4 (b)



Fig.4(c)

DISCUSSION

Liver abscess is common in the tropical regions like the Indian subcontinent. The common etiologic agents are *E. histolytica* (amoebic), bacterial (pyogenic), *Mycobacterium tuberculosis* and various fungi. They tend to affect younger population especially males. In this era of mass movement of populations in and out of areas with poor sanitary conditions, more cases of amoebic liver abscess and its thoracic complications are being seen in countries where they were previously uncommon. Although less described; amoebic liver abscess can present with complications and unusual manifestations.

Amoebic liver abscess arises from the hematogenous spread of the trophozoites of *Entamoeba histolytica* from the intestinal mucosa to the liver through the portal vein. The disease is suspected in endemic areas in persons presenting with fever, pain abdomen and liver tenderness.⁴ Occasionally it ruptures into the pleural cavity as signaled by abrupt exacerbation of pain, sometimes a tearing sensation, followed by rapidly progressive respiratory distress and sepsis, occasionally with shock.² These patients presented with upper abdominal pain, right side chest pain and breathlessness which is a common presenting symptom in most of the studies.⁵ The level of alkaline phosphatase is elevated in more than 75% of patients, whereas the levels of transaminases are elevated in 50%. Our patient had elevated alkaline phosphatase. Amoebic liver abscess occurs most commonly in the age group of 20 to 45 years. It has also been noted frequently at the extremes of age; men are

proportionately affected, with reported male to female ratio of approximately 10:19.⁶ The atypical presentation in our case is massive pleural effusion causing completely opaque right hemithorax which is very uncommon presentation. The rupture is into the right pleural space in more than 90% of patients. The diagnosis of amoebic liver abscess with transdiaphragmatic rupture is suggested by the discovery of anchovy paste or chocolate sauce pleural fluid on diagnostic thoracentesis. Amoebas can be demonstrated in the pleural fluid in fewer than 10% of patients but in our patient amoebas were not demonstrated in pleural fluid. Approximately one third of patients with transhepatic rupture also have bacterial infection of their pleural space.³ In our patient leukocytosis may suggest that patients have secondary infection and sterile pus culture might be due to prior antibiotic course. Although acute pericarditis is mostly reactive in these cases due to irritation by surrounding pleural effusion; it may occur due to rupture of abscess in pericardial cavity. In our case; pericarditis was reactive as pericardial effusion was not seen.

CONCLUSION

Amoebic liver abscess produces pleural effusion through diaphragmatic irritation or when the abscess ruptures through the diaphragm into the pleural space. In this situation the pleural fluid is described as “chocolate sauce” or “anchovy paste.” The thoracic complication of amoebic liver abscess are not uncommon but causing massive empyema with reactive pericarditis is a rare finding.

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