Amoebic liver abscess presenting as left empyema thoracis

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Abstract
Amoebiasis is an important parasitic infection in developing countries like ours. Apart from intestinal infection, it can lead to some serious complications like pleural empyema, hepatopulmonary fistula and cardiac tamponade. Here, we present an interesting and rare case of an amoebic liver abscess rupturing into the left pleural space and presenting as left sided empyema thoracis.

Keywords: Amoebiasis, hepatic abscess, empyema thoracis, tube thoracostomy.

Introduction
Infection with Entamoeba histolytica occurs worldwide but is more prevalent in the tropical and subtropical countries. If intestinal infections are far more commonly encountered, tissue infections are quite serious. Liver abscess is the most common extraintestinal complication of amoebiasis. Pleuropulmonary complications (i.e., thoracic amoebiasis) are the second most common tissue complication and probably the most important, often associated with liver abscesses. They can manifest as pleural effusion, lung abscess or pleural empyema. Recently, invasive amoebic infections have been reported in increased numbers in male homosexuals and immunocompromised patients.

Case Report
A 20 year old male presented to us with complaints of left sided chest pain, upper abdominal pain since 1 month and high grade fever with chills and rigors, breathlessness & cough with expectoration since 10 days.

He was a rickshawpuller by occupation. He had history of I.V. drug abuse, smoking and alcohol intake since 5 years. On examination, patient was febrile and pale. On auscultation, breath sounds were decreased on left side. Chest X-ray showed left sided pleural effusion. (Fig – 1)

On pleural aspiration, chocolate coloured pus was aspirated.
He was sent for a CT scan chest. On CECT chest, there was a hepatic abscess in segment II with its rupture into the left pleural cavity via a rent of 69mm in the dome of the left diaphragm. Multiple splenic abscesses were also present. (Fig – 2a, 2b)

Fig 2: CECT Chest showing hepatic abscess in segment II with its rupture into the left pleural cavity via a rent of 69mm in the dome of the left diaphragm.
Lab investigations revealed Hb - 10 G%, TLC – 12,300/mm³, DLC- P30, L70, E0, B0 ; ESR – 140 mm at the end of first hour; S. Bilirubin – 0.8 mg%; SGOT – 56 IU; SGPT – 64 IU; S. ALP-106 mg. He was diagnosed as HIV reactive.

The pus showed the presence of trophozoites.

He was put on metronidazole and tube thoracostomy was done for drainage of left sided empyema.

His general condition improved dramatically.

Discussion

Amebiasis, as an infectious disease is one of the leading causes of mortality and morbidity worldwide. More than 500 million people are infected by the parasite, Entamoeba histolytica. The primary infection results in acute amebic colitis, which is commonly benign. Tissue infection is always secondary to colic amebiasis. It is due to embolization of E.histolytica through the portal vein, inside the liver, leading to focal necrosis and subsequently an abscess formation.  

Pleuropulmonary complications occur in approximately 20% of patients with amebic liver abscesses. It is the third most common manifestation of amoebiasis in the body after amoebic colitis and liver abscess. It may be primary or secondary. At present, the term 'primary' lung involvement simply denotes the absence of liver amoebiasis. This could result from haematogenous or lymphatic spread directly from the primary source of intestinal ulceration. 'Secondary' pleuropulmonary amoebiasis always follows amoebic liver abscess, which may manifest itself or may be occult.

The theoretical mechanisms of thoracic dissemination include: direct rupture of a hepatic abscess through the diaphragm; lymphatic spread through the diaphragm; hematogenous dissemination of trophozoites; or inhalation of E. Histolytica cysts. Pleural effusion in association with amoebic liver abscess, arises by two mechanisms. The first occurs when an amebic abscess produces diaphragmatic irritation and a sympathetic pleural effusion, similar to that seen with pyogenic liver abscesses. Amebic liver abscess also produces pleural effusion 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 disease is suspected in endemic areas in persons presenting with fever, pain abdomen and liver tenderness. Occasionally, its rupture into the pleural cavity is signalled by abrupt increase in pain, sometimes a tearing sensation, followed by rapidly progressive respiratory distress and sepsis, which may even lead to shock. The level of alkaline phosphatase is elevated in more than 75% of patients, whereas the levels of transaminases are elevated in 50%. Demonstration of E. Histolytica although diagnostic, is demonstrable only in one fourth of the patients. The sputum, aspirated fluid and the stools should also be examined for the parasitic trophozoites.

The prognosis, at present, for a patient with pleuro-pulmonary amoebiasis is very good. With early diagnosis and adequate treatment, a patient of pleuro-pulmonary amoebiasis can be saved from the significant mortality and morbidity which is associated with the disease. Lethal ruptures of hepatic abscesses or pleural empyemas in the pericardium have previously been reported (mainly in patients with left sided amoebic pleural empyemas), highlighting that great diligence is required from physicians to diagnose pleural amoebiasis, a rare but a potentially fatal condition. Mortality can reach up to 15% in endemic areas.

Conclusion

Hepatic abscesses usually rupture into the right pleural cavity. This rare case shows an amoebic liver abscess presenting as a left sided empyema. Thus, ruptured hepatic abscess should also be kept in mind while dealing with a case of left sided empyema. As it can be a potentially fatal condition, clinicians should have a high index of suspicion in such cases especially in endemic areas so as to diagnose it early and treat effectively.
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References