Rupture of Left Lobe Amoebic Liver Abscess into the Left Lung- A Case Report

Abstract- Amoebic infection is endemic in tropical and subtropical countries and still remains a common cause of chronic morbidity in these areas. We report a case of amoebic liver abscess (ALA) in a 30-year-old male which had ruptured into the left lung parenchyma and pleural space. This was followed by thoracotomy and evacuation of pus with repair of the diaphragmatic defect and parenchymal defect.
Rupture of amoebic liver abscess into left lung is very rare and the clinician should suspect this presentation.

Keywords: **Amoebic liver abscess, pleuropulmonary involvement, thoracotomy, hepato-bronchial fistula**

1. Introduction

Amoebiasis is a common infection in developing countries caused by the protozoan *Entamoeba histolytica*. **Amoebic liver abscess** (ALA) is the most frequent extraintestinal presentation of the disease and occurs in 3-9% of patients with intestinal amoebiasis.
The lung happens to be the second most common extraintestinal site of amoebic involvement after the liver. Usually the lower lobe, and sometimes the middle lobe of the right lung, are affected, but it may affect any lobe of the lungs. The abscess can rupture into the pleura to cause respiratory distress. [1]

Pleuropulmonary amoebiasis is **uncommon** and easily confused with other illnesses and is treated as pulmonary TB, bacterial lung abscesses, and carcinoma of the lung. [1]. Around 22% of ALA rupture leading to further complications. The exact incidence of pleuropulmonary amoebiasis is unknown, however it is estimated to be involving around 50% of the complicated cases [11]. Increase in morbidity and mortality is attributed to low index of suspicion of pleuropulmonary amebiasis causing a delay in diagnosis. The prognosis improves with early diagnosis and prompt treatment. Here we report a case of left sided pleuropulmonary amoebiasis which is very rare.

2. Case Report

A previously healthy 30-year-old Male resident of Hooghly, West Bengal, India presented with pain in the Right Hypochondrium for 2 months, intermittent fever with chills and rigour, for 30 days duration associated with anorexia. There was a history of diarrhoea 15 days prior to the start of these symptoms. On physical examination, the patient was alert, conscious and cooperative with tachypnoea. He was febrile and tachycardiac, without any elevated blood pressure and oxygen saturation was 98%. There were no known risk factors such as alcoholism, diabetes, long-term steroid use. The abdomen showed right upper abdominal fullness with guarding and tenderness in the region. Examination of the respiratory system revealed decreased air entry in left lower lung. On patient admission, routine blood tests revealed elevated total leucocyte count at 13,900 per mm3 with neutrophilia, haemoglobin 11.0 g/L, elevated PT INR 1.9. Rest of the investigations including Alkaline phosphatase, Hepatic transaminases, bilirubin level and blood gas were within normal limits.
Imaging studies were conducted. Chest Xray showed left sided pleural effusion [Fig. 1]. Ultrasonography of the
abdomen showed a huge liver abscess in the left lobe of the liver. Computed Tomography (CT) scan was done which showed liver abscess 12*9 cm in the left lobe of liver with hepatomegaly. [Fig. 2]
After admission patient was started on Piperacillin- Tazobactam 4.5 gm IV TDS and Metronidazole 750mg TDS and was planned for pigtail catheterisation. Patient was given 4 units of FFP to correct the INR, which was corrected on admission day 6. On admission day 7, patient started having paroxysmal bouts of productive cough with typical anchovy sauce pus sputum. There were no signs of peritonitis. Contrast enhanced CT scan of Thorax was done which showed liver abscess has ruptured into the lung parenchyma and left pleural cavity. [Fig. 3] Anchovy sauce pus was aspirated from the 5th Intercostal space in the midaxillary line, which confirmed that the pus had percolated into the pleural space. [Fig. 4]
Due to severe respiratory distress, immediate tube thoracostomy was done on the left side under aseptic measures and local anaesthesia which resulted in minimal evacuation of pus and pleural fluid. A decision to

proceed towards open thoracotomy and drainage was taken on suspecting hepatobronchial fistula due to rupture of amoebic liver abscess.

Intraoperative Management and Findings

Patient was placed supine.

A Left Anterolateral thoracotomy incision was made on the 8th intercostal space after proper antiseptic and draping under general anaesthesia. Left lung was collapsed, pus was sucked out and the defect on the left dome of diaphragm was identified. [Fig. 5] A fistulous tract extending up to the left the lobe of liver was located along with a defect in the lower lobe of the left lung. A malecot catheter was placed within the abscess cavity of left lobe and was brought out through the left lateral abdominal wall. [Fig. 6]

The diaphragmatic defect was repaired in double layer in continuous fashion with 2-0 Prolene suture. [Fig. 7, 8]

The necrotic part of the lower lobe of left lung was debrided and the bronchopleural fistula was ligated with 3-0 PDS suture. [Fig. 9]

Left pulmonary cavity was washed with warm normal saline and two chest drains were placed in the apex and base of the left thoracic cavity. Intercostal muscles were closed with 2-0 Vicryl and skin was closed with 2-0 linux.

The postoperative diagnosis remained the same i.e., Liver Abscess which ruptured into lungs and the patient was shifted to the intensive care unit and was kept in propped-up position to avoid dissemination of the ruptured material into lung parenchyma. In the postoperative period, patient was started on Cefoperazone-Sulbactam (2g q12h) and Metronidazole (750 mg) till the 7th post-operative day. Chest Drains were removed on 5th post-operative day. Malecot Catheter was removed on 13th post-operative day. Overall, patient had an uneventful post-op period and was discharged on 14th post-operative day.

3. Discussion

Liver abscess formation is the commonest extraintestinal manifestation with characteristic 'anchovy-sauce' pus.[1]

Amoebiasis is a major parasitic infection in developing countries. It is a common cause of recurrent diarrhoea and blood-tinged or mucoid stool. An estimated 60,000 to 100,000 people die every year from amoebiasis worldwide.

With treatment, the death rates vary between 2 and 3%. Transmission is usually via contaminated food or water

but can be associated with sexual contact through faecal oral contact. The lung is the second most common extraintestinal site of amoebic involvement after the liver. It is due to embolization of *E. histolytica* inside the liver through the portal vein, leading to focal necrosis and then an abscess. Pleuropulmonary complications (i.e., thoracic amoebiasis) are the second most frequent tissue complication, often associated with liver abscesses. They manifest in pleural effusion, lung abscess and, rarely, pleural empyema.

Usually, the lower and middle lobes of the right lung, are affected, but it may affect any lobe of the lungs.

Theoretically, the mechanisms of thoracic amoebiasis are as follows [1,3]. First, the infection usually spreads to the lung by rupture of an amoebic liver abscess through the diaphragm. Second, the infection may disseminate to the thorax directly from the primary intestinal lesion through hematogenous or lymphatic spread. Lastly, inhalation of dust containing cysts of E. histolytica is also a hypothetical route. Infection spreads to the thorax through the ruptured diaphragm into the pleural space or even in the lung parenchyma, producing hepatobronchial fistula.

Left lobe amoebic liver abscesses are of particular concern because risk of rupture into the pericardium is increased. However, the rupture of left lobe liver abscess into the left lung parenchyma is extremely rare.

Immediate intervention is required to repair the hepatobronchial fistula as bile might enter the lung and cause permanent damage to the alveoli.

The diagnosis of thoracic amebiasis is suggested by the combination of an elevated hemidiaphragm, hepatomegaly, pleural effusion, and involvement of the lung base in the form of haziness and obliteration of costophrenic and cardio- phrenic angles. Diagnosis of thoracic amoebiasis by finding *E. histolytica* in stool specimens is of limited value. In a limited number of cases, amoebae might be found in aspirated pus or expectorated sputum. Liver enzymes are usually normal and neutrophilic leucocytosis may or may not be found. Serology tests are of immense value in diagnosis. Anti- amoebic antibody can be detected by indirect hemagglutination (IHA) or enzyme-linked immunosorbent assay (ELISA). Amoebic antigen can be detected from serum and pus by ELISA.[6]

Recently, the detection of *E. histolytica* DNA in urine or saliva provides a new approach for the diagnosis of amoebiasis. [4,5]

Aspiration and drainage of pus from thoracic empyema is needed for confirmation and therapeutic purposes. Amoebic liver abscess has been recommended to be treated with metronidazole or tinidazole plus a luminal amebicide viz. iodoquinol even if intestinal infection is not documented. [7,8] Most amoebic liver abscesses respond to medical treatment, and metronidazole at 750mg for 7 to 10 days is the amoebicidal treatment of choice. [10]

Metronidazole can be used intravenously if required. Needle aspiration or catheter drainage is helpful for large abscess (over 5-10 cm), in particular if the diagnosis is uncertain, if there is an initial lack of response, or if a patient is very ill, suggesting abscess rupture. [2] Though the treatment options vary from medical to open drainage, it is now universally accepted that aspiration and/or surgical drainage of ALA together with anti-amoebic therapy is effective. [9].

4. Conclusion

The physicians must be aware of the epidemiology of this disease, and consider it in patients presenting with symptoms indicating pleuropulmonary amoebiasis. Medical management is considered as the first line however in ruptured ALA prompt intervention via surgery or image-guided drainage is necessary and should not be delayed.

5. Informed Consent

The patient gave informed consent

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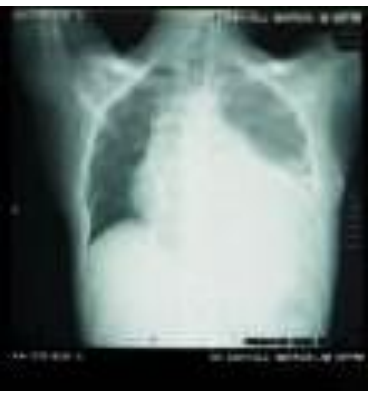

Figure 1. Left Sided Pleural Effusion seen on Radiograph of Upper Neck and Chest in Anteroposterior view

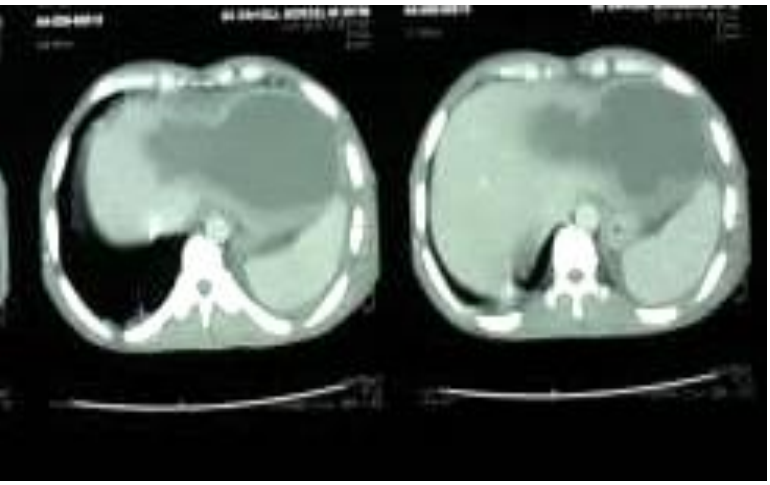

Figure 2 – Initial abdominal Computed Tomography (CT) scan showing Left Lobe liver Abscess 12*9 cm in the left lobe with left sided reactive pleural effusion.

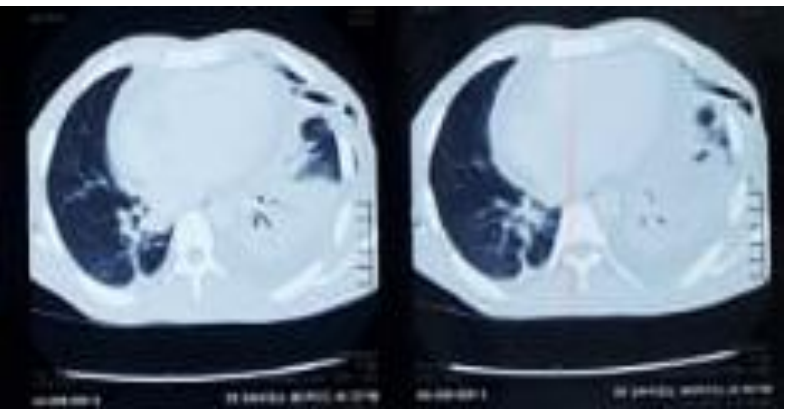

Figure 3 – Contrast enhanced CT (CECT) scan of Thorax on Day 7 of admission showing liver abscess which has ruptured into the lung parenchyma and left pleural cavity

Figure 4 – Anchovy sauce pus aspirated from the left 5th Intercostal space

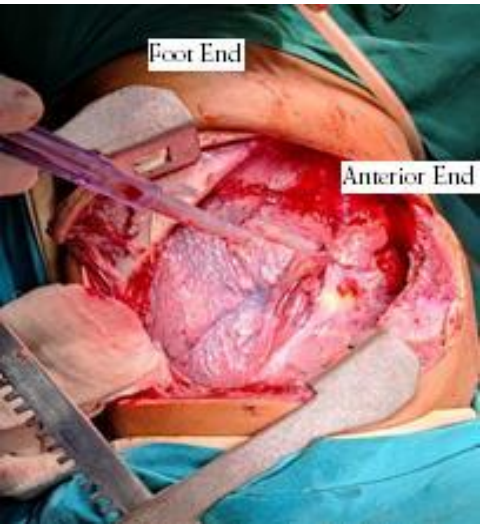

Figure 5 - Pus Being Aspirated from the Left Pleural cavity

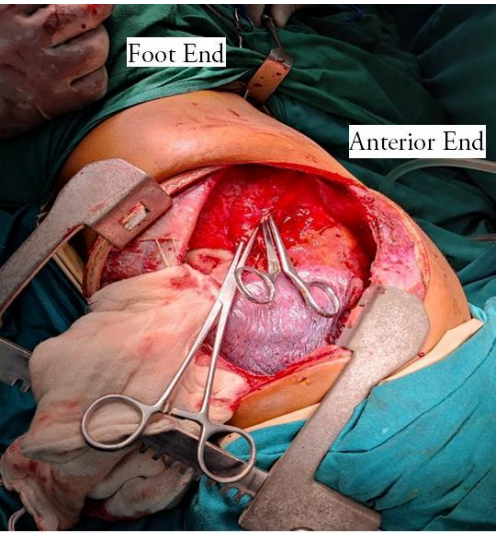

Figure 6 - Roberts Artery forceps passed through the defect in the diaphragm and brought out via the left subphrenic space for placement of Malecot Catheter

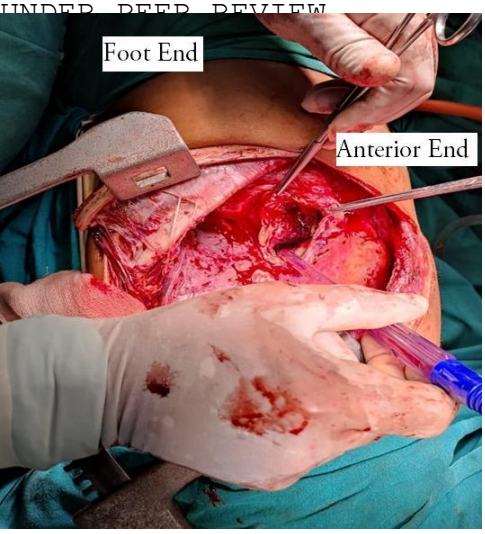

Figure 7 - Rent in the Diaphragm leading to the liver abscess cavity shown with the help of instruments and pus being sucked out from the abscess cavity.

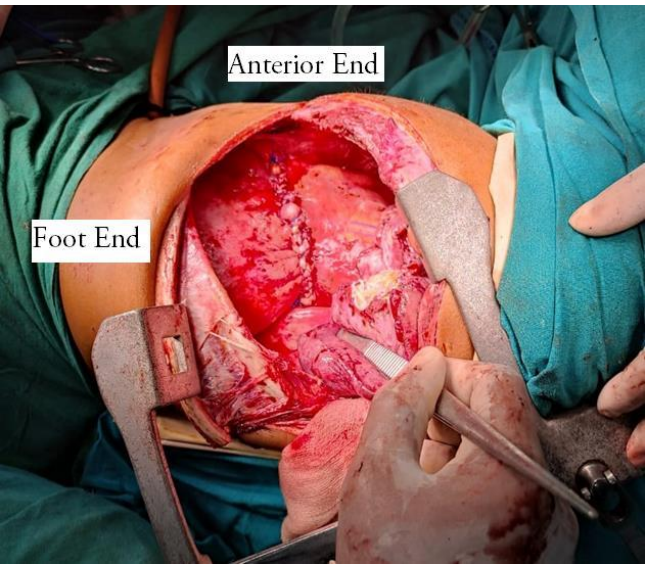

Figure 8 - Diaphragmatic Defect repaired with 2-0 Prolene in double layer continuous fashion

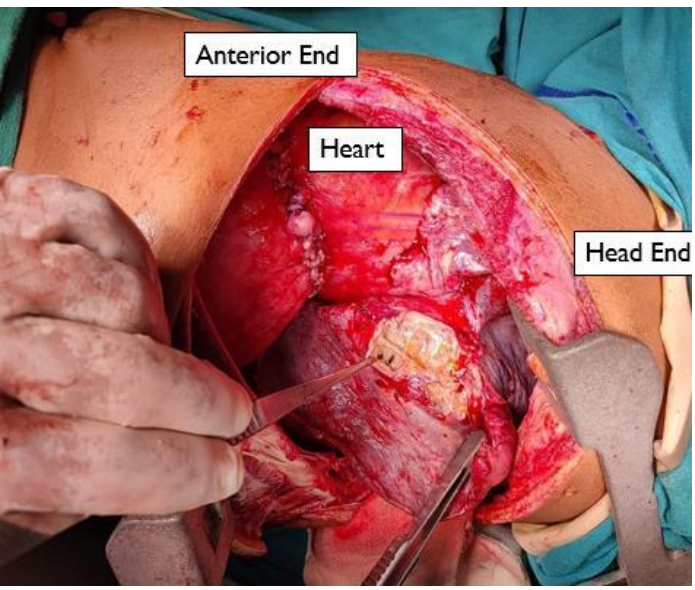

Figure 9 - Defect in the lung parenchyma is shown via forceps. The defect was repaired with 3-0 PDS. Chest drains were placed in the basal and apical pleural cavity.