CASE REPORT

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Amoebic Liver Abscess and Inferior Vena Cava Thrombosis – A Rare Case Report

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Abstract

Amoebic liver abscess is the most common extra-intestinal manifestation of infection with Entamoeba histolytica. It is a common disease especially in endemic areas, but it is a rare cause of inferior vena cava thrombosis, with only a few cases appearing in literature. Imaging techniques, serological tests, image-guided interventional procedures, and appropriate therapeutic regimens have significantly reduced mortality, yet the disease is associated with many complications and can be fatal if untreated. We describe one such case of inferior vena cava (IVC) thrombosis which presented as a rare complication of liver abscess. The case responded well to radiological intervention and pharmacological treatment including anti-thrombotic medication.

Key words: Amoebic liver abscess, complication, inferior vena cava thrombosis.

Introduction

Amoebic liver abscess is a collection of pus in the liver in response to the intestinal parasite *Entamoeba histolytica*. This parasite causes amebiasis, an intestinal infection that is also called as amoebic dysentery. Once infection occurs, the parasite may get carried by the blood stream from the intestine to liver. This infection occurs worldwide. Amoebiasis is the second leading cause of death from parasitic disease worldwide. It can be diagnosed easily by ultrasound and amoebic serology, but CT scan of abdomen is the best tool to diagnose its complications. Complications include rupture of liver abscess into adjacent pleural, pericardial, peritoneal cavities, and rarely into gastrointestinal tract. Vascular complications in the form of portal vein, hepatic vein and inferior vena cava thrombosis is also known to occur rarely.

Case report

A 36-year-old male, known case of hypertension, presented with history of fever with chills from the past 8 - 10 days along with history of breathlessness and pain in abdomen from the past two days. On examination, the patient was conscious but with a toxic look, mild icterus was present, blood pressure was 120/70 mmHg, pulse rate was 128 per minute with normal volume, respiratory rate was 28 per minute, SpO2 92% on room air, and bilateral air entry diminished in chest with a two fingers tender, palpable liver. So we summarised this case of pyrexia, breathlessness, icterus with hepatomegaly with the differential diagnosis of pneumonia with septicaemia, malaria fever, liver abscess.

The haematological investigations revealed a haemoglobin of 12.7 gm%, TLC - 15,360/cumm, platelets - 1.34 lacs/cumm, total bilirubin - 4.3 mg/dl, direct bilirubin - 3.8 mg/dl and indirect - 0.5 mg/dl, SGOT - 83 IU/L, SGPT - 73 IU/L, ALP - 387 U/L, total protein - 5.8 gm/dl, albumin - 2.8 gm/dl, globulin - 3.0 gm/dl, serum sodium - 139 meq/l, serum potassium - 4.0 meq/l, blood urea - 23 mg/dl, serum creatinine - 0.9 mg/dl, RBS - 118 mg/dl, Typhi dot IgM negative, PS for malaria and MP serology-negative, serum procalcitonin - 4.10, serum lipase - 12 iu/l, PT with INR - 1.25, APPTc - 28.6, APPTt - 31.6, amoebic serology - 1.754 (positive), anti HAV - negative, anti-HEV - negative, anti HCV - negative, HbsAg - negative, Elisa for dengue IgM - negative, HIV 1 and 2 - negative, rapid test for leptospira IgM - negative, blood and urine culture - sterile. Arterial blood gas analysis showed a pH - 7.381, pCO2 - 25.8 mmHg, pO2 - 67 mmHg, BE - 10 mmol/l, HCO3 - 15.3 mmol/l sO2 - 93% suggestive of compensatory metabolic acidosis. Chest X-ray showed mild raised right-sided diaphragm with bilateral haze in both lower zones. Abdomen ultrasound suggestive of a large liver abscess (10.8 x 7.5 x 10.2 cm) with suprahepatic inferior vena cava thrombus (2.8 x 1.4 cm). 2D echocardiography showed no RWMA with an ejection fraction of 65% and no vegetations on cardiac valves. We performed USG guided pigtail insertion and drainage of abscess with 120 cc of anchovy pus, which was sent for amoebic serology. Patient was started on injection monocef 2 gm twice daily, injection metrogyl 750 mg thrice daily, inj fragmin 5,000 units subcutaneous twice daily, and injection albumin 20% OD. Nebulisation and incentive spirometry was advised along with oxygen supplementation via nasal

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prongs in propped-up position. The patient started showing dramatic improvement, but chest X-ray still showed left-sided plural effusion. Hence, for further evaluation CECT chest and abdomen was advised which revealed hepatomegaly with partially liquefied abscess with pigtail in situ, moderate right pleural and mild left pleural effusion with minimal ascites and non visualised right hepatic vein ? thrombosed and a small thrombus in the IVC. Patient was discharged in stable condition on tablet levoflox 750 mg OD, tablet metrogyl 800 mg TID for 10 days , tablet ceftum 500 mg BID, cap superia DSR before breakfast and tab warfarin 5 mg per day and was advised to monitor PT with INR weekly and keep INR in the range of 2.5 to 3 for 6 months. Patient improved dramatically, pigtail catheter was removed after few days and all the medications were stopped after 14 days. Warfarin was stopped and the patient was shifted to tablet afogatran 110 mg BID. He was discharged in a stable condition.

Discussion

Amoebic liver abscess is the most common extra-intestinal manifestation of infection with *Entamoeba histolytica*. ALA develops in less than 1% of patients infested with *E. histolytica*, but still represents a large number of patients, especially in endemic areas. It is a rare cause of IVC thrombosis with only few cases reported in literature. The diagnosis of amoebic liver abscess relies on the identification of a space-occupying lesion of the liver and positive amoebic serology. Ultrasound abdomen is the preferred and easy choice to diagnose, but CT scan is ideal to detect liver abscess, particularly smaller lesions and associated complications. The rate of various complications was reported to be 10.3% including rupture in the pleural, pericardial and peritoneal cavity, rupture into bile ducts and vascular thrombosis. Though thrombosis of hepatic vena cava is rare, referred to as obliterative hepatocavopathy, is described mostly in autopsy studies. However, a recent case report described this complication prospectively but the exact pathophysiology of IVC thrombosis in amoebic liver abscess is uncertain. Proposed mechanisms include external mechanical compression, thrombotic state associated with inflammatory process of amoebiasis and an adjacent spread of inflammation. In our case, we suspected that the inflammatory process in the wall of the amoebic abscess spread and caused injury to the IVC wall, leading to inflammation followed by thrombosis. Hepatic abscess in the close proximity to the IVC or hepatic veins should be investigated using CT or Doppler ultrasonography. Coagulation profile should be assessed in order to rule-out any pre-existing thrombogenic state. The management of amoebic liver abscess with IVC thrombosis mainly includes anti amoebic, antibiotics and drainage of abscess but in few cases, anticoagulation therapy may be needed to achieve complete resolution. The management of complicated amoebic liver abscess is operative. Extension of thrombus up to the right atrium mandates aggressive management with thrombectomy to reduce chances of pulmonary embolism.

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