

Amebic Liver abscess Complicated by Inferior Vena Cava Thrombosis: A Case Report

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SUMMARY

Amebic liver abscess is the most common extraintestinal 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 (IVC) obstruction, with only a few cases appearing in the literature. The authors describe a case of amebic liver abscess in a patient who developed a rare vascular complication of inferior vena cava thrombosis. The case responded to conservative treatment and radiological intervention.

KEY WORDS:

Amebic liver abscess, extraintestinal, inferior vena cava, vascular complication, thrombosis

INTRODUCTION

Amebiasis occurs in 10% of the world's population and is most common in tropical and subtropical regions¹. Hepatic involvement is common and may develop in 3-9% of cases, with rates of infection being higher for men¹. The organism enters the liver through the portal circulation, where an abscess may develop. Complications of an amebic liver abscess (ALA) include its rupture into the pleural, pericardial, and peritoneal spaces. Vascular complications in the form of thrombosis or compression resulting in either hepatic venous outflow obstruction or inferior vena cava (IVC) obstruction are rarely seen in ALA. There have been only a handful of case reports of ALA with IVC obstruction. We present a case of inferior vena cava thrombosis in a patient with amebic liver abscess.

CASE REPORT

A 47-year-old male presented with high grade fever with occasional chills and right-sided abdominal pain for 15 days, yellowish discoloration of eyes and swelling of the feet for 7 days. The pain was continuous, dull aching with no radiation. He reported normal bowel function. He had a 10-year history of alcoholism and was a labourer.

On examination, he was febrile (oral temperature 39.2° C) and icteric. Blood pressure was 114/80 mm Hg, pulse rate 90 beats/min, and respiratory rate 22 breaths/min. His abdomen was moderately distended, soft and tender in the right hypochondrium. There were few prominent back veins and the venous flow was below upward. The liver was enlarged 6 cm below the right costal margin and it was firm, tender, and smooth. There was no ascites or splenomegaly.

Hematological investigations revealed a hemoglobin level of 10.4 gm/dL, leucocyte count 11,600/mm³ with

predominantly polymorphic leukocytosis. The biochemical investigations showed a raised blood urea of 64 mg/dL, serum creatinine of 1.4 mg/dL. Liver function tests showed total bilirubin of 8.4 mg/dL, direct bilirubin of 6 mg/dL, alkaline phosphatase (ALP) of 1134 IU/L, aspartate transferase (AST) of 46 IU/L and alanine transferase (ALT) of 75 IU/L. Ultrasound of the abdomen showed hepatomegaly with a heterogeneously hypoechoic rounded lesion measuring 15 × 10 cm mainly in the right lobe and involving the inferior surface of liver, compressing the common bile duct [Fig 1A]. The abscess cavity was abutting the IVC with the possibility of thrombus-like lesion in the inferior vena cava [Fig 1B]. Abnormal color fill in the IVC was found on colour doppler [Fig 2A]. A computerized tomography scan of the abdomen revealed an abscess cavity and confirmed the findings of thrombus in the IVC [Fig 2B]. The patient tested positive for amoebic antibody by enzyme linked immunosorbent assay (ELISA) test. A provisional diagnosis of amebic liver abscess was made; the patient was treated with intravenous metronidazole 750 mg every eight hours and ceftriaxone 2gm 12 hourly. Under ultrasound guidance, brownish fluid (anchovy sauce-like) was aspirated and pigtail catheter (14 Fr) drainage of the abscesses was performed. There were no trophozoites seen on the microscopic evaluation of the pus and no organisms were isolated on culture. A coagulation workup was done including proteins C and S that showed non-specific derangement, probably associated with inflammation. Liver function tests returned to the normal range. The patient was discharged in good condition and follow up ultrasound done at the end of three months showed no residual abscess and the IVC was normal in caliber, color fill and flow pattern [Fig 2C].

In this particular case, an amebic liver abscess presented with two unusual complications of jaundice and transient IVC thrombosis. Based on the typical ultrasound and CT scan findings, all possibilities except that of liver abscess were excluded.

DISCUSSION

Amebic liver abscess (ALA) is the most common extraintestinal manifestation of amebiasis. ALA develops in less than 1% of patients infested with *Entamoeba histolytica*, but this still represents a large number of patients². The disease should be suspected in anyone with a history of residency in or travel to an endemic area and fever, right upper quadrant pain, and substantial hepatic tenderness.

The diagnosis of ALA relies on the identification of a space occupying lesion of the liver and positive amebic serology¹.

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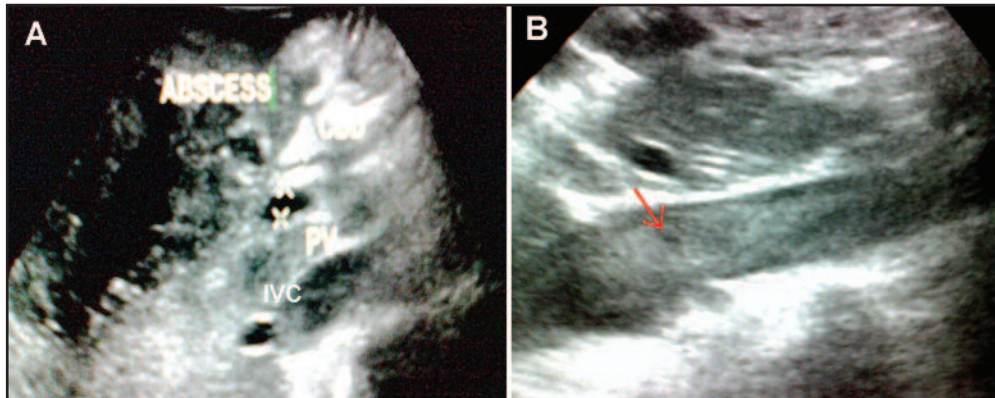


Fig. 1: (A). A large heterogenous cavity involving inferior surface of right lobe of liver, compressing common bile duct; (B). Inferior vena cava shows increased echogenicity (red arrow).

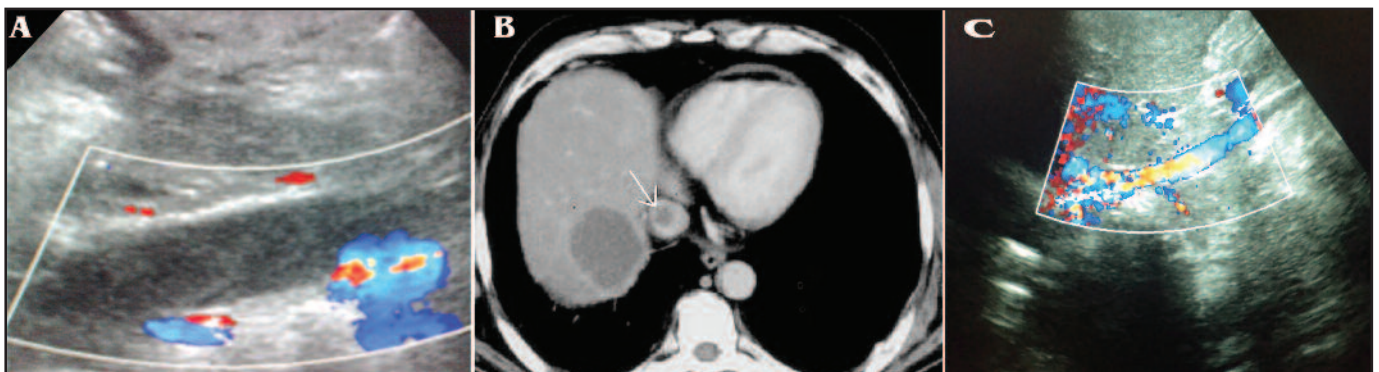


Fig. 2: (A). IVC lumen showing no colour filling; (B). CT scan of the abdomen shows a portion of liver abscess and intraluminal filling defect (white arrow), in the inferior vena cava, suggestive of thrombosis; (C). Repeat ultrasound at three month showed no residual abscess with normal color fill and flow pattern in IVC.

Ultrasound of the abdomen should be performed routinely in patients presenting with abdominal pain and fever of more than one week duration especially in endemic areas. Computed tomography is ideal to detect liver abscesses, particularly smaller lesions and its associated complications. The rate of various complications described in an ALA was reported to be 10.3%³. Complications associated with amebic liver abscess include rupture into pleural, pericardial and peritoneal cavity, vascular thrombosis, and rupture into the bile ducts. Though rare, thrombosis of the hepatic inferior vena cava, referred to as obliterative hepatocavopathy, is a reported complication of hepatic amebiasis described mostly in autopsy studies⁴. However, a few recent case reports described this complication prospectively.

The exact pathophysiology of IVC thrombosis occurrence in hepatic amebiasis is uncertain. Proposed mechanisms include external mechanical compression, a thrombotic state associated with the inflammatory process of amebiasis, an adjacent spread of inflammation⁵. Inflammatory processes associated with the amebic abscesses could potentially predispose a patient to a thrombotic state.

In our case we suspect that the inflammatory process in the wall of the amebic abscess may spread and cause injury to the IVC wall, leading to inflammation followed by thrombosis. External mechanical compression due to a large abscess with resultant sluggish circulation was also considered. Hepatic abscess in close proximity to the IVC or hepatic veins should be investigated using CT or Doppler

ultrasonography. Coagulation system should be assessed in order to rule out a pre-existing thrombogenic state.

The management of ALA with IVC thrombosis mainly includes antibiotics and drainage of the abscess, but in a few cases, anticoagulation therapy may be needed to achieve complete resolution. The management of complicated ALA is still evolving with controversies regarding operative treatment but most of the literature supports non-operative management to achieve a better outcome. Extension of thrombus up to right atrium mandates aggressive management with thrombectomy to reduce chances of pulmonary embolism.

To conclude, we believe that in our case the strategically placed abscess in the anteroinferior surface of right lobe of the liver led to compression of the biliary radicles anteriorly at the porta and the IVC posteriorly, giving rise to obstructive jaundice and IVC thrombosis. Both of the complications were resolved following the drainage of the abscess.

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