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Case Report/Case Series

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Vascular complications of amebic liver abscess – Computed tomography case series with review of the literature

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Abstract:

Amebiasis is a parasitic infection with amebic liver abscess (ALA) being the most common extraintestinal manifestation. Common complications of ALA include rupture into the pleural, pericardial, or peritoneal cavity. Uncommonly, they can cause vascular complications such as thrombosis of the hepatic vein and inferior vena cava which may further extend to the right atrium or may embolize resulting in pulmonary thromboembolism. In this study, we report three patients with vascular complications in ALA. The presence of vascular pathology in ALAs should not be missed. With its detection and prompt treatment, the progression of vascular complications can be prevented.

Keywords:

Amebic liver abscess, hepatic vein thrombosis, inferior vena cava thrombosis

Introduction

Amebiasis is a parasitic infection caused by *Entamoeba histolytica* with amebic liver abscess (ALA) being the most common extraintestinal manifestation. It is the fourth leading cause of mortality globally with the prevalence ranging from around 8% to 14%.[1] The organism crosses the intestinal mucosa and then reaches the liver through the portal circulation. The common complications of an ALA include rupture into the pleural, pericardial, or peritoneal cavity. Inferior vena cava (IVC) or hepatic venous outflow obstruction or thrombosis is some of the uncommon consequences. The thrombus may extend into the right atrium or may embolize resulting in pulmonary thromboembolism. These vascular problems, albeit they are uncommon, can be life-threatening.[2-6]

We present three such cases of vascular complications in patients with ALA referred to our department.

Case Reports

Case 1

A 49-year-old male presented with pain in the epigastrium with fever and vomiting for 5 days with leukocytosis and low platelet count. Liver function tests were deranged. The patient was hepatitis C virus reactive. On ultrasonography, hepatomegaly with two hypoechoic lesions in the right lobe of the liver was seen (4 cm × 4 cm in Segment VIII and 3 cm × 3 cm in Segment VI subcapsular in location). Contrast-enhanced computed tomography (CT) abdomen [Figure 1a-c] revealed hepatomegaly with two nonenhancing and ragged-edged hypodense lesions with the presence of multiple septa with perilesional edema (Segment VIII-4.3 cm × 3.9 cm × 3.5 cm with

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wedge-shaped surrounding hypodense area, Segment VI-3.4 cm × 3.3 cm × 2.6 cm with thinning of overlying hepatic mantle suggestive of impending rupture). A hypodense filling defect was noted in one of the tributaries of the middle hepatic vein communicating with the lesion in Segment VIII-suggestive of thrombosis of a tributary of the middle hepatic vein. Under ultrasound guidance, brownish fluid (anchovy sauce-like) was aspirated. The patient tested positive for amebic antibody by enzyme-linked immunosorbent assay (ELISA) test and was started on intravenous metronidazole for 10 days, followed by diloxanide furoate for 20 days. No percutaneous drainage was attempted as the patient showed good response to medical treatment in a week.

Case 2

A 6-year-old male presented to a surgical emergency with chief complaints of fever and abdominal pain for 7 days with severe anemia, leukocytosis, low platelet count, deranged liver function tests, and raised C-reactive protein values. Ultrasonography revealed hepatomegaly with a hypoechoic lesion in the left lobe of the liver (3 cm × 3 cm) with free fluid in the pelvis, Morrison's space, and perisplenic region. Contrast-enhanced CT abdomen [Figure 1d-f] revealed left-sided pleural effusion with free fluid in the pelvis, perisplenic region, and bilateral paracolic gutters. The liver showed a nonenhancing hypodense lesion (4.7 cm × 4 cm × 4.9 cm) with ragged edges and multiple septa, incomplete wall, and interrupted enhancement of the edges in the left lobe (subcapsular) that seems to be communicating with the peritoneal cavity suggestive of rupture with diffuse hypodensity in the left lobe. The left hepatic vein was hypodense suggestive of thrombosis. The patient tested positive for amebic antibody by ELISA test. A provisional diagnosis of ALA was made, and the patient was managed medically with metronidazole and diloxanide furoate. Percutaneous drainage with a pigtail catheter was also done under ultrasonography (USG) guidance. The patient improved with treatment.

Case 3

A 16-year-old male patient was presented in emergency with chief complaints of pain in the right hypochondrium, fever for 7 days, and vomiting for 1 day. The patient had tachycardia, icterus, and tenderness in the right hypochondrium. Blood investigations revealed mild anemia with leukocytosis with low platelet count. Prothrombin time was deranged. Liver function tests were elevated. Ultrasonography revealed hepatomegaly with a large (9 cm × 7 cm × 9 cm), heterogeneously hypoechoic lesion with internal echoes in Segments VII and VIII of the right lobe of the liver suggestive of the liver abscess with free fluid in the pelvis and Morrison's space. The abscess was compressing the intrahepatic IVC with no flow on the color Doppler. Contrast-enhanced CT abdomen [Figure 2a-f] was performed which confirmed free fluid in the pelvis, bilateral paracolic gutters, and Morrison's space with hepatomegaly with nonenhancing and ragged-edged hypodense lesions with multiple septa with no definite wall abutting the liver capsule (10.8 cm × 10.6 cm × 8.7 cm) with perilesional edema in Segments VII and VIII of the liver with a suspicious rent into the pleural cavity in its superomedial aspect. The lesion was causing eccentric compression over intrahepatic IVC. A hypodense filling defect was also seen extending from the right hepatic vein into IVC suggestive of thrombosis with extension till the right atrium. The patient tested positive for amebic antibody by ELISA test. He was put on medical treatment with metronidazole followed by diloxanide furoate with drainage of the abscess under USG guidance. On follow-up, the patient showed good response.

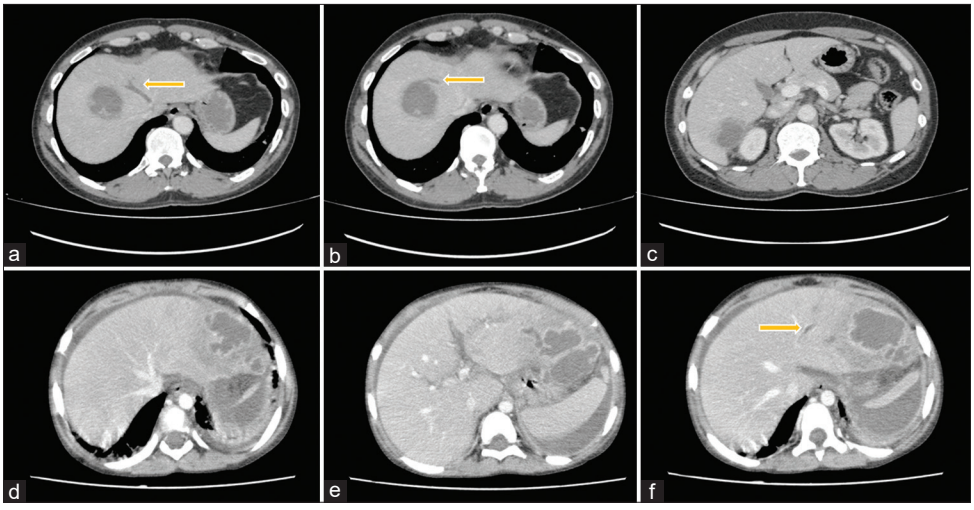

Figure 1: Case No. 1 (a-c) two liver abscesses in Segments VI and VIII, respectively, with thrombosis of the middle hepatic vein (arrow). Case No. 2 (d-f) left liver lobe abscess with thrombosed left hepatic vein (arrow)

Discussion

Amebiasis is one of the leading parasitic causes of death worldwide. The vascular complications may include hepatic vein thrombosis (Case 1, 2, and 3) which may extend into IVC or right atrium (Case 3). Previous studies with IVC and/or hepatic vein thrombosis in ALA are discussed in Table 1, with only few case reports of right atrial thrombosis. Such complications have rarely been reported in pediatric patient (Case 2). Gupta *et al*. reported a case of a 6-year-old boy with an ALA with thrombosis of the middle and left hepatic veins with a thrombus in the IVC which extended up to the right atrium.[13] A similar case was reported by Aneja *et al*. where a 7-year-old child with ALA who developed pulmonary thromboembolism was promptly diagnosed and managed.[14] The proposed mechanisms for thrombotic events include proximity of the abscess with venous structures or external mechanical compression with resultant sluggish flow. The prolonged endothelial cell activation would induce severe local inflammation, leading to necrosis that spreads directly to the adjacent vessel wall causing luminal thrombosis.[2-5]

Ultrasonography should be performed in patients presenting with abdominal pain and fever for more than 1-week duration for detection, characterization of the lesion, and further follow-up.[3] Contrast-enhanced CT is an ideal method for diagnosing hepatic abscesses with a sensitivity of 97%, however, its CT appearances are nonspecific. The classical features of the round or oval hypodense lesion with thick enhancing wall and peripheral edema are rather suggestive of the healing process. The abscesses designated as aggressive types

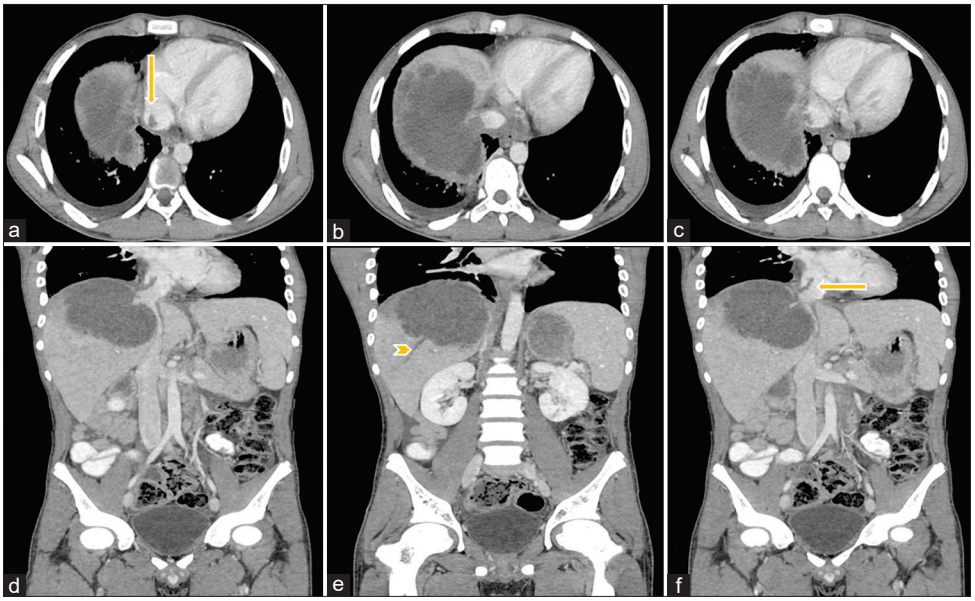

Figure 2: Case No. 3 (a-f) right liver lobe abscess with thrombosis of right hepatic vein (arrowhead) extending into inferior vena cava and further into right atrium (arrow)

Table 1: Review of literature with 9 case reports/series of vascular complications in amebic liver abscess

Author	Case	Complication and mortality
Sarda *et al*.[4]	Three cases of ALA	IVC obstruction Follow-up for 3 months, no mortality reported
Marak *et al*.[7]	Three cases of liver abscess	IVC and portal vein thrombosis. No mortality reported
Lal *et al*.[8]	Four cases of liver abscess	IVC and hepatic vein thrombosis 1 mortality reported
Babu and Singh[9]	36-year-old male	IVC thrombosis. No mortality reported
Khan and Ameen Rauf[10]	46-year-old man	IVC obstruction with low attenuation thrombus partially occluding the lumen with extension into the cavity of the right atrium. No mortality reported
Zia-ur-Rehman *et al*.[11]	32-year-old man	Thrombus in the hepatic veins IVC and floating thrombus extending into the right atrium. No mortality reported
Touré *et al*.[12]	52-year-old man	Thrombus at confluence of hepatic veins extending into IVC and right atrium. No mortality reported
Gupta *et al*.[13]	6-year-old boy	Thrombus in the middle and left hepatic veins together with a thrombus in the IVC which extended up to the right atrium. No mortality reported
Aneja *et al*. (2021)[14]	7-year-old	Right hepatic vein and IVC thrombus with pulmonary thromboembolism. No mortality reported

IVC: Inferior vena cava, ALA: Amebic liver abscess

are commonly multiple in number with severe clinical symptoms and laboratory profiles. The most common risk factors for rupture are large size, left lobe location, and lack of a mature wall. The thickness (<1 cm) of liver tissue between the abscess and liver margin is an objective measure to assess impending rupture. The incidence of colitis was higher with abscesses lacking a mature wall.[6,8] Delay in diagnosis could be fatal and an acceptable outcome is achieved with timely diagnosis. The diagnosis of ALA relies on the identification of the lesion on ultrasound with confirmation of lesion and its associated complications on contrast-enhanced CT abdomen.[4] The presence of vascular pathology in ALAs should not be missed. With its detection and prompt treatment, the progression of vascular complications can be prevented. Management of an ALA with vascular complications is usually medical with percutaneous drainage.[15] However, in a few cases, anticoagulation therapy and surgical intervention may be needed to achieve complete resolution.[3,9,10]

Conclusion

The vascular complications of ALA such as hepatic vein and IVC thrombosis are rare but could be life-threatening. The presence of vascular pathology in ALAs should not be missed. With its detection using radiological modalities such as USG and computer tomography, the progression of vascular complications can be prevented. Early initiation of medical treatment as well as radiological interventions helps in averting complications and results in the timely recovery of the patient.

Author contributions statement

BA (Lead): Conceptualization (lead), reviewing, and editing the final draft (equal), supervised the case management (equal), LK: writing original draft (lead), supervised the case management (equal), reviewing and editing the final draft (equal). SM: reviewing and editing the final draft (equal).

Conflicts of interest

None Declared.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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