Research Article

Amoebic Liver Abscess Complicated By Hepatocaval-Right Atrial Thrombosis And Transdiaphragmatic Pleuropulmonary Rupture: A Case Report

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Abstract

Background: Amoebic liver abscess (ALA) is the most common extraintestinal manifestation of Entamoeba histolytica infection. Thoracic rupture is a recognized complication, whereas extension of hepatic venous thrombosis through the inferior vena cava (IVC) into the right atrium is exceptionally rare and potentially life-threatening. We report an unusual case in which both complications occurred simultaneously.

Case presentation: An 18-year-old man presented with high-grade fever, chills, abdominal distension, and abdominal tenderness. Leukocytosis was present. Ultrasonography showed a hepatic abscess with associated IVC thrombosis, prompting contrast-enhanced CT. CT demonstrated a large, thick-walled, peripherally enhancing abscess measuring approximately 6.8 cm and involving segments I, IVa, VII, and VIII, with hepatomegaly. Thrombus involved the middle and right hepatic veins and extended contiguously through the intrahepatic IVC to the right atrium. Transthoracic echocardiography confirmed a freely mobile right atrial thrombus. Superiorly, the abscess breached the right hemidiaphragm through an approximately 27-mm defect and extended directly into the adjacent pleural/subpleural compartment and posterior right lower lobe, where focal consolidation with surrounding ground-glass opacity was present. Indirect hemagglutination assay for E. histolytica was positive at a titre of 1:250. The patient was resuscitated and treated with intravenous metronidazole and low-molecular-weight heparin. Because of the freely mobile intracardiac thrombus and the associated embolic risk, open surgical thrombectomy was performed together with operative drainage of the liver abscess. Characteristic anchovy-sauce material was obtained. The patient recovered uneventfully and was discharged on postoperative day 10. At 1 month, follow-up ultrasonography showed no significant residual hepatic or vascular abnormality.

Conclusion: ALA may rarely produce simultaneous hepatocaval-intracardiac thrombosis and transdiaphragmatic pleuropulmonary rupture. CT is essential for defining the full anatomic extent of disease, whereas echocardiography provides critical information about intracardiac thrombus mobility. A freely mobile right atrial thrombus should prompt urgent multidisciplinary assessment because surgical thrombectomy may be required in selected patients.

Keywords: amoebic liver abscess; Entamoeba histolytica; hepatic vein thrombosis; inferior vena cava thrombosis; right atrial thrombus; transdiaphragmatic rupture; pleuropulmonary extension; thrombectomy.

Introduction

Amoebiasis remains an important parasitic disease in tropical and subtropical regions, with Entamoeba histolytica responsible for invasive disease. Amoebic liver abscess is its most common extraintestinal manifestation and occurs predominantly in men. Alcohol use has also been repeatedly associated with ALA in endemic populations, although the mechanism is not fully understood. [1,2] Most uncomplicated abscesses respond well to nitroimidazole therapy, but a subset behaves aggressively, with rupture into adjacent compartments or development of vascular complications.

Thoracic spread is a recognized consequence of a superiorly located abscess. In the classic series by Ibarra-Perez, transdiaphragmatic rupture accounted for a substantial proportion of thoracic complications of ALA and could involve the pleural space, lung, airways, or pericardium.[3] With modern cross-sectional imaging, direct diaphragmatic breach and contiguous pleuropulmonary extension can usually be demonstrated, allowing these findings to be distinguished from reactive change or hematogenous pulmonary infection.

Vascular involvement represents a different and less familiar complication pathway. Hepatic and portal venous thrombosis may occur adjacent to an ALA, but extension into the IVC is uncommon, and propagation into the right

atrium has been reported only in isolated case reports. [4-9] The clinical importance of intracardiac extension lies in its potential for pulmonary embolization, tricuspid inflow obstruction, and hemodynamic compromise, particularly when the thrombus is mobile. Because of the rarity of this presentation, no standardized treatment algorithm exists; reported management ranges from anti-amoebic therapy and anticoagulation to thrombolysis and surgical thrombectomy. [5-9]

We describe an 18-year-old man with a large ALA complicated by two simultaneous routes of extrahepatic spread: contiguous thrombosis of the middle and right hepatic veins with propagation through the IVC into the right atrium, and direct transdiaphragmatic rupture into the right pleuropulmonary compartment. The case emphasizes the complementary roles of CT and echocardiography in defining complications that have immediate implications for management.

Case Presentation

An 18-year-old man presented with high-grade fever and chills associated with progressive abdominal distension. He had a history of chronic consumption of locally produced alcohol. On examination, the abdomen was distended and tender. Laboratory evaluation showed leukocytosis, consistent with an active inflammatory or infective process.

Initial abdominal ultrasonography demonstrated a hepatic abscess and an intraluminal thrombus within the IVC. In view of the suspected central venous complication, the patient underwent multiphasic contrast-enhanced CT for comprehensive assessment. No diagnostic needle aspiration or ultrasound-guided therapeutic aspiration was performed before CT.

CT showed hepatomegaly, with the liver measuring approximately 207 mm craniocaudally. A large, predominantly hypoattenuating, thick-walled collection measuring approximately 6.8 cm in maximum dimension involved segments I, IVa, VII, and VIII. The lesion showed marked peripheral wall enhancement, most conspicuous on the arterial phase, with nonenhancing central liquefactive contents. The adjacent hepatic parenchyma demonstrated heterogeneous arterial-phase hyperenhancement consistent with transient hepatic attenuation differences related to perilesional inflammation and altered regional perfusion.

Thrombotic involvement of the hepatic venous outflow tract was extensive. Nonenhancing intraluminal thrombus was present in the middle and right hepatic veins and extended contiguously into the intrahepatic IVC, with cranial propagation through the cavoatrial junction into the right atrium. Given its continuity with the inflammatory process, the thrombus was considered infection-associated rather than bland thrombosis. Transthoracic two-dimensional echocardiography confirmed a freely mobile right atrial thrombus.

A second route of spread was seen superiorly. The abscess breached the right hemidiaphragm through an approximately 27-mm defect and extended directly into the adjacent pleural/subpleural compartment and posterior segment of the right lower lobe. The pulmonary component consisted of focal consolidation with surrounding ground-glass opacity. Because this abnormality was in direct anatomic continuity with the hepatic abscess through the diaphragmatic defect, it was interpreted as contiguous pleuropulmonary extension rather than a separate septic embolic lesion.

Serologic testing for E. histolytica by indirect hemagglutination assay was positive at a titre of 1:250, supporting the diagnosis of ALA in the appropriate clinical and imaging setting. The patient was resuscitated and started on intravenous metronidazole. Low-molecular-weight heparin anticoagulation was also initiated. Exact drug doses were not available in the records reviewed for this report.

Because of the extensive hepatocaval thrombosis and, in particular, the freely mobile right atrial component with its potential for pulmonary embolization, operative management was favored over initial percutaneous drainage. Open surgical thrombectomy was performed, together with operative drainage of the hepatic abscess. Drainage yielded thick brown material with the characteristic 'anchovy-sauce' appearance of ALA. Microbiological culture or histopathologic examination of the excised thrombus was not available.

The postoperative course was uncomplicated. The patient remained hemodynamically stable, improved clinically, and was discharged on postoperative day 10. At 1-month follow-up, abdominal ultrasonography showed no significant residual hepatic collection or vascular abnormality.

Discussion

This case represents the severe end of the clinical spectrum of ALA. Its main significance is not the presence of a large hepatic abscess alone, but the simultaneous occurrence of two distinct complications: central venous propagation to a freely mobile right atrial thrombus and direct transdiaphragmatic pleuropulmonary rupture. Either can be clinically important on its own; together they create a combined risk of sepsis, pulmonary embolization, respiratory compromise, and cardiovascular deterioration.

Venous thrombosis in ALA is likely multifactorial. A large abscess adjacent to a hepatic vein or the IVC can produce local venous compression and stasis, while intense perilesional inflammation may extend to the venous wall and promote endothelial injury and thrombophlebitis. In an MDCT study of 62 patients with ALA, Priyadarshi et al. identified venous thrombosis in 43 patients (69%). Portal venous thrombosis was present in 39, hepatic venous thrombosis in 37, and IVC thrombosis in only four.[4] These data suggest that local venous thrombosis is not unusual in complicated ALA, whereas central propagation into the IVC is distinctly less common.

The arterial-phase perfusion abnormality in our patient is also relevant. Segmental and perilesional perfusion changes have been described in association with venous thrombosis in ALA. [4] Although such hyperenhancement is nonspecific, it should prompt careful assessment of the portal and hepatic venous systems when seen adjacent to a large inflammatory lesion. In our patient, CT allowed the entire thrombotic pathway to be followed from the middle and right hepatic veins through the IVC to the right atrium.

Right atrial extension is the most clinically important vascular feature of this case. Previous reports have documented IVC thrombus extending into the right atrium and have emphasized the risk of pulmonary embolization.[5-9] Khan and Rauf described ALA with IVC and right atrial thrombus requiring surgical removal.[5] Rehman et al. reported a young man with hepatic vein and IVC thrombus extending into the right atrium who was successfully treated with metronidazole, drainage, anticoagulation, sternotomy, and thrombectomy.[6] Siddiqui et al. similarly reported a free-floating right atrial thrombus arising from IVC thrombosis in a patient with ALA, with a favorable outcome after thrombectomy and abscess drainage.[7] Taken together, these reports establish right atrial propagation as an exceptionally rare but recognized complication of ALA.

The dynamic information provided by echocardiography was crucial in our patient. CT demonstrated the extent of the thrombus, but echocardiography showed that the right atrial component was freely mobile. This finding substantially increased concern for embolization and influenced the decision to proceed with surgery. There is no universally accepted treatment algorithm for ALA-associated right atrial thrombosis because the condition is too rare for comparative trials. Published approaches have included anti-amoebic therapy with anticoagulation, thrombolysis in selected cases, and open thrombectomy. [5-9] Management therefore needs to be individualized, taking into account thrombus size and mobility, hemodynamic status, evidence of pulmonary embolism, bleeding risk, response to medical treatment, and available cardiothoracic expertise.

In the present case, open thrombectomy was chosen because of the large thrombus burden and the freely mobile right atrial component. Anticoagulation was used to address the thrombotic process, while metronidazole and operative drainage provided treatment of the underlying infection and source control. Because the excised thrombus was not subjected to microbiological culture or histopathologic examination, we use the term infection-associated thrombus rather than claiming microbiologically proven septic thrombosis. This is a limitation of the case, but the close anatomic continuity between the abscess, hepatic venous thrombosis, IVC thrombus, and right atrial extension strongly supports a common pathophysiologic process.

The thoracic complication followed a different route. Superior right-lobe abscesses can erode through the diaphragm and extend into the pleural cavity or lung. Ibarra-Perez reported that transdiaphragmatic rupture accounted for the majority of thoracic complications in a large historical series.[3] Contemporary reviews likewise

recognize pleural effusion or empyema, pulmonary consolidation, lung abscess, and hepatobronchial fistula as manifestations of thoracic extension of ALA. [2]

Distinguishing direct pleuropulmonary spread from septic pulmonary embolism was particularly important in this patient because the mobile right atrial thrombus independently created an embolic risk. The posterior right lower-lobe consolidation and surrounding ground-glass opacity were immediately contiguous with the liver abscess through a visible 27-mm diaphragmatic defect and adjacent subpleural extension, strongly supporting direct transdiaphragmatic spread. In contrast, multifocal peripheral nodules, wedge-shaped opacities, cavitation, or pulmonary arterial filling defects would have raised greater concern for embolic disease.

The case also demonstrates a practical imaging approach. Ultrasonography is an effective first-line examination for detecting ALA and may identify large-vessel thrombosis. Once IVC involvement is suspected, contrast-enhanced CT should be used to define the hepatic lesion, trace the hepatic veins and IVC to the cavoatrial junction, and assess the diaphragm, pleural space, and lung bases. Multiplanar reconstructions are particularly helpful for demonstrating venous continuity and a diaphragmatic breach. Echocardiography then provides complementary real-time assessment of any intracardiac component, especially its mobility and relation to the tricuspid valve.

Despite the extent of disease, the patient had a favorable outcome after timely multidisciplinary treatment. Following resuscitation, anti-amoebic therapy, anticoagulation, surgical thrombectomy, and operative abscess drainage, he recovered without reported perioperative complication and was discharged on postoperative day 10. At one month, ultrasonography showed no significant residual hepatic or vascular abnormality. The case illustrates that even extensive hepatocaval-intracardiac thrombosis with thoracic rupture can have a good outcome when the full complication pattern is recognized early and treated decisively.

Conclusion

Amoebic liver abscess can rarely cause simultaneous vascular and thoracic complications. In this patient, thrombus propagated from the middle and right hepatic veins through the intrahepatic IVC into the right atrium, where echocardiography demonstrated a freely mobile component, while the abscess simultaneously ruptured through the right hemidiaphragm with direct pleuropulmonary extension. Contrast-enhanced CT was central to defining both routes of spread, and echocardiography provided the dynamic information that guided urgent surgical management. When evaluating a complicated ALA, radiologists should deliberately assess the hepatic veins, IVC, cavoatrial junction, right atrium, diaphragm, pleural space, and adjacent lung. A mobile right atrial thrombus warrants immediate multidisciplinary evaluation because surgical thrombectomy may be lifesaving in selected patients.

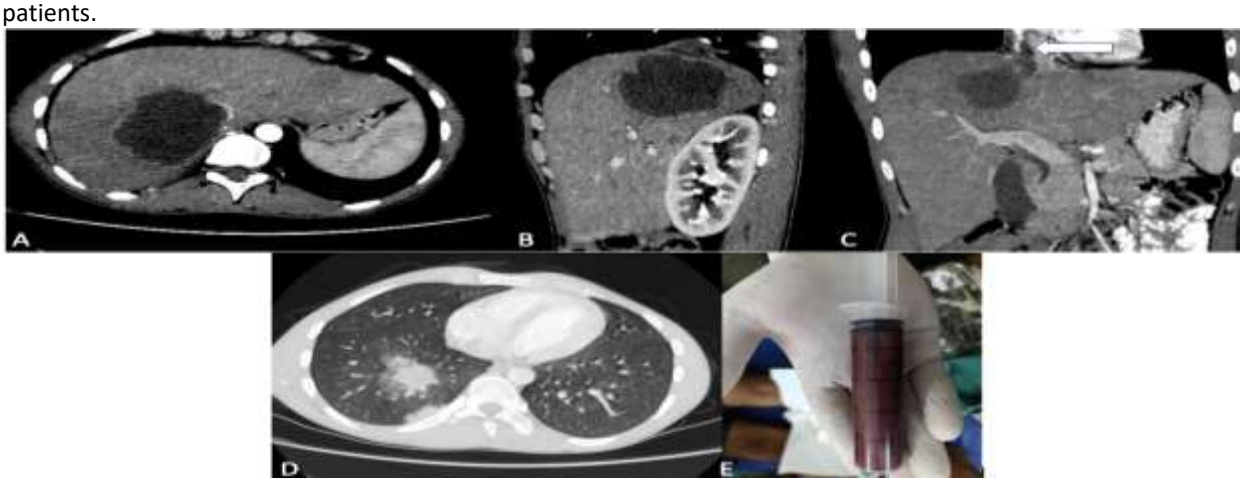

Figure 1 (A–E). Complicated amoebic liver abscess with vascular and thoracic extension. (A) Axial contrast-enhanced CT (CECT) image shows a large, thick-walled, peripherally enhancing abscess in the right hepatic lobe. (B) Sagittal CECT image demonstrates superior extension of the abscess toward the right hemidiaphragm. (C) Coronal CECT image shows hepatic venous and inferior vena caval thrombosis with cranial extension into the right atrium (arrow). (D) Axial lung-window image demonstrates right lower-lobe consolidation with surrounding ground-glass opacity, consistent with pleuropulmonary extension. (E) Percutaneous aspiration yielded thick, reddish-brown, "anchovy sauce–like" material characteristic of an amoebic liver abscess.

References

1. Stanley SL Jr. Amoebiasis. Lancet. 2003;361(9362):1025-1034. doi:10.1016/S0140-6736(03)12830-9.
2. Priyadarshi RN, Kumar R, Anand U. Amebic liver abscess: clinico-radiological findings and interventional management. World J Radiol. 2022;14(8):272-285. doi:10.4329/wjr.v14.i8.272.
3. Ibarra-Perez C. Thoracic complications of amebic abscess of the liver: report of 501 cases. Chest. 1981;79(6):672-677. doi:10.1378/chest.79.6.672.
4. Priyadarshi RN, Kumar P, Kumar R, Anand U, Shyama. Venous thrombosis and segmental hypoperfusion in amebic liver abscess: MDCT demonstration and its implications. Abdom Radiol (NY). 2020;45(3):652-660. doi:10.1007/s00261-020-02409-6.
5. Khan S, Rauf MA. Amoebic liver abscesses complicated by inferior vena cava and right atrium thrombus. Trop Doct. 2009;39(3):177-180. doi:10.1258/td.2008.080343.
6. Rehman Z, Alvi AR, Bibi S. Hepatic vein and inferior vena caval thrombus extending into the right atrium: a rare complication of amoebic liver abscess. J Coll Physicians Surg Pak. 2010;20(1):57-59. PMID:20141696.
7. Siddiqui M, Gupta A, Kazmi A, Chandra D, Grover V, Gupta V. Inferior vena caval and right atrial thrombus complicating amoebic liver abscess. Interact Cardiovasc Thorac Surg. 2013;17(5):872-874. doi:10.1093/icvts/ivt350.
8. Barcat D, Constans J, Jarnier P, Le Metayer P, Gosse P, Conri C. Thrombosis of the inferior vena cava and right atrium in amoebic abscess of the liver. Arch Mal Coeur Vaiss. 1998;91(2):271-273. PMID:9749257.
9. Sodhi KS, Ojili V, Sakhuja V, Khandelwal N. Hepatic and inferior vena caval thrombosis: vascular complication of amebic liver abscess. J Emerg Med. 2008;34(2):155-157. doi:10.1016/j.jemermed.2007.05.045.