

Amoebic liver abscess with sympathetic empyema thoracis: A Case Report And Review Of Literature

G Ogbole, A Adeyinka

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Abstract

We report a case of Amoebic liver abscess in a 42 year-old male alcoholic complicated by Empyema thoracis. He had ultrasound guided drainage of the abscess cavity with significant clinical improvement and abscess regression.

INTRODUCTION

Amoebiasis is a parasitic infection caused by *Entamoeba histolytica*, an organism that lives up to its formidable name by the invasion of colonic tissue leading to amoebic colitis and amoebic liver abscess (ALA). ALA is the most common extra intestinal manifestation of infection by *E histolytica*, and it is associated with significant morbidity and mortality¹. Empyema is usually a complication of pneumonia but may arise from adjacent infection involving the oropharynx, oesophagus, mediastinum, or a subdiaphragmatic tissue, like the liver²

Pleuro-pulmonary penetration of amoebic liver abscess occurs in 15%-20% cases. It develops when a right lobe abscess penetrates the diaphragm and produces an empyema or broncho-pleural fistula. Such involvement is associated with right lower chest pain, usually accompanied by persistent cough³

We present a case of amoebic liver abscess complicated with empyema thoracis, highlighting the role of imaging in diagnosis and treatment

CASE REPORT

A 42 year old male bricklayer, presented with a 2-week history of fever, associated with Jaundice and a 1-week history of abdominal pain. The fever was high grade, intermittent and associated with chills and rigors. Abdominal pain was dull in nature and generalized and began from the periumbilical region with no known relieving or aggravating factors.

The pain was unrelated to meals but produced a dragging sensation to the right side and was associated with loss of

appetite and early satiety.

He has a significant history of alcohol ingestion for greater than 10 years and multiple sexual partners. He is also known to abuse the use of analgesics.

Examination revealed a young man, who was mildly pale, dehydrated, icteric, febrile to touch with grade III finger clubbing but no peripheral lymph node enlargement or pedal edema. He had coarse crepitations in the right lower lung zone laterally and left lower zone posteriorly. His abdomen was full, moved with respiration with epigastric and right hypochondrial tenderness.

The liver was enlarged with a span of 20cm in the mid-clavicular line. It was hard, irregular and tender.

The spleen was mildly enlarged and measured 6cm below the coastal margin in the mid-clavicular line. There was mild ascites, (shifting dullness). Ascitic tap yielded serous fluid.

An initial assessment of chronic liver disease, possibly primary liver cell carcinoma (PLCC) was made with a background bronchopneumonia.

He was rehydrated with Intravenous (IV) fluids and commenced on IV Ciprotab, IV Methronidazole and Tramadol

However an abdominal ultrasound done 2 days later revealed the liver to be markedly enlarged with a span of 20.5cm and extending to the right iliac fossa and crossing the midline. It contained a fairly rounded, well defined, thick walled mass of mixed echogenicity in its postero-superior aspect,

measuring 11.6 x 9.6cm with an estimated volume of 550mls. It had shaggy inner margins with a sonolucency in its anterior portion. (Fig.1) No intrahepatic duct dilatation was seen. The gall bladder, spleen, pancreatic bed, para-aortic areas and both kidneys were all within normal limits. These features were consistent with a hepatic abscess.

Chest radiography showed a homogenous opacity in the right lower lung zone obliterating the ipsilateral hemidiaphragm and right cardiac margin with a well defined upper border giving a meniscus sign, the remaining lung fields were clear. There was however no mediastinal shift; features were in keeping with a right pleural effusion with possible underlying lung collapse. (Fig 2)

Liver function tests were slightly deranged with elevated bilirubin and liver enzymes but tumour makers (CEA1.7 μ g/l, AFP-1.9ku/l) were within normal limits. Patient's symptom subsided 5 days after commencement of antibiotics.

Aspiration of right pleural fluid collection yielded purulent fluid, which was cultured and grew *Kblesiella* spp. sensitive to Ciprofloxacin. An intercostal chest tube was inserted to ensure adequate drainage. This was later followed by an ultrasound guided percutaneous drainage of the hepatic abscess. Pre-aspiration estimated abscess volume was 650 ml. The procedure was done using an 18 G Echocoat needle. 570ml of 'anchovy sauce' chocolate colored viscid fluid was aspirated. Post aspiration residual volume was 186ml.

Patient felt a lot relieved and continued antibiotics. A repeat ultrasound scan 5 days later showed re-organization of the abscess cavity, with a thicker wall and some increase in volume estimated at 290mls (Fig. 3). A follow up scan two months latter, revealed a reduced volume estimated at 180ml with a clearer content (Fig. 4). The right pleural collection had also resolved with only minimal blunting of the costophrenic angle. He was however subsequently lost to follow up.

Figure 1

Figure 1a: Longitudinal ultrasound scan showing the liver containing a well defined thick walled, roundish area of mixed echogenicity in its postero-inferior aspect, with estimated volume of 550mls.

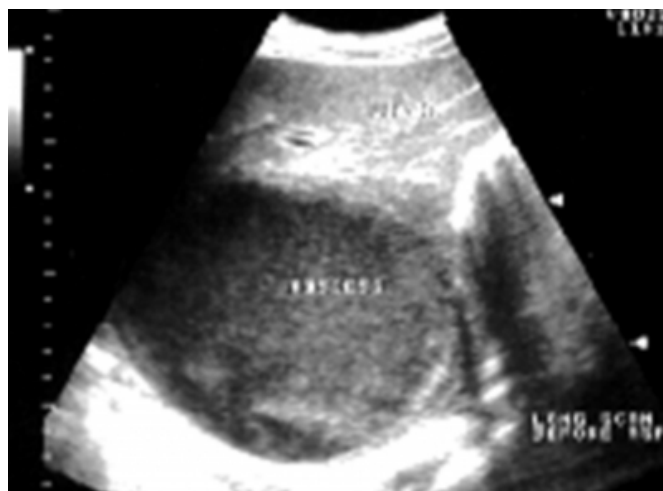


Figure 2

Figure 1b: Real time ultrasound scan during aspiration showing tip of echo coat needle within the abscess cavity (arrow).

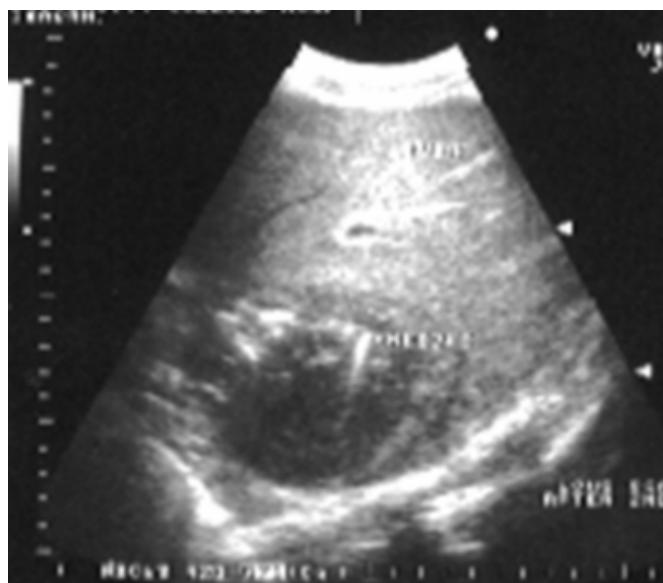


Figure 3

Figure 2: Frontal chest radiograph showing a homogenous opacity in the right lower lung zone obliterating the ipsilateral hemi-diaphragm and right cardiac margin with a well defined upper border and meniscus sign, there is no mediastinal shift.



Figure 4

Figure 3: Showing abscess cavity with irregular inner wall and central sonolucency 5days post aspiration, estimated volume -290mls.

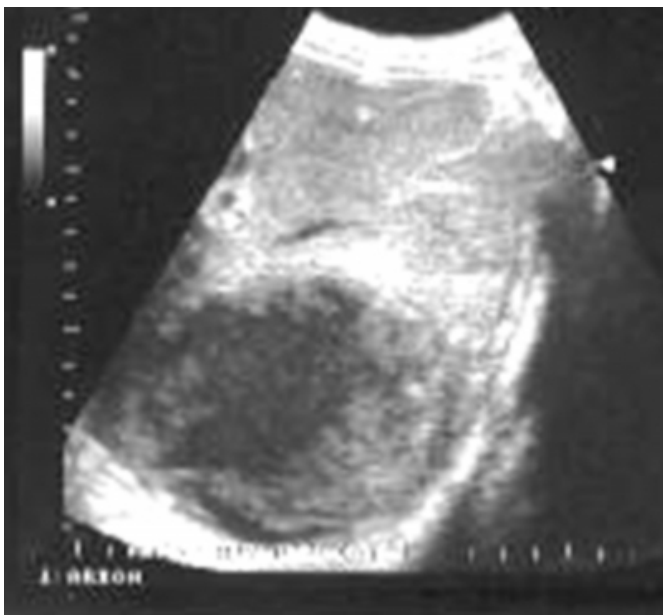


Figure 5

Figure 4: Showing abscess cavity with smooth inner wall and central sonolucency, 2months post aspiration. Estimated volume 180mls



DISCUSSION

Amoebiasis can be considered the most aggressive disease of the human intestine, being the second or third leading cause of death amongst the parasitic diseases, surpassed only by malaria and schistosomiasis. It is responsible in its invasive form for clinical syndromes, ranging from the classic dysentery of acute colitis to extra-intestinal disease, with emphasis on hepatic amoebiasis, unsuitably named amoebic liver abscess⁵

Amoebic liver abscess is an enigma as it has been observed in people with no evidence of previous amoebic colitis or history of tropical travel. The patient presented though living in the tropics, has no history suggestive of colitis. Nevertheless it may occur several years after colonic amoebiasis or after the patient had left the tropics.

In a series by Archampong and Clark, a third of autopsy cases had lesions in the liver⁷. It is assumed that the infection reached the liver through the portal tract. Unlike in portal pyaemia, the abscess is most often unilocular but coalescence of multicystic abscesses is a possibility and most frequently is located in the right lobe, situated contiguously with the liver capsule.

Variable in size, in some cases it may occupy more than 80% of the whole liver surface This may be explained by the larger volume of the right lobe, which receives most of the venous drainage from the right colon, a segment of the

bowel frequently affected by intestinal amebiasis ⁸. The amoebic lesions of the left lobe are less common, and multiple abscesses may also occur in advanced cases of amoebiasis.

ALA develops in less than 1% of patients infected with *E. histolytica*, but this still represents a large number of patients ⁷. Amoebic liver abscess occurs most commonly in the age group of 20 to 45 years consistent with the present case. It has also been noted infrequently at the extremes of age; men are disproportionately affected, with reported male to female ratio of approximately 10:1 ⁸.

ALA may present as an acute process or as a chronic indolent disease. Most patients present with an acute illness and duration of symptoms less than 2 weeks as in this case where the main presenting features were abdominal pain, fever, and anorexia. Diffuse abdominal pain, pleuritic chest pain, and radiation of right upper quadrant pain to the right shoulder are not uncommon. Epigastric pain is commonly seen in left lobe abscesses. Fever is of moderate degree in most instances, while high fever with chills is suggestive of secondary bacterial infection as seen in this case. Cough with or without expectoration and pleuritic chest pain is also seen in ALA. ⁹. During the course of illness one-third of patients may develop clinical jaundice. Severe icterus is usually due to a large abscess or multiple abscesses, or to an abscess situated at the porta hepatis ⁹.

Jaundice raises diagnostic problems and brings in the possibilities of intra-hepatic obstruction or viral hepatitis. Similar findings were evident in our patient. Tender hepatomegaly is detected in up to 80% of patients. The liver surface is generally smooth. Upper abdominal guarding and rigidity is seen in a minority of cases with features of generalised peritonitis. Ascites developing in a patient with ALA suggests development or presence of inferior vena cava obstruction, and cough with copious expectoration suggests rupture into the communication with the right lower lobe bronchus. ⁹

On plain chest radiography, an elevated right hemidiaphragm with associated right "sympathetic" pleural fluid collection may raise the first suspicion ¹⁰ as in the case presented. The diagnosis is now made most readily and easily by ultrasonography of the liver ¹¹. The classic appearance is a non-homogeneous, hypoechoic, round or oval mass with well defined borders. However, radioisotope scan, CT and MR imaging are also diagnostic ¹¹. The findings may however be indistinguishable from those of a

hydatid cyst or a tumour. Leakage of the abscess may occur into the pleural cavity, with empyema thoracis. Pleuropulmonary amoebiasis is usually caused by a ruptured right lobe liver abscess. No rupture was however demonstrated in this case.

Approximately 10% of patients with ALA develop Pleuropulmonary Amoebiasis. A hepatobronchial fistula is an unusual problem characterized by the expectoration of sputum resembling anchovy paste. The trophozoites of *E. histolytica* may be found in the sputum sample. ¹²

Intra-abdominal extension following perforation into the peritoneal cavity is usually associated with shock and generalised peritonitis and may occur in up to 7% of cases.

Apart from rupturing into the pleural cavity, peritoneal cavity and lung, the pericardium, the skin of the thoraco-abdominal wall, the hepatic flexure of colon or even the duodenum and the stomach are possible viscera that may be affected. It may present as a "collar-stud" or "hour-glass" abscess below and above the diaphragm. Complete resolution of an amoebic liver abscess may take up to two years ¹³.

Occasionally, percutaneous diagnostic needle aspiration may be needed to differentiate between amoebic and pyogenic liver abscess. The absence of serum antibodies to *E. histolytica* after 1 week of symptoms is strong evidence against the diagnosis of invasive amoebiasis of the colon or liver.

Serum antibodies to amoebae are detected in 85-95% of all patients who present with invasive amoebiasis or liver abscess. However, as antibodies persist for many years, ELISA or IHA cannot differentiate acute from remote infection in areas of high endemicity ⁴.

Amoebicides effective in both tissues and the intestinal lumen include; nitroimidazole derivatives-metronidazole, tinidazole, and ornidazole. They are the drugs of choice in invasive amoebiasis. Oral or intravenous metronidazole or tinidazole also leads to rapid clinical improvement of amoebic liver abscess ¹³.

Open surgical drainage is rarely indicated and may be required in the setting of a large abscess with a poor yield on needle aspiration or clinical deterioration despite attempted needle aspiration, and in complicated ALA.

Surgical mortality is, however, very high. Hence, it is only

used when the cavity has ruptured into adjacent viscera or peritoneum. After clinical cure, patients show few symptoms and sonographic follow-up demonstrates evidence of persistent hypoechoic lesion¹³. The mean time for disappearance of the sonographic abnormality is 6-9 months. Relapses are very uncommon and the sonographic abnormality does not warrant continued therapy¹⁴.

The pattern of resolution that have been seen on sonographic follow-up include: type I, where complete disappearance of the cavity occurs within 3 months (29.8%); type II, where a rapid reduction till 25% of the original cavity size and then a delayed resolution occurs (5.9%)¹⁴.

CORRESPONDENCE TO

Godwin Ogbole Email: gogbole@yahoo.com Department of Radiology University College Hospital, Ibadan, Nigeria

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Author Information

Godwin I. Ogbale, FWACS

Department of Radiology, University College Hospital

Abiodun O. Adeyinka, FWACS

Department of Radiology, University College Hospital