

CASE REPORT

Management of a ruptured amoebic liver abscess at Edward Francis Small Teaching Hospital in The Gambia: A Case series report

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Abstract

Introduction: Amoebic liver abscess caused by an anaerobic parasitic amoeba, *Entamoeba histolytica*, causes mainly intestinal infection in man, but it can also cause extra-intestinal infections, the commonest of which is amoebic liver abscess.

Its treatment is mainly non-surgical, as it responds well to medications. However, few cases present as surgical emergencies, when the abscess ruptures, and has to be managed surgically.

Case presentation: We present two cases of ruptured amoebic liver abscess, a 34yrs old woman and an 18yrs old man, both of which were managed surgically. For the first one, the diagnosis was suspected before surgery, but the second one presented as an acute abdomen.

Conclusion: Amoebic liver abscess can be complicated by rupture into the peritoneal cavity resulting in peritonitis, however the outcome can be good, even in resource poor settings.

Keywords: Liver abscess, Amoebic, Ruptured, Gambia

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Introduction

Amoebic liver abscess (ALA) is caused by *Entamoeba histolytica*, an anaerobic parasitic amoeba, which causes mainly amoebic dysentery in man, an intestinal infection. It can also cause extra-intestinal infections, the commonest of which is amoebic liver abscess.

Infection results from ingestion of food or drinking water contaminated by cysts of *E. histolytica*, which enter the intestines and cause the symptomatic infection. It can also enter the mesenteric venules to the portal circulation then to the liver, where it can cause single or multiple abscesses.

ALA is reported to be common in tropical countries¹ and is prevalent mainly among the lower socioeconomic class living in unhygienic conditions.²

The common complications of ALA include rupture into pleural cavity and peritoneal cavity; rarely, vascular complications are seen in the form of thrombosis or compression resulting in either hepatic venous outflow obstruction or inferior vena cava (IVC) obstruction³

Case Descriptions:

Case 1:

A case of a 34-year-old female who was referred on account of a two-week history of abdominal pain and distension, and a week's history of yellowness of the eyes.

The abdominal pain was said to be of gradual onset, located in the epigastric region, initially dull in nature, with no known relieving or aggravating factors. However, after a few days of the abdominal

pain the patient started having progressive abdominal distention with low grade fever and several episodes of vomiting.

About a week prior to presentation, she started having yellowing of her eyes, but no associated history of body itching or pale stool. There was also bilateral lower limb swelling.

On examination, she was found to be acutely ill- looking, in painful distress, very pale, deeply jaundiced and with bilateral pitting pedal oedema, and tachycardic.

Her abdomen was mildly distended, moved with respiration, and demonstrated generalized tenderness with guarding. Bowels sounds were present, but hypoactive.

Digital rectal examination revealed an empty rectum. The rest of the systems were unremarkable.

She had an abdominal ultrasound scan done from the referring center, which revealed a hepatomegaly and a right lobe mass with a collection, suggestive of an abscess, and a large collection of free fluid in the abdomen and pelvis with strands and septae suggestive of peritonitis.

An emergency exploratory laparotomy was performed after adequate resuscitation, and findings were as follows: a huge cavity (10 x15cm) in the right lobe of the liver involving segments 5,6,7 & 8, filled with necrotic liver tissues. There was about 3 liters of chocolatey-like fluid (anchovy sauce) in the peritoneal cavity. The bowels and the rest of the intra-abdominal organs appeared normal.

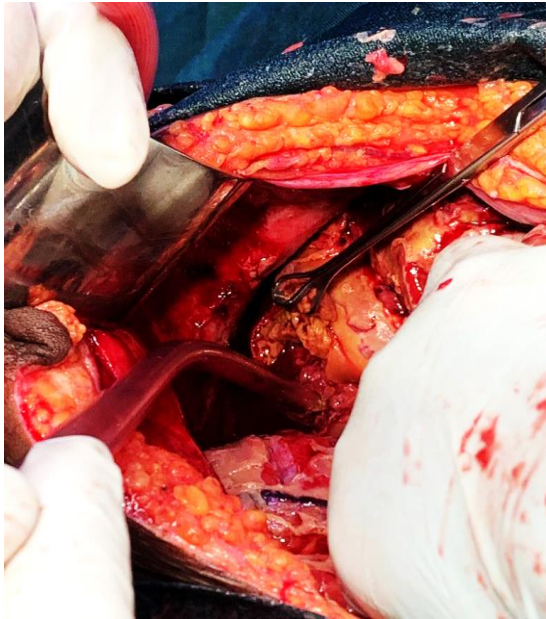


Fig 1: Intra-operative findings of cavity in liver with chocolatey like fluid in the cavity

Toileting of the right lobe of the liver was done with 2L of warm normal saline. The cavity was irrigated with 200mls (1g) metronidazole, and the abdomen was closed using the mass closure technique, leaving an abdominal drain in situ in the Morisson's pouch. The skin was closed with interrupted mattress sutures using 2/0 nylon.

The post-operative period was unremarkable. She was on nil by mouth for 72hrs, during which time she received 3L of intravenous fluids daily (2L 5% dextrose and 1L Ringer's lactate) and 500mg intravenous metronidazole six hourly and 400mg intravenous ciprofloxacin twelve hourly. The fluids were reduced as she was started on graduated feeds from post-operative day 4. She made good recovery and the drain was removed on post-operative day 9. She was discharged after ten days, on oral Metronidazole 800mg three times daily for 5 days, and followed up at the Surgical Outpatient Department (SOPD) after a week.

She later had serial ultrasound guided aspirations for a residual abscess in the liver, by the Gastroenterologists till she was finally discharged, after resolution of the abscess.

CASE 2:

An 18-year-old male presented with a 3-week history of gradual onset abdominal pain which was non-radiating and not relieved by pain medication. It was associated with fever and intermittent non-bilious vomiting, (2 episodes per day).

He also had a 2-week history of non-bloody diarrhoea, which stopped about two weeks prior to presentation. A week prior to presentation, he noticed a gradual distension of his abdomen, associated with severe pain on the right side of his abdomen.

On examination, he was acutely ill looking, mildly pale, in painful distress, warm to touch, dehydrated, and was tachycardic.

Abdomen was mildly distended with tenderness on the right upper quadrant. Bowel sounds were diminished. Rectal examination revealed an empty rectum.

Other systems or regions were unremarkable.

Patient was resuscitated and taken for emergency exploratory laparotomy, and findings were as follows: about 500mls of chocolate-like fluid (anchovy sauce) in the peritoneal cavity, with a distorted right lobe of the liver, mainly segments 5 and 6. The nearby small bowel and colon were attached to the affected lobe of the liver, but the rest of the intra-abdominal viscera were normal.



Fig 2: Intra-operative findings of distorted right lobe of liver

The abdominal cavity, including the distorted right lobe of the liver, was irrigated with 2L of warm normal saline, and a tube drain left in situ, in the Morisson's pouch, and the abdomen was closed using the mass closure technique, and skin closed using interrupted 2/0 nylon stitches.

Post-operatively, he was continued on intravenous antibiotics, ceftriaxone, 1g twelve hourly and metronidazole 500mg six hourly. Intravenous fluids were continued till post-operative day 2, after which he was started on graduated feeding. The abdominal drain was removed on post-operative day 7 and he was eventually discharged on post-operative day 8, on oral metronidazole 800mg three times daily for 5 days, to be followed up at the surgical out-patient department (SOPD) weekly till finally discharged.

Discussion:

Incidence of Amoebic liver abscess in The Gambia is not well documented in

published literature. However, about 5 decades ago, a country-wide stool survey of The Gambia showed the prevalence of *Entamoeba histolytica* cysts to range from 13.7% up-country in the dry season to 52.3% near the coast⁴. Bittaye et al conducted a study on liver abscess in general, including both amoebic and pyogenic liver abscesses, at the main referral teaching hospital in The Gambia, and reported 35 cases in three years⁵.

The majority of infection by *E. histolytica* are asymptomatic, only 10 – 20% progress to develop symptomatic infection⁶, and ALA develops in < 1% of patients infested with *E. histolytica*³

Amoebic liver abscesses are more likely to be solitary lesions than multiple lesions, and they are more commonly found in the right lobe of the liver than in the left lobe⁷. This could be because the right lobe has more blood supply¹

There is no consensus on how a ruptured amoebic liver abscess associated with diffuse amoebic peritonitis can be optimally managed.⁸

Literature shows that conservative management for amoebic liver abscess is recommended, even for abscesses that have ruptured. A study done in Pakistan for example revealed a high mortality for ruptured amoebic liver abscesses that had surgical intervention compared to those managed conservatively.¹⁰

The mortality rate is low when the abscess is limited to liver but increases as the abscess gets complicated.³

Some studies have reported higher mortality rates of between 20 – 50% following surgical therapy in patients with ALA that ruptured into the peritoneum compared to between 0–5% following

conservative medical therapy, consisting of ultrasound-guided PCD combined with antimicrobial agents⁸

Most cases of extra-intestinal abscess occur without concurrent intestinal infection, resulting in lower sensitivity of stool studies for the diagnosis of amebic liver abscess⁶. Stool samples for both cases were tested for cysts of *E. histolytica*, but both turned out negative. This is consistent with the study done by Bittaye et al, where the stool samples of all the 35 patients with liver abscesses tested negative for ova or parasites⁵.

ALA may mimic many different conditions like acute cholecystitis, perforated peptic ulcer, acute hepatitis, cirrhosis, hydatid cysts, pancreatic pseudocysts, pneumonia, acute pleurisy with effusion, empyema, chronic lung disease, malignancy, tuberculosis and pyrexia of unknown origin.² In case 2, unlike case 1, ALA was not suspected prior to surgery. The diagnosis was only made when the exploratory laparotomy was performed with findings as stated above.

Occasionally, diagnostic or therapeutic aspiration is required. Aspiration of the amebic liver abscess reveals "anchovy paste," chocolate-colored fluid consisting of necrotic hepatocytes⁶.

Drainage is typically reserved for those that do not show prompt clinical response to antiparasitic therapy by about 72 hours or those at high risk for impending abscess rupture, typically defined by a cavity with a diameter >5–10 cm

In a study by Priyadarshi et al. where 32 of 117 (27.3%) patients with ruptured ALA had diffuse intraperitoneal spread, all patients were managed with Percutaneous

drainage along with antimicrobial agents. The success rate was 97%, with a mortality rate of 3%.⁸ In another study by Monga et al., the mortality rate following surgical therapy for ruptured ALA was 33%.⁸ A retrospective study done in Pakistan revealed that mortality in cases of ruptured ALA treated surgically was 37.5% compared to 5% in those treated conservatively.¹⁰ Again, in another retrospective study done in India, mortality in those treated conservatively was zero compared to 40% in those treated surgically.¹¹

For our case 1, the diagnosis was suspected prior to surgery, because she came with an ultrasound report of a suspected liver abscess, but because she presented as an emergency with an acute abdomen, we decided to treat surgically, and had a good outcome.

Despite improvements in surgical techniques, anesthesia, critical care, and wide availability of antimicrobial agents, the mortality rate of surgical treatment is still reported to be quite high⁸.

Conclusion:

Even in settings where resources are not adequate, the outcome of surgical management of ruptured liver abscess can be good, with appropriate pre-operative resuscitation and management, as seen in our two cases managed surgically. Surgical intervention should not be delayed when such a patient presents with an acute abdomen.

This might not reflect the true outcome of such cases, as the number is quite small. Further observation is required with larger numbers.

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