

COMPLICATION AMOEBIC LIVER ABSCESS STILL A SIGNIFICANT HEALTH PROBLEM IN THE TROPICS: 3 - CLINICAL CASE REPORTS

E. J. PETERS, A. U. ETIUMA, D. U. EDUWEM and V. O. ANSA

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ABSTRACT

Amoebic liver abscess still poses a serious clinical problem in tropical countries. Here we describe three complicated cases to illustrate the magnitude this disease condition could assume in the tropics. Limited access to health facilities as well as poverty and ignorance result in patients presenting late, often with complications, which compounds the problem of differential diagnoses. The need for improved sanitary conditions, and qualitative health care delivery in the tropics to positively alter the epidemiology of this disease becomes more imperative.

KEY WORDS: Amoebic liver abscess, complications, tropics.

INTRODUCTION

Human Amoebiasis is caused by *Entamoeba histolytica*. It affects mainly the gastrointestinal tract but may extend to the liver and other organs. Though Amoebiasis has been considered essentially a tropical disease, there is significant interterritory transmission because of increased global movement. It is probably the most widely distributed of protozoal diseases and in man only 10% of intestinal infections are symptomatic (Huston, et al 1999) Severe infection occurs in pregnant women, very young children, the malnourished, persons on prolonged steroids and the immunocompromised.

Amoebic liver abscess is the most common extra-intestinal form of amoebiasis. (Edington et al, 1981, Greenstein et al 1985) it may occur without intestinal symptoms. The liver abscess may in turn give rise to pericardial, pleuropulmonary, cerebral, genitourinary, free peritoneal, or cutaneous disease which may occur many years after exposure in up to 70% of cases. (Huston et al 1999).

Clinically, the symptoms and signs of amoebic liver abscess are often non-specific. Findings of trophozoites of *Entamoeba histolytica* in Stool is helpful but its absence does not rule

out amoebiasis. Serological tests and other specific diagnostic tests are rarely available in most tropical hospitals. Poverty and ignorance result in late presentation and often makes early diagnosis difficult. The resultant large abscess cavity and other rare complications make diagnosis difficult especially in these parts of the world where primary liver cell carcinoma and tuberculosis are common. We described three recently encountered cases to highlight some of the complications that may develop in this condition.

CASE REPORTS

CASE 1

Mr. U. Y, a 30 year-old unemployed male presented with a history of abdominal pain, epigastric swelling and general body weakness of one month's duration. There was associated low-grade fever, mild jaundice and loss of appetite. He had lost weight and was passing loose stools, which was occasionally, blood stained. There was no vomiting. He takes local gin 2-3 shot's daily (20mls) and he had been drinking for the past 25 years. There was no history of tobacco consumption. The source of drinking water was stream and he used pit toilet.

✉ E. J. PETERS, Department of Medicine, University of Calabar Teaching Hospital, Calabar, Calabar - Nigeria
A. U. ETIUMA, Department of Surgery, University of Calabar Teaching Hospital Calabar, Calabar - Nigeria
D. U. EDUWEM, Department of Radiology, University of Calabar, Calabar - Nigeria
V. O. ANSA, Department of Medicine, University of Calabar, Calabar - Nigeria

Examination revealed a chronically ill looking young man, markedly wasted, pale mildly jaundiced, with grade 2 finger clubbing. There was an epigastric mass left of the midline measuring 2x3cm, tender, and fluctuant (fig 1). The Spleen and kidneys were not palpable. Bowel sounds were normal. Investigations revealed a normal chest X-ray and an abdominal ultrasound scan demonstrated a large circumscribed echopoenic lesion in the antero-superior part of the (Lt) lobe of the liver measuring (8.6 x 7.2 x 5.2cm) extending anteriorly through a fistulating tract to a subcutaneous position in the epigastrium (fig 2). The surrounding hepatic

parenchyma showed signs of diffuse inflammatory changes. The kidneys, gall bladder, and spleen were normal. His liver function test showed a total bilirubin of 10.0. Umol/l, conjugated bilirubin of 5.0 umol/l, aspartate transaminases-64 iu/, Alanine transaminases 54 iu/l and alkaline phosphates of 10 iu/l. The full blood count result showed a pcv of 27%, white count of 6.0×10^9 /L, with anisocytosis and microcytosis, stool microscopy revealed cysts of *Entamoeba histolytica*.

The aspirate from the epigastric swelling yielded chocolate-coloured pus. The patient was admitted in hospital and fully resuscitated with intravenous fluid. He was transfused initially with 2 pints of blood and the abscess was later drained by aspiration under ultrasonic guidance. He also received intravenous metradinazole 500mg 8hrly for 10days and intravenous Ampiclox 500mg 6hrly for 7 days.

He responded to treatment given and was discharged home after 14 days. Post discharge monitoring of cavity by ultrasound scan was carried out regularly and revealed no further abnormality.

CASE 2

A 51 years old male retired teacher, presented at the University of Calabar Teaching Hospital, Calabar with a 2-month history of dull aching rightsided abdominal pain and a one-week history of productive cough. No preceding history of diarrhea, fever or jaundice. He is a known hypertensive for 15years and drinks water from stream and used pit toilet.

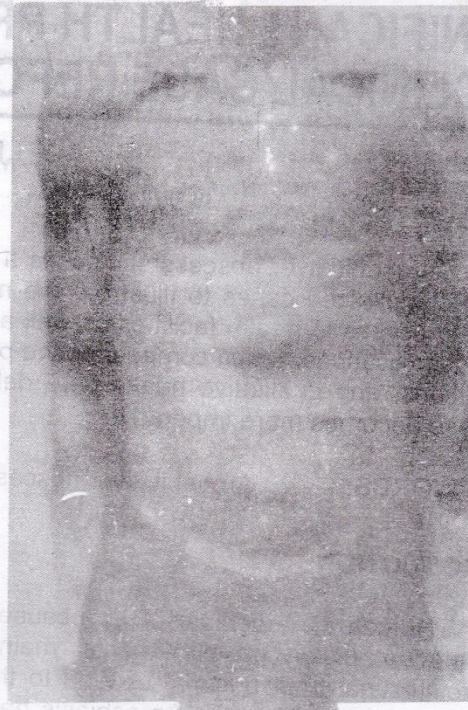


Figure 1:



Figure 2:

Examination showed an otherwise stable man with mild pallor and massive hepatomegaly. A Plain chest radiograph showed gross elevation of the right hemi-diaphragm with normal lung fields. An Abdominal ultrasound scan (USS) showed three circumscribed cystic cavities within the liver. The largest of these cystic masses was located in the inferior aspect of the right lobe, and it measured 13.2 x 9.2m. Other intra abdominal organs were normal. There was no ascities. Stool microscopy revealed cysts of *Entamoeba histolytica* and the Liver function test showed marginal increases in the transminases only. A full blood count revealed mild anemia (PCV 26%), total white count 8.2×10^9 /L, with a normal differential count. A diagnosis of multiple Amoebic liver abscess was made and the patient was commenced on intravenous Metrodinazole 500mg 8hourly x 72hrs then tablets 800mg 8hourly x 10/7 and tablets chloroquine i/bd. X 10/7.

Patient's condition improved. A repeat USS showed only minimal reduction in the size of the cavities. He was however discharged home on drugs after 18 days of admission after showing



Figure 3:

signs of full recovery

He represented 3 weeks later through casualty to surgical team with a 5-day history of cough productive of copious purulent sputum. There was mild fever, chest pain and difficulty in breathing. Patient also had 3 days history of persistently worsening abdominal pain and distension.

A chronically ill patient was found with severe dyspnoea (RR: 36 cycles/min), afebrile, pale ++ with a tinge of jaundice. He had grade II finger clubbing, bilateral pitting pedal edema up to the ankles and the pulse rate was 96-beats/min, regular and good volume. Blood pressure was 180/130mmHg as patient was previously uncontrolled hypertensive. Clinical and radiological examination of the chest showed a massive right pleural effusion. (Fig 3) Abdominal examination revealed generalized moderate tenderness, and guarding. There was tender hepatomegaly of 18cm below the right costal margin with a span of 24 cm, and ascites. Other systems were normal except muscle wasting in the limbs.

A diagnosis of amoebic liver abscess with pleuropulmonary and abdominal rupture in a hypertensive was made. A full blood count result, showed Pcv: 19%, white count 11.3×10^9 /L, Neutrophil 81%, lymphocyte 19%, Erythrocyte sedimentation rate 145 mm/hr (Westergren). Sputum and abdominal fluid aspirate culture yielded no growth after 48hours. HIV screening, liver function test, clotting profile showed no significant abnormalities. It was not possible to repeat the abdominal USS.

The patient was managed with 2 pints of fresh blood transfusion, and a pre-exploratory chest tube insertion under local anesthesia drained 2 liters of chocolate coloured non-foul smelling pus from the right pleural cavity. Exploratory laparotomy was then carried out under general anesthesia and the findings included a huge Lt. Hepatic abscess adjoining the bare area, a transdiaphragmatic rupture on the right and an intrabdominal rupture via a rent in the hepatic capsule. Five liters of pus was drained from the abdomen. The abscess cavities were drained and cleaned out with gauze, and peritoneal lavage with warm saline was carried out. Thorough consecutive treatment with

Metronidazole tablets 400mg 8 hourly continued for 14 days and Ampiclox 500mg x 6 hourly for 7 days led to good recovery and hepatic cavity monitored by ultrasound 3 months later showed no abnormality.

CASE 3

A 30-year-old lady was referred from a private clinic on account of Rt.- Sided lower chest wall pain which "extended to the Rt. Loin with inability to lie down on that side as well as a demonstrable progressive liver enlargement.

Earlier, she presented with complaints of fever and rigors for three weeks and Rt. Hypochondrial pain radiating to the umbilicus. She was a known peptic ulcer patient, diagnosed, one year earlier.

On examination, she was acutely ill with marked pallor, and mild jaundice. She was moderately dehydrated and a pulse rate of 130/min regular with low volume. Her temperature was 38°C, blood pressure 90/50 mmHg, and there was moderate respiratory distress as shown by a respiratory rate of 38 cycles/min.

Her Abdomen was moderately distended and tender. Tenderness was maximum in the right hypochondrium and right lumbar region with generalized rebound tenderness. There was tender hepatomegaly 12cm with a liver span of 20cm. The Spleen and kidneys were not palpably enlarged and bowel sounds were normal.

Digital rectal examination and proctoscopy showed no abnormality except presence of altered blood.

Investigations done revealed a full blood count with Hemoglobin of 9.0g/L, white count of $5 \times 10^9/L$, normal differential, and erythrocyte sedimentation rate was 2mm/hr (Westergren). Abdominal ultrasound scan showed a solitary abscess cavity in the Rt. Lobe of the liver measuring 72 x 48mm.

Stool analysis showed formed stool without blood and mucus and Cysts of *E. histolytica* were seen. HIV Screening was negative and hepatitis B surface antigen urea, electrolytes and liver function test were normal.

The patient was resuscitated with intravenous fluids blood transfusion (2 pints), intravenous Metronidazole, Ampiclox, intravenous

Ranitidine, Nil by mouth and urethral catheterization.

An emergency abdominal exploration was done and the findings included a massively enlarged liver with a large right lobe abscess containing chocolate coloured pus; Straw coloured ascites and perisplenitis. All other organs were grossly normal.

An Intra operative diagnosis of giant Amoebic liver abscess was made Drainage and a thorough peritoneal lavage was carried out and the patients: Postoperative recovery was uneventful and he was discharged home 12 days after surgery. Follow up abdominal ultrasound 6 months later confirmed a normal liver without any residual cavity.

DISCUSSION

These three cases amply illustrate common patterns of complicated amoebic liver disease seen in the tropics. They include cutaneous, pleuropulmonary and peritoneal complications. These complications often result from primary lesions in the liver; although rarely secondary lesions from other tissues such as lungs, brain, genital organs and skin have been reported. (Pleorde 1991, Greenstein et al 1985)

Although these complicated patterns of amoebic liver abscesses are frequently seen in the tropical and subtropical regions, they nevertheless, have a worldwide distribution (Herman P. and Costal MLV 2001). The high incidence in these countries could easily be attributable to the poor sanitary environment prevalent in these countries. Mcleod in Durban, South African demonstrated a high prevalence of this condition in areas with poor sanitary conditions. (Mcleod et al 1966). In 10% of patients the abscess may rupture into the peritoneal cavity, pleural cavity, and pericardium or subcutaneous tissue Rarely, it can rupture into various hollow abdominal organs like stomach and colon; resulting in severe hemorrhage.

In over 80% of patients with an insidious onset, half of those presenting with acute manifestation have a single abscess. (Peters et al 1981). Most commonly, this is localized in the posterior portion of the right lobe of the liver.

because this lobe receives most of the blood draining the right colon.

The right pleural cavity and lungs are involved by direct extension of the abscess from the liver in about 20% of patients with liver abscess (Thompson 1985). Occasionally it perforates directly into the bronchus resulting in expectoration of large amounts of exudates as exemplified by our second case in which the patient presented with cough productive of copious purulent sputum. A massive pleural effusion may result if it ruptures into the pleural cavity. Peritoneal complications of amoebic liver abscess often results from the rupture of the abscess into the peritoneal cavity or perforation of a colonic ulcer. This is a fairly common complication as it accounts for about 20-30% of complicated cases (Vakil et al 1970). The condition is often fatal if not timely managed as the patient will develop generalized peritonitis and shock, as seen in our third patient.

The cutaneous complication of amoebic liver abscess results from a direct extension of the abscess through a fistulous tract to the overlying subcutaneous tissue.

The most dangerous complication of amoebic abscess, is the direct extension of the abscess to the pericardium. Fortunately, it is uncommon in this environment in our experience. This condition is often associated with abscess in the left lobe of the liver.

Amoebic liver abscess may occur after a long latent period; in which case, a stimulus is needed to trigger the multiplication of the entamoeba.

Immune-depressed conditions and prolonged steroid treatment have been implicated in this regard (Kanami et al 1969; Sacua 1967; Hamide et al 2002, Joshi et al 2002) However, none of the cases we reported was immunocompromised. Although jaundice is uncommon in Amoebic disease, it can be seen and when present implies a grave prognosis, (Pleorde 1991, Lewis et al 1969). All three cases had jaundice and this probably reflects the severity of their illness. The prognosis in uncomplicated amoebic abscess is excellent with appropriate medical therapy; the mortality being as low as 1% in some series (Scharschmidt 1985, Adams et al 1972; Ender

2001). However, with extension or rupture into the pleural, pericardial or peritoneal spaces, the mortality increases sharply to 20% (Thompson 1985, Vakil et al 1970, Greenstein et al 1985).

In conclusion, Amoebic liver disease is still a common problem in the tropics. The condition may assume a complex picture. Diagnosis may become a problem owing to absence of basic scanning facilities. Clinician should have a high index of suspicion and the importance of public and personal hygiene is emphasized.

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