

## Case Report

### Case report: Huge amoebic liver abscesses in both lobes

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#### Summary

We describe the case of a patient who returned to China from Africa and underwent emergency open surgical drainage with evacuation of 600 mL of anchovy sauce-like fluid from hepatic lesions. Computed tomography scans and surgical findings indicated abscesses in both hemilivers and communication between them. Bacteriological investigation of the fluid yielded negative results, but DNA assay of the pus detected 18S rRNA genes of *Entamoeba histolytica*. Serum anti-amoebic antibodies were detected using an indirect fluorescent-antibody test. Consequently, anti-amoebic drugs were administered and drainage was performed, leading to improvement in the patient's condition. As is evident from this case, an amoebic liver abscess in the left hepatic lobe is rare but treatable.

**Keywords:** *Entamoeba histolytica*, amoebiasis, amoebic liver abscess

#### 1. Introduction

*Entamoeba histolytica* is a causative agent of amoebic dysentery and extra-intestinal abscesses. It is prevalent in developing countries where its fecal-oral spread is difficult to control. *E. histolytica* is responsible for approximately 50 million cases of invasive amoebiasis annually with a mortality of 40,000 to 110,000 (1). Invasive amoebiasis is a major health problem worldwide and is second to malaria among protozoan causes of death (2).

The prevalence of *E. histolytica* infection in China has not been definitively ascertained. Recent data have revealed a higher seroprevalence of *E. histolytica* infection in HIV/AIDS patients in China (3) and approximately 0.7-2.7% of the Chinese population is reported to suffer from the amoebiasis (4). Liver abscesses are the most common non-enteric complication of amoebiasis. Presented here is a case of amoebic liver abscesses in both lobes in a patient with

high fever and continuous abdominal pain.

#### 2. Case report

This case involved a 57-year-old Chinese man who served as a doctor for ten years in the Republic of Cote d'Ivoire. He had fever, anorexia, and dull and continuous epigastric pain. He had been hospitalized at a local clinic in Cote d'Ivoire for three weeks. He presented with chills, a temperature of up to 39°C, and epigastric pain upon hospitalization. The fever and abdominal pain persisted and edema and respiratory distress developed during the final ten days of treatment. The patient had no history of diarrhea or vomiting. At the local clinic, he was diagnosed with malaria and treated with empiric antimalarial and antityphoid drugs to no effect. He was then sent back to China and admitted to the hospital.

Upon examination, he was febrile (38.5°C) and presented with hepatomegaly and pitting edema. Ultrasonography of the abdomen revealed multiple hypoechoic lesions in both hemilivers. Computed tomography (CT) scans revealed these to be multiple lesions. Results indicated pleural effusion on both sides and two hypodense lesions in the liver, 9.9 × 9.5 × 10 cm on the right and 13 × 9 × 9 cm on the left (Figure 1). Whole blood analysis revealed a leukocyte count of 13,620/mm<sup>3</sup>, mild normochromic normocytic anaemia (96 g/L), thrombocytosis (40,100/mm<sup>3</sup>),

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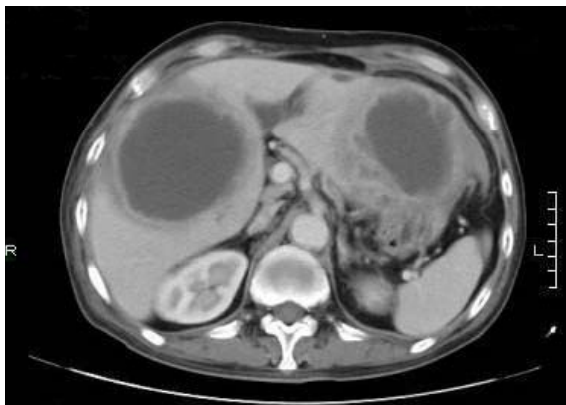
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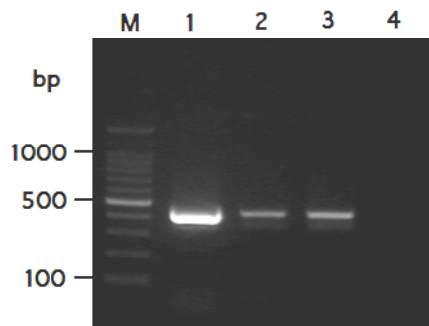
and high erythrocyte sedimentation rate (82 mm/h). The patient's renal function was normal. Data on the patient's liver function revealed slightly decreased liver function indicating hypoglycemia and hypoproteinemia. Liver biochemistry results were abnormal. The patient tested positive for hepatitis B surface antigen and anti-hepatitis B core IgG and anti-hepatitis B eAg antibodies. However, the patient tested negative for hepatitis B eAg and anti-hepatitis B surface antigen antibodies. PCR was performed to confirm the HBV viral load. The patient tested negative for anti-HIV and anti-hepatitis C virus antibodies. Sera tests for infection with *Schistosoma japonicum*, *Echinococcus granulosus*, and *Fasciola hepatica* were negative.

The patient was heterosexual with no history of intravenous drug abuse and was not an active smoker or drinker. He had no changes in toilet habits and no history of yellow fever and tuberculosis. He had malaria 12 years ago.

A serum indirect fluorescent-antibody test (IFA) for *E. histolytica* was performed (5). The patient's anti-*E. histolytica* antibody titer was 1:1,024 (Figure 2).



**Figure 1.** Abdominal computed tomography scan showing lesions of  $9.9 \times 9.5 \times 10$  cm in the right hemiliver and  $13 \times 9 \times 9$  cm in the left. Lesions were hypodense with rim enhancement.

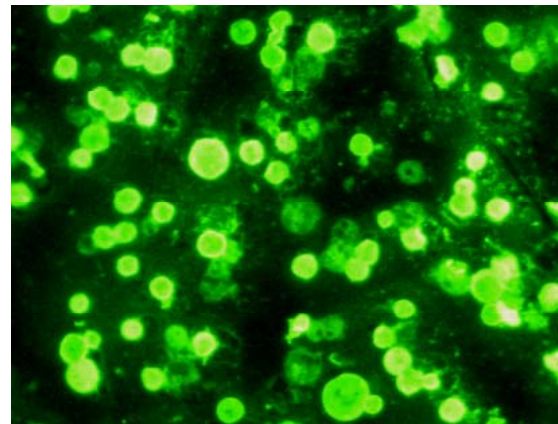


**Figure 3.** PCR amplification of 18S rRNA genes from liver pus DNA. The *E. histolytica* 18S primer was used. Templates are genomic DNA from *E. histolytica* HK9 (lane 1), liver pus from the patient (lanes 2 and 3), and a negative control (lane 4). M, DNA size marker (100 bp ladder).

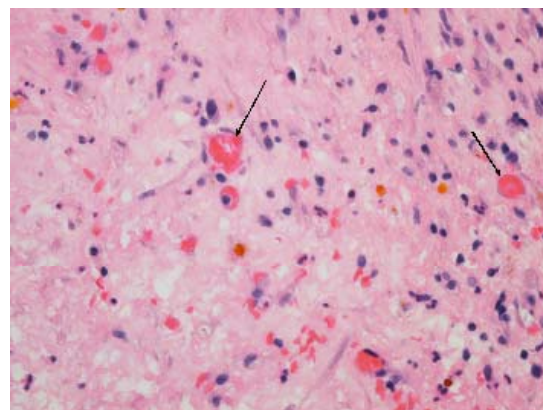
Ornidazole and levofloxacin were not effective. Two weeks of subsequent treatment with chloroquine caused the patient's fever to go down. Pleural effusion and edema gradually decreased. However, abdominal pain still persisted. Open surgical drainage was performed. Two pigtail catheters were placed into the lesions, and 600 mL of thick anchovy sauce-like pus was drained from the lesions. The diagnosis of an amoebic liver abscess was confirmed by DNA assay by detecting 18S rRNA genes (6) (Figure 3). Histopathological examination of necrotic inflammatory exudates revealed multiple trophozoite-like cells of *E. histolytica* (Figure 4). After aspiration and pigtail catheter drainage of the abscesses, cultures of the pus were bacteriologically sterile. A CT examination 3 weeks after drainage revealed that the abscesses had decreased markedly in size (Figure 5). The pigtail catheters were removed and the patient was discharged.

### 3. Discussion

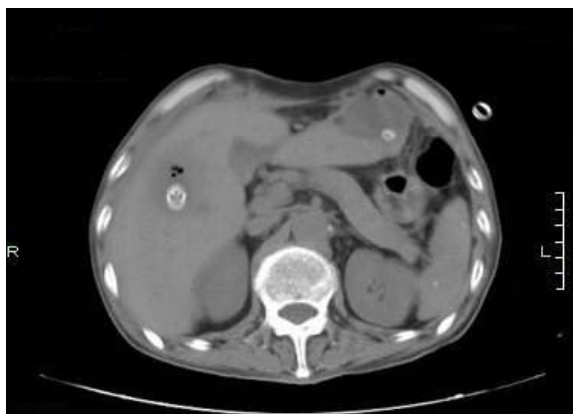
Hepatic amoebiasis is the most serious consequence



**Figure 2.** Detection of serum anti-*E. histolytica* antibodies using IFA. Original magnification:  $\times 100$ .



**Figure 4.** Hematoxylin/eosin-stained section from the patient's liver. Numerous trophozoite-like objects (arrows) are present in the peripheral region of the abscess.



**Figure 5.** Abdominal computed tomography scan after surgery showing insertion of two catheters into the hepatic lesions.

of invasive amoebiasis since various complications associated with amoebic liver abscesses include rupture of the abscess into the pleural, pericardial, and peritoneal cavities and the bile ducts. The early detection of *E. histolytica* is crucial to reducing morbidity and mortality (7). The current patient lived in the Republic of Cote d'Ivoire for over ten years, which may be a major factor for infection with *E. histolytica* (8). Hepatic amoebiasis is a result of trophozoites entering mesenteric venules and traveling to the liver through the hepatoportal system. Amoebic liver abscesses are often seen in young men and more often involve the right hemiliver than the left.

Amoebic liver abscesses are difficult to distinguish from bacterial abscesses or other liver diseases. Although epidemiological information may indicate that the patient has come from an area where amoebiasis is endemic, acute onset of fever, abdominal pain, and hepatomegaly are common to both amoebic and bacterial abscesses. Ultrasonography, abdominal CT, and magnetic resonance imaging are not specific for the differentiation of an amoebic liver abscess from a pyogenic liver abscess, necrotic hepatoma, or echinococcal cyst. Helpful clues to an accurate diagnosis include the presence of epidemiologic risk factors for amoebiasis and the presence of serum anti-amoebic antibodies.

In general, nitroimidazoles, and metronidazole in particular, are the mainstay of therapy for invasive amoebiasis. Nitroimidazoles with longer half-lives (namely, tinidazole, secnidazole, and ornidazole) are better tolerated and cause fewer side effects, allowing shorter periods of treatment. That said, complicated amoebic abscesses may require drugs with drainage according to the principles for treatment of

extraintestinal amoebiasis (9).

A review of the current case suggests that a primary diagnosis of amoebiasis would have led to prompt management of the condition with minimal morbidity. The combination of serological tests with target gene detection by PCR amplification of the parasite offers the best approach to diagnosis. Absence of diarrhea and parasites in the stool should not exclude the possibility of amoebiasis. Amoebiasis should be considered in patients from a population with a high prevalence of the condition should they present with a high fever and abdominal pain.

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