CASE REPORT

UNDIAGNOSED AMEBIC BRAIN ABSCESS

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Abstract. We report a case of amebic brain abscess due to *Entamoeba histolytica*. The patient was a 31-year-old man who presented with amebic liver abscess. His clinical course deteriorated in spite of proper drainage and treatment. He developed delirium, lethargy and then expired. With a history of heroin addiction, withdrawal syndrome from heroin was suspected. At autopsy, amebic abscesses were detected in the liver, large intestine, meninges and brain. A 19 cm amebic liver abscess was found in the right lobe of the liver. A 4 cm amebic brain abscess was found in the right occipital lobe. Microscopically, the tissue sections from the affected organs were confirmed to have degenerated *E. histolytica* trophozoites. Involvement of the brain in amebic liver abscess should be suspected in patients with neurological signs and symptoms.

INTRODUCTION

Entamoeba histolytica is the causative agent of amebiasis. It is an important pathogenic amoeba affecting people worldwide. More than 50 million persons worldwide are believed to be infected with *E. histolytica* (Walsh, 1986). The disease causes 40,000-100,000 deaths annually but the majority of patients (90%) are asymptomatic (Ackers and Mirelman, 2006). Transmission of the disease is from the cysts passed in the stool by infected human hosts. When ingested, the cysts reach the small intestine where the trophozoites are released from the cyst (excystment) and migrate to the large intestine. Clinical amebiasis occurs when trophozoites living in the intestinal lumen, penetrate the colonic mucosa. The clinical symptoms range from dysentery to extraintestinal disease manifested as abscesses of various organs (Ackers and Mirelman, 2006). A common site of extraintestinal abscess formation is the liver, spreading through the portal vein (Haque *et al*, 2003). Distant spread of trophozoites can be seen, but is rare, such as to the lungs and brain in the form of an amebic abscess.

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CASE REPORT

We report here a case of clinically diagnosed amebic liver abscess with a complication of amebic brain abscess which remained undiagnosed until death. A 31-year-old man was referred from another hospital because of epigastric pain for 2 months. Five months prior to admission, he had mucous and bloody stools for 2 weeks. He did not seek treatment. The symptoms subsided spontaneously leaving only a slight pain at the right hypochondriac region. He was diag-

nosed as having hepatitis and treatment was given. A history of heroin addiction was also noted. On admission, he appeared chronically ill, cachectic and moderately pale but had no jaundice. His vital signs were as follows: T 38.5ºC, PR 100/minute, RR 20/minute and BP 130/70 mmHg. The heart and lungs were unremarkable. Other pertinent findings included an enlarged liver down to the right iliac crest with upward extension of liver to the fourth intercostal space (by percussion). His relevant laboratory findings included a Hb of 7.5 g%, WBC 11,000 cells/mm^3, neutrophils 73%, lymphocytes 26%, eosinophils 1%. The liver function test was within normal limits. Chest radiography showed an elevated right hemi-diaphragm and an enlarged shadow of the liver. The diagnosis of liver abscess was made. Diagnostic liver aspiration revealed anchovy-like pus, with the presence of *E. histolytica* trophozoites.

He was treated with an antiamebic drug. On the following day, liver aspiration was repeated and yielded 600 ml of anchovy-like pus. He had high fever (T 39.5ºC) and complained of fatigue, epigastric distension, pain at the right hypochondriac region and headache. Two days later, he developed severe headache. On the fourth day of hospitalization, his condition deteriorated. He became lethargic with delirious episodes and progressed rapidly to a comatose state and expired. The clinical diagnosis was amebic liver abscess with underlying heroin addiction.

A whole body autopsy was performed. The liver was markedly enlarged. It weighed 2,500 g. There was a 19 cm solitary abscess occupying almost the whole right lobe of the liver (Fig 1). The abscess contained 1,200 ml of anchovy-like pus mixed with yellow creamy fluid and necrotic tissue. The compressed liver parenchyma was tan-brown and firm. The remaining liver tissue, especially in the left lobe, was congested. The superior part of the right lobe was adhered to the diaphragm which was elevated. Adhesion of the posterior part of the liver to the right kidney, right adrenal gland, part of the gall bladder and hepatic flexure was also noted. The large intestine, particularly the cecum and sigmoid colon, showed several undermined-edge ulcers, each measuring 0.5 cm in diameter. The brain weighed 1,400 g; it was markedly congested and swollen. There was thick yellow purulent fluid covering the meninges at the base of the brain. In the right occipital lobe, a 4-cm abscess was noted (Fig 2a). It contained 500 ml of thick anchovy-like fluid mixed with thick yellow purulent fluid. The adjacent brain parenchyma was congested. The heart, lungs and other organs were within normal limits. Microscopically, the large abscess of the liver showed degenerated amebic trophozoites at the rim of the abscess infiltrated with chronic inflammatory cells, plasma cells and eosinophils. The margin of the abscess was demarcated by fibrous connective tissue which separated the area of abscess and the compressed hepatocytes. The large intestine, especially the cecum and sigmoid colon, had ulcers with amebic trophozoites. The tissue sections from the brain, particularly at the right occipital lobe, revealed acute suppurative meningitis and an amebic brain abscess with the presence of degenerated *E. histolytica* (Fig 2b).

DISCUSSION

Cases of amebiasis disseminated to the brain are very rare. Fewer than 0.1% of amebic liver abscess cases disseminate to the brain (Stanley, 2003). Just over 50% of amebic brain abscess are associated with intestinal symptoms (Lombardo *et al*, 1964). Orbison *et al* (1951) collected 64 cases of amebic brain abscess from the literature

and reported five more cases. The patients developed sudden onset headaches, vomiting, seizures and mental changes (Orbison *et al*, 1951). More cases of amebic brain abscess have been documented since then (Lombardo *et al*, 1964; Hughes *et al*, 1975; Becker *et al*, 1980; Banerjee *et al*, 1983; Schmutzhard *et al*, 1986; Ohnishi *et al*, 1994; Shah *et al*, 1994; De Villiers and Durra, 1998; Di Rocco *et al*, 2004; Sundaram *et al*, 2004; Solaymani-Mohammadi *et al*, 2007; Sayhan Emil *et al*, 2008).

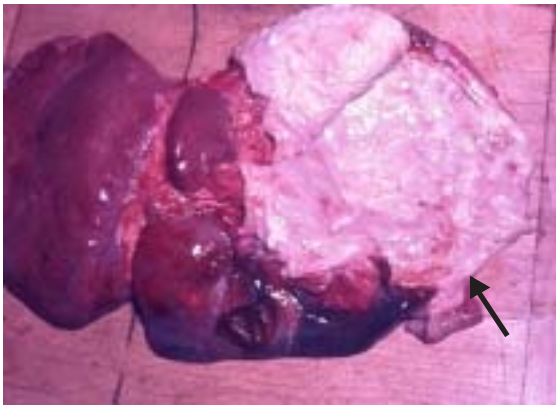

Fig 1–A huge liver abscess of the right lobe showing (yellow) creamy pus (arrow). Cut surface of the abscess showing a solitary mass, pus and necrotic tissue.

Lesions within the brain have been reported as single or confluent abscesses (Banerjee *et al*, 1983; Solaymani-Mohammadi *et al*, 2007; Yamasaki *et al*, 2007) as well as multiple abscesses (Hughes *et al*, 1975; Di Rocco *et al*, 2004; Sayhan Emil *et al*, 2008). The right side of the brain is more likely to be involved, particularly the posterior-parietal area (Shah *et al*, 1994), occipital lobe (Di Rocco *et al*, 2004), parieto-occipital lobe (Yamasaki *et al*, 2007) and right cerebellar peduncle (Sayhan Emil *et al*, 2008). In our case, the amebic brain abscess was in the right occipital lobe. The finding of amebic trophozoites in the brain lesion is rare, although some reports described trophozoites in the brain abscess (Becker *et al*, 1980; Shah *et al*, 1994; Di Rocco *et al*, 2004; Sayhan Emil *et al*, 2008). The diagnosis of amebiasis

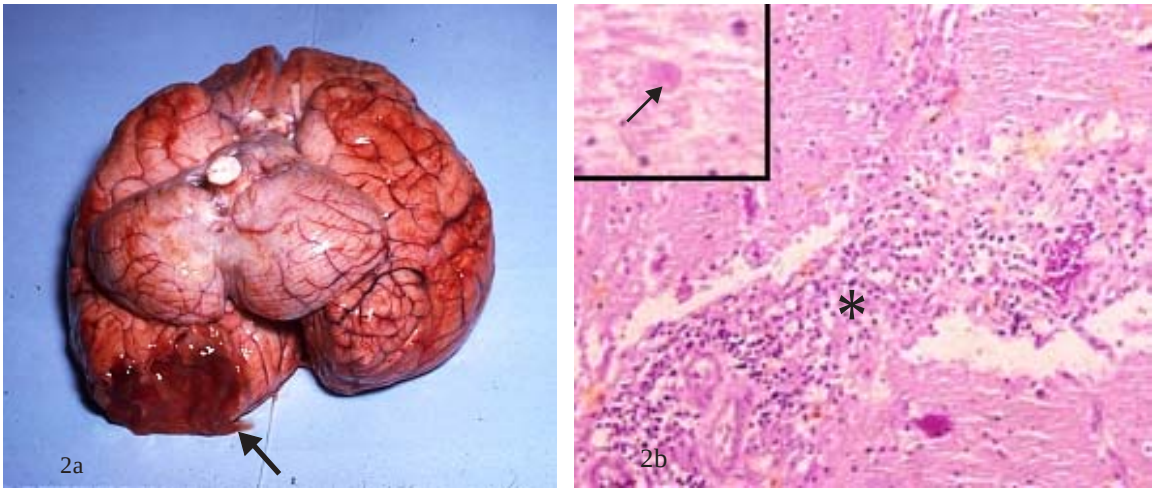

Fig 2–Ventral aspect of the brain showing a 4 cm abscess of the right occipital lobe (2a), containing thick anchovy-like fluid mixed with thick (yellow) purulent fluid (arrow). Adjacent brain parenchyma showed congestion. Tissue section of the meninges showed purulent meningitis (*) (2b), H&E 100X. A degenerated amebic trophozoite is noted (inset), H&E 400X.

of the brain is possible with a combination of computed tomography or magnetic resonance imaging findings, positive serological results and response to antiamebic drugs (Shah *et al*, 1994). For other extraintestinal amebiasis, serological methods, such as indirect hemagglutination assay, latex agglutination, countercurrent immunoelectrophoresis, indirect immunofluorescence, radioimmunoassay and ELISA have been used. A positive serological test for *E. histolytica* in patients with neurological symptoms may suggest amebiasis of the brain (Haque *et al*, 2003). Recently, polymerase chain reaction has been reported to be useful in the diagnosis of amebiasis in cases where amebic trophozoites cannot be demonstrated by microscopy (Roy *et al*, 2005; Solaymani-Mohammadi *et al*, 2007).

Generally, the outcomes of amebic brain abscess cases are good with correct diagnosis and proper antiamebic treatment. Antiamebic drug include metronidazole is administered intravenously with the help of abscess drainage (Ohnishi *et al*, 1994; Shah *et al*, 1994; Di Rocco *et al*, 2004; Sundaram *et al*, 2004; Sayhan Emil *et al*, 2008).

In this case, an amebic brain abscess was not suspected during the initial lethargic, delirious state. Instead, the patient was diagnosed as having heroin withdrawal symptoms. Although, brain involvement in amebic liver abscess (or intestinal amebiasis) is rare, it is a serious condition and may be fatal. It should be suspected when there are associated headaches and mental changes. Clinical diagnosis of brain abscess in this case may have been possible with proper neurological evaluation followed by neuroimaging.

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