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 (ex-cystment) 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.

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 chroni-
cally ill, cachectic and moderately pale but
had no jaundice. His vital signs were as fol-
lows: T 38.5°C, PR 100/minute, RR 20/minute
and BP 130/70 mmHg. The heart and lungs
were unremarkable. Other pertinent find-
ings included an enlarged liver down to the
right iliac crest with upward extension of
liver to the fourth intercostal space (by per-
cussion). His relevant laboratory findings
included a Hb of 7.5 g%, WBC 11,000 cells/
mm³, 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. Diag-
nostic liver aspiration revealed anchovy-like
pus, with the presence of *E. histolytica*

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 39.5°C) and
complained of fatigue, epigastric distension,
pain at the right hypochondriac region and
headache. Two days later, he developed se-
vere headache. On the fourth day of hospi-
talization, his condition deteriorated. He
became lethargic with delirious episodes
and progressed rapidly to a comatose state
and expired. The clinical diagnosis was
americ 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 com-
pressed 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 under-
mined-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 an-
chovy-like fluid mixed with thick yellow
purulent fluid. The adjacent brain paren-
chyma was congested. The heart, lungs and
other organs were within normal limits. Mi-
croscopically, the large abscess of the liver
showed degenerated amecic trophozoites at
the rim of the abscess infiltrated with chronic
inflammatory cells, plasma cells and eosino-

DISCUSSION

Cases of amebiasis disseminated to the
brain are very rare. Fewer than 0.1% of ame-
bic liver abscess cases disseminate to the
brain (Stanley, 2003). Just over 50% of ame-
bic brain abscess are associated with intesti-
nal symptoms (Lombardo *et al*, 1964).
Orbison *et al* (1951) collected 64 cases of
americ 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).

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
of the brain is possible with a combination of computed tomography or magnetic resonance imaging findings, positive serological results and response to antiamoebic drugs (Shah et al., 1994). For other extraintestinal amebiasis, serological methods, such as indirect hemagglutination assay, latex agglutination, countercurrent immunoelctrophoresis, 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 antiamoebic treatment. Antiamoebic 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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