Entamoeba histolytica meningoencephalitis diagnosed by trophozoites in cerebrospinal fluid

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Abstract

Entamoeba histolytica meningoencephalitis has not been described in the modern literature, which is distinct from that caused by free-living amoebae. We report the first case of *E. histolytica* meningoencephalitis without liver or brain abscesses. Cerebrospinal fluid revealed 2 + very motile trophozoites. Our patient was successfully treated with intravenous metronidazole.

Keywords: Amoebiasis, cerebrospinal fluid, *Entamoeba histolytica*, meningoencephalitis, trophozoites

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Case Report

A 16-month-old Polynesian boy presented to the emergency room with a 4-day history of cough, dyspnoea, wheezing, anorexia, fever and a 1-day history of non-bloody, mucoid diarrhoea. A month earlier, he had undergone surgical debridement of scabies-induced pyoderma of his feet. He also had recurrent otorrhoea.

Pertinent physical findings were mild respiratory distress with retractions (respiratory rate of 50 breaths/min, pulse oximetry of 98% on room air), wheezing, normal abdominal and neurological examinations and healing sores on anterior right foot. Laboratory abnormalities included marked leucocytosis of 45.2×10^9/L—89% neutrophils, haemoglobin 1.09 mmol/L, haematocrit 0.228, platelets 630×10^9/L and electrolyte disturbances. He was hospitalized for bronchiolitis, acute gastroenteritis and severe dehydration. Ampicillin, crystalloids, bronchodilators and methylprednisolone were given.

On day 2, his abdomen became very distended with absent bowel sounds. Abdominal X-ray revealed air–fluid levels. He was transferred to the intensive care unit for an acute abdomen and anuria. Ceftriaxone and metronidazole were added. Blood culture grew *Streptococcus pyogenes;* a repeat culture was negative. Computed tomography of abdomen without contrast showed ascites without free air.

On day 3, an exploratory laparotomy revealed ascites of 700 mL, copious peritoneal fibrinous exudates throughout, inflamed liver without abscess and 15 cm of inflamed jejunum with petechiae. Peritoneal fluid culture grew *S. pyogenes.* He remained intubated and critically ill.

On day 4, stool returned positive for *Entamoeba histolytica* 2+ cysts—he was switched to oral metronidazole. Stool cultures were negative. On day 6, he received a dose of dexamethasone on self-extubation.

On day 8, he developed tonic–clonic seizures. Computed tomography with/without contrast of the brain and abdomen revealed no intracranial abnormality or liver abscess. Subsequently, he had prolonged seizures. When he was stabilized, a lumbar puncture revealed bloody cerebrospinal fluid (CSF) with leucocytes 3/μL, erythrocytes 3096/μL, protein 1.73 g/L, glucose 3.6 mmol/L, negative Gram stain and culture; direct wet mount revealed 2+ very motile trophozoites. He received intravenous metronidazole for 2 weeks. No further seizures were noted. On follow up he was doing well without any sequelae.

Invasive amoebiasis causes extra-intestinal disease via haematogenous spread to the liver, lung, brain and rarely other organs [1]. Legrand described 24 cases and Lombardo *et al.* described 17 cases of cerebral amoebiasis associated with liver abscesses [2,3]; neither mentioned finding trophozoites in the CSF. A case of *E. histolytica* liver abscess with encephalitis was diagnosed by PCR of the CSF, which was negative for trophozoites [4]. Our patient would be the first case of *E. histolytica* meningoencephalitis without liver or brain abscesses, with trophozoites in the CSF.

Computed tomography can be negative in the early stages of encephalitis and cell count in the CSF may be falsely low, if measured more than 30 minutes after the lumbar puncture is performed [5]. Several risk factors undoubtedly contributed to the development of fulminant invasive amoebiasis: very young

age, malnutrition, compromised innate immunity and treatment with high-dose corticosteroids [1].

Hence, in endemic areas, clinicians need to be actively looking for cerebral amoebiasis in a child with intestinal amoebiasis and new-onset seizures. It may be under-recognized because of a lack of clinical suspicion and of laboratory capacity.

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Conflicts of Interest

None declared.

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