Acanthamoeba encephalitis in an immunocompetent child and review of the imaging features of intracranial acanthamoebic infections in immunocompetent patients

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Abstract

Granulomatous amoebic encephalitis caused by Acanthamoeba is a rare entity mainly affecting immunocompromised patients. We reported a case of Acanthamoeba encephalitis of a 1-year-old immunocompetent child and described the CT and MRI findings of the brain, while reviewing the relevant literatures. The imaging findings of Acanthamoeba meningoencephalitis in immunocompetent patients are non-specific and pose a diagnostic challenge.

Keywords: Acanthamoeba; encephalitis; granulomatous; magnetic resonance imaging; computed tomography

INTRODUCTION

Acanthamoeba is a widespread free-living amoeba found in diverse habitats predominantly in water and soil. It exists in the form of replicating trophozoite or dormant cyst. The Acanthamoeba cyst is highly resistant to extreme temperature, disinfection and desiccation, which accounts for its ubiquitous distribution in our environment. Rarely, Acanthamoeba may be pathological mainly as an opportunistic infection in immunocompromised individuals. The recognised types of infection caused by Acanthamoeba include granulomatous amoebic encephalitis, meningitis, amoebic keratitis as well as cutaneous, nasopharyngeal and disseminated infections. Here, we reported an Acanthamoeba encephalitis in an immunocompetent child and discussed its imaging findings.

CASE REPORT

A 1-year-old child presented with two episodes of left-sided focal seizures associated with fever. She was previously well. The routine biochemical study showed normal full blood count, coagulation profile and C-reactive protein. The child was provisionally treated as meningoencephalitis and started on intravenous ceftriaxone and acyclovir empirically. However a week later, she developed left-sided hemiparesis. MRI of the brain was performed and revealed ill-defined high signal areas (T2W and FLAIR) with diffusion restriction on DWI sequence in both frontal lobes and right basal ganglia consistent with acute infarcts (Figure 1). A few days later, she developed a right-sided focal seizure, which warranted an urgent neuroimaging. The CT and MRI scans demonstrated intraparenchymal haemorrhagic transformation of the earlier detected lesions associated with perilesional oedema and significant mass effect causing midline shift to the left (Figure 2).

He was intubated and underwent craniotomy and evacuation of the right temporo-parietal haemorrhage. Samples were sent for histopathological analysis, which demonstrated multiple amoebic-like structures within the brain parenchyma and near the blood vessels. The culture, however, came back as negative. Polymicrobial therapy was initiated. A follow-up MRI scan at 3 months showed persistent multiloculated collections of high signal intensity in both T2W and FLAIR in the right frontal and parietal lobes. These collections showed minimal rim enhancement on contrast-enhanced T1W images (Figure 3). No diffusion restriction was
Figure 1. MRI images on day 1 of admission. The axial (A) FLAIR and (B) T2 W images showing ill-defined high signal changes in both frontal lobes and right basal ganglia. The (C) ADC and (D) DWI images demonstrate diffusion restriction of these lesions.

noted in DWI sequence (Figure 3). Re-craniotomy was performed and the collections were aspirated as much as possible and revealed pale yellow jelly like material. Microscopic study of the wet mount found a few slow moving amoeboid-looking microorganisms compatible with *Acanthamoeba* spp trophozoites. Subsequently, the PCR with DNA sequencing of the sample were also consistent for *Acanthamoeba* spp.

She received 106 days of intravenous fluconazole and trimethoprim, and oral rifampicin, which all were subsequently converted to oral therapy. Serial imaging of the brain showed partial resolution of the intraparenchymal lesions. However, neurologically, she had residual swallowing incoordination, spastic quadriplegia with dystonia and left focal seizures.

**DISCUSSION**

*Acanthamoeba* encephalitis affecting immunocompetent patient is extremely rare. The exact prevalence or incidence of this entity
in Malaysia or South East Asia is unclear. To the best of our knowledge, there were no previous published report on *Acanthamoeba* encephalitis in Malaysia although a few cases of *Acanthamoeba* keratitis were reported.\(^3\,4\) There is no obvious link between this entity affecting immunocompetent patients and the patients’ socioeconomic status. It is important to note that *Acanthamoeba* has also been isolated in treated water.\(^5\)

The description of imaging findings of *Acanthamoeba* encephalitis was not extensive in the literature and was mainly described in sporadic case reports. Presence of multifocal lesions involving the cortex or corticomedullary junctions associated with white matter oedema and mild mass effect is a recognized pattern albeit nonspecific of granulomatous amoebic encephalitis.\(^6\,8\) Diencephalon, brain stem, thalamus and cerebellum may also be affected.\(^6\,8\) Post contrast imaging of the brain lesions reveals variable appearance from none to avid. Ring, target-like, punctate, heterogeneous and gyriform enhancement have been described in the literature.\(^8,9\)

Review of the reported cases of acanthamoebic meningoencephalitis specific to immunocompetent patients with description of imaging findings showed 3 out of 16 cases had normal cross-sectional brain imaging (Table 1).\(^9\,22\) The diagnosis in these cases was made from the CSF cytology or culture. One case showed a small pneumatocele with normal imaging findings of the brain and meninges.\(^13\) However, the pneumatocele indicated a fistula between the intracranial cavity and the nasopharynx, hence explained the route of spread into the CNS, rather than as a sequela of the acanthamoebic infection itself. Non-specific cerebral oedema and hydrocephalus may also be the only imaging findings in intracranial acanthamoebic infection.

![Figure 2. CT and MRI images after 1-week.](image)

Figure 2. CT and MRI images after 1-week. The (A) axial plain CT brain shows haemorrhagic transformation of the lesions with significant mass effect. The (B) FLAIR image shows high signal intensity and the (C) GRE image shows blooming artifact of the haemorrhage.

![Figure 3. MRI images at 4 months.](image)

Figure 3. MRI images at 4 months. (A) Axial T1W post gadolinium image shows hyperintense collection with minimal rim enhancement in the right frontal lobe. (B) Coronal FLAIR image shows the collection as hyperintense signal. (C) Axial ADC map shows no evidence of diffusion restriction.
Table 1: Summary of neuroimaging findings of cases of intracranial *Acanthamoeba* infections in immunocompetent patients

<table>
<thead>
<tr>
<th>Age /Sex</th>
<th>Diagnosis</th>
<th>Imaging descriptions</th>
</tr>
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<tbody>
<tr>
<td>Case 1 8yrs/M</td>
<td>CSF cytology</td>
<td>CT brain showed communicating hydrocephalus and basal cisterns enhancement.</td>
</tr>
<tr>
<td>Case 2 8yrs/M</td>
<td>CSF cytology</td>
<td>MRI brain showed multiple small round enhancing lesions in both supratentorial (including deep gray matter) and infratentorial (cerebellum).</td>
</tr>
<tr>
<td>Case 3 3yrs/M</td>
<td>CSF cytology with positive culture</td>
<td>Normal CT brain</td>
</tr>
<tr>
<td>45yrs/F</td>
<td>CSF cytology</td>
<td>Initial CT and MRI brain were normal. Subsequent CT brain showed obstructive hydrocephalus.</td>
</tr>
<tr>
<td>26yrs/M</td>
<td>CSF cytology</td>
<td>MRI brain and spine showed multiple lesions in supratentorial region and intramedullary of the spinal cord at T7/8 level.</td>
</tr>
<tr>
<td>64yrs/M</td>
<td>CSF cytology and positive culture</td>
<td>Pneumotocoele with no imaging features of meningitis or intracranial lesions. Radionuclide cisternography confirm CSF rhinorrhoea but no morphological defect detected in CT or MRI.</td>
</tr>
<tr>
<td>63yrs/M</td>
<td>Positive CSF culture</td>
<td>Normal CT Brain</td>
</tr>
<tr>
<td>15yrs/F</td>
<td>CSF cytology and positive culture</td>
<td>Normal MRI brain</td>
</tr>
<tr>
<td>25yrs/M</td>
<td>Cytology of the ependymal cyst.</td>
<td>CT brain showed an anterior interhemispheric cyst of CSF density. On MRI, the cyst showed high signal in T2W and low signal in T1W.</td>
</tr>
<tr>
<td>17yrs/M</td>
<td>CSF PCR and culture</td>
<td>MRI brain showed multiple infarcts and basal meningeal contrast enhancement on post contrast imaging.</td>
</tr>
<tr>
<td>38yrs/M</td>
<td>Autopsy specimen cytology</td>
<td>The initial lesion was in the right frontal lesion, which was hypodense in CT brain. On MRI, it was hypointense in T1W and hyperintense in T2W imaging. Post contrast imaging showed ring like and gyriform enhancement. MR spectroscopy showed lipid peak and supression of NAA, choline and creatinine. Subsequently more lesions developed in ipsilateral frontal and contralateral temporal and parietal lobes.</td>
</tr>
<tr>
<td>5yrs/F</td>
<td>Post mortem CSF cytology with positive culture.</td>
<td>CT brain showed diffuse cerebral oedema</td>
</tr>
<tr>
<td>38yrs/M</td>
<td>Serology study. Cytology, PCR and immunofluorescent study of resected brain tissue</td>
<td>CT brain revealed a right temporal lesion. Selected image of T1 W post gadolinium MRI imaging showed the lesion was hypointense with patchy and irregular rim enhancement with perilesional oedema.</td>
</tr>
<tr>
<td>63yrs/M</td>
<td>Cytology of biopsy taken from the brain lesion</td>
<td>CT showed multiple hypodense areas resembling watershed infarcts in the left parietal, occipital and frontal lobes. MRI revealed mass like lesions associated with surrounding oedema.</td>
</tr>
<tr>
<td>4yrs/M</td>
<td>CSF cytology and culture</td>
<td>CT brain showed mild hydrocephalus.</td>
</tr>
<tr>
<td>41yrs/M</td>
<td>Cytology of the excised specimen</td>
<td>MRI showed tumour like lesion in the right frontoparietal lobe.</td>
</tr>
<tr>
<td>1yr/F</td>
<td>Cytology of resected brain specimen and aspirated fluid of intracranial collection. Positive PCR study</td>
<td>Multiple infarcts in CT and MRI in both frontal lobes and right thalamus. DWI showed diffusion restriction. Subsequently, there was haemorrhagic transformation of the infarcts. Collections with rim enhancement and lack of diffusion restriction gradually developed post operatively.</td>
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in immunocompetent group. A single case has reported acanthamoebic infection within an anterior inter-hemispheric cyst as a very unusual manifestation. Of the cases with intraparenchymal brain involvement, the location of the brain lesions were variable and may involve supratentorial and/or infratentorial regions. Random location suggest haematogenous mode of spread of the infection in these cases. Most of these lesions were hypodense on CT scan; hypointense on T1W, hyperintense on T2W and hyperintense on FLAIR sequences of MRI imaging. In the present case, multiple lesions were initially present in both frontal lobes and the right basal ganglia. Two cases in particular were presented with imaging features of infarcts as the initial presentation, similar to the current case. It was reported that several common infectious agents increase the risk of ischaemic stroke, as the pathogens are thought to promote inflammation, increasing atherosclerosis and subsequent predisposing patient to ischaemic stroke. In addition, histopathological analysis demonstrates predilection of Acanthamoeba trophozoites to aggregate in the perivascular spaces causing cuffing of inflammatory cells around the affected vessels associated with intraluminal thrombi resembling panarteritis explaining the possible mechanism of the initial manifestation of an acute infarct, prior to the development of the cerebritis or abscess collections.

The present case demonstrated unprovoked haemorrhagic transformation of the areas of infarcts seen on the initial scan. Intralesional haemorrhage is a recognized imaging and macroscopic findings of amoebic encephalitis. The haemorrhage is most likely caused by underlying necrotizing angitis, which is described in association with severe amoebic encephalitis. In addition, Acanthamoeba has been reported to cause microbial intracranial aneurysm which can rupture and cause haemorrhage.

Despite variable imaging findings, patient with neurological deficit associated with radiographic evidence of cerebral haemorrhage or acute infarcts, when there is no obvious underlying risk factors of infarction and haemorrhage, the patients should be investigated for systemic infection, particularly in young person with persistent febrile illness. Other than the more usual, viral, bacterial or fungal infections, potential parasite like Acanthamoeba should be borne in mind.

DISCLOSURE

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Conflict of Interest: None

REFERENCES


