Case Report

Acanthamoeba meningoencephalitis with hydrocephalus in an immunocompetent patient: A case report

Rahul Singh, Anurag Sahu, Kulwant Singh, Ravi Shankar Prasad, Nityanand Pandey

From Department of Neurosurgery, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India

ABSTRACT

Acanthamoeba is an unusual cause of meningoencephalitis having high mortality and it usually affects immunocompromised patients. We present a case of cerebrospinal fluid proven *Acanthamoeba* meningoencephalitis in a young male without immunosuppression. The patient presented with a chief complaint of fever for 1 month duration with features of meningoencephalitis, which progressed to rapid deterioration, and development of features of raised intracranial pressure. His initial contrast-enhanced computed tomography brain was normal but later on, magnetic resonance imaging of the brain showed features of hydrocephalus. Ventriculoperitoneal shunting was done followed by chemotherapy for *Acanthamoeba* and the patient recovered from illness.

Key words: *Acanthamoeba* meningoencephalitis, Hydrocephalus, Immunocompetent, Ventriculoperitoneal shunt

Acanthamoeba meningoencephalitis is caused by free-living ameba *Acanthamoeba*. These usually infect immunocompromised persons and have a high fatality. *Acanthamoeba* causes granulomatous encephalitis and meningoencephalitis along with keratitis, sinusitis, and cutaneous lesions [1,2]. *Acanthamoeba* infection of the brain has a high mortality [3]. *Acanthamoeba* meningoencephalitis presenting as hydrocephalus in an immunocompetent individual is a rare disorder. Ventriculoperitoneal shunting along with anti-*Acanthamoeba* medications is curative and leads to the resolution of symptoms in such patients.

Several case reports are present in the literature regarding the infection of *Acanthamoeba* meningoencephalitis in immunocompetent patients, but the patients reported did not presented with hydrocephalus. We present a rare case of *Acanthamoeba* meningoencephalitis with hydrocephalus diagnosed at cerebrospinal fluid (CSF) examination in an immunocompetent young patient.

CASE REPORT

A 15-year-old boy from Gaya, Bihar, presented with a chief complaint of headache and fever for 1 month. The fever was insidious in onset, progressive, occured daily, and was associated with chills, rigors, fast breathing, and palpitation. There was an evening rise of temperature that was relieved after taking medication. There was a history of loss of consciousness 4 months back for 8 h. He also gave a history of sewage cleaning 4 months back. There was no history of similar illness in the family. No history of any tuberculosis, diabetes mellitus, or any other chronic systemic illness. The child was fully immunized. No history of any delayed developmental milestones. The boy belongs to low socioeconomic status.

On examination, the patient was well oriented to time, place, and person. General examination revealed fever of 100°F, pulse rate of 90/min, respiratory rate of 15/min, and oxygen saturation (SpO_2) of 95%. Neck rigidity was present with positive Kernig and Brudzinski signs. Pupils were bilaterally constricted and reactive to light. Other neurological and systemic examinations were normal.

Tuberculous meningitis is common in India, so the patient was initially suspected to have extrapulmonary tuberculosis (tuberculous meningitis). After CSF biochemical report, contrast-enhanced computed tomography (CECT) scan evaluation, and the presence of evening rise in temperature for 1 month along with the signs of meningitis, a strong suspicion of tuberculous meningitis was made and the patient was started empirically on anti-tubercular drugs (rifampicin, pyrazinamide, ethambutol and isoniazid, and IM streptomycin).

Lumbar puncture was done; CSF was clear under mild pressure.

CSF examination revealed total cell count = 600 (N90L10), CSF glucose = 30 mg/dL, and CSF protein = 97 mg/dL. CSF was sterile after culture for 48 h. CSF-GeneXpert was negative. CSF adenosine deaminase was 4.30 U/L. Acid-fast bacillus staining was negative.

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Correspondence to: Anurag Sahu, Department of Neurosurgery, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India. E-mail: anuragsahubhuneurosurgery@gmail.com

CSF Gram staining showed Gram-positive cocci. CSF for fungal stain was negative. CSF examination revealed motile organisms resembling *Acanthamoeba* (Fig. 1). Blood culture was sterile. Urine routine/microscopy was normal and culture was sterile. The chest X-ray was normal. The Mantoux test was negative after 48 h. CECT scan head was normal with normal fundus findings on fundoscopy. The erythrocyte sedimentation rate (ESR) was 40 mm/hr. Total white blood cell count was $9.96 \times 10^3/\mu l$ at admission with differential leukocyte count of N67L20.7M6.8E5.3B0.2. The patient was negative for human immunodeficiency virus (HIV).

Based on the above findings, a diagnosis of primary amebic meningoencephalitis was made. The initial CECT brain did not show any signs of hydrocephalus (Fig. 2) and the fundus examination showed hyperemic disc. The patient was started on phenytoin (5 mg/kg/day IV/oral for 6 months), amphotericin B (1.5 mg/kg/day IV for 3 days followed by 1 mg/kg/day IV for 11 days), rifampicin (10 mg/kg/day IV/oral for 4 weeks), ketoconazole (5 mg/kg/day IV/oral for 8 weeks), and cotrimoxazole (20 mg/kg/day for 8 weeks).

The patient improved initially but after 7 days, there was a fall in the Glasgow Coma Scale (E4V4M5) and signs of raised intracranial tension (vomiting and bradycardia) appeared. Magnetic resonance imaging brain of the patient was done and it revealed hydrocephalus with periventricular ooze (Fig. 3). The fundus examination showed papilledema. The patient was prepared for surgery and neuronavigation-guided right-sided medium pressure ventriculoperitoneal (MPVP) shunting was done. The patient improved in the post-operative period and signs of meningeal irritation were resolved. The patient became well oriented to time, place, and person (E4V5M6) and was discharged on the 7th post-operative day on oral rifampicin (for 4 weeks), ketoconazole (for 8 weeks), and cotrimoxazole (for 8 weeks). Repeat CT scan after 1 month revealed resolved hydrocephalus. CSF examination at 1-month follow-up was negative for *Acanthamoeba*. The patient was followed up in the outpatient department after 15 days, 1 month, and 3 months with no symptoms.

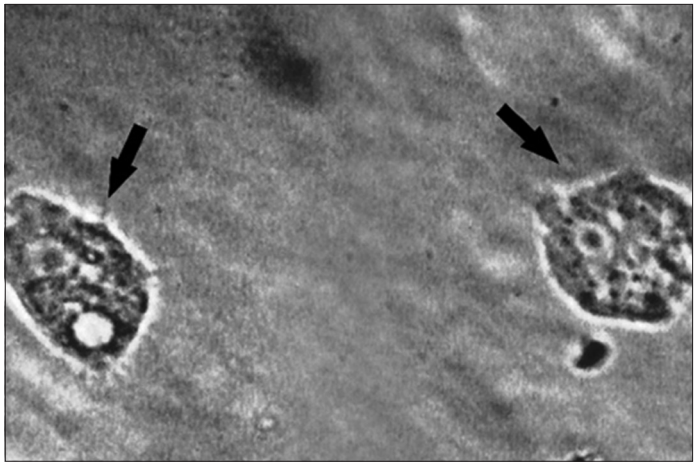

Figure 1: *Acanthamoeba* trophozoites in cerebrospinal fluid (black arrow)

DISCUSSION

Amebic infections of the brain are of two types, namely, primary amebic encephalitis caused by *Naegleria fowleri* and granulomatous amebic encephalitis caused by both *Acanthamoeba* species and *Balamuthia mandrillaris* [4]. *Acanthamoeba* occurs ubiquitously in the air, water, soil, contaminated contact lens solutions, dialysis units, and sewage and air-conditioning units [5]. This patient gives a history of sewage cleaning 4 months back. It usually infects weak and immunocompromised individuals. In this case, the patient was immunocompetent. Conditions predisposing include diabetes mellitus, splenectomy, hypoalbuminemia, renal failure, bone marrow failure, lymphoproliferative, hematoproliferative disorders, splenectomy, hypoproteinemia, corticosteroid use, immunomodulatory drug use, chemotherapy, and HIV infection [6]. It has also been reported in immunocompetent individuals from low socioeconomic status [7]. In this case, the boy belonged to low socioeconomic status.

Case reports of *Acanthamoeba* infecting immunocompetent patients [8-11] show different imaging findings and outcome (Table 1). Hydrocephalus was not present in any of these

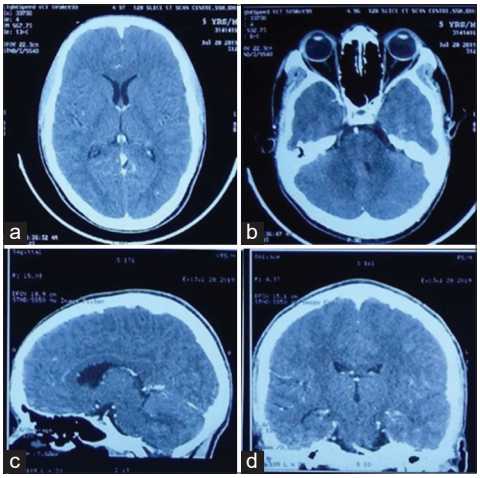

Figure 2: Contrast-enhanced computed tomography scan brain (a) and (b) axial sections, (c) sagittal section, and (d) coronal section showing normal findings and absence of hydrocephalus

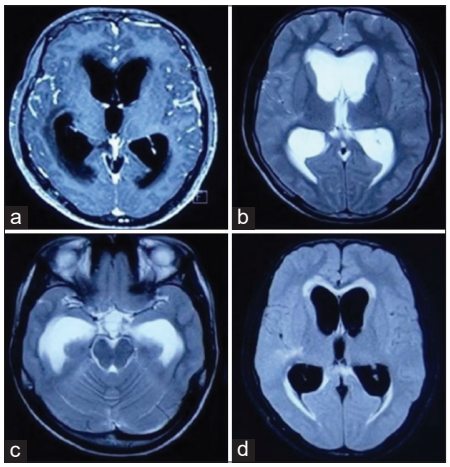

Figure 3: Magnetic resonance imaging brain of patient. (a) is axial T1, (b and c) are axial T2, and (d) is axial fluid-attenuated inversion recovery images showing hydrocephalus with periventricular ooze

Table 1: Case reports of *Acanthamoeba* meningoencephalitis in immunocompetent patients

Author	MRI/CT findings and diagnosis	Immune status	Outcome
Lackner *et al.* [8]	MRI showed multiple infarcts and basal meningeal contrast enhancement Diagnosis: Acute granulomatous *Acanthamoeba* encephalitis based on CSF culture	Immunocompetent	Recovery
Reddy *et al.* [9]	MRI showed a right parietal lesion hypointense on T1-weighted image, hyperintense on fluid-attenuated inversion recovery scan and showing target-like appearance on contrast imaging Diagnosis: *Acanthamoeba* meningoencephalitis on autopsy histopathological examination of brain and CSF examination	Immunocompetent	Death
Khanna *et al.* [10]	Contrast-enhanced computed tomography brain showed normal features Diagnosis: *Acanthamoeba* meningoencephalitis on CSF examination	Immunocompetent	Recovery
Das *et al*. [11]	MRI brain showed left middle cerebral arterial territory infarcts in first case and Normal MRI in the 2nd case Diagnosis: *Acanthamoeba* encephalitis based on CSF culture in both cases	Immunocompetent	Residual right side hemiparesis in the 1st patient and recovery in the 2nd patient

MRI: Magnetic resonance imaging, CT: Computed tomography, CSF: Cerebrospinal fluid

case reports of *Acanthamoeba* brain infection. Diagnosis of *Acanthamoeba* brain infection was made on the basis of CSF examination, CSF culture, or histopathological examination of the affected brain tissue in these case reports. Histopathological examination reveals vasculitis, trophozoites, or cysts in perivascular spaces, lymphocytic infiltration, and granulomatous lesions [11,12].

CSF examination revealed raised, reduced glucose, and raised CSF protein in this patient. In various previous reports, CSF analysis had shown mild-to-moderate pleocytosis with lymphocytes and neutrophils, decreased glucose, and elevated total protein [6]. To confirm the diagnosis of *Acanthamoeba* meningitis, CSF wet mount, Giemsa-Wright staining technique, culture, and polymerase chain reaction are used [13]. Hanging drop technique helps in identifying the trophozoites showing characteristic movement in fresh CSF samples [13]. In this patient, the CSF examination revealed motile organisms resembling *Acanthamoeba*.

The treatment regimen for *Acanthamoeba* meningoencephalitis is controversial. Drugs used are ketoconazole, fluconazole, sulfadiazine, albendazole, amphotericin-B, rifampicin, and trimethoprim-sulfamethoxazole [14]. Reports have shown that a combination of rifampicin with trimethoprim-sulfamethoxazole and ketoconazole has good results [15,16]. This patient recovered completely with amphotericin B (1.5 mg/kg/day intravenous [IV] in two divided doses for 3 days followed 1 mg/kg/day for 11 days), rifampicin (10 mg/kg/day IV/PO for 28 days), ketoconazole (5mg/kg/day IV/PO for 8 weeks) and cotrimoxazole (20 mg/kg/day IV/PO for 8 weeks), and ventriculoperitoneal shunting.

This patient was having rapidly progressing hydrocephalus for which MPVP shunting was done. Hydrocephalus in *Acanthamoeba* meningoencephalitis has been reported in a few studies [17,18]. A combination of chemotherapy with a ventriculoperitoneal shunt has resolved the disease in this patient. In a nutshell, one must keep in mind while managing patients with meningoencephalitis causing progressive hydrocephalus that *Acanthamoeba* can be a causative agent in an immunocompetent patient also.

CONCLUSION

Acanthamoeba meningoencephalitis is a rare cause of rapidly progressing hydrocephalus in immunocompetent patients as reported in this case report. Ventriculoperitoneal shunting along with a complete course of amphotericin B, rifampicin, ketoconazole, and cotrimoxazole combination chemotherapy heralds the successful recovery of the patient.

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