Case Report

A rare case of survival from primary amebic meningoencephalitis

Parshotam Lal Gautam[1], Shruti Sharma, Sandeep Puri[2], Raj Kumar[3], Vandana Midha[2], Rajinder Bansal[4]

Abstract

Primary amebic meningoencephalitis (PAM) is a rare and fatal disease of central nervous system (CNS) caused by *Naegleria fowleri*, an ameba found in soils and warm waters. It enters the CNS after insufflation of infected water by attaching itself to the olfactory nerves.The infection is usually difficult to diagnose and has a poor prognosis.The present case is one such case in which CSF examination led us to the diagnosis of PAM and finally to a favorable outcome when treated with Amphoterracin B and antibiotics.

Keywords: Amphotericin B, central nervous system, cerebrospinal fluid, *Naegleria fowleri*, primary amebic meningoencephalitis

Access this article online
Website: www.ijccm.org
DOI: 10.4103/0972-5229.94432
Quick Response Code:

Introduction

Primary amebic meningoencephalitis (PAM) is caused by *Naegleria fowleri*, an ameba ubiquitous in soils and warm waters. It enters the central nervous system after insufflation of infected water by attaching itself to the olfactory nerve. It migrates into the olfactory bulbs of the forebrain and multiplies by feeding on nerve tissue.[1,2] *N. fowleri* amebae migrate through the cribriform plate, along the fila olfactoria and blood vessels, and into the anterior cerebral fossae, where they cause extensive inflammation, necrosis, and hemorrhage leading to high mortality. About 310 cases have been reported with a high case fatality rate of approximately 95%. Seven survivors of PAM are reported in the western literature and from India; two survivors have been reported so far the present case being the third one.[3-5] Very few are successfully treated and we are reporting one such case.

From:
Intensivist, Critical Care Division [1] Departments of Anaesthesia and Critical Care Division, [2]Department of Medicine, [3]Department of Microbiology, [4]Department of Neurology, Dayanand Medical College and Hospital, Ludhiana, Punjab, India

Correspondence:
Dr. Shruti Sharma, Intensivist, Critical Care Division, Dayanand Medical College and Hospital, Ludhiana, India. E-mail: shruti_sarit@yahoo.co.in

Case Report

A 73-year-old male, a known case of type II diabetes mellitus, diabetic nephropathy, coronary artery disease (postangioplasty in 2005), head injury (9 years back), and CSF (cerebrospinal fluid) rhinorrhea (diagnosed 1 year back now recovered) presented with history of fever, neck pain, seizures, and altered sensorium since 3 days. Fever was not associated with chills or rigors. On examination neck rigidity was present. Pupils were equal size reacting to light bilaterally. There was no evidence of any cranial nerve involvement or focal neurological deficit. Deep tendon reflexes were normal and plantars were extensor bilaterally. The patient was given injection lorazepam 4 mg and phenytoin 1500 mg over 30 minutes intravenously to control the seizures. Orotracheal intubation was accomplished to control airway and assist ventilation. He was given dextrose 25% 50 ml to treat suspected hypoglycemia prior to shifting to our hospital casualty and the Random blood sugar (RBS) at presentation was 426 mg/dl. Treatment was started with injection ceftriaxone and vancomycin considering him a case of bacterial meningitis. Investigations at the time of admission showed hemoglobin 11 gm/dl, total leucocyte count of 32,000/mm^3 (polymorphs 75%, lymphocytes 15%, eosinophils 4%, monocytes 4%, and basophils 3%), blood urea 67 mg/dl, serum creatinine

1.6 mg/dl, and blood RBS as 212 mg%. Arterial blood gas analysis showed metabolic acidosis. CT head showed prominent sulci suggestive of cerebral atrophy. CSF done few hours after the patient presented revealed negative gram stain and microscopy showed 70 cells/mm^3 with polymorhs 92%, and lymphocytes 8%. CSF biochemistry analysis showed sugar 165 mg/dl, proteins 120 mg/dl, globulin +++, and chloride 116 mg/dL. CSF adenosine deaminase was 13.3 units/l and CSF fungal and bacterial culture showed no growth. A latex agglutination test for *Staphylococci, H. Influenza, S. Pneumoniae, E. Coli, and N. Meningitis* was negative. CSF for the agglutination test for *Cryptococcus* antigen was also negative. He developed ventilator-associated pneumonia (VAP) with growth of *Acinetobacter* in endotracheal secretions on the fourth day of admission, so injection piperacillin and tazobactum was added according to culture and sensitivity reports. The conscious level did not improve so a repeat lumbar puncture was done on the 5th day of admission. Gram stain was negative and the biochemistry revealed high proteins. This time the wet preparation of the CSF revealed trophozoites of *Naegleria* species with an irregular shape, vacuolated cytoplasm, and a single nucleus located centrally or slightly eccentrically. Giemsa stain confirmed the presence of amebae and CSF culture for pyogenic organism and the fungus was negative. On a non-nutrient agar medium with a lawn culture of E. Coli revealed growth of trophozoites of *N. fowleri*, when examined under the microscope, after 48 hours. Treatment was started with injection Amphotericin B (1 mg/kg/day) and oral Rifampicin (600 mgOD) apart from other antibiotics with careful monitoring of urine output, renal, and liver function tests. The patient improved and was extubated on the 10th day of admission. His CSF sample examined 2 weeks later revealed the persistence of *N. Fowleri*. Treatment was continued with Amphotericin B for a month along with other supportive treatment. He had a full recovery without any neurological deficit. CSF examination done after 4 weeks was normal.

Discussion

N fowleri is one of several species in the genus *Naegleria* and the only one known to produce human disease.[6,7]

This form of nervous system infection by ameba was first documented in Australia in 1965. *N fowleri* trophozoites are motile and move by extending a blunt lobopodium (pseudopodium) and destroy tissue with which they come into contact. Trophozoites replicate by binary fission. When the *N fowleri* trophozoites are exposed to a change in ionic concentration, such as placement in distilled water, they transform into biflagellates or multiflagellates. Trophozoites encyst in response to unfavorable conditions.[8]

Most *N. fowleri* infections have occurred in children and young adults in summers that have had recent exposure to swimming or diving in warm fresh water. The symptoms of PAM are indistinguishable from acute bacterial meningitis. The illness begins suddenly with the abrupt onset of fever, headache, nausea, and vomiting. Altered mental status occurs in about two-third of patients and is followed by rapid deterioration to coma and death.[9]

Diagnosis is based on the patient's history and examination of the CSF. The CSF glucose level may be low or within the reference range, but the protein is usually elevated. CSF gram stain is negative for bacteria. CSF wet mount is positive for motile trophozoites and is of paramount importance for the diagnosis. Additional methods of diagnosing *N. fowleri* infection include polymerase chain reaction (PCR), monoclonal antibodies, DNA probes, and isoenzyme profile analysis. Limited data are available on imaging studies. Amphotericin-B (intrathecal and intravenous) is the drug of choice. The patients may benefit from Rifampicin and Tetracyclin. Sometimes Metronidazole and Ornidazole are also added for better results.[4,5,9]

Our patient is one of the few survivors of PAM who was diagnosed and responded well to treatment with Amphotericin B, and oral Rifampicin. The age group of our patient is atypical for presentation of this disease. There was no history of exposure to pond water or swimming prior to the infection but probably the patient was harboring the pathogens in the nose since a long time and CSF rhinorrhea predisposed the patient to PAM. His symptoms led us to start him on antibiotic treatment for bacterial meningitis, but lack of improvement and repeat CSF examination led us to the diagnosis. So, the amebic infection should be suspected in a patient who has sign and symptoms of encephalitis/meningitis and in whom CSF examination is negative for pyogenic, fungal, tubercular, and viral infection with increased polymorphs and high proteins, but wet mount direct CSF examination is showing motile trophozoites. Ultimately a timely diagnosis and early start of treatment may lead to a favorable outcome.

References

1. Cabanes PA, Wallet F, Pringuez E, Pernin P. Assessing the risk of primary amoebic meningoencephalitis from swimming in the presence of environmental Naegleria fowleri. Appl Environ Microbiol 2001;67:2927-31.
2. Centers for Disease Control and Prevention (CDC). Primary amebic

meningoencephalitis--Arizona, Florida, and Texas, 2007. MMWR Morb Mortal Wkly Rep 2008;57:573-7.
3. Gupta N, Bhaskar H, Duggal S, Ghalaut PS, Kundra S, Arora DR. Primary amoebic meningoencephalitis: first reported case from Rohtak, North India. Braz J Infect Dis 2009;13:236-7.
4. Jain R, Prabhakar S, Modi M, Bhatia R, Sehgal R. Naegleria meningitis: a rare survival. Neurol India 2002;50:470-2.
5. Singh SN, Patwari AK, Dutta R. Naegleria meningitis. Indian Pediatr 1993;35:101-25.
6. Schuster FL. Cultivation of pathogenic and opportunistic free-living amebas. Clin Microbiol Rev 2002;15:342-54.
7. Available from: http://emedicine.medscape.com/article/223910-overview last updated on 2009 Aug 3. [Last cited on 2010 Feb 13].
8. Parija SC, Jayakeerthee SR. Naegleria fowleri: a free living amoeba of emerging medical importance. J Commun Dis 1999;31:153-9.
9. Niu MT, Duma RJ. Amebic infection of the nervous system. Handb Clin Neurol;8:309-37.{cited in literature as Handbook of Clinical Neurology vol.8:309-337

How to cite this article: Gautam PL, Sharma S, Puri S, Kumar R, Midha V, Bansal R. A rare case of survival from primary amebic meningoencephalitis. Indian J Crit Care Med 2012;16:34-6.

Source of Support: Nil, **Conflict of Interest:** None declared.