



# Primary Amebic Meningoencephalitis

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## Abstract

This case is reported with the intention of highlighting the presentation of primary amebic meningoencephalitis as acute meningitis, a rare differential diagnosis with presence of free living amoebas in the CSF. ©

## INTRODUCTION

Infection of humans with free living amebas is an infrequent but often life threatening occurrence in both normal and immunocompromised individuals. Fatal CNS infection by free living amebas present with features similar to acute pyogenic meningitis. High index of suspicion is required to diagnose primary amebic meningoencephalitis in patients of acute pyogenic meningitis without e/o bacteria on different investigations with positive h/o bath or contact with swimming pool or lakes. CNS invasion by free living amebas, Naegleria, Acanthamoeba and Balamuthia mandrillaris formerly known as leptomyxid amebas has been reported in approximately 400 patients world wide and only four of them survived with high dosages of Amphotericin B and Rifampin. The prognosis is uniformly poor. This case report highlights primary amebic meningoencephalitis presenting as acute pyogenic meningitis.<sup>1,2</sup>

## CASE REPORT

A 30 years male admitted on 28/12/2002 with continuous high grade fever since 15 days without chills and rigors. He had also generalized headache and h/o vomittings since 8 days. Vomittings were 6-8 times per day, projectile, containing ingested food and gastric juices with yellowish green colour. He had h/o generalized weakness of the body. There was no history of convulsions, cough, haemoptysis, ear discharge and burning micturition. No h/o pulmonary tuberculosis in past. No h/o visit/bath to swimming pool or poater lake. No h/o sexually transmitted disease in the past.

Clinical examination revealed febrile but conscious,

oriented, irritable patient with a pulse rate of 110/minute, regular BP - 110/70 mmHg and RR-20/minute. There was no pallor, icterus, cyanosis, clubbing, oedema feet, lymphadenopathy and JVP was not raised. Skin and ENT examination was normal and no other significant finding noted.

Central nervous system examination showed marked neck stiffness and bilateral extensor plantar without signs of focal neurological deficit. Examination of other system revealed no abnormality. The provisional diagnosis of acute pyogenic meningitis was made.

## Investigations

Hb-12 gm%, TLC - 16000/cumm, with neutrophils 80% and lymphocytes 20%. ESR 30 mm at the end of one hour by Wintrobe method, peripheral smear was normocytic normochromic and negative for malarial parasite. Widal was negative. Findings of urine examination, LFT, KFT, USG abdomen, stool examination and X-ray chest were normal. ELISA for HIV and blood culture were negative. Examination of fundus showed bilateral papilloedema. CSF examination revealed proteins 226 mg%, Sugar 26%, plenty of pus cells (3+), occasional lymphocytes HPF without any e/o bacteria. Positively free living, motile amebas were seen and impression of acute pyogenic meningitis versus primary amebic meningoencephalitis was made. CAT scan report showed e/o generalized cerebral edema with increased vascularity in post-contrast study (suggestive of meningitis).

Patient was treated with Mannitol, Cefotaxim, Amikacin and Metronidazole in appropriate doses initially. Azithromycin was also given (7.5 mg/kg) for 5 days. Cefotaxim and Amikacin were discontinued after 10 days due to no clinical response. Crystalline penicillin with cloxacillin were also given for 10 days. Nursing care, nutrition were maintained and antipyretics were given round the clock. In spite of administration of antibiotic and antiamebic treatment, patient's condition deteriorated with persistent fever, s/o meningeal irritation and altered consciousness. Second CSF was

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*Figs. 1 and 2 : Photograph showing presence of Amoeba in cerebrospinal fluid a pt. of primary amoebic meningo-encephalitis. (Marked with ←)*

done on 20/1/2003 which revealed 50% polymorphs and occasional lymphocytes but no free living, motile amoeba. Sugar was 85 mg%, proteins 154 mg%, pus cells were present. In Gram staining, no bacteria were seen. Treatment was continued and thereafter patient's condition deteriorated rapidly and patient died on 24/1/2003 due to terminal cardiorespiratory arrest. Culture of second CSF sample was negative.

## DISCUSSION

Primary amoebic meningoencephalitis is caused by free living amoebas of genera *Naegleria fowleri*, *Acanthamoeba* and *Balamuthia mandrillaris* formerly known as leptomyxid amoeba.<sup>1,2</sup> These are free living, amebizoic and opportunistic protozoa that are ubiquitous in nature. These amoebas are found in soil, water, air samples from all over world. Man gets infected from inhalation of trophozoites or cysts on exposure to polluted water in ponds, swimming pools and manmade lakes. Raised temperatures during hot summer months or warm water from power plants facilitates the growth of *Naegleria fowleri* (a thermophilic amoeba that grows well in tropical and subtropical climates).<sup>3</sup> Human infection involves brain, lung, skin and eyes and has increased significantly since last 10 years.

CNS infection occurs in two forms:

- Primary amoebic meningoencephalitis, (PAME) caused by *Naegleria fowleri*
- Granulomatous amoebic encephalitis, (GAE) caused by Spp of *Acanthamoeba* and *Balamuthia mandrillaris*.<sup>4</sup>

Incubation period for *N fowleri* is 3 to 7 days and that for *acanthamoeba* is unknown but perhaps more than 10 days PAME produced by *N fowleri* is characterised by acute fulminant meningoencephalitis with severe headache, high grade fever, nausea, vomitings, altered consciousness with signs of meningeal irritation. Cranial nerve palsies, seizures and coma may follow and death occurs within a week if not treated early. The portal of entry to CNS is through olfactory neuroepithelium.

Victims are healthy young individuals with H/o recent water related activities. GAE caused by *Acanthamoeba* species and *Balamuthia mandrillaris* occurs in immunocompromised host i.e. in patients of AIDS, Chronic debilitated, malnourished, those on steroids and chemotherapy drugs and organ transplant patients. The course is typically subacute with altered mental status, headache, fever, neck stiffness, seizures, focal neurological signs such as cranial nerve palsies and coma leading to death within 1 week to several months after onset. *Balamuthia* infection can also occurs in immunocompetent host. *Acanthamoeba* keratitis known to occur in contact lens wearer resulting in local signs of corneal ulcer and finally visual loss. The portal of entry to CNS is through haematogenous spread from respiratory tract or skin ulceration.<sup>1,3</sup>

Diagnosis depends on clinical features, positive H/o contact with lake water and negative cultures for bacteria, viruses, fungi and CSF detection of free living amoebas and observation of amoeba in water of wash of rhinopharynx and pharyngeal cavity and serological testing with monoclonal antibodies. Special culture media are required for culture of amoeba.<sup>5</sup> Serological test performed are indirect Immunofluorescence assay (IIA), dot immunobinding assay (DIBA) and enzymed linked immunotransfer blot technique (EITB). Neuroimaging findings Nonspecific brain edema favouring *N fowleri* suggestive of meningitis and punctuate enhancing lesions due to *acanthamoeba*. Successful outcome depends on early diagnosis and aggressive treatment with Amphotericin B and Rifampicin and only four survivals have been reported till date.<sup>1</sup>

Azithromycin in the dose of 7.5 mg/kg/day for 5 days can be effective in the treatment of PAME against *N fowleri* and may be useful additional therapy for PAME.<sup>6</sup> Also consensus has made that Amphotericin B and Ketoconazole remain the main drugs with proven activities against pathogenic *Naegleria* Spp. Successful treatment with Cotrimaxazole has been documented.

The present case is reported with the intention of highlighting the presentation of primary amebic meningoencephalitis as acute pyogenic meningitis, a rare differential diagnosis with presence of free living amebas in the CSF. Culture and immunological studies were not done due to unavailability of these facilities. However live recording of freely motile amebas was done (Figs.). So one must have high index of suspicion in any case of acute pyogenic meningitis in patients with a positive history of contact with lake water and negative bacterial culture reports.

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