PRIMARY AMOEBIC MENINGO ENCEPHALITIS- A CASE STUDY

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ABSTRACT

Primary amoebic meningoencephalitis (PAM) mimicks acute bacterial meningitis in presentation. However because it is very rare as compared to pyogenic meningitis, physicians may miss the diagnosis initially. Recent exposure to diving, swimming or splashing in warm fresh water should suggest the possibility of amoebic meningoencephalitis, and prompt examination of the cerebrospinal fluid (CSF) for *N. Fowleri*.Timely diagnosis requires a high index of suspicion in any individual presenting with acute onset of meningitis-like symptoms. We present a fatal case of PAM masquerading as an acute upper respiratory infection but a quick transition to pyogenic meningitis, and encephalitis.

INTRODUCTION

Primary amoebic meningoencephalitis is caused by the small, pathogenic, free-living amoeba *Naegleria Fowleri*, *Acanthamoeba species* and *Balamuthia mandrillaris*. The first of these, *N. Fowleri* produces a rare and sporadic acute central nervous system infection that culminates in the death of the host within 5 to 8 days[1]. The world over about 310 cases have been reported with a high case fatality rate of approximately 95% so far[2]. Fowler and Carter first described the potential for free-living amoebae to cause disease in 1965, and shortly thereafter, Butt reported several cases in Florida, coining the term primary amoebic meningoencephalitis (PAM) [3].

CASE PRESENTATION

At 49 years old, previously healthy, serving military officer was admitted to PNS Shifa on 8th May 2012 with a two day history of high grade fever and sudden severe headache. There was no history of swimming or exposure to recreational fresh water sources as stated by the attendants. He was a regular golf player. The general condition deteriorated further with development of vomiting and acute confusional state. He had high grade fever (102^0F), was restless and confused. The neck was supple; Kernig's sign positive/equivocal, fundi oculi normal and systemic examination was unremarkable. CT scan with contrast was normal. The peripheral blood picture was unremarkable with TLC of $10x10^9$ and a DLC within normal limits of distribution. CSF was turbid with increased protein content 4.5 g/l, low glucose and cell count was 12,000/HPF mostly neutrophils. Gram stain and culture from two different labs did not reveal any growth.

Considering the differential diagnosis of acute meningitis/viral encephalitis, cerebral malaria or Legionella disease, treatment for provisional diagnosis of acute pyogenic meningitis was started immediately. Due to the rapidly deteriorating condition of the patient, lack of bacterial growth in culture and negative gram staining in CSF, another CSF specimen was sent for *Nigleria Fowleri*. It was reported positive by two microbiologists independently in two different laboratories within next 24 hours. He was started on antiamoebic treatment with Amphotericin B at a maximum dose of 1 mg/kg BW IV infusion divided into the 8 hourly dose schedule. As an adjunct to Amphotericin B , IV Rifampicin was also started at a dose of 600 mg once daily dose. However, his condition continued to deteriorate; necessitating ventilator support and finally ended fatally on the 9th day of his admission.

DISCUSSION

N. Fowleri is a thermophilic amoeba that grows in tropical and subtropical climates, tolerating temperatures up to 45°C. It is a ubiquitous inhabitant of warm fresh water and soil, but it does not multiply at temperatures

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below 40°C and does not survive in seawater, its pathogenic strains were not isolated during a study in Malaysia[4] although rarely associated with clinical disease, exposure to this amoeba is common, as exemplified by the widespread presence of anti-*N. Fowleri* antibodies in the general population[5]. Elevated summer temperatures and warm water discharge from power plants to facilitate the growth of *N. Fowleri*[6]. In another study 67% water samples from geothermal sources were found to harbour *N. Fowleri*[7]. It may also occur via inhalation of contaminated dust[8]. Two fatal cases of confirmed PAM have been reported from a study at Agha University Hospital Karachi[9].

Its incidence is underestimated because of difficulty in making a definitive diagnosis. This is due in part to the fact that wet preparations of CSF are seldom examined under high magnification. Males are affected 3 times more frequently[10]. A case report of 8 fatal cases of PAM in India reported contact with water source in only 4 of the cases[11]. PAM is almost uniformly fatal, with only 10 survivors currently reported in the medical literature[12].

Naegleria species typically cause PAM in children and healthy adults who have been swimming in polluted pond water or inadequately chlorinated swimming pools, as well as man-made or natural freshwater lakes. Organisms enter through the olfactory neuroepithelium at the level of the cribriform plate and invade the amyelinic submucosal nervous plexus[13]. There is possibility of ritual ablution for prayer (Wudu,Ablution) using contaminated tap water as a possible mechanism of infection in our set up[14].

Symptoms begin after a 3–7 day incubation period. Another case of confirmed PAM in an infant without history of swimming has been reported from India[15] usual history is with abrupt onset, sore throat, headache, nausea, vomiting, malaise and fever. Early findings may also include irritability, hallucinations, meningismus, cerebellar ataxia and cranial nerve palsies, although focal neurologic deficits are usually absent. Seizures progressing to coma are frequently followed by death within 4 to 6 days[16].

Primary care physicians in endemic areas should caution their patients to be aware of symptoms after prolonged swimming in warm, fresh water lakes[17]. Frequent isolation of free living Amoeboea was identified as a public health risk[18] Public health authorities should advise closure of (or avoidance of submersion in) hot spring recreational sites where *N. Fowleri* is present, and of waterways and rivers infected with this organism during times of high temperature [>30°C] and low water levels. Careful maintenance of swimming pool filters and adequate chlorination, to at least 1–2 ppm, are important preventive measures[19].

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