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Review Article

Primary Amoebic Meningoencephalitis in Children: Kerala Experience and Emerging Public Health Concerns

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Abstract

Primary Amoebic Meningoencephalitis (PAM) is a rare but devastating central nervous system infection caused by Naegleria fowleri, a thermophilic free-living amoeba found in warm freshwater bodies. The disease primarily affects healthy children and young adults following nasal exposure to contaminated water during swimming or diving. Kerala has recently witnessed an alarming rise in reported PAM cases, bringing renewed attention to this highly fatal disease. Clinical manifestations mimic acute bacterial meningitis, often leading to delayed diagnosis and treatment. Rapid neurological deterioration, failure to respond to antibiotics and a history of freshwater exposure are important diagnostic clues. Laboratory diagnosis depends on prompt cerebrospinal fluid examination demonstrating motile trophozoites, while PCR remains the most sensitive confirmatory test. Current treatment strategies involve aggressive multidrug therapy, intracranial pressure control and intensive care support. Despite advances in therapy, mortality remains extremely high. This review summarizes the epidemiology, pathophysiology, clinical profile, diagnostic approach, treatment protocols and preventive strategies for PAM, with special emphasis on the recent Kerala experience and its implications for public health surveillance.

Keywords: Primary Amoebic Meningoencephalitis; Public Health Surveillance; Kerala

Introduction

Primary Amoebic Meningoencephalitis is an acute fulminant infection of the brain caused by Naegleria fowleri, popularly known as the "brain-eating amoeba". Although rare, PAM is associated with an exceptionally high mortality rate, often exceeding 95% [1]. The organism thrives in warm freshwater environments such as ponds, lakes, hot springs, poorly maintained swimming pools and stagnant water bodies. Human infection occurs when contaminated water forcefully enters the nose, allowing trophozoites to migrate along the olfactory nerve through the cribriform plate into the brain. The disease predominantly affects otherwise healthy children and young adults. Because its clinical presentation closely resembles bacterial meningitis, early diagnosis is frequently missed [2]. Patients usually present with fever, headache, vomiting, neck stiffness and rapidly progressive encephalopathy. Delay in diagnosis and treatment often results in death within days. In recent years, Kerala has emerged as a major hotspot for PAM in India, with repeated cluster events and increasing public concern. Rising temperatures, recreational freshwater exposure and greater awareness may have contributed to the apparent increase in cases [3,4].

Epidemiology and Kerala Experience

Naegleria fowleri infections have been reported worldwide, particularly in tropical and subtropical climates. The first documented cases were reported from Australia in 1965 by Fowler and Carter. Since then, sporadic cases have been identified across Asia, North America and Europe.

India has reported occasional PAM cases over several decades; however, Kerala has experienced a remarkable surge during recent years. Government data between 2023 and 2025 documented more than 200 cases with significant mortality. Most cases occurred during the summer months between March and June, corresponding with higher environmental temperatures and increased freshwater recreational activities.

Children and adolescents constitute the majority of affected patients. Freshwater ponds, lakes, wells and stagnant water bodies have been repeatedly implicated. Clustering of cases in specific districts suggests possible environmental factors and highlights the need for systematic surveillance.

Pathogenesis and Clinical Features

Naegleria fowleri exists in trophozoite, cyst and flagellated forms. The trophozoite stage is responsible for human disease. After entering the nasal cavity, trophozoites adhere to the olfactory mucosa and penetrate the cribriform plate. They subsequently migrate into the olfactory bulbs and frontal lobes, producing extensive hemorrhagic necrosis and inflammatory damage [5].

The incubation period ranges from one to nine days. Early symptoms include fever, severe headache, nausea, vomiting and loss of smell or taste. Neck stiffness and photophobia soon develop, followed by altered sensorium, seizures, coma and death. The rapid clinical progression is a hallmark of PAM.

A key diagnostic clue is the absence of response to empirical antibiotic therapy. Any patient with acute meningitis and recent freshwater exposure should immediately raise suspicion for PAM.

Diagnosis

Timely diagnosis is critical because survival depends on early initiation of therapy. A detailed exposure history is often the most important clinical clue. Patients frequently report swimming, diving or nasal water exposure in warm freshwater within two weeks before symptom onset.

Cerebrospinal fluid findings resemble bacterial meningitis, with neutrophilic pleocytosis, elevated protein, low glucose and increased opening pressure. Gram stains and bacterial cultures are usually negative. The most important diagnostic test is direct wet mount examination of fresh cerebrospinal fluid, which may reveal rapidly motile trophozoites.

Polymerase Chain Reaction (PCR) assays provide highly sensitive and specific confirmation. Neuroimaging findings include diffuse cerebral edema, basilar meningeal enhancement, hydrocephalus and hemorrhagic lesions, although these findings are nonspecific.

Treatment and Intensive Care Management

Management of PAM requires immediate aggressive therapy. Amphotericin B remains the cornerstone of treatment and may be administered intravenously as well as intrathecally or intraventricularly. Combination therapy with azithromycin, fluconazole, rifampicin and miltefosine has shown improved survival in selected cases [6].

Supportive intensive care measures are equally important. Raised intracranial pressure should be treated with mannitol, hypertonic saline, external ventricular drainage, controlled hyperventilation and therapeutic hypothermia when indicated. Some centers have also reported benefit from corticosteroids and pentobarbital coma in severe cases [7,8].

Despite these interventions, survival remains uncommon. Globally reported survivors typically received early diagnosis, multidrug therapy including miltefosine and aggressive intracranial pressure management.

Prevention and Public Health Importance

Preventive strategies are essential because effective treatment remains limited. Public education regarding the risks of swimming in warm stagnant freshwater during summer months is crucial. Individuals should avoid forceful water entry into the nose by using nose clips or keeping the head above water.

Swimming pools and recreational water facilities should be adequately chlorinated and maintained. Distilled or boiled water must be used for nasal irrigation and sinus rinsing. Environmental surveillance and water quality monitoring may help identify high-risk areas.

Kerala's recent experience demonstrates the importance of clinician awareness and rapid laboratory support. Establishing standard operating procedures for evaluating acute meningitis with freshwater exposure may improve outcomes and reduce diagnostic delays [9,10].

Conclusion

Primary Amoebic Meningoencephalitis remains one of the most lethal infectious diseases affecting the central nervous system. Increasing reports from Kerala underscore the growing public health significance of Naegleria fowleri infection in tropical regions. High clinical suspicion, rapid cerebrospinal fluid examination, early multidrug therapy and aggressive neurocritical care offer the only realistic chance of survival.

Healthcare professionals must maintain awareness of PAM, particularly during summer months and in patients with a history of freshwater exposure. Strengthening surveillance systems, improving diagnostic capacity and conducting community education programs are essential steps toward reducing mortality from this devastating disease.

Conflict of Interest

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Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement

Informed consent was obtained from all participants included in the study.

Authors' Contributions

All authors contributed equally to this paper.

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