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The Brain-Eating Amoeba: A Silent but Deadly Threat

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ABSTRACT

Naegleria fowleri is a free-living amoeba (FLA) that invades the central nervous system (CNS) and causes an acute and fatal infection known as primary amoebic meningoencephalitis (PAM). It is for this reason that it is also known as the "brain-eating amoeba". PAM is characterized by a high-mortality rate of 98%, causing death within a fortnight of initial exposure. Although PAM can be considered exceedingly rare, it has been suggested that the neurological illnesses caused by these free-living amoebas are underreported and often misdiagnosed, most likely because of a poor autopsy rate or insufficient knowledge of their pathophysiology. With this background, the review provides a comprehensive summary of Naegleria fowleri, including its life cycle, pathogenesis, virulence factors, laboratory diagnosis, treatment options, and preventive strategies. It is particularly relevant for microbiologists and infectious disease specialists interested in understanding the pathogen's molecular mechanisms and diagnostic. Medical practitioners and neurologists can benefit from the information to enhance early detection and treatment approaches for Primary Amoebic Meningoencephalitis (PAM). Epidemiologists and public health officials will find the discussion useful for tracking cases and developing preventive strategies to mitigate the risks of exposure. Healthcare policymakers can utilize this review to design response protocols for handling rare but fatal infections effectively. Additionally, researchers specializing in molecular biology and immunology may find valuable insights into the host-pathogen interactions and potential therapeutic interventions. Lastly, this review can serve as an important awareness resource for the general public, particularly swimmers and individuals engaging in freshwater recreational activities, helping them understand the risks and preventive measures associated with this deadly amoeba.

Keywords*: Naegleria fowleri; Primary Amoebic Meningoencephalitis (PAM);Brain-eating amoeba.*

INTRODUCTION

Certain pathogens can infiltrate the central nervous system by crossing the blood-brain barrier through specific molecular mechanisms. Among these are opportunistic protozoa and free-living amoebas (FLA), which are widespread in various environments (1). Members of the genus Naegleria, particularly the thermophilic freshwater amoeboflagellate Naegleria fowleri, belong to the Phylum Percolozoa, Class Heterolobosea, Order Schizopyrenida, and Family Vahlkampfiidae. This organism thrives in warm aquatic habitats and poses a potential threat to human health (2).

Of the approximately 30 species in the genus Naegleria, N. fowleri is the only known human pathogen (3). N. fowleri was named after Malcolm Fowler of Adelaide Children's Hospital in Australia, who, along with R.F. Carter, described Primary Amoebic Meningoencephalitis (PAM) following four deaths at the hospital attributed to amoebic invasion of the meninges causing brain damage and inflammation (3). N. fowleri has been found on all continents except Antarctica, in various water bodies such as lakes, ponds, rivers, hot springs, spas, and swimming pools, as well as in domestic water systems, air-conditioning systems, humidifiers, cooling towers, and soil environments worldwide (2).

The life cycle of Naegleria spp. comprises three stages: trophozoite, flagellate, and cyst. Trophozoites are the reproductive stage of the parasite and cause disease in humans. They divide by binary fission and grow at temperatures between 35–46 °C. Trophozoites are elongated, measuring 22 µm X 7 µm, with a large nucleus, dense central nucleolus, mitochondria, pseudopodia, a single contractile vacuole, endoplasmic reticulum, ribosomes, and amoebastomes. Their granular cytoplasm contains ingested red blood cells and leukocytes, along with cytoplasmic organelles. Their primary food sources are bacteria, algae, and yeast. Cysts are spherical structures, measuring 7-12 µm, with a thick endocyst and a thin ectocyst. They can survive temperatures as low as 4°C, remain dormant in winter, and resume reproduction during summer. In the presence of water, a cyst can transform into a pear-shaped flagellate, measuring 10-16 µm, which does not divide or feed.

These flagellate forms thrive at 27–37 °C and are usually present in warm waters or during summer. These flagellate forms can spontaneously revert to the trophozoite stage (4,5).

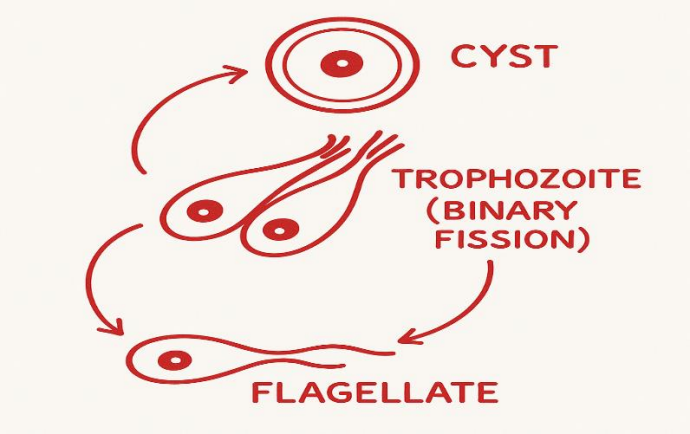

Figure 1. Life cycle of N. fowleri

Epidemiology:

Primary amoebic meningoencephalitis (PAM) was first documented in Florida, USA, in 1962. In 1966, the disease was formally named Primary Amoebic Meningoencephalitis (PAM) by Butt (6). PAM is a severe haemorrhagic-necrotizing meningoencephalitis that predominantly affects immunocompetent children and young adults. The occurrence of PAM is highest during the summer months when the thermophilic Naegleria fowleri thrives in warmer water temperatures, coinciding with increased recreational water activities (7). Global data from 2018 indicates that 39 countries have reported N. fowleri infections, with the USA, Pakistan, Mexico, India, Australia, and the Czech Republic being the most affected (8).

Recent observations indicate a rise in reported cases in Asia. However, this deadly condition is often misdiagnosed and likely underreported. In India, the first confirmed PAM case in Kerala was reported in the Alappuzha district in March 2016. Since then, seven more cases have been reported in the state, with a 100% mortality rate (9). Recent studies in India suggest a high fatality rate, with only four out of sixteen diagnosed cases surviving. Notably, most affected individuals had a history of using ponds and groundwater for swimming and bathing, environments where the presence of Naegleria has been confirmed in North India (9, 10).

Figure 2. Endemic areas of N. fowleri

Pathophysiology and Clinical Manifestations of PAM:
PAM is a haemorrhagic-necrotizing meningoencephalitis caused by N. fowleri, primarily affecting immunocompetent, healthy children and young adults with recent exposure to warm, contaminated freshwater sources. These sources include heated swimming pools, geothermal waters, warm ponds, and industrial cooling water discharged into sewage systems (11).

Transmission to humans typically occurs during hot summer months through the contact of nasal mucosa with water containing the parasite, often during recreational activities. Most patients report a history of such exposure approximately one week before the onset of neurological symptoms. Due to the lack of specific early clinical signs, PAM is frequently diagnosed post-mortem. Clinical manifestations can appear 5 to 7 days after initial exposure, with the illness progressing rapidly, sometimes within 24 hours. The non-distinctive clinical features often lead to misdiagnosis as pyogenic, bacterial, or viral meningitis (12).

Following nasal exposure, amoebic trophozoites migrate along the olfactory nerve, cross the cribriform plate, and reach the olfactory bulbs. This invasion triggers an inflammatory response and results in necrotic haemorrhages. Superficial haemorrhages are observed in the cortex, with lesions predominantly located at the base of the orbitofrontal and temporal lobes, the base of the brain, hypothalamus, midbrain, pons, medulla oblongata, and the upper spinal cord (13). N. fowleri inflicts severe damage to the central nervous system (CNS) due to its inherent pathogenicity and the intense immune response it elicits (5).

The limited understanding of its virulence factors and pathogenic mechanisms includes both contact-dependent and contact-independent mechanisms (5). Contact-dependent mechanisms involve adhesion and phagocytic food-cups. Trophozoites traverse the nasal epithelium by adhering to basement membrane (BM) components. This adhesion activates signal transduction pathways, facilitating CNS entry and proliferation. Axenically maintained N. fowleri (initially low in pathogenicity) can become significantly more virulent after passage through a mouse brain, exhibiting altered proteins involved in cytoskeletal rearrangement and stabilization. Contact phagocytosis, utilizing amoebastomes, causes severe tissue destruction. The Nfa1 gene in N. fowleri encodes a protein linked to locomotion and amoebastome formation, contributing to increased cell adhesion, phagocytosis, and cytotoxicity.

Contact-independent mechanisms involve cytolytic molecules secreted by the amoeba. Matrix metalloproteinases (MMPs) are endopeptidases that cleave gelatin and type IV collagen, aiding parasite invasion into the CNS by degrading the extracellular matrix (ECM) and simplifying passage from nasal chambers to the olfactory bulb. N. fowleri also degrades tight junction proteins (TJPs), disrupting the stability of the blood-brain barrier (BBB). Studies have shown the secretion of cysteine proteases that degrade TJPs such as claudins-1 and occludins. Furthermore, pore-forming, membrane-bound proteins and peptides can lyse nucleated cells and compromise host cell membrane integrity by depolarization. Phospholipases (A, C), sphingomyelinase, neuraminidase, and elastase contribute to host cell lysis, damage cytoplasmic membranes, and cause demyelination and neurodegenerative processes in PAM. N. fowleri produces nitric oxide (NO) in vitro and exhibits increased resistance to NO toxicity. Elevated levels of heat shock protein 70 (HSP70) in highly virulent N. fowleri help resist cellular stress and play a role in regulating the pathogen within the host immune system (14).

Neuroinflammation and its Association with PAM Pathogenesis:
N. fowleri can exacerbate host injury through the induction of a robust immune response. Trophozoites reach the olfactory bulb approximately 72 hours post-infection and trigger the production of reactive oxygen species (ROS), activate the epidermal growth factor receptor (EGFR) pathway, and induce the release of IL-8 and IL-1β. By 102 hours post-infection, eosinophils and neutrophils begin to surround trophozoites in the olfactory bulb. Eosinophils can produce pro-inflammatory cytokines such as TNF-α, IL-6, IL-8, and eotaxin. Between 108 and 120 hours post-infection, the number of neutrophils and macrophages increases, aiming to eliminate pathogens through degranulation, proteolytic enzymes, antimicrobial peptides, and ROS. N. fowleri also induces the production of neutrophil extracellular traps (NETs), composed of nuclear or mitochondrial DNA combined with histones and proteins from cytoplasmic granules. Neutrophils, in the presence of TNF-α, can destroy N. fowleri. However, this process also leads to extensive tissue damage.

To cross the BBB, N. fowleri releases cysteine proteases. It also induces the expression of leukocyte adhesion molecules and the production of NO, further altering the permeability of the BBB, allowing more leukocytes to enter the CNS. Macrophages release TNF-α, IL-1, and NO, which can combine to form peroxynitrite, a potent neurotoxin. Microglia, the resident immune cells of the brain, perform antigen-presenting and pro-inflammatory functions through the interaction of toll-like receptors (TLRs) with pathogen-associated molecular patterns (PAMPs), resulting in the production of IL-1β, IL-6, TNF-α, ROS, and reactive nitrogen species (RNS), which are neurotoxic and cause cell destruction through lipid peroxidation. The amoeba also promotes the activation of astrocytes, which play a role in regulating the immune system within the brain. Overall, these pro-inflammatory cytokines contribute to brain tissue destruction, breakdown of the BBB, and significant inflammation (15).

Clinical presentation:

A patient with Primary Amoebic Meningoencephalitis (PAM) typically presents with initial symptoms including frontal or bitemporal headaches, a high fever ranging from 38.5 to 41 °C, and nuchal rigidity accompanied by positive Kernig and Brudzinski signs. Other early symptoms may include nausea, vomiting, irritability, and restlessness. Subsequently, the patient develops photophobia and various neurological abnormalities such as diplopia, lethargy, seizures, and confusion, potentially progressing to a coma. Cerebral edema can lead to palsies of the third, fourth, and sixth cranial nerves. In some cases, myocardial necrosis and cardiac arrhythmias may also occur. Naegleria fowleri trophozoites can sometimes be detected in the cerebrospinal fluid (CSF). Increased intracranial pressure, ranging from 300 to 600 mmHg, can result in brain herniation and death (7, 16, 17). The prognosis for PAM is extremely poor, with a mortality rate of 98%, and death typically occurs within 7 to 10 days following infection (18). Due to the rapid onset and progression of PAM, the body has a limited chance to develop an effective humoral immune response against the amoebae (19). Post-mortem examination reveals cerebral hemispheres that are soft, swollen, edematous, and severely congested. The white matter shows demyelination, and the olfactory bulb exhibits inflammatory exudates and hemorrhages. The leptomeninges are congested, diffusely hyperemic, with limited cellular infiltration. Trophozoites can be detected at the base of the brain, hypothalamus, midbrain, subarachnoid space, and perivascular spaces (19).

Diagnosis:

Table 1. Diagnostic modalities for N. fowleri

Diagnostic Modalities for N. fowleri
CT Scan
MRI
Culture
Microscopy
Immunofluorescence assay (IF)
Enzyme-linked immunosorbent assay (ELISA)
Flow cytometry (FC)
Reverse transcription PCR (RT-PCR)
Clinical evaluation (History + features)

Initial Stages: Contrast-enhanced computerized tomography (CT) scans and magnetic resonance imaging (MRI) during the early stages of infection often reveal cerebral edema, effacement of cortical sulci, and obliteration of the cisterns around the midbrain and subarachnoid space (20).

Laboratory Diagnosis: Cerebrospinal fluid (CSF) obtained via lumbar puncture is crucial for diagnosis. CSF examination typically reveals a grey to yellow-white, or red color. The red blood cell count is significantly elevated, reaching up to 24,600 cells/mm³. The polymorphonuclear leukocyte count can range widely from 300 to 26,000 cells/mm³. Protein concentration in the CSF is increased, ranging from 100 to 1,000 mg/100mL, while glucose concentration is significantly reduced, often falling to 10 mg/100mL or even lower. Naegleria fowleri trophozoites may be detected in the CSF (5).
CSF stained with Giemsa-Wright or trichrome stain reveals the presence of amoeba. Cultivation of N. fowleri involves transferring a few drops of CSF into a non-nutrient or low-nutrient agar plate seeded with Enterobacter aerogenes, Escherichia coli or other Gram-negative bacilli. Nelson's growth medium is most recommended consisting of:
Page'saline (0.4mgMgSO4+ 0.4mgCaCl2+ 14.2mgNa2HPO4+ 13.6mgKH2PO4+ 12mgNaCl+ 100ml DW)

Liver infusion- 0.17 g

- Glucose-0.17 g
- Medium is autoclaved for 25 min at 121°C.
- Sterile, heat-inactivated, foetal calf serum is added.

Table 2. Nelson's Growth Medium Composition for Naegleria fowleri

Component	Details
Base Medium	Page's saline (0.4 mg $MgSO_4$ + 0.4 mg $CaCl_2$ + 14.2 mg Na_2HPO_4 + 13.6 mg KH_2PO_4 + 12 mg NaCl in 100 ml distilled water)
Liver Infusion	0.17 g
Glucose	0.17 g
Autoclaving	Medium is autoclaved for 25 minutes at 121°C
Supplement	Sterile, heat-inactivated fetal calf serum is added 1% peptone

Supplementing Nelson's medium with 1% peptone improves amoeba's growth. Culture is incubated at 37 °C and monitored every day throughout the week. Trophozoites develop within first three days and begin to encyst after 7 to 10 days. To transform trophozoite, flagellation can be done by combining one drop of amoebae culture or sedimented CSF with one ml

of DW for 1-2 h. Liquid encystment medium which is modified from Page's amoeba saline can also be used. It includes cultivation of amoeba in Nelson's growth medium supplemented with 10% fetal bovine serum and incubating at 37°C. Once trophozoites develop, wash 2 × 106 cells twice in Phosphate Buffer Saline (PBS) (pH 7.4). Then incubate in 24-well plates with 5 mL of encystment medium; pH 6.8 at 37°C.

Other tests:

- Microscopy
- Immunofluorescence assay (IF)
- Enzyme-linked immunosorbent assay (ELISA)
- Flow cytometry (FC)
- Reverse transcription polymerase chain reaction (RT-PCR)
- Clinical features+ recent H/o contact with water confirm infection with N. fowleri infection

Treatment:

Given the rapid progression and high fatality rate of Primary Amoebic Meningoencephalitis (PAM), prompt diagnosis and immediate treatment are critical for improving patient outcomes. Enhancing physician awareness of PAM and improving diagnostic capabilities are essential for enabling early intervention. A coordinated global response, encompassing public education, effective environmental management strategies, and robust scientific research efforts, is imperative to address this significant emerging threat effectively (21).

Currently, early treatment regimens for PAM often include fluconazole, amphotericin B, and oral rifampicin. In vitro studies have also demonstrated the inhibitory effects of antibacterials such as neomycin, roxithromycin, clarithromycin, rokitamycin, zeocin, hygromycin, and erythromycin on Naegleria fowleri. Notably, chlorpromazine has shown rapid and potent activity against N. fowleri trophozoites, exceeding that of fluconazole and amphotericin B in laboratory settings. Furthermore, drugs conjugated with metal nanoparticles exhibit significant potential for PAM treatment and warrant further in vivo evaluation. Recent research has indicated that amphotericin B (AmB) and nystatin (NYS) conjugated with silver nanoparticles (AgNp) demonstrate enhanced efficacy against N. fowleri compared to the drugs administered alone (21).

Prevention and Control:

Given the limitations of current treatments, preventative measures are paramount in avoiding PAM. Research into a DNA vaccine utilizing a lentivirus to express the N. fowleri Nfa1 gene is ongoing. As N. fowleri is susceptible to chlorine and is effectively inactivated at a concentration of one part per million, consistent chlorination of swimming pools and water parks is strongly recommended for PAM prevention (22). Amoeba-monitoring programs, such as the one developed by the South Australian High Commission, routinely assess residual chlorine levels and total coliform counts to detect the presence of N. fowleri in water sources. Therefore, meticulous chlorination of pools, water parks, domestic water supplies, medical instruments, and hygiene devices is crucial. Additionally, the use of boiled water for rinsing, flushing, or irrigating nasal passages is essential, and purified water should be used in neti pots. Avoiding recreational water activities such as swimming and diving, particularly during hot summer months, and the use of nose plugs are encouraged to prevent the amoeba from entering the nasal cavity (23).

CONCLUSION:

Naegleria fowleri is a pathogenic free-living amoeba that causes the devastating necrotizing and hemorrhagic meningoencephalitis known as PAM, with a high fatality rate of approximately 95% in infected individuals. This amoeba is prevalent in countries experiencing hot summers, where human contact with contaminated water is more likely. Infection initiates with the entry of the infective trophozoite form through the nostrils into the nasal passages, subsequently reaching the brain and causing central nervous system infection, typically leading to death within 3 to 7 days. Tissue damage results from a combination of contact-dependent and contact-independent mechanisms, as well as the host's immune response. PAM is characterized by its rapid progression and high mortality, often resulting in death within two weeks of initial exposure. Diagnosis is challenging yet critical for any chance of patient survival, with most cases currently diagnosed post-mortem. The development of safe and effective treatments remains a significant challenge, with current options including amphotericin B and other investigational drugs. Further in vitro and in vivo studies are urgently needed to identify more effective therapeutic strategies for PAM. At present, the implementation of robust prevention strategies offers the best approach to mitigating the risk of this deadly infection.

Continued research into this parasite is vital to enhance our understanding of its biology, improve diagnostic methods, and develop effective strategies for controlling, treating, and ultimately preventing this devastating pathogen.

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