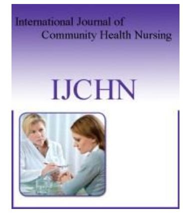
International Journal of Community Health Nursing



Journal homepage: www.mcmed.us/journal/ijchn

AMBOEA EATING BRAIN DISEASES

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Article Info
Received: 20/10/2025; Revised: 03/11/2025
Accepted: 01/12/2025

ABSTRACT

Struma ovarii is a rare ovarian teratoma characterized predominantly by thyroid tissue, comprising approximately 1–5% of ovarian teratomas and less than 1% of all ovarian tumors. While most cases are benign, a small percentage may undergo malignant transformation, typically to papillary or follicular thyroid carcinoma. Its clinical presentation can range from asymptomatic pelvic masses to features of hyperthyroidism due to functional hormone secretion. Diagnosis involves a combination of imaging, thyroid function assessment, and histopathological examination. Management primarily includes surgical intervention, with additional thyroidectomy and radioactive iodine treatment in malignant or metastatic cases. Prognosis is favorable, especially for benign tumors, though vigilant monitoring is essential due to rare recurrence or malignant progression. Awareness of this unique entity is crucial for improving diagnostic accuracy and optimizing patient outcomes.

Key words: Struma, ovarian teratoma, thyroid tissue, hyperthyroidism, malignant transformation, papillary thyroid carcinoma, follicular carcinoma, ovarian tumor, gynecologic oncology, thyrotoxicosis, ovarian mass, thyroid function.

INTRODUCTION

Amoeba eating brain disease, or Primary Amoebic Meningoencephalitis (PAM), is a rare but deadly infection caused by the amoeba Naegleria fowleri. It enters the body through the nose from contaminated warm freshwater and travels to the brain, causing rapid and severe damage.

Definition

The disease people refer to as "brain-eating amoeba" is Primary Amoebic Meningoencephalitis (PAM). It is caused by a microscopic organism called Naegleria fowleri, a free-living amoeba found in warm freshwater.

Naegleria fowleri Found in It usually lives in: Warm freshwater lake The official medical name for the resulting disease is Primary Amboea Meningoencephalitis (PAM). This is a rare, severe, and almost always fatal infection of the central nervous system the official medical name for the resulting disease is Primary Amoebic Meningoencephalitis (PAM).

Life cycle of Amboea brain eating disorder

N. fowleri exists as trophozoites (feeding / reproductive), flagellates (transient dispersal form), and cysts (environmental, hardy form). The trophozoite is the infectious and tissue-invading form. Human infection occurs almost exclusively via the nasal route when contaminated water is forcibly introduced into the nasal cavity (swimming, diving, water sports, or nasal irrigation). From the olfactory mucosa the organism migrates along olfactory nerve bundles into the olfactory bulbs and then deeper into cerebral tissue.

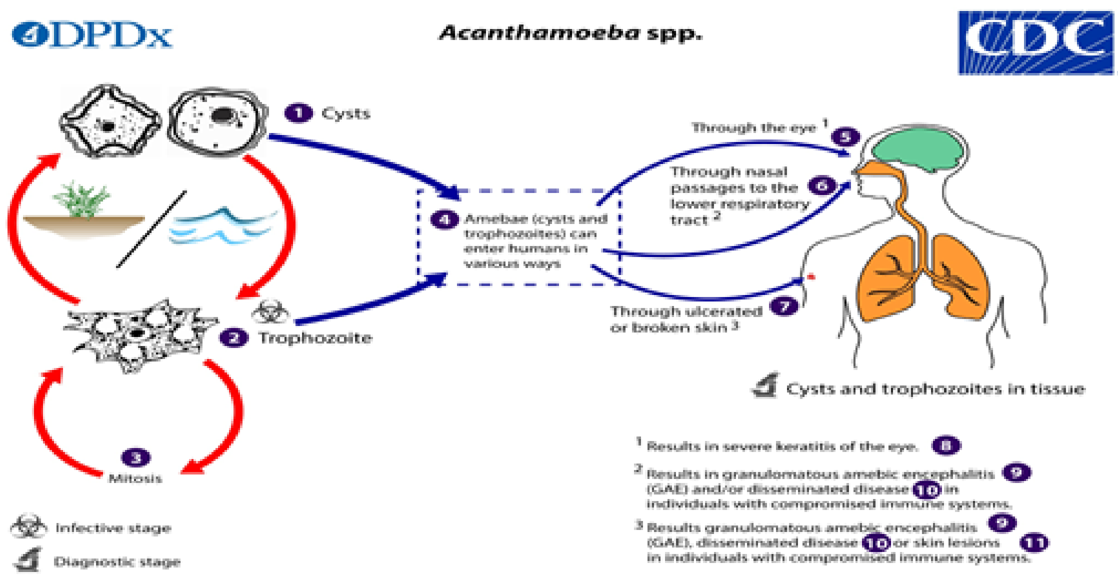

Figure 1: Life Cycle of Pam

Primary Amoebic Meningoencephalitis

Organism: Naegleria fowleri, a free-living, single-celled amoeba (a microscopic organism).

Habitat: It thrives in warm freshwater environments, especially during hot summer months. This includes lakes, rivers, hot springs, and improperly maintained swimming pools or hot tubs.

Infection Route: Infection occurs when water containing the amoeba is forcefully introduced into the nose, typically during activities like swimming, diving, or using unsterilized water for nasal rinsing

Crucially: You cannot get the infection by swallowing the water, and it is not spread from person

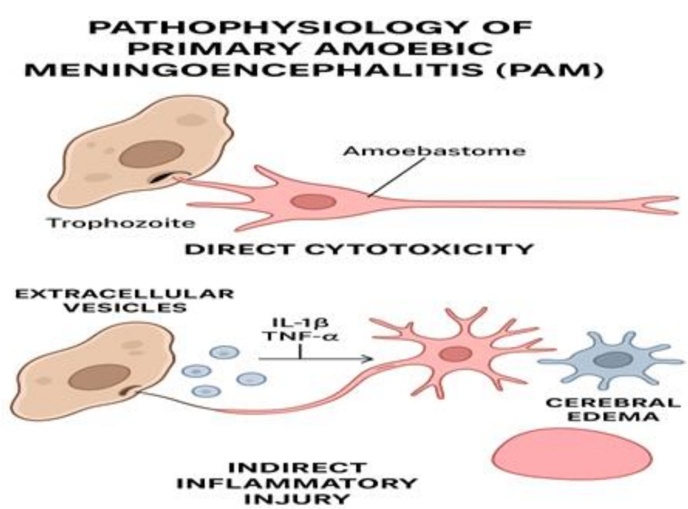

Figure 2: Pam

Pathophysiology of Primary Amboea Meningoencephalitis

Pathophysiology — stepwise, mechanistic view (core of review)

1. Initial attachment and mucosal traversal

After nasal exposure, trophozoites adhere to the olfactory epithelium. Adhesion is mediated by surface lectins and other adhesins that bind host glycoconjugates. Early tissue penetration requires both motility and enzymatic degradation of epithelial barriers.

2. Neural invasion via the olfactory nerve

The amoeba traverses the cribriform plate along olfactory nerve axon bundles to reach the olfactory bulbs.

This direct neural route partially bypasses systemic immunity and enables rapid CNS seeding — a key reason disease progresses so quickly.

3. Contact-dependent cytolysis (direct trophozoite effects)

Once in neural tissue, trophozoites cause direct cell injury through multiple factors: Hydrolytic proteases (including cysteine proteases and metalloproteinases) that degrade extracellular matrix and host cell membranes.

Phospholipases and pore-forming activities that disrupt neuronal and glial cell membranes. Surface molecules and amoebas tomes (food-cup like structures) can phagocytose host cells. These contact-dependent mechanisms produce focal necrosis and hemorrhage.

4. Contact-independent mechanisms: secreted factors & extracellular vesicle

Recent work shows trophozoites release soluble cytotoxic factors and extracellular vehicles (EVs) that can degrade tight junctions and promote paracellular invasion and distant tissue damage without direct contact. EVs may carry proteases, lipids, and regulatory molecules that modulate host responses and facilitate deeper spread. This dual mechanism (contact-was dependent + contact-independent) broadens the tissue injury footprint.

5. Host immune response and secondary injury the innate immune response (neutrophils, microglia, macrophages) is rapidly recruited but is often ineffective at clearing trophozoites. Instead, a vigorous inflammatory response with cytokine and chemokine release (e.g., IL-1β, TNF-α) contributes to cerebral edema, increased intracranial pressure, and secondary neuronal damage. This combination of direct amoebic cytotoxicity and immune-mediated injury explains the rapid necrotizing meningoencephalitis and often fatal cerebral edema.

6. Pathological hallmark: Gross and histopathology show extensive hemorrhagic necrosis centered in olfactory bulbs and frontal lobes early on, with trophozoites present at lesion margins. Widespread necrosis, neutrophilic infiltrates, and vascular congestion are typical.

INCUBATION PERIOD

Incubation typically 1–9 days post-exposure.

SIGN AND SYMPTOMS

Early symptoms are nonspecific (headache, fever, nausea), then rapidly progress to stiff neck, photophobia, altered mental status, seizures, and coma.

Rapid deterioration over days is characteristic — clinicians should suspect PAM in any rapidly progressive meningoencephalitis with recent warm-freshwater exposure or nasal irrigation with non-sterile water.

Diagnosis

CSF: often shows neutrophilic pleocytosis, high protein, low glucose — resembling bacterial meningitis. Crucially, wet mount microscopy of fresh, non-refrigerated CSF can reveal motile trophozoites; this is the fastest presumptive test. Confirmation is via PCR and immunofluorescence assays at reference labs (e.g., CDC). Neuroimaging shows hemorrhagic necrosis but is nonspecific. Early suspicion and direct CSF exam dramatically increase the chance of timely diagnosis.

Treatment and outcomes

1. Amphotericin B (intravenous and intrathecal) has been the mainstay.
2. miltefosine (an antileishmanial with amoebicidal activity) has been used in combination with amphotericin B, azoles (fluconazole/ketoconazole/posaconazole), rifampin, and aggressive management of intracranial pressure.

Prevention and Measures

1. Avoid forcing warm freshwater up the nose; use nose clips or avoid submerging the head in warm stagnant water.
2. For nasal irrigation, use sterile, boiled (and cooled), or appropriately filtered water — not tap or unboiled well/river water.
3. Proper pool maintenance and chlorination reduce risk. Public health surveillance, water system monitoring, and clinician awareness are central to mitigation.
4. Recent outbreak reports emphasize the importance of water quality and safe nasal practices.

CONCLUSION

Naegleria fowleri infection causes a rapidly progressive, often lethal meningoencephalitis through a combination of direct trophozoite cytotoxicity (proteases, phospholipases, pore-forming activities), paracrine injury via extracellular vesicles and secreted factors, and a harmful host inflammatory response. Early clinical suspicion after nasal freshwater exposure, rapid CSF examination, swift molecular confirmation, and immediate combination therapy offer the best—but still limited—chance of survival. Continued research into diagnostics, therapeutics, and prevention strategies is urgently needed

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