

Free-Living Amebas: *Naegleria*, *Acanthamoeba* and *Balamuthia*

Three genera of free-living amebas, *Naegleria*, *Acanthamoeba*, and *Balamuthia* are known to infect humans. *Naegleria fowleri* causes an acute and almost fatal encephalitis, which, fortunately, is rare. Several species of *Acanthamoeba* and *B. mandrillaris* can cause lung and skin infections, as well as encephalitis, in immunocompromised patients. In addition, *Acanthamoeba* may cause an ulcerative keratitis, which is usually associated with improper sterilization of soft contact lenses. These amebas live freely in soil and in fresh and coastal waters. The resistant cysts can be transported in dust.

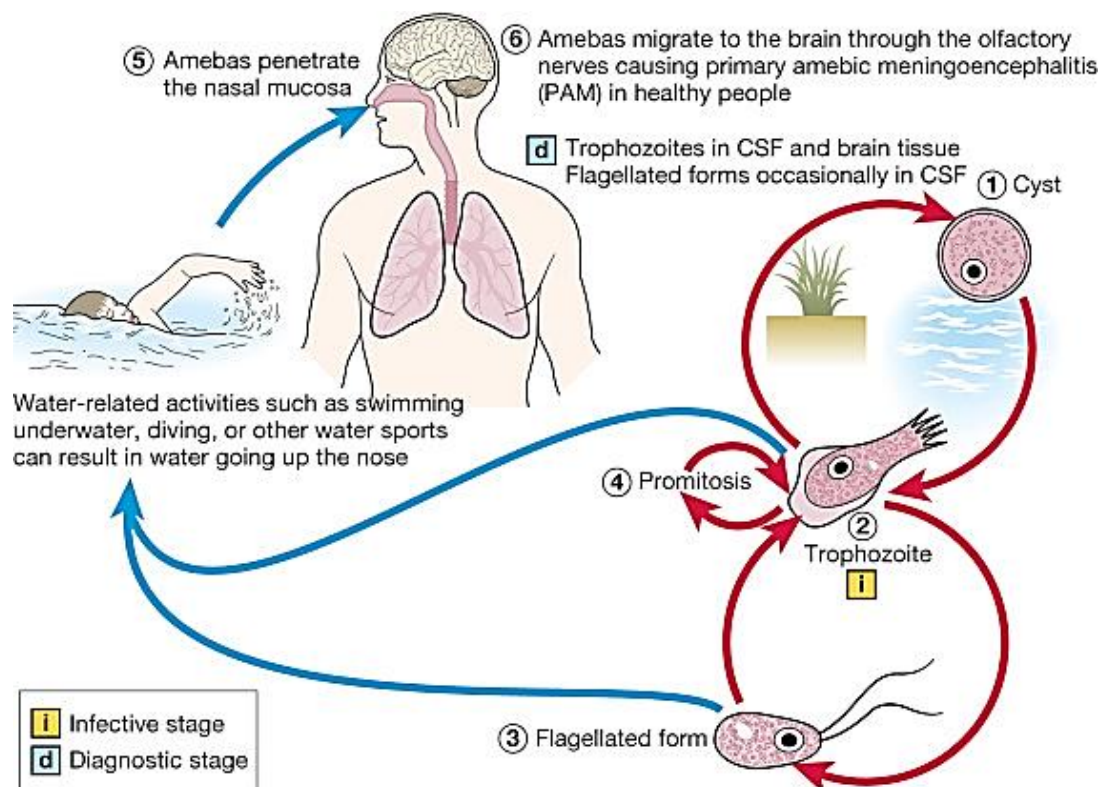
Naegleria fowleri

Structure

The trophozoites are 10 to 15 μm in diameter and produce broadly rounded lobopodia. Cysts are single-walled, spherical and 8 to 12 μm in diameter. The trophozoites can also transform to a flagellated form.

Multiplication and Life Cycle

The trophozoites are free-living inhabitants of soil and warm fresh water. They reproduce by binary fission.



Pathogenesis and Clinical manifestation

Amebas splashed or inhaled onto the olfactory epithelium migrate up the olfactory nerve to the brain and spread via the subarachnoid space. Clinical manifestation *Naegleria fowleri* causes primary amebic meningoencephalitis, a rare, rapidly fatal disease with sudden onset of headache, fever, stiff neck, lethargy, and coma in otherwise healthy people.

Epidemiology

The organism is found worldwide in soil and warm fresh water. Infectious cysts may be carried in dust.

Diagnosis

Diagnosis

Primary amebic meningoencephalitis cannot be distinguished clinically from acute pyogenic or bacterial meningoencephalitides. The disease usually occurs in children and young adults in good health who have recently swum in warm water. Computed tomography of the brain shows obliteration of the cisternae surrounding the midbrain and of the subarachnoid space over the cerebral hemispheres. The disease may be

diagnosed rapidly by examining one or two drops of fresh cerebrospinal fluid under a light microscope for *N. fowleri*. The organism may also be cultured from cerebrospinal fluid or brain tissue for a definitive diagnosis. Diagnoses have been made by examining paraffin-embedded brain tissue sections stained with hematoxylin and eosin..

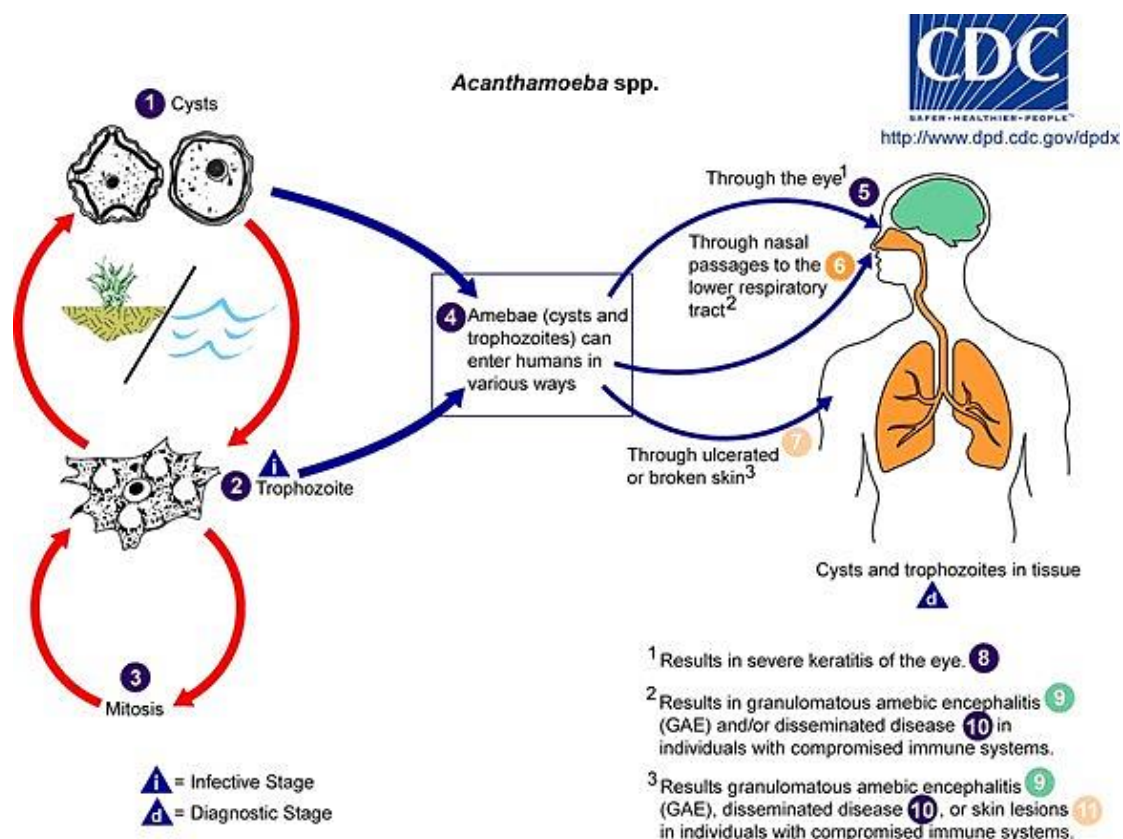
Acanthamoeba Species

Structure

Acanthamoeba castellanii: trophozoites are 25 to 40 μm in diameter with characteristic spine-like pseudopodia. Cysts are double-walled, usually polygonal and spherical, and 15 to 20 μm in diameter.

Multiplication and Life Cycle

The trophozoites are free-living inhabitants of soil and of fresh and salt water. They reproduce by binary fission.



Pathogenesis and Clinical Manifestation

Encephalitis is caused by the hematogenous spread from superficial or pulmonary lesions to the brain. Keratitis results from contamination of superficial corneal scrapes. Clinical Manifestation, *Acanthamoeba* species

and *Balamuthia mandrillaris* usually act as opportunistic pathogens in immunocompromised or debilitated individuals in whom they cause pneumonitis or dermal ulcerations. From these lesions the amebas may spread to the brain to cause slowly progressive, and usually fatal encephalitis called granulomatous amebic encephalitis. In healthy individuals, *Acanthamoeba* spp can cause an ulcerating keratitis, which is often associated with the use of improperly sterilized contact lenses.

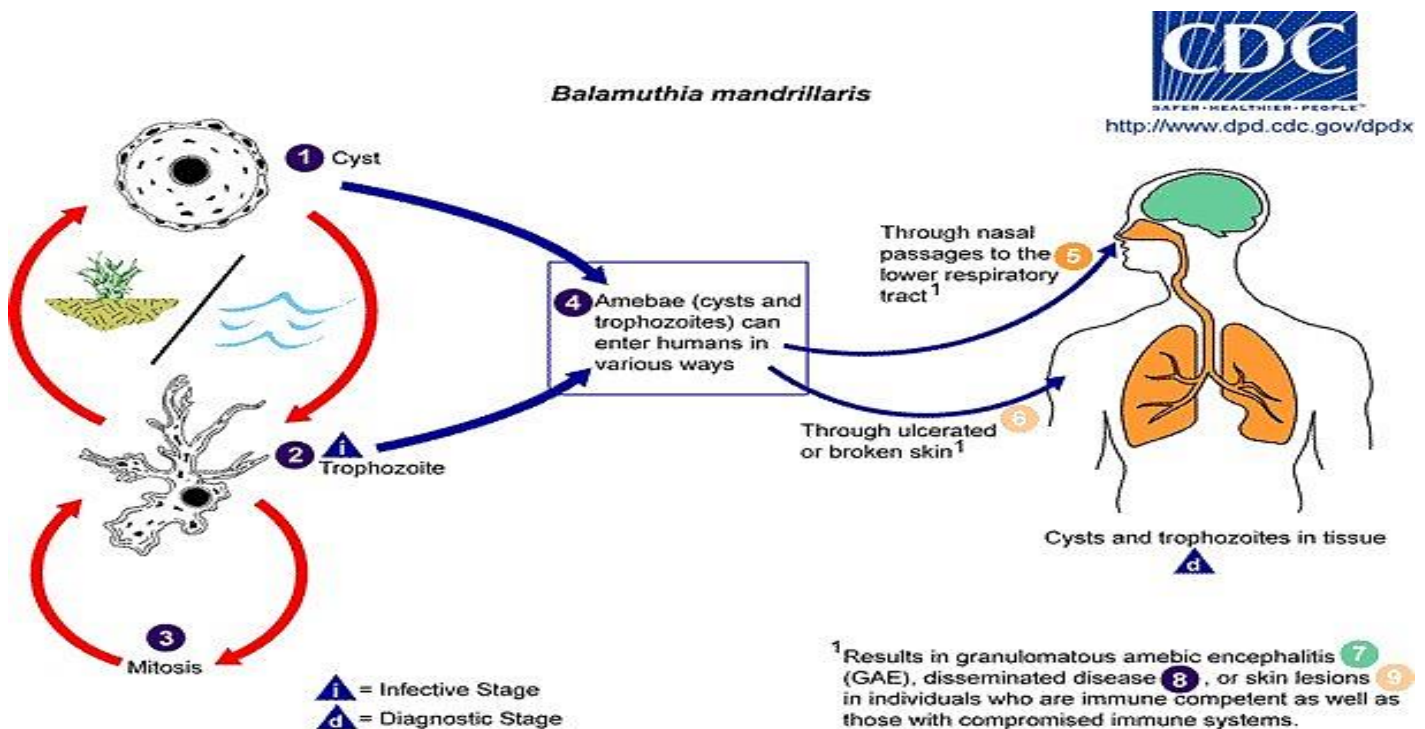
Epidemiology

Acanthamoeba and *B. mandrillaris* organisms live worldwide in soil and fresh and salt water. They may contaminate contact lens solution, physiotherapy pools, air-conditioning units, etc.

Diagnosis

In many cases, granulomatous amebic encephalitis is not diagnosed until after or, at best, shortly before death. Immunosuppression or other predisposing factors may provide important clues. Computed tomography and magnetic resonance imaging of the brain are important diagnostic tests, as is examination of cerebrospinal fluid and brain biopsy specimens. The diagnosis usually is made after examination of brain tissue with light a microscope. Amebic “dermatitis” is often diagnosed by microscopic examination of a skin biopsy. Both trophozoites and cysts are usually visible.

In the case of amebic keratitis, scrapings of the corneal ulceration and biopsy specimens may contain amebic trophozoites and cysts. Both light and electron microscopy may be useful. Amebic cysts in the corneal stroma may be demonstrated by staining with hematoxylin and eosin, trichrome, calcofluor-white, or immunofluorescence techniques. Alternatively, amebas may be cultured at 37°C on non-nutrient agar with Page's saline containing *Escherichia coli*, *Enterobacter aerogenes*, or other Gram-negative bacteria. Cysts and trophozoites may be identified on the basis of morphology and locomotion; isoenzyme electrophoresis may be used to further classify species.



Blastocystis hominis

was previously considered as a yeast, but recently it has been reclassified as a protozoan.

Habitat

It is a strict anaerobic protozoa found in large intestine of humans.

Morphology

B. hominis has 3 morphological forms:

Vacuolated form

is usually seen in stool specimen. It measures 8 µm in diameter and is characterized by its large central vacuole, which pushes the cytoplasm and the nucleus to the periphery. It multiplies by binary fission.

Amoeboid form

is a polymorphous cell slightly larger than the vacuolated form occasionally seen in the feces. It multiplies by sporulation.

Granular form

measures 10–60 µm in diameter and is seen exclusively in old cultures.

Pathogenicity and Clinical Features

The pathogenicity of *B. hominis* is doubtful its believed to cause the irritable bowel syndrome. However, recent studies have shown the parasite to be associated with diarrhea. Clinical manifestations include diarrhea, abdominal pain, nausea, vomiting, fever, and chills. More than half of the patients suffering from infection with *B. hominis* have been found to be immunologically compromised.

Diagnosis

The condition is diagnosed by demonstration of the organism in stool smear stained by Giemsa or iron hematoxylin or Trichrome stains.

