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Granulomatous Amebic Encephalitis (GAE): A severe disease.

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Commentary

GAE caused by *Acanthamoeba spp.* is more common in chronically sick, debilitated people, immunocompromised people, such as those with HIV/AIDS, and people who have taken broad-spectrum antibiotics or chemotherapy drugs. Personality changes, headaches, low-grade fevers, nausea, vomiting, lethargy, diplopia, hemiparesis, seizures, decreased levels of awareness, and coma are some of the clinical symptoms. Some individuals may have palsies of the third and sixth cranial nerves. GAE might be mistaken for bacterial, tuberculous, or viral meningitis. GAE is more common in chronically sick, debilitated people, whether caused by *Acanthamoeba spp.* or *B. mandrillaris*. Many have had immunosuppressive therapy, have AIDS, or have been prescribed broad-spectrum antibiotics or chemotherapy drugs. The incubation time is unclear, and the sickness may take many weeks or months to manifest. Hematogenous invasion and penetration into the brain occurs as a result of dissemination from the original target, which is most likely the lower respiratory tract or the skin.

Acanthamoeba Keratitis (AK) is a persistent corneal infection linked to contact lens use, corneal abrasion or hypoxic damage, and contaminated water exposure. GAE caused by *Acanthamoeba* was previously only diagnosed after autopsy. Because *Acanthamoeba spp*. have very rarely been isolated from the CSF or detected on wet mount, brain biopsy is the most accurate diagnosis method; both an immunohistochemical stain and PCR testing are available to help in the examination of brain tissue. *Acanthamoeba* exposure causes measurable serum antibodies, although it's unclear if this leads to protective immunity. Although high titers to *Acanthamoeba* were recently utilised as evidence of *Acanthamoeba* GAE in an immunocompetent patient, serologic tests are typically not helpful for diagnosis. Antibodies to *Acanthamoeba* have been found in the sera of people who haven't had any signs of the disease, as well as in individuals who have had GAE, skin infections, or both.

With or without contrast enhancement, neuroimaging often reveals single or many space-occupying lesions in the brain. Toxoplasmosis, primary CNS lymphoma, TB, neurocysticercosis, nocardiosis, aspergillosis, bacterial brain abscess, and GAE related to Balamuthia are among the differential diagnoses in these individuals. These organisms can't be distinguished reliably on radiography, thus tissue staining with *Acanthamoeba*-specific antibodies is necessary for a definite diagnosis. *Acanthamoeba* skin infections are common among GAE patients. In individuals suspected of having GAE, skin nodules or ulcers should be biopsied and tested for *Acanthamoeba*. Brain and skin biopsy tissues have been successfully cultivated with *Acanthamoeba. Acanthamoeba spp.* have been found in brain tissue by PCR, including formaldehyde-fixed and paraffin-embedded tissue. Early diagnosis and therapy are critical for effective AK treatment. Although *in vivo* confocal microscopy for cyst visualisation is becoming more commonly used for the diagnosis of AK, it still relies on a high clinical suspicion and the demonstration of *Acanthamoeba* in corneal scrapings or biopsy specimens by histopathologic examination, culture, or molecular mechanisms such as PCR or DNA probes. Even with therapy, *Acanthamoeba* infection of the CNS is typically deadly, with only a few documented survivors, all of whom experienced lasting neurological impairments. The time of diagnosis, the virulence and sensitivity of the *Acanthamoeba* strain, and most importantly the immunological state of the afflicted person all have a role in the prognosis.

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