DRUG INFORMATION ALERT

Brain-Eating Amoeba (aka *Naegleria fowleri)*: What A Pharmacist Needs To Know

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A rare amoeba has taken the life of yet another person this summer. *Naegleria fowleri*, commonly referred to as "the brain-eating amoeba", is a thermophilic, unicellular organism that resides in soil and freshwater. This particular species is the only amoeba in its genus that has the ability to infect humans.[1] Although rare (~200 reported cases worldwide), this fulminant infection is almost always fatal due to primary amebic meningoencephalitis.[1-4]

N. fowleri infections acutely present as headache, fever > 38.2°C (101°F), nausea, vomiting, and stiff neck. Other common symptoms include mental status changes such as lethargy, stupor, disorientation, confusion, delirium, obtundation, restlessness, irritability and combativeness. As the infection progresses, other symptoms become apparent such as photophobia, anorexia, sleep disturbances, sore throat, rhinitis, diplopia, blurred vision, seizures, hallucinations, ageusia, parosmia, and hearing difficulties. It is imperative to direct patients complaining of any combination of the symptoms described above to the nearest acute care facility for immediate evaluation. In most reported cases, patients have died within 48 to 72 hours of symptom presentation. However, it has taken seven to twelve days for some patients to expire.[2, 5] One rapidly-identified case was managed with a combination of intravenous amphotericin B and fluconazole and resulted in successful treatment.[1] Those few patients who have survived have received a combination of systemic and intrathecal amphotericin B, and some patients received concurrent therapy with other antimicrobials including systemic and intrathecal miconazole, fluconazole, rifampin, or sulfisoxazole.[6] Once infected, *N. fowleri* infections are not transmitted from one person to another.[5]

Given its primary environment, *N. fowleri* is more common in the warm summer months, with 85% of reported cases occurring from June to September. The organism is typically contracted from warm, free-standing freshwater and can also be found in soil, geothermal water sources, thermal discharges of industrial plants, heated pools with poor chlorination, water heaters $\leq$47°C (116°F), aquariums, and sewage. *N. fowleri* has a three-stage life cycle. The cyst form resides in water and soil. This stage can progress to the trophozoite form that subsequently infects humans by entering the body through the nose and migrating to the brain via the olfactory nerve. Once in the brain, the trophozoite

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begins to lyse red bloods cells and leukocytes, as well as other cells, leading to hemorrhagic necrosis. The trophozoite form can revert between a non-feeding flagellated form and itself. The trophozoite form is most commonly found in the cerebrospinal fluid while the flagellated form is uncommonly found.[1, 5-7]

While *N. fowleri* infections, and subsequent primary amebic meningoencephalitis, are incredibly rare, the incidence of primary amebic meningoencephalitis has increased in recent years. *N. fowleri* was first successfully linked to primary amebic meningoencephalitis in 1937. As of 2007, there have been more than 120 reported cases in the U.S. Males account for most of the reported cases.[3, 7] This summer alone (Summer 2011), three immunocompetent individuals have died: a 16-year-old female in Florida, a 9-year-old boy in Virginia, and a 20-year-old man in Louisiana. The girl is suspected to have contracted the disease after swimming in fresh water, the boy after attending a fishing camp, and the man after using a neti pot filled with improperly disinfected water. The incidence of *N. fowleri* infections is more common in southern states, including Texas. Since 2000, ten cases have been reported in Texas, including the 2010 death of a 7-year-old boy and the 2007 deaths of a 12-year-old boy and 22-year-old man. These Texas deaths occurred after exposure to freshwater.[3, 8] The highest annual U.S. infection rate occurred in 1980 when eight cases were reported.[7]

Since primary amebic meningoencephalitis is a rare disease, health care providers have a low index of suspicion for diagnosing this condition.[3] Pharmacists, as the most readily accessible health care provider, should be well-informed about the signs, symptoms, and surrounding circumstances associated with this infectious agent so they are able to answer patient questions and quickly refer patients to acute care facilities as necessary.

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