Figure. (A) Hemorrhagic meningoencephalitis due to *Naegleria fowleri*. Subarachnoid space (top) is expanded by inflammatory infiltrate and amoeba. Cortex demonstrates perivascular infiltrates and hemorrhage. Hematoxylin and eosin; $\times 100$. (B) *N. fowleri* invading the Virchow–Robin space. Hematoxylin and eosin; $\times 400$. (Inset, B) Two *N. fowleri* organisms are demonstrated, each 15 μm in diameter. A foamy microvesicular nucleus and large eosinophilic nucleolus are seen. Hematoxylin and eosin; original magnification $\times 1,000$. (C) Immunofluorescence profile of brain tissue demonstrates reactivity of anti-*N. fowleri* antibodies to *N. fowleri* trophozoites.

***Naegleria fowleri* meningoencephalitis**

Darin T. Okuda, MD; and Stephen Coons, MD

A previously healthy 5-year-old boy presented with severe headache and neck stiffness. On examination, he exhibited somnolence and nuchal rigidity. The results of CSF studies were as follows: red blood cells, $400/\text{mm}^3$; white blood cells, $1,150/\text{mm}^3$ (91% polymorphonuclear cells); glucose level, 49 mg/dL; protein

level, 108 mg/dL; and the presence of living amoeba. MRI of the brain revealed diffuse high T2 signal intensities within the sulci and cerebellar folia. During the next 24 hours, his medical and neurologic status declined rapidly. Death occurred from brainstem compression due to cerebellar swelling. Postmortem examination of brain tissue was consistent with the diagnosis of *Naegleria fowleri* hemorrhagic meningoencephalitis (figure).¹

The Centers for Disease Control confirmed the presence of *N. fowleri* in one of the city water tanks providing service to the patient's home.²

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