■ ETIOLOGY

Neurocysticercosis is the most common parasitic disease of the CNS worldwide. Humans acquire cysticercosis by the ingestion of food contaminated with the eggs of the parasite *T. solium* **(Chap. 235)**. Toxoplasmosis **(Chap. 228)** is a parasitic disease caused by *T. gondii* and acquired from the ingestion of undercooked meat and from handling cat feces.

■ CLINICAL PRESENTATION

The most common manifestation of neurocysticercosis is new-onset partial seizures with or without secondary generalization. Cysticerci may develop in the brain parenchyma and cause seizures or focal neurologic deficits. When present in the subarachnoid or ventricular spaces, cysticerci can produce increased ICP by interference with CSF flow. Spinal cysticerci can mimic the presentation of intraspinal tumors. When the cysticerci first lodge in the brain, they frequently cause little in the way of an inflammatory response. As the cysticercal cyst degenerates, it elicits an inflammatory response that may present clinically as a seizure. Eventually the cyst dies, a process that may take several years and is typically associated with resolution of the inflammatory response and, often, abatement of seizures.

Primary *Toxoplasma* infection is often asymptomatic. However, during this phase, parasites may spread to the CNS, where they become latent. Reactivation of CNS infection is almost exclusively associated with immunocompromised hosts, particularly those with HIV infection. During this phase, patients present with headache, fever, seizures, and focal neurologic deficits.

■ DIAGNOSIS

The lesions of neurocysticercosis are readily visualized by MRI or CT scans depending on the stage of the lesion. There are four stages of neurocysticercosis: (1) the vesicular stage, (2) the colloidal stage, (3) the granulonodular stage, and (4) the nodular-calcified stage. Lesions with viable parasites appear as cystic lesions, and the scolex can often be visualized on MRI. Cystic lesions with small