stage. ***E.*** Postcontrast T1-weighted imaging demonstrates enhancing lesions in the left putamen and in the genu of the internal capsule near the foramen of Monro. ***F.*** These lesions demonstrate no surrounding edema on FLAIR imaging, typical for this stage of the disease. ***G.*** The nodular-calcified stage. Computed tomography scan demonstrates typical parenchymal brain calcifications. *(Courtesy of Aaron Kamer, MD; with permission.)*

MRI findings of toxoplasmosis consist of multiple lesions in the deep white matter, the thalamus, and basal ganglia and at the gray-white junction in the cerebral hemispheres. With contrast administration, the majority of the lesions enhance in a ringed, nodular, or homogeneous pattern and are surrounded by edema. In the presence of the characteristic neuroimaging abnormalities of *T. gondii* infection, serum IgG antibody to *T. gondii* should be obtained and, when positive, the patient should be treated.

TREATMENT

Infectious Focal CNS Lesions

Anticonvulsant therapy is initiated when the patient with neurocysticercosis presents with a seizure. There is controversy about whether or not anthelmintic therapy should be given to all patients, and recommendations are based on the stage of the lesion. Cysticerci appearing as cystic lesions in the brain parenchyma with or without pericystic edema or in the subarachnoid space at the convexity of the cerebral hemispheres should be treated with anticysticidal therapy. Cysticidal drugs accelerate the destruction of the parasites, resulting in a faster resolution of the infection. Albendazole monotherapy is recommended for patients with one to two parenchymal cysts. The dose of albendazole is 15 mg/kg per day in two daily doses for 10–14 days. A combination of albendazole plus praziquantel is recommended for patients with more than two viable cysts. Viable cysts are defined as those in the vesicular or colloidal stages (see above). The recommended dose of praziquantel is 50 mg/kg per day for 10–14 days. Prednisone or dexamethasone is begun prior to anticysticidal therapy to reduce the host inflammatory response to degenerating parasites. Only cysts in the vesicular stage, where the cyst contains living larva