

22. Immunocompromise

The availability and efficacy of highly active anti-retroviral treatment has rendered the CNS manifestations of HIV—HIV encephalitis, progressive multifocal leukoencephalopathy (PML), and toxoplasmosis—less common. The recognition of these entities on MRI is nevertheless important, HIV encephalitis being the most frequent. In HIV encephalitis there can be focal or diffuse hyperintensity of subcortical and deep white matter on T2WI (Fig. 22.1 A, B). The areas of involvement are poorly visualized on T1WI (Fig. 22.1. C) and rarely enhance. With treatment, the signal intensity changes in the white matter may resolve, however atrophic changes typically remain. Such atrophy is in fact the most common MRI finding in HIV encephalitis, demonstrated in Figure 22.1 C along with the loss of gray-white matter differentiation, another common finding. DTI (diffusion tensor imaging) changes (decreased fractional anisotropy) may precede those of conventional MRI. Spectroscopic findings in HIV-associated neurocognitive disorders (AIDS dementia complex) include a decrease in NAA (from neuronal loss), an increase in choline (from

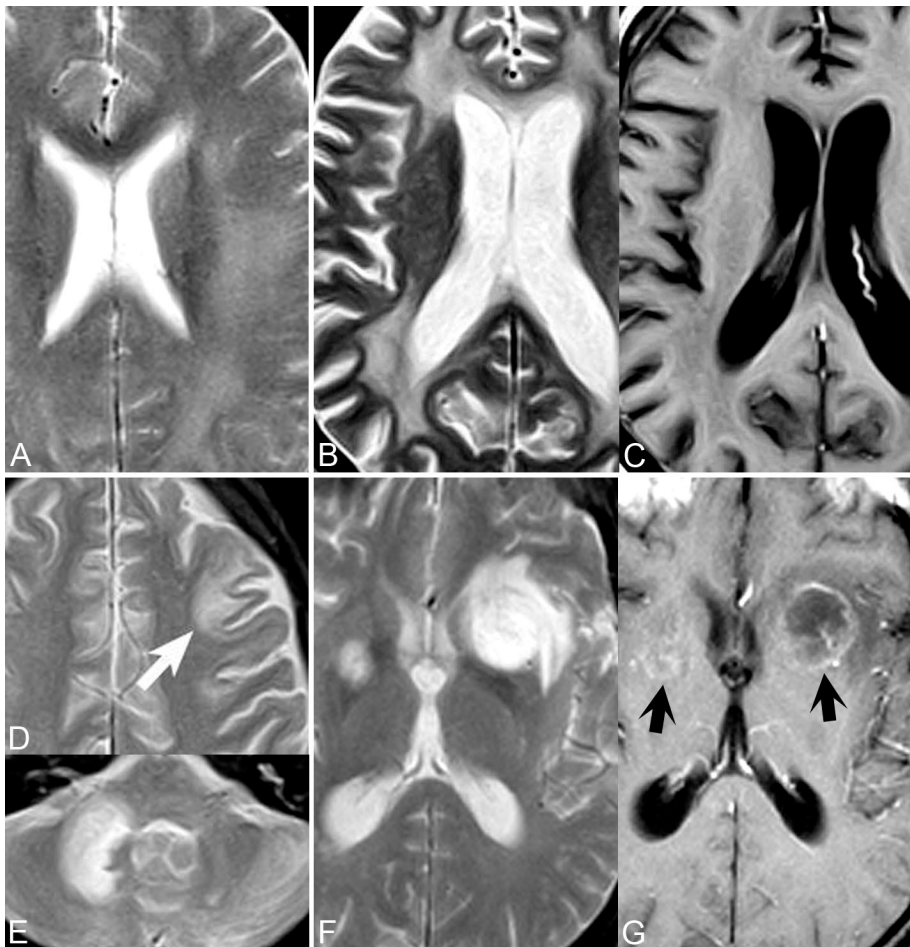


Fig. 22.1

membrane turnover), and increased myo-inositol (a marker for neuroglial activation). PML (progressive multifocal leukoencephalopathy) is another CNS disease associated with immunosuppression and AIDS. It is caused by the JC polyomavirus which infiltrates and destroys oligodendrocytes while largely sparing the axons. Without treatment, PML is a rapidly progressive, fatal disease. Initial clinical findings include focal neurologic defects, in contrast to the diffuse encephalopathy seen in HIV encephalitis. MR findings mirror this relationship, PML being more asymmetric, presenting with a unifocal or multifocal pattern localized to the parietal or frontal lobes (Fig. 22.1 D) as opposed to the diffuse, more symmetric pattern of HIV encephalitis. PML may occasionally involve the brainstem or cerebellum (Fig. 21.1 E). The demyelinated lesions of PML appear as high SI on T2WI (Fig. 22.1 D, E, white arrow) and low SI on T1WI. These lesions typically do not enhance. In PML DWI may reveal central areas of increased diffusion, correlating with necrosis and reduced cellularity, and areas of mild restricted diffusion peripherally, correlating with ongoing tissue injury.

Toxoplasmosis is the most common intracranial opportunistic infection in HIV and is caused by an obligate intracellular protozoan transmitted through insufficiently cooked meat and cat feces. Clinical features in immunocompromised adults include fever, headache, seizures, mental status changes, and focal neurologic signs. MRI evidence of disease is commonly seen in the thalamus, basal ganglia, and gray-white matter junctions of the frontal and parietal lobes. Figure 22.1 F demonstrates the characteristic appearance of toxoplasmosis on T2WI consisting of multiple hyperintense lesions with surrounding peripheral edema. A contrast enhanced T1WI (Fig. 22.1 G) reveals faint rim enhancement (black arrows)—indicative of active disease—surrounded by low SI edema. Differential considerations for toxoplasmosis include necrotic metastases, pyogenic abscesses, and lymphoma. Unlike these, toxoplasmosis demonstrates increased central diffusion, likely from the decreased immune response within the lesion. Metastases and pyogenic abscesses may also be differentiated on the basis of clinical history, although CNS lymphoma, which frequently occurs in HIV patients, may prove more difficult. Lymphoma however does typically present with solitary lesions that are larger, with less edema, and that enhance with a more irregular rim than those of toxoplasmosis. The inability to distinguish these two on imaging may necessitate empiric treatment for toxoplasmosis with MRI followup. Neonatal toxoplasmosis is associated with the triad of chorioretinitis, intracranial calcifications, and hydrocephalus.