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Imaging of the brain in the HIV-positive child

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Abstract The prevalence of human immune-deficiency virus (HIV) infection around the world, coupled with increasing population movement, make it likely that many physicians will treat HIV-infected patients. New treatment protocols for the specific manifestations of acquired immune-deficiency syndrome (AIDS) make distinguishing the different neurological diseases of great importance. The pattern of disease in children differs from those of adults both in its distribution and etiology. This article encapsulates the salient aspects relating to the imaging of the brain in HIV-positive children, paying particular attention to recent advances and the different features of the various pathological conditions affecting the HIV-infected brain in children.

Introduction

Despite the presence of improved screening and drug regimens for pregnant females that reduce vertical human immune-deficiency virus (HIV) transmission, a significant number of HIV patients are in the pediatric age group. This population is further increased by the antiretroviral regimens that are increasingly effective in prolonging life [1]. The wider use of CT, MR, and nuclear medicine studies in diagnosing the manifestation of HIV, combined with the presence of effective treatment agents for these symptoms, requires the radiologist to be familiar with the nuances of HIV in the pediatric patient.

HIV encephalopathy

HIV encephalopathy is a broad term that refers to the clinical deterioration of higher functions and associated white matter disease and cerebral atrophy. As both cerebral atrophy and white matter disease are the radiological manifestation of clinical encephalopathy, they will be discussed in this section.

HIV encephalopathy is divided into two types: (1) progressive encephalopathy which is comparable to the adult acquired immune-deficiency syndrome (AIDS) dementia complex [2] and refers to the step-wise deterioration of mental status and higher functioning of the child. It is associated with severe immunodeficiency [3–6]; (2) static encephalopathy where the child has better higher functions, but does not keep up with the age-appropriate milestones [7].

Diffuse atrophy (Fig. 1) and bifrontal white matter abnormalities on MRI (Fig. 2) are particularly common in the setting of HIV encephalopathy. The severity of clinical encephalopathy is related to the extent of white matter involvement and cerebral atrophy. Mild atrophy is associated with static encephalopathy, while severe atrophy is associated with progressive encephalopathy [3, 8]. The extent of atrophy may reverse with antiretroviral administration [9–11].

Disorders in the white matter, which are thought to be due to alteration in the blood-brain barrier [12, 13],

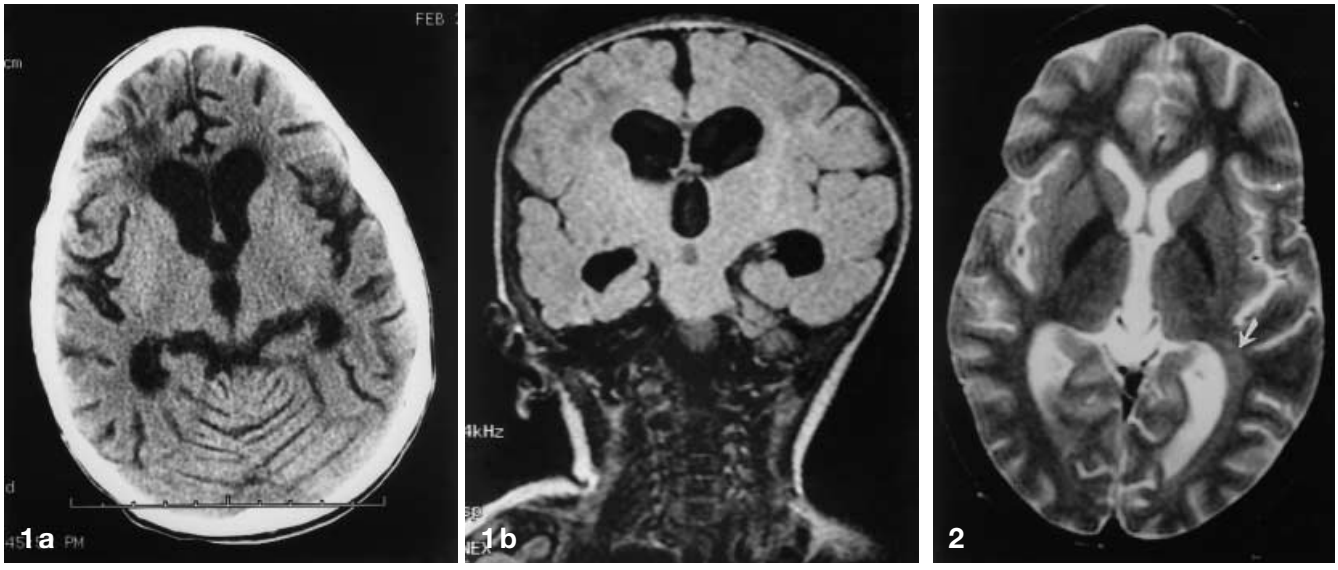

Fig. 1 Axial CT (**a**) and coronal T1-weighted MR (**b**) images demonstrate parenchymal atrophy with ventricular dilatation out of proportion to sulcal atrophy in an HIV positive child

Fig. 2 Axial T2-weighted MR image in an HIV-positive child demonstrates non-specific abnormal T2 signal in the left periatrial white matter. A combination of gliosis and edema secondary to the HIV virus or the papovirus associated with PML are the most likely causes

are a sign of the advanced disease and affect up to 44 % of individuals [14, 15], while cerebral atrophy occurs in 57 % to 86 % of patients [16–19]. The United States Public Health Service recommends that a baseline study be obtained upon confirmation of the diagnosis of HIV. This will document both deterioration and response to therapy.

There are three broad patterns of cerebral atrophy recognized:

1. Central atrophy shows ventriculomegally to a disproportionate extent as compared with cortical atrophy. This is due to the preferential tropism of the virus for the basal ganglia [20], causing necrosis and subsequent atrophy in that region [21–24] (Fig. 1 a,b).
2. Generalized atrophy that affects particularly the frontal lobe [25].
3. Necrotizing encephalopathy that causes encephalomalacia and is associated with dilated cardiomyopathy of AIDS [26, 27].

The extent of cerebral atrophy can be assessed on CT or MRI and, with newer software, can be volumetrically measured rather than visually estimated.

Table 1 Classical anatomic distribution of disease states in the HIV-positive child

Predominant anatomic region	Disease state
Arachnoid villi and fourth ventricle outflow tract	Bacteria, mycobacteria and fungi cause blockage and hydrocephalus
Basal ganglia	Arteriopathy with stenosis, atrophy, calcifications, lymphoma, toxoplasma
Centrum semiovale	White matter degeneration
Cerebellum	Calcifications
Circle of Willis	Arteriopathy with aneurysmal dilatation
Corpus callosum	Lymphoma
Frontal lobe	Arteriopathy with stenosis, atrophy
Posterior parietal lobe	Progressive multifocal leukoencephalopathy
Temporal/occipital region	Encephalopathy
White matter – periventricular	CMV, lymphoma, toxoplasma (at the cortico-medullary junction), white matter degeneration
White matter – sub-cortical frontal	Calcifications

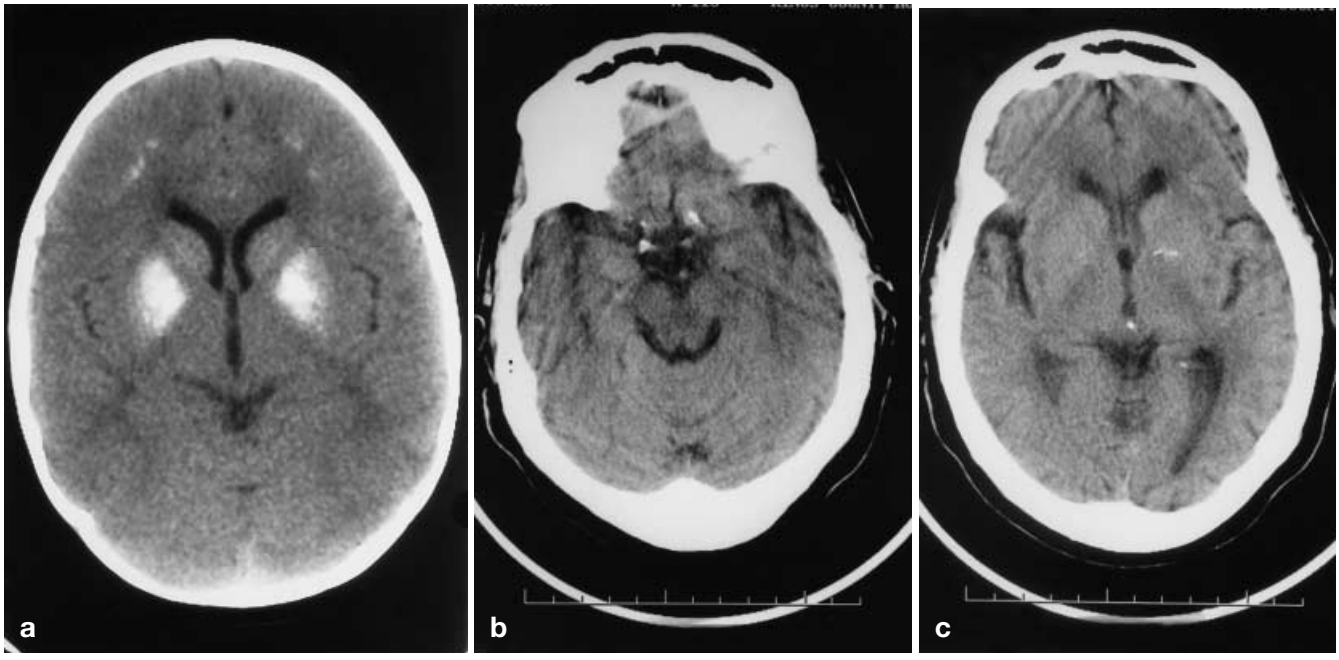

Fig. 3 Axial unenhanced CT images (**a**) in a HIV-positive child illustrates the basal ganglia and subcortical calcifications commonly seen. A second child (**b,c**) demonstrates calcific vasculitis in the circle of Willis (**b**) and lenticulostriate vessels (**c**)

MR is better than CT in detecting white matter lesions [28, 29] with abnormalities usually seen in the periventricular white matter and the centrum semiovale. The lesions have no mass effect and are of low attenuation on unenhanced CT, low signal intensity on T1-weighted MRI, and increased signal intensity on T2-weighted MRI images (Fig. 2). White matter lesions do not enhance with the administration of IV contrast. Care must be taken in interpreting these images, though, as normal myelination of the centrum semiovale is not complete until 18 months of age and may be delayed until further in the ventricular trigone [2]. This increases the relative signal of these normal areas on a T2-weighted image and may be confused with pathological changes in the white matter. On nuclear medicine scans, areas of white matter pathology show decreased radiotracer uptake [30, 31].

Currently, clinical imaging of HIV encephalopathy has focused on its associated findings, namely, white matter disease and cortical atrophy. However, positron emission tomography (PET) has recently been used to detect early disease in the face of a normal MRI. ^{18}Fluorudeoxyglucose (FDG) PET may show both hyper- and hypometabolism in the posterior lobar regions on a background of globally decreased metabolism [32], which may be a harbinger of incipient neurological deficit before the onset of MR changes.

The differential diagnosis of white matter disease related to primary HIV includes progressive multifocal leukoencephalopathy (PML), lymphoma and toxoplasmosis. A combination of contrast-enhanced MRI, nuclear medicine studies, or a biopsy is used to differentiate these conditions. The imaging appearance of these conditions is discussed further in the respective sections.

Cranial calcifications

Up to 33 % of HIV-infected children show basal ganglia calcifications, and 90 % show a calcific vasculitis at autopsy, probably on the basis of alterations in the blood-brain barrier [2, 7, 17–19, 33] (Fig. 3). These calcifications are usually bilateral and symmetrical, involving the globus pallidus and putamen. The subcortical frontal white matter and the cerebellum may also calcify [18]. White matter and cortical calcifications may also be seen, but are invariably associated with basal ganglia calcifications. The extent and progression of calcification correlates with the presence of encephalopathy and its progression [7, 8]. Calcifications related to HIV disease are usually not seen before 10 months of age. Calcifications seen prior to this stage are related to congenital infections such as toxoplasma, CMV, rubella, HSV, or syphilis rather than HIV [2]. Calcifications are optimally assessed on sonography in early life or unenhanced CT later.

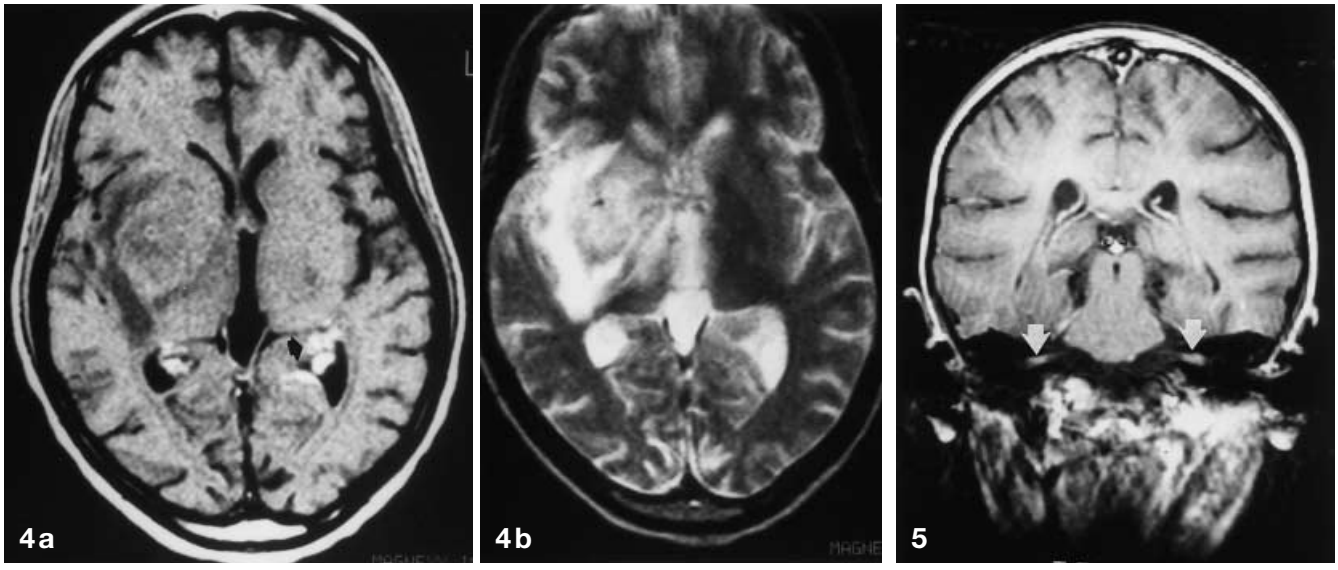

Fig. 4 Axial T1-weighted contrast enhanced images (**a**) and axial T2-weighted images (**b**) in an HIV-positive patient with multifocal lymphoma. The enhancing periventricular component (*arrow*) and involvement of the basal ganglia are common for this disease

Fig. 5 Coronal T1-weighted contrast-enhanced images in this HIV-positive patient with lymphoma demonstrate pathological enhancement of cranial nerves III, V, VII, and VIII

Malignancies

The most common malignancy related to HIV infection of the CNS is a high-grade B-cell lymphoma. This lymphoma is associated with Epstein-Barr virus infection and is either primary or metastatic. Primary lymphoma is found in 4 % of infected children, presents mostly between the ages of 5 and10 years, and usually arises in the periventricular white matter (most common), basal ganglia or corpus callosum [34–37] (Fig. 4). It may be multifocal or involve the cranial nerves (Fig. 5). CNS lymphoma may be associated with lymphocitic interstitial pneumonitis (LIP) and infiltration of the gut-associated lymphoid tissue [34, 37]. It should be noted that the overall incidence of HIV-associated malignancies is less common in children than in adults.

Primary lymphoma is hypodense on an unenhanced CT scan and enhances uniformly. In AIDS, a peripheral location, hemorrhage and ring enhancement are also common. Ring enhancement is due to necrosis and may be present even without chemotherapy administration [38, 39]. Mass effect may be less than expected for the size of the lesion [2]. Lymphomatous meningitis is rarer and is associated with extracranial lymphoma.

Differentiating lymphoma from toxoplasmosis often poses a diagnostic challenge. Lymphoma is commoner in the pediatric population than toxoplasma, and the presence of lesions with the above-mentioned imaging findings in the corpus callosum is highly suggestive of lymphoma. Thallium SPECT and more recently ^{18}FDG PET, can be used to differentiate the two entities. Lymphoma shows increased radiotracer uptake (toxoplasmosis shows decreased uptake) and is, in most cases, larger than 1 cm [30, 31, 40].

Cerebrovascular disease

Pediatric patients with HIV infection have a 1.3 % annual risk of developing cerebrovascular disease, with 25 % of patients showing evidence of cerebrovascular disease at autopsy [26, 27, 41]. Cerebrovascular disease has a wide range of manifestations from arterial stenosis to aneurysmal dilatation and may be due to the primary effects of HIV or secondary to opportunistic infections.

The fibrosing arteritis that is found in the liver, kidneys, spleen, and lungs of HIV patients also affects the brain, particularly in the basal ganglia and frontal regions [41–43]. This arteriopathy is due to the direct effects of HIV and causes arterial stenosis. At the other end of the spectrum are multiple aneurysms of the branches of the circle of Willis, which are associated with thrombotic and hemorrhagic events [43].

Cerebrovascular accidents may also occur due to either the arteriopathy of HIV or, in the setting of meningoencephalitis, due to tuberculosis, varicella zoster virus, herpes simplex virus, syphilis or *Candida* [43, 44]. Infarcts caused by these are due to thrombosis of the large and medium-sized vessels [2].

Intracranial hemorrhage is associated with HIV-induced thrombocytopenia and clotting abnormalities. Embolic events are rare and are associated with the dilated cardiomyopathy of AIDS [26, 27].

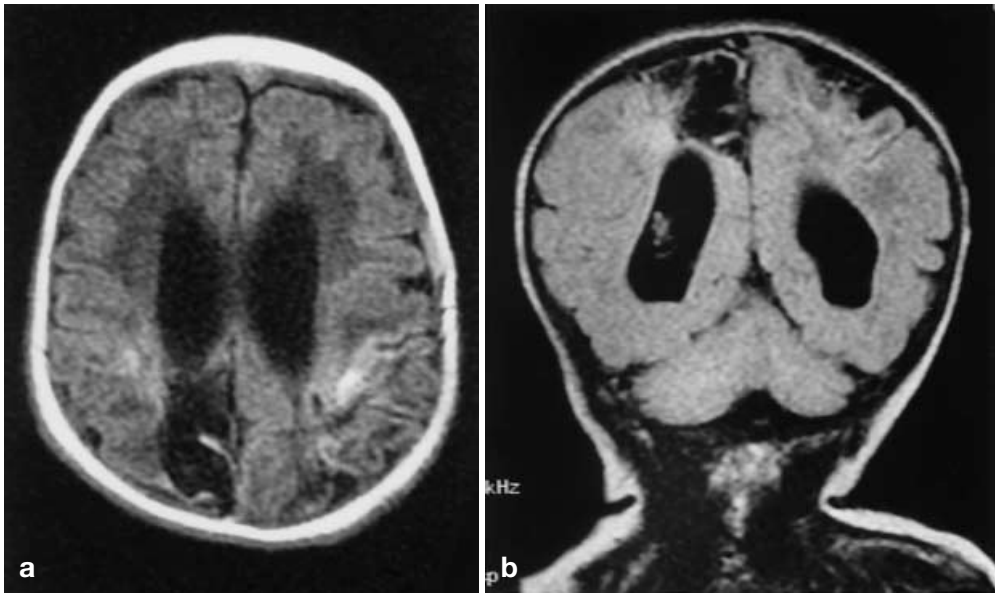

Fig. 6 Axial (**a**) and coronal (**b**) T1-weighted images illustrate encephalomalacia in the right posterior frontal and anterior parietal lobes in this HIV-positive child following an infarct. Increased T1 signal represents the laminar necrosis associated with infarcted cortex

CT and MRI are the primary imaging modalities for cerebrovascular disease. A pre-contrast CT scan may demonstrate low-density regions. Basal ganglia and cortical enhancement may be seen in subacute infarctions which, rarely, may be confused with an infectious process. Infarctions will proceed to form areas of encephalomalacia with volume loss (Fig. 6).

MR diffusion weighted images are the best sequences to detect infarction in the acute stage becoming positive as early as 30 min after the event. T1-weighted images will initially show decreased signal intensity. As early as 8 h after the event there may be increased signal on T2-weighted images. T2-weighted images will ultimately revert to low signal. In the acute phase, contrast administration may show meningeal enhancement to be replaced by parenchymal enhancement in the subacute phase. The value of thrombolysis in the acute period has not yet been proven in this population.

MR or flouroscopic angiography best demonstrates either stenosis or aneurysmal dilatation. In neonates, cranial ultrasonography may be used for diagnosis and may demonstrate lack of flow in the affected vessel.

Infections

Progressive multifocal leukoencephalopathy (PML)

PML is caused by the JC virus which belongs to the papovirus family. While many children have minor sporadic symptoms, very few manifest clinical symptoms [2]. Distinguishing pure white matter disease related to primary HIV from PML can be difficult due to the incomplete and sometimes inconsistent myelination pattern in the young brain. PML is rarer than pure white matter abnormalities in children and tends to be more focal, asymmetric and commoner in the posterior parietal lobe, while white matter disease is generally bilateral, symmetrical, diffuse, and is commoner in the periventricular white matter and centrum semiovale. Both demonstrate no mass effect, are of low attenuation on unenhanced CT, low signal intensity on T1-weighted MRI, and increased signal intensity on T2-weighted MRI images with no contrast enhancement. Preliminary results with ^{18}FDG PET show potential in differentiating PML and lymphoma, with the former showing decreased metabolism and lymphoma showing increased metabolism [48, 49].

Toxoplasmosis

Reactivation toxoplasmosis is primarily a disease of older children, while congenital toxoplasmosis in the HIV patient is little different from that in the immunocompetent patient. Anatomically, foci of toxoplasma infection are most commonly found in the basal ganglia and the corticomedullary junction of the periventricular white matter.

Contrast-enhanced CT shows inhomogeneous or ringlike enhancement with mass effect and edema. On MRI, toxoplasmosis shows increased signal on T2-weighted images and decreased signal on T1-weighted images, which enhance with contrast administration. MRI is more sensitive than CT for the detection of foci of toxoplasma [2]. The differential diagnosis for such lesions is toxoplasmosis, tuberculosis, abscesses, or lymphoma. As mentioned previously, nuclear medicine

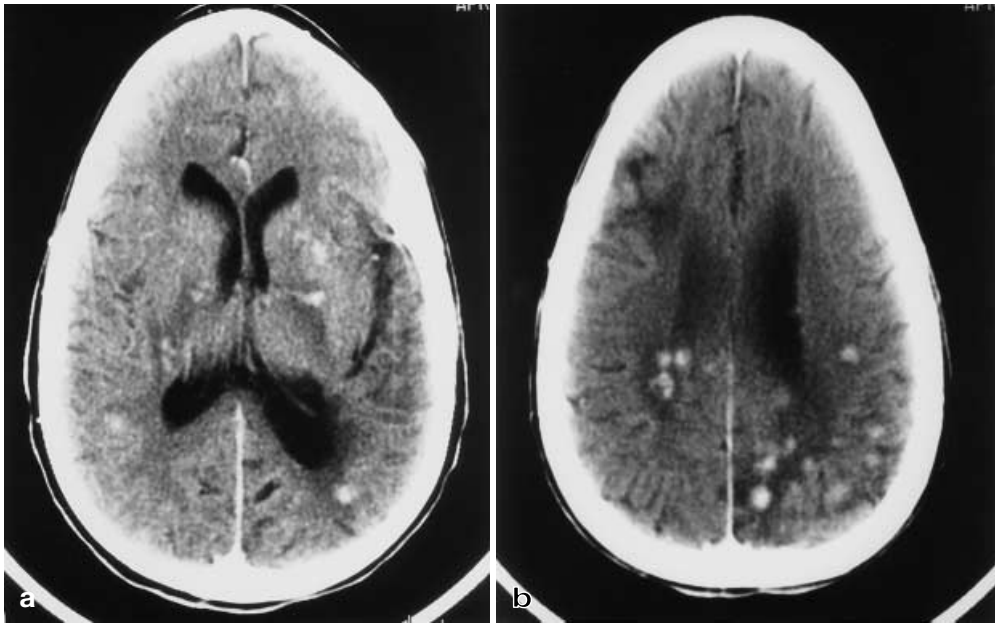

Fig. 7 Axial (**a**,**b**) contrast enhanced CT images in a child with tuberculous meningitis demonstrate numerous nodular foci of enhancement with involvement of the basal ganglia, periventricular white matter, and subcortical white matter

studies can distinguish between toxoplasma infection and lymphoma [30, 31, 50]. The combination of CT and MRI in the appropriate clinical setting should provide sufficient information to make a definitive differentiation between all these lesions.

Newly diagnosed toxoplasmosis should be followed up with an imaging study after 2 weeks of therapy. Resolution ranges from complete resolution to encephalomalacia. The appearance of new lesions while on antitoxoplasma therapy should suggest lymphoma or tuberculosis.

Meningitis

In addition to the usual causes of meningitis in children, HIV-positive children are also at risk for fungal, mycobacterial and nocardial meningitis (Fig. 7). Tuberculous meningitis is usually secondary, with milliary tuberculosis being rare. Both tuberculosis and nocardia are usually associated with pulmonary disease.

Fungi and tuberculous meningitis can have findings similar to those in lacunar infarcts because of venous thrombosis of meningeal vessels, non-communicating hydrocephalus due to obstruction of the outflow of the fourth ventricle, or, more commonly, communicating hydrocephalus from purulent material obstructing either the basal cisterns or the arachnoid villi [44, 51].

Both contrast-enhanced CT and MRI are often normal, but may show meningeal enhancement, which is more frequently seen with MRI [2]. Cine MRI can be used to determine the level of obstruction in hydrocephalus [44].

Unlike other fungi, *Cryptococcus neoformans* shows perivascular pseudocyst formation, cryptococcomas and no meningeal enhancement on contrast-enhanced imaging studies.

CMV

This infection is usually asymptomatic and, like toxoplasma, the congenital form does not differ in presentation in the immunocompetent and immunocompromised child. On MRI, CMV is seen as areas of increased signal on T2-weighted sequences in the periventricular region. Post-contrast images may show enhancement.

Conclusion

Between 15 and 60 % of infants born to HIV-positive mothers will become infected with HIV despite maternal antiretroviral therapy [4]. Of these, 23 % will develop AIDS in the 1st year of life [52]. Many conditions affecting the HIV-infected child can now be palliated, allowing for longer survival. In order to make the correct diagnosis and institute the appropriate therapy, the radiologist must have a good clinical history, a knowledge of the anatomical and demographical differences between diseases, and use a combination of imaging modalities.

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