Neuroimaging Findings in Patients with AIDS

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An increasing number of patients are presenting with central nervous system complications of human immunodeficiency virus infection. New imaging technologies such as magnetic resonance imaging, magnetic resonance proton spectroscopy, single-photon emission computed tomography, and positron emission tomography are playing an ever-increasing role in the diagnosis of these complications. As therapeutic modes improve, imaging may assume a growing role in monitoring the responses to therapy among these patients.

Approximately 40%–90% of patients with AIDS will develop CNS manifestations during the courses of their illnesses [1–3]. As a consequence, neuroimaging has come to play an important role in the treatment of AIDS [4].

Patients with AIDS develop a variety of CNS lesions, and the diagnosis of these lesions may require the application of several imaging techniques including CT, MRI, single-photon emission computed tomography (SPECT), or magnetic resonance spectroscopy (MRS). Because the sensitivity of MRI is superior to that of CT and because MRI allows acquisition of images in multiple planes, it has become the "gold standard" in neuroimaging.

Patients with AIDS may have CNS lesions due to different pathological processes that occur synchronously or sequentially; occasionally, more than one pathological process is responsible for the lesion [1, 5, 6]. In addition, patients who have developed AIDS as a consequence of intravenous drug addiction remain at risk for developing CNS diseases common to intravenous drug abusers and may have a higher risk of developing AIDS-related CNS diseases.

In the differential diagnosis of brain diseases in patients with AIDS, some generalizations about the imaging appearance of CNS lesions are helpful because the clinical presentation of these lesions is often more dependent on their anatomic location than on their etiology. Imaging reveals CNS abnormalities in these patients that can be broadly classified into four categories: focal lesions with mass effect; focal lesions without significant mass effect; diffuse global CNS abnormalities; and ventriculitis, meningitis, and infarcts.

Focal Lesions with Mass Effect

Focal brain lesions develop in 15%–20% of patients with AIDS. As there are effective therapeutic regimens for most of the lesions in this category, they are the most important lesions to identify. Diagnosis based on imaging can be confounded by the similarity in appearance of many of the CNS lesions associated with AIDS and by alterations in the typical patterns of enhancement and edema that these same lesions would produce in patients who are not immunocompromised.

Contrast enhancement on CT scans or MR images depends on vascularity and disruption of the blood-brain barrier. The inflammatory response can be weak in patients whose immune status is markedly compromised, and there may be correspondingly poor contrast enhancement of infectious lesions that would otherwise show intense enhancement in patients with normal immune responses. However, tumors enhance secondary to an abnormal blood-brain barrier within tumor vessels or secondary to increased vascularity. In general, tumors that normally enhance will continue to enhance despite a severely dysfunctional immune system, but enhancement of infectious or inflammatory lesions can be variable.

The amount of vasogenic edema surrounding a lesion is also influenced by the immune response. Both tumors and infectious lesions are affected, and in the setting of severe immune dysfunction, the amount of adjacent edema and mass effect evoked...
Figure 1. MR images of the brain of a patient with toxoplasmosis. A, T₂W fast spin echo axial MR (3000/102/2) image reveals multiple bilateral high signal-intensity masses (arrows) in the basal ganglia. The lesions elicit considerable surrounding edema, and some of the lesions have low signal-intensity rims. B, T₁W non-contrast-enhanced axial MR (600/10/1) image; the lesions are of low signal intensity, with some lesions having high signal-intensity rims secondary to hemorrhage (arrows). C, Contrast-enhanced T₁W (600/10/1) image; the lesions are ring enhancing (arrows). D, CT scan, obtained 2 months after MRI was performed, shows complete resolution of the lesions following the initiation of antitoxoplasmic therapy. T₂W = T₂ weighted; T₁W = T₁ weighted; numbers in parentheses are repetition time in milliseconds/echo time in milliseconds/excitations.
in response to either type of lesion can be less than would normally be expected. The most common focal mass lesions encountered in patients with AIDS generally induce considerable surrounding edema and enhance on CT and MRI following the administration of intravenous contrast agents.

Toxoplasmosis, the most common opportunistic CNS infection in patients with AIDS (this infection occurs in 13.4%–33% of AIDS patients with CNS complications [2, 3]), is caused by the protozoan *Toxoplasma gondii*. In the immunologically intact host, *T. gondii* generally causes a self-limited infection. After the initial infection, an intact immune system keeps the organism dormant. In the immunocompromised host, the latent organism can reactivate and cause encephalitis and brain abscesses.

Once the infection is diagnosed, it may be controlled with lifelong drug therapy [7]. Therapy is often begun empirically as soon as CT scans or MR images show focal parenchymal lesions of any sort because the infection is so common in this population [7, 8]. Patients who respond to antitoxoplasmic medications are presumed to have toxoplasmosis. The response to drug therapy is typically rapid, with noticeable regression of the lesions apparent on imaging studies within 10 days to 2 weeks [8, 9]. Patients who are not compliant or who discontinue therapy will relapse.

Toxoplasmic lesions begin as foci of encephalitis that progress rapidly to parenchymal abscesses with central necrosis and surrounding inflammation. Toxoplasmosis typically manifests on CT scans and MRIs as nodular (small-encephalitis) and/or ring-enhancing (large-abscess) lesions within the brain parenchyma. The enhancing ring, when present, may be somewhat thicker and more ill defined than that seen in association with a typical bacterial abscess.

The lesions are associated with surrounding edema and tend to be multiple at presentation [9–11]. However, a significant percentage of patients present with solitary lesions [11]. Toxoplasmic lesions are most often seen in the basal ganglia [12] and cerebral hemispheres [11] (figure 1). On nonenhanced T1-weighted (T1W) MR images, the lesions are of low signal intensity, and on T2 weighted (T2W) MR images, the lesions are mildly-to-moderately hyperintense in relation to the brain parenchyma and can be difficult to separate from the surrounding edema. The presence of small hemorrhages may be a sign of toxoplasmosis (figure 2), and calcifications can occasionally be seen at the sites of treated lesions.

Primary lymphoma of the CNS also occurs frequently in patients with AIDS [2, 3, 13, 14] and is the most common...
Figure 3. MR image of a patient with primary CNS lymphoma. 

A, T₂W fast spin echo axial MR (3000/102/2) image shows a hyperintense mass with surrounding edema in the right occipital lobe (arrows). The lesion extends along white matter tracts adjacent to the occipital horn of the right lateral ventricle. B, T₁W contrast-enhanced axial MR (600/10/2) image shows both peripheral and central enhancement. C, T₁W contrast-enhanced axial MR (600/10/2) image of another patient with primary CNS lymphoma shows an enhancing lesion involving the ependyma of the anterior horns of both lateral ventricles and the corpus callosum (arrows). The lesion either originated within the corpus callosum or spread along the corpus callosum to cross the midline. T₂W = T₂ weighted; T₁W = T₁ weighted; numbers in parentheses are repetition time/echo time/excitations.
lac and lipids

CNS neoplasm in these patients. This lymphoma is nearly always of the high-grade B-cell type, and the cells contain the Epstein-Barr virus [15, 16] (before the AIDS epidemic, most primary CNS lymphomas were of the T-cell variety). Primary CNS lymphoma can present as a solitary lesion, even in patients with AIDS, but it most often presents multifocally [11, 17]. In fact, autopsy studies have shown that CNS lymphoma is multifocal in 80%-100% of AIDS patients [18].

On CT scans and MR images, lymphoma most commonly manifests as an enhancing, space-occupying mass with surrounding edema. However, much of the time the lesions undergo central necrosis and present as ring-enhancing masses [11]; some studies suggest that most of these lesions are ring enhancing [17, 19]. On non-contrast-enhanced T1W images, typical lesions are isointense in relation to brain parenchyma, while on T2W images the lesions are isointense to hyperintense.

Occasionally, lesions may be hypointense on T2W images [11]. On nonenhanced CT, a small percentage of the lesions are of increased attenuation with respect to the brain parenchyma (as is commonly observed in cases of primary CNS lymphoma in patients who do not have AIDS). Lesions that are of increased attenuation on non-contrast-enhanced CT scans have a high probability of being lymphomas [11] and probably correspond to the lesions that are hypointense on T2W MR images. Primary CNS lymphoma most often occurs centrally (basal ganglia or corpus callosum) or in the periventricular white matter, and subependymal spread or ventricular encasement is a characteristic finding [11] (figure 3).

The imaging characteristics of lymphoma and toxoplasmosis overlap to such a significant degree that it is nearly impossible to differentiate the lesions on the basis of their appearance on CT scans or MR images alone. Patients with a few solid lesions or ring-enhancing subependymal or periventricular lesions (particularly those with subependymal extension) tend to have lymphomas, whereas patients with multiple ring-enhancing lesions (particularly those that are hemorrhagic) in the basal ganglia and cerebral hemispheres are more likely to have toxoplasmosis. The fact that toxoplasmosis and lymphoma may occur synchronously in the same patient or even together in the same lesion further confuses the issue [6]. When patients are treated empirically for toxoplasmosis, all lesions must be monitored for therapeutic responses. Differential diagnosis can be particularly difficult for patients who present with solitary lesions.

Imaging techniques including positron emission tomography (PET), MRS, and SPECT have all been reported as being helpful in differentiating primary CNS lymphoma from toxoplasmosis. When SPECT imaging is performed with thallium-201, lymphomas have a propensity to take up the radiotracer, whereas inflammatory lesions do not [20, 21]; however, the specificity of SPECT is still unknown [22, 23]. On PET scans, lymphomas have a similar propensity to take up 18F fluorodeoxyglucose [24], but PET is not widely available, and its specificity is also unknown. MRS is another technique that can aid in the differential diagnosis of these lesions [25].

Spectra produced by toxoplasmic lesions show that these lesions tend to have markedly elevated lactate and lipid peaks with depletion of normal metabolites (figure 4), while the spectra produced by lymphomas show that these lesions tend to have elevated choline peaks and mildly to moderately elevated lactate and lipid peaks (figure 4). However, even on MRS there may be considerable overlap between the spectra of these lesions. We have found that lymphomas with central necrosis may have spectra very much like the spectra of toxoplasmic lesions.

Cryptococcosis, an infection caused by the yeast Cryptococcus neoformans, is also common in patients with AIDS (~5% of these patients present with cerebral cryptococcosis [26, 27]). The initial infection is probably pulmonary, but the organism is neurotropic, and the infection quickly spreads to the CNS. Once in the CNS, the infection can manifest as meningitis, focal encephalitis...
Figure 5. CT scan of a patient with cryptococcosis. A, contrast-enhanced CT image at the level of the basal ganglia reveals multiple well-defined, bilateral nonenhancing lesions of low attenuation in the basal ganglia (arrows). B, $T_2W$ spin echo axial MR (2500/100/1) image (of the same patient) at approximately the same level shows the lesions to be uniformly hyperintense (arrows). The lesions do not induce edema. C, $T_1W$ non-contrast-enhanced axial MR (800/20/2) image shows the same lesions to be of uniformly low attenuation (arrows); these soap bubble–like lesions are gelatinous pseudocysts. $T_2W = T_2$ weighted; $T_1W = T_1$ weighted; numbers in parentheses are repetition time/echo time/excitations.
Figure 6. MR image of a patient with a cryptococcal brain abscess. A, T₂W fast spin echo axial MR (3000/102/2) image at the level of the top of the lateral ventricles shows two well-defined lesions of high signal intensity (one in the right frontal lobe adjacent to the right lateral ventricle [arrow] and another small lesion in the right occipital lobe [curved arrow]). B, contrast-enhanced T₁W MR (600/10/2) image at the same level shows both lesions to be of low signal intensity with thin rim enhancement. T₂W = T₂ weighted; T₁W = T₁ weighted; numbers in parentheses are repetition time/echo time/excitations.

Figure 7. CT scans of a patient with a tuberculous brain abscess. A and B, contiguous contrast-enhanced CT scans at the level of the basal ganglia reveal a multiloculated lesion of low attenuation with thin rim enhancement in the left temporal lobe (arrows). The lesion elicits considerable surrounding edema.
Figure 8. MR image of a patient with systemic lymphoma that metastasized to the oculomotor nerves. T₁W fat-saturated contrast-enhanced MR (600/15/2) axial image shows enlarged, enhancing third cranial nerves (arrows). Cytological examinations of the CSF were also diagnostic of lymphoma. T₁W = T₁ weighted; numbers in parentheses are repetition time/echo time/excitations.

or a cerebral mass, or an abscess. However, meningitis is by far the most common presentation, even in the immunocompetent host. The immune response is subdued (probably because of the mucinous capsule the organism elaborates), and meningeal enhancement on imaging is rare. For AIDS patients who have cerebral cryptococcosis, >70% of CT scan findings are normal or consist only of generalized atrophy [28].

C. neoformans can proliferate within the subarachnoid spaces to the point that the perivascular spaces around perforating blood vessels become filled with organisms and mucoid exudate. The dilated perivascular spaces appear as nonenhancing cystic masses on CT scans and MR images and are termed “gelatinous pseudocysts.” These cyst-like lesions have a predilection for the basal ganglia but can be present in the brain stem or in the convexities (figure 5). They appear as areas of low attenuation on CT scans, low signal intensity on T₁W images, and high signal intensity on T₂W images (these signal characteristics are similar to those for the CSF).

When C. neoformans infects the brain parenchyma, encephalitis results. The areas of encephalitis may evolve into cryptococcal masses or abscesses (figure 6). However, focal parenchymal masses occur in a relatively small percentage of patients with cerebral cryptococcosis [28] and generally appear as discrete nodular or ring-enhancing masses on CT scans and MR images.

Although the incidence of tuberculosis is higher among patients with AIDS [29], the overall incidence of neurotuberculosis still

Figure 9. MR image of a patient with progressive multifocal leukoencephalopathy. A, T₁W fast spin echo axial MR (4000/102/1) image shows ill-defined white matter lesions in both frontal lobes (arrows); no mass effect is seen. B, contrast-enhanced T₁W axial MR (66/10/1) image at the same level shows the lesion to be of low signal intensity with minimal, if any, contrast enhancement.
appears to be low [4, 30]. Bishburg et al. [30] reported an increased incidence (10 of 420 patients in their series) of neurotuberculosis among HIV-infected patients who were also drug abusers. Neurotuberculosis can present either as meningitis or as focal parenchymal lesions. As has been observed for fungal lesions of the CNS, focal neurotuberculosis may present as areas of cerebritis, granulomas (enhancing nodules), caseating granulomas (lesions with irregular ring enhancement), or as a tuberculous abscess (a lesion with thin ring enhancement and mass effect and edema) [31, 32]. Granulomas may have little or no mass effect and little associated edema. They are typically multiple. Abscesses are generally solitary (figure 7) [33]. The simultaneous occurrence of basilar meningitis in association with focal parenchymal lesions or with an abscess greatly increases the likelihood of a diagnosis of neurotuberculosis.

As noted above, both tuberculosis and cryptococcosis can rarely present as abscesses. These lesions are low attenuation on CT scans, low signal intensity on T\textsubscript{1}W-MR images, and high signal

**Figure 10.** MR image of a second patient with progressive multifocal leukoencephalopathy. *A*, T\textsubscript{2}W fast spin echo axial MR (3000/96/1) image reveals bilateral ill-defined white matter lesions in the cerebellar hemispheres (arrows); there is no significant mass effect. Contrast-enhanced T\textsubscript{1}W images showed no evidence of enhancement. *B*, an axial image at the level of the pons (same scan as *A*) shows ill-defined hypertense signal throughout the pons (arrow). *C*, contrast-enhanced T\textsubscript{1}W axial MR (600/10/1) image at the same level shows no evidence of enhancement. T\textsubscript{2}W = T\textsubscript{2} weighted; T\textsubscript{1}W = T\textsubscript{1} weighted; numbers in parentheses are repetition time/echo time/excitations.
diffuse, ill-defined, abnormally increased signal intensity in the white matter (arrows). T₂ W = T₂ weighted; numbers in parentheses are repetition time/echo time/excitations.

intensity on T₂W-MR images. They have thin enhancing rims, usually induce considerable surrounding edema, and can be identical in appearance to pyogenic abscesses. Although infection with HIV-1 does not predispose to pyogenic abscess formation, a considerable percentage of HIV-infected patients have acquired the infection through drug abuse, and those patients who continue to abuse intravenous drugs are at risk for the development of pyogenic abscesses.

Neurosyphilis, which has been reported to occur in 1.5%–1.8% of patients with AIDS [34, 35], can rarely present as focal syphilitic lesions (gummas). Berger et al. [36] have reported two such lesions in HIV-infected patients, and Brightbill et al. [37] recently reported that among their patients with neurosyphilis, 2 (6%) of 35 had gummas. Findings on imaging are nonspecific, with lesions presenting as nodular, enhancing masses in the cortex or dura. The lesions have mass effect and elicit surrounding edema [36-38]. Metastases from systemic lymphomas may present as enhancing focal masses, dura-based masses, or infiltrative lesions of cranial nerves (figure 8).

Focal Lesions Without Significant Mass Effect

The lesions in this category generally involve only white matter, do not induce significant edema, and generally do not enhance. Focal lesions in the white matter are characteristically of decreased attenuation on CT images, low signal intensity on T₁W MR images, and high signal intensity on T₂W images, and they appear nonspecific. Similar-appearing lesions are seen in patients with multiple sclerosis and postviral demyelinating syndromes (such as acute disseminated encephalomyelitis) and in aging patients. Aside from these more typical etiologies, white matter lesions in patients with AIDS may result from a number of other pathological processes.

Progressive multifocal leukoencephalopathy is the most serious focal lesion without significant mass effect. Åström et al. [39] first described it in 1958 among patients with lymphoma. It is now known to be caused by the JC virus, a papovavirus. Eighty percent of adults are seropositive for antibodies to the JC virus; however, like T. gondii, this virus causes few problems in the immunocompetent host. In 1%–3.8% of patients with AIDS, immune system failure allows reactivation of the virus. CNS manifestations are caused by infection and destruction of oligodendrocytes, resulting in secondary demyelination. The lesions tend to be multifocal at presentation, primarily involve the white matter, and progress in size and number. The lesions cause focal neurological deficits, and there is no proven therapy. Death usually occurs within months of reactivation of the virus, but remission can occur if the patient’s immune status improves.

On CT, the lesions have low attenuation. On MRI, they are of low signal intensity on T₁W images and high signal intensity on T₂W images. They exhibit little or no mass effect. Although ring enhancement has been reported [40], these lesions generally do not enhance on CT or MRI. They often occur at the interface between the gray matter and the white matter and have a scalloped contour secondary to involvement of peripheral U fibers (figure 9). The parietal lobe is predominantly affected, but these lesions also occur in the periventricular white matter, posterior fossa, brain stem, spinal cord, and even the basal ganglia (where traversing white-matter tracts are thought to be involved). In our experience, two thirds of lesions have occurred in an occipital or subtemporal location (figure 10). MR proton spectroscopy may also be useful in distinguishing progressive multifocal leukoencephalopathy from other focal brain lesions [25].

Similar-appearing lesions have also been attributed to direct infection of the CNS with herpes simplex virus and cytomegalovirus (CMV). As well, similar lesions may be seen in patients with neurosyphilis [37].

Diffuse Global CNS Abnormalities

In our practice, the most common imaging manifestation of HIV infection is global atrophy. This condition manifests as dilated, prominent subarachnoid spaces, basilar cisterns, and ventricular enlargement that is out of proportion in relation to the patient’s stated age (hydrocephalus ex vacuo). It is seen on both CT scans and MR images. The finding of diffuse cerebral atrophy can be accompanied by diffuse, confluent, ill-defined areas of abnormally increased signal intensity on T₂W MR images of the periventricular white matter (figure 11) and ab-
normally diminished attenuation on CT scans. No enhancement is noted. SPECT and MRS are more sensitive in detecting these global abnormalities; MRS shows a corresponding decrease in N-acetylaspartate (a neuronal marker), consistent with neuronal loss [41]. The findings may be accompanied by encephalopathy (AIDS dementia complex).

These findings were initially attributed to CMV infection, as autopsy studies have shown that a high percentage of AIDS patients had concomitant CMV infection of the CNS. It was subsequently recognized that HIV is neurotrophic and directly infects the CNS; these global abnormalities were then attributed to a subacute encephalitis caused by HIV. It now seems likely that both HIV and CMV can cause subacute encephalitis and encephalopathy [42]; these conditions have an identical nonspecific imaging appearance. CMV encephalopathy appears to manifest itself late in the illness, whereas HIV dementia (although usually presenting late) can occasionally be the AIDS-defining illness.

Ventriculitis, Meningitis, and Infarcts

In addition to subacute encephalitis, CMV infection can manifest as a more virulent ventriculoencephalitis [42-44]. This is most often seen in concert with CMV infection elsewhere in the body (such as CMV retinitis [44]) and is usually a late manifestation of AIDS. The infection appears to begin in the ependymal or subependymal region and spreads into the adjacent brain. The ventricles are enlarged, and hypoattenuation on CT or increased signal intensity on T2W MRI is noted in the periventricular white matter. Ill-defined periventricular enhancement can also be seen following injection of contrast medium (figure 12). The imaging findings may be similar in appearance to those associated with CNS primary lymphoma. Dural enhancement, consistent with meningitis, may also occasionally be seen [42].

Meningitis—particularly basilar meningitis—and hydrocephalus occur often in the setting of neurotuberculosis [33]. Meningitis manifests on CT scans and MR images as prominent enhancement and thickening of the meninges (figure 13) and it can be associated with infarcts secondary to vascular stenosis or occlusion.

Neurosyphilis, which is being diagnosed with increased frequency among patients with AIDS, can cause infarcts secondary to arteritis. In a recent study [37], 23% of patients with neurosyphilis were noted to have cerebral infarcts, and 20% were noted to have white matter lesions. The differential diagnosis of cerebral infarction in a young patient with AIDS should include neurosyphilis.
### Table 1. Imaging characteristics of focal CNS mass lesions in patients with AIDS.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Nonenhanced CT scans</th>
<th>Nonenhanced MR images</th>
<th>Enhancement pattern</th>
<th>Surrounding edema</th>
<th>Location of lesion</th>
<th>No. of lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toxoplasmosis</td>
<td>Low attenuation; occasional hemorrhagic lesions</td>
<td>Low signal intensity on T1W image; high signal intensity on T2W image; occasional hemorrhagic lesions</td>
<td>Nodular (small); ring (small to large)</td>
<td>++</td>
<td>Basal ganglia; grey-white junction</td>
<td>Single to many</td>
</tr>
<tr>
<td>Primary CNS lymphoma</td>
<td>Varies, but high attenuation is characteristic</td>
<td>Low to isointense signal on T1W image; low to isointense to decreased signal on T2W image</td>
<td>Uniform to ring (necrotic)</td>
<td>++</td>
<td>Periventricular white matter; basal ganglia; subependymal spread characteristic</td>
<td>One to a few</td>
</tr>
<tr>
<td>Cryptococcosis (pseudocyst)</td>
<td>Low attenuation</td>
<td>Low signal intensity on T1W image; high signal intensity on T2W image</td>
<td>None</td>
<td>None</td>
<td>Perivascular spaces (particularly basal ganglia)</td>
<td>One to many</td>
</tr>
<tr>
<td>Tuberculosis (granuloma)</td>
<td>Low attenuation</td>
<td>Low signal intensity on T1W image; high signal intensity on T2W image</td>
<td>Ring or nodular</td>
<td>None</td>
<td>Variable</td>
<td>One to many</td>
</tr>
<tr>
<td>Syphilis (gumma)</td>
<td>Low attenuation</td>
<td>Low signal intensity on T1W image; high signal intensity on T2W image</td>
<td>Uniform</td>
<td>++</td>
<td>Cortex or dura</td>
<td>One to a few</td>
</tr>
<tr>
<td>Abscess (cryptococcal, tuberculous, or bacterial)</td>
<td>Low attenuation</td>
<td>Low signal intensity on T1W image; high signal intensity on T2W image</td>
<td>Thin ring</td>
<td>+++</td>
<td>Varies</td>
<td>One to a few</td>
</tr>
</tbody>
</table>

NOTE. MR = magnetic resonance; T1W = T1 weighted; T2W = T2 weighted; ++ = mild to moderate surrounding edema; +++ = large amount of surrounding edema.

### Table 2. Imaging characteristics of other CNS lesions in patients with AIDS.

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Diagnosis</th>
<th>Nonenhanced CT scans</th>
<th>Nonenhanced MR images</th>
<th>Enhancement pattern</th>
<th>Surrounding edema</th>
<th>Location of lesion</th>
<th>No. of lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal lesion without mass effect</td>
<td>Progressive multifocal leukoencephalopathy</td>
<td>Low attenuation</td>
<td>Low signal intensity on T1W image, high signal intensity on T2W image</td>
<td>Rare rim enhancement</td>
<td>None</td>
<td>White matter</td>
<td>Single to a few</td>
</tr>
<tr>
<td>Diffuse abnormality</td>
<td>Infection with HIV and CMV</td>
<td>Low attenuation, atrophy</td>
<td>Low signal intensity on T1W image, high signal intensity on T2W image</td>
<td>None</td>
<td>None</td>
<td>Deep white matter</td>
<td>Diffuse, ill defined</td>
</tr>
<tr>
<td>Ventriculitis, meningitis, and infarcts</td>
<td>Infection with CMV</td>
<td>Low attenuation</td>
<td>Low signal intensity on T1W image; high signal intensity on T2W image; atrophy</td>
<td>Linear ependymal enhancement and ill-defined periventricular enhancement</td>
<td>+++</td>
<td>Ependyma, periventricular spaces</td>
<td>Confluent</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Thickened meninges, infarcts, hydrocephalus</td>
<td>Thickened meninges; infarcts; hydrocephalus</td>
<td>Uniform</td>
<td>++</td>
<td>Meninges (particularly basilar meninges)</td>
<td>Confluent</td>
<td></td>
</tr>
<tr>
<td>Neurosyphilis Infarcts</td>
<td>Infarcts</td>
<td>Variable</td>
<td>Variable</td>
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<td>Variable</td>
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<td>Variable</td>
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</table>

NOTE. CMV = cytomegalovirus; MR = magnetic resonance; T1W = T1 weighted; T2W = T2 weighted; ++ = mild to moderate surrounding edema; +++ = large amount of surrounding edema.
Figure 13. MR image of a patient with tuberculous meningitis and hydrocephalus. T,W contrast-enhanced axial MR (600/20/2) image reveals thickened enhancing meninges surrounding the brainstem (arrows) and dilated temporal horns of the lateral ventricles (large arrows). An arachnoid cyst in the left middle fossa (curved arrows) is an incidental finding. T,W = T₁ weighted; numbers in parentheses are repetition time/echo time/excitations.

Tables 1 and 2 summarize the CT and MRI findings with respect to brain abnormalities in patients with AIDS.

Diagnostic imaging studies serve as an adjunct to clinical acumen and laboratory studies in the management of AIDS. Future research in the areas of functional neuroimaging (SPECT and PET) and MRS will result in improved diagnostic specificity and will impact positively on patient care.

Acknowledgment

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References


