MR of the CNS in Patients with AIDS

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Introduction

The acquired immunodeficiency syndrome (AIDS) is a disease whose manifestations result from a breakdown in the cell mediated human immune system. Therefore, typical clinico-radiologic findings include largely uncontrolled infections and neoplastic proliferations of cells peculiar to this specific depression in the host's defence network.

It is well recognized that magnetic resonance (MR) imaging is superior to any other imaging modality with regard to sensitivity of detection of pathologic change in the central nervous system (CNS) in patients with AIDS.[1-7] The excellent diagnostic sensitivity of MR coupled with the extraordinary breadth of expression of one or multiple concomitant disease processes in individuals with AIDS reveal a profoundly striking and sometimes confounding picture of a devastating disease process.

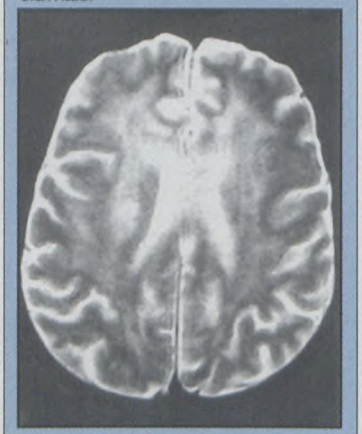
This as yet usually (uniformly) fatal disease presents with classic as well as unique pathology on MR in isolation or in combination with one another.[8-15] For the most part, the MR findings are non-specific and may be representative of infection and/or neoplasia. This review presents many of the typical as well as unusual manifestations of AIDS on enhanced MR imaging of the CNS. All cases were proved either based upon the presumptive evidence of a positive response to medical therapy (e.g. toxoplasmosis) or on the basis of direct inspection of tissue from surgical biopsy or CSF analysis (e.g. progressive multifocal leukoencephalopathy, lymphoma, cytomegalovirus infection, cryptococcosis).

Primary Human Immuno-deficiency Virus (HIV) Infection

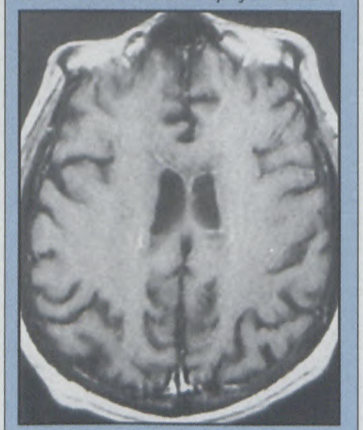
HIV is an RNA virus of the retrovirus group. It is a highly neurotropic virus which avidly infects the tissues of the CNS. The HIV particles have been found in the CNS primarily within macrophages and multinucleated giant cells. The pathologic lesion consists of microglial nodules associated with multinucleated giant cells. HIV particles have also been observed in neurons and glial cells.

Early in the course of the disease, MR imaging may be normal. The most common abnormality encountered on imaging of the brain in patients with AIDS is generalized atrophy without focal pathologic change: patchy white matter disease eventually appears that may affect the cerebral hemispheres, cerebellum, brainstem, cerebral commissures[16-20] and spinal cord.[17,21-23] This white matter disease, seen more commonly pathologically at necropsy than at imaging, is believed to be due to active HIV encephalomyelitis. This HIV involvement does not demonstrate enhancement after IV gadolinium administration which helps to differentiate this entity from other pathologic processes also found in AIDS patients that do typically enhance (eg. toxoplasmosis, lymphoma). The cerebral imaging abnormalities are often accompanied by the AIDS dementia complex, the most common neurologic problem in patients with AIDS.[24] (Figure 1) These patients present with decreased memory function, inability to concentrate, psychomotor retardation, and/or seizures. Approximately 50% of patients with AIDS dementia complex will also have involvement of the spinal cord manifesting clinically as varying degrees of paraparesis. The clinical

Figure 1: HIV encephalitis in 48 year-old male.



A) Axial T2-weighted (2400/80) spin echo acquisition. Demonstrated are bilateral hyperintense patchy white matter lesions in the periventricular regions extending into the subcortical areas. Mild cerebral atrophy is noted.



B) Axial T1-weighted (500/20) spin echo acquisition after IV Gd-DTPA administration. No abnormal enhancement of the cerebral parenchyma can be identified. This is a typical although nonspecific finding in HIV encephalitis.

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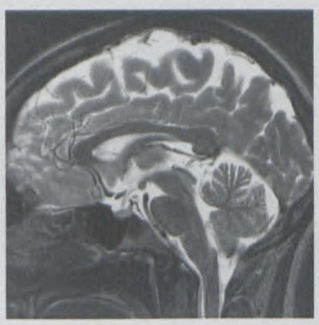

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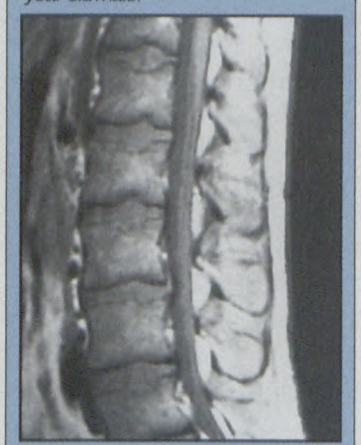
syndrome usually becomes manifest before MR abnormalities are detected. Thus, MR cannot be used to predict with certainty which patients with AIDS will develop the AIDS dementia complex.

Depending upon the variable severity of the findings, the differential diagnosis of this MR appearance in AIDS patients includes viral encephalomyelitis due to secondary infection e.g. cytomegalovirus, Herpes simplex virus, progressive multifocal leukoencephalopathy (PML), and ischaemic white matter disease of the type frequently seen in association with the vasculopathy common to CNS AIDS.[26,27]

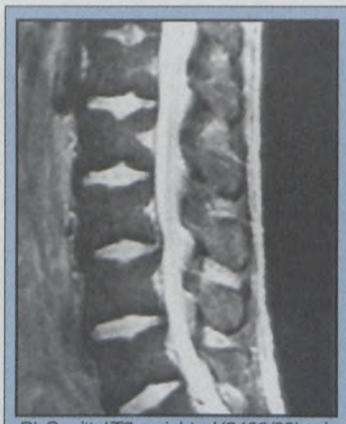
Secondary Viral Infection

Secondary infection by virus particles other than HIV is an important component of the clinicoradiologic picture in patients with AIDS. Cytomegalovirus (CMV) is a herpes virus that can be found in the tissues of up to 90% of AIDS patients at autopsy. Infection of the CNS occurs in approximately 33% of AIDS patients and may affect the cerebrum, cerebellum, spinal cord, spinal nerve roots and leptomeninges (Figure 2).[25,26,27]

Figure 2: CMV polyradiculitis in 25 year-old male.

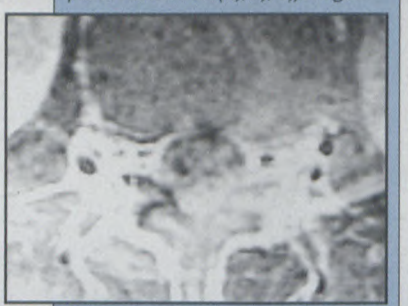


A) Sagittal T1-weighted (500/20) spin echo acquisition.



B) Sagittal T2-weighted (2400/80) spin echo acquisition.

C) Axial T1-weighted (500/20) spin echo acquisition at the L3 level. No abnormality can be identified on these pre-enhancement (A), B), C)) images.

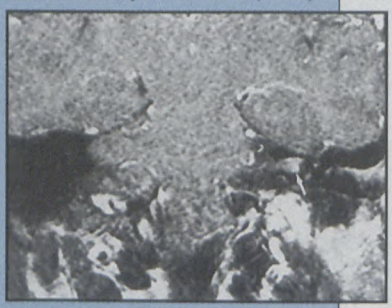


D) Axial T1-weighted (500/20) spin echo acquisition at the L3 level after IV Gd-DTPA administration. This image reveals striking enhancement of all of the intrathecal nerve roots. This is a typical although not specific finding of CMV polyradiculitis.

Typically, CMV involvement of the cranial nerves/spinal nerve roots demonstrate enhancement with IV gadolinium, whereas CMV infection of the parenchyma of the brain and spinal cord usually does not. This is an important observation because such active disease of nerves and nerve roots cannot be seen on T1- or T2-weighted acquisitions alone, and may be missed if enhancing agents are not used in this setting. Similarly, herpes virus may affect these same tissues, occasionally presenting with related signs and symptoms (Figure 3).[28,29] Uncommonly, Herpes simplex may present in patients with AIDS as a necrotizing encephalitis preferentially involving the temporal lobes.

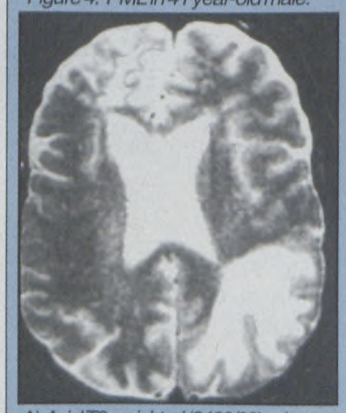
Figure 3: Herpes virus infection producing the Ramsay-Hunt syndrome in 24 year-old male.

A) Coronal T1-weighted (500/20) spin echo image. The normal appearing left internal auditory canal is seen (arrow).



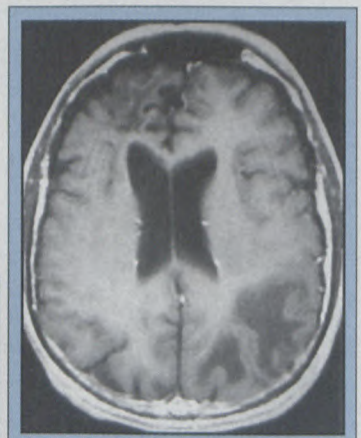
B) Coronal T1-weighted (500/20) spin echo image after IV Gd-DTPA administration. Noted is intense enhancement (arrow) within the left internal auditory canal. Herpes infection at this location often results in enhancement during the acute/subacute phases of the infectious process.

Figure 4: PML in 41 year-old male.



A) Axial T2-weighted (2400/80) spin echo image. Identified are bilateral areas of abnormal hyperintensity involving the deep white matter extending into the subcortical areas resulting in atrophy of the right frontal lobe and some mild mass effect in the left parieto-occipital region.

PML is caused by infection with the JC papovavirus. These viruses directly infect oligodendrocytes. Dysfunction of the myelin producing oligodendrocytes results in lesions secondary to demyelination. There is an absence of significant perivenous inflammation. PML may begin as a single focus, but eventually progresses to encompass many foci that may become confluent. Any site may be affected in the brain and cerebellum. While the deep white matter (including the cerebral commissures) is the predominant site of involvement, the gray matter may also become incorporated into this process.[30,31] PML lesions typically do not enhance after IV gadolinium administration (Figure. 4). If enhancement does occur, complicating factors should be considered such as superinfection with another organism, frank tissue necrosis, or superimposed infarction.



B) Axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration. No enhancement is present. This is a classic appearance of PML. If enhancement is present, superinfection should be considered.

Parasitic Disease

By and large, the type of parasitic infection encountered in AIDS patients and its incidence will depend upon the presence of endemic infectious agents in the index population. In North America, *Toxoplasma gondii* predominates in AIDS

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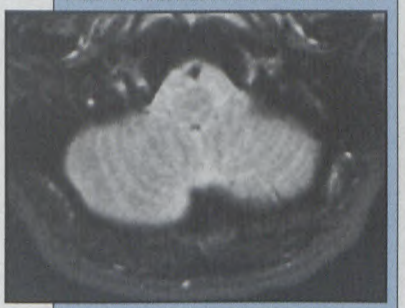
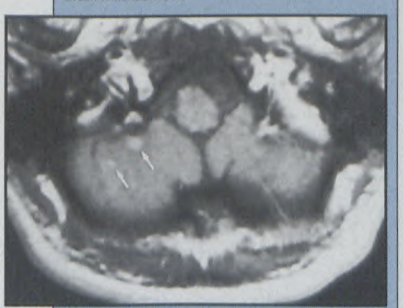
patients. This organism is presumed to be a protozoan. It is an obligate intracellular organism that usually causes subclinical infection in immunocompetent individuals characterized clinically by lymphadenopathy and low grade fever. Toxoplasmosis in AIDS patients is believed to be due to reactivation of quiescent areas of previous infection that occurred prior to the contraction of AIDS.

Figure 5: Toxoplasmosis in 30 year-old male: unenhanced versus enhanced MRI.



A) Axial T1-weighted (500/20) spin echo acquisition following IV Gd-DTPA administration.

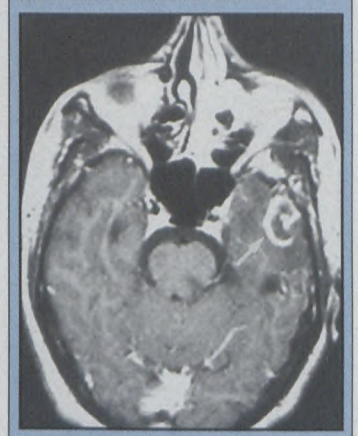


B) Two lesions are identified in the right cerebellar hemisphere (arrows). These foci of toxoplasmosis would have been less missed without the use of a paramagnetic contrast agent.

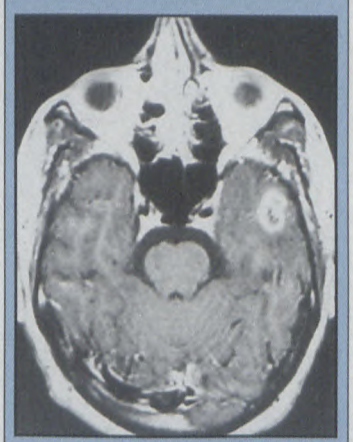
Toxoplasmosis is one of the most common neurologic opportunistic infections in patients with AIDS. Microscopically, toxoplasmosis lesions are necrotizing abscesses consisting of three zones. The inner zone contains necrotic material with few organisms. The middle zone is hypervascular and contains many inflammatory cells and organisms. The outer zone has few vascular changes and mostly encysted organisms. The foci tend to be multiple, and may affect any area of the brain [32,33] or more rarely the spinal cord.[34,35]

Toxoplasmosis lesions enhance after IV gadolinium administration with a solid or ring-like configuration (Figure 5). Although a multiplicity of enhancing lesions favours toxoplasmosis, and even though toxoplasmosis is more common, there is no absolute imaging criterion that specifically distinguishes toxoplasmosis from enhancing foci due to other aetiologic agents (e.g. fungal abscesses, tuberculous granulomata, multicentric lymphoma).[31,36,37] Even solitary enhancing lesions statistically will most likely represent *Toxoplasma* infection. Repeat imaging on medical treatment (e.g. sulfadiazine, pyrimethamine) may be confirmatory by showing a positive response to therapy and provides a reasonable alternative to surgical biopsy in areas where toxoplasmosis is common (Figure 6). Lesions which do not decrease in size indicate the presence of other or concurrent pathologic processes which will usually require surgical biopsy to establish a specific diagnosis.

Figure 6: Toxoplasmosis in 34 year-old male: effect of medical treatment.



A) Initial axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration. An incomplete ring enhancing lesion (arrow) is noted in the left temporal lobe.



B) Repeat axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration following only seven days of appropriate medical therapy. A dramatic reduction in size in the left temporal lesion is revealed, confirming successful empirical therapy. No other form of therapy (e.g. steroids) was administered during the interim between the scans.

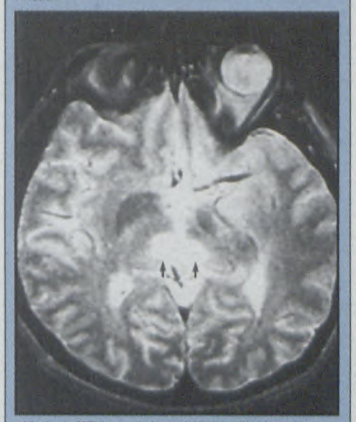
Bacterial Infection

Early in the clinical experience with AIDS, bacterial organisms were not thought to play a major role in regard to superinfections of the CNS in patients with AIDS. The reason for this is that in AIDS it is the cellular rather than the humoral immune response that is primarily disrupted, and it is the latter which largely accounts for the immune reaction to bacteria. Nevertheless, several bacterial organisms break these rules and are encountered with some frequency in AIDS patients.

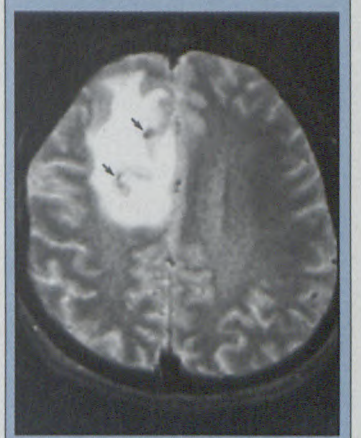
For example, sexually transmitted bacteria such as the syphilis spirochete, may gain access to the CNS in AIDS patients and pursue a more rapid course of active disease than would be usual in the otherwise immunocompetent individual. Neurosyphilis may affect neural tissue directly as well as the vascular system serving the CNS.[38,39] In meningovascular syphilis, secondary infarction and hemorrhage often accompany the vasculitis (Figure 7). In its parenchymatous form, neurosyphilis may manifest CNS gumma formation that enhances in a ring-like or solid, nodular configuration (Figure 7).

Granulomatous bacterial infections can recur from reactivation of quiescent foci of prior infection and may dominate the clinical picture in some patients with AIDS.[40,41] This is dramatically illustrated in the rapidly escalating incidence of tuberculosis (TB) in world populations. Such TB may be typical or atypical, drug susceptible or drug resistant, and may affect the entire

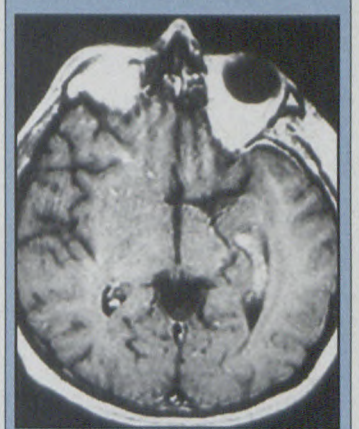
Figure 7: Neurosyphilis in 29 year-old male.



A) Axial T2-weighted (2400/80) spin echo acquisition. Bithalamic hyperintensity is noted (arrows).



B) Axial T2-weighted (2400/80) spin echo image acquired at a higher level. Diffuse hyperintensity is identified in the right frontal lobe, with several internal regions of hypointensity (arrows).



C) Axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration acquired at the same level as A). No abnormal enhancement is seen. This is consistent with bithalamic infarcts caused by arteritis accompanying meningovascular syphilis.

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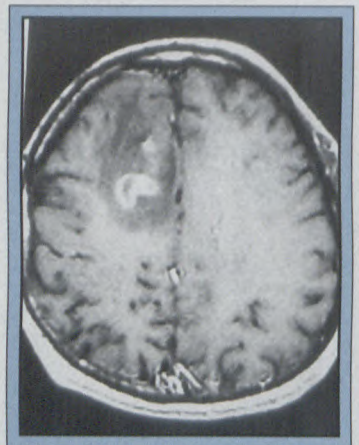
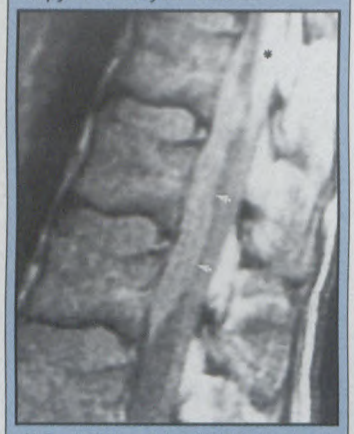


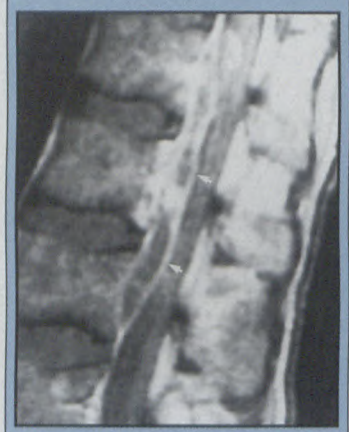
Figure 7 D) Axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration acquired at the same level as B).

craniospinal axis. Because of the poor immune response in AIDS patients and the nature of the tuberculous disease process, the progress of TB may be fulminant and its extent of involvement may be overwhelming. Granuloma or frank abscess formation may occur within the CNS parenchyma or epidural/subdural space in the cranium or spine. In addition, a diffuse meningitis may also be a feature of TB, often occurring concomitantly with the parenchymal deposit(s). After IV gadolinium administration, solid granulomas and phlegmonous epidural inflammatory response will enhance homogeneously, while caseating granulomas and frank TB abscess/empyema formation will show central non-enhancement (Figure 8).

Figure 8: Spinal tuberculous epidural empyema in 30 year-old male.



A) Sagittal T1-weighted (500/20) spin echo image acquired in the upper lumbar region. An area of hyperintensity (arrows) is seen in the anterior spinal canal. The process extended throughout the thoracic spine as well (not shown). The tip of the conus (asterisk) is also identified. Note the normal appearance of the vertebral bodies ad intervertebral discs.

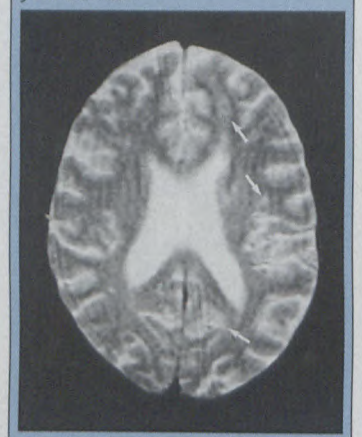


B) Sagittal T1-weighted (500/20) spin echo image after IV Gd-DTPA administration. Demonstrated is enhancement surrounding the periphery (arrows) of a tuberculous epidural abscess. While not specific, the appearance is typical for spinal abscess formation. The normal appearance of the intervertebral discs and the extensive disease are not uncommon in patients with AIDS.

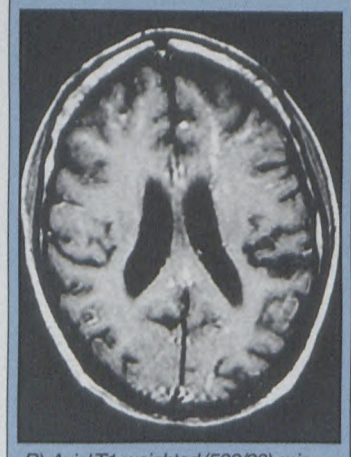
Fungal Infection

Although theoretically any opportunistic fungus might affect the CNS in AIDS patients, certain fungi are characteristically observed. *Cryptococcus neoformans* is probably the most commonly encountered fungus in this setting.[6,7,41] *Cryptococcus* is an opportunistic pathogen that is very neurotropic. In host tissues it has a variable yeast morphology. Its surrounding polysaccharide capsule is nearly immunologically inert and helps mask fungal surface antigens. The predilection for CNS involvement may be related to poor phagocytic response in the CNS, the presence of nutritional factors in CSF (i.e. asparagine and creatine), and perhaps to the absence of inhibitory factors in CSF that are found in serum. *Cryptococcus* may affect the brain and meninges focally or diffusely and can be difficult or impossible to identify on unenhanced images. The reason for this difficulty is because of the generalized, nonspecific grey and white matter disease (e.g. PML, HIV encephalitis) observed in many advanced cases of CNS AIDS. Following IV gadolinium administration, however, the foci of fungal infection are strikingly revealed as areas of parenchymal and/or leptomeningeal enhancement (Figure 9). One exception to this general rule is seen in the nonenhancing or poorly enhancing gelatinous pseudocysts typically seen involving the perivascular spaces penetrating the basal ganglia in association with cryptococcal infection (Figure 10).[42]

Figure 9: Cryptococcal infection in 31 year-old male.



A) Axial T2-weighted (2400/80) spin echo acquisition. Noted is diffuse cerebral atrophy and scattered, ill-defined foci of hyperintensity (arrows) in the hemispheric white matter.



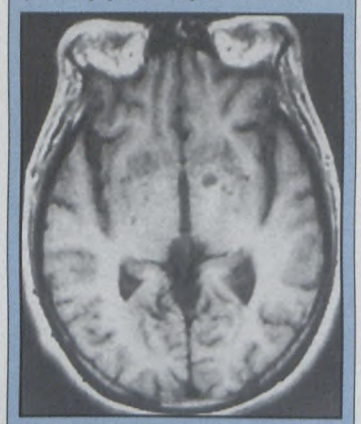
B) Axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration. Enhancing lesions are seen throughout the deep white matter, the corticomedullary junctions, the grey matter and even the corpus callosum. Once again, while not specific, this nevertheless suggests diffuse infection and is a pattern that has been described in cerebral cryptococcal disease.

Nevertheless, virtually any fungus that gains access to the CNS in AIDS patients may progress fulminantly. Other fungi that have been reported to affect the CNS in AIDS patients include *Aspergillus, Rhizopus, Coccidioides, Histoplasma* and *Candida* species.[5,7,8,10,11,13,15]

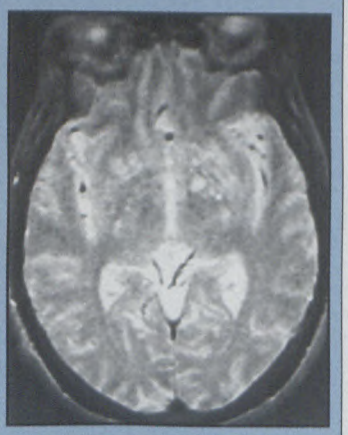
Neoplasia

The classic neoplastic process seen in patients with CNS AIDS is that of lymphoma. Lymphoma is second in frequency only to toxoplasmosis as a cause of cerebral masses in patients with AIDS. Typically, these lymphomas are of the large cell immunoblastic or small noncleaved cell type.[43,44] Lymphoma may be primary, affecting the CNS

Figure 10: Cryptococcal gelatinous pseudoycysts in 35 year-old male.



A) Unenhanced axial T1-weighted (500/20) spin echo image shows multiple hypointense areas scattered within the basal ganglia bilaterally.



B) Axial T2-weighted (2400/80) spin echo image shows that the regions of abnormality in the basal ganglia identified in Fig. A) are hyperintense on this imaging sequence. These findings are typical of cryptococcal gelatinous pseudo cysts.

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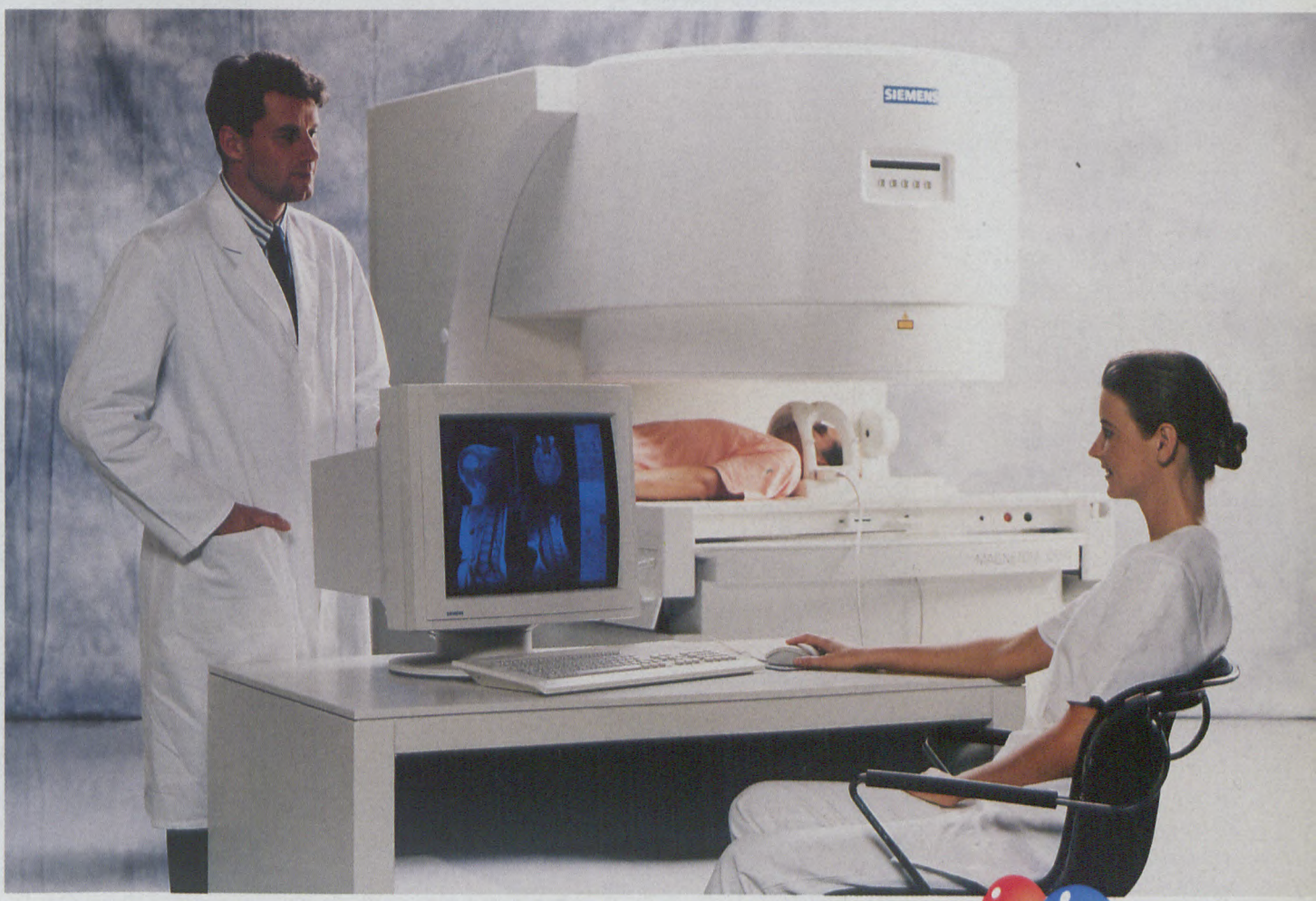

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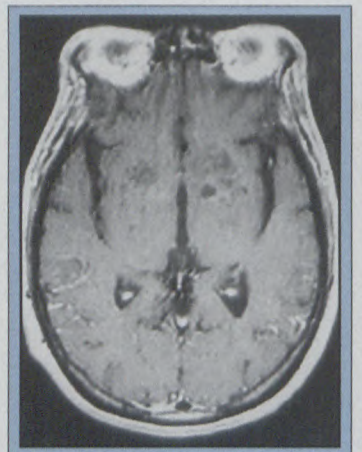


Figure 10 C) Axial T1-weighted (500/20) spin echo image after IV Gd administration shows poor or nonenhancement of the basal ganglia gelatinous pseudocysts.

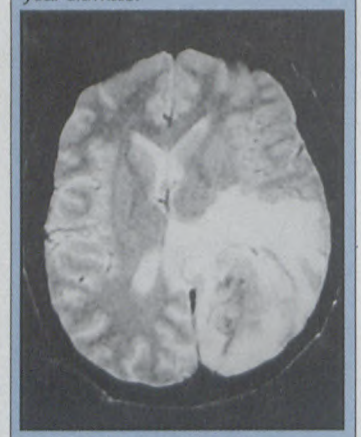
parenchyma (Figure 11),[45] or alternatively metastatic from a peripheral lymphomatous focus affecting the ependymal surfaces (Figure 12), nerve roots, and/or leptomeninges (Figure 13).[46] While primary CNS lymphoma may be solitary, in some instances primary lymphoma in AIDS patients can be multifocal. Solid, nodular enhancement may be seen in parenchymal deposits; however, CNS foci of lymphoma often show central non-enhancement due to necrosis. Ependymal or leptomeningeal CNS spread from a peripheral lymphomatous source typically shows extensive nodular and/or sheetlike regions of enhancement following IV gadolinium administration, and may affect both the cranium and the spinal canal.

Other neoplasms have uncommonly been seen to affect the CNS in association with AIDS patients. For instance, metastatic Kaposi's sarcoma to brain parenchyma has been reported and is definitely related to AIDS.[47] Other published neoplastic conditions (e.g. plasmacytoma, astrocytoma) are so rare that they may be only incidental to the condition of AIDS.

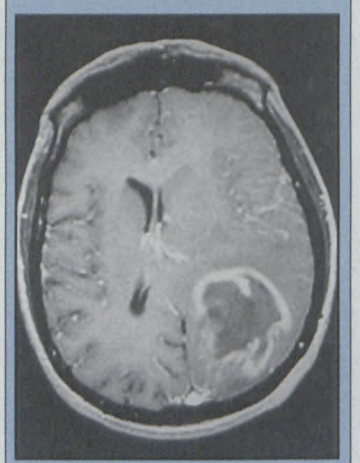
Cerebrovascular Lesions

Cerebrovascular accidents may be seen in association with virtually any infectious agent (e.g. bacterial, viral, parasitic, or fungal).[48-50] These vascular

Figure 11: Primary lymphoma in 35 year-old male.



A) Axial T2-weighted (2400/80) spin echo acquisition. Shown is a large mixed intensity mass in the left parieto-occipital area surrounded by edema and accompanied by mass effect.



B) Axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration. A ring-enhancing pattern is revealed with irregular margins and of varying thickness. No other lesions could be identified. While this is more typical of neoplasm than it is of most inflammatory processes, this lesion still cannot be differentiated from some other lesions often seen in AIDS patients, such as toxoplasmosis. Often following a failure of medical therapy for toxoplasma, such lesions come to surgical biopsy for tissue confirmation.

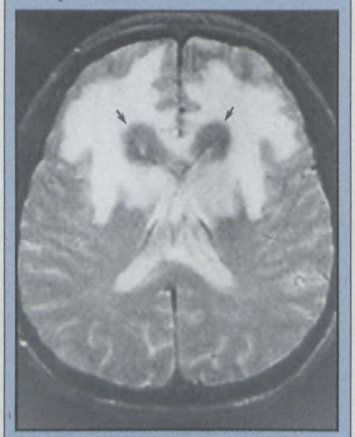
accidents have been seen on MR in up to 34% of AIDS patients presenting with neurologic complaints.[48] The majority were non-hemorrhagic cerebral infarctions, although frank hemorrhages may also be seen. For this reason, T1-weighted images should always be obtained before IV gadolinium administration so that hemorrhage (i.e. methemoglobin) will not be confused with enhancement. In most documented cases, the cerebrovascular lesions were related to a local arteritis. The other major factor for cerebrovascular accidents was found to be emboli from a peripheral source (e.g. cardiac). Finally, in cases of CNS hemorrhage, an associated systemic bleeding tendency must be considered in patients with AIDS (e.g. associated with thrombocytopenia).[48]

Conclusion

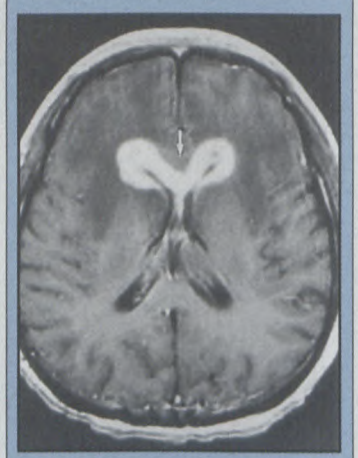
MR has proved to be the most sensitive single imaging method for the demonstration of CNS disease in AIDS patients. The administration of IV gadolinium increases the sensitivity of the MR examination to disruptions in the blood-CNS barrier present in many pathologic processes affecting AIDS patients both in the presence and in the absence of T2-weighted MR signal changes in the cranium and spine. At the same time, the use of a paramagnetic contrast agent distinguishes enhancing from unenhancing pathology revealed on abnormal T2-weighted acquisitions.[51] In some cases the judicious use of a contrast agent materially assists in focusing the differential diagnosis and thereby potentially positively affects patient management.[52] Unfortunately, it must still be concluded that the patterns of contrast enhancement observed in most instances remain non-specific with regard to a particular etiology, either inflammatory or neoplastic.

Because MR imaging of the CNS has not proven to be efficacious to survey for early, silent disease in HIV positive patients, MR should probably be reserved for those AIDS patients who are symptomatic.[53,54] As an initial diagnostic imaging examination, MR has proved to be valuable in such patients in directing further clinical diagnostic tests (e.g. CSF sampling), surgical procedures (e.g. stereotactic or open biopsy), and imaging studies (e.g. arteriography in cases of suspected arteritis).

Figure 12: Metastatic periventricular/ependymal immunoblastic lymphoma in 40 year-old male.



A) Axial T2-weighted (2400/80) spin echo acquisition. Bilateral hyperintensity is noted in both frontal lobes surrounding periventricular caps (arrows) of hypointensity.



B) Axial T1-weighted (500/20) spin echo image after IV Gd-DTPA administration. The periventricular areas are seen to enhance intensely. Note that the enhancement extends beneath the corpus callosum (arrow) and not through it. This pattern is quite peculiar to ependymal spread of lymphoma metastatic from a peripheral focus. This diagnosis is almost always confirmed by the known presence of peripheral lymphoma deposits.

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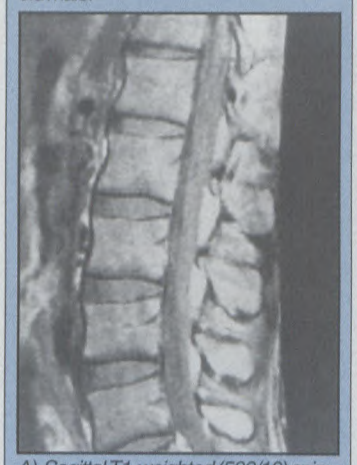
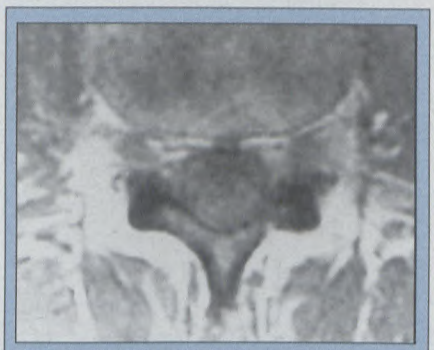
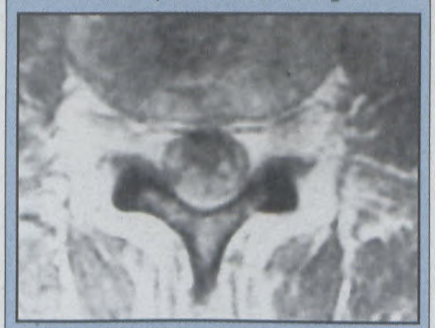
Figure 13: Metastatic subarachnoid dissemination of peripheral (non-CNS) non-Hodgkin's lymphoma in 31 year-old male.



A) Sagittal T1-weighted (500/10) spin echo acquisition.



B) Axial T1-weighted (500/10) spin echo image acquired at the L3 level. No abnormality can be identified on these pre-enhancement images.



C) Axial T1-weighted (500/10) spin echo image acquired at the same level as in after IV Gd-DTPA administration. Enhancement is noted of virtually all structures (roots) within the thecal sac. While this is compatible with the diagnosis of subarachnoid spread of lymphoma (lymphomatous meningitis), even in the face of known peripheral lymphoma the final diagnosis must rest with cellular proof obtained from cerebrospinal fluid samples. This is necessitated by the identical appearances of subarachnoid tumour dissemination and infectious polyradiculitis in some AIDS patients. (Compare with Figure 2).

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