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Encephalopathy in AIDS Patients: Evaluation with MR Imaging

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The presence and extent of encephalopathy were evaluated in 47 patients with AIDS or AIDS-related complex (ARC) by the use of MR imaging. Twenty-nine (62%) of the patients showed some form of white matter disease, exhibited as high signal intensity on T2-weighted images. Focal white matter lesions were seen in 23 (49%) of the patients, while a diffuse white matter process was observed in six patients (13%). Of the 29 patients who had white matter disease on MR scans, 17 (36%) had a suggestion of white matter involvement on an initial CT study. Meanwhile, 12 (26%) of the patients had a normal CT scan on the initial examination. MR findings showed predominant disease in the subinsular and peritrigonal white matter areas. Marked cerebral atrophy was observed in 17 (36%) of 47 patients, cerebellar atrophy in 18 (38%), and brainstem atrophy in seven patients (15%). Pathologic findings showed that toxoplasmosis was present in eight patients (17%), and primary CNS lymphoma was present in three patients (6%). Cryptococcal meningitis was noted in two (4%) of the patients at autopsy, and *Mycobacterium tuberculosis* was seen in one (2%) of the patients at autopsy.

MR imaging has been shown to be a valuable technique for the detection of encephalopathy in AIDS patients.

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The human immunodeficiency virus (HIV-1) is not only lymphocytic but also neurotrophic [1–3]. Approximately one third of AIDS patients have neurologic signs or symptoms during the course of the illness, and approximately 10% develop neurologic complaints prior to the manifestation of the syndrome [4–6]. CNS lymphomas and infections due to opportunistic organisms often produce neurologic disease in these patients [4–6]. However, it is clear that many cases of neurologic disease are caused by direct CNS infection with HIV-1 [3, 4, 6–8].

Neurologic imaging studies have proved important in suggesting the diagnosis and treatment [9–16]. The spectrum of CNS disease in AIDS is wide and comprises viral syndromes, nonviral infections, neoplasms, cerebrovascular accidents, and myelopathy associated with or without peripheral neuropathies [4–6, 10]. Imaging studies are important tools for establishing the diagnosis and monitoring the progression of the illness [4, 10–12].

The purpose of this paper is to compare MR and clinical findings in 47 patients with AIDS/ARC studied retrospectively at the University of Illinois-Cook County Hospitals during a period of 26 months.

Materials and Methods

The cranial MR and CT scans of 60 patients with a presumptive diagnosis of AIDS or ARC were reviewed. CT and MR scanning were used to evaluate new disease or progression of existing disease, or to reevaluate recurrent neurologic symptoms.

The medical records of the 60 patients were accessed from the files of the University of Illinois and Cook County Hospitals Medical Records Department and reviewed for criteria of

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TABLE 1: Summary of Clinical Data and Imaging Results in 47 Patients with AIDS

Case No.	Age	Sex	Clinical Diagnosis	Demyelination	
				Focal	Diffuse
1	47	M	ARC (HIV+): admitted for heme + stools; 3rd day acute mental status changes, right hemiparesis and aphasia	+	−
2	43	M	AIDS S/P PCP, syphillis, candidiasis, positive Pseudomonas sepsis: admitted for dementia × 2 mo	−	−
3	49	M	AIDS: admitted with weight loss, cough, memory loss × 2 mo		+
4	39	M	AIDS S/P neurosyphillis, toxo titers, chronic Pseudomonas, otitis media: admitted with right arm weakness	+	−
5	41	M	AIDS S/P PCP, candidiasis: presented with nausea and vomiting	+	−
6	31	M	AIDS S/P candidiasis, duodenal AFB: presented with tonic clonic seizures	+	−
7	29	M	AIDS: admitted for candidiasis, weight loss, hepatitis; positive toxo titers, class IV cytology CSF	+	−
8	22	M	ARC: admitted for RLE, anesthesia, RUE weakness; normal CT, normal lumbar puncture, Epstein-Barr viral titer = 1:640	−	−
9	57	M	AIDS with headaches; negative lumbar puncture	−	−
10	30	M	AIDS with bilateral lower extremity sensory loss	−	−
11	42	M	AIDS, shortness of breath, PCP, mental status changes with memory loss	−	−
12	31	M	AIDS with acute mental status changes and memory loss	−	+
13	39	M	AIDS with acute mental status changes	−	−
14	43	M	AIDS with new-onset headaches, mental status changes × 1 mo, memory loss; neurologic exam normal	−	−
15	40	M	AIDS: admitted for weight loss, fatigue, swelling under left arm; node biopsy positive for lymphoma		
16	30	M	ARC with history of ulcerative colitis: admitted for bloody diarrhea		
17	57	M	AIDS with cytomegaloviral retinitis retroperitoneal lymph adenopathy, mental status changes		
18	50	M	AIDS; admitted for flank pain, increased temperature, acute mental status changes, candidiasis	+	
19	39	M	AIDS: admitted for dementia with mental status changes, urinary incontinence; lumbar puncture and cultures negative		+
20	25	M	AIDS, PCP, candidiasis: admitted with acute bilateral sensory loss neurologic exam negative		+
21	32	M	AIDS: presented with memory loss and dizziness; neurologic exam normal		+

Atrophy				Sinusitis					Lymphoid Hyperplasia		Lesions (Toxo/Lymph)
CBA	CBLLA	VA	BSA	FS	ES	MS	MAS	SS	NP	Parotid Gland	
+		+	−	−	+	−	−	+	−	−	CT: lesion in left occipital; clinical toxo
+	+								+		Cutaneous lymphoma
−	+										Clinical toxo
−	+						+			+	Clinical toxo
−	+	+			+	+				+	
+	+	+									
−	+	+							+	+	CNS lymphoma
−	+	+								+	
−									+		
−	−	+							+		
−	+	+	+					+			
−	−		+	+	+			+			
−		+								+	
+	+										
+		+									Non-Hodgkin lymphoma
+	+	+									
+	+	+	+		+						
											Lacunar infarct
+	+	+	+								Diffuse white matter disease
+	+	+	+								
+		+									

TABLE 1—*Continued*

Case No.	Age	Sex	Clinical Diagnosis	Demyelination	
				Focal	Diffuse
22	21	M	AIDS: presented with bilateral lower extremity sensory loss, pulmonary TB		
23	32	M	ARC with sudden-onset right-sided sensory loss, weakness of right leg, memory loss	+	
24	35	M	AIDS with PCP lesions on brain, toxo; acute mental status changes, Cryptococcus	+	
25	37	M	ARC with recent mental status changes; lesions on CT scan; toxo	+	
26	45	M	AIDS with memory loss × 2 weeks, dizziness, fatigue		
27	37	M	AIDS S/P CNS, toxo, new ocular findings; visual and auditory perception loss		+
28	52	M	AIDS with mental status changes, focal motor LLE and memory loss	+	
29	23	M	AIDS with positive toxo titers and CT lesion: admitted for seizure disorder and left leg hyperreflexia	+	
30	39	F	AIDS with positive CSF cryptococcal antigens: presented with acute mental status changes	+	
31	30	M	AIDS with Kaposi sarcoma and new-onset headaches		
32	27	M	AIDS: admitted for persistent headaches; CT showed right hypodense lesion in caudate nucleus	+ (basal ganglia)	
33	25	M	AIDS with bilateral 6th nerve palsy, confusion, ataxia, white spot on fundus		
34	47	M	AIDS with new-onset seizures, loss of memory, weakness	+	
35	28	M	AIDS with *Mycobacterium kansasii*, new-onset generalized seizures	+	
36	53	M	ARC with new-onset memory loss and acute mental status changes	+	
37	33	M	AIDS with loss of dexterity and strength, disorientation	+	
38	40	M	ARC with nightly fevers, bilateral sinus tenderness	+	
39	42	M	ARC: presented with RUE weakness, mental status changes, lethargy		
40	24	M	ARC: presented with mental status changes, weight loss		
41	31	M	AIDS with dementia, memory loss		
42	35	M	AIDS with bilateral leg weakness, dysphagia		
43	33	M	AIDS with grand mal seizures	+	
44	39	M	ARC with bilateral visual and mental status changes	+	
45	21	M	AIDS with mental status changes and lower extremity weakness	+	
46	49	M	AIDS with right-sided weakness	+	
47	32	M	AIDS with decreased mental status/RLE weakness	+	

Note.—CBA = cerebral atrophy, CBLLA = cerebellar atrophy, VA = vermian atrophy, BSA = brainstem atrophy, FS = frontal, ES = ethmoid, MS = maxillary, MAS = mastoid, SS = sphenoid, NP = nasopharyngeal, Toxo = toxoplasmosis, Lymph = Lymphoma, ARC = AIDS related complex, PCP = pneumocystis carinii pneumonia, AFB = acid fast bacillus, RLE = right lower extremity, LLE = left lower extremity, RUE = right upper extremity, and PML = progressive multifocal leukoencephalopathy.

Atrophy				Sinusitis					Lymphoid Hyperplasia		Lesions (Toxo/Lymph)
CBA	CBLLA	VA	BSA	FS	ES	MS	MAS	SS	NP	Parotid Gland	
+	+	+			+						Cryptococcus; biopsy positive for toxo
+	+		+								Clinical toxo
	+	+									Clinical toxo
+											
	+										Clinical toxo
											Cryptococcus meningitis
											CNS lymphoma
									+	+	Cytomegaloviral retinitis
+	+		+		+	+	+	+	+		
+					+	+	+	+			
+						+					Right cerebellar lesion
+						+					PML
						+			+		
						+	+				Toxo
											CNS lymphoma

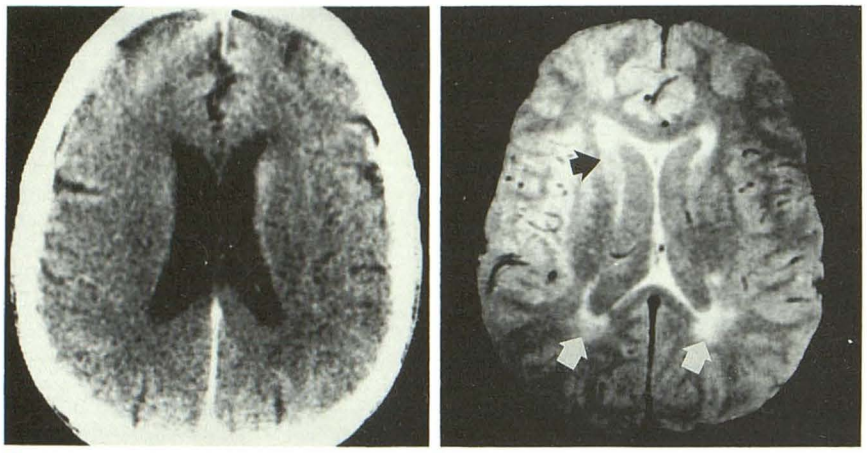

Fig. 1.—Case 28: 52-year-old man who was admitted with lower extremity weakness and central neurologic signs.

***A*, Contrast-enhanced CT scan is normal.**

***B*, Axial T2-weighted MR image (2000/40) shows hyperintense foci in periventricular region (*arrows*). Autopsy revealed loss of myelin in periventricular white matter. These changes resulted from direct HIV infection.**

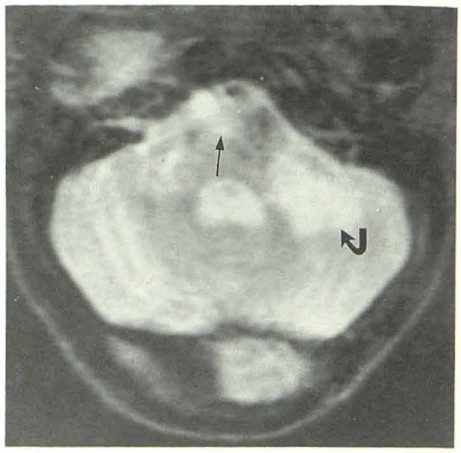

Fig. 2.—Case 25: 37-year-old man admitted with mental status changes. Axial T2-weighted MR image (2000/80) shows foci of hyperintensity involving the pons (*straight arrow*), left middle cerebellar peduncle, and adjacent cerebellum (*curved arrow*). These findings represent a case of AIDS encephalopathy with associated clinical toxoplasmosis. The patient's symptoms were relieved with antitoxoplasmoid medications.

AIDS and ARC as established by the Center for Disease Control (CDC) [17]. Forty-seven patients were established as fitting the CDC criteria for AIDS and/or ARC, of which 38 were positively diagnosed with AIDS and nine were diagnosed with ARC. Thirteen patients were not included in this study because their medical charts lacked the CDC criteria for AIDS or because of the presence of other neurologic illnesses. Risk factors included homosexuality, IV drug use, and bisexual practices. Forty-six patients were men and one was a woman. The ages ranged from 21 to 57 years old. The MR examinations were reviewed by two attending neuroradiologists and one radiology fellow in training. The MR studies were performed on a 1.5-T superconductive magnet (General Electric, Milwaukee). Technical factors were as follows: sagittal, axial, and coronal images; 5-mm section thickness; 256 × 128 or 256 × 256 matrix, a short repetition time (TR) of 800 msec; a long TR of 2000–2500 msec, echo times (TE) of 20, 70, 80; two excitations; 20- or 24-cm field of view, and a spin-echo (SE) multisection acquisition technique. The short TR, short TE provided T1-weighted images. The SE sequence (axial and coronal) of long TR and short TE provided proton-density images, and the long TR and long TE provided the T2-weighted images. The CT studies were performed on General Electric 8800 and 9800 CT/T scanners (General Electric, Milwaukee) using a 5- or 10-mm slice thickness. Twenty patients had contrast-enhanced CT studies with 150 ml of 60% meglumine diatriazoate, while 27 examinations were done without contrast enhancement.

Results

Table 1 is a summary of the clinical data and imaging results for the 47 patients included in the study. All were members of AIDS risk groups: 18 were homosexuals, 27 had a history of IV drug abuse, and two were in both risk categories. White matter disease, which was the single most common MR finding, was classified as either diffuse or focal according to neuroanatomic extension into particular brain structures. Of the 47 patients studied, 23 (49%) exhibited focal white matter disease manifested by high-intensity signals on T2-weighted images (Figs. 1 and 2). Six (13%) of the patients exhibited diffuse high signal intensity. Diffuse white matter disease was further subdivided, and three (6%) of the patients exhibited diffuse unilateral white matter involvement (Fig. 3), while three patients (6%) demonstrated bilateral white matter involvement (Fig. 4). Seventeen (36%) of our patients had white matter lesions on initial CT imaging, while 12 patients (26%) whose initial CT scan demonstrated cortical atrophy revealed white matter lesions on MR images. The histological diagnoses of 11 patients (23%) are known. Six patients were diagnosed at autopsy; five patients were diagnosed from biopsy specimens. Four of the autopsy-diagnosed cases revealed multinucleated giant cells with HIV viral particles along with changes in the white matter, and all demonstrated white matter disease on MR. One of the biopsy-proved cases resulted in the diagnosis of progressive multifocal leukoencephalopathy, which correlated with the white matter disease demonstrated on the patient's MR scan.

One interesting observation was that of prominent involvement of the external and extreme capsules and of the white matter along and in the vicinity of the optic radiation (Figs. 5 and 6). Atrophy of the cerebral cortex and cerebellar cortex is a common manifestation of AIDS-related CNS pathology. Of the 47 patients, 17 (36%) manifested cerebral atrophy with or without ventricular enlargement. Seven (15%) were noted to have brainstem atrophy and 18 (38%) were noted to have cerebellar atrophy. Sixteen (34%) of these patients were noted to have vermian atrophy. Mass lesions were subdivided into toxoplasmosis (Fig. 7) and nontoxoplasmoid infections.

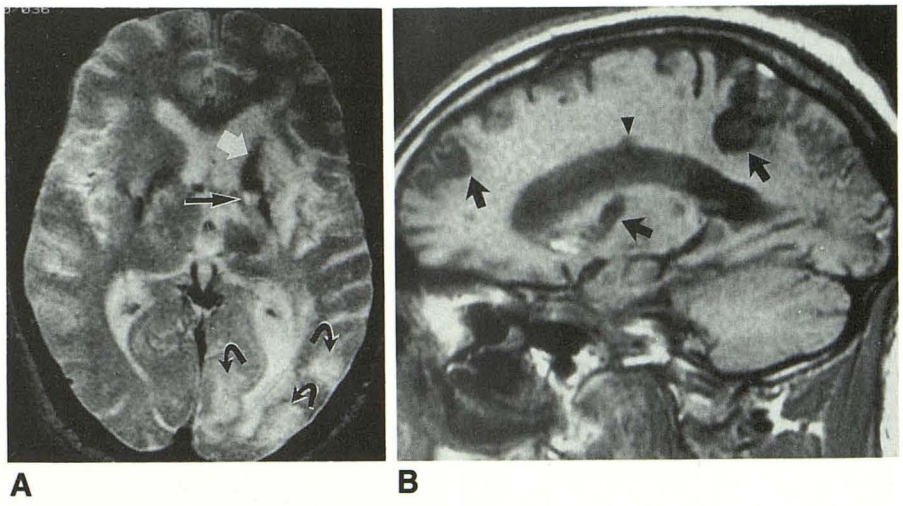

Fig. 3.—Case 27: 37-year-old man admitted with visual and auditory perception loss.

A, Axial T2-weighted MR image (2000/80) shows diffuse unilateral area of hyperintensity involving left subinsular and paratrigonal regions. Notice that left external and extreme capsules, along with basal ganglia and paraventricular structures, are also involved. Small foci of hyperintensity (*straight arrow*) seen at genu of internal capsule represent minimal involvement. These findings represent a demyelination secondary to HIV infection. There are several foci of hyperintensity in the parietooccipital lobe, which represent presumed toxoplasmoid abscesses (*curved arrows*). This case represents HIV infection with associated clinical toxoplasmosis. Patient's symptoms were relieved with antitoxoplasmoid medications. *Large white arrow* represents noninvolvement of anterior limb of internal capsule.

B, Sagittal T1-weighted MR image (600/20) shows numerous presumed abscesses involving the deep white matter and basal ganglia (*arrows*). Note a small hypointense region involving the corpus callosum adjacent to the lateral ventricle (*arrowhead*). This is thought to represent another abscess.

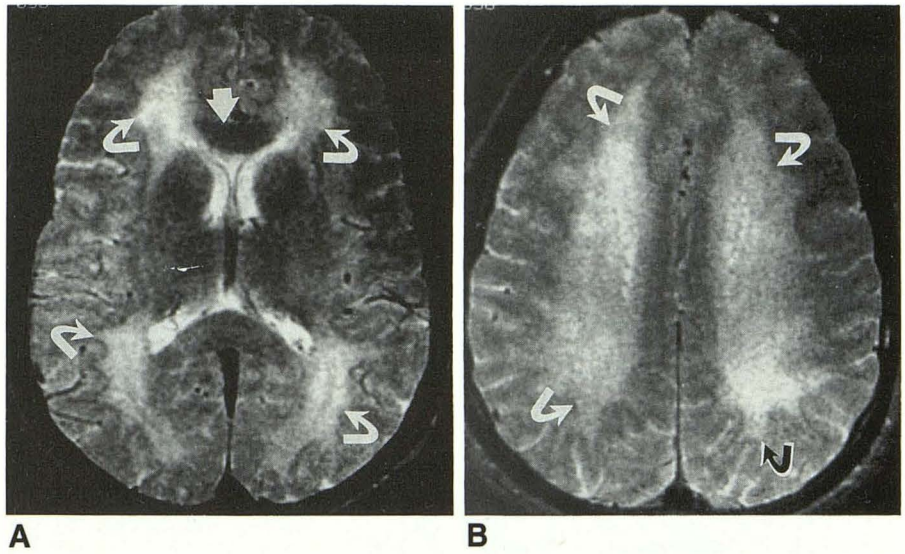

Fig. 4.—Case 19: 39-year-old man admitted with AIDS dementia complex.

A and *B*, Axial T2-weighted MR images (2000/80) show bilateral almost symmetrical demyelination of white matter (*curved arrows*) involving the paraventricular and centrum semiovale. Notice sparing of corpus callosum (*straight arrow* in *A*). These changes were thought to represent direct involvement by HIV resulting in subacute encephalopathy of AIDS.

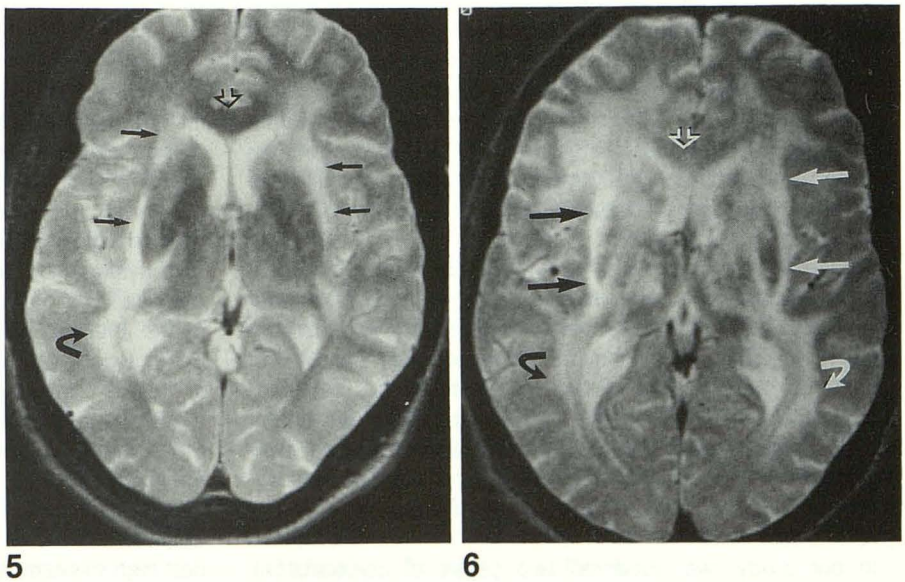

Fig. 5.—Case 21: 32-year-old man with dizziness and a normal neurologic examination. Axial T2-weighted MR image (2000/80) shows a demyelinating process that predominates in the external and extreme capsules (*straight arrows*). Notice involvement along the right optic radiation (*curved arrow*). The internal capsules and basal ganglia are almost spared. Note also the sparing of corpus callosum fibers (*open arrow*). Although no biopsy was performed, these lesions were thought to represent HIV demyelination.

Fig. 6.—Case 3: 49-year-old man with weight loss and AIDS dementia complex. Axial T2-weighted MR image (2000/80) shows demyelination along external and extreme capsules (*straight arrows*) and optic radiations (*curved arrows*). The basal ganglia and internal capsules are also involved bilaterally, although to a lesser extent. The corpus callosum fibers are spared (*open arrow*). This was a case of clinical toxoplasmosis. The patient's symptoms were alleviated after treatment with antitoxoplasmoid medication.

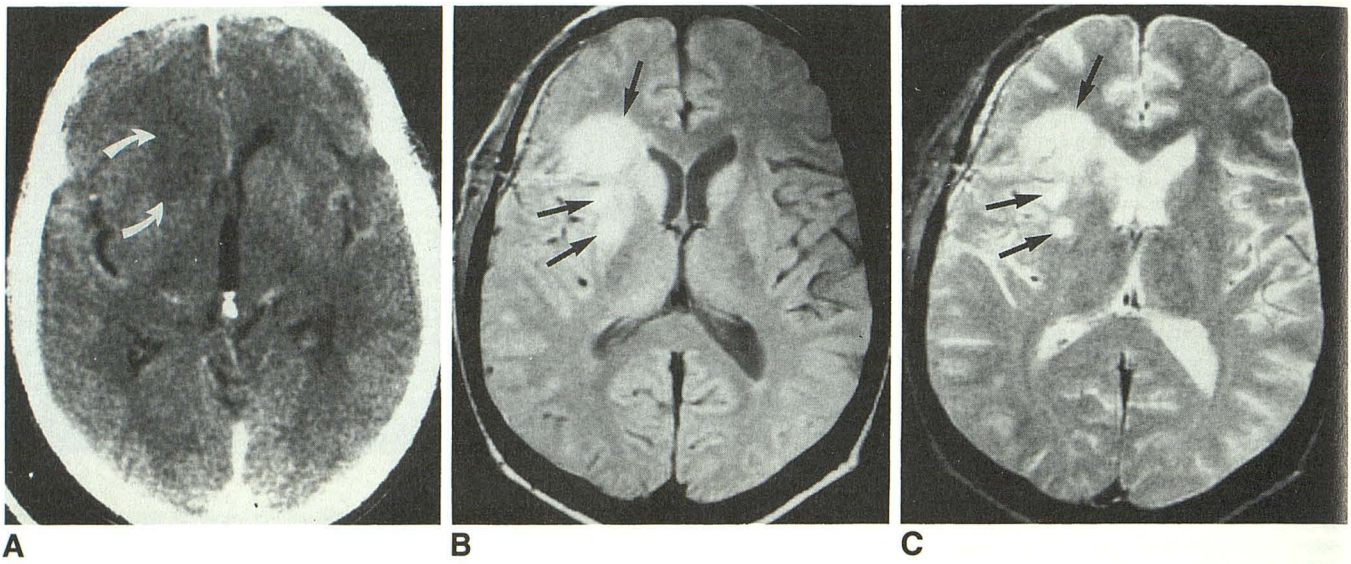

Fig. 7.—Case 24: 35-year-old man admitted with mental status changes. This is a biopsy proved case of toxoplasmosis.
***A*, Contrast-enhanced CT scan shows ill-defined nonenhancing mass (*arrows*) in right frontal lobe with compression of adjacent frontal horn.**
***B* and *C*, Proton-density (*B*) and T2-weighted (*C*) (2000/80) show three cerebral abscesses (*arrows*) not evident on CT scan. Notice postcraniotomy changes and slight amount of epidural fluid on the right side.**

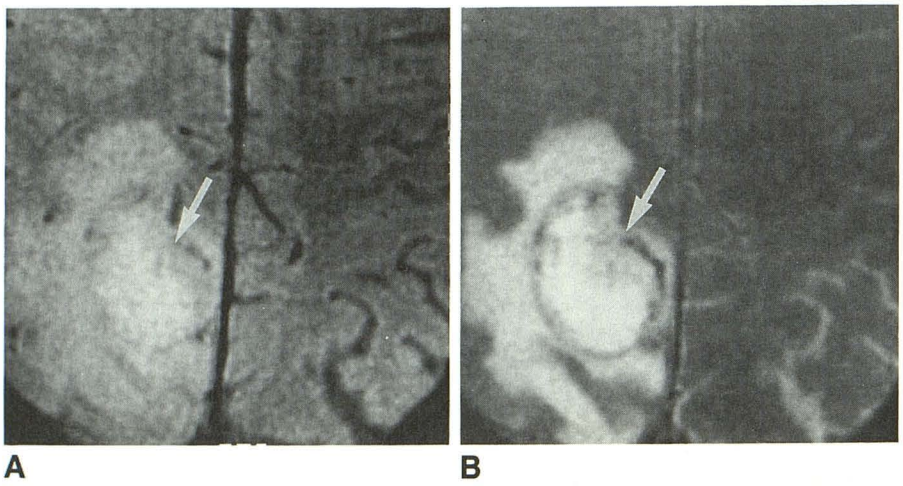

Fig. 8.—Case 46: 49-year-old man with biopsy proved toxoplasmosis.
***A* and *B*, Proton-density (*A*) and T2-weighted (*B*) axial MR images (2000/80) show abscess (*arrow*) with marked peripheral edema surrounding it. Biopsy revealed toxoplasmosis.**

Of 47 patients, eight (17%) were diagnosed as having toxoplasmosis and of these, two cases were biopsy proved (Figs. 7 and 8), one diagnosed at autopsy and five diagnosed clinically after lesions were observed to regress on CT and MR scans following treatment with antitoxoplasmoid medications.

Three (6%) of 47 patients were diagnosed as having CNS lymphoma. One case was autopsy proved, one case was biopsy proved, and the third case was presumptive based on class IV neoplastic lymphocytes on cytology smear of the CSF (Figs. 9 and 10). Primary CNS lymphoma exhibited enhancement on MR scans after administration of gadopentetate dimeglumine (Fig. 9). We also observed two cases of systemic non-Hodgkin lymphoma: One case involved the scalp and the other was diagnosed by biopsy of the axillary lymph node.

In our study, we observed two cases of cryptococcal meningitis that were both diagnosed at autopsy without any lesions detected on nonenhanced MR images. One case of mycobacterial tuberculous meningitis was demonstrated at autopsy in one patient. Two cases of CNS infarction were observed. It is quite difficult to assay the significance in one of these patients who was 57 years old and may have had underlying atherosclerotic disease. On autopsy this patient exhibited HIV particles around the small arteries in the region of the infarcts. The other case of CNS infarction was most likely secondary to involvement of the basal ganglia with infectious toxoplasmosis.

Among the 47 patients studied, MR findings revealed ethmoid sinusitis in seven (15%), maxillary sinusitis in six (13%), sphenoid sinusitis in five (11%), and otomastoiditis in five (11%). Radiologic findings revealed pansinusitis in three patients (6%). Eleven (23%) of the patients in our study group had high-intensity signals on T2-weighted images in the na-

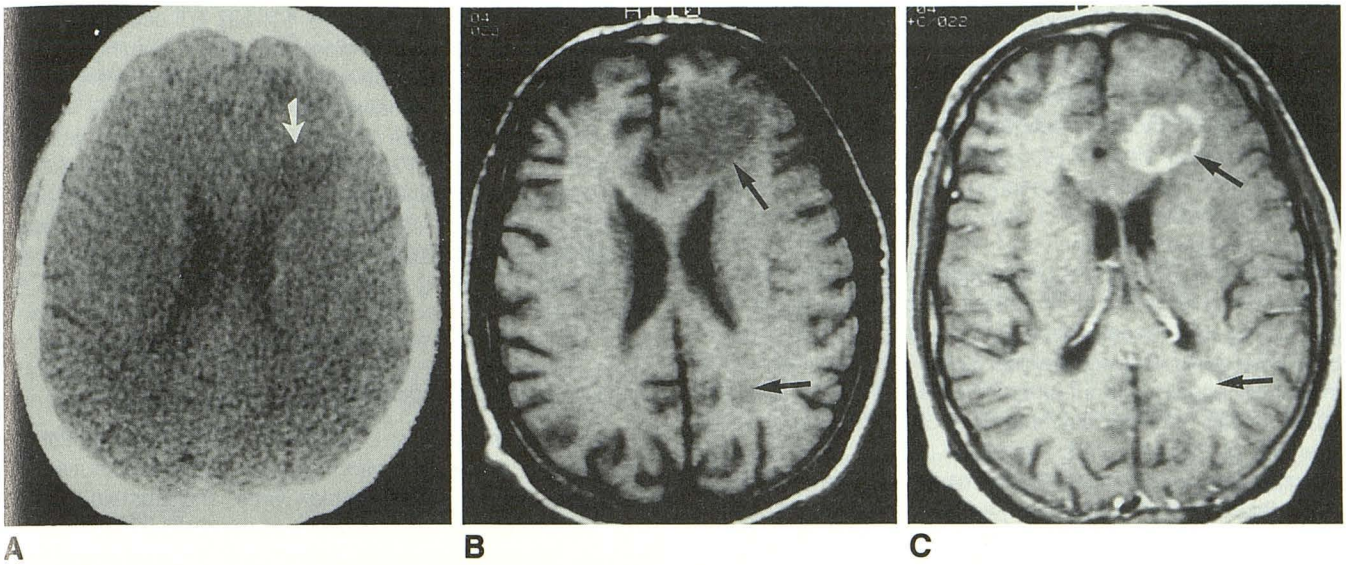

Fig. 9.—Case 47: 32-year-old man admitted with mental status changes along with right lower extremity weakness.
***A*, Contrast-enhanced CT scan shows ill-defined nonenhancing lesion (*arrow*) involving left frontal lobe.**
***B* and *C*, Pre- (*B*) and post- (*C*) gadopentetate dimeglumine enhanced T1-weighted MR images show two lesions (*arrows*) with enhancement. At autopsy, primary CNS lymphoma was confirmed.**

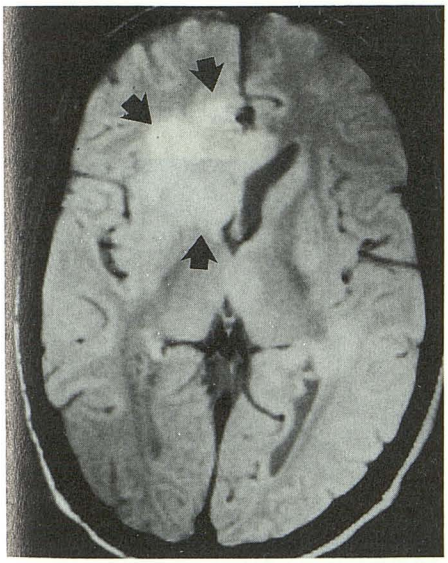

Fig. 10.—Case 32: 27-year-old man admitted with persistent headaches. CT scan (not shown) revealed a hypodense lesion in the head of the right caudate nucleus. Proton-density MR image (2000/20) shows hyperintense lesion involving the right caudate nucleus and its surrounding tissue with involvement of the corpus callosum. Biopsy proved primary CNS lymphoma.

sopharynx and parotid gland, suggesting lymphoid hypertrophy and/or hyperplasia.

Discussion

Neuroimaging studies play an important role in the evaluation of AIDS patients [4, 5, 10–14]. It is imperative that the radiologist correlate findings with the patients' symptomatology. MR is superior to CT in the evaluation of AIDS patients with encephalopathy because of its sensitivity for detecting white matter lesions and inflammatory diseases of the CNS [10, 13–15, 17]. Many others have demonstrated the importance of MR in the evaluation of deep-seated parenchymal lesions even when the CT scan was normal (Fig. 1) [10, 11, 13, 15, 17].

White Matter Disease

The AIDS dementia complex is the most common CNS entity observed in AIDS patients [4, 5, 18, 19]. It is heralded by a clinical triad of motor, cognitive, and behavioral dysfunctions and its chief imaging manifestation is that of atrophy [4, 5, 18, 19]. Generalized cortical atrophy, with or without ventricular dilatation, accompanies memory loss and other forms of cognitive impairment [18, 19]. Although the pathophysiological process is unknown, it is currently believed that the atrophy and dementia are most likely caused by diffuse encephalitis, most commonly from HIV but also from cytomegalovirus, herpes, papovaviruses, and, less often, toxoplasmosis [4, 5, 18, 19]. The pathologic hallmark of HIV encephalitis is the presence of multinucleated giant cells located primarily in the white matter and less commonly in the gray matter [7, 8, 18, 19]. This results in myelin pallor and rarefaction of central cerebral white matter [7, 8]. Cytomegalovirus was initially considered to be the etiological agent, but more recently it has been shown that direct invasion of the neural elements by HIV is the cause [4, 7, 19]. Both CT and T2-weighted MR scans may demonstrate periventricular lesions associated with HIV encephalitis (Figs. 1, 5, and 6) [10, 18, 20]. Their appearance on both CT and MR may be nonspecific, resembling that of other nonfocal mass lesions in AIDS (Fig. 10) [10, 11, 13, 15, 18].

Progressive Multifocal Leukoencephalopathy

Progressive multifocal leukoencephalopathy (PML), characterized by chronic demyelination of the CNS, is caused by papovaviruses, Jakob-Creutzfeldt disease, and less frequently by simian virus-40 [4, 6, 8, 21]. Before the emergence of AIDS, PML was primarily seen in patients with lymphoproliferative disorders [4–6, 21]. Various studies have reported the observance of this entity in 1–5% of AIDS patients [4, 5, 7, 11, 13, 21]. The illness often begins with subtle signs of personality change, memory deficits, or cognitive impairment [4, 5, 21]. CT reveals decreased density in the cerebral hemispheres or the cerebellum, often affecting the white matter but usually without enhancement [10–12, 14, 21]. MR often reveals multiple asymmetric foci of increased signal intensity on T2-weighted images [4, 10–13, 18, 20, 21]. Definite diagnosis is made by examining a biopsy specimen of the lesion.

Toxoplasmosis

Toxoplasmosis, the infection caused by the protozoan *Toxoplasma gondii*, is a common infection in humans and other animals [4–6]. Serologic evidence of past infection is found in 20–70% of adults [4, 6]. In the immunocompetent individual, acute infection is subclinical and produces a self-limited febrile illness with lymphadenopathy. CNS toxoplasmosis accounts for 10–15% of infections in AIDS patients and may present as a subacute meningoencephalitis, diffuse encephalitis, or a space-occupying lesion [4, 6, 10–14]. The cerebral hemispheres are nearly always involved [4, 7, 8]. Fifty to seventy-five percent of toxoplasmosis cases involve the subcortical structures, most often the basal ganglia (Figs. 3, 6, 7A) [4, 7, 8]. Most cases evolve in a subacute manner with focal findings developing over a period of days to weeks during which nonlocalizing features such as headaches, lethargy, and cognitive impairment are evident [4–6]. CT often reveals enhancing lesions with surrounding edema and evidence of mass effect [10–15]. The lesions may not enhance on CT scans (Fig. 7A). MR, which is more sensitive in identifying focal mass lesions, especially in the cortex and subcortical deep gray matter, shows the *Toxoplasma* abscess as low intensity on T1-weighted images (Fig. 3B) and medium- to high-intensity signals on proton-density and T2-weighted images (Figs. 7B, 7C, 8A, 8B) [11–15]. Autopsy reports have shown three histologic types of abscesses: organized, necrotizing, and chronic [7]. The clinical diagnosis of toxoplasmosis is suggested by a rising serum specific antibody titer for *Toxoplasma* but definite diagnosis depends on examination of a biopsy specimen of the lesion (Figs. 7 and 8).

Cryptococcosis

Cryptococcus neoformans, the soil fungus, may cause CNS disease in normal hosts but is more likely to do so in patients with underlying immunodeficiency. There are many reports of CNS cryptococcosis in AIDS patients [4–8]. The disease manifests as a subacute to chronic granulomatous meningitis with nonlocalizing features [4–6, 11, 13]. CT scans are often normal or reveal diffuse enhancement of the meninges along with nonspecific cortical atrophy [4, 10–15, 18]. More rarely, intraparenchymal cryptococcomas may occur [4, 6, 10–15, 18]. MR images may be normal or show either mild enlargement of the ventricle or cortical atrophy [10–13, 18]. The CSF protein is generally elevated, often with accompanying pleocytosis. CSF culture, Indian ink staining, and the cryptococcal antigen assay are all useful in the diagnosis. Definite diagnosis is made by examining a biopsy specimen or at autopsy [4, 5, 7, 8].

Aseptic Meningitis and Other Infections

Aseptic meningitis is common in AIDS patients and is usually due to HIV infection but may also be caused by cytomegalovirus, herpes simplex virus, and Epstein-Barr virus. Very rarely, fungal infections of the CNS include aspergillosis, coccidioidomycosis, histoplasmosis, and candidiasis [4, 7, 8]. *Mycobacterium tuberculosis* and *Mycobacterium avium-intracellulare* meningitis are not observed frequently [4, 5, 7].

Tumors and Primary CNS Lymphoma

Primary brain lymphomas account for less than 2% of primary brain tumors [4–6]. Although much less common than

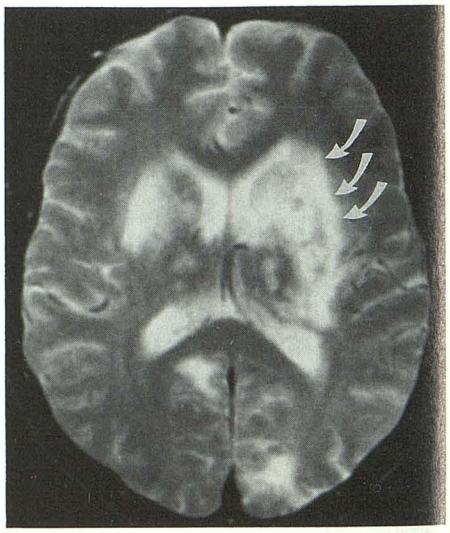

Fig. 11.—Case 7: 29-year-old man with positive toxoplasmosis titers combined with a malignant lymphocyte profile on CSF analysis. Axial T2-weighted MR image (2000/80) shows hyperintense lesions involving basal ganglia, with left greater than right (*arrows*). Notice involvement of internal capsules; left external and extreme capsules with involvement of insular cortex. Although this patient's serology was positive for toxoplasmosis, his CSF demonstrated evidence of malignant lymphocytes secondary to primary CNS lymphoma. This case emphasizes the importance of doing a biopsy of a mass lesion to differentiate toxoplasmosis from primary CNS lymphoma.

toxoplasmosis, this entity is now seen in 6% of AIDS patients [6, 10, 15, 19]. Primary CNS lymphoma generally presents as an expanding cerebral mass lesion (Figs. 9 and 10), which may be indistinguishable from toxoplasmosis on imaging studies (Fig. 11) [10–14]. Pathologically, these tumors are of high grade, either the immunoblastic sarcoma type or the small, noncleaved cell type [4–8]. On CT scans these lesions are hypodense (Fig. 9A) and often enhance irregularly in a nodular or ringlike pattern. Surrounding edema may be prominent [10–14]. MR images reveal regions of low intensity on T1-weighted scans (Fig. 9B), and there is enhancement after IV administration of gadopentetate dimeglumine (Fig. 9C). The lesion, along with surrounding edema, becomes hyperintense on proton-density and T2-weighted images (Fig. 10) [10–15, 17].

Other Lesions

Lymphoid hyperplasia is an entity known to accompany other lesions in patients with AIDS. It most often involves intraparotid, adenoidal, or pharyngeal lymph nodes [22, 23]. MR demonstrates enlargement of lymph-bearing tissues with intermediate signal intensity on T1-weighted images and moderate to high signal intensity in T2-weighted images [22, 23].

Although sinusitis and otomastoiditis (unexplained serous otitis media) have not formerly been identified as an added feature of AIDS, one has to consider immunodeficiency in a patient with unexplained sinusitis and otomastoiditis.

Summary

On the basis of our study and on the findings of previous reports [10, 20], we recommend that in patients who have observed neurologic complaints or who fit the CDC criteria for AIDS or ARC, an MR scan with and without gadopentate dimeglumine should be the initial diagnostic procedure. If an enhancing mass is identified on the initial MR or CT scans and the patient is also deteriorating clinically after treatment for presumptive toxoplasmosis, a stereotaxic biopsy is indicated to establish diagnosis and guide treatment [25, 26]. If both MR and CT are negative, then repeat studies should be obtained. In this study, MR was far superior in demonstrating white matter disease even when the CT scan was normal. Twelve (26%) of our patients whose initial CT scans were reported as normal or as showing only mild cortical atrophy demonstrated white matter disease on MR.

White matter disease is the most common finding in subacute encephalopathy of AIDS and, according to our study, preferentially strikes the subinsular and peritrigonal white matter fibers. It has been observed that the demyelination that is demonstrated at autopsy is most likely secondary to invasion of neural tissue by HIV-1 along with intimal changes of the small blood vessels, leading to myelin loss. Other viral syndromes, such as progressive multifocal leukoencephalopathy, cytomegaloviruses, and herpes encephalitis, as well as toxoplasmosis and primary CNS lymphoma, can only be excluded by examining a biopsy specimen.

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