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Abnormalities of the Brain in AIDS Patients: Correlation of Postmortem MR Findings with Neuropathology

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The ability of MR to detect CNS lesions in AIDS patients was evaluated by postmortem scanning of 10 formalin-fixed brains. Nine patients had premortem mental status changes and five had focal neurologic deficits. The brains were imaged and sectioned in corresponding planes. MR images showed atrophy in eight of the 10. All grossly identified lesions and areas of MR abnormality were histologically evaluated. Areas of infarction and necrosis associated with cytomegalovirus (CMV) or *Toxoplasma gondii* were seen as foci of increased signal intensity. Severe ventriculitis and focal gliosis were also visible by MR. Neither CT nor MR was able to detect diffuse CMV- or HIV-associated microglial nodules. Dementia without focal neurologic signs correlated best with the presence of diffuse microglial nodules at pathology.

Our results demonstrate the usefulness of correlating postmortem MR imaging with neuropathology, and the relevance of postmortem findings to the interpretation of MR images in living patients.

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Patients with AIDS may develop numerous neurologic abnormalities, and they frequently have neurologic symptoms at initial presentation. Neuroradiologic imaging studies, including CT and MR, are important in the clinical assessment of these patients. The most frequent neuroradiologic finding in AIDS is cerebral atrophy [1–3]. Although CT is usually effective for detecting parenchymal CNS mass lesions, diffuse diseases that do not produce mass effect—such as infection by cytomegalovirus (CMV), human immunodeficiency virus (HIV), or papovavirus—are more difficult to detect by CT [1–5]. MR is reportedly more sensitive for the detection of these infections [1, 4], but neither MR nor CT has yet been able to differentiate the various causes of encephalopathy in AIDS. Nevertheless, some investigators have proposed that certain radiologic patterns are suggestive of specific diseases [4, 5]. Several prior investigations have involved retrospective analysis of neuroradiologic studies in correlation with pathologic findings at the time of autopsy [1–5]. Interpretation of this type of study is difficult because of the progression of neurologic disease in AIDS that occurs between the time of radiologic examination and the time of death and pathologic examination. Correlation of radiologic studies with biopsy results may be misleading as well, since there is a high frequency of coexistent pathologic processes in the CNS of AIDS patients, making sampling error a critical problem. These limitations make it difficult to determine both the sensitivity and specificity of neuroradiologic imaging studies.

To better understand the usefulness of MR in the diagnosis of neurologic abnormalities in AIDS, we compared postmortem MR characteristics of 10 brains from AIDS patients with detailed gross and microscopic neuropathologic examinations. This approach eliminates the problem of interval changes between radiologic study and pathology, and allows for extensive histologic sampling of the nervous system, including all radiologically abnormal areas. These studies provide important MR-neuropathologic correlation that will be useful in clinical evaluations.

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Differences in MR signal characteristics might be anticipated with the use of formalin-fixed brains as compared with unfixed tissues, but previous studies have confirmed the validity of this method for the detection of CNS lesions [6–10].

Materials and Methods

The study population consisted of 10 men ages 24–51 with clinical and serologic diagnoses of AIDS. The cases selected were 10 sequential AIDS patients who came to autopsy during the period from July to December 1987. Only cases in which a full autopsy was permitted were included. Nine of the 10 patients had premortem mental status changes, including dementia and decreased level of consciousness (Table 1). Five had focal neurologic deficits, including weakness, ataxia, and seizures. One patient had several premortem MR examinations 1–4 months before death and four patients had premortem CT (pre- and postcontrast) examinations within 2 months of death. These studies were also reviewed.

Complete autopsies were performed in all cases, with postmortem times ranging from 4–48 hr. At autopsy, all patients had opportunistic infections identified in the systemic autopsy; CMV and *Pneumocystis carinii* pneumonia were each detected in eight patients. The brains were placed in 20% neutral buffered formalin and fixed for at least 2 weeks.

After fixation, the formalin was drained from each specimen and 5-mm-thick, interleaved, proton-density-weighted and T2-weighted images, 2800/20,70/2 (TR/TE/excitations), were obtained with a 1.5-T superconducting magnet (Signa, General Electric, Milwaukee, WI). We used a 16-cm field of view with a 256 × 256 matrix corresponding to a spatial resolution of 0.62 mm. Images of every specimen were acquired in the anatomic coronal plane. Transaxial images of the brainstem were also acquired to correspond with routine sectioning methods used in neuropathologic examination. In a prospective viewing of the MR studies, all lesions were identified jointly by three neuroradiologists. Brain parenchymal volume was also assessed on the MR studies. The pattern of cerebral atrophy (central and/or peripheral) and its severity (none, mild, moderate, or severe) were graded subjectively in each case. Dilatation of the ventricular system (especially the lateral and third ventricles) was interpreted as *central* atrophy; prominence of the supratentorial sulci with narrowing of the gyri was interpreted as *peripheral* atrophy. After the histopathologic studies were completed, the MR studies were reviewed again with knowledge of the pathologic results to evaluate any discrepancies.

Each brain was sectioned in 5-mm-thick slices directly corresponding to the MR images. The cerebral hemispheres were sectioned in the coronal plane, the brainstem was sectioned transversely, and the cerebellar hemispheres were sectioned in the sagittal plane. Each slice was examined visually for any lesions by two neuropathologists. The size of the ventricular system was evaluated and any dilatation observed was subjectively graded as none, mild, moderate, or severe. Blocks of tissue were taken for histology from standard, representative regions of the brain (frontal, temporal, parietal, and occipital cortex, hippocampus, thalamus, hypothalamus, basal ganglia, periventricular white matter, midbrain, pons, medulla, cerebellar cortex, and deep gray and white matter) and spinal cord, as well as from regions of gross and/or MR abnormality. If lesions were found on visual examination, 3-mm-thick blocks of those regions were sampled for histologic studies. If no lesions were seen by gross inspection, yet the MR indicated an abnormality, successive 5-mm slices were sectioned to a thickness of 2.5 mm in an attempt to discern the lesion. If still no abnormality was seen, tissue blocks were taken through the region of MR abnormality and embedded for histologic study. All blocks were routinely stained with hematoxylin and eosin (H and E). Immunoperoxidase stains for glial fibrillary acidic protein (GFAP) were employed when H and E studies were unremarkable but abnormalities were seen by MR. When appropriate, further studies were performed, including immunoperoxidase stains for CMV, HIV, and toxoplasma [11]; acid-fast stain (Fite) for mycobacteria; and mucicarmine stain for cryptococcus.

Results

Atrophy was the only abnormality detected in the four premortem CT scans. Eight of the 10 brains demonstrated varying degrees of atrophy by postmortem MR. The locations and histologic features of all lesions seen by MR and by pathology are described in Table 2. All focal abnormalities that were detected in the MR scans were less than 1 cm in size and hyperintense relative to normal white matter on the proton-density- and T2-weighted sequences. The lesions were more conspicuous on the T2-weighted images, except when adjacent to a sulcus or ventricle filled with high-signal-

TABLE 1: Neurologic Findings

Case No.	Mental Status Changes	Physical Findings
1	Hallucinations, delusions	Headache*
2	Decreased level of consciousness	Seizures, painful polyneuropathy
3	None	None
4	Memory problems	Seizures; diplopia; slurred speech; snout, glabellar, Babinski +
5	Confusion, hallucinations	None
6	Dementia	Ataxia, nausea, vomiting; left-sided weakness
7	Decreased level of consciousness	None
8	Anxiety, decreased concentration	Parkinsonism (phenothiazine), bilateral upper extremity weakness
9	Decreased level of consciousness	None
10	Dementia	Weakness, seizure

* History of Guillain-Barré, 1981.

TABLE 2: Lesions Identified by MR and Pathology

Case No.	MR Focal High-Intensity Lesions	Pathologic Findings
1	Right inferior basal ganglia	Vessels/Virchow-Robin spaces
	NS	Microglial nodules (CMV), diffuse
	NS	Cryptococcal cyst, occipital white matter
	NS	Cryptococcal meningitis
2	Right inferior basal ganglia	CMV necrosis, prominent vessels
	Right dentate nucleus, cerebellum	CMV necrosis
	Left dentate nucleus, cerebellum	CMV necrosis
	NS	CMV necrosis, cervical spinal cord
	NS	Microglial nodules (CMV), diffuse
	NS	CMV ventriculitis, diffuse, mild
	NS	Acid-fast bacilli, choroid plexus
3	Inferior basal ganglia, bilateral	Vessels/Virchow-Robin spaces
	Cerebellar white matter, bilateral (one each)	NS
4	Left anterior basal ganglia	Vessels/Virchow-Robin spaces
	Right cerebellar white matter, small	NS
	NS	Microglial nodules (HIV), diffuse
5	White matter lateral to right posterior thalamus	NS
	Right cerebellar white matter	Gliosis, mild
	Inferior basal ganglia, bilateral	Vessels/Virchow-Robin spaces
	NS	Gliosis, mild, right frontal white matter
6	Periventricular, diffuse	CMV ventriculitis
	Periventricular white matter, right inferior	CMV ventriculitis, no focal lesion
	Periventricular white matter, left thalamus	CMV ventriculitis, no focal lesion
	Right inferior anterior internal capsule	Microglial nodules (CMV), aggregate
	Right inferior lateral thalamus	NS
	Right lateral thalamus–internal capsule	Microglial nodules (CMV), aggregate
	Left superior putamen	NS
	Occipital white matter	CMV necrosis
	Cerebellar white matter, bilateral (four foci)	CMV necrosis, toxoplasmosis
	Right medulla (decreased signal intensity)	CMV necrosis, toxoplasmosis
	NS	CMV necrosis, hippocampus
	NS	CMV necrosis, cerebellar cortex
	NS	CMV necrosis, dentate nucleus cerebellum
7	Deep cerebellar white matter, bilateral	NS
	NS	Microglial nodules (CMV), diffuse
	NS	Subependymal gliosis, occipital
8	Putamen, bilateral, multiple	Microglial nodules (HIV), multiple, necrosis
	Right frontal white matter	Gliosis, mild
	Occipital white matter	NS
9	Left thalamus	NS
	Left temporal deep white matter	NS
	Basal ganglia, bilateral	NS
	Right middle cerebellar peduncle	NS
	Left cerebellar white matter	NS
	Pons (two central foci and one in left upper pons)	NS
	NS	Microglial nodules,* diffuse
10	Right inferior caudate	NS
	Thalamus, bilateral	Vessels/Virchow-Robin spaces
	Left temporal white matter	Vessels/Virchow-Robin spaces
	Pons, mid (two foci)	Microglial nodules,* aggregate
	NS	Microglial nodules,* spinal cord
	NS	Microglial nodules,* hypothalamus

Note.—NS = not seen.

* Origin of microglial nodules (CMV or HIV) could not be determined.

intensity formalin. Many of the lesions detected by MR were located in the deep white and gray matter of the cerebrum and cerebellum. Several lesions were identified in the subcortical white matter of the cerebral hemispheres (frontal, temporal, and occipital lobes) and in the brainstem.

Comparison of MR Findings and Pathology

Atrophy. Atrophy was a frequent postmortem MR finding (80%). The atrophy was central in distribution in one case, peripheral in two, and mixed central and peripheral (with a central predominance) in five (Table 3). At autopsy, two of the brains had moderate dilatation of the lateral ventricles, four had mild dilatation, and four were judged to have normal-sized ventricles. The presence of ventricular enlargement at autopsy correlates well with central atrophy as identified by MR ($r_s = 0.955$; $p < .001$, Spearman rank correlation coefficient). The brain weights ranged from 1140–1550 g (mean = 1386). Brain weight did not correlate with either ventricular enlargement at autopsy ($r_s = 0.224$; NS, Spearman rank correlation coefficient) or with atrophy seen by MR ($r_s = 0.405$; NS, Spearman rank correlation coefficient).

MR true-positive focal lesions. A summary of the identification of focal lesions by MR and pathology is presented in Table 4. Representative cases are illustrated in Figures 1 and 2. MR detected 17 (81%) of 21 regions of necrosis/infarction; 15 were associated with CMV infection, two with HIV, and four with toxoplasmosis coexistent with CMV. In four foci of MR abnormality (in two cases), the only pathology was the presence of microglial nodules (MGNs), without significant necrosis. In these foci, multiple MGNs formed *aggregate* lesions that were greater than 3 mm in size, rather than single

TABLE 3: Evaluation of Atrophy by MR and Pathology

Case No.	Atrophy by MR	Ventricles at Autopsy	Brain Weight (g)
1	Central, mild	Mild dilatation	1550
2	None	Normal	1550
3	Peripheral, mild	Normal	1140
4	Central > peripheral, moderate	Mild dilatation	1500
5	Peripheral, moderate	Normal	1460
6	Central > peripheral, moderate	Moderate dilatation	1400
7	Central > peripheral, moderate	Moderate dilatation	1350
8	None	Normal	1450
9	Central > peripheral, mild	Mild dilatation	1260
10	Central > peripheral, mild	Mild dilatation	1150

TABLE 4: Detection by MR of Lesions Listed by Pathologic Features

	Histology	MR+	MR−
Infarction/necrosis	21	17	4
Microglial nodules	Innumerable	Four foci	Innumerable
Ventriculitis	2	1	1
Meningitis	1	0	1
Focal gliosis	4	2	2
Other	1 (choroid plexus AFB)	0	1
	1 (cryptococcal cyst)	0	1

Note.—AFB = acid-fast bacilli.

MGNs sparsely scattered throughout the parenchyma. One of two cases of ventriculitis (associated with CMV) was seen by MR. Two of four foci of *focal* gliosis were seen by MR. The majority of the lesions detected by MR were in the cerebral and cerebellar white matter (15/21).

MR false-negative for detection of pathologic findings. MR imaging failed to detect the vast majority of MGNs, which occurred diffusely in seven (70%) of 10 cases, not only in the deep white and gray matter, but also in the cerebral and cerebellar cortex. In four cases diffuse MGNs were associated with CMV, in one case diffuse MGNs were associated with HIV, and in two cases the specific causative agent producing the MGNs could not be identified. MR was also relatively insensitive in detecting gliosis; two of four cases of focal gliosis were not detected. *Diffuse* white matter gliosis not associated with other lesions was seen histologically in six cases, and was not recognized by MR. Lesions located near CSF spaces were poorly detected by MR. These included: (1) regions of necrosis in the cerebral and cerebellar cortex adjacent to sulci, (2) CMV necrosis in the pineal gland, (3) mild CMV ventriculitis, (4) choroid plexus mycobacteria, and (5) cryptococcal meningitis and a single cryptococcal cyst. The small size (70 μm) of the latter lesion is the most likely explanation for its remaining undetected by MR. Pathologically identified lesions were also located predominantly in the deep structures of the cerebral hemispheres and cerebellum,

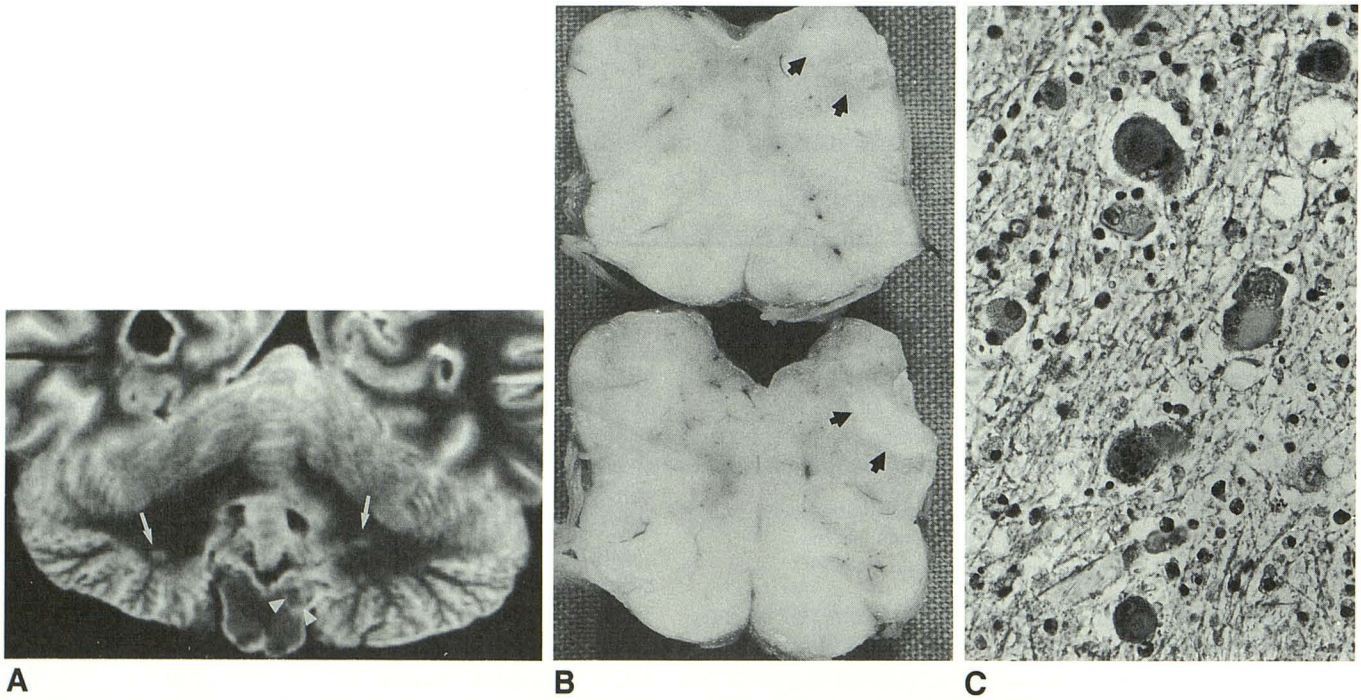

Fig. 1.—Case 6.
***A*, T2-weighted MR image (2800/70/2) shows foci of increased signal intensity bilaterally in cerebellar white matter (*arrows*), on ventricular surface of medulla, and surrounding a low-signal-intensity lesion (*arrowheads*) in left dorsolateral medulla.**
***B*, Gross photograph shows necrotic lesion in left dorsolateral medulla (*arrows*). Histology showed CMV and toxoplasmosis.**
***C*, Photomicrograph of a cerebellar white matter lesion shows cytomegalic cells and necrosis. (×500)**

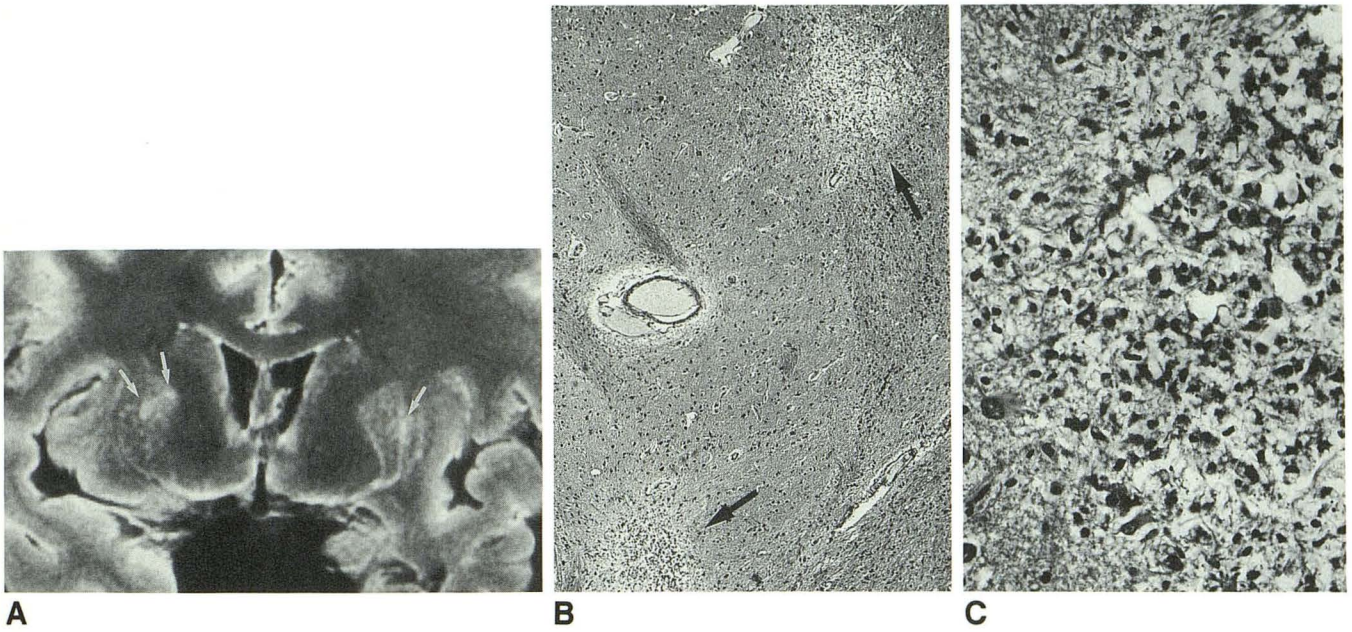

Fig. 2.—Case 8.
***A*, T2-weighted MR image (2800/70/2) shows multiple foci of increased signal intensity in putamen, bilaterally (*arrows*).**
***B* and *C*, Photomicrographs of these lesions show large microglial nodules with necrosis (*arrows*). On immunoperoxidase studies, many of the mononuclear cells contained HIV antigens. (*B* = ×50, *C* = ×500)**

but lesions were seen in all regions of the CNS, including cerebral and cerebellar cortex and brainstem.

MR false-positive rate for detection of foci of increased signal intensity. Several foci of increased signal intensity could be easily recognized as autopsy saw cuts, tips of the ventricular horns (especially occipital), and deep sulci. Lenticulostriate vessels were seen by MR in five cases as linear/punctate foci of increased signal intensity. MR identified 18 additional foci of increased signal intensity for which no abnormalities were histologically identified. Three were small and punctate and may represent vessels. All of these foci were less than 2 mm in size.

Correlation with clinical findings. Of the nine patients with mental status changes, MR showed atrophy in seven. The two patients without MR evidence of atrophy suffered from (1) decreased level of consciousness (case 2) and (2) anxiety and decreased ability to concentrate (case 8). In addition, one patient who had no mental status or neurologic changes (case 3) also had atrophy on MR. There were no specific MR findings that correlated with mental status changes. In contrast, mental status changes were present in all of the seven patients whose brains revealed diffuse MGNs at autopsy. This does not, however, reach statistical significance, since the overall proportion of cases with MGNs and the overall proportion of cases with mental status changes is so high. The presence of diffuse MGNs, associated with either CMV or HIV, was generally not detected by MR studies. There were four foci of MR abnormality where MGNs were the only pathologic findings (see above section "MR true-positive focal lesions"). In the same brains, however, there were widespread MGNs that were *not* detected by MR. No other pathologic features were consistently associated with specific clinical findings.

Discussion

We examined the capability of MR to detect the pathologic changes in the brains of AIDS patients. MR readily detected foci of infarction or necrosis, and identified some, but not all, areas of severe ventriculitis and focal gliosis. It has been previously hypothesized that infarcts are particularly prominent on T2-weighted MR scans because of the astrocytic reaction surrounding the central region of necrosis [7]. Reactive astrocytes produce abundant cytoplasmic filaments (which are visible with GFAP immunostaining) and may absorb proteinaceous edema fluid, both contributing to increased intracellular water content.

MR did not detect *diffuse* MGNs (seen histologically in seven of 10 cases) or diffuse gliosis (seen in six of 10 cases). This may be due to the small size of such lesions (usually 50–200 μm), which is much smaller than the in-plane-resolution (0.62 mm) of our MR scans. In the two cases of this series in which MGNs were detected by MR without associated infarction, necrosis, or edema, multiple MGNs formed *aggregate* lesions that were greater than 3 mm in size at histology. Volume averaging of the signal from adjacent normal brain parenchyma on 5-mm-thick MR sections likely further contributes to the inability to demonstrate MGNs by MR. Other small lesions, such as a cryptococcal cyst measuring 70 μm in

diameter, were also not detected by MR. The muted inflammatory response in the brains of immunosuppressed patients may also contribute to the decreased size of CNS lesions and their lack of identification by MR.

MR registered several false-positive findings. While some of these represented vessels, perivascular spaces, or artifacts related to the autopsy procedure, others remain unexplained. While it is possible that small areas of abnormality may have been missed on gross visual examination of the brain slices, an attempt was made to submit all areas of MR abnormality for histologic examination. Nevertheless, a few small lesions may have escaped histologic examination. Alternatively, these foci may represent normal vessels and/or Virchow-Robin spaces, or less likely, subtle lesions not detected by the histopathologic methods used in this study.

The use of postmortem MR to identify CNS lesions has been validated by a variety of studies in the literature [6, 8–10]. Studies using NMR spectroscopy of unfixed postmortem brain tissue have shown no change in T1 values and slight decreases in T2 values over time (up to 24 and 90 hr) [12, 13]. Fixation of CNS tissue, however, causes a decrease in T1 values, with cortex decreasing more than white matter [6, 13]. Thus, in postmortem scans of formalin-fixed brains, several investigators have shown that the T1 relaxation time of gray matter is less than that of white matter, while in the living brain the T1 of white matter is less than that of gray matter [6, 8, 10, 13]. Formalin fixation appears to have less of an effect on T2 relaxation. T2-dependent images were found to be the most useful images in our study. The premortem MR scans from one of our cases (1–4 months before death) did not reveal any additional lesions or other significant differences in appearance when compared with the postmortem scan of the brain.

The patients in our study generally had mild, nonfocal neurologic symptoms and did not have severe CNS disease. None had CNS tumors. Although toxoplasmosis is the most common focal lesion detected by CT in living AIDS patients [4, 14–17], it was seen only once in our postmortem series, appearing as a plaquelike parenchymal lesion in combination with CMV necrosis. Other opportunistic and viral infections are also common in AIDS patients and may cause focal lesions; in our series there was one case of cryptococcus and one case with acid-fast bacilli in the choroid plexus. While viral infections may cause focal CNS lesions, frequently they are associated with diffuse encephalopathy [5, 18, 19]. In our series, four cases had diffuse CMV encephalitis, two had diffuse HIV encephalitis, and two had diffuse MGNs that could not be ascribed to a specific disease. CMV may also cause diffuse ventriculitis [19, 20], and the central predominance of atrophy in some of our cases may be related to CMV involvement of the periventricular regions.

The majority of AIDS patients develop cognitive dysfunction during the course of their disease [18, 21]. In our series, nine of 10 patients demonstrated premortem mental status abnormalities. Although many of the brains revealed discrete foci of abnormal, increased signal intensity in the deep white matter on MR, the focal lesions detected in each case seemed too small and few in number to account for the clinical impression of encephalopathy. All seven of the patients with diffuse MGNs seen histologically, however, had mental status changes.

Cerebral atrophy is frequently seen by premortem and postmortem CT and MR scans in AIDS patients, particularly those with mental status changes [1–3, 5]. Atrophy was seen in 80% of our postmortem brains, and overall had a central predominance. The radiologic identification of atrophy correlated well with ventricular dilatation seen at autopsy, but neither of these features correlated with brain weight. All of the brain weights were within the normal range. The lack of correlation with brain weight may reflect the mild atrophy seen in many cases, but may also be expected in such a small group, as normal brain weights in the larger population show wide variability [22].

The focal lesions identified by MR in this study were predominantly located in the deep white and gray matter of the cerebrum and cerebellum. In addition to a true preponderance of deep lesions at pathologic examination, it is also likely that any lesion with prolonged T2 relaxation time will be more conspicuous against a background of white matter (having lower signal intensity) than gray matter (having higher signal intensity) on T2-weighted images. A similar distribution has been described in previous studies of CNS pathology in AIDS (see, for example [3, 23]). Focal lesions identified by MR in our series most often included regions of infarction or necrosis. In our series, infarctions occurred in clinically, radiologically, and pathologically advanced cases of CMV and/or HIV encephalitis. The high frequency of infarcts associated with CMV is consistent with the hypothesis that such infarcts result from CMV infection of endothelial cells followed by occlusion of the vascular lumen [24]. While most of the CMV-infected cells in these brains were glial or neuronal, occasional cytomegalic cells were morphologically and immunocytochemically identifiable as endothelial cells [19, 20].

While the detection and localization of focal lesions is important for diagnostic and prognostic purposes, particularly when surgical or radiation therapy is considered, the identification of diffuse lesions associated with encephalopathy is more problematic. Two of the diseases that frequently cause diffuse brain injury in AIDS patients are HIV and CMV infection. While each of these viral infections has some specific histologic features, both can result in diffuse changes without any associated necrosis or focal lesions [3, 19]. In our series MR did not detect *diffuse* MGNs, and thus did not detect diffuse viral encephalitis. A similar result was reported in a review of premortem CT ($n = 22$) and MR ($n = 7$) scans in 22 patients with HIV encephalitis [3]. In several cases, diffuse white matter hyperintensity was present on MR in encephalopathic AIDS patients [1, 5]. This pattern would be consistent with the pathologic findings of a viral encephalitis, but would not help to distinguish between CMV or HIV encephalitis. The coexistence of multiple pathologic processes in the CNS of AIDS patients further complicates the situation. In our series, three of eight patients had more than one infectious agent identified in the brain at autopsy.

In conclusion, our data indicate that focal hyperintense lesions without mass effect on T2-weighted images of postmortem AIDS brains most frequently represent necrosis or infarction, often associated with CMV infection. MR did not

detect the diffuse MGNs accompanying diffuse viral encephalopathy. Since focal lesions associated with tumor or opportunistic infection are readily detected by MR, the presence of a negative MR scan or diffuse white matter hyperintensity in an encephalopathic AIDS patient should suggest the possibility of a viral encephalitis or metabolic abnormality. Our study clearly demonstrates the usefulness of correlating postmortem MR imaging with neuropathology and its relevance to the interpretation of MR in living patients.

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The reader's attention is directed to the commentary on this article, which appears on the following pages.