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CT and MR Evaluation of Intracranial Involvement in Pediatric HIV Infection: A Clinical-Imaging Correlation

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Purpose: To review the cranial CT and MR examinations of 29 children with perinatally transmitted HIV infection and correlate the imaging findings with clinical and pathologic data. **Methods:** 28 children were examined with CT, four with MR. **Results:** CT abnormalities were seen in 25 children studied (89%), including cerebral atrophy (25 children), basal ganglia calcification (10 children), periventricular frontal white matter calcification (four children), cerebellar calcification (one child), white matter low attenuation areas (two children), intracranial hemorrhage (three children) and cerebral infarction (one child). Intracranial calcifications were only seen in association with cerebral atrophy and were never seen prior to 1 year of age. Calcifications in the periventricular white matter or cerebellum were always associated with basal ganglia calcifications. MR abnormalities were seen in all four children studied; cerebral atrophy (four children), areas of high signal intensity in white matter (four children), loss of normal posterior pituitary high signal intensity (one child). Cerebral atrophy appeared to be a nonspecific finding that was seen in some children in the absence of neurologic signs and symptoms. All children with intracranial calcifications had developmental delay. Intracranial hemorrhage was seen in children with severe thrombocytopenia. Focal intracranial infections were unusual and neoplastic lesions were not found. **Conclusions:** Cerebral atrophy, basal ganglia calcifications, and focal white matter lesions were the most common abnormalities seen neuroradiologically in our series of HIV-infected children; cerebral atrophy was a nonspecific finding.

Index terms: Acquired immunodeficiency syndrome (AIDS); Brain, computed tomography; Pediatric neuroradiology

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Neurologic abnormalities are common in children with human immunodeficiency virus (HIV) infection (1-4). Intracranial involvement may be due to primary HIV infection or secondary to complications of immunodeficiency. Estimates are that 50% of infected children may have a subcortical dementia or focal neurologic signs or symptoms (4, 5). Nevertheless, the attention given to the neuroimaging findings in children with AIDS has lagged far behind that focused on adults. In this report we describe the cranial

computed tomography (CT) and magnetic resonance (MR) appearance of 29 children with perinatally transmitted HIV infection and correlate imaging findings with clinical and pathologic data.

Patients and Methods

Eighty-three children who fulfilled the criteria for perinatally transmitted HIV infection (6) were evaluated at our institution between January 1987 and June 1991. Cranial imaging studies were performed on 29 of these children during the study period. Forty-four cranial CT scans were performed in 28 children. Eighteen children had a single examination and 10 had two or more examinations: two examinations, six children; three examinations, two children; and four examinations, two children. Each examination included a noncontrast CT. Additionally, 12 exams also included contrast-enhanced CT. Four cranial MR studies were performed in four children. Twenty-five children were imaged with CT only, three with CT and MR, and one with MR only.

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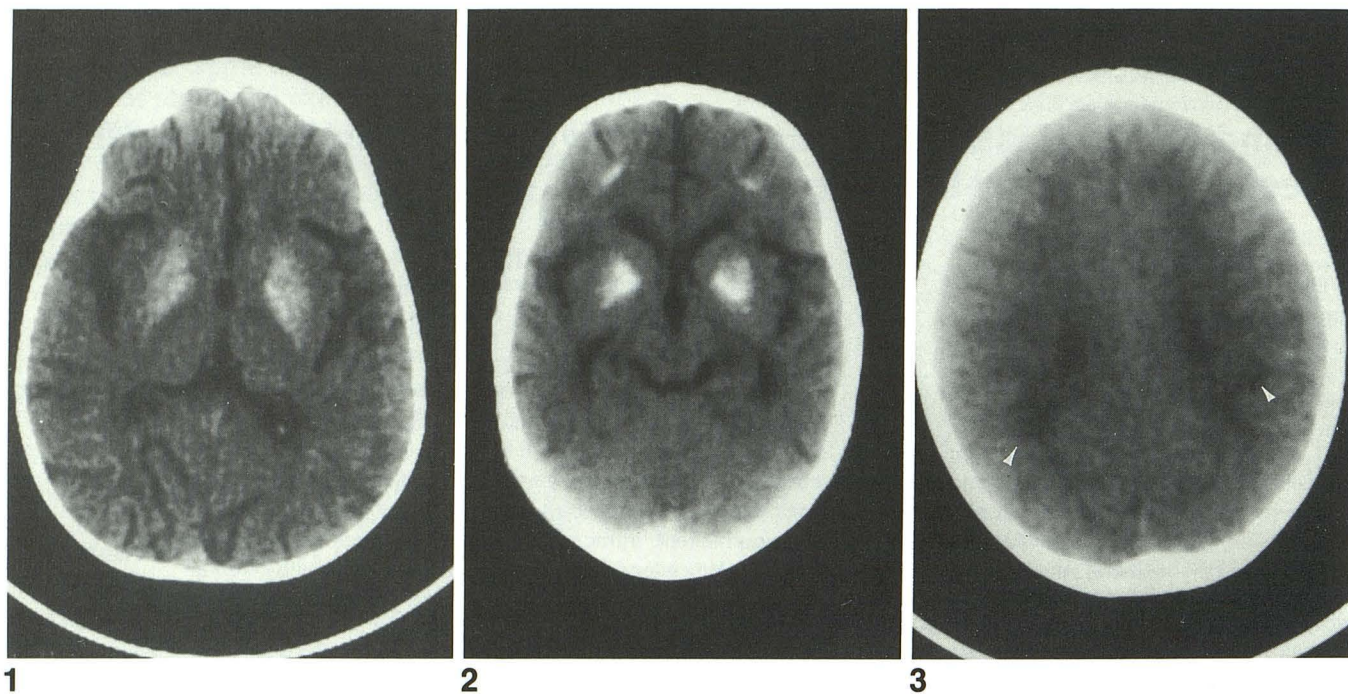


Fig. 1. Basal ganglia calcification. Two-year-old with encephalopathy and weakness. Noncontrast CT shows symmetrical calcification in the basal ganglia.

Fig. 2. Frontal white matter calcification. Three-year-old with encephalopathy and hypereflexia. Noncontrast CT demonstrates symmetrical calcification in the basal ganglia and frontal white matter.

Fig. 3. Focal white matter lesions. Four-year-old with encephalopathy, weakness, and hypereflexia. Noncontrast CT shows focal areas of low attenuation in the parietal white matter (arrowheads).

Seventeen patients were female and 12 were male. The age range was 1 month to 8 years (median age at time of first CT or MR examination = 11 months). All 29 children were classified as having symptomatic HIV infection (6); of these, 26 met diagnostic criteria for AIDS and three did not. Two of these children also had associated perinatally transmitted cytomegalovirus infection.

Indications for CT or MR included the following: change in mental status (11 children), sepsis (nine children), focal neurologic deficit (eight children), fever of unknown origin (six children), failure to thrive (six children), seizure (six children), diabetes insipidus (one child) and head banging (one child). Clinical records, including classification of HIV infection (6) and neurologic examination findings were retrospectively reviewed. The following information was retrieved from neurologic and developmental examinations: presence or absence of weakness, hypertonia, hypotonia, ataxia, and encephalopathy, defined as no further acquisition or progressive loss of developmental milestones, language or socially adaptive skills. The neurologic examination was considered abnormal if one or more of the above findings were present. In all cases, the neurologic evaluation was performed within 1 week of the CT. These children all lacked evidence for perinatal asphyxia.

CT was performed with contiguous 1-cm thick sections obtained from foramen magnum to vertex in children over 3 months of age (40 examinations), while 5-mm thick sections were obtained at 5-mm intervals in children 3 months of age or less (four examinations). Three milliliters

per kilogram (maximum dose 120 mL) of 60% iodinated contrast material (lothalamate meglumine) was administered by means of intravenous bolus injection on the contrast-enhanced exams.

MR was performed on a 1.5-T superconducting magnet. Proton-density and T2-weighted images were acquired in the axial plane and T1-weighted images were acquired in the sagittal plane with the following parameters: 2800/30, 2800/80, 600/20 (repetition time (TR)/echo time (TE) msec). Contiguous 6- to 7-mm thick sections were obtained. No contrast media was administered.

The CT and MR scans were retrospectively reviewed by one of the authors who was blinded to the clinical neurologic status. Abnormal findings were divided into seven categories: 1) cerebral atrophy, defined as dilatation of the ventricular system and prominence of the sulci; 2) intracranial calcification; 3) focal areas of white matter abnormality; 4) cerebral infarction; 5) intracranial hemorrhage; 6) abnormal contrast enhancement on CT; and 7) abnormal signal intensity on MR.

Results

CT Findings

Six of 28 children (21%) had a normal initial CT scan, two of these had abnormalities on subsequent scans (atrophy in two, basal ganglia calcifications in one). Abnormalities noted on the

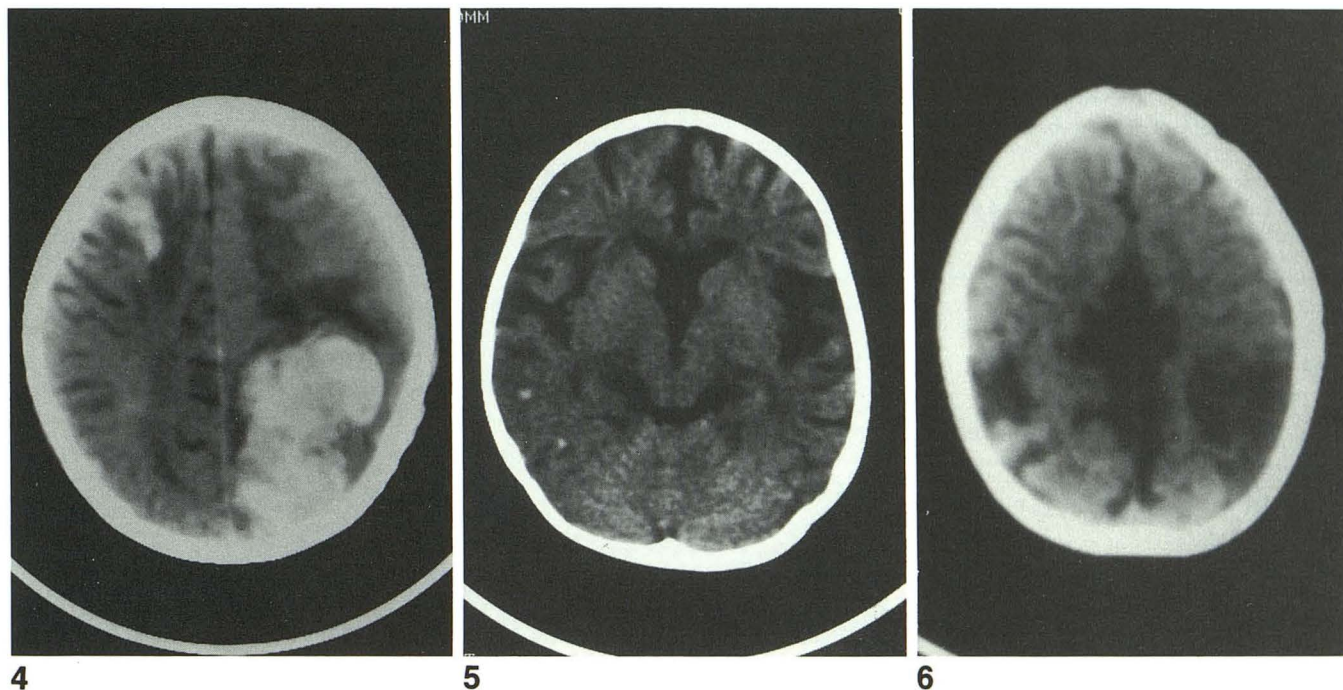


Fig. 4. Intracranial hemorrhage. Four-year-old with disseminated intravascular coagulopathy, focal neurologic deficits, and seizures. Noncontrast CT demonstrates a large parenchymal hemorrhage in the left parietal-occipital region and a smaller hemorrhage in the right frontal lobe.

Fig. 5. Intracranial hemorrhage. Sixteen-month-old with autoimmune thrombocytopenia and seizures. Noncontrast CT shows multiple punctate parenchymal hemorrhages.

Fig. 6. Cerebral infarction. Six-month-old with encephalopathy and hypereflexia (Table 3, case 5). Noncontrast CT demonstrates bilateral parietal-occipital infarcts.

noncontrast CT scans included the following: cerebral atrophy (25 children), basal ganglia calcifications (10 children, Fig. 1), periventricular white matter calcification (four children, Fig. 2), cerebellar calcification (one child), peripheral white matter low attenuation areas (three children, Fig. 3), intracranial hemorrhage (three children, Figs. 4 and 5), cerebral infarction (one child, Fig. 6).

Ten children with two or more CT exams had cerebral atrophy on the initial study. In seven of these, the severity of atrophy increased on follow-up exams, over a 4- to 48-month interval (Fig. 7).

Intracranial calcifications were always seen in association with cerebral atrophy. The calcifications were bilateral and symmetrical in all cases. Calcifications in the periventricular frontal white matter were always associated with basal ganglia calcifications. Cerebellar calcifications were also seen in association with calcifications in the basal ganglia. Intracranial calcifications were not observed in the two children with perinatally transmitted cytomegalovirus infection.

White matter low attenuation areas were also always seen in association with cerebral atrophy.

The distribution was patchy and bilateral. There was no associated edema or mass effect. The lesions were primarily observed in the parietal-occipital white matter. Two of the three children had contrast-enhanced CT scans. The lesions did not enhance.

Review of the 11 contrast-enhanced CT scans revealed a small, focal area of contrast ring enhancement in the right frontal white matter in one child (Fig. 8). There was no associated edema or mass effect. Abnormal contrast enhancement was not observed in any other case.

MR Findings

MR scans were obtained on four children. Abnormalities noted on MR included cerebral atrophy (four children), areas of peripheral white matter high signal intensity on proton-density and T2-weighted images (four children, Fig. 9), and absence of the normal high signal intensity in the posterior pituitary on T1-weighted images (one child, Fig. 10). The areas of high signal intensity in the peripheral white matter were bilateral but not always symmetric. There was no associated

Fig. 7. Increasing cerebral atrophy. Three-year-old with encephalopathy and hyperreflexia.

A, Noncontrast CT shows mild symmetrical enlargement of the cerebrospinal fluid spaces.

B, Noncontrast CT in same child 6 months later demonstrates increased size of the cerebrospinal fluid spaces.

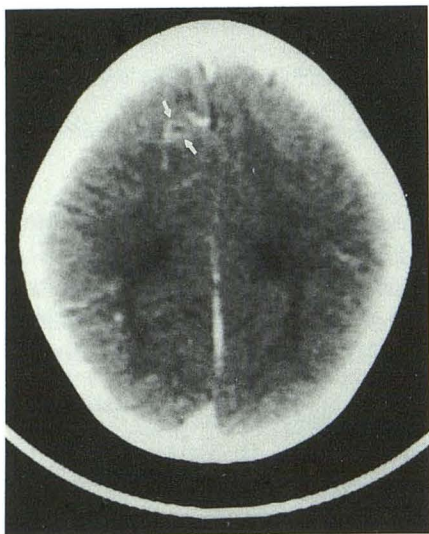
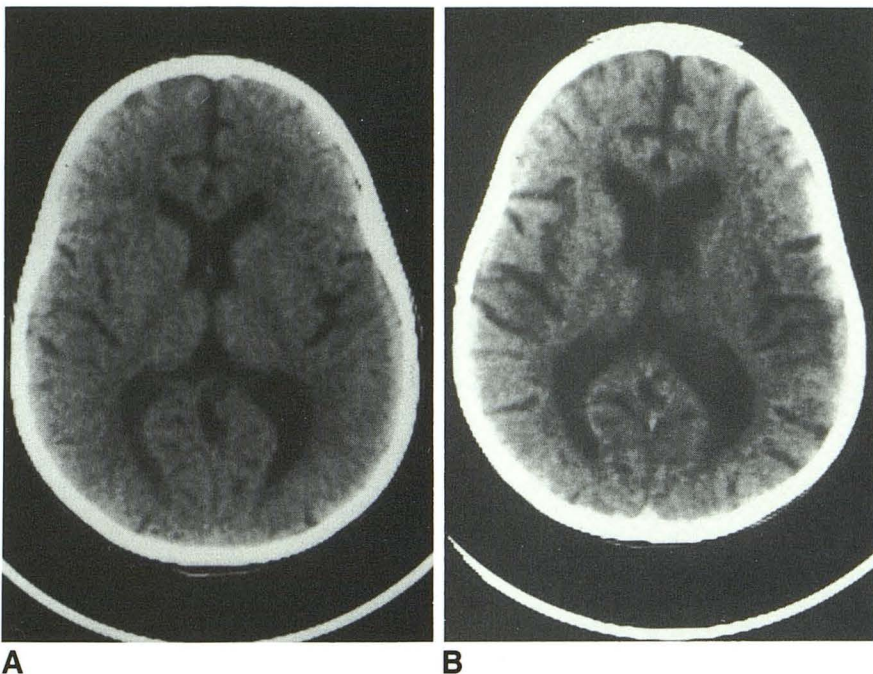


Fig. 8. Abnormal contrast enhancement. Eighteen-month-old with encephalopathy, weakness, and hyperreflexia (Table 3, case 2). Contrast-enhanced CT shows a ring-enhancing lesion in the right frontal lobe (arrows). There was no associated edema or mass effect. The child died 1 month following CT evaluation and post-mortem examination revealed only scattered foci of inflammatory cell infiltrates.

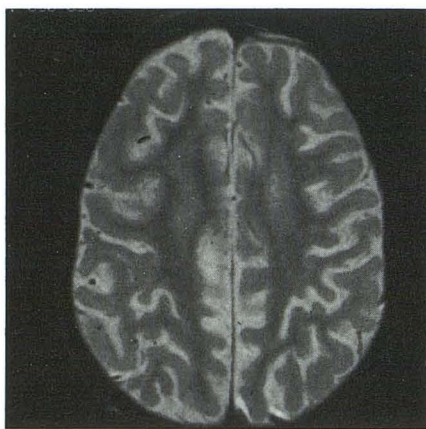


Fig. 9. Focal white matter lesions. Four-year-old with encephalopathy and hyperreflexia. Proton-density MR demonstrates patchy areas of white matter high signal intensity.

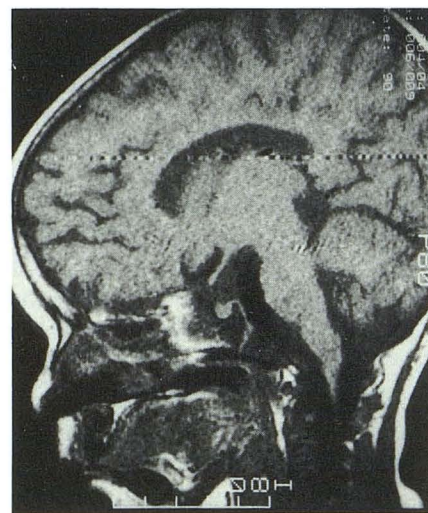


Fig. 10. Three-year-old with severe encephalopathy and diabetes insipidus (Table 3, case 1). T1-weighted midline sagittal image shows absence of hyperintense posterior pituitary signal. The child died 1 month following MR evaluation. The pituitary gland appeared normal on gross pathologic inspection.

edema or mass effect. Three children with focal white matter abnormalities on MR had been examined with CT; in one of these, the studies were performed less than 1 week apart. The CT scans in all three had demonstrated only atrophy.

Clinical Findings

The CT findings were compared with patient age (Table 1). Intracranial calcifications were never seen prior to 1 year of age, even in children with clinical encephalopathy. The age of children

TABLE 1: CT abnormalities in 28 children with HIV infection compared with patient age (%)

Patient Age	No Abnormalities	Cerebral Atrophy	Low Attenuation Areas in WM ^a	Calcifications		Hemorrhage	Infarction	Enhancing Lesion
				Basal Ganglia	Frontal WM ^b			
<1 year n = 16	5 (31)	11 (69)	1 (6)				1 (6)	
≥1 year n = 19 ^c		18 (95)	2 (11)	10 (53)	4 (21)	3 (16)		1 (5)

^a Denotes peripheral white matter.^b Denotes periventricular frontal white matter.^c Seven of these children were initially studied prior to 1 year of age and are included in both groups.

TABLE 2: CT abnormalities in 28 children with HIV infection compared to neurologic examination abnormalities (%)

Neurologic Examination	No Abnormalities	Cerebral Atrophy	Low Attenuation Areas in WM ^a	Calcifications		Hemorrhage	Infarction	Enhancing Lesion
				Basal Ganglia	Frontal WM ^b			
Normal n = 8	4 (50)	4 (50)	1 (12)					
Abnormal n = 24 ^c	2 (8)	22 (92)	2 (8)	10 (42)	4 (17)	3 (25)	1 (4)	1 (4)

^a Denotes peripheral white matter.^b Denotes periventricular frontal white matter.^c Four of these children had a normal neurologic examination at the time of the initial CT and are included in both groups.

at the time intracranial calcifications were first noted on CT ranged from 1 to 6 years; median age, 1 year and 8 months. Three of these children had CT scans prior to 1 year of age (range, 3 to 11 months) that did not show calcifications.

The CT findings were also compared with the neurologic examination findings (Table 2). The neurologic abnormalities noted in these children included: encephalopathy (24 children), hypereflexia (17 children), weakness (16 children), ataxia (2 children). Four of six children (67%) with a normal initial CT scan had a normal neurologic examination, while two had clinical encephalopathy. Eight of 12 children (67%) with cerebral atrophy as an isolated CT abnormality had an abnormal neurologic examination at the time of CT; encephalopathy in eight, hypereflexia in six, weakness in five, ataxia in one. The other four children had prior or concurrent systemic infections; *Pneumocystis carinii* pneumonia in three, cytomegalovirus enteritis in one. All seven children with increased severity of atrophy on follow-up examinations had delayed motor and language development. All 10 children with intracranial calcifications also had developmental delay. Four of 10 had loss of previous developmental milestones at the time of CT scan. Children with focal white matter lesions or infarctions on CT or MR all had clinical encephalopathy. They did not,

however, present with sudden focal neurological deficits.

Pathology Findings

Seventeen of these children have subsequently died. Histopathologic examination of tissue obtained by autopsy was performed in six children. The time between the premortem CT or MR examination and death in the autopsied children ranged from 1 to 13 months. Neuroimaging findings were compared with results of histopathologic evaluation in these six children (Table 3).

The brain was small in five of six children, with weights ranging between 50%–85% of values expected for age. Microglial nodules, multinucleated giant cells, and perivascular infiltration by mononuclear inflammatory cells were seen in the white matter in three children (Fig. 11). In an additional child, multinucleated giant cells were present in the absence of inflammatory cell infiltrates. Focal white matter gliosis was present in five children. Of these, two also showed microscopic foci of calcification in the basal ganglia (Fig. 11). One child additionally demonstrated calcification in the media of medium-sized arteries of the brain (Fig. 11). Multiple abscesses in the cerebral cortex, basal ganglia, thalamus, and mid-brain were noted in the child. Silver methenamine

TABLE 3: Lesions identified by CT, MR, and pathology in autopsied patients

Case No.	Interval between CT or MRI and Death	CT or MR Findings	Pathologic Findings
1	1 month	Atrophy white matter lesions abnormal posterior pituitary NS	Small brain Gliosis NS Vascular and parenchymal calcification
2	1 month	Atrophy Focal area of abnormal contrast enhancement	Small brain microglial nodules multinucleate giant cells perivascular inflammation NS
3	2 months	Atrophy	Small brain multinucleate giant cells gliosis
4	8 months	Atrophy NS	Small brain microglial nodules multinucleate giant cells perivascular inflammation gliosis Parenchymal calcification
5	11 months	Atrophy parietal-occipital infarction white matter lesions NS	Small brain Focal parietal-occipital atrophy (healed infarction) Gliosis Multiple abscesses <i>Candida albicans</i>
6	13 months	NS	Small brain multinucleate giant cells microglial nodules perivascular inflammation gliosis

Note.—NS = not seen.

stain demonstrated pseudohyphae of *Candida albicans* in the abscesses.

Discussion

The nervous system is a primary site of HIV infection in the pediatric age group (3, 7). As a result, neurologic dysfunction is common in infected infants and children. The presence of encephalopathy, defined as a subcortical dementia, has been reported in 30%–50% of affected children (3, 5). The majority of children (90%) in our study had encephalopathy. Clinical manifestations of this encephalopathy include developmental delay, cognitive impairment, and pyramidal tract signs (4, 8). Symptoms may be static,

characterized by failure to develop beyond a given point, or progressive, distinguished by loss of previously acquired milestones (3, 4). Motor signs, including hypereflexia, spastic-ataxic gait, or frank paraparesis may accompany the encephalopathy (2, 3).

Previous reports of cranial imaging findings in children with HIV infection have described cerebral atrophy as the most common finding (3, 8–10). This was true in our population as well. Atrophy was observed in 90% of children evaluated with CT or MR (Fig. 7). These findings correlate well with histopathologic reports of HIV-infected brains that demonstrate diminished weight for age and infiltration of microglial nodules and multinucleated giant cells that harbor

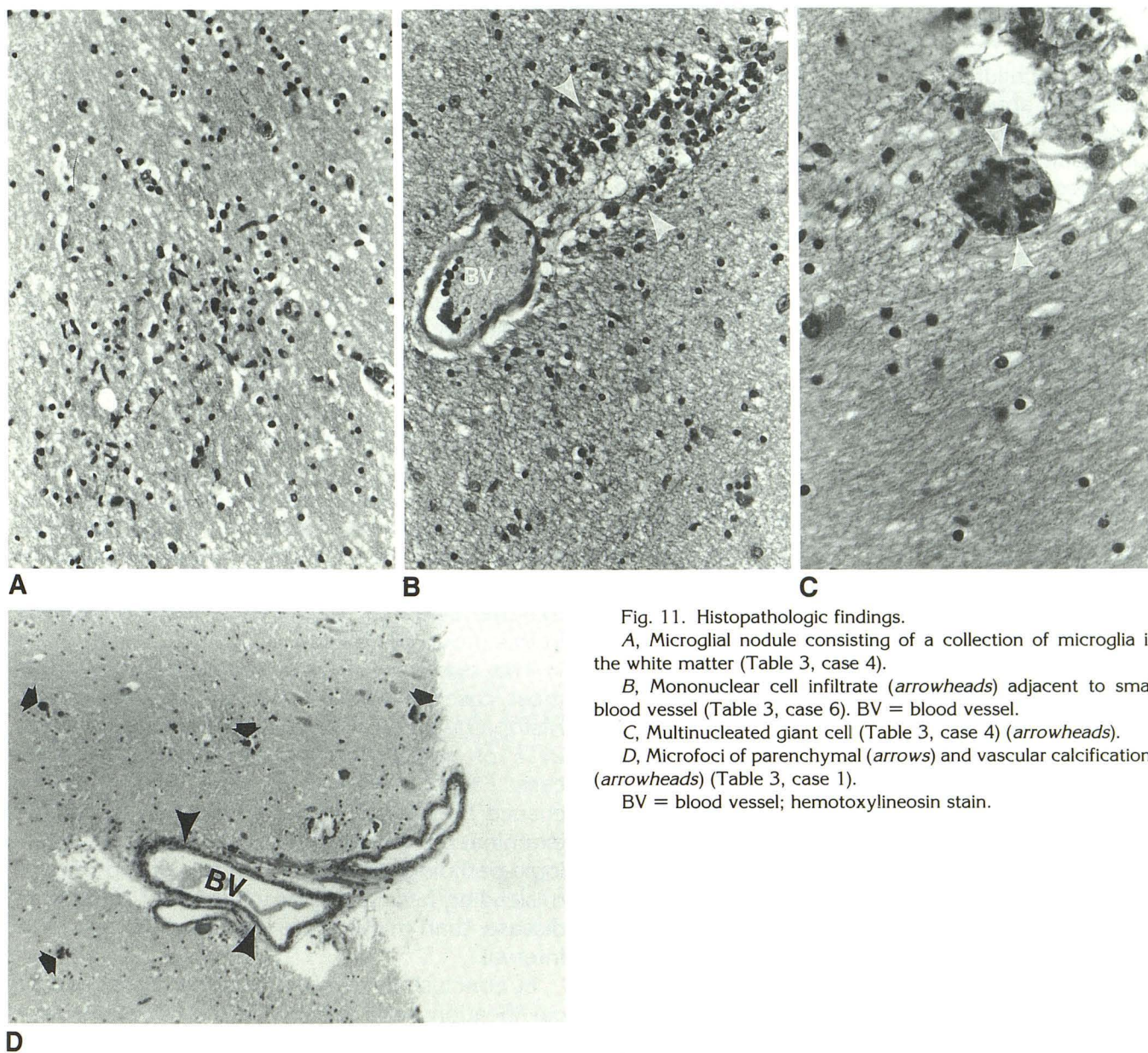


Fig. 11. Histopathologic findings.
 A, Microglial nodule consisting of a collection of microglia in the white matter (Table 3, case 4).
 B, Mononuclear cell infiltrate (arrowheads) adjacent to small blood vessel (Table 3, case 6). BV = blood vessel.
 C, Multinucleated giant cell (Table 3, case 4) (arrowheads).
 D, Microfoci of parenchymal (arrows) and vascular calcifications (arrowheads) (Table 3, case 1).
 BV = blood vessel; hematoxylineosin stain.

viral particles predominantly in the deep white matter (Fig. 11) (3, 5, 11, 12). Nutritional and metabolic factors may also contribute to the development of cerebral atrophy. We noted atrophy on CT in the absence of neurologic signs and symptoms in four children. All four had prior systemic illnesses requiring prolonged hospitalization, and poor weight gain.

Focal areas of peripheral and deep white matter disease have been reported in both children and adults with AIDS (Figs. 3 and 4) (4, 9, 13). The changes are secondary to direct involvement of the brain by HIV resulting in areas of demyelination and gliosis (11, 14). Scattered areas of gliosis were noted on histopathologic evaluation in both

children who had focal areas of white matter abnormality on CT or MR and were autopsied (Fig. 11).

Intracranial calcifications have also been previously noted in HIV-infected children (3, 8, 15, 16). The calcifications have been observed primarily in the basal ganglia and to a lesser extent in the frontal white matter. We noted similar findings, as basal ganglia calcifications were present twice as frequently as frontal white matter calcifications (Figs. 1 and 2). Additionally, frontal white matter calcifications were always associated with basal ganglia calcifications. These calcifications were never seen prior to 1 year of age or when the neurologic examination was normal.

Conversely, they were observed on CT in slightly over one half of children over 1 year of age and in 42% of children with encephalopathy or other neurologic abnormality. Pathologically, the calcifications have been noted in the parenchyma and surrounding small and medium-sized blood vessels accompanied by inflammation (Fig. 11) (3, 5). The changes appear to be related to HIV infection of the central nervous system (CNS). Calcification of the arterial wall, termed calcific arteriopathy, has also been described (Fig. 11) (17). The presence of intracranial calcifications represents an important difference between children and adults with HIV infection, inasmuch as calcifications have not been a prominent feature in adults (5, 9).

Another important difference between infected children and adults has been the rarity of focal intracranial neoplastic or infectious lesions reported in children (8, 18, 19). Our findings support these previous observations. A focal enhancing lesion was only observed in one child in our study group (Fig. 8). This area may have represented an intracranial focus of infection, however no organisms were seen and special immunofluorescent stains were not performed on postmortem examination 1 month following CT. Intracranial abscesses were noted on postmortem examination in one child who died of systemic candidiasis; however, no imaging examination had been performed immediately prior to death; CT was performed 11 months prior to death. Intracranial neoplasms were not identified in any child.

Intracranial hemorrhage and infarction are infrequent complications of pediatric AIDS (20, 21). Intracranial hemorrhage has been reported in children with AIDS in the setting of a hemorrhagic diathesis from severe thrombocytopenia as in our three cases (Figs. 4 and 5). Hemorrhage has also been noted in adult AIDS patients associated with CNS neoplasia (21). Two types of nonhemorrhagic infarction have been reported. These include multifocal microinfarctions or large areas of encephalomalacia as seen in our case (Fig. 6). Proposed etiologies for nonhemorrhagic infarction include arteriopathy and necrotizing encephalopathy (22).

Central diabetes insipidus has been observed with a variety of lesions that damage the hypothalamohypophyseal tract, including encephalitis. Prior reports have indicated that the high signal intensity in the posterior pituitary on T1-weighted images is absent in the presence of central dia-

betes insipidus as was observed in our case (Fig. 10) (23, 24). This has led to speculation that the absence of high signal intensity reflects altered functional integrity of the posterior pituitary (23). This MR finding has not been previously reported in children with AIDS encephalopathy.

This study does not propose to compare the relative efficacy of CT and MR in identifying intracranial lesions in children with AIDS. Combined imaging studies were only available in three children. Additionally, progression of neurologic disease may occur between the time of imaging evaluation and the time of death and pathologic examination. Each imaging examination has been shown to have an advantage over the other in the identification of different intracranial lesions commonly seen in these children. MR is more sensitive in detecting white matter abnormalities, while CT is better at demonstrating intracranial calcifications (11, 13, 25). Further prospective analysis in a large pediatric population is required to better define the role of each imaging modality in this group of children.

The absence of pathologic confirmation in most cases is another limitation of the study. Histopathologic correlation was available in only 21% of cases. Additionally, in three of these cases, a long interval of time (8–13 months) ensued between the final premortem CT or MR examination and death. Any attempt at radiologic-pathologic correlation in this group may be misleading because of progression of neurologic disease that might have occurred in that time interval.

In conclusion, cerebral atrophy, basal ganglia calcifications, and focal white matter lesions were the most common abnormalities seen on cranial imaging evaluation in our population of HIV-infected children. The calcifications were not seen in the first year of life nor in neurologically normal children. Cerebral atrophy was a nonspecific finding.

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