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Deep Gray Matter Involvement in Children with Acute Disseminated Encephalomyelitis

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PURPOSE: To review the frequency, distribution, and extent of deep gray matter disease in children with acute disseminated encephalomyelitis. **METHODS:** The MR examinations of 10 patients, who were discharged with the clinical diagnosis of acute disseminated encephalomyelitis between 1986 and 1992, were retrospectively reviewed. Locations of abnormal signal in the cerebral and cerebellar cortices, white matter, and deep gray matter nuclei were recorded. Pre-contrast and postcontrast images were compared, when available, to assess degree of enhancement (if any). **RESULTS:** Six patients had foci of prolonged T2 relaxation in the deep gray matter, ranging in size from less than 1 cm to 4 cm. The caudate heads were involved in 4 patients, caudate body in 3, globus pallidus in 3, putamina in 3, and thalami in 4. In 1 patient, the thalami were involved nearly symmetrically, with mild mass effect. Asymmetric subcortical white matter involvement was present as well. Prolonged T2 relaxation was present within the cerebral cortex in 4 patients and was associated with subcortical white matter abnormality in 3 and more central white matter disease in 1. Nine of 10 patients demonstrated foci of T2 prolongation in white matter, most commonly involving the subcortical region, corona radiata, and centrum semiovale. Three patients also had periventricular foci. Of the 3 patients receiving gadolinium, one showed no enhancement. Two of the patients showed enhancement of some but not all lesions. One patient, who had normal brain MR findings and symptoms of myelopathy, underwent spine MR which demonstrated focal linear areas of T2 prolongation in the spinal cord at levels C-1 to C-2 and T-6. **CONCLUSION:** Involvement of deep gray matter was common in our small series. The finding of T2 prolongation in these structures does not preclude the diagnosis of acute disseminated encephalomyelitis in the proper clinical setting. Because thalamic involvement is reported to be rare in multiple sclerosis, it may prove useful in distinguishing between acute disseminated encephalomyelitis and the initial presentation of multiple sclerosis.

Index terms: Demyelinating disease; Gray matter; Brain, magnetic resonance; Myelitis; Pediatric neuroradiology

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Acute disseminated encephalomyelitis, also known as postinfectious or postvaccinal encephalomyelitis, is an uncommon inflammatory demyelinating disease of the central nervous system. Most cases are related to recent vaccination or infection, although the process may

occur without an apparent precipitating event (1, 2). Although classically considered a white matter disease, gray matter involvement is not uncommon and has been noted in both the pathologic and radiologic literature (1-4). However, deep gray matter involvement is thought rare (5, 6). Recent case reports have reported thalamic and basal ganglia involvement with acute disseminated encephalomyelitis (5, 6). In our practice, we also have noted deep cerebral gray matter involvement on magnetic resonance (MR) studies of a number of patients who ultimately have been discharged with diagnoses of acute disseminated encephalomyelitis. We have therefore reviewed our experience to investigate the frequency, distribution, and extent

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of deep gray matter disease in acute disseminated encephalomyelitis.

Materials and Methods

The MR examinations of 10 patients who were ultimately discharged with the clinical diagnosis of acute disseminated encephalomyelitis between 1986 and 1992 were retrospectively reviewed. Ages of the patients (4 girls and 6 boys) ranged from 2 months to 13 years. All patients presented with acute neurologic deficits about 1 to 3 weeks after a viral illness. Symptoms included cranial nerve palsies, hemiparesis, seizures, and spastic diplegia. No signs or symptoms of disease outside the central nervous system were elicited. Cerebrospinal fluid revealed mild pleocytosis in 8 of 10 patients. Cultures were negative for bacteria, fungi, and virus. Brain MR was performed in all patients during peak symptoms, before the initiation of steroid therapy. Eight patients had MR on a 1.5-T system, two patients on 1.0-T systems. Spin-echo T1-weighted images (400–617/16–25/2 [repetition time/echo time/excitations]) were obtained in sagittal and axial planes in all patients and in the coronal plane in three patients. Spin-echo T2-weighted images (2500–3000/30–60/70–120) were obtained in axial planes. Three patients had T1-weighted axial and coronal images performed after intravenous administration of gadopentetate dimeglumine. Axial computed tomography (CT) (10-mm contiguous images) before and after administration of intravenous iodinated contrast was performed 1 week before and 1 day before MR in two patients. Noncontrast CT 2 days before MR was performed in a third patient. One patient with normal brain MR findings underwent total spine MR consisting of gradient-echo 3-mm sagittal images (600/15/35, $\theta = 20^\circ$) and cardiac-gated T2-weighted 5-mm axial images (1800/30/80). Two patients had follow-up MR, one patient 8 months and the other 4 weeks after acute presentation.

A clinical diagnosis of acute disseminated encephalomyelitis was made in each, and steroid therapy was initiated. All patients showed dramatic improvement within 48 to 72 hours, with recovery of full neurologic functions within 2 months. All 10 patients are now completely healthy and have had no subsequent episodes 7 months to 7 years after the acute events.

We retrospectively reviewed the MR studies. Locations of abnormal signal in the cerebral and cerebellar cortices, white matter, and deep gray matter nuclei were recorded. Precontrast and postcontrast images were compared, when available, to assess degree of enhancement (if any).

Results

Nine of 10 patients demonstrated foci of T2 prolongation in white matter, most commonly involving the subcortical region, corona radiata, and centrum semiovale. Three patients had periventricular foci. Six patients had foci of pro-

longed T2 relaxation in the deep gray matter, ranging in size from less than 1 cm to 4 cm. The caudate heads were involved in 4 patients, caudate body in 3, globus pallidus in 3, putamina in 3, and thalami in 4. In 1 patient, the thalami were involved nearly symmetrically, with mild mass effect; asymmetric subcortical white matter involvement was present as well. Prolonged T2 relaxation was present within the cerebral cortex in 4 patients, associated with subcortical white matter abnormality in three, and with more central white matter disease in one.

Of the three patients receiving gadolinium, one showed no enhancement. A second patient showed enhancement of some, but not all, foci of prolonged T2 within the subcortical white matter, thalamus, and basal ganglia (Fig 1). The third patient who received gadolinium demonstrated patchy enhancement of some but not all supratentorial lesions (Fig 2).

Two patients had follow-up MR. In one patient, follow-up 8 months after acute presentation showed resolution of all abnormal T2 signal. Gadolinium was not given. In the second patient, 4-week follow-up showed improvement of some areas of T2 prolongation, yet there was a development of new lesions. In addition, there was near total resolution of supratentorial enhancement but development of patchy asymmetric enhancement in the cerebellar white matter (Fig 2).

Of the three patients who had both CT and MR, CT reflected the MR abnormality in one. In the second patient, the cortical disease was underestimated. The CT of the third patient was normal. Contrast enhancement was not apparent in either of the two patients receiving iodine.

One patient had normal brain MR findings. Because the patient had signs and symptoms of myelopathy, a spine MR scan was obtained, demonstrating small (less than 1 cm) focal linear areas of T2 prolongation in the spinal cord at levels C-1 to C-2 and T-6 (Fig 3). See Table for compilation of findings.

Discussion

Acute disseminated encephalomyelitis is an inflammatory demyelinating disorder often following infections with measles, chickenpox, rubella, smallpox, infectious mononucleosis, herpes zoster, mumps, and influenza. Rabies and vaccine immunizations also have been associated with acute disseminated encephalomyeli-

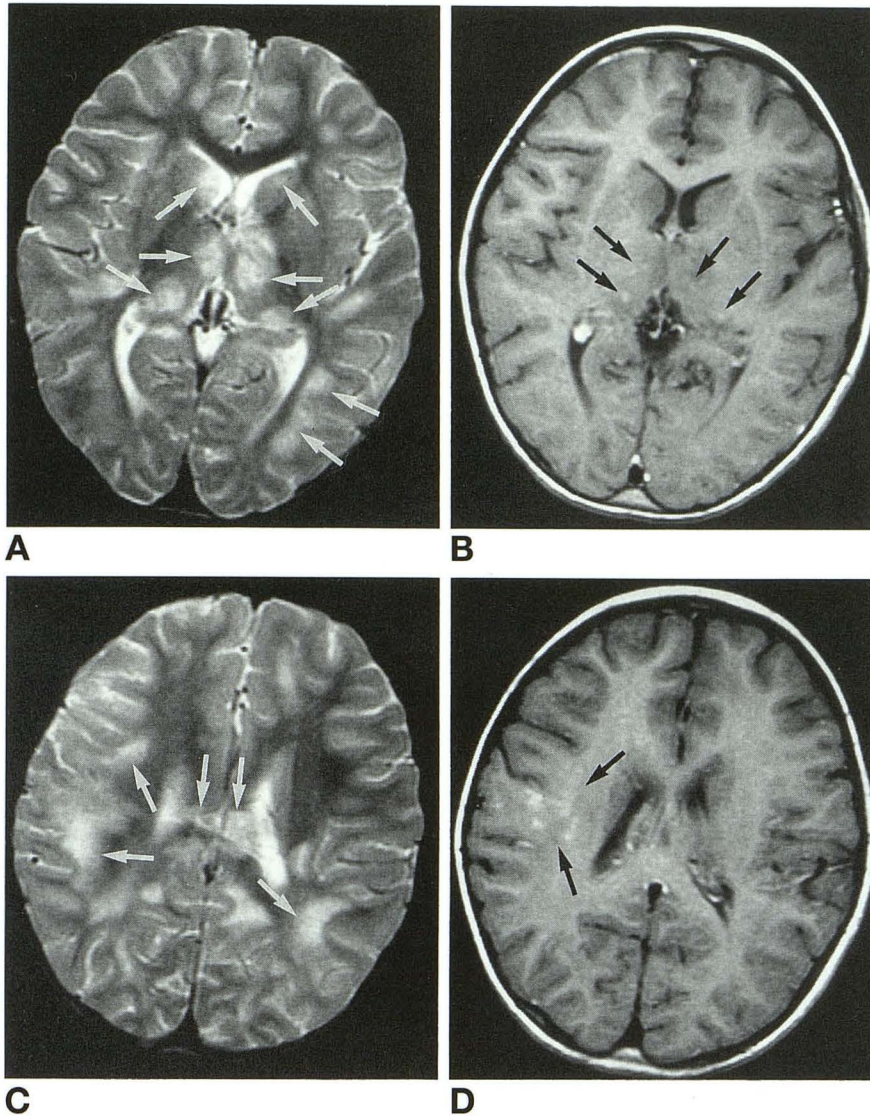


Fig 1. Case 1.

A, T2-weighted axial (2500/80) image shows multiple areas of T2 prolongation involving the thalami, caudate heads, and subcortical white matter (arrows).

B, T1-weighted axial (600/20) image postcontrast at same level as A shows patchy enhancement of some of the thalamic lesions (arrows).

C, T2-weighted axial (2500/80) images show T2 prolongation involving the body and splenium of the corpus callosum and subcortical white matter (arrows).

D, T1-weighted axial (600/20) image postcontrast at same level as C shows patchy enhancement of subcortical white matter in the posterior right frontal lobe (arrows).

tis. Symptoms usually develop within 3 weeks after the onset of a viral infection or immunization. An identical syndrome may occur in the absence of clinically apparent antecedent infection or immunization, possibly because of a preceding subclinical viral infection (7).

Children 6 to 10 years of age are most commonly affected, presenting with an acute onset of motor signs and symptoms, seizure activity, and, sometimes, altered consciousness. Other signs may include neck stiffness, strabismus, and symptoms of transverse myelitis (flaccid paraplegia, incontinence, and absent plantar and deep reflexes). In most patients the disease is monophasic, lasting from 2 to 4 weeks, with no relapse or recurrence of neurologic signs or symptoms. Recurrent attacks have been reported (7). Permanent neurologic deficit re-

mains in 10% to 20% of patients, and 15% to 20% die (8). Cerebrospinal fluid analysis can be entirely normal or may show mild leukocytosis, elevated protein, and/or oligoclonal IgG (immune gamma) bands (1, 7).

Although the pathogenesis of acute disseminated encephalomyelitis remains unknown, the leading hypothesis is that the disorder results from an autoimmune reaction to myelin triggered by a virus (or vaccine) (9, 10). In theory, a molecular (presumably protein) focus on the surface of myelin has a similar chemical structure to one of the surface antigens of the infecting virus, resulting in the production of antiviral antibodies, which can be misdirected against myelin by the immune system. This hypothesis is supported by the striking similarity of acute disseminated encephalomyelitis to experimen-

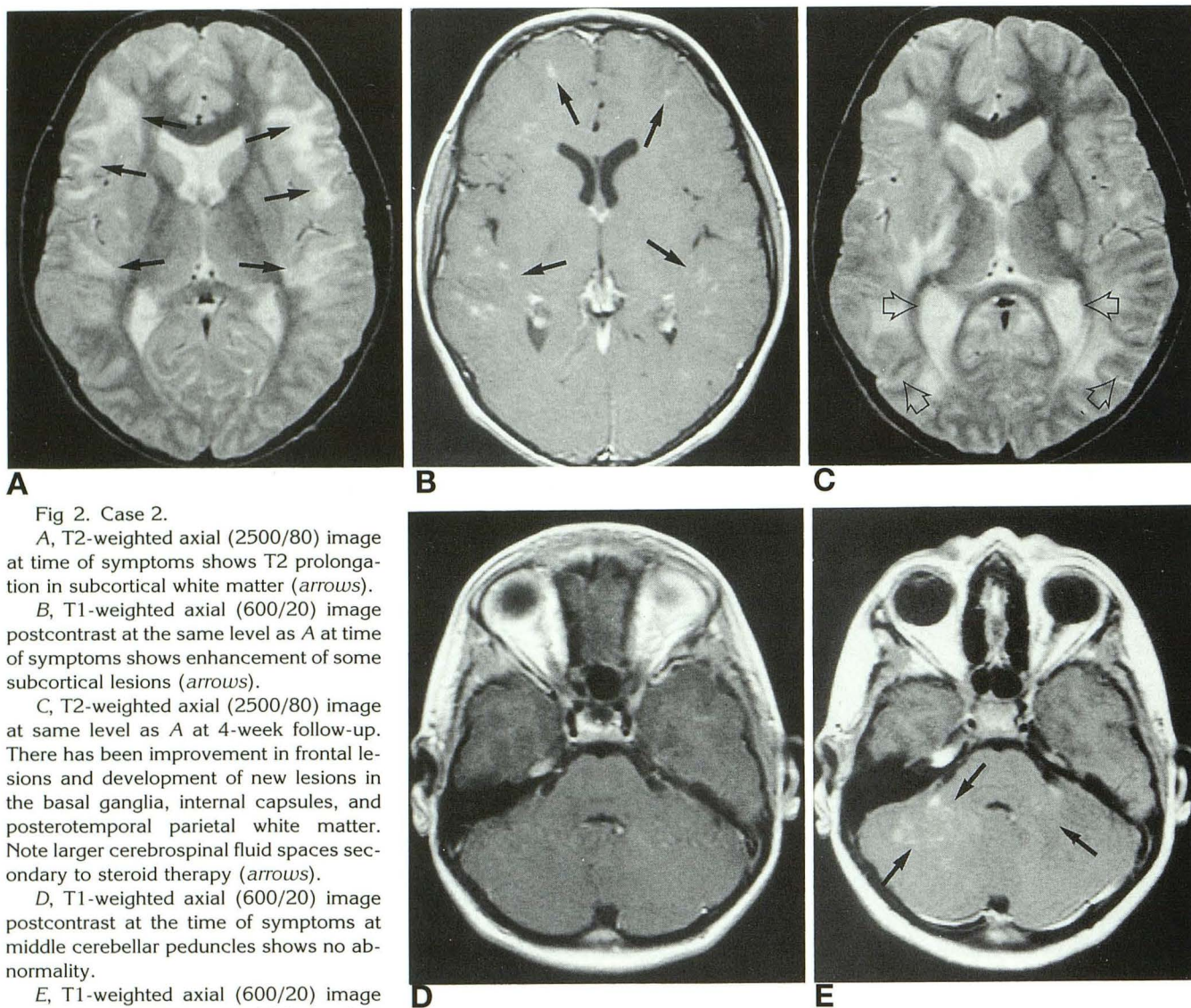


Fig 2. Case 2.

A, T2-weighted axial (2500/80) image at time of symptoms shows T2 prolongation in subcortical white matter (arrows).

B, T1-weighted axial (600/20) image postcontrast at the same level as A at time of symptoms shows enhancement of some subcortical lesions (arrows).

C, T2-weighted axial (2500/80) image at same level as A at 4-week follow-up. There has been improvement in frontal lesions and development of new lesions in the basal ganglia, internal capsules, and posterotemporal parietal white matter. Note larger cerebrospinal fluid spaces secondary to steroid therapy (arrows).

D, T1-weighted axial (600/20) image postcontrast at the time of symptoms at middle cerebellar peduncles shows no abnormality.

E, T1-weighted axial (600/20) image postcontrast (fourth week of follow-up) at same level as D shows multiple new areas of enhancement (arrows).

tal allergic encephalomyelitis, in terms of both timing and histology. The fact that no virus can be consistently isolated from patients with acute disseminated encephalomyelitis also supports this hypothesis and is evidence against direct viral toxicity as the mechanism of injury (10).

The histopathologic hallmark of acute disseminated encephalomyelitis, regardless of cause, is a zone of demyelination around veins in association with infiltration of vessel walls and perivascular spaces by lymphocytes, plasma cells, and monocytes. The myelin sheaths show degradation with relative sparing of the axons. At the edge of lesions there is microglial cell proliferation; these cells contain

phagocytized and degraded myelin. This same intense inflammatory response is also known to occur in gray matter (2, 8). Demyelination probably contributes little to the MR characteristics of the affected brain tissue. In fact, both the lipid protons of myelin and the water associated with it are effectively invisible on MR because of their short relaxation times (11). The increase in free water concentration that accompanies the demyelination process secondary to inflammation, edema, and loss of myelin results in increased T1 and T2 relaxation times (11). If the insult is severe enough, a breakdown in the blood-brain barrier occurs, resulting in contrast enhancement of the lesion. This same

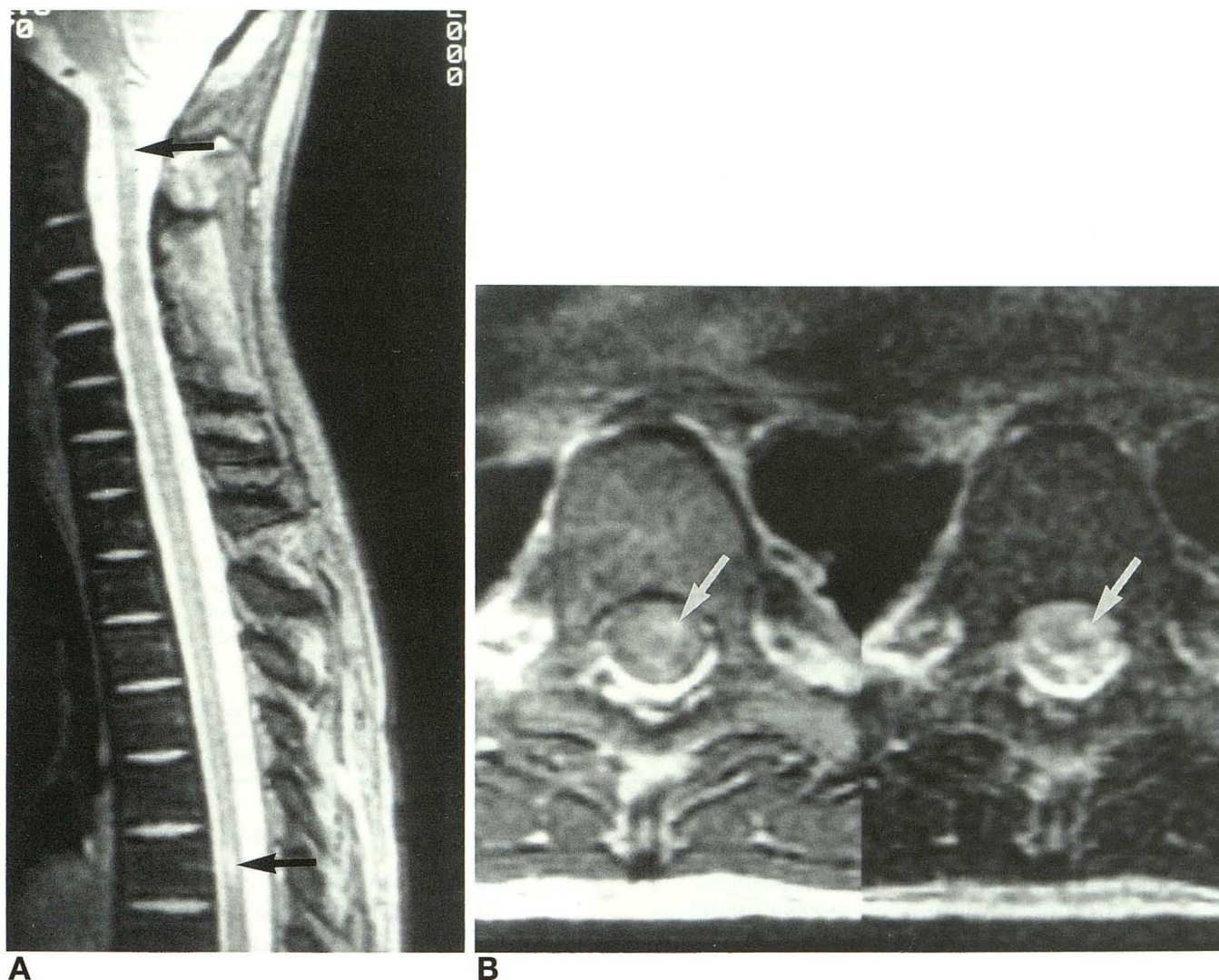


Fig 3. Case 10.

A, Multiplanar gradient-echo sagittal (600/15, 20° flip angle) image shows linear T2 prolongation posterior to C-2 and T-6 levels (arrows).

B, T2-weighted axial images (1800/30/80) at T-6 confirm cord abnormality (arrows).

breakdown of the blood-brain barrier has been reported in other demyelinating diseases, such as multiple sclerosis and central pontine myelinolysis (12, 13).

The resolution of MR abnormalities is likely the result of resorption of abnormally increased interstitial water and, if frank demyelination has occurred, remyelination (14). Some lesions may remain visible on MR as areas of T2 prolongation. These areas probably represent the astrocytic hyperplasia and gliosis known to occur in late stages of acute disseminated encephalomyelitis. Regions of astrocytosis and tissue damage (with loss of normal tissue) have longer T2 relaxation times than normal brain. Thus,

some white matter lesions seen in asymptomatic patients may be the sequelae of past acute disseminated encephalomyelitis (15).

Multiple radiologic reports have emphasized only the white matter lesions of acute disseminated encephalomyelitis; this is not surprising, given that the main histopathologic abnormality is demyelination (16, 17, 18). However, gray matter also contains myelin, as well as oligodendrocytes, the main function of which is to form and maintain myelin. Thus, MR signal abnormalities might be expected in deep gray matter structures from a perivenous demyelinating process such as acute disseminated encephalomyelitis. Gray matter lesions also have

Ten patients with acute disseminated encephalomyelitis

Patient	Age/ Sex	Clinical Data	Imaging	White Matter Involvement	Cortical Involvement	Basal Nuclei Involvement	Enhancement	Other Areas of T2 Prolongation
1	4 y/M	Spastic diplegia	MR 2/13/91 MR 10/23/91	Body CC, subcorti- cal white matter Total resolution on follow-up		Globus pallidus L Putamen-bilateral patchy asym- metric Caudate-bilateral heads Thalamic-bilateral patchy asym- metric <1-1.5 cm	2/13/91 Patchy thalamic R posterior frontal subcortical white matter 10/23/92 No contrast	1-cm foci dorsal brain stem cerebellar white matter
2	8 y/M	Cranial nn palsy	MR 7/17/92 MR 8/13/92	7/17-subcortical frontal, parietal, temporal, body CC 8/13-improved frontal, ↑ 'd post-periven- tricular, internal capsule, CR, splenum CC ↑ Posterior frontal/ parietal sub- cortical ↑ Patchy cerebellar white matter ↑ Internal capsule	Bifrontal cortex	Caudate-R body on follow-up Left posterior pu- tamen on fol- low-up Right putamen/ globus pallidus on follow-up	7/17 patchy subcortical and CR 8/13 near total resolution su- pratentorial enhancement ↑ Patchy asym- metric cerebellar enhancement	
3	9 y/M	Cranial nn palsy	MR 10/22/92	Centrum semiovale Bilateral/CR periventricular (posterior/anterior) External capsule/ internal capsule (posterior L)		Caudate-heads bilateral diffusely symmetric	No contrast given	Dorsal midbrain Bilateral middle cerebellar peduncles
4	5.5 y/F	Spastic hyper- reflexia	MR 3/8/90 CT 3/6/90	Multiple subcortical cerebellum and cerebrum			No contrast given	
5	13 mo/F	Coma	Ct 2/25/92 MR 3/4/92	Bilateral asym- metric centrum semiovale R CR Minimal L frontal subcortical		Thalamic-bilateral asymmetric 4.0 cm R 3.5 cm L	CT-MR, no en- hancement	
6	5 y/F	Mental status changes hemipar- esis	MR 10/26/86	Bilateral asym- metric sub- cortical frontal/temporal/ parietal	L posterior frontal parietal		No contrast given	
7	7 y/M	↓ Mental status cranial nn palsy	MR 5/8/91 CT 5/7/91	Bilateral asym- metric sub- cortical frontal-temporal extreme capsule	Bilateral asym- metric frontal/ insular/temporal/ Parietal at gray-white junction		CT, no en- hancement	<1-cm middle and R pontine lesion R middle cere- bellar pe- duncle le- sions

Table continued

Patient	Age/ Sex	Clinical Data	Imaging	White Matter Involvement	Cortical Involvement	Basal Nuclei Involvement	Enhancement	Other Areas of T2 Prolongation
8	12 mo/M	L hemiparesis	MR 12/27/91	Patchy subcortical Bilateral asymmetric centrum semiovale, CR Periventricular (posterior) external capsule	Minimal L frontal Minimal R posterior temporal R frontal parietal	Globus pallidus-bilateral asymmetric Putamen-bilateral asymmetric Caudate heads bilateral body on R Thalami-bilateral asymmetric	No contrast given	
9	2 mo/M	Acute onset mental status changes Quadripareisis	MR 5/10/89	Minimal bilateral asymmetric CR Body CC	R posterior frontal/parietal	Globus pallidus-bilateral patchy asymmetric <2 cm Putamen-bilateral patchy asymmetric <2 cm Caudate-head and body bilateral patchy asymmetric <2 cm Thalami-bilateral patchy asymmetric <2 cm	No contrast given	L dentate nucleus R middle cerebellar peduncle
10	13 y/M	Seizure myelopathy	MR brain/ T spine 10/23/91				No contrast given	Focal linear ↑ T2 cord C-1-C-2, T-6

Note.—CR indicates corona radiata; CC, corpus callosum.

been described in extrapontine myelinolysis and multiple sclerosis (19, 20).

The fact that gray matter involvement in acute disseminated encephalomyelitis is not rare should be kept in mind when interpreting imaging studies of children with acute neurologic signs and symptoms. Deep gray matter involvement should not militate against the diagnosis or be thought of as atypical for acute disseminated encephalomyelitis. The neuroimaging differential diagnosis of basal ganglia and thalamic lesions consists of toxic, metabolic, and hypoxic-ischemic causes (21). The pattern of involvement in patients with acute disseminated encephalomyelitis is somewhat distinctive, being mostly bilateral, asymmetric, and always (in our cases) associated with asymmetric white matter disease. Most metabolic and toxic abnormalities are bilateral and symmetric. Other disorders, such as mitochondrial cytopathies, viral encephalitis, and vasculitis, can have similar imaging findings; however, differentiation can be made on a clinical basis in most cases.

Differentiation of acute disseminated encephalomyelitis from the initial presentation of multiple sclerosis is not possible, even by the combination of clinical features, cerebrospinal fluid analysis, and MR (15). In fact, in our experience, a significant but unknown fraction of patients presenting initially as acute disseminated encephalomyelitis will go on to develop relapses, establishing a clinical diagnosis of multiple sclerosis. Because the prognoses of acute disseminated encephalomyelitis and multiple sclerosis differ significantly, their distinction at the time of initial presentation is important. Prior series comparing MR findings in acute disseminated encephalomyelitis with those in multiple sclerosis showed significant overlap. It was suggested that bilateral symmetric involvement in the occipital and parietooccipital white matter, cerebellar peduncles and cerebellar white matter should suggest acute disseminated encephalomyelitis as a diagnosis (15); unfortunately, these findings are not commonly seen in acute disseminated encephalomyelitis and, indeed, were uncommon in this series. Basal ganglia

involvement, seen in 50% of patients in the present series, can be seen in up to 25% of patients with multiple sclerosis (11) and thus is of little use in distinguishing these entities. The presence of thalamic involvement in 40% of the patients in this series suggests that thalamic involvement potentially may be a useful sign, because thalamic involvement with multiple sclerosis is rare (11, 20). Although a single report does note MR evidence of thalamic involvement with multiple sclerosis, the images showed T2 shortening, not prolongation (22).

Theoretically, contrast-enhanced MR should help differentiate multiple sclerosis from acute disseminated encephalomyelitis. Because acute disseminated encephalomyelitis is usually a monophasic illness, all lesions theoretically should enhance at the same time. However, as illustrated in two of our patients and in a prior report, enhancement can occur in some, but not all, lesions of acute disseminated encephalomyelitis. Caldemeyer et al suggested that, although acute disseminated encephalomyelitis is usually clinically a monophasic disease, some of the demyelinating lesions may occur in silent areas of the central nervous system (23). Thus, although the demyelination develops over a period of time, a monophasic illness results because of the occurrence of silent lesions. Alternatively, it is possible that the lesions are produced at the same time but are of different magnitudes, with different amounts of damage to the blood-brain barrier.

Although a monophasic illness in the vast majority of cases, acute disseminated encephalomyelitis does evolve over a variable period of time, usually 2 to 4 weeks. Therefore, the appearance of new lesions within this period is possible. The appearance of new lesions or progression of prior lesions with clinical improvement was noted in one of our cases. This has not been reported in acute disseminated encephalomyelitis. A prior case has shown progression of lesions on 2-week follow-up; however, a coincident progression of clinical symptoms was noted (15). Several prior cases have shown persistence of lesions as long as 18 months after acute presentation despite clinical improvement. These cases illustrate that extensive white matter signal abnormalities may be present in patients with acute disseminated encephalomyelitis but without clinical deficits, and that clinical recovery may precede the normalization of the MR examination (14, 15). Rarely,

neurologic disturbances recur for up to 18 months after a typical presentation of acute disseminated encephalomyelitis without subsequent development of multiple sclerosis. The criteria of the Poser Committee require a minimum of 1 month between separate episodes to qualify as relapses of multiple sclerosis. Others believe this should be a minimum of 6 months when the clinical features are more typical of acute disseminated encephalomyelitis (15).

Isolated involvement of the spinal cord with a presentation of transverse myelitis, seen in one of our patients, was an atypical presentation of acute disseminated encephalomyelitis. Lesions of the spinal cord are usually associated with encephalitis but may be seen alone. Symptoms referable to cord involvement usually occur with a longer latency after infection than the encephalitis. Clinical symptoms often begin with mid- or low-back pain, then progress to urinary hesitancy and finally to paralysis. Most patients make a moderate to complete recovery (7). Our results do not allow us to comment on the frequency of spinal involvement in acute disseminated encephalomyelitis, because the spinal cord was imaged in only one patient.

In summary, 10 cases of clinically proved acute disseminated encephalomyelitis are reviewed. Involvement of deep gray matter was common in our small series. In fact, we no longer consider this finding atypical. The finding of T2 prolongation in these structures should not discourage the diagnosis of acute disseminated encephalomyelitis in the proper clinical setting. Although our series is small, it suggests that thalamic involvement may prove useful in distinguishing between acute disseminated encephalomyelitis and the initial presentation of multiple sclerosis.

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