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A M Wang, J H Morris, W F Hickey, S B Hammerschlag, G V O'Reilly and C L Rumbaugh

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Unusual CT Patterns of Multiple Sclerosis

Ay-Ming Wang[1]
James H. Morris[2]
William F. Hickey[2]
Steven B. Hammerschlag[3]
Gerald V. O'Reilly[3]
Calvin L. Rumbaugh[1, 3]

In three patients with histologically confirmed, acute demyelinating disease (multiple sclerosis), serial cranial computed tomography revealed unusual findings. Computed tomography showed contrast-enhancing lesions with mass effect, which suggested brain tumors and the need for surgical biopsy. Steroids were administered to each patient; in two, the large mass lesions resolved dramatically. These and other rapid changes may help in the recognition of multiple sclerosis.

Computed tomographic (CT) abnormalities have been reported in 30% of patients with cerebral demyelinating disease. These include focal areas of reduced attenuation in white matter, atrophy (enlarged sulci, ventricular dilatation), and contrast-enhanced lesions without mass effect [1–4]. The most common abnormality is a focal area of decreased attenuation in white matter [5].

Contrast enhancement of demyelinating lesions is unusual [1] and is probably related to local breakdown of the blood-brain barrier in the acute phase of the disease [6–8]. Contrast-enhanced lesions with mass effect are even more unusual. Only two reports describe multiple sclerosis presenting as a mass with enhancement on the CT scan [9, 10]. The three patients described below were all proved by biopsy to have acute demyelinating disease. In each case, initial CT evaluation showed contrast enhancement with mass effect; sequential scans demonstrated rapid changes in these patterns over a very short period of time.

Case Reports

Case 1

A 39-year-old woman was admitted to Brigham and Women's Hospital with rapid onset of left lower facial weakness, dysarthria, and a sensation of heaviness of the left arm. Two weeks after the onset, CT demonstrated an area of low density and homogeneous contast enhancement in the white matter of the right parietal lobe, just superior to the body of the right lateral ventricle (figs. 1A and 1B).

Six weeks after onset, she developed a weakness in her left leg. Repeat CT showed obvious changes from the earlier study; the low density area had enlarged, and its rim was now markedly enhanced (figs. 1C and 1D). Treatment with dexamethasone was begun but resulted in no clinical improvement. A right internal carotid angiogram showed a relatively avascular mass in the right parietal lobe. This was presumed to be a brain tumor. Craniotomy and brain biopsy were performed in order to confirm the diagnosis.

The biopsy specimen consisted of fragments of identifiable, normal-appearing gray and white matter, together with pale, yellow tan tissue. Histologic examination showed normal cortex and white matter containing some reactive hypertrophic astrocytes. Some of the tissue was characterized by large, pleomorphic, hypertrophic astrocytes interspersed among foamy macrophages; the latter contained aminosalicylic acid–positive material embedded in a fibrillary background. Several blood vessels within the lesion had prominent cuffs of normal-appearing lymphocytes. Scattered lymphocytes and occasional plasma cells were also seen within the damaged parenchyma. The lesion was quite sharply

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[1]Department of Radiology, Harvard Medical School, and Brigham and Women's Hospital 75 Francis St., Boston, MA 02115. Address reprint requests to A. M. Wang.

[2]Department of Pathology, Harvard Medical School, and Brigham and Women's Hospital, Boston, MA 02115.

[3]Department of Radiology, Harvard Medical School, and Beth Israel Hospital, Boston, MA 02115.

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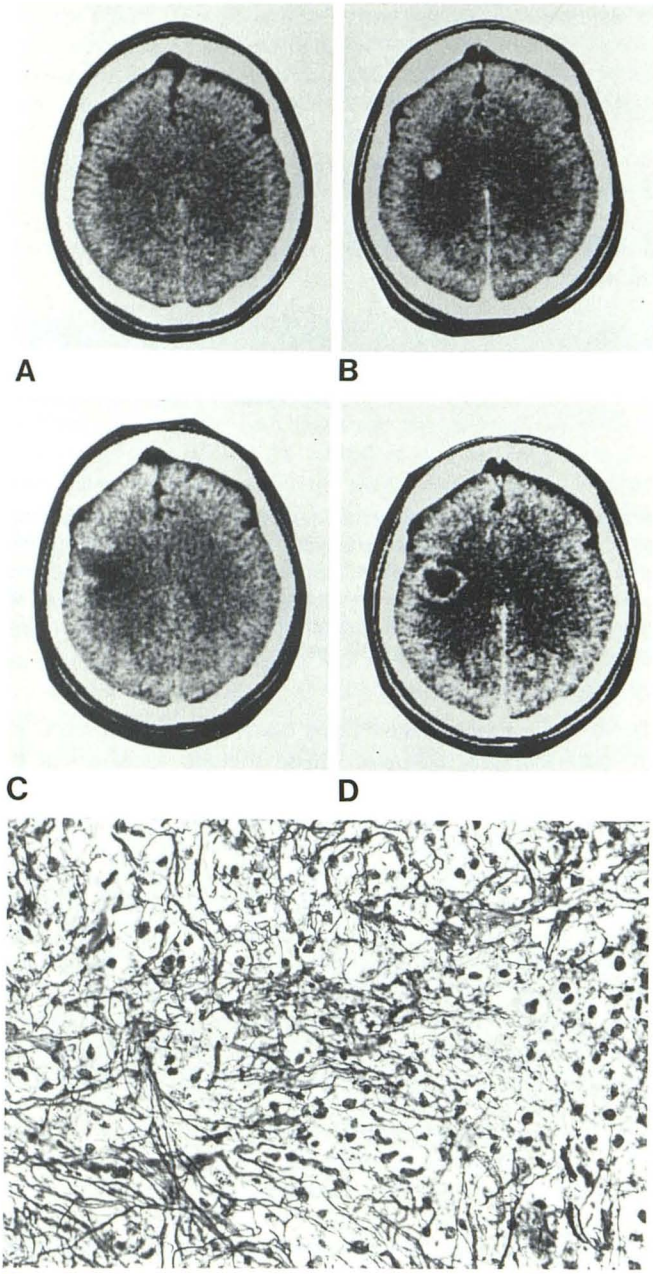

Fig. 1.—Case 1. CT scans 2 weeks after onset of symptoms before (**A**) and after (**B**) contrast enhancement. Homogeneous, contrast-enhanced low-density lesion in white matter of right parietal lobe. Scans 3 weeks later without (**C**) and with (**D**) contrast enhancement show rim enhancement. **E**, Bodian (silver) stain for axons shows numerous normal-appearing demyelinated axons in middle of lesion. (Bodian ×200.)

demarcated from adjacent white matter. It showed an almost complete absence of myelin despite nearly normal axon content (fig. 1E), thereby establishing the diagnosis of acute demyelinating disease. At a follow-up examination 2 months later, the patient was asymptomatic without treatment.

Case 2

A 30-year-old woman fell while jogging and subsequently noticed some left leg weakness. At Brigham and Women's Hospital 2 weeks later, she had left-sided hyperreflexia, decreased position sense in the left leg, and poor three-dimensional construction. CT revealed a large (8 × 3 cm), right, parasagittal lesion near the corpus callosum and above the right lateral ventricle (figs. 2A and 2B). The cigar-shaped lesion, which homogeneously enhanced with contrast material, produced a mass effect in the surrounding white matter (figs. 2C and 2D). The patient was presumed to have a brain tumor, possibly a microglioma; she was admitted to the hospital and treated with dexamethasone.

Four weeks after the first CT scan, a second scan showed that the lesion had almost disappeared (figs. 2E and 2F). In the interim, a needle biopsy of the brain was unsuccessfully attempted. Three weeks after the second scan, the patient developed mental confusion, with a decrease of memory and right hand weakness. Dexamethasone was discontinued.

Three weeks later, a third CT scan was obtained; this revealed periventricular contrast enhancement as well as multiple contrast-enhanced, new lesions with low-density areas in the cerebral hemispheric white matter (figs. 2G and 2H).

Successful needle biopsy of the original parasagittal lesion was performed (8 weeks after the onset of new symptoms and 3 weeks after the third CT scan). Histologic examination revealed normal cerebral cortex, white matter containing hypertrophic astrocytes, and tissue similar to that seen in case 1. The lesion was seen to be sharply demarcated from the adjacent white matter. It was characterized by the presence of reactive astrocytes and foamy macrophages; the tissue contained vessels with perivascular cuffs of lymphocytes and had a normal content of axons together with an almost total lack of stainable myelin (figs. 2I and 2J). The diagnosis of acute demyelinating disease was made.

Two weeks after this second biopsy, the patient's condition again worsened. Her level of alertness decreased, and she developed incontinence and aphasia. Dexamethasone was restarted. A month later, while she was still on this medication, her general condition deteriorated further, and a right optic neuritis developed. A fourth CT study showed fewer contrast-enhanced lesions, together with a diminution of the low-density areas in the cerebral white matter. The patient died 3 months later. Autopsy demonstrated multiple demyelinating plaques.

Case 3

A 21-year-old woman was admitted to Beth Israel Hospital with sudden vision impairment, speech difficulties, and unstable gait of 2 weeks duration. She showed a left homonymous hemianopsia and a right superior quadrantanopsia. CT (figs. 3A and 3B) revealed multiple lesions throughout the cerebral hemisphere, as well as periventricular contrast enhancement. A large, contrast-enhanced lesion with surrounding edema was noted in the right occipital region. Right internal carotid and left vertebral angiograms demonstrated luminal irregularity in numerous small arteries throughout the right cerebral hemisphere and posterior fossa. Early venous drainage was also present in these areas. Biopsy of the right occipital lobe 1 week after admission revealed acute demyelinating disease.

A second CT study (figs. 3C and 3D) with contrast enhancement was performed 1 week after the biopsy procedure and after the patient had been receiving dexamethasone for 1 week. The scan showed a decrease in periventricular contrast enhancement and complete resolution of the diffuse contrast-enhanced cerebral lesions. A low attenuation area involving the right occipital lobe was again seen, but no contrast enhancement was identified. On a follow-up examination 1 year later, there was no change in the visual impairment, but the unstable gait and dysarthria had disappeared. The patient was no longer receiving therapy.

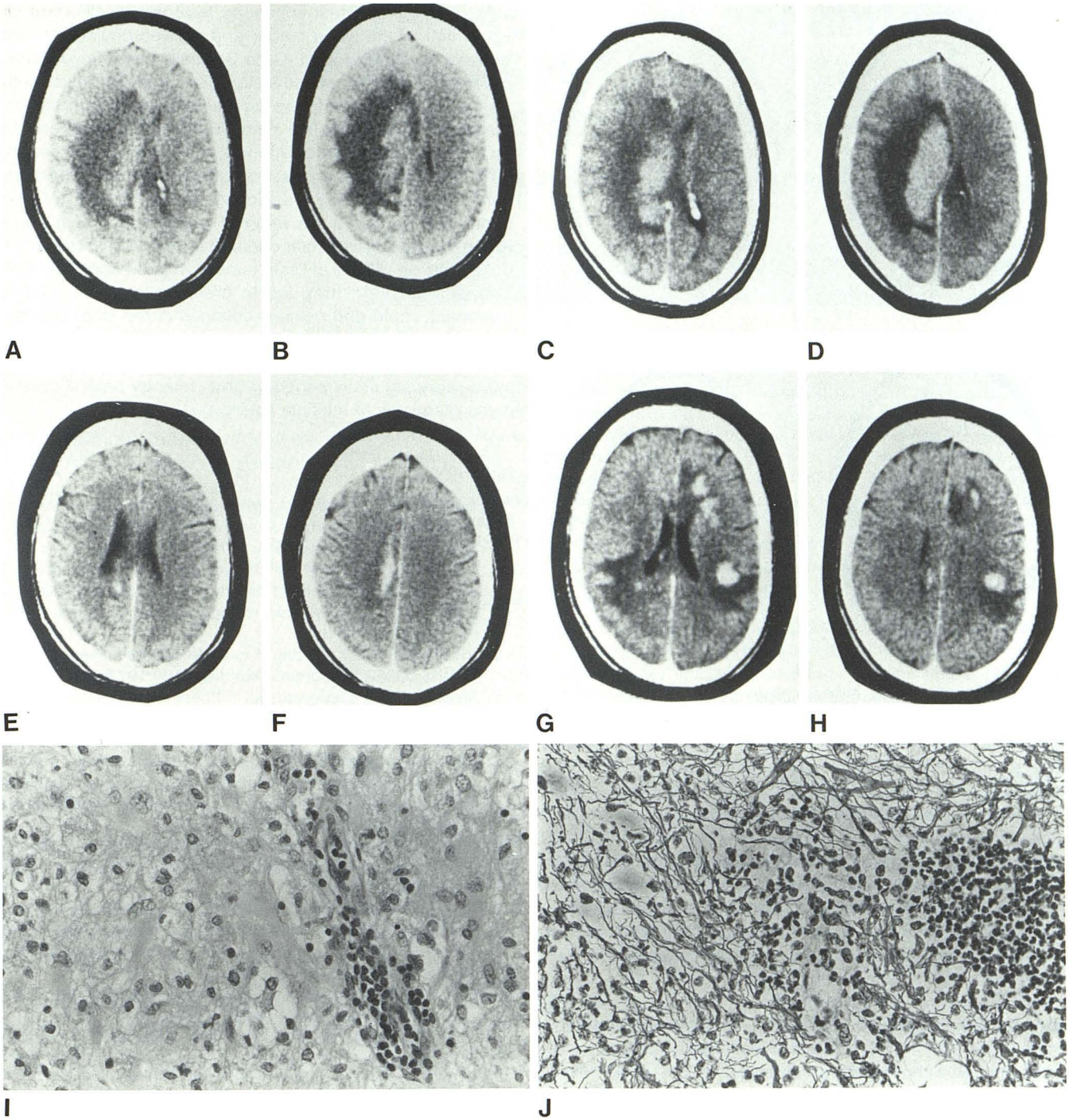

Fig. 2.—Case 2. Initial CT scans without (**A** and **B**) and with (**C** and **D**) contrast enhancement. Enhancing mass in white matter of right frontoparietal region is 8 × 3 cm. Prominent zone of edema borders mass. **E** and **F**, 3 weeks later with contrast enhancement. Mass in white matter of right frontoparietal region has diminished greatly, contemporary with steroid treatment. **G** and **H**, 6 weeks later. Contrast enhancement shows several lesions, decrease in density of white matter, and slight periventricular enhancement. **I**, Tissue from lesion. Small blood vessel with cuff of normal-appearing lymphocytes. Parenchyma contains prominent reactive hypertrophic astrocytes, numerous foamy macrophages, and complete loss of stainable myelin. (H and E ×540.) **J**, Demyelinated axons in lesion adjacent to blood vessel, which is surrounded by cuff of normal-appearing lymphocytes and foam macrophages. (Bodian ×200.)

Discussion

Multiple sclerosis is a progressive, demyelinating disease that usually has its onset in early adult life. The disease is characterized by irregular periods of exacerbation and remission and by brain lesions, which appear pathologically as demyelinating plaques with gliosis. On CT, multiple sclerosis may manifest itself in one of several ways: nonspecific, cortical atrophy; transient, focal lesions that are low in density, but that enhance with contrast; lesions low in atten-

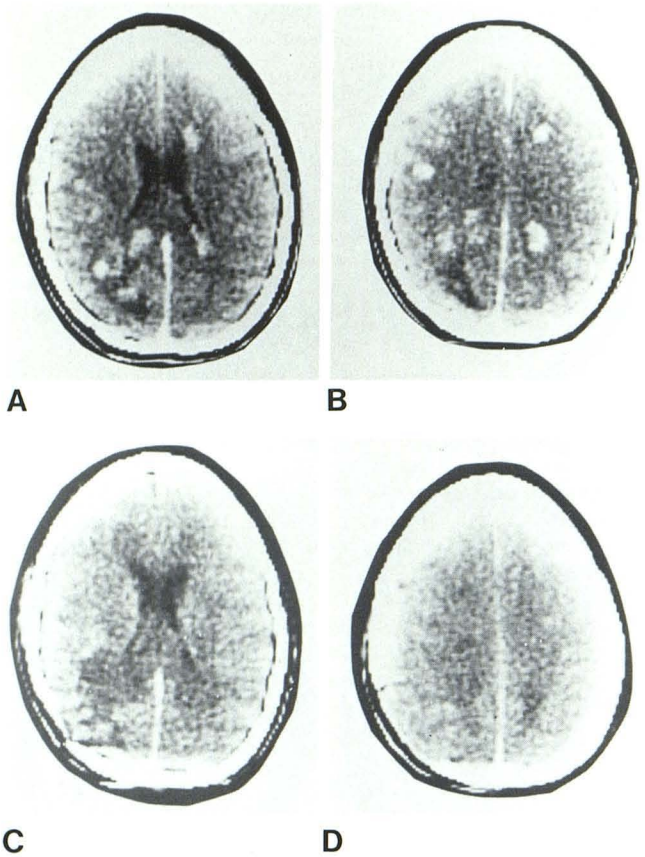

Fig. 3.—Case 3. **A** and **B**, Admission scans. Several contrast-enhanced lesions throughout white matter of cerebral hemispheres, periventricular contrast enhancement, and single large, low-density mass in white matter of right occipital region with scattered areas of enhancement. **C** and **D**, 2 weeks later after 1 week of steroid treatment. Almost complete resolution of diffusely contrast-enhanced cerebral lesions, together with decrease in periventricular contrast enhancement. Persistent area of low density involved white matter of right occipital lobe, with absence of contrast enhancement.

uation that do not enhance with contrast; and, rarely, mass lesions with contrast enhancement. Only two cases with mass effect have been reported [9, 10]. Both cases showed low-density lesions with rim enhancement. The first had the diagnosis proven at autopsy; the second patient had the diagnosis established by biopsy.

The CT findings in our three patients were unusual. In case 1, the initial lesion showed homogeneous contrast enhancement, but this changed to rim enhancement in a 4 week interval without steroid treatment. In case 2, an initial large, contrast-enhanced cerebral mass showed dramatic diminution, as well as alterations in the pattern of contrast enhancement with 4 weeks of steroid treatment. Six weeks later, multiple contrast-enhanced lesions with bilateral involvement of the white matter and periventricular contrast enhancement developed. Case 3 initially had a large mass, several contrast-enhanced lesions, and periventricular contrast enhancement; a follow-up study 2 weeks after steroid therapy showed complete resolution of the lesions except for one area of decreased attenuation without enhancement.

In our three patients, histologically proven acute cerebral demyelinating disease was found. In each case, a large, contrast-enhanced mass with rim enhancement or multiple lesions with periventricular contrast enhancement raised the diagnostic possibility of brain tumor, lymphoma, or infection. This possibility, in turn, led to brain biopsy. After steroid treatment, rapid and marked changes in the size, number, and pattern of contrast-enhanced lesions in the white matter were demonstrated. These changes, which took place during a relatively short period of time, may be helpful criteria in the diagnosis of multiple sclerosis.

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