

Acute Aphasia in Multiple Sclerosis

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Acute aphasia is rare in multiple sclerosis. We describe 3 patients with multiple sclerosis who had acute exacerbations presenting as aphasias. One patient had a mixed transcortical aphasia, 1 had a transcortical motor aphasia, and 1 had a Broca aphasia. Magnetic resonance imaging scans of the brain with contrast enhancement revealed new white matter lesions in the left hemisphere in all 3 patients. Two of the 3 patients had a good response to treatment with methylprednisolone sodium succinate.

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Multiple sclerosis (MS) primarily affects the white matter of the brain and spinal cord.^{1,2} Aphasia rarely occurs as a clinical manifestation of MS.² Since aphasia is usually associated with diseases of the gray matter, it is not an expected presentation of MS. Only a handful of case reports in the literature describe this entity.³⁻⁶ We describe 3 patients with acute MS exacerbations presenting as aphasias with lesions compatible with their new symptoms demonstrated on magnetic resonance imaging (MRI) scans (Table). The patients were followed up at the Washington University Multiple Sclerosis Clinic, St Louis, Mo. The clinic provides care to more than 1300 patients with MS. All 3 patients were diagnosed as having clinically definite MS.^{2,7}

REPORT OF CASES

CASE 1

A 24-year-old left-handed woman with relapsing-remitting MS had a history of exacerbations that included left hemiparesis, dysarthria, poor coordination, and weakness. She was admitted with a 5-day history of right leg weakness and a 1-day history of "trouble getting words out." On examination, her speech was slightly dys-

arthric and her language was nonfluent. Naming was impaired, and she had difficulty following complex commands. Her sentences consisted of only 3 to 4 words, and her reading was impaired. Repetition was intact. She had a new right-sided hemiparesis, along with bilateral hemianesthesia and dysmetria.

An MRI scan of the brain revealed interval formation of large confluent foci in the deep white matter of both cerebral hemispheres, with extensive involvement of the periventricular white matter adjacent to the frontal and occipital horns on fluid attenuation inversion recovery images and T2-weighted sequences. Post-gadolinium T1-weighted images demonstrated a small area of contrast enhancement in the left frontal area corresponding to a 2 × 3-cm area of hyperintensity on T2-weighted images.

The patient was treated with 5 g of intravenous methylprednisolone sodium succinate over 5 days. Her aphasia and right-sided hemiparesis gradually improved over several months. At a follow-up visit more than 1 year later, she reported only occasional word-finding difficulty when speaking quickly.

CASE 2

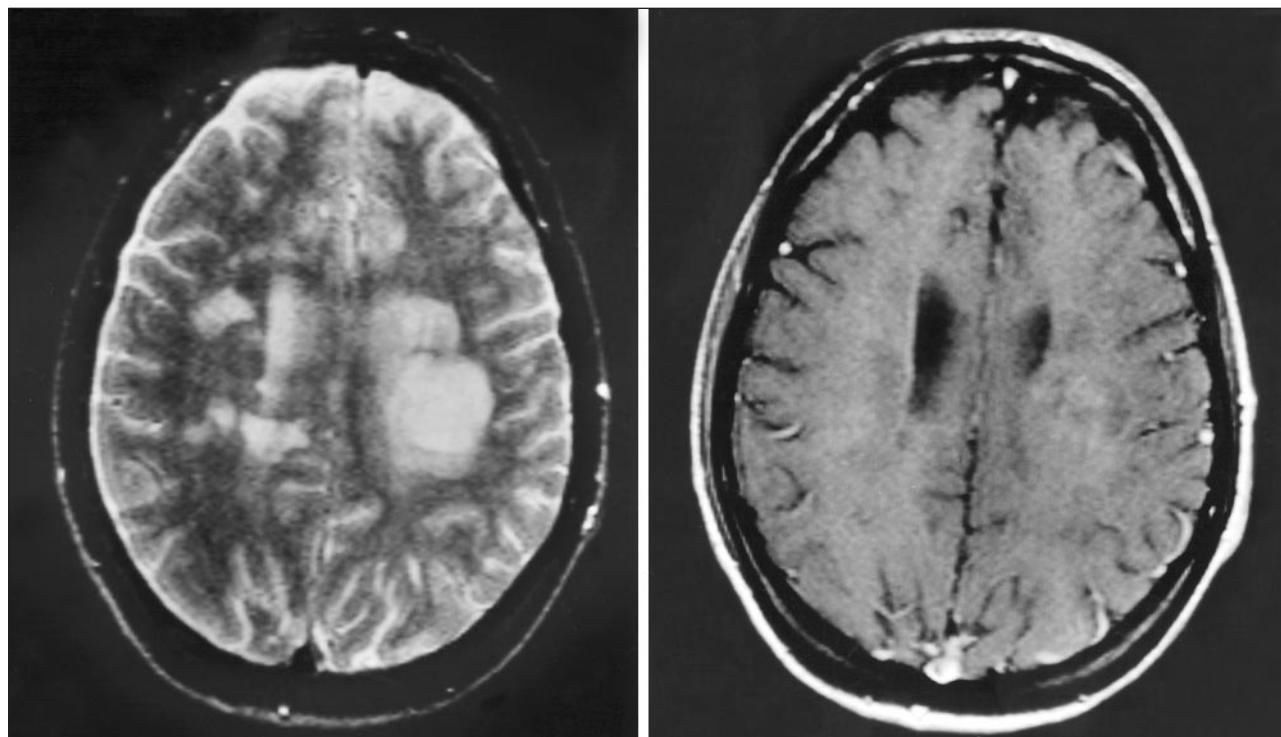
A 45-year-old right-handed woman with an 8-year history of relapsing-remitting MS had

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Data on 3 Patients With Relapsing-Remitting Multiple Sclerosis

Patient No./ Age, y/Sex	Type of Aphasia	White Matter Finding on MRI*
1/24/F	Mixed transcortical	3 × 2-cm Lesion with small area of contrast enhancement in left frontal lobe
2/45/F	Broca	3 × 2-cm Lesion with trace contrast enhancement in left temporal lobe
3/25/M	Transcortical motor	5 × 3-cm Foci with contrast enhancement in left parietal lobe

*MRI indicates magnetic resonance imaging.



Patient 3. Magnetic resonance imaging scans of the brain. Left, T2-weighted sequence with a large left parietal lesion. Right, T1-weighted postgadolinium sequence with contrast enhancement in the left parietal lesion.

been stable since her last hospitalization for right leg weakness 1 year earlier. She presented with a 2-week history of poor memory and language impairment. On examination, her language was nonfluent, with paraphasic errors and prominent anomia. Repetition was mildly impaired. Comprehension was intact. She had poor recall on memory testing. The findings of the rest of her neurologic examination were remarkable for patchy hypesthesia, spasticity, and an unsteady gait.

An MRI scan of the brain revealed extensive periventricular white matter disease, including a 3×2 -cm area in the white matter of the left temporal lobe on T2-weighted images. Postgadolinium T1-weighted images revealed a trace amount of contrast enhancement in the left temporal lobe lesion.

The patient was treated with 5 g of intravenous methylprednisolone sodium succinate over 5 days. She had incomplete improvement in her aphasia and was transferred to a rehabilitation unit. The course of her MS later became progressive, and she is now in a nursing home, with marked cognitive impairment.

CASE 3

A 25-year-old right-handed man with a 4-year history of relapsing-remitting MS presented with an acute exacerbation associated with aphasia. Six weeks before admission, he developed right hand weakness and right foot drop. These symptoms resolved spontaneously after 1 week. Two weeks later, he developed paresthesias in the right side of his face and in his right arm and leg. Four days before admission, his speech became slurred and he began to have difficulty "finding words." On examination, he demonstrated dysarthria, impaired fluency with word-finding difficulty, paraphasic errors, and right-sided hemiparesis with bilateral spasticity and hypesthesia. Comprehension and repetition were intact.

An MRI scan of the brain (Figure) revealed multiple white matter lesions in the periventricular areas and the pons. There was also a new 5×3 -cm left frontoparietal subcortical lesion that was most prominent on T2-weighted images and demonstrated contrast enhancement on T1-weighted postgadolinium sequences.

The patient was treated with 5 g of intravenous methylprednisolone sodium succinate over 5 days. Examination a few months later demonstrated complete resolution of his aphasia.

COMMENT

Patients with MS often develop cognitive abnormalities that have been reported to occur with a frequency of up to 65%.^{2,8} These abnormalities include memory deficits, psychomotor slowing, visual spatial impairment, and poor conceptional reasoning. Language deficits are relatively uncommon. Moreover, cognitive deficits in MS typically develop progressively over time. The unusual aspect of our 3 cases is the development of acute language impairment.

Although cortical lesions produce most cases of aphasia, a variety of subcortical white matter lesions may cause similar clinical syndromes. Subcortical strokes and intraparenchymal hemorrhages involving the putamen, internal capsule, caudate nucleus, and thalamus have been associated with a variety of aphasias.⁹⁻¹¹ The concept of diaschisis, whereby impaired function occurs in one area of the brain secondary to an acute focal lesion in a more distant part of the brain, has been cited as one explanation for subcortical lesions causing aphasias.^{12,13} According to this concept, lesions of the white matter tracts that are anatomically connected to the cortical language centers could produce aphasias that are sometimes difficult to distinguish from aphasias that are associated with cortical lesions. Several studies have demonstrated cortical hypoperfusion in aphasic patients with acute strokes in the subcortical regions.^{12,13} However, the type of aphasia produced by a subcortical lesion may not always be predicted from the location of the lesion.¹²

All 3 of our patients presented with primarily motor aphasias as part of an acute MS exacerbation. Patient 1 had a mixed transcortical aphasia with more motor than sensory involvement; patient 2 had a Broca aphasia; and patient 3 had a transcortical motor aphasia. Friedman et al⁵ reported a case of global aphasia and Arnett et al⁶ reported a case of conduction aphasia in MS. However, the majority of previously reported cases of aphasia in MS have described motor aphasias.^{3,4,6} Achiron et al⁴ performed serial MRI scans on 2 patients with MS who developed acute motor aphasias. Each of their patients had interval development of a large plaque (>5 cm) located in the white matter of the dominant hemisphere. One patient had a 6-cm plaque in the left frontotemporal region. The other patient had a 5-cm plaque in the left centrum semiovale.

In our series, all 3 patients had MRI scans that demonstrated bilateral disease associated with 2- to 5-cm lesions in the left hemisphere and showed contrast en-

hancement on postgadolinium T1-weighted images. The MRI lesions in each patient occurred only in the white matter, with sparing of the gray matter. All 3 patients had relapsing-remitting MS at the time of their exacerbations. Two of our 3 patients had significant improvement in their aphasia after treatment with intravenous methylprednisolone. However, it is unclear if the clinical improvement was the result of treatment with methylprednisolone, or simply spontaneous recovery.

The experience in our center with acute aphasia in MS is consistent with the paucity of reported cases in the literature. We have identified only 3 cases in our MS clinic population during a period of more than 7 years. Despite the possibility that acute aphasia in patients with MS can represent an exacerbation of the disease, the clinician must always exclude cerebrovascular events, seizures, and infectious origins. This report provides further evidence that aphasia may present as an acute exacerbation in MS and is often associated with some recovery.

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