

Case Reports

Magnetic Resonance Imaging of Ataxic Hemiparesis Localized to the Corona Radiata

David H. Gutmann, MD, PhD, and Steven Scherer, MD, PhD

A 61-year-old woman developed right hemiparesis with homolateral cerebellar-type ataxia. Computed tomography and magnetic resonance imaging demonstrated left corona radiata lesions, not present on magnetic resonance imaging 1 year earlier. No brainstem lesions were identified, suggesting that ataxic hemiparesis can result from lesions in the corona radiata. (*Stroke* 1989;20:1571–1573)

Ataxic hemiparesis syndrome (AHS) was first described by Fisher and Cole.¹ This rare syndrome is manifested by weakness and ataxia characteristic of cerebellar dyscoordination (defective postural fixation and limb ataxia) on the same side of the body. Fisher and Cole¹ originally speculated that the lesion might lie in either the internal capsule or the corona radiata. Subsequent pathologic analysis of three cases in 1978 demonstrated small infarcts in the basis pontis at the level of the junction of the upper one third and the lower two thirds of the contralateral pons.² Reports of additional cases of AHS using computed tomography (CT) have shown lesions in a variety of supratentorial and infratentorial locations, most commonly in the posterior limb of the internal capsule and corona radiata.^{3–7} Since these observations were made by CT alone, which may fail to demonstrate small nonhemorrhagic infarcts, their exact correlation with AHS has been challenged.^{8,9} Kistler et al⁸ described a patient with AHS in whom a CT scan showed a lesion in the corona radiata only, while magnetic resonance imaging (MRI) revealed a pontine lesion that appeared to better explain the clinical findings. We describe a patient with AHS in whom premorbid and postictal MRI scans indicated recent infarction in the corona radiata.

Case Report

A 61-year-old right-handed woman with a history of hypertension and noninsulin-dependent diabetes mellitus presented to the Graduate Hospital emergency room after 4 days of right-sided weakness and clumsiness. Her medical history included glau-

coma, but her visual fields were persistently normal. Vague complaints of dizziness 1 year prior to admission prompted an MRI scan, which was normal. Four days before admission, the patient developed a staggering gait and clumsiness that progressed over the next 2 days to right-sided weakness, incoordination, and a vague right-sided numbness. On the day before admission, she experienced two episodes of garbled speech lasting several minutes and characterized by an inability to vocalize words without an impairment in verbal speech comprehension. She denied diplopia or other visual disturbances, meningismus, nausea, or vomiting.

On admission, examination showed her to be afebrile; her blood pressure was 138/70 mm Hg and her pulse was 64/min. The general medical examination was remarkable only for a fourth heart sound and a crescendo-decrescendo systolic murmur. There were no carotid bruits, and her neck was supple. She was alert, with normal cognition and fluent speech. The cranial nerves were intact. Motor examination showed 4/5 weakness of the right deltoid, triceps, and dorsal interossei. The sensory examination revealed that she was intact to cold, pin, vibration, graphesthesia, and stereognosis. Defective postural fixation was demonstrated by marked cerebellar-type ataxia with past-pointing on finger-to-nose and heel-to-shin testing as well as an abnormal Holmes rebound response of her right arm. Limb ataxia was manifested by slow, inaccurate, and dysrhythmic rapid alternating and fine movements of her right hand. She listed to the right while standing, and her gait was mildly hemiparetic. Deep tendon reflexes were brisker on the right side in both the arm and the leg, and her right plantar response was equivocal.

On the day of admission, a CT scan revealed a nonenhancing hypodensity of the left corona radiata that appeared as two adjacent lesions on an MRI scan obtained 48 hours later (Figure 1). The MRI

From the Department of Neurology, The University of Pennsylvania, Philadelphia, Pennsylvania.

Address for correspondence: David H. Gutmann, MD, PhD, Department of Neurology, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, PA 19104.

Received January 18, 1989; accepted March 13, 1989.

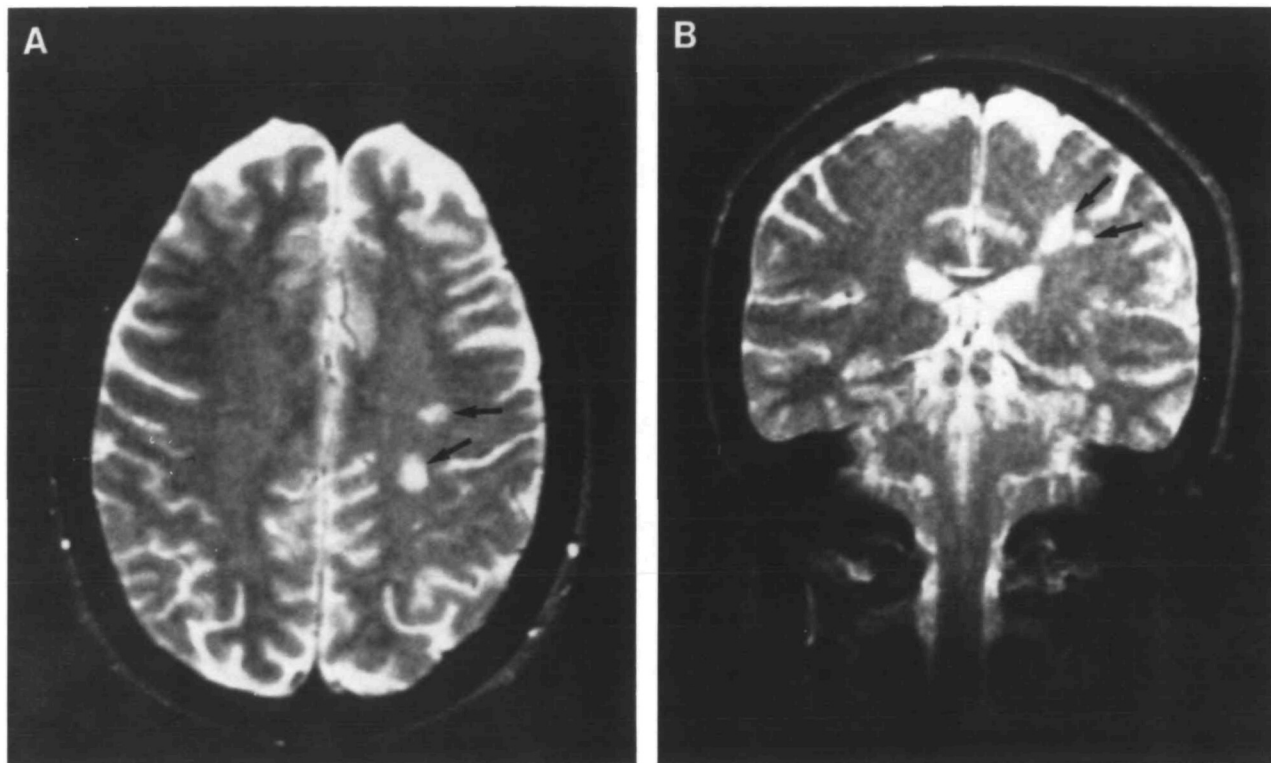


FIGURE 1. Magnetic resonance images of patient with ataxic hemiparesis 1 year after normal scan. Two small lesions are demonstrated in left corona radiata in axial plane (A). Coronal view (B) better delineates extent of these lesions. Both images are T2-weighted (resonance time, 2400 msec; echo time, 80 msec) produced on 1.5-T General Electric Signa unit (Milwaukee, Wisconsin) with spin-echo multisection imaging sequences.

scan also showed a small lesion at the tip of the left occipital lobe but no lesions in the brainstem. Complete blood count was normal, erythrocyte sedimentation rate was 18 mm/hr, and antinuclear antibody titer was <1:2. The rapid plasma reagin test was positive at a titer of 1:8, and the fluorescent *Treponema* antibody test was positive. Cerebrospinal fluid examination revealed three leukocytes, a protein concentration of 52 mg/dl, a glucose concentration of 114 mg/dl, and a nonreactive VDRL. An echocardiogram was unremarkable, while carotid Doppler ultrasound studies revealed a 50% stenosis in the left internal carotid artery. Over the next 5 days, there was partial resolution of her right hemiparesis and ataxia.

Discussion

Since Fisher's pathologic study,² there have been numerous descriptions of AHS in patients whose CT scans demonstrated lesions in only the internal capsule or corona radiata.³⁻⁵ However, the possibility of a concomitant pontine lesion could not be excluded on the basis of these CT findings. Rothrock et al⁹ reviewed a series of cases of lacunar strokes evaluated by MRI and reported one case of AHS with two normal MRI scans that failed to identify any lesions. Recently, Helwig et al¹⁰ reported three cases of AHS studied by MRI. Lesions were identified in the posterior limb of the internal cap-

sule, the midpons, and the red nucleus. None of these lesions were apparent on CT scans.

In our patient, who presented with a right hemiparesis and homolateral cerebellar-type ataxia, the initial CT scan demonstrated only a single left corona radiata lesion. It was suspected, based on the report of Kistler et al,⁸ that MRI would demonstrate a pontine lesion that was undetected on CT scan. Despite this, MRI failed to demonstrate any brainstem pathology and confirmed the presence of the left corona radiata lesion. These results provide strong support for the notion of Fisher and Cole¹ (as well as the findings of others) who described an association between corona radiata lesions and AHS. The significance of our patient's left occipital lesion is unknown; Goldman perimeter examinations of her visual fields have persistently been normal.

Lesions responsible for AHS have also been reported in the rostral mesencephalon and thalamus.^{6,7,9} The "cerebellar" qualities of the limb ataxia and postural fixation do not imply localization to the cerebellum, but rather imply that pathways affected in AHS are relatively preserved in pure hemiparesis. To produce homolateral ataxia and hemiparesis, it has been suggested that lesions must interrupt the fibers connecting the ventrolateral nucleus of the thalamus with the precentral cortex as they pass through the corona radiata and internal capsule. Alternatively, a lesion could pro-

duce AHS by disrupting the corticopontine fibers descending from the precentral cortex through the posterior capsular area to make connections in the basis pontis.⁵

Acknowledgments

We thank Drs. Donald Silberberg and Michael Kushner for their critical review of this manuscript.

References

1. Fisher CM, Cole M: Homolateral ataxia and crural paresis: A vascular syndrome. *J Neurol Neurosurg Psychiatry* 1965; 28:48–55
2. Fisher CM: Ataxic hemiparesis: A pathologic study. *Arch Neurol* 1978;35:126–128
3. Sage JI, Lepore FE: Ataxic hemiparesis from lesions of the corona radiata. *Arch Neurol* 1983;40:449–450
4. Sanguineti I, Tredici G, Beghi E, Aiello U, Bogliun G, DiLelio A, Tagliabue M: Ataxic hemiparesis syndrome: Clinical and CT study of 20 new cases and a review of the literature. *Ital J Neurol Sci* 1986;7:51–59
5. Iragui VJ, McCutchen CB: Capsular ataxic hemiparesis. *Arch Neurol* 1982;39:528–529
6. Bendheim PE, Berg BO: Ataxic hemiparesis from a midbrain lesion. *Ann Neurol* 1981;9:405–407
7. Murthy JMK: Ataxic hemiparesis—Ventrolateral nucleus of the thalamus: Yet another site of lesion (letter). *Stroke* 1988; 19:122
8. Kistler JP, Buonanno FS, DeWitt LD, Davis KR, Brady TJ, Fisher CM: Vertebral-basilar posterior cerebral territory stroke—Delineation by proton nuclear resonance imaging. *Stroke* 1984;15:417–426
9. Rothrock JF, Lyden PD, Hesselink JR, Brown JJ, Healy ME: Brain magnetic resonance imaging in the evaluation of lacunar stroke. *Stroke* 1987;18:781–786
10. Helwig S, Larsson H, Henricksen O, Sorensen PS: Ataxic hemiparesis: Three different locations of lesions studied by MRI. *Neurology* 1988;38:1322–1324

KEY WORDS • ataxia • magnetic resonance imaging