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. 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 Cole 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. 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. Since these observations were made by CT alone, which may fail to demonstrate small nonhemorrhagic infarcts, their exact correlation with AHS has been challenged. Kistler et al 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 glaucoma, 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 was normal. 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
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 capsule, 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


KEY WORDS • ataxia • magnetic resonance imaging
Magnetic resonance imaging of ataxic hemiparesis localized to the corona radiata.
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Stroke. 1989;20:1571-1573
doi: 10.1161/01.STR.20.11.1571

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