

Dysnomia, Ataxia, Choreoathetosis, Sensory Impairment, and Gait Imbalance after Lentiform Nucleus Stroke

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We describe a patient in whom dysnomia, ataxia, choreoathetosis, sensory impairment, and severe gait imbalance resulted from an isolated left lentiform nucleus-posterior limb of internal capsule lacunar stroke. Each of these deficits has been described as a consequence of unilateral basal ganglia or internal capsule infarction in prior reports, but the combination of these findings in one patient is unusual. Accurate localization of the lesion to the anterior circulation has potential therapeutic implications. **Key Words:** Aphasia—ataxia—choreoathetosis—lentiform nucleus—stroke.

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Case Report

A 43-year-old right-handed woman with a history of hypertension abruptly developed dysarthria with weakness of the right face and arm. On examination, she had particular difficulty naming parts of objects and made phonemic errors during spontaneous speech. Her verbal comprehension and ability to repeat were unimpaired. She did not have a visual field deficit. There was right facial weakness in an upper motor neuron pattern and slight weakness of the right hand. There was increased tone in the right arm, with bradykinesia, and prominent ataxia and dysmetria (out of proportion to the weakness). Choreoathetoid movements of the right hand were observed when her arm was outstretched. Appreciation of pinprick was impaired in her right arm and leg but

joint position sensibility was not impaired. Deep tendon reflexes were symmetrically brisk, and plantar responses were flexor. She tilted to the right when standing and was unable to walk unassisted as a result of severe gait instability. She was indifferent towards her postural deficits, and persisted in her attempts at ambulation, despite requiring full assistance.

Brain magnetic resonance imaging showed an area of signal change consistent with acute infarction involving the left lentiform nucleus and the adjacent posterior limb of the internal capsule (Fig 1).

Discussion

Pure motor stroke, pure sensory stroke, ataxic-hemiparesis and clumsy-hand dysarthria are among the common "lacunar" syndromes caused by small deep infarctions.¹ Language deficits, choreoathetosis, and postural instability have each been described as consequences of unilateral basal ganglia or posterior internal capsule infarction,²⁻⁷ but the combination of signs and symptoms exhibited by this patient are unusual.

Lesions of the dominant basal ganglia and adjacent posterior limb of the internal capsule can result in a heterogeneous group of both fluent and non-fluent aphasia, as well as micrographia.^{2,3} Our patient had a language disorder with preservation of her ability to repeat suggesting a sub-cortical aphasia. Pseudochoreoathetosis related to

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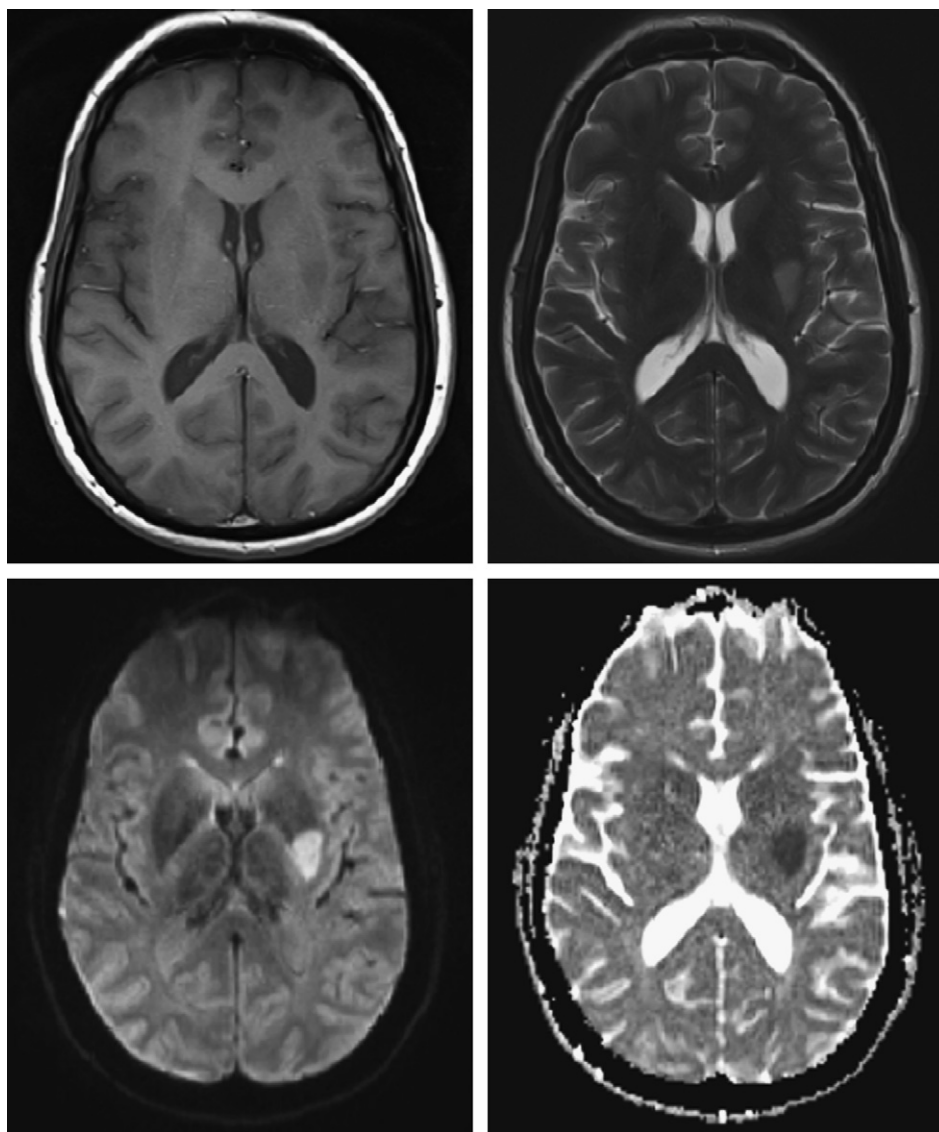


Figure 1. Brain magnetic resonance imaging obtained 1 day after onset of symptoms showing acute infarction involving left lentiform nucleus and adjacent posterior limb of internal capsule (upper left, T1-weighted; upper right, T2-weighted; lower left, diffusion-weighted, and lower right, apparent diffusion coefficient map images).

proprioceptive deficits is a well known sequelae of posterolateral thalamic infarction⁸, however, acute choreoathetosis with preservation of joint position sensation following lesions of the contralateral posterior limb of the internal capsule has also been reported.⁵ In our patient, the movement disorder was restricted to the arm (reported in one case previously)⁶ and resolved within 24 hours. Severe postural deficits following acute unilateral basal ganglia lesions, without significant motor weakness or evidence of cerebellar, vestibular, or posterior column dysfunction, were observed in 9 patients.⁷ As in our case, these patients tended to tilt and had a lack of awareness of the deficit and an absence of a correcting reflex, noted as key features distinguishing their falls from those caused by corticospinal or cerebellar deficits. Our patient's striking gait instability was out of proportion to her mild hemiparesis, and appendicular ataxia.

Anatomic localization of the lesion responsible for our patient's deficits presented a challenge. Prior to neuroimaging, a left thalamic lesion was considered because of the combination of a language disturbance, contralateral ataxia, sensory deficits and inappropriate gait instability that resembled thalamic astasia. Differentiating a lesion affecting the thalamus from one involving the lentiform nucleus can be clinically important. The arterial supply of the ventral thalamus is largely from thalamoperforators originating from the posterior cerebral or posterior communicating arteries, whereas the blood supply to the putamen is from lenticulostriate arteries originating from the middle cerebral artery. A carotid stenosis ipsilateral to a lentiform nucleus infarction could be symptomatic whereas, depending on the configuration of an individual patient's circle of Willis, a similar carotid stenosis on the side of a thalamic

infarction would not be responsible for the patient's symptoms.

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