

Pure Hemiparkinsonism Secondary to Contralateral Lacunar Stroke in the Substantia Nigra

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There are different etiologies of hemiparkinsonism. A few patients with hemiparkinsonism secondary to infarction in the contralateral substantia nigra have been reported in the literature, and only one of these patients presented with pure hemiparkinsonism. This paper reports the unusual case of a patient presenting with pure hemiparkinsonism secondary to a lacunar stroke in the substantia nigra. A 66-year-old man who presented with a 5-year history of left-sided hemiparkinsonism manifested by tremor, rigidity, and bradykinesia located mainly on the upper extremity. Magnetic resonance imaging of the brain revealed a lacunar infarct located in the right substantia nigra. Patients experiencing sudden hemiparkinsonism should be investigated to rule out any abnormality in the contralateral substantia nigra. This case points to the significance of neuroimaging in the evaluation of unilateral parkinsonism. **Key Words:** Hemiparkinsonism—substantia nigra—lacunar stroke—midbrain infarction—nigrostriatal pathway lesion.

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Case Report

A 66-year-old male, who had a medical history of mitral valve replacement 20 years ago, took warfarin. The patient's current condition started 5 years ago; the patient experienced a sudden onset of left arm tremor predominantly in the hand; in addition, the patient noticed a decrease in dexterity on the same hand. At that time, he did not seek medical attention, and therefore, radiological tests were not performed. These symptoms have remained at the same intensity and have not progressed. He denies any symptoms on the left leg or in the right limb. These symptoms do not change with alcohol intake but increase

and are bothersome during stress or emotional states, and for this reason he visited us to have an evaluation.

On examination, the patient was alert and oriented, and his cranial nerves were intact. Rest and postural tremors were observed in the left arm, predominately in the hand; these tremors increased on mental stress (counting backward). Mild cogwheel rigidity was present on the left arm and was exaggerated by active movement of the right hand. Gait was normal except for a mild decrease in arm swing on the left side. There were no motor, cerebellar, or sensory abnormalities in the limbs.

Brain magnetic resonance imaging showed 2 lesions in the midbrain at the level of the inferior colliculus. One lesion was localized in the area corresponding to the right substantia nigra; very likely this lesion was responsible for the patient's symptoms. The other lesion was smaller and was located in the tegmentum. The lesion was hypointense on fluid-attenuated inversion recovery sequence, which was compatible with an old ischemic event (Fig 1, A-D).

The patient considered that his symptoms were not disabling, and for this reason he preferred to wait before taking any treatment.

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Received September 2, 2015; revision received September 29, 2015; accepted October 27, 2015.

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1052-3057/\$ - see front matter

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<http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2015.10.027>

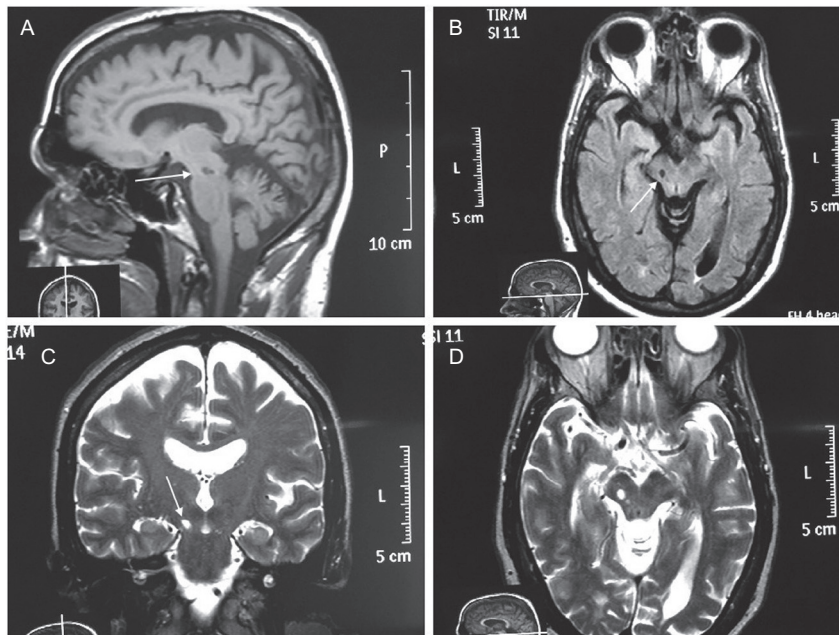


Figure 1. (A) Sagittal MRI T1 sequence; 2 hypointense lesions are observed in the substantia nigra at the level of inferior colliculus (white arrow). (B) Axial MRI fluid-attenuated inversion recovery; the lesion in the midbrain looks hypointense, which is compatible with an old infarct (white arrow). (C) Coronal MRI T2 sequence; the hyperintense lesion is observed in the right midbrain. (D) Axial MRI T2 sequence; 2 hyperintense lesions are observed: the biggest lesion, which is the cause of the clinical picture, is located in the right substantia nigra, and the smaller one is located in the tegmentum. Abbreviation: MRI, magnetic resonance imaging.

Discussion

In the current case, even though functional tests (e.g., DaTSCAN) were not performed to confirm dysfunction of dopaminergic pathways, there is enough information to believe that the lesion observed in the substantia nigra is the cause of the patient's clinical status. The location of the lesion correlates with the clinical information observed in this patient. Moreover, the sudden onset and lack of progress of symptoms strongly suggest that the hemiparkinsonism was caused by the ischemic lesion observed in the midbrain. More information that confirms an infarction in the midbrain is the characteristic of the lesion observed in the magnetic resonance imaging.

Few cases of hemiparkinsonism secondary to infarction in the substantia nigra have been reported in the literature, and usually other neurological signs associated with hemiparkinsonism are observed.¹⁻⁵ The presence of pure hemiparkinsonism due to ischemic stroke is an exceptional phenomenon.

Hemiparkinsonism is not rare in idiopathic Parkinson's disease, especially in early stages. In the current case, the acuteness of onset and lack of progression are

the distinguishing features that suggest a different cause of hemiparkinsonism.

This case suggests that patients experiencing sudden and nonprogressive hemiparkinsonism should be investigated to rule out a stroke in the contralateral nigrostriatal pathways.

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