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LETTER TO THE EDITOR

# Transient ischemic attacks presenting as hemiballismus due to carotid stenosis: Evidence indicating a blood-brain barrier defect in the subthalamic nucleus

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Dear Editor,

Hemichorea-hemiballismus (HCHB), a hyperkinetic movement disorder characterized by involuntary movements on one side of the body, occurs in various conditions including cerebrovascular diseases (1). While persistent involuntary movements caused by ischemic or hemorrhagic stroke are well-documented, transient or continuous involuntary movements have emerged as an atypical presentation of carotid stenosis (2–8). Typically, HCHB results from a lesion in the contralateral basal ganglia, especially the subthalamic nucleus (STN) (1). The etiology of HCHB from ischemic events is not well understood, though it is hypothesized to involve striatal dysfunction due to cerebral hemodynamic alterations or microembolic ischemia (2–5). Numerous reports have indicated that carotid stenosis can lead to hypoperfusion in the basal ganglia and subcortical areas without causing acute ischemic lesions, thus triggering HCHB (2–5). The blood supply to the STN comes from the anterior choroidal arteries, which branch off from the internal carotid arteries or the posterior communicating arteries. Infarction in the STN induces continuous HCHB (1). Here, we present a case of a patient with transient ischemic attacks (TIAs) manifesting as HCHB due to carotid stenosis and contrast enhancement of the contralateral STN on magnetic resonance imaging (MRI).

A 72-year-old woman presented to the neurology outpatient clinic due to involuntary movements, including bending of the right arm, occasional deviation of her mouth to the right, and bending of the right leg. These symptoms, which had been ongoing for about 20 days, completely resolved spontaneously within an hour. There was no altered consciousness or focal neurologic deficits. The attacks occurred during the day and ceased during sleep, with stress and walking acting as provoking factors. The patient had no history of chronic disease or medication use, no family history of involuntary movements or other neurological disorders, and she smoked and consumed three to four glasses of wine daily. Cranial MRI, including diffusion-weighted images (DWI), showed no evidence of acute ischemic stroke. Laboratory tests—including electrolytes, glucose, hormones, liver, and kidney function—revealed no abnormalities during this period. Further investigations and neurology follow-ups were scheduled as she experienced less frequent attacks.

Three weeks after the initial evaluation, the patient presented with dysarthria upon awakening and right-sided weakness. An MRI with contrast agent showed an acute infarction limited to the left internal capsule on DWI and apparent diffusion coefficient (ADC) sequences (Fig. 1), and contrast enhancement in the left STN on axial and coronal T1-weighted

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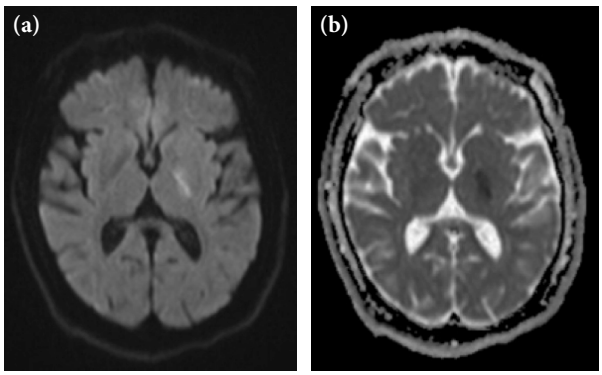


**Figure 1.** Diffusion-weighted imaging **(a)** and apparent diffusion coefficient **(b)** sequences on magnetic resonance imaging reveal an acute infarct within the internal capsule.

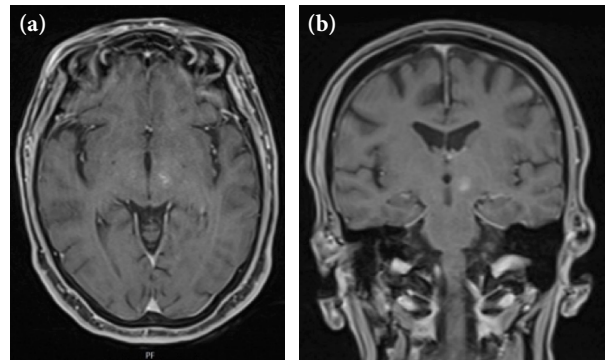


**Figure 2.** Axial **(a)** and coronal **(b)** T1-weighted magnetic resonance imaging sequences show contrast enhancement in the subthalamic nucleus.

sequences (Fig. 2). Intravenous (IV) thrombolytic therapy was initiated after identifying an occlusion at the bifurcation of the left carotid artery by magnetic resonance (MR) angiography. Subsequent digital subtraction angiography demonstrated 90% stenosis of the left internal carotid artery. Carotid balloon angioplasty and endovascular thrombectomy were performed. Due to the need for carotid stenting, the patient was administered dual antiplatelet therapy with acetylsalicylic acid and clopidogrel. During follow-up, carotid stenting was not performed because the stenosis caused by the plaque in the left internal carotid artery was less than 50%, and the antiplatelet therapy was continued. No involuntary movements have been observed since the onset of the stroke and throughout the six-month follow-up period.

First described by Fischer in 1962, limb shaking syndrome is a hemodynamic TIA clinically characterized by brief, dysrhythmic, flailing, or jerking movements of limbs contralateral to an occlusion of the internal carotid artery (9). Neurologists are more familiar with limb-shaking TIAs, which are a rare form and typically occur with a precipitating factor, such as hyperventilation, postural change, or exercise. These factors may compromise cerebral perfusion and must be excluded in the differential diagnosis. Involuntary movements are more commonly associated with metabolic issues and stroke and may be overlooked as TIAs, especially when the patient does not report a postural hemodynamic trigger. We report a case of HCHB as a symptom of TIAs associated with carotid stenosis, which causes a blood-brain barrier (BBB) defect in the STN. There have been a few reports of carotid stenosis causing involuntary movements, including HCHB, without evidence of an MRI lesion (3–5, 8). These involuntary movements have occurred as paroxysmal attacks (2–4, 7) or persistent movements (5, 6, 8). Some patients' symptoms resolved a few months after undergoing revascularization, or even sooner (4–6). While various structural lesions, even those outside the basal ganglia, have been associated with HCHB, the STN and the pallidus subthalamic pathways appear to play a critical role in the expression of this movement disorder (1). The pathophysiology of HCHB caused by carotid stenosis is not fully understood. It is known that the basal ganglia are sensitive to ischemia. Perfusion studies and resolution of such involuntary movements after carotid revascularization suggest that a functional disconnection causes this symptom due to hemodynamic insufficiency rather than a lesion in the basal ganglia (3–6, 8). Given the arterial border zone position of the STN between the anterior and posterior circulation, hemodynamically compromising carotid stenosis can lead to hypoperfusion in this area, which is vulnerable to ischemia and causes a BBB defect. In the present case, the absence of basal ganglia lesions on initial MRI and the resolution of symptoms after a stroke with evidence of a BBB defect in the STN supports this theory. The contrast enhancement in the STN suggests that these attacks were TIAs due to hypoperfusion in the STN caused by carotid stenosis. The ipsilateral pyramidal tract, red nucleus, pallidum, and pallidothalamic pathways must be intact to manifest HCHB (1). Given the clinical course of our patient, it is likely that damage to the pallidothalamic pathways led to the recovery from HCHB. In some patients, the fogging effect phenomenon may be observed, characterized by a change in density from hypodense to isodense in the ischemic region of the brain compared to the surrounding normal brain. This can be detected in the second and third weeks after an ischemic stroke. The fogging effect may result from the invasion of lipid-laden macrophages, proliferation of capillaries, and decreased water mass within the

ischemic area and can lead to underestimation of the true infarct size (10). As in our patient, contrast-enhanced studies should be performed to avoid diagnostic errors related to the fogging effect phenomenon. The patient underwent left carotid balloon angioplasty and endovascular thrombectomy following IV thrombolytic treatment. She had no further symptoms after the stroke. Considering the characteristics of the symptoms, they were not triggered by exertion. Furthermore, the frequency of attacks decreased even when the patient stood for extended periods during the day. The duration of the attacks, the absence of symptoms with maneuvers causing cerebral hypoperfusion, and the clinical course excluded transient ischemic attacks and epileptic seizures causing limb tremors. All cases reported in the literature had vascular risk factors (2–6, 7) and even previous strokes (3, 4). In contrast, our patient had no chronic diseases, but advanced age, smoking, and alcohol consumption were identified as risk factors.

Our report highlights the importance of the STN in the pathophysiology of HCHB and the hemodynamic changes associated with carotid stenosis. Recognizing this unusual form of carotid stenosis is crucial, as early diagnosis and appropriate treatment will reverse cerebral hemodynamic insufficiency and reduce the likelihood of a stroke. This case also underscores the importance of contrast-enhanced brain MRI for the early detection of BBB defects in patients with involuntary movement disorders and vascular risk factors.

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