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Case Report
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A Case Report of Cerebral Infarction in the Basal Ganglia Region with Isolated Manifestation of Behavioral and Cognitive Impairment

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Abstract

Cerebral infarction in the basal ganglia region is a common condition with various clinical syndromes such as hemiplegia and/or hemianesthesia. In this paper, one case of cerebral infarction in the left basal ganglia region with isolated manifestation of acute behavioral and cognitive impairment was reported, in an attempt to help clinicians better understand this type of acute cerebral infarction.

Introduction

Because of the presence of the basal nuclei in the basal ganglia region, including the internal capsule and many nuclei involved in motor functions, clinicians often interpret cerebral infarction in this region simply as "the triad of hemiplegia, hemianesthesia, and hemianopia" or "involuntary limb movements" and then locate it accordingly [1]. However, the non-motor symptoms such as mental and behavioral abnormalities, cognitive decline, atypical aphasia, and hemispatial neglect that can be caused by lesions in this region are not sufficiently emphasized and evaluated. In this report; we aimed to discuss a case of cerebral infarction in the left basal ganglia region with isolated manifestation of acute behavioral and cognitive impairment.

Case Presentation

The patient is an 81-year-old female. She was admitted to the hospital on March 26th, 2020 due to "sudden unresponsiveness with reduced speech for two days". Two days ago, after breakfast, the patient was found by her family to be apathetic and less responsive to the outside world than when waking up in the morning, she showed a significant decrease in speech and lack of words when asked by her family, but no obvious difficulty in understanding language. In the following two days, the patient continued to have these symptoms without relief and often sat for hours without active speech or activity, so she was sent to our hospital by her family. During the course of the disease, the patient had no gibberish, choking and coughing with water, limb weakness, or urinary and fecal incontinence. Personal history: History of hypertension for 3 years, with blood pressure reaching up to 180/110 mmHg. She used to take 2.5 mg of levamlodipine benzoate tablets orally at bedtime for antihypertensive treatment, but blood pressure control was not monitored. Neurological examination: Clear consciousness, apathetic expression, unresponsiveness, reduced speech, relevant but inaccurate answers to questions, MMSE score of 17, MoCA score of 12. Pupils equal, round, and reactive to light, 3 mm in diameter. Normal eye movement in all directions with no nystagmus. Symmetrical nasolabial folds. Angle of the mouth not deviated when the teeth were shown. Normal pharyngeal reflex. Tongue centered when stuck out. Cervical tenderness. Meningeal irritation sign (-). Normal muscle tone and muscle strength of all four limbs. Mild paralysis test in both upper and lower limbs (-). No hemihypalgesia. Bilateral tendon reflexes symmetrical, no pathological signs induced. Lack of coordination in finger-to-nose test and heel-knee-shin test. Difficulty in standing sign with closed eyes (-). Slow pace but normal gait when walking. Auxiliary examination: No abnormalities in routine blood test, electrolytes, liver function, kidney function, blood glucose, blood lipids, blood sedimentation, homocysteine, ultrasensitive C-reactive protein, tumor markers, thyroid function, complete set of autoimmune antibodies, and anti-neutrophil cytoplasmic antibodies. ECG: Sinus rhythm, heart rate of 70 beats/min, normal ECG. Chest CT scan and abdominal ultrasound showed no unusual findings. Lumbar puncture showed intracranial pressure of 160 mmH2O. The routine, biochemical and viral nucleic acid tests of cerebrospinal fluid were normal. Cranial MRI scan showed acute ischemic changes in the lenticular nucleus of

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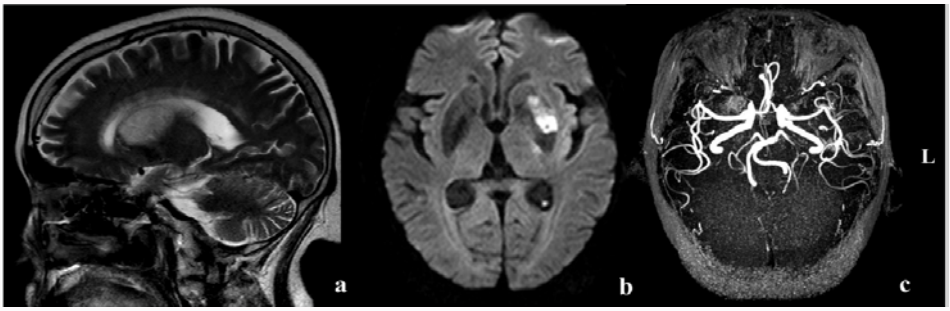

Figure 1: a) Sagittal cranial MRI shows long T2 signal in the lenticular nucleus of the left basal ganglia; b) DWI shows restricted diffusion of the left globus pallidus; c) MRA shows no significant stenosis in the left internal carotid artery system.

the left basal ganglia, and further MRA did not reveal significant stenosis of the internal carotid artery and vertebrobasilar system (Figure 1). Diagnosis: 1. Cerebral infarction (acute stage, in the left basal ganglia region); 2. Hypertensive disease (grade 3, very high risk), with most likely TOAST classification of small-vessel occlusion. After admission, the patient was given antiplatelet, blood pressure monitoring, statin therapy, and cognitive rehabilitation. Two weeks later, the patient's mental status improved, spontaneous speech and active activities increased significantly, and the MMSE and MoCA retests recovered to 24 and 21 points. Ability to independently care for herself in daily life.

Discussion

In clinical practice, mental and behavioral abnormalities and cognitive impairment due to organic focal lesions are often located in the frontal lobe, thalamus, medial temporal lobe, and hippocampus [2]. In the present case, the patient had a sudden onset and a clear previous history of hypertension, so it was easily misdiagnosed as acute cerebrovascular disease in these areas based on her clinical presentation. However, acute cerebral infarction in the left basal ganglia region was later confirmed by cranial MRI.

Previous studies have reported few cases of acute unilateral infarction in the basal ganglia region manifested with behavioral and cognitive abnormalities but completely persevered motor functions, and most cases were acute ischemic lesions in the unilateral lenticular nucleus, predominantly on the left side. Reiji Koide et al. [3] reported a case of infarction of the left globus pallidus with behavioral abnormalities as the only manifestation in 2013. They also suggested that compared with Multi-Infarct Dementia (MID), which is more common in clinical practice, the cognitive decline caused by cerebral infarction of the left globus pallidus belongs to Strategic Infarct Dementia (SID). They also believed that the pathogenesis of such infarcts is mostly penetrating lesions of the middle cerebral artery, with good prognosis. That is, the behavioral and cognitive impairment are often partially or completely recovered after timely intervention. Giroud et al. [4], on the other hand, divided the infarcts in the lenticular nucleus into two different clinical syndromes according to the anatomical regions: Putamen infarction, which is characterized by dystonia and cognitive decline, and globus pallidus infarction, which is characterized by mental and behavioral abnormalities and cognitive impairment. They also concluded that the globus pallidus infarction result in mental and behavioral abnormalities, usually manifesting as apathy and unresponsiveness, whereas the range of cognitive impairment is mainly associated with attention, short-term memory, and executive function. Su Hyun Kim et al. [5] analyzed and explained the behavioral and cognitive impairment alone caused by infarction of the left globus pallidus: Since the function of the cortical projections to different regions of the globus pallidus is relatively independent, the disruption between the frontal cortex and the ventral circuit of the globus pallidus may be the mechanism by which a frontal lobe syndrome is produced. In addition, the frontopontine bundle of the anterior limb of the internal capsule is damaged by compression due to infarction of the anterior part of the globus pallidus, thus affecting frontal lobe function, which has also been speculated to be a mechanism of the condition. In addition to behavioral and cognitive impairment, a case of motor aphasia alone due to the lenticular nucleus infarction has been reported in China. The author suggested that the lenticular nucleus is also directly involved in the regulation and output of speech functions [6]. The present case showed mental symptoms such as apathy and unresponsiveness, reduced spontaneous speech, and normal comprehension after the onset of the disease. The cognitive function assessment showed impaired attention, memory, and executive function, and imaging examination confirmed that the ventral lesions of the left globus pallidus, among other features, were basically consistent with the above report.

In conclusion, the possibility of infarction in the basal ganglia region, especially in the globus pallidus of the dominant hemisphere, should be considered in patients with sudden onset of mental and behavioral abnormalities and cognitive impairment, in order to avoid missing the best time window for treatment. Meanwhile, neuropsychological assessment should also be carefully performed for cerebral infarction in the lenticular nucleus to avoid missing the possible behavioral and cognitive impairment, and timely targeted treatment and rehabilitation should be provided to achieve early return to normal life.

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