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Case Report

Cognitive and behavioral dysfunctions caused by caudate infarction: a case report and literature review

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Abstract: The caudate nucleus is important in the modulation of motor functions and may influence cognitive and mental functions. However, cases of cognitive and behavioral disorders caused by caudate stroke are rare in practice. We report a case of 79-year-old male patient who presented with abrupt onset of cognitive and behavioral dysfunctions. His prominent symptoms included rambling, memory impairment, emotional indifference, reduced words, and lack of care for his family and surroundings. Computed tomography and magnetic resonance imaging of the brain revealed an acute infarction in the head of the left caudate nucleus. The present case and review contribute to the growing body of evidence that the caudate nucleus plays a crucial role in controlling cognitive and behavioral processes.

Keywords: Caudate nucleus, stroke, cognitive impairment, behavioral disorder, computed tomography, magnetic resonance imaging

Introduction

It is generally believed that mental and behavioral disorders can be caused by acquired frontal and temporal lesions. The caudate nucleus is important in the modulation of motor functions. However, the eventual effects of damage to the caudate nucleus on cognitive and behavioral abnormalities are not well known. There is a growing body of evidence indicating that the caudate nucleus plays a crucial role in the control of cognitive and behavioral processes. Here we report a case of a patient with caudate head infarction who presented with cognitive and behavioral abnormalities as prominent clinical features.

Case report

The patient was a 79-year-old right-handed male, retired cadres, with 12 years of education. He was admitted to the hospital reporting a sudden onset of potential cognitive impairments for the past 20 days. Twenty days prior to admission, he noted an abrupt onset of rambling, which was not related to his surrounding environment. Along with impaired intelligence, his faculties of memory and understanding were also affected. He could not remember what he had just done and also could not fully understand the plot of a simple television program. Along with behavioral changes, he experienced emotional indifference, reduced words, and lack of care for his family and surroundings. He felt weak on the right side of his body, and was without fever, head ache, limb tic, or disturbance of consciousness. His previous medical history included hypertension for the past 20 years. He had no history of smoking and reported only occasional alcohol use. The results of physical examination included: blood pressure 120/90 mmHg; awake; cooperative and retarded; fluent speech; fully oriented to person, place, and time; impaired abilities of memory, computation, and judgement; normal cranial nerve function; soft neck; weak right arm and leg with motor power grade V-; without Barbinski and Chaddock signs; and normal sensory and coordination. Laboratory tests results for factors related to cognition or ischemic stroke were all within normal ranges. His Mini-Mental State Examination (MMSE) score was 22/30, and his

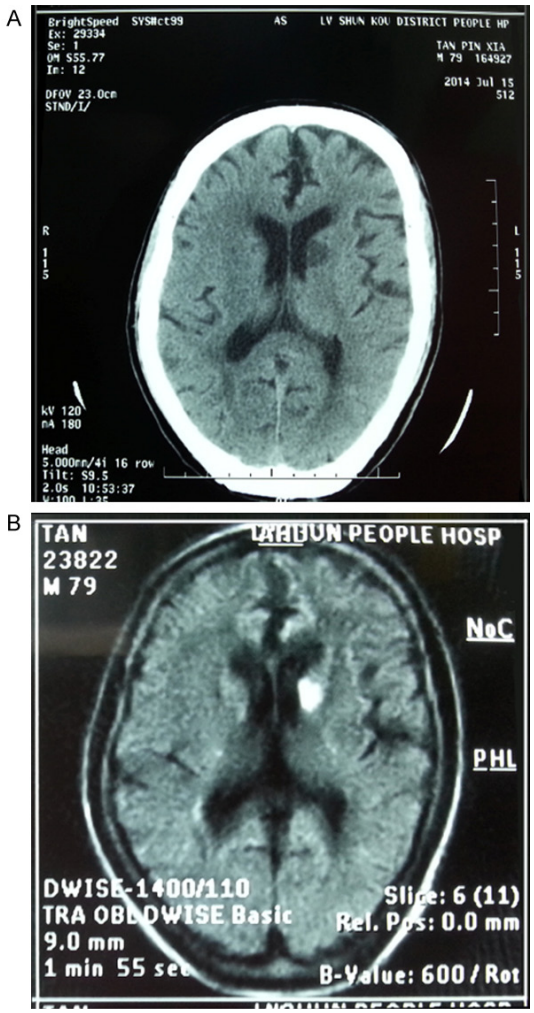

Figure 1. CT and MRI images. An acute infarction was found in the left caudate head, with slight white matter changes. A. Axial CT image. B. Axial diffusion-weighted image.

Montreal Cognitive Assessment (MoCA) score was 19/30. Computed tomography (CT) and magnetic resonance imaging (MRI) scanning were conducted 5 and 6 days after symptom onset, respectively. MRI scans included diffusion weighted imaging (DWI), and magnetic resonance angiography (MRA). Both examinations confirmed that a new infarction was located at the head of the left caudate nucleus (**Figure 1**). MRA showed that the intracranial segment of the left vertebral artery was slimmer than that of the right. Duplex sonography of the carotid and vertebral arteries showed no apparent stenosis or unstable plaques. Electroencephalography (EEG), electrocardiography (ECG), and ultrasound cardiography (UCG) results were normal. Considering these findings altogether, he was diagnosed with an acute caudate infarction. The patient received anti-platelet aggregation therapy, improving cerebrocirculation and cerebrometabolism, as well as lipid regulation therapy. All symptoms improved gradually before he was discharged.

Discussion

Patients with cognitive and behavioral abnormalities as the main presentation can be easily misdiagnosed with an intracranial infection such as encephalitis. Cases of cognitive and behavioral disorders caused by caudate stroke are rare, and thus, may not receive enough attention in practice. The most prominent symptoms of the patient in the present case of caudate infarction were behavioral and cognitive changes. Further investigation of caudate stroke can enrich our understanding of the functions of the caudate nucleus. Thus, the related behavioral and cognitive abnormalities along with the clinical features of caudate vascular lesions are reviewed below. The relevant behavioral and cognitive abnormalities are summarized in **Table 1**.

The most common symptom of caudate nucleus stroke is abulia. Kumral *et al*. [1] reported that one-half of patients with caudate stroke had abulia, which is characterized by decreased spontaneous activity and language, lack of initiative, indifference, psychic akinesia, and prolonged latency in responding to questions and other stimuli. The mechanism of abulia is unclear, and it is probably related to the impairment of the frontal-striatal-thalamic-cortical circuits. Other causes of frontal lobe damage also result in abnormal behavior and abulia, suggesting that the frontal lobe may play a certain role in abnormal behavior and abulia. In addition, some patients with lesions in caudate nucleus have exhibited restlessness, disinhibition, impulsivity, confusion, agitation, and hyperactivity [1, 2]. Another result of a caudate vascular lesion was affective symptoms with psychotic features. Depression was observed relatively frequent in patients with caudate lesions [1], suggesting the caudate nucleus may regulate emotions.

Different degrees of memory impairment are also a common symptom of caudate stroke. The patient in the present case had memory impairment as the first clinical presentation.

Table 1. Cognitive and behavioral abnormalities with caudate infarction

Study (No. of patients)	Left lesion		Right lesion	
	Caplan (18)	Kumral (25)*	Caplan (18)	Kumral (25)*
Behavioral findings				
Abulia	6/10	5/11	4/8	6/11
Confusion		7/11		7/11
Psychic akinesia		2/11		3/11
Restlessness		1/11		2/11
Agitation, hyperactivity	3/10		4/8	
Depression		3/11		2/11
Cognition findings				
Memory impairment	2/10		0/8	
Verbal amnesia		3/11		0/11
Visual amnesia		0/11		4/11
Visual-verbal amnesia		3/11		2/11
Contralateral neglect	0/10	1/11	3/8	6/11
Nonfluent aphasia	1/10	4/11	0/8	1/11
Transcortical motor aphasia	1/10	1/11	0/8	0/11
Global aphasia		1/11		1/11

*3 patients had bilateral caudate infarcts in the study of Kumral *et al*. Caplan (18) (Caplan *et al.* [2]); Kumral (25) (Kumral *et al*. [1]).

Kumral *et al*. [1] reported that one-third of patients with a left caudate lesion had verbal amnesia, while patients with a right caudate lesion had visual amnesia, suggesting the caudate nucleus can integrate verbal and visual memories. Mizuta [3] reported that patients with left caudate infarction induced by occlusion of the recurrent artery of Heubner showed lower scores on both motor procedural and declarative memory tasks, suggesting procedural and declarative memory may be associated with the left caudate nucleus.

Krumral *et al.* [1] also reported that visuospatial and motor neglect were present in one-fourth of patients with a right caudate vascular lesion. Karnath *et al.* [4] investigated 140 patients with right hemisphere stroke (unselected, consecutively admitted over 7 years). Voxel-wise statistical analysis demonstrated that the right superior temporal cortex, the insula and subcortical putamen, and caudate nucleus are typically associated with spatial neglect. These structures form a coherent corticosubcortical anatomical network in the genesis of spatial neglect.

Previous studies have reported that dysarthria was a common symptom in patients with caudate vascular lesions [1, 2]. Krumal *et al.* [1] observed dysarthria without a side predominance in one-third of caudate stroke patients. It is speculated that uniform speech patterns may be caused by the interruption of the corticostriatocerebellar loops and corticolingual pathways, which results in dysarthria.

Kumral *et al.* [1] reported that one-half of patients with a left caudate lesion had minor and transient linguistic deficits, while only one patient had global-type aphasia. Notably, different types of aphasia such as transcortical, non-fluent aphasia, characterized by semantic and verbal paraphasias and perseverations without comprehension impairment, occur in patients with left caudate vascular lesions [1, 2, 5] Gronholm *et al.* [6] reported that more than three-fourths of ischemic stroke patients with language and speech impairment (score of 1-3 on item 9 of the NIHSS) had a subcortical lesion in the left caudate nucleus and the adjacent corona radiate. It is probable that this was the result of acute damage to the fiber tracts connecting the caudate nucleus with frontal and temporal language areas.

In addition to the influence on neuropsychological signs, caudate stroke can also cause hemiparesis and hemichorea. The characteristics of motor deficits were facial and upper extremity weakness and clumsiness. Generally, hemiparesis caused by a caudate nucleus lesion is considered nonpyramidal hemimotor syndrome, which is caused by impairment of the frontopontine fibers running through the anterior limb of the internal capsule.

Notably, there are three major arteries supplying blood to the head of caudate nucleus, which are: the Heubner's artery, anterior lenticulostriate arteries, and lateral lenticulostriate arteries that supply the inferior, anterior, and the major part of the caudate head, respectively. Thus, small-artery diseases could be a main cause of caudate ischemic stroke. Hypertension, diabetes, and hypercholesterolemia are usually associated risk factors. In the present case, MRA showed no obvious big vascular abnormalities, which also supports the aforementioned statement. In addition, clogging of perfo-

rators with atherosclerotic plaque due to large artery disease may be another important cause of caudate infarction. Cardiac embolism is another potential cause of caudate infarction.

In conclusion, caudate nucleus lesions can cause various cognitive and behavioral abnormalities. The present case of caudate head infarction involved cognitive damage and behavioral abnormalities as the main presentations, which is unusual in clinical practice. To enhance diagnostic precision, it should be emphasized that the caudate nucleus may play an important role in influencing cognitive and behavioral processes, in addition to motor regulation.

Disclosure of conflict of interest

None.

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