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Bilateral Caudate Nucleus Infarction Resulting from Left Ventricular Diastolic Dysfunction

Case Report

Asuman Orhan Varoglu

Department of Neurology, Medical School of Ataturk University, 25240 Erzurum, Turkey

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Abstract: Bilateral caudate infarct is a very rare neurological situation, usually caused by small-artery disease resulting from a cardiac embolism. The most prominent clinical features of caudate vascular lesions are behavioral and cognitive abnormalities. We report here a case of bilateral infarction of the caudate nucleus and right parietal lobe with loss of consciousness and left hemiparesia resulting from left ventricular diastolic dysfunction (LVDD). A 50-year-old woman was admitted to our clinic with symptoms of mental status change. One day ago before appearing at our clinic, the patient was hospitalized because of food intoxication and diarrhea. Her neurological examination revealed that orientation-cooperation was impaired, and motor weakness was found in the left extremities, predominantly in the upper limb. The lesions detected by CT and MRI were located on the bilateral caudate nucleus and right parietal lobe. Transthoracic echocardiography revealed LVDD. Therefore, the patient was diagnosed as having had an ischemic stroke. Three days after the treatment, all neurological deficits had improved and the patient was discharged. Attending physicians should be alert to the presence of permanent or intermittent complications of LVDD in patients with ischemic cerebral events, especially in patients with the bilateral caudate nucleus infarcts.

Keywords: *Bilateral caudate nucleus • Left ventricular diastolic dysfunction (LVDD) • Ischemic cerebral event*

1. Introduction

It is well known that caudate nucleus damage is very rare neurological situation. The most common presentations of caudate nucleus lesions are neuro-behavioral and cognitive abnormalities [1]. Caudate nucleus lesions usually involve neighboring structures; depending upon the extent of involvement of these structures, the clinical features of caudate nucleus lesions may be variable [1]. There have been few reports In the literature concerning bilateral caudate infarct [1,2]. The caudate nucleus has an important role in the basal ganglia-thalamo-cortical circuit. This connection is disrupted in bilateral caudate infarcts [3]. The most common cause of bilateral caudate infarct has been reported as cardiac emboli [1].

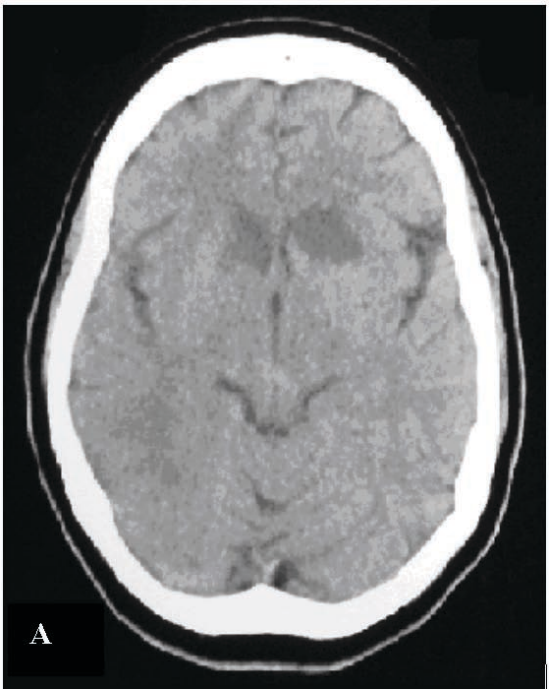
We report here a case of bilateral infarction of the caudate nucleus and right parietal lobe with confusion and left hemiparesia resulting from left ventricular diastolic dysfunction (LVDD).

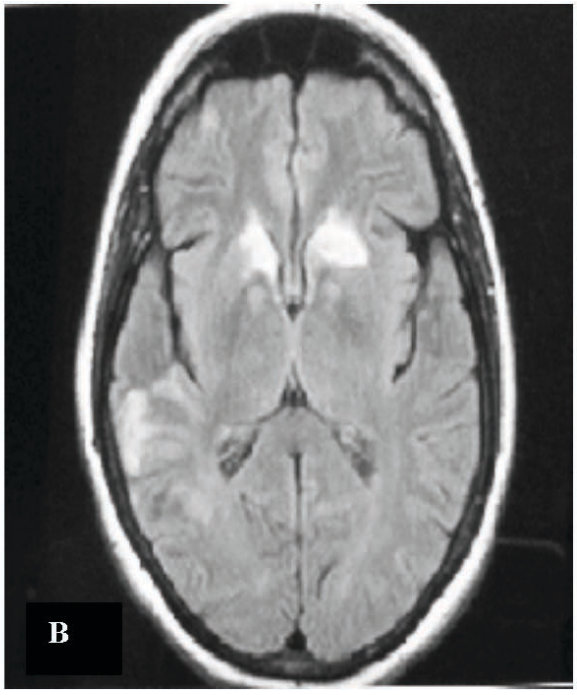
2. Case Report

A 50-year-old woman was admitted to our clinic with sudden impairment of mental status and motor weakness in the left extremities. At admission, her blood pressure was 135/85 mmHg. A neurological examination revealed that orientation-cooperation was impaired, and motor weakness, the Babinski sign, and clumsiness were present in the left extremities, predominantly in the upper limb. Routine laboratory investigations, such as Na, K, P, glucose, blood urine nitrogen (BUN), creatine, plasma triglyceride (TG), low density protein (LDL), and high density lipoprotein (HDL), were within normal limits. The patient's medical history contained no diagnosis of disease at that point. A CT scan showed hypodense lesions at the bilateral caudate and right parietal regions (Figure 1A). In addition, a T2 hyperintense lesion was detected on an MRI (Figure 1B). The diffusion-weighted Magnetic Resonance Imaging (DWI) and the apparent diffusion coefficient (ADC) map investigations supported an infarction in the entire area examined. Her

* *E-mail: asumanorhan@yahoo.com*

Figure 1. A) Brain CT reveals bilateral infarction of the caudate nucleus and right parietal lobe; B) MRI fluid attenuated recovery image reveals hyperintense lesions at whole corresponding areas.





acute confusional state was improved one day after admission.

There were no memory disturbances. The Mini-Mental State Examination score was 27/30. An electroencephalography was evaluated as normal. Carotid Doppler USG findings were also normal. A transthoracic echocardiography showed left ventricular diastolic dysfunction. All hematological marker levels, such as fibrinogen, antithrombin, and proteins C and S, were also found to be normal. Factor V Leiden and prothrombin G20210A gene mutations were not detected in this patient. Therefore, the patient was diagnosed as having suffered an acute ischemic stroke at the bilateral caudate nucleus and right parietal lobe. Three days after initiation of the treatment, the patient's neurological status had improved. Approximately six months later, the present patient was re-evaluated in an outpatient clinic, and the neurological examination was normal.

3. Discussion

Bilateral caudate nucleus damage is an extremely rare neurological condition [1,2]. As reported in previous studies, patients usually exhibit clinical features including behavioral and cognitive abnormalities [1,4]. Behavioral abnormalities result from damage to the basal ganglia-thalamocortical loops. The caudate nucleus is the primary area that bears striatal efferent projections to the cortex, including the frontal, parietal, and temporal lobes [5]. However, neurobehavioral changes were not found in the case presented here.

Confusion is an extremely rare sign, with lesions confined to the caudate nucleus. Kumral et al. reported that acute confusion as a stroke onset sign was observed in a patient with bilateral caudate infarcts [1]. Similarly, acute confusion was found at stroke onset in the present case. The causes of acute confusion might have been both right parietal infarction and bilateral caudate infarctions.

Motor abnormalities are seen in patients with lesions spreading to the anterior limb of the internal capsule. The characteristics of motor deficit are facial and upper extremity weakness, and clumsiness in the acute caudate ischemic events [4]. In our patient, motor weakness was detected in the left extremities, predominantly in the upper limb. Therefore, we assumed that the lesion extended to the anterior limb of internal capsule.

In patients with acute bilateral caudate infarcts, the most common source of emboli is cardiac [1]. In the present case, transthoracic echocardiography was used to evaluate the cardiac source of emboli, and showed left ventricular diastolic dysfunction. Recent reports have revealed that LVDD is a risk factor for ischemic stroke [6,7]. Although the mechanisms of the association between LVDD and ischemic stroke remain unclear,

there are some tentative explanations in the literature. The first explanation suggests that LVDD may be caused by existing latent heart failure and/or silent myocardial ischemia [8]. In a second possible evaluation, Lantelme et al. [7] reported that left ventricular diastolic dysfunction may lead to left atrial dilatation with subsequent risk of atrial fibrillation (AF) and cerebral embolism. Therefore, we speculated that, in our case, LVDD might be a cause of the bilateral caudate infarcts as induced by atrial dilatation or latent heart failure or silent myocardial ischemia.

It is well documented that occult intermittent nondiagnosed AF might also lead to the development of ischemic stroke [9]. In addition, a recent report revealed that transient arrhythmias, and atrial fibrillation in particular, could be involved in the stroke mechanism [10].

In conclusion, physicians should take into account of the presence of transient or permanent left ventricular diastolic dysfunction as the possible source of cardiac emboli in patients with an ischemic cerebral event.

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