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Bilateral caudate nuclei infarctions following the unilateral left anterior cerebral artery occlusion

Eun Ji Chung, M.D., Gi Youn Bae, M.D., Sung ll Sohn, M.D., Hyon Ah Yi, M.D.,
Hyung Lee, M.D., Yong Won Cho, M.D., Jeong Geun Lim, M.D., Sang Doe Yi, M.D.

Department of Neurology, Keimyung University School of Medicine,
Daegu, Korea

Abstract : Bilateral caudate nuclei infarctions are rare patterns of infarction. A 75-year-old female presented with acute abnormal behavior. She had atrial fibrillation. Brain computed tomography (CT) angiography showed non-visualization of the bilateral anterior cerebral arteries (ACAs). After using of intravenous recombinant tissue plasminogen activator (rt-PA), there were high signal intensities of the bilateral caudate nuclei on diffusion weighted magnetic resonance images and recanalization of the left ACA with hypoplasia of the A1 segment of the right ACA on conventional angiography. This case demonstrates the bilateral caudate nuclei infarctions can be induced by a single cardioembolic occlusion of the ACA in a patient with hypoplasia of the contralateral ACA.

Key Words : Bilateral caudate nuclei, Unilateral ACA occlusion

Introduction

The caudate nuclei have four different blood supplies from the anterior cerebral artery and middle cerebral artery [1]. The lateral lenticulostriate artery from the middle cerebral artery (MCA) supply major part of head of the caudate nucleus, anterior limb of the internal capsule, and the putamen. Inferior part of the caudate nucleus is supplied by the Heuber's artery. Anterior part of head of the caudate nucleus is supplied by the anterior lenticulostriate artery branching from the ACA, and the lateral

Corresponding Author: Sung Il Sohn, M.D., Department of Neurology, Keimyung University School of Medicine
216, Dalseongno, Jung-gu, Daegu, 700-712 KOREA
Tel: +82-53-250-7075 E-mail: docsohn@dsmc.or.kr

caudate nucleus is supplied by the medial lenticulostriate artery branching from the proximal MCA.

About 2% of the general populations have only one ACA [2], while perforators from the MCAs are always bilateral. Bilateral caudate nuclei infarctions can be expected in the person who has single ACA supplying caudate nuclei bilaterally.

There are several case reports of bilateral caudate nuclei infarctions [3,4], but incidence is very rare. We report our experience of bilateral caudate nuclei infarctions developed by the occlusion of the A1 segment of the ACA.

Case

A 75 year-old female visited our hospital with the chief complaint of abnormal behavior for 70 minutes. She had history of hypertension and atrial fibrillation. On neurologic examination, consciousness was alert, but she answered barely when we tried to make her speak, which seemed to be a sign of abulia. Speech content and volume was decreased. Weakness of bilateral ankle joints were detected, but no other neurologic abnormalities were seen.

Brain CT angiography showed non visualization of the bilateral ACAs. Under the diagnosis of acute occlusion of the bilateral ACAs, intravenous rt-PA injection was done within 3 hours after symptom onset. Diffusion-weighted magnetic resonance image (MRI) showed high signal intensities of the bilateral caudate nuclei. Magnetic resonance angiography showed absence of the bilateral ACAs. Perfusion defect of the bilateral ACA territory was detected on perfusion weighted image (Fig. 1). We underwent conventional angiography which showed spontaneously recanalized left ACA and hypoplasia of the A1 segment of the right ACA (Fig. 2). On the second day of her admission, initial symptoms were resolved. Follow-up MRI at seventh day still showed bilateral caudate nuclei infarctions, predominantly on left (Fig. 3). Because she complained of easy forgetfulness and losing her way in the hospital, we carried out neuropsychological test on seventh day which reveled impairment of naming in Korean-

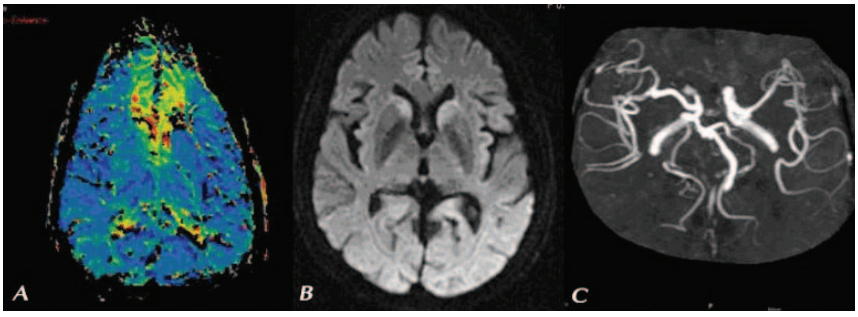

Fig. 1. Brain MRI and MRA of the patient after rt-PA injection. (A) Perfusion weighted image showed perfusion defect of the bilateral ACA territories. (B) Diffusion weighted image showed acute infarctions of the bilateral caudate nuclei. (C) Non-visualization of the bilateral ACAs was seen on magnetic resonance angiography.

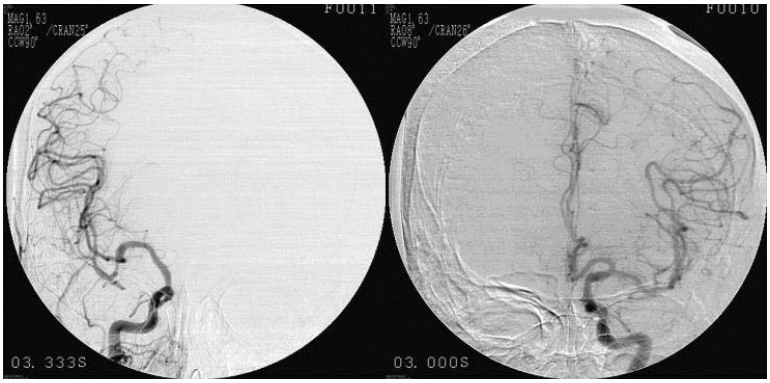

Fig. 2. Conventional angiography showed spontaneously recanalized left ACA and hypoplasia of the A1 segment of the right ACA.

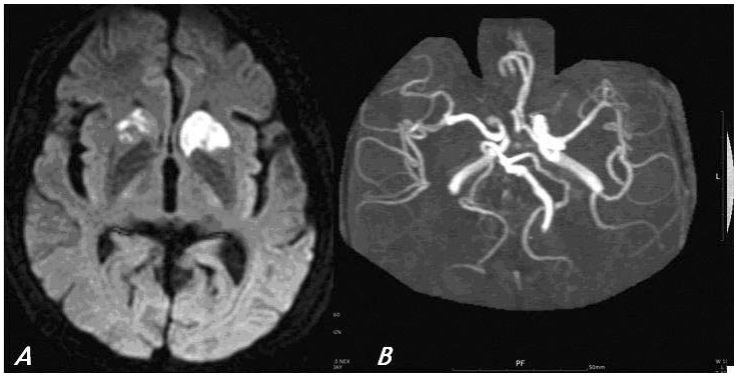

Fig. 3. Follow-up MRI at the 7th admission day. (A) Diffusion weighted MRI still showed the bilateral caudate nuclei infarctions, predominantly on left. And newly developed putaminal infarction on left side. (B) Follow up MRA showed hypoplasia of the A1 segment of the right ACA.

Boston naming test (K-BNT) for language & related function, planning impairment in Rey-Osterrieth complex figure test (RCFT) copy for visuospatial function. 24 points in Korean version of Mini-Mental State Exam (K-MMSE), 1 point in clinical dementia rating (CDR), 4 points in global deterioration scale (GDS) were seen. On the eleventh day, she left hospital while showing no signs of deficit in the neurological examination. Her neuropsychological symptoms passed away within 6 months. On follow up neuro-psychological test, there were improvement in visual recognition, attention and naming, which was in normal range.

Discussion

Isolated bilateral caudate infarctions are rare condition. The bilateral ACA occlusion can cause the bilateral caudate nuclei infarctions. The common etiologies of the bilateral ACA territory infarctions are vasospasm after subarachnoid hemorrhage [5], and cardiogenic embolism, artery to

artery embolism [6], vasculitis accordingly. In the case of patients with unilateral hypoplastic A1 segment, thrombosis of the contralateral ACA or distal ICA can bring about the bilateral ACA infarctions. Besides, there are more chances for embolus to enter the only dominant ACA [5]. A single A1 segment is known as the normal variation, which accounts for 2% of general populations [2]. Our case had hypoplastic A1 segment of the right ACA on conventional angiography and atrial fibrillation on electrocardiogram. Owing to clear recanalization after intravenous rt-PA, it is presumed that the bilateral caudate nuclei infarctions were developed by the cardioembolic occlusion of the A1 segment.

As clinical features of the caudate nucleus infarction, memory decline, abulia and akinesia are frequent neurological abnormalities [7]. Dysarthria, aphasia, confabulations can be another neurobehavior consequences of the caudate nucleus infarction. Particularly, severe and more persistent abulia and global dementia symptoms are possible for the bilateral caudate nuclei infarctions [6]. Abulia and akinesia can be explained by interruption of the limbofrotal connection. And memory deficits are caused by dysfunction of the corticocaudate connections [8]. Weakness develops when a lesion extends into the anterior limb of the internal capsule and the putamen, interrupting the striatopontine fibers. Our case presented that abulia and bilateral ankle weakness which were resolved after reperfusion by IV rt-PA seems to have relation with the limbofrontal connection and the striatopontine fibers.

Summary

We experienced a case of bilateral caudate nuclei infarctions due to the unilateral ACA A1 occlusion. Embolism or large artery thrombosis can be a cause of bilateral caudate nuclei infarctions with patients who have anatomical variation of the A1 segment.

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