Basilar Artery Fenestration Versus Hypoglycemia-Induced Vertebrobasilar Insufficiency: A Case Report

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Abstract

Purpose: Vertebrobasilar insufficiency (VBI) is a common transient neurological condition related to posterior circulation hemodynamic insufficiency. However, it is rarely seen as an initial presentation in basilar artery (BA) fenestration or hypoglycemia. We present this case to further clarify the association between BA fenestration and hypoglycemia-induced VBI, as well as the difficulty in diagnosis, especially in acute clinical settings.

Case Report: Herein, we report a case with BA fenestration, in which the patient suffered from transient episodes of focal neurological deficits, including dysarthria, focal limbs weakness, and ataxia with subsequent total recovery. Apart from hypoglycemia, no other abnormal laboratory surveys were found. This concurrent finding of hypoglycemia with transient focal neurological deficit poses a difficulty in differentiating between hypoglycemia-induced VBI and true acute ischemic stroke in the clinical setting. Subsequent brain imaging studies revealed no evidence of acute infarction and no evidence of atherosclerosis changes in vessels but BA fenestration was observed. We prescribed antiplatelets for the prevention of future strokes. However, currently, no consensus exists regarding the prevention of cerebral ischemia with BA fenestration.

Conclusion: BA fenestration-induced VBI and hypoglycemia-induced VBI are rarely reported and their mechanisms of action remain uncertain and controversial. However, BA fenestration-induced VBI may pose a risk for future cerebral ischemic events and warrants further investigations.

Keywords: basilar artery fenestration; hypoglycemia; vertebrobasilar insufficiency; stroke; ischemia.

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INTRODUCTION

Clarifying the correct pathophysiology of vertebrobasilar insufficiency (VBI) can be quite challenging especially in the presence of "stroke mimics", such as hypoglycemia1. However, different

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pathophysiology lead to different treatment strategies and different disease outcomes. The clinical presentation of VBI can vary greatly2 and brain imaging is usually required to rule out other active intracranial lesions. However, different imaging modalities may lead to different results. We report a case of basilar artery (BA) fenestration presenting with VBI with concurrent hypoglycemia, making diagnosis difficult in the acute emergency setting.

CASE PRESENTATION

A 75-year-old Asian female with a past medical history of type 2 diabetes mellitus visited our emergency department after experiencing a choking episode at breakfast, followed by slurred speech 1.2 hours later. This was preceded by a sudden onset of bilateral lower limb weakness and gait unsteadiness 7 hours before, and the symptoms then spontaneously subsided. Upon arrival, her blood pressure was 187/80 mmHg and her consciousness was clear. Neurological examination revealed dysarthria, mild left limb weakness (4+/5 strength), and bilateral positive Babinski signs. Her blood tests were unremarkable except for hypoglycemia (42 mg/dL). Her previous medication for sugar control included metformin 2000 mg/day, glimepiride 4mg/day, vildagliptin 100 mg/day, and pioglitazone 30 mg/day. A non-contrast head computed tomography (CT) showed no signs of intracranial hemorrhage. Glucose 50% 80 ml intravenous injection and aspirin 300 mg were prescribed. After 30 minutes her blood sugar was 85 mg/dL and her neurological symptoms recovered within hours.

Based on her history and the clinical signs and symptoms of transient brainstem focal neurological deficits, VBI was diagnosed. However, the etiology remains uncertain. Subsequent examination with brain magnetic resonance imaging (MRI) after admission showed no evidence of acute infarction. However, magnetic resonance angiography (MRA) revealed an abnormality of the proximal basilar artery, and an aneurysm was suspected (Figure 1) without obvious atherosclerosis changes to the vessels. A computed tomography angiography (CTA) was arranged, which revealed a BA fenestration at the level of the anterior inferior cerebellar artery (Figure 2). There was neither visible atherosclerosis nor stenosis found in the posterior

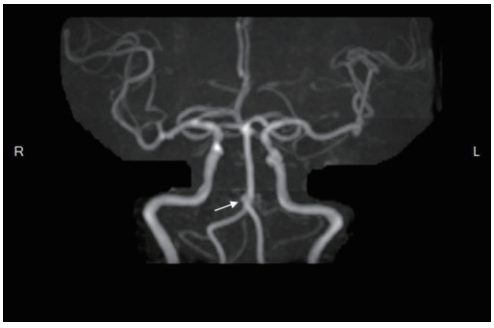

Fig. 1. Brain magnetic resonance angiography: a suspected proximal basilar artery aneurysm (white arrow).

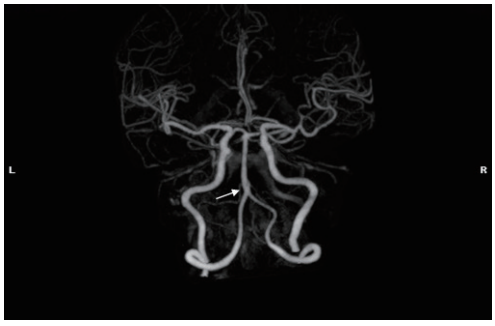

Fig. 2. Brain computed tomography angiography: a fenestration of the proximal basilar artery (white arrow).

Table 1. Transcranial Doppler Sonogram for Vertebrobasilar Blood Flow

	Systolic velocity (cm/s)	Diastolic velocity (cm/s)	Mean velocity (cm/s)	Resistance index
Right vertebral artery	33.0	6.9	16	0.79
Left vertebral artery	64.6	16.7	33	0.74
Basilar artery	58.4	17.0	31	0.71

circulation by both the CTA and a carotid color ultrasound. Low blood flow over the right vertebral artery was noted on a transcranial Doppler examination compared with the left vertebral artery (Table 1). A cardiac source of thromboembolic events was also excluded by an electrocardiogram. We kept the patient on aspirin 100 mg/day. The patient was subsequently discharged without any focal neurological deficits after observation for 3 days.

DISCUSSION

VBI refers to transient neurological deficits caused by hypoperfusion of the cerebral tissue in the posterior circulation3. Common signs and symptoms include dizziness and syncope, dysarthria, headache, nausea or vomiting3, ataxia, imbalance, and weakness in both sides of the body2. VBI is usually caused by hemodynamic insufficiency and embolism. Risks factors that predispose patients to VBI include those that exacerbate atherosclerosis, such as smoking, hypertension, hyperlipidemia, and age2. In our patient, lower blood flow over the right vertebral artery compared with the left vertebral artery was observed on a transcranial Doppler examination, which may be suggestive of hemodynamic insufficiency.

The reported case presented with transient focal neurological deficits with dysarthria, limb weakness, and ataxia. Initial laboratory data revealed hypoglycemia with negative brain CT findings. Hypoglycemia is a well-known "stroke mimic"1 with focal presentations widely reported. The mechanism of cerebral dysfunction causing transient focal neurological deficits in patients with hypoglycemia remains uncertain. Several mechanisms have been postulated, including hypoglycemia-induced focal cerebral hypoperfusion4. Our patient presented with acute onset focal neurological deficits in the absence of vascular occlusion and infarct on follow-up brain MRIs, with lower blood flow over the vertebrobasilar system on transcranial Doppler examination, which may be suggestive of vertebrobasilar insufficiency. The finding of concurrent hypoglycemia provides evidence that hypoglycemia might be another possible cause of the symptoms. Previous studies have reported controversial findings regarding changes in cerebral blood flow during hypoglycemia4. Hypoglycemia-induced hypoperfusion was seen in single-photon emission CT scans in some studies5. We postulate that in our case, hypoglycemia-induced hypoperfusion, in turn induced VBI. However, it is difficult to distinguish hypoglycemia-induced VBI and true acute ischemic stroke within an emergency setting.

Further brain MRI examination revealed no evidence of acute infarction and no evidence of obvious atherosclerosis changes to the vessels were observed on a brain MRA but a suspected proximal BA aneurysm was observed, which was subsequently diagnosed as BA fenestration on a brain CTA. Although catheter angiography remains the gold standard in the workup for diagnosing intracranial vascular lesions, brain imaging such as MRA and CTA are used to detect the degree of cerebral artery stenosis and vascular abnormalities6. The diagnostic value of CTA was in accordance with MRA7. However, in our case, CTA provided better spatial resolution and a BA fenestration was easily identified. The authors suggest conducting a brain CTA examination if the lesion cannot be identified in a previous MRA.

BA fenestration is a rare congenital vascular malformation resulting from incomplete fusion of the paired arteries during embryonic development8. The association between posterior circulation infarction and BA fenestration has been previously reported; however, the mechanism remains uncertain8. BA fenestration presenting with VBI is rarely reported. It is speculated that local hemodynamic and blood flow turbulence caused by BA fenestration might lead to vascular endothelium damage9. This in turn causes hypoperfusion leading to brain ischemia or atherosclerosis causing thrombus formation10. In our case, transient brainstem signs with choking and dysarthria are the typical presentation of VBI. We postulate a possible association between BA fenestration-induced hypoperfusion, which leads to VBI and causes the clinical manifestation of transient focal neurological signs seen in our patient. However, additional studies are necessary to determine the exact association and mechanisms of VBI with BA fenestration.

Distinguishing hypoglycemia-induced VBI and true acute ischemic stroke is important to prevent unnecessary cerebrovascular evaluation and treatment, which can also cause the patient unnecessary emotional distress1. However, it is not always easy to do in clinical practice. We recommend correcting hypoglycemia as well as

prescribing antiplatelets in the first-line emergency setting. We also prescribed the conventional treatment of aspirin as a prophylaxis for stroke.

In conclusion, we report a case of VBI which initially presented with hypoglycemia but was subsequently diagnosed as BA fenestration. In our case, BA fenestration was diagnosed via a brain CTA examination. Currently, the mechanisms of BA fenestration and hypoglycemia-induced VBI remain uncertain and controversial. There is also no consensus regarding the treatment of cerebral ischemia with BA fenestration. However, BA fenestration-induced VBI may pose a risk for future cerebral ischemic events and thus warrants further investigation to differentiate this disease entity from hypoglycemia-induced VBI.

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