Dissection of the Posterior Inferior Cerebellar Artery in A Young Adult with Cerebellar Infarct

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Abstract- The posterior inferior cerebellar artery (PICA) is frequently involved in dissection of the vertebral artery (VA); however, isolated PICA dissection has rarely been reported. A 37-year-old man experienced acute and progressive drowsiness, vertigo, occipital headache, vomiting, and ataxia. There was no precedent trauma or chiropractic manipulation. Neurologically, he had dysmetria of the left extremities. His NIHSS score was 3. Brain magnetic resonance imaging showed an acute cerebellar infarct in the left PICA territory. Magnetic resonance angiography showed a faint signal adjacent to the junction of the left VA and PICA, suggesting a vascular shadow. Catheter angiography showed focal stenosis with a post-stenotic fusiform aneurysmal dilatation of the left proximal PICA that was highly suggestive of dissection with pseudoaneurysm formation. He was treated with clopidogrel and was free of neurological symptoms 3 months after the stroke event. Isolated PICA dissection may be considered in patients with PICA territory infarct or subarachnoid hemorrhage. Treatment depends on the manifestations; ruptured dissecting aneurysms are often treated with surgery or embolization, and infarcts are usually treated with antithrombotic agents.

Key Words: Posterior inferior cerebellar artery, Dissection, Young stroke

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INTRODUCTION

Dissection of the posterior inferior cerebellar artery (PICA) is often extended from the intracranial vertebral artery (VA)[1,2]. Isolated PICA dissection has been rarely reported with variable clinical course and prognosis[1,3]. The clinical presentations of dissection between the isolated PICA and the VA are usually indistinguishable. Dissection of the PICA may lead to infarct, subarachnoid hemorrhage (SAH), or both, depending mainly on the dissecting plane, and the segment of the PICA[1,3,4]. Herein, we describe a case with PICA dissection presenting with cerebellar infarct.

CASE REPORT

A 37-year-old man experienced an acute onset and progressive course of drowsiness, occipital headache, vertigo, vomiting, and ataxia. He had undergone an

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ureterorenoscope lithotripsy for his renal and ureteral stones 1 week prior to the cerebrovascular event at a regional hospital. He had no remarkable medical or familial history. Conventional cardiovascular risk factors, including hypertension, diabetes mellitus, hyperlipidemia, and cardiac diseases, were absent. There was no trauma nor chiropractic manipulation preceding the onset of neurologic symptoms.

On examination, he was afebrile with normal blood pressure and pulse rate. His neck was supple without carotid bruit. The heart sounds were normal. Neurologically, he was oriented but lethargic. Motor and sensory functions of the extremities were intact. Dysmetria of the left extremities was noted, and the National Institute Health Stroke Scale score was 3.

Blood tests for complete blood counts, glucose, lipids, renal, and liver function were unremarkable. Coagulation tests for protein C, protein S, plasminogen,

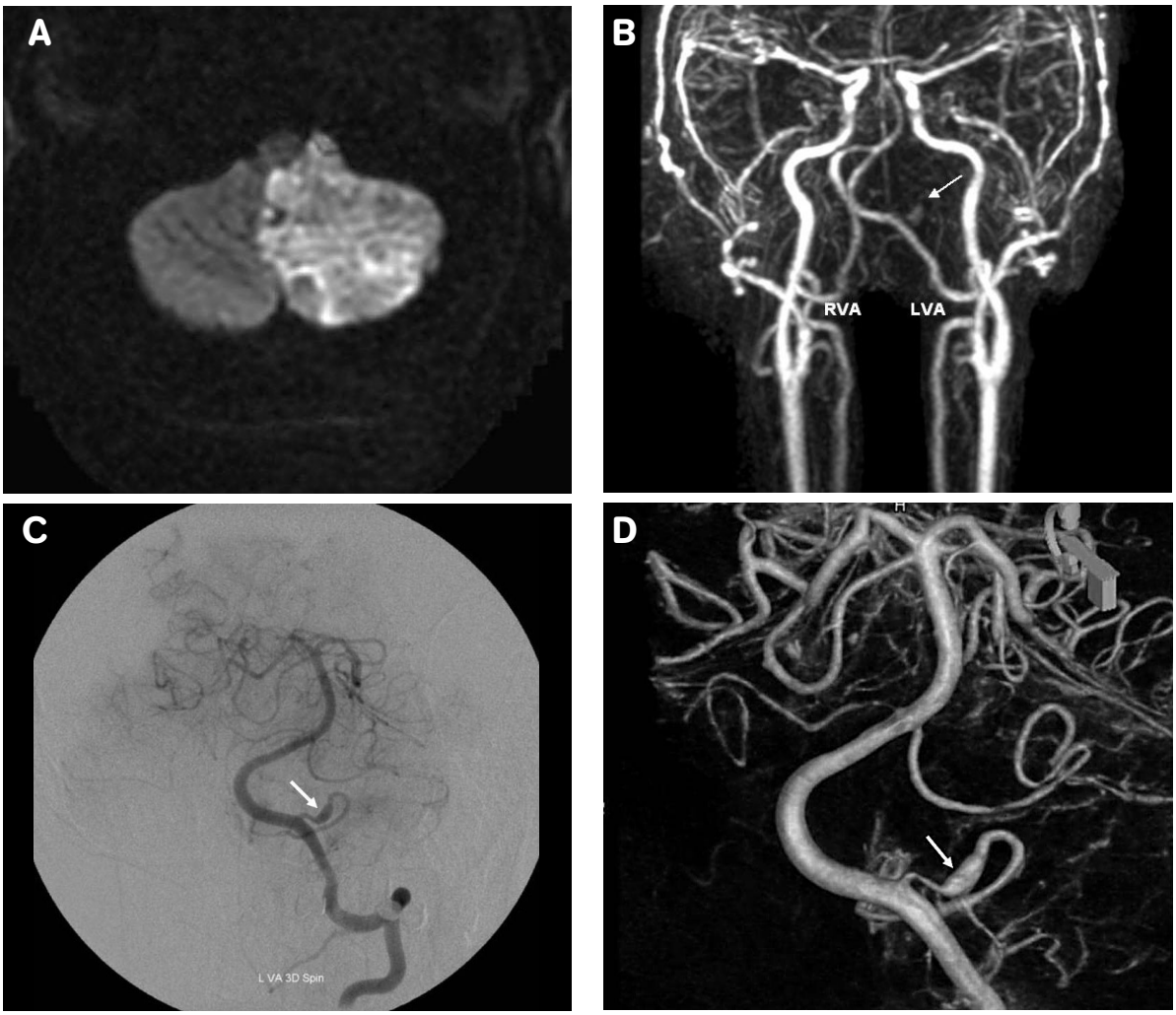

Figure. (A) Diffusion-weighted magnetic resonance (MR) imaging showed an acute cerebellar infarct in the left posterior inferior cerebellar artery (PICA) territory. (B) MR angiography showed a faint signal adjacent to the junction of the left vertebral artery (VA) and the PICA, suggesting a vascular shadow (arrow). (C) Catheter angiography showed a fusiform aneurysmal dilatation of the left proximal PICA. (D) 3D reconstruction angiography clearly demonstrated an isolated left PICA dissection-sparing VA.

and anticardiolipin antibody were within normal limits. C-reactive protein was 0.15 mg/L. Computed tomography (CT) of the brain showed a hypodensity lesion at the left inferior cerebellum with a mild mass effect. Magnetic resonance imaging (MRI) of the brain showed a high signal intensity at the left inferior cerebellum on a diffusion-weighted image that was compatible with an acute cerebellar infarct in the left PICA territory (Fig. A). Magnetic resonance angiography (MRA) showed a faint signal adjacent to the junction of the left VA and PICA, suggesting a vascular shadow (Fig. B). Duplex and transcranial Doppler sonography did not reveal focal stenosis or abnormal hemodynamics of the VA-basilar artery (BA) complex. The transthoracic echocardiography did not find intracardiac thrombi, or valvular problems.

Catheter angiography performed 9 days after the onset of the stroke showed a focal stenosis with a poststenotic fusiform aneurysmal dilatation of the left proximal PICA suggesting dissection with a pseudoaneurysm formation (Fig. C-D). His extracranial and intracranial VAs and BA were normal. He was treated with clopidogrel after admission. He had a near-normal neurological status on discharge, and he was free of neurologic symptoms 3 months after the stroke event.

Follow-up cerebral angiography 3 months after the stroke event revealed no significant changes. Conservative treatment was preferred than operation or endovascular intervention because this PICA was the predominant supply artery.

DISCUSSION

The PICA is the largest, but frequently inconstant one of all cerebellar arteries[5]. These characteristics make clinical presentations and treatments of PICA dissection variable. The first or the anterior medullary segment of the PICA is the most frequently affected site[1], and often results from extension of VA dissection. The VA has a higher incidence of internal elastic lamina defects near the origin of the PICA, which would promote the occurrence and progression of arterial dissection[6]. Furthermore, the intradural artery is more susceptible to rupture than the extradural portion due to its thicker internal elastic lamina, lack of external elastic lamina, thinner adventitia, and fewer elastic fibers in the media[7]. Infarct tends to occur in the proximal PICA dissection, while SAH more in the peripheral ones[1,7].

PICA dissection may lead to infarct or SAH or both, and the clinical presentations are variable and indistinguishable between isolated PICA and intracranial VA dissection. SAH is similar to that of other intracranial ruptured aneurysms. Infarct often presents with lateral medullary syndrome or cerebellar infarct. Isolated PICA dissection accounts for only 2.9% of the lateral medullary infarct[5]. Hemorrhagic events are much more common than infarct in PICA dissection[1,7-9]. The long-term clinical outcome depends mainly on the clinical status on admission[1,8] and the rebleeding event. PICA dissection seems to have a less ominous natural history compared to VA dissection[8].

Catheter angiography still serves as the standard for the diagnosis of posterior circulation dissection. In our case, the diagnosis of PICA dissection was also established by catheter angiography. Catheter angiography should be considered in patients with suspected dissection, even if the VA-BA is normal in MR angiography or sonography. The angiographic pattern of PICA dissection is usually focal stenosis followed by saccular or fusiform dilatation, and there might be pearl and string sign, with retention of contrast medium[1,7,10]. A more pronounced fusiform dilatation with or without a saccular pouch at the site of dissection was regarded as a pseudoaneurysm[7]. Fusiform dilatation with or without pseudoaneurysm is prominent in SAH cases, whereas stenosis without dilatation is dominant in ischemic cases[1,11]. The most common erroneous diagnosis is a ruptured saccular aneurysm of unusual shape with intraluminal thrombosis, associated vasospasm of its parent artery[10].

Treatment of PICA dissection depends on the existence of SAH, the involved segment, the existence of pseudoaneurysm, the rebleeding, the collateral circulation of the PICA, and of course the physical and neurological condition of the patients. The medullary perforators are of much importance, so supratonsillar (cranial)

loop, or the distal third segment of the PICA is served as the landmark for safe distal occlusion of the PICA[1,12,13].

Although the outcome did not differ significantly between endovascular treatment and conservative treatment in SAH group[7], patients presented with SAH usually need a more aggressive treatment. Ruptured dissecting aneurysms should be treated as soon as possible to prevent early re-bleeding[10]. There are still controversial in the timing and treatment plan for the ruptured dissecting aneurysms, and both early and delayed interventions are proposed.

Surgical interventions of the dissecting aneurysms include wrapping, trapping, and resection with reconstruction[14]. Sacrifice or reconstruction of the PICA depends on the dissecting segment, balloon occlusion test[8], and neurological deficits before intervention. Most dissecting aneurysms of the distal PICA are treated by microsurgical trapping[15], and these deconstructive procedures with parent artery occlusion or aneurysm trapping would require bypass such as occipito-PICA anastomosis sometimes[9].

In contrast to surgical intervention, endovascular therapy has gradually become a major therapeutic option in PICA dissection and may be a rational approach in patients at high risk of re-bleeding, especially those with pseudoaneurysms[7]. Endovascular treatment with coils or embolization may give a favorable outcome, but complications with rupture, re-rupture, and ischemic event may occur[8,15]. Intra-aneurysmal embolization of distal PICA with parent artery preservation should be avoided to prevent re-rupture[15]. Besides, the medullary perforators of PICA could not be fully investigated under angiography and could be sacrificed by endovascular treatment[9]. This circumstance could probably be avoided by direct inspection and surgical reconstructive procedure[9].

There is still no consensus about the antithrombotic therapy in patients with intracranial dissections. Although many clinicians used anticoagulant therapy in non-ruptured non-aneurysmal intracranial artery dissections, antiplatelets are usually considered in non-ruptured intracranial artery dissections, including VA or PICA dissection[3,16,17]. However, angiography should be performed and followed-up in ischemic type to search for pseudoaneurysm which may require interventional treatment[1,9]. Evidences are required for better understanding and guiding treatment strategies in these patients.

In conclusion, isolated PICA dissection should be considered in patients with PICA infarct or SAH, especially in patients with young strokes. Treatment depends on clinical manifestations. Ruptured dissecting aneurysms are treated with surgery or embolization, and infarcts are usually treated with antithrombotic agents.

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