

Case Report

Cerebellar hemorrhage after embolization of ruptured vertebral dissecting aneurysm proximal to PICA including parent artery

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Abstract

Background: Some complications related to vertebral artery occlusion by endovascular technique have been reported. However, cerebellar hemorrhage after vertebral artery occlusion in subacute phase is rare. In this report, we describe a patient who showed cerebellar hemorrhage during hypertensive therapy for vasospasm after embolization of a vertebral dissecting aneurysm.

Case Description: A 56-year-old female with a ruptured vertebral dissecting aneurysm proximal to the posterior inferior cerebellar artery developed cerebellar hemorrhage 15 days after embolization of the vertebral artery, including the dissected site. In this patient, the preserved posterior inferior cerebellar artery fed by retrograde blood flow might have been hemodynamically stressed during hypertensive and antiplatelet therapies for subarachnoid hemorrhage, resulting in cerebellar hemorrhage.

Conclusion: Although cerebellar hemorrhage is not prone to occur in the nonacute stage of embolization of the vertebral artery, it should be taken into consideration that cerebellar hemorrhage may occur during hypertensive treatment.

Key Words: Cerebellar hemorrhage, dissecting aneurysm, embolization, posterior inferior cerebellar artery, vertebral artery

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INTRODUCTION

Dissecting aneurysms of the vertebral artery (VA) cause subarachnoid hemorrhage (SAH) or ischemia. For the treatment of a ruptured dissecting aneurysm of VA, several options have been reported.^[6] Recently, endovascular treatment for a dissecting aneurysm has been commonly performed.^[1,5] Some complications related to VA occlusion have been reported.^[3,7,8] In this report, we describe a patient who showed cerebellar hemorrhage during hypertensive and antiplatelet therapies for vasospasm after

embolization of a VA dissecting aneurysm proximal to the posterior inferior cerebellar artery (PICA). We discuss the mechanism of cerebellar hemorrhage and perioperative management of patients with a ruptured VA dissecting aneurysm after occlusion of the parent artery.

CASE REPORT

A 56-year-old female suffered a sudden onset of consciousness disturbance. She was brought to our hospital by ambulance. Computed

tomography (CT) demonstrated SAH and ventricular enlargement [Figure 1a]. Three-dimensional CT angiography (3D-CTA) revealed a dissecting aneurysm of the right VA proximal to PICA [Figure 1b]. On an angiogram, the right VA was dominant and thicker than the left [Figure 2a]. We considered occlusion of the right VA to be a possibility because the left VA was not hypoplastic and slightly thickened. Therefore, endovascular embolization of the aneurysm including VA with preservation of PICA was planned.

Endovascular embolization of the dissecting aneurysm was performed under local anesthesia. A guiding catheter with balloon was advanced to the right VA. A microcatheter (Excelcior SL-10, Stryker, MI, USA) was advanced to the right VA on the distal side of the aneurysm with the aid of microguide-wire (Transend EX, Stryker) via the guiding catheter. The balloon of the guiding catheter was inflated for flow control of VA. A Guglielmi detachable coil (4 mm × 8 cm) was initially placed in the aneurysm and right VA proximal to PICA. Then, the aneurysm cavity was embolized roughly with larger coils. After partial embolization of the ruptured portion of VA, the micro-catheter was pulled and placed in the proximal VA, and the VA was tightly embolized. The dissecting aneurysm and VA were embolized with a total of 12 platinum coils. A postoperative angiogram [Figure 2b] showed complete occlusion of the right VA proximal to PICA including the dissecting aneurysm. Left vertebral angiography (VAG) revealed opacification of the distal right VA and right PICA [Figure 2c]. The aneurysm was not opacified.

Postoperatively, to prevent thrombosis, intravenous administration of heparin was begun after embolization and continued for 7 days. She was treated with fasudil hydrochloride hydrate (90 mg/day) for vasospasm, and was kept in a hypertensive state. Her systolic blood pressure was kept higher than 140 mmHg. The intravenous administration of 80 mg of sodium ozagrel was performed to prevent vasospasm and thrombosis. These treatments for vasospasm and thrombosis was started 2 days after the VA occlusion. Fifteen days after embolization, the patient vomited. CT demonstrated right cerebellar hemorrhage [Figure 3a]. Emergency angiography showed complete occlusion of the aneurysm and no recanalization of the right VA. The right PICA was patent and fed by retrograde blood flow from the contralateral VA [Figure 3b]. Therefore, conservative treatment with strict blood pressure control less than 130 mmHg was performed. Administration of fasudil hydrochloride hydrate and sodium ozagrel was stopped. Her symptoms of nausea and vertigo gradually improved. The patient was transferred to another hospital for rehabilitation with mild cerebellar ataxia and bilateral abducens palsy on the 39th day.

DISCUSSION

The goal of treatment for a ruptured dissecting aneurysm is the isolation of the lesion from the blood circulation to prevent rerupture.^[4] Endovascular occlusion of a dissected site with platinum coils is a safe and reliable treatment for VA dissecting aneurysms.^[3] Although the endovascular technique is an effective treatment, some complications

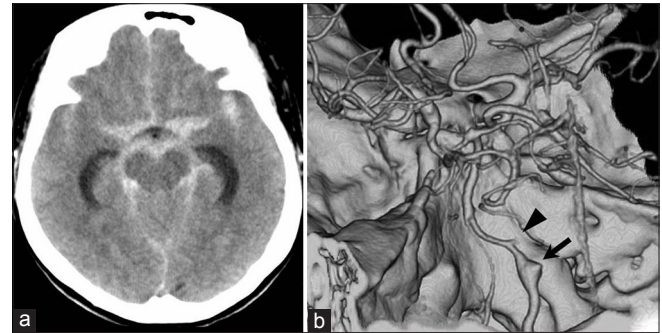


Figure 1: (a) CT on admission showing SAH and ventricular enlargement. (b) 3D-CTA showing a dissecting aneurysm of the right VA (arrow). Origin of PICA is demonstrated (arrowhead). The right VA is thicker than the left

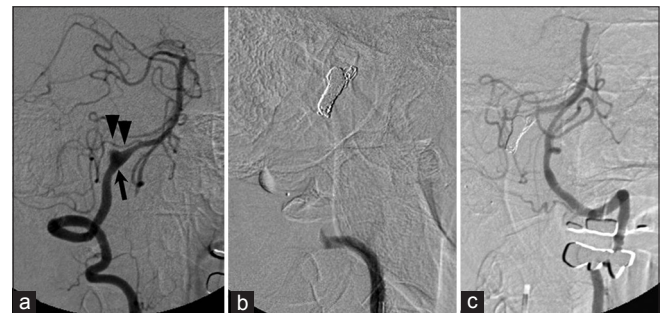


Figure 2: (a) Angiogram showing a dissecting aneurysm (arrow) in the right VA proximal to PICA (arrowheads). (b) Postoperative right VAG demonstrating complete occlusion of the aneurysm and VA. (c) Left VAG showing opacification of the distal right VA and right PICA via blood flow through the VA union

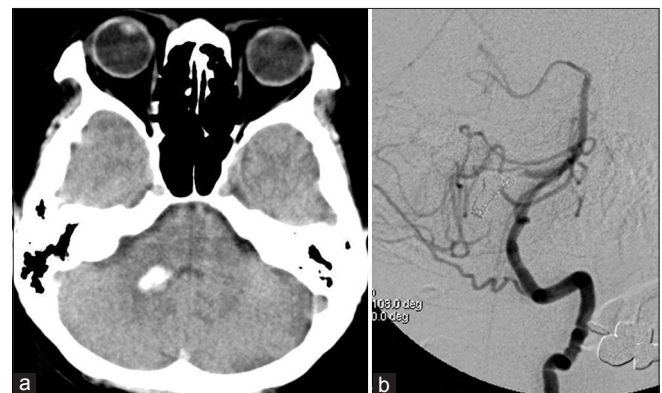


Figure 3: (a) CT obtained on the 15th day after VA occlusion showing hemorrhage in the right cerebellar hemisphere. (b) Angiogram after cerebellar hemorrhage showing no recanalization of the right VA including the dissecting aneurysm and preserved right PICA

have been reported.^[3,7,8] We previously reported a patient with a VA dissecting aneurysm distal to PICA who showed cerebellar hemorrhage 23 h after embolization.^[7] In such a case, blood circulation in the VA system might change after embolization of the aneurysm. The preserved PICA might have been hemodynamically stressed postoperatively, resulting in cerebellar hemorrhage.

In patients with VA dissecting aneurysms who underwent VA occlusion, major perioperative complications were rebleeding from the aneurysm and ischemia.^[8] Concerning hyperperfusion, there was a report of a patient who showed hyperperfusion after percutaneous transluminal angioplasty (PTA) for VA.^[2] In that patient, the hyperperfusion phenomenon resolved within a week. Terada, *et al.*^[9] reported four patients with hemorrhagic complications after PTA or stenting for intracranial arterial stenosis. These patients showed hemorrhage within 3 days.

In the present case, PICA originated distal to the embolization site. PICA was fed by retrograde blood flow from the contralateral VA after embolization of the aneurysm. Hemodynamic stress on PICA fed via VA union might not be so high. Commonly, hemodynamic stress such as hyperperfusion improves within a week.^[2] Although the hemorrhage did not occur immediately after embolization of the affected VA, hemodynamic stress on PICA might have increased due to hypertensive therapy for vasospasm in the present case. Another possibility is a hemorrhagic transformation in an infarction. An infarction might develop due to occlusion of small arteries from VA after VA occlusion. Then the hemorrhage occurred during hypertensive and antiplatelet therapies for vasospasm and thrombosis.

Even in a case such as ours, a rare complication such as cerebellar hemorrhage should be taken into consideration during the postoperative period. Although induced hypertension and antiplatelet therapy are effective for preventing vasospasm after SAH, careful control of the blood pressure is essential in acute and subacute stages after VA occlusion.

REFERENCES

1. Ahn JY, Han IB, Kim TG, Yoon PH, Lee YJ, Lee BH, *et al.* Endovascular treatment of intracranial vertebral artery dissections with stent placement or stent assisted coiling. *AJNR Am J Neuroradiol* 2006;27:1514-20.
2. Bando K, Satoh K, Matsubara S, Nakatani M, Nagahiro S. Hyperperfusion phenomenon after percutaneous transluminal angioplasty for atherosclerotic stenosis of the intracranial vertebral artery. *J Neurosurg* 2001;94:826-30.
3. Kai Y, Hamada J, Morioka M, Todaka T, Mizuno T, Ushio Y. Treatment of dissecting vertebral aneurysm. *Interv Neuroradiol* 2001;7:155-60.
4. Kitanaka C, Morimoto T, Sasaki T, Takakura K. Rebleeding from vertebral artery dissection after proximal clipping. *J Neurosurg* 1992;77:466-8.
5. Lee JM, Kim TS, Joo SP, Yoon W, Choi HY. Endovascular treatment of ruptured dissecting aneurysms -long-term follow-up results, benefits of early embolization, and predictors of outcome. *Acta Neurochir (Wien)* 2010;152:1455-65.
6. Mizutani T, Aruga T, Kirino T, Miki Y, Saito I, Tsuchida T. Recurrent subarachnoid hemorrhage from untreated ruptured vertebrobasilar dissecting aneurysms. *Neurosurgery* 1995;36:905-13.
7. Nomura M, Kida S, Kita D, Kaneko T, Higashi R, Yoshikawa J, *et al.* Cerebellar hemorrhage after embolization for a ruptured vertebral dissecting aneurysm. *Surg Neurol* 2000;53:239-42.
8. Taha MH, Sakaida H, Asakura F, Maeda M, Toma N, Yamamoto A, *et al.* Endovascular management of vertebral artery dissecting aneurysms: Review of 25 patients. *Turk Neurosurg* 2010;20:126-35.
9. Terada T, Tsuura M, Matsumoto H, Masuo O, Tsumoto T, Yamaga H, *et al.* Hemorrhagic complications after endovascular therapy for atherosclerotic intracranial arterial stenoses. *Neurosurgery* 2006;59:310-8.