

De Novo Vertebral Artery Dissection after Endovascular Trapping for Ruptured Dissecting Internal Carotid Artery Aneurysm: Case Report

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Abstract

The authors present an extremely rare case of a 54-year-old female patient with subarachnoid hemorrhage due to a rupture of a dissecting internal carotid artery (ICA) aneurysm, who developed *de novo* vertebral artery dissection in the spasm period after endovascular trapping of the ICA. Interestingly, postoperative cardiopulmonary monitoring showed high global end-diastolic volume index and mean arterial pressure, which could contribute to this *de novo* dissection via hemodynamic stress in the cerebral circulation. Spontaneous intracranial artery dissection of more than two arteries is rare, and we believe this is the first case of *de novo* dissection occurring on a circulating vessel different from that of the initial dissection. The clinical implications are discussed in relation to postoperative hemodynamic stress with a review of the literature.

Key words: subarachnoid hemorrhage, dissection, *de novo*, endovascular trapping, hemodynamic stress

Introduction

Spontaneous intracranial artery dissection is recognized as a cause of focal neurological deficits due to ischemia or subarachnoid hemorrhage (SAH). In general, surgical or endovascular intervention should be performed to prevent recurrent hemorrhage in patients with SAH, and occlusion of the affected vessel with or without bypass is recommended.^{1,2)} Interestingly, there have been several case reports showing contralateral vertebral artery (VA) dissection after the treatment of the ruptured VA dissection.^{3–6)} We herein present the case of *de novo* intracranial VA dissection after endovascular trapping for the ruptured internal carotid artery (ICA) dissection, and the clinical implications are discussed in relation to postoperative hemodynamic stress with a literature review.

Case Report

A 54-year-old female patient was referred to our neurosurgery department for sudden onset of severe headache and seizure. Emergent computed tomography (CT) demonstrated massive SAH mainly in the right basal cistern with an

intracerebral hematoma, and CT angiography detected an intimal flap at the supraclinoid portion of the right ICA (Fig. 1A). Digital subtraction angiography (DSA) clearly detected a fusiform-shaped dissecting aneurysm at the ophthalmic segment of the ICA involving the posterior communicating artery (Fig. 1B, C). She had no episode of head trauma or predisposing causes for the dissection including collagen disease or fibromuscular dysplasia. The patient underwent emergent endovascular trapping of the right ICA without bypass under general anesthesia (Fig. 1D). The collateral supply was well developed through the anterior communicating artery, and there was no change in intraoperative monitoring of regional oxygen saturation of the brain.

The patient underwent triple-H therapy (hypervolemia, hypertension, and hemodilution) to prevent delayed cerebral ischemia following vasospasm. Moreover, postoperative hemodynamic monitoring was performed for the assessment of volume management using PiCCO plus (Pulsion Medical Systems, Munich, Germany).⁷⁾ Surprisingly, follow-up MR imaging and DSA 7 days after the intervention showed segmental narrowing of the left VA with an intramural hematoma, which suggested that the intracranial VA dissection occurred postoperatively (Fig. 2). PiCCO monitoring clearly showed that global end-diastolic

volume index (normal range, 680–800 ml/m²) and mean arterial pressure, indicators of preload, were high throughout the postoperative period (Fig. 3). Therefore, we speculated that postoperative hemodynamic stress could have contributed to the *de novo* VA dissection. Fortunately,

this *de novo* dissection was asymptomatic, and the patient was treated conservatively under strict control of blood pressure, achieving spontaneous healing of the dissection on MRI 3 months later.

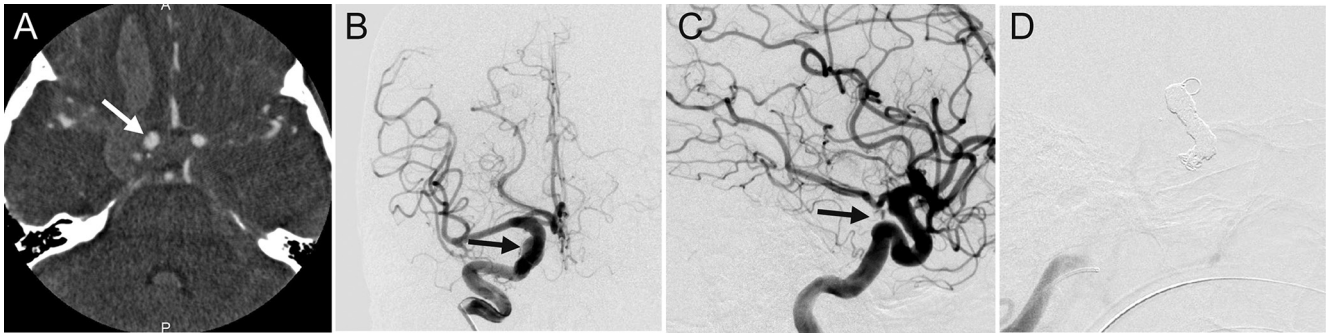


Fig. 1 Computed tomography angiography showing an intimal flap in the right internal carotid artery (ICA, arrow in A). Digital subtraction angiography showing a dissecting aneurysm with a double lumen (arrow in B) and extravasation of the contrast media at the ophthalmic segment of the right ICA (arrow in C). Postoperative angiography showing complete obliteration of the ICA with detachable coils (D).

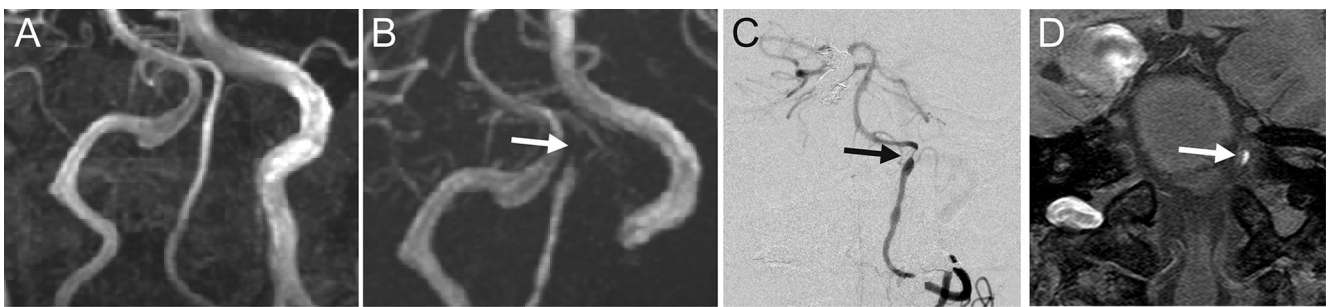


Fig. 2 Initial magnetic resonance (MR) angiography showing no abnormal findings on the left vertebral artery before the intervention (A), but segmental stenosis detected 1 week after endovascular trapping of the ICA (arrow in B and C). MR imaging showing an intramural hematoma on T₁-weighted image (arrow in D). ICA: internal carotid artery.

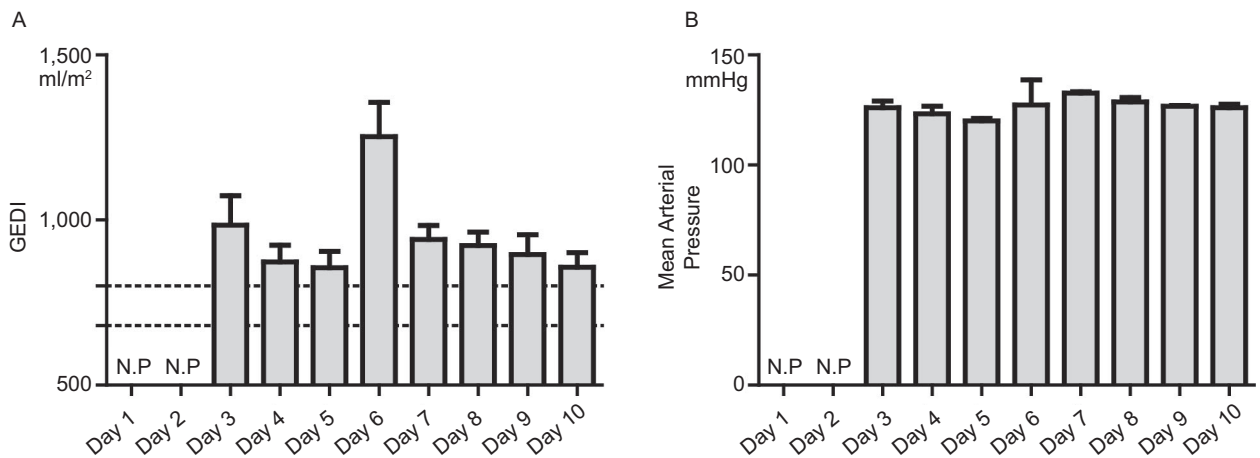


Fig. 3 Postoperative hemodynamic monitoring using PiCCO (Pulsion Medical Systems, Munich, Germany). Global end-diastolic volume index (A: GEDI; normal range, 680–800 ml/m²) and mean arterial pressure (B) as indicators of preload are high throughout the postoperative period. N.P.: not performed.

Discussion

Arterial dissection is a pathologic process characterized by splitting of the media or subadventitial layer of the arterial wall, which leads to formation of an aneurysm with or without relevant narrowing of the arterial lumen. It is well known that spontaneous intracranial or cervical artery dissection may occur, resulting in cerebral ischemia or SAH. Although several mechanisms including genetic and environmental factors have been suggested for the development of spontaneous artery dissection, the cause is unclear in most cases.^{8,9)}

Only five cases of *de novo* intracranial arterial dissection have been reported in the English literature (Table 1),³⁻⁶⁾ and all showed contralateral VA dissection after intervention for the affected VA dissection. Among them, four patients initially developed SAH and were treated with proximal occlusion or trapping of the affected VA. The interval from the initial dissection to onset of the *de novo* dissection was reportedly 8 hours to 13 months (median, 3 weeks), and conservative therapy was performed in four cases probably because of the inability to trap the remaining VA. The clinical outcome varied depending on severity (symptomatic or asymptomatic). Some previous papers also reported similar cases of bilateral VA dissection presenting with different time courses, but details were not described.^{10,11)} Moreover, surgical intervention to treat dissecting aneurysms of the bilateral VAs on one side reportedly carries the risk of rupture of the contralateral lesion.⁶⁾ Therefore, increased hemodynamic stress could be important in the development of *de novo* dissections in the contralateral VA. The actual incidence of *de novo* dissection on the contralateral VA remains unclear, but could be more frequent than previously reported because many patients with intracranial vertebrobasilar artery dissection are asymptomatic or have only minor symptoms.¹¹⁾

This is the first case of *de novo* intracranial dissection occurring on a circulating vessel different from that of the initial dissection. We also found that systemic hemody-

amic parameters determined with PiCCO were high in the postoperative period. Although it is uncertain why *de novo* dissections preferably occur in the VA under these conditions, we believe that postoperative aggressive volume loading for vasospasm as well as hemodynamic changes in the cerebral circulation contributed to this *de novo* VA dissection. Sagoh et al. reported an interesting case showing a ruptured VA dissecting aneurysm associated with occlusive ICA dissection at the same time.¹²⁾ Although the correlation of these two dissecting vessels was not described in their report, initial ICA occlusion might have contributed to the rupture of the VA dissection, which supports our hypothesis. Moreover, it is reported that multiple arteries can be involved in patients with spontaneous intracranial or cervical arterial dissections, and the reported incidence of spontaneous multi-vessel dissection is 10% to 15%.^{8,13)} These reports raise a possibility that initial dissection could contribute to another dissection, and further accumulation of cases is needed.

Recent Japanese surveys of spontaneous cervicocephalic arterial dissection (SCAD-1, SCADS-2) have suggested that in Japan, intracranial VA dissections occur more frequently.¹⁴⁾ This is completely different from findings in American populations showing that cervical ICA dissections occur more frequently; the reason for this difference has not been clarified.¹⁵⁾ Actually, all case reports of *de novo* VA dissection are Japanese articles, and genetic or environmental factors might be involved in the mechanism of *de novo* VA dissection in addition to the hemodynamic stress after the initial insult. Taken together, careful follow-up might be necessary focusing on *de novo* dissections after the initial dissection.

Conclusion

It is important to consider the risk of *de novo* dissection following SAH due to intracranial vessel dissection. Genetic or environmental factors might be involved in the mechanism of *de novo* VA dissection in addition to the

Table 1 Reported cases showing *de novo* dissection after intervention for the initial dissection

Case no.	Author	Age, Sex	Initial dissection	Initial onset	Initial treatment	<i>De novo</i> dissection	Interval	2nd onset	2nd treatment	Outcome
1.	Kubo et al. ⁵⁾	49,F	Left VA	SAH	Proximal occlusion	Right VA	3W	Asympt	Proximal occlusion	GR
2.	Otawara et al. ⁶⁾	51,F	Right VA	SAH	Surgical trapping	Left VA	1Mo	Asympt	Conserve	GR
3.	Inui et al. ³⁾	36,M	Left VA	Infarct	Conservative	Right VA	13Mo	Infarct	Conserve	D
4.	Inui et al. ³⁾	45,M	Left VA	SAH	Endovascular trapping	Right VA	2W	Infarct	Conserve	SD
5.	Katsuno et al. ⁴⁾	39,M	Left VA	SAH	Surgical trapping	Right VA	8H	SAH	Conserve	D
6.	Present	54,F	Right ICA	SAH	Endovascular trapping	Left VA	1W	Asympt	Conserve	GR

Asympt: asymptomatic, Conserve: conservative, D: dead, F: female, GR: good recovery, H: hour, ICA: internal carotid artery, Infarct: infarction, M: male, Mo: month, SAH: subarachnoid hemorrhage, SD: severely disabled, VA: vertebral artery, W: week.

hemodynamic stress after the initial insult.

Conflicts of Interest Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices in the article. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

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