

# **Anterior Spinal Artery as a Collateral Channel in Patients With Acute Bilateral Vertebral Artery Occlusions**

## **—Two Case Reports—**

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### **Abstract**

Retrograde flow through the anterior spinal artery (ASA) from the cervical vertebral artery (VA) to the intracranial distal VA due to disrupted perfusion caused by bilateral VA occlusion is rare. We report two cases of hemodynamic vertebrobasilar circulatory insufficiency caused by bilateral VA occlusion. In these patients, the ASA filled in the retrograde direction, and provided collateral support to the ipsilateral posterior inferior cerebellar artery. The patients were treated with drip intravenous infusion of edaravone and/or argatroban. One patient had a good collateral supply from the posterior communicating artery and recovered almost completely within one month, but the other did not and lapsed into a coma, with generalized hyperreflexia, pin-point pupils, and ataxic respiration. Severe calcified lesions on three-dimensional computed tomography angiography at the occlusion site in the second patient indicated direct surgery including right superficial temporal artery to superior cerebellar artery anastomosis, rather than the endovascular approach. Retrograde flow through the ASA may be observed in this type of critical situation, and may be an important source of collateral supply to the posterior fossa territory.

Key words: anterior spinal artery, hemodynamic vertebrobasilar circulatory insufficiency, collateral channels, angioplasty, ischemia

### **Introduction**

Proximal vertebral artery (VA) occlusion is usually compensated by anastomotic flow to the upper part of the artery via the deep cervical, thyrocervical, occipital, and ascending pharyngeal arteries or reflux from the circle of Willis via the posterior communicating artery.<sup>3)</sup> Occlusion or severe stenosis of the unilateral VA tends to be asymptomatic if there is sufficient flow through the contralateral VA.<sup>7)</sup> On the other hand, patients with bilateral distal VA occlusions are not generally supposed to have such compensatory collateral channels, so occlusion in these patients may subsequently lead to the anterior spinal artery (ASA) syndrome.<sup>11,18)</sup> However, the ASA may conversely be essential in supplying blood to the posterior circulation system under such severe conditions.<sup>5)</sup>

We report two cases of retrograde flow through the ASA from the cervical VA to the intracranial distal VA because of disrupted perfusion from the

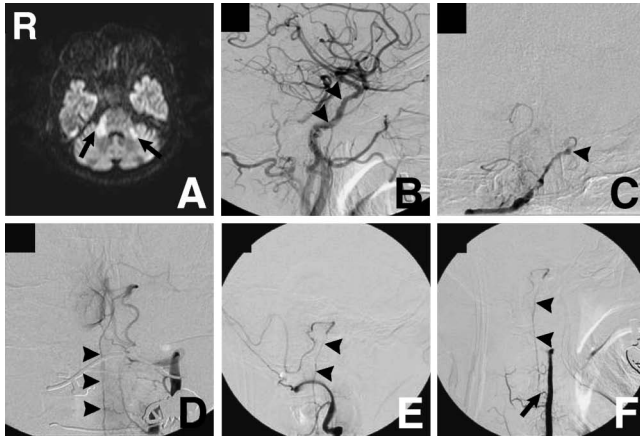
bilateral VAs.

### **Case Reports**

**Case 1:** A 76-year-old man with a 10-year history of diabetes and hypertension was transferred to our hospital on August 16, 2006, with a diagnosis of cerebellar infarction. No neurological abnormalities were noted except for mild dysarthria. Diffusion-weighted magnetic resonance (MR) imaging revealed hyperintensity spots on the bilateral middle cerebellar peduncles (Fig. 1A). Left common carotid angiography revealed contrast filling of the mid-basilar artery from the posterior communicating artery (Fig. 1B). Vertebral angiography revealed complete occlusion at the intracranial level just before the posterior inferior cerebellar artery (PICA) orifice on the right (Fig. 1C), and at the extracranial level on the left, with reversal of blood flow through the ASA arising from the left radicular artery (Fig. 1D-F). The patient was treated for 14 days with drip in-

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**Fig. 1** Case 1. A: Diffusion-weighted magnetic resonance image of the middle brainstem and cerebellum showing lateral hyperintensity spots on both middle cerebellar peduncles (arrows). B: Left common carotid angiogram, lateral view, showing contrast filling of the mid-basilar artery from the posterior communicating artery (arrowheads). C: Right vertebral angiogram, anteroposterior (AP) view, showing the occluded vertebral artery just before the posterior inferior cerebellar artery orifice (arrowhead). D, E: Left vertebral angiograms, AP (D) and lateral (E) views, showing the anterior spinal artery (arrowheads) filled in the retrograde direction, and providing collateral support to the ipsilateral posterior inferior cerebellar artery. F: Left vertebral angiogram, lateral view, at the cervical level showing the anterior spinal artery (arrowheads), arising from the radicular artery (arrow), and ascending the ventral aspect of the cord as a collateral flow to the basilar artery.

travenous infusion of edaravone and 7 days of argatroban. He recovered almost completely within one month.

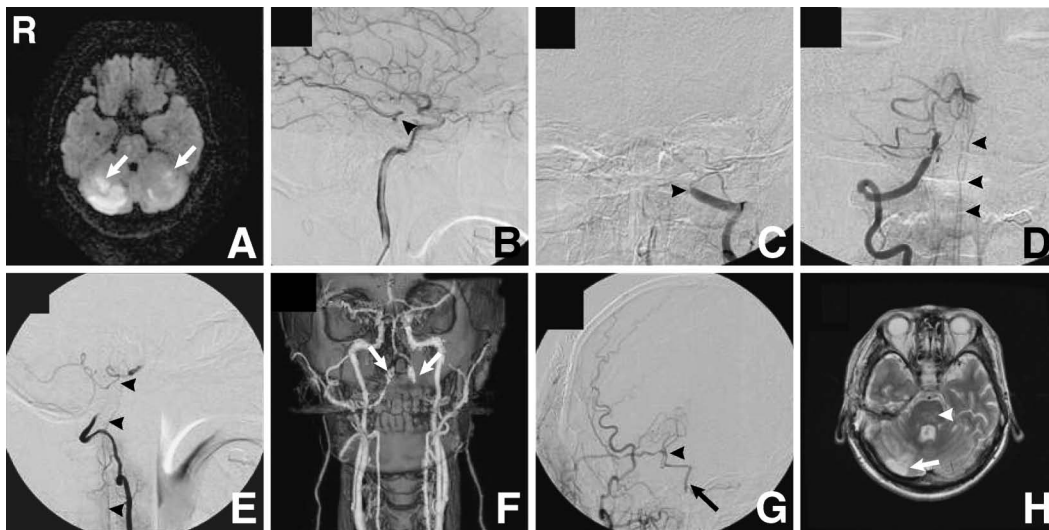
**Case 2:** A 48-year-old man developed vertigo, nausea, and vomiting on September 10, 2007. On admission, his neurological state was scored as 14 points on the Glasgow Coma Scale (E-3, V-5, M-6) with right hemiparesis. Diffusion-weighted MR imaging revealed multiple hyperintensity spots in the bilateral cerebellar hemispheres (Fig. 2A). Left common carotid angiography revealed exclusive contrast filling of the top of the basilar artery (Fig. 2B). Vertebral angiography revealed complete occlusion at the extracranial level on the left (Fig. 2C), and at the intracranial level just before the PICA orifice on the right, with reversal of blood flow through the

ASA (Fig. 2D, E). The patient was immediately treated with drip intravenous infusion of argatroban and low molecular weight dextran. Over the next 6 hours he lapsed into a coma, scored as 7 points on the Glasgow Coma Scale (E-1, V-2, M-4), with generalized hyperreflexia, pin-point pupils, and ataxic respiration. The absence of evidence of hydrocephalus on MR imaging led us to assume that his neurological deterioration was paralleled by the progressive hypoperfusion to the brainstem, although his neurological state was refractory to the induced hypertension caused by dopamine administration. Three-dimensional computed tomography angiography showed severe calcified lesions at the occlusion site of both VAs (Fig. 2F), so we performed direct angioplastic surgery including right superficial temporal artery (STA) to superior cerebellar artery anastomosis, rather than the endovascular approach. Follow-up angiography 2 weeks later showed abundant contrast filling to the basilar artery via the anastomotic STA (Fig. 2G) as well as reduced reversal flow of the ASA. Postoperative MR imaging obtained 1 month later showed small infarctions in the brainstem and right cerebellar hemisphere (Fig. 2H). The patient became almost self-ambulant 3 months later.

## Discussion

The ASA arises from the distal VA, courses medially to unite with the contralateral counterpart, and then courses inferiorly to supply the central part of the anterior two-thirds of the spinal cord. The mean outer diameter of the ASA is 0.7–0.8 mm.<sup>1)</sup> Antegrade flow through the ASA has been demonstrated angiographically in 3 of 200 cases,<sup>20)</sup> 24%,<sup>2)</sup> or 51% of the cases examined,<sup>17)</sup> but usually requires more sophisticated techniques and facilities.<sup>2)</sup> The ASA is very long and has numerous segmental connections at multiple levels, so is an important potential pathway for collateral arterial flow.<sup>4)</sup> In such cases, pathological reversals of blood flow direction in the spinal cord arteries occur under peculiar conditions.<sup>4,5,7)</sup> One of the most common reversals of flow takes place after the occlusion of both VAs before/after their penetration into the endocranium. A total of 12 such cases have been reported including our cases (Table 1).<sup>8,9,13,14,21)</sup> The patients were 7 males and 5 females aged from 28 to 81 years (mean 60.9 years). The treatment was conservative in 6 cases, endovascular angioplasty in 4 cases, and direct surgery in one case.

The interruptions in blood supply created by VA occlusions and the absence of collateral circulation through the posterior communicating artery may



**Fig. 2** Case 2. **A:** Diffusion-weighted magnetic resonance (MR) image of the middle brainstem and cerebellum showing multiple hyperintensity spots on both cerebellar hemispheres (arrows). **B:** Left common carotid angiogram, lateral view, showing contrast filling of the bilateral posterior cerebral arteries from the posterior communicating artery (arrowhead). **C:** Left vertebral angiogram, anteroposterior (AP) view, showing the occluded vertebral artery at the extracranial level (arrowhead). **D, E:** Right vertebral angiograms, AP (**D**) and lateral (**E**) views, showing the anterior spinal artery (arrowheads) filled in the retrograde direction, and providing collateral support to the ipsilateral posterior inferior cerebellar artery. **F:** Preoperative three-dimensional computed tomography angiogram showing severe calcifications in the occluded bilateral vertebral arteries (arrows). **G:** Postoperative right common carotid angiogram, AP view, showing contrast filling of the mid-basilar artery (arrow) via the patent anastomotic artery (arrowhead). **H:** Follow-up axial T<sub>2</sub>-weighted MR image showing small infarcted areas in the pons (arrow) and right cerebellar hemisphere (arrowhead).

cause flow reversal in the ASA, but little is known about the clinical implications of this type of flow reversal. The reported cases share certain similarities in that no large territorial infarction with mass effect or fatal brainstem infarction was observed, and no deaths occurred, but the delicate balance between the extent of perfusion via the ASA and the degree of collateral supply from the posterior communicating artery varies considerably from patient to patient.<sup>13)</sup> Based on the angiographical analysis in our 2 cases, the occurrence of neurological deterioration seemed to parallel the development of the total blood supply from the anterior circulation system. However, the angiographical evaluation is far from being adequate to predict the outcome, as shown in Table 1. The period from the unilateral VA occlusion to the contralateral VA occlusion is also essential, since this must affect the total maturity of the collateral flow systems. The presence of an anatomical variant of the ASA also makes the clinical entities more complicated.<sup>15)</sup>

Advanced development of the ASA might generally have advantageously reduced the likelihood of an

ischemic event in these patients. Even in patients undergoing therapeutic occlusion of the VA for dissecting aneurysms, examining the ASA may be useful for predicting the likelihood of the lateral medullary syndrome developing in patients without a PICA or with a low origin PICA,<sup>12)</sup> because the ASA may be involved in preventing the propagation of thrombus in the VA distal to the site of occlusion and supplying blood to its perforating arteries in such high-risk patients. However, evidence that the ASA cannot provide absolute protection can be seen in Table 1.<sup>8,9,13,14,21)</sup>

Symptomatic, angiographically defined basilar artery occlusion and bilateral distal VA occlusions are associated with a mortality rate as high as 70% and significant morbidity in survivors.<sup>6)</sup> Current knowledge clearly suggests that the prognosis is grave in symptomatic patients with significant distal VA and basilar artery disease, and prompt restoration of blood flow preferably by endovascular angioplasty improves the outcome.<sup>6,10,16)</sup> In our Case 2, the severe calcified lesions at the occlusion site contraindicated endovascular angioplasty. The surgical

**Table 1 Summary of reported cases of bilateral vertebral artery occlusions with collateral anterior spinal artery (ASA) channels**

Case No.	Author (Year)	Age (yrs)	Sex	Presentation	Perfusion via ASA	Collateral supply from PcomA	Treatment	Comment	Outcome*
1	Karasawa et al. (1972) <sup>14)</sup>	28	F	headache, tinnitus, vomiting	total BA system	nil	conservative	reversed ASA flow filled BA trunk	?
2	Uchiyama et al. (1995) <sup>21)</sup>	66	F	vertigo, vomiting	part of BA	nil	conservative		GR
3	Garnier et al. (2003) <sup>8)</sup>	60	M	?	total BA system	?	conservative	reversed ASA flow filled BA trunk	?
4	Hott et al. (2004) <sup>9)</sup>	68	M	?	total BA system	?	?	reversed ASA flow filled BA trunk on MR angiography	?
5	Kang et al. (2007) <sup>13)</sup>	62	F	dizziness, gait disturbance	PICA and rt AICA	bil SCA filling	IA thrombolysis with stent-assisted angioplasty	reversed flow of ASA disappeared after recanalization	MD
6		64	M	rt hemiparesis, rt facial nerve palsy	part of BA	nil	IA thrombolysis with stent-assisted angioplasty	reversal of ASA flow direction after recanalization	MD
7		75	F	lt hemiplegia, lt facial nerve palsy	not perfused	BA trunk filling	conservative		VS
8		63	F	dizziness, gait ataxia	not perfused	bil SCA filling	stent-assisted angioplasty	reversal of ASA flow direction after recanalization	GR
9		40	M	dizziness, gait ataxia	PICA and rt AICA, lt AICA	nil	conservative	increased perfusion through ASA after occlusion	MD
10		81	M	quadriparesis, aphasia	not perfused	nil	IA thrombolysis with stent-assisted angioplasty	reversal of ASA flow direction after recanalization	VS
11	Present Case 1	76	M	dysarthria	lt PICA	BA trunk filling	conservative		GR
12	Present Case 2	48	M	consciousness disturbance	rt PICA	BA top filling	STA-SCA anastomosis	reversed flow of ASA resided	MD

\*According to Glasgow Coma Scale. AICA: anterior inferior cerebellar artery, BA: basilar artery, GR: good recovery, IA: intraarterial, MD: moderate disability, MR: magnetic resonance, PcomA: posterior communicating artery, PICA: posterior inferior cerebellar artery, SCA: superior cerebellar artery, STA: superficial temporal artery, VS: vegetative state.

indications remain unclear,<sup>19)</sup> especially in patients with acute deterioration caused by brainstem ischemia. We would like to emphasize the clinical significance of acute anastomotic surgery, which may be essential in the restoration of blood flow in selected patients. Until methods to evaluate the precise and simultaneous ischemic damage caused by VA occlusion are developed, we should incorporate therapeutic modalities including angioplasty as early in the course as possible, especially in a patient with clinical progression such as our Case 2. Also, these reversals of flow may cause the 'steal' phenomenon, resulting in some degree of cord ischemia.<sup>4)</sup>

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