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Case Report

A case of vertebral artery stump syndrome treated by parent artery occlusion via collateral anastomosis[☆]

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ABSTRACT

The treatment for vertebral artery stump syndrome (VASS) remains controversial. Here, we report a case of VASS in which cervical vertebral artery (VA) occlusion was performed. A 35-year-old man was admitted for left VA cerebellar infarction caused by left cervical VA dissection with severe stenosis on angiography, and was administered aspirin (100 mg/day). One month after discharge, the patient experienced recurrent stroke with cerebellar infarction. Digital subtraction angiography revealed that the origin of the left VA was occluded and that the VA received antegrade and retrograde flow via the left ascending and deep cervical arteries. The patient was diagnosed with a retrograde flow-induced thrombosis and recurrent embolic stroke. Planned parent artery occlusion (PAO) was performed using a left radial approach under local anesthesia. The patient did not experience stroke recurrence after the procedure. PAO via collateral anastomosis is an option for recurrent stroke treatment when anastomosis via the muscle branch is well developed.

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Introduction

Vertebral artery stump syndrome (VASS) is strongly associated with posterior circulation ischemic stroke [1,2] after vertebral

artery (VA) origin occlusion. Owing to its rarity, effective treatments for VASS remain controversial. To date, there have been no reports on parent artery occlusion (PAO) of the VA via collateral anastomosis.

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Here, we report a case of VASS in which cervical VA occlusion was performed using the radial artery approach and ascending carotid artery under local anesthesia.

Case report

Medical history and examination

A 35-year-old male, that underwent posterior fusion of a dislocated C5 vertebra, presented to our emergency department with sudden onset of headache and dizziness. Magnetic resonance imaging (MRI) revealed left cerebellar infarction; therefore, the patient was admitted to our hospital. Angiography indicated occlusion in the cortical segment of the left posterior inferior cerebellar artery (PICA) at the anterior inferior cerebellar artery (AICA)-PICA junction. Single antiplatelet therapy (SAPT) was initiated using aspirin, and the patient was discharged with no neurological deficits (Figs. 1A–C).

One month later, the patient presented to the emergency department with dizziness, nausea, and gait disturbances. Neurological examination revealed that the patient was conscious; however, he exhibited motor ataxia and coordination difficulties on the left side. Diffusion-weighted imaging (DWI) of the brain was negative, and the patient was discharged. However, a head MRI the following day revealed a left cerebellar infarction, and the patient was admitted to our hospital. Repeated angiography confirmed occlusion at the origin of the left VA along with collateral circulation from the anterior cervical artery (ACA), deep cervical artery (DCA), and inferior thyroid artery (Figs. 1D–G). Despite comprehensive investigations, the underlying cause of stroke remained unclear. It was hypothesized that vascular occlusion resulted from the dissection of the left VA after the patient underwent C5 fusion, leading to retrograde blood flow via collateral flow of the muscular branch-induced turbulent flow. As a result, the thrombus induced by turbulent blood flow (inferior thyroid artery) caused an embolic stroke, followed by cerebellar infarction induction (VASS) (Fig. 1H). Endovascular treatment was considered because the patient was refractory to medical treatment. The VA orifice was occluded on the second angiogram; therefore, antegrade reperfusion of the VA was challenging. Coil embolization of the retrograde flow below the collateral channel was planned.

Endovascular procedure

A 4Fr FUBUKI hard guiding sheath (Asahi Intec, Seto, Aichi, Japan) was inserted through the left radial artery under local anesthesia, and a Guidepost (Tokai Medical Products, Aichi, Japan) distal access catheter was carefully navigated into the proximal part of the ACA using an Excelsior SL-10 (Stryker, USA) over a CHIKAI 14 (Asahi Intec, Seto, Aichi, Japan). Subsequently, SL-10 was navigated into the VA anastomosis of the ACA (Fig. 2A), and endovascular occlusion of the parent vessel was performed using ten coils (Fig. 2B). The disappearance of the retrograde flow was confirmed angiographically (Fig. 2C, D).

Postoperative course

No neurological abnormalities were observed. On postoperative day 1 and 4, DWI presented no ischemia or indications of stroke. Magnetic resonance angiography (MRA) revealed disappearance of the VA origin. The patient was discharged 5 days after treatment without neurological deficits. Follow-up angiography performed 3 months later showed good collateral blood flow from the ACA, DCA, and inferior thyroid artery, with the disappearance of retrograde blood flow, followed by the discontinuation of antiplatelet therapy (Fig. 2E). Two years postoperatively, recurrent stroke did not occur in the patient.

Discussion

In 2013, Kawano et al. [1] reported the following VASS diagnostics: (i) acute ischemic stroke in the posterior circulation, (ii) occlusion of VAO confirmed by MRA ultrasound, CT angiography, and/or conventional angiography, (iii) presence of retrograde blood flow distal to the occlusion in the ipsilateral VA, (iv) absence of other causes of ischemic stroke, including intracranial vertebrobasilar artery lesion or embolic diseases, excluding arterial embolism. In addition to these criteria, Zhang et al. [2] suggested that (v) the residual stenosis of the VA was < 30% after EVT. In the present case, medical treatment was initiated in the acute phase, and there was no significant stenosis (> 30%) in the AICA-PICA segment on DSA 10 days after onset, and there was a low possibility of cerebral infarction due to hypoperfusion. These findings demonstrated that the cause of stroke was VASS.

In addition, there are 2 major processes of the VA blind-end due to occlusion: gradual stenosis and occlusion of the VA due to atherosclerotic changes caused by factors such as hypertension, diabetes, and dyslipidemia. The second process was acute occlusion of the VA, which is induced by dissection or trauma. In the present case, the patient was young and had no atherosclerotic disease, and angiography showed no atherosclerotic changes in the other cerebral vessels. These findings indicated that acute occlusion of the VA occurred around the position of the previous surgery, resulting in the VASS.

During initial stroke, dissection of the VA is expected to induce pseudo-occlusion. In the present case, thrombus formation occurred because of the turbulent blood flow generated by the antegrade and collateral retrograde flows. Initially, antiplatelet therapy was administered.

However, angiography revealed VA occlusion during the second stroke. This finding shows that the stagnation of blood flow at the distal blind-end of the VA occlusion produces thrombosis, resulting in the development of another stroke.

However, an optimal treatment for VASS has not yet been established. Endovascular VAS treatment prevents recurrent stroke [3,4] and reperfusion therapy prevents acute ischemic stroke [5,6]. However, antiplatelet agents [1,7] and anticoagulants [1,8] remain the foundation of treatment.

We chose antiplatelet agents for stroke prevention because of slight antegrade blood flow in the VA. However, the patient

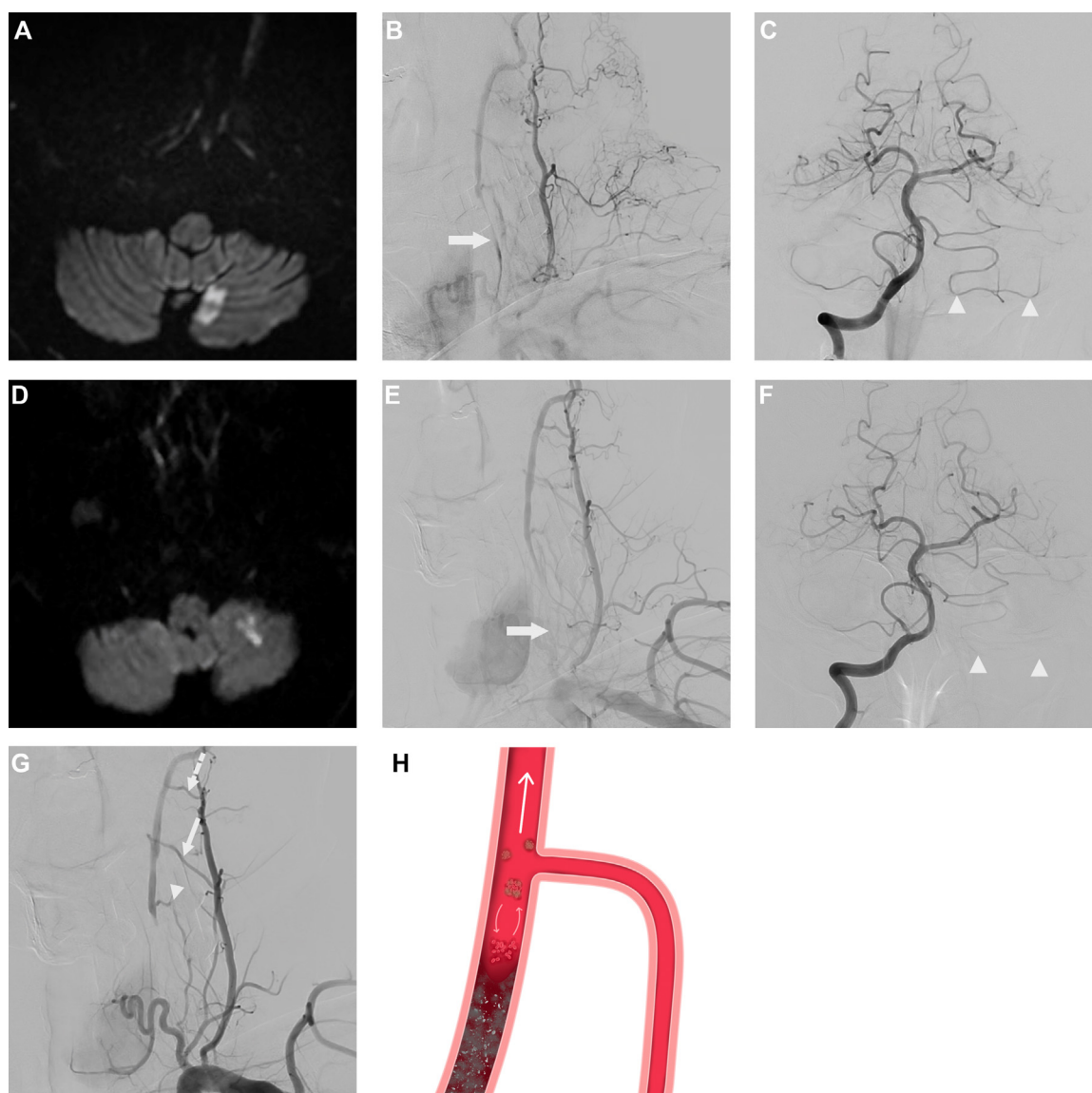


Fig. 1 – (A) Diffusion-weighted image on admission reveals left cerebellum infarction. **(B)** Oblique view of left subclavian arteriography reveals slow antegrade flow of the VA in the delayed phase (arrow). **(C)** Frontal view of the right vertebral arteriography reveals that the PICA was an AICA-PICA with no stenosis (arrowhead). **(D)** Diffusion-weighted image on the second admission reveals left cerebellar infarction. **(E)** Oblique view of left subclavian arteriography shows no flow of VA (arrow). **(F)** Frontal view of the left vertebral arteriography. The PICA shows the occlusion as an arrowhead. **(G)** Oblique view of the left subclavian artery showing collateral blood flow from the inferior thyroid artery (arrowhead), ascending cervical artery (arrow), and deep cervical artery (dashed arrow). Furthermore, origin of the vertebral arteries was not found. **(H)** This image shows that retrograde blood flow via collateral flow of the muscular branch induces turbulent flow proximal to the vertebral artery. VA, vertebral artery; AICA, anterior inferior cerebral artery; PICA, posterior inferior cerebral artery.

experienced recurrence 1 month later. Therefore, endovascular treatment was provided.

In the present case, because it was difficult to identify the origin of the VA on angiography, we considered that revascularization of the VA was difficult and chose to occlude the parent vessel at the blind-end with coil embolization. There are 2 methods of PAO: PAO with clipping [3] and coil embolization [4]. Clipping can occlude the V3 portion of the VA. However, it may be anatomically difficult to approach and is highly invasive. Moreover, preservation of collateral circula-

tion is particularly desirable in cases where external forces are routinely applied to the VA to reduce the risk of contralateral VA dissection. Additionally, in young patients, there may be potential for aneurysm formation due to hemodynamic stress [9]. Coil embolization of the vessel is relatively easier, but care must be taken to ensure sufficient embolization, as insufficient embolization increases the risk of thrombus formation. It is important not to inadvertently occlude the target vessel, particularly when dealing with multiple collateral pathways.

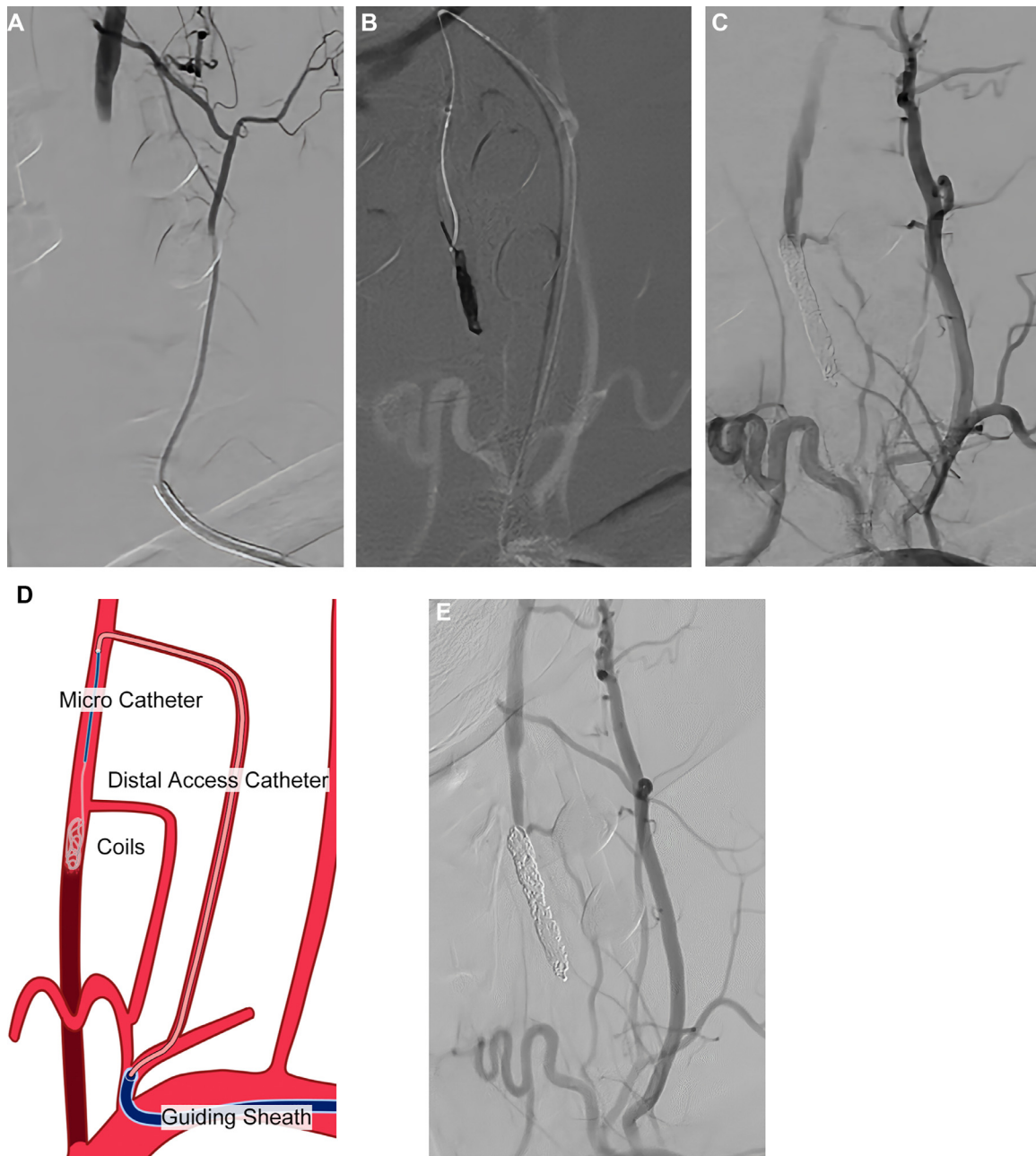


Fig. 2 – (A) The angiography via an ascending cervical artery showing the blind end at the VA proximal. (B) The parent vessel occluded by coils at the blind end is shown. (C) Subclavian arteriography shows complete disappearance of blood flow at the blind end. (D) Schema in which the catheter was inserted into the left vertebral artery and parent artery occlusion was achieved using coils. (E) Follow-up angiography 3 months after treatment showing remodelling of the collateral flow.

Two approaches are possible: via the contralateral VA or the ipsilateral muscular branches. The former approach is relatively easier to navigate because of larger vessels, but it also increases the risk of vascular injury or cerebral infarction. Moreover, vessel stretching during the procedure may move the patient, making continuous procedures challenging under local anesthesia. In contrast, the approach via the muscular branches reduces the risk of injury to the contralateral VA; however, the tortuosity or small caliber of the muscle

branches can make navigation into the occlusion site challenging. In the present case, it was feasible to navigate the microcatheter into the target vessel via transradial artery access, as a relatively straight muscular branch was present, and a distal access catheter was used.

There are no previous reports in which PAO for the VASS was performed under local anesthesia, with radial artery access, using a muscular branch approach. Advantages include minimal invasiveness, reduced risk of stroke, preservation of

collateral flow; and the possibility of discontinuing antithrombotic drugs in the future. The disadvantages include the possibility of another stump from the remaining collateral circulation and coil-induced thrombus formation. However, a long-term follow-up is necessary to determine the effectiveness of this treatment.

Conclusion

The treatment of VASS requires thorough and individualized consideration for each case, taking into account the unique characteristics and challenges presented by the patient's condition. In refractory VASS cases, targeted embolization of turbulent flow serves as a promising therapeutic option for the treatment of recurrent VASS, depending on the pattern of collateral circulation.

Data availability statement

All data generated or analyzed during this study are included in this article and/or its online supplementary material files. Further inquiries can be directed to the corresponding authors.

Author contributions

Conception and design: Sakamoto and Matsuda. Data acquisition: Sakamoto and Oiwa. Data analysis and interpretation: Sakamoto, Matsuda, and Oiwa. Drafting of the article: Sakamoto and Matsuda. Critical revision of the article: Sakamoto Matsuda. Sakamoto, Matsuda, and Mizutani reviewed the submitted version of the manuscript. Approved the final version of the manuscript on behalf of all authors: Matsuda. Statistical analysis: Sakamoto, Matsuda. Administrative/technical/material support: Matsuda, Matsumoto, and Mizutani. Study supervision: Mizutani.

Ethical approval

Ethical approval was not necessary for the preparation of this article.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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