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Case Report

Basilar Artery Occlusion Caused by Extracranial Vertebral Artery Dissection on Its Entry into the Transverse Foramen of the C6 Vertebra: Case Report

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Objective: Basilar artery occlusion (BAO) is an infrequent form of acute life-threatening stroke and may occur secondary to vertebral artery dissection (VAD). VAD, which occurs spontaneously and sometimes results from mechanical stress or blunt force trauma to the neck, sometimes occurs in the V1−V2 junction, but there are not many reported cases of those. Herein, we report a pictorially illustrative and clinically informative case of VAD in the V1–V2 junction following BAO.

Case Presentation: The patient was a 27-year-old woman who was transferred to our hospital with abrupt severe unconsciousness. On admission, she presented with generalized convulsions and respiratory arrest, and pan-scan CT and CTA indicated BAO. We performed mechanical thrombectomy and achieved recanalization of the basilar artery, and she was diagnosed with BAO secondary to the right VAD at the entry of the C6 transverse foramen (V1–V2 junction). In hindsight, she had scapula and back pain before the onset. She recovered with a modified Rankin scale score of 3 after 90 days from the onset.

Conclusion: VAD sometimes occurs at its entry into the transverse foramen of the C6 vertebra. In this case, VAD may be affected by minor trauma and potentially histological fragility due to the embryonic development process. Although BAO is sometimes difficult to diagnose because it presents with various symptoms, BAO secondary to VAD should be considered in cases of abrupt severe unconsciousness preceded by neck, scapula, or back pain in young and healthy persons.

Keywords basilar artery occlusion, vertebral artery dissection, V1−V2 junction, C6 transverse foramen, mechanical thrombectomy

Introduction

Basilar artery occlusion (BAO) is an infrequent form of life-threatening stroke with high disability and mortality rates.[1,2] Some studies have been conducted on mechanical thrombectomy (MT) for BAO, and the efficacy of MT for BAO has been recently reported.[3–5] Early recanalization of BAO has been shown to reduce mortality and improve clinical outcome.[1,3–9] Additionally, BAO may sometimes occur in conjunction with artery-to-artery embolism secondary to vertebral artery dissection (VAD) and result in severe stroke in young patients.[1,2,6,10] VAD occurs spontaneously or secondary to external blunt force trauma.[8,10–13] Regardless of whether it is spontaneous or secondary to trauma, it has been reported that VAD most commonly occurs at the V1−V2 junction at the level of the C6 vertebra (43%).[14] On the other hand, some studies have shown that VAD tends to occur more frequently in the V2 (transverse foramen from C6 to C2) or V3 (from the transverse foramen of C2 to the entry into dura) segment (60%–77%) and less commonly at the V1 (from subclavian artery to entry into transverse foramen of C6 vertebra)–V2 junction (9%–22%).[11–13] While there are some reports on its frequency, the number of reported cases of VAD in the V1−V2 segment is limited.

Herein, we report a pictorially illustrative and clinically informative case of VAD at the very V1−V2 junction. It presented as BAO, with the patient experiencing a sudden

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onset of severe unconsciousness with generalized seizure. Due to resuscitation efforts and limited available information, the diagnosis was delayed. The patient's prehospital history (preceding scapula and back pain) suggested VAD, and further investigation revealed the occurrence of VAD at the V1–V2 junction, which corresponds to the entry of the vertebral artery (VA) into the transverse process at the level of the C6 vertebra.

Case Presentation

The patient was a 27-year-old woman with an unremarkable past medical and family history. She presented with sudden and severe unconsciousness, and was transferred to our hospital. According to her family, she had scapula and back pain, and received a massage the night before onset. Additionally, she complained of headache and visual impairment on the morning of the disease onset day.

On admission, she presented with generalized seizure, respiratory arrest, low blood pressure, and bradycardia. She was unconscious, showed decerebrate rigidity, and had Glasgow Coma Scale of 4 (E1V1M2), and her pupillary light reflex was lost. Her right eye showed adduction and depression, and the right oculomotor paralysis was suspected, but anisocoria was absent (both pupil diameters were 3.5 mm). She underwent intratracheal intubation, and pan-scan CT and CTA were performed to evaluate her general condition. Hyperdense artery sign, a sign of basilar artery occlusion (**Fig. 1A**); defect of enhancement from BA trunk to BA top; and roughness of the vessel wall of the right VA were evident on the CT and CTA images (**Fig. 1B**). We considered that severe unconsciousness occurred due to BAO and suspected that it was caused by the right VAD. Accurate diagnosis and recanalization were crucial if BAO had occurred; therefore, we decided to perform DSA and MT when it was necessary. Although the time since the onset was within 4.5 hours, tissue plasminogen activator injection was not administered because the VAD was suspected as the cause of the BAO.

We punctured the right common femoral artery, inserted a 7-Fr long sheath, and induced a 7-Fr FUBUKI guiding catheter (Asahi Intecc, Aichi, Japan) to the left VA. To achieve the fastest recanalization of the BA and dissolve brainstem ischemia to preserve life, we selected the unaffected side of the left VA as the approach route. Pre-VA angiography showed BAO (**Fig. 2A**), and we decided to perform MT. By using the system of Catalyst 6 (Stryker, Kalamazoo, MI, USA)/Trevo Trak 21 (Stryker)/CHIKAI 0.014 (Asahi Intecc), MT was performed using a direct aspiration first-pass technique (ADAPT).[10] As a result, successful recanalization with thrombolysis in cerebral

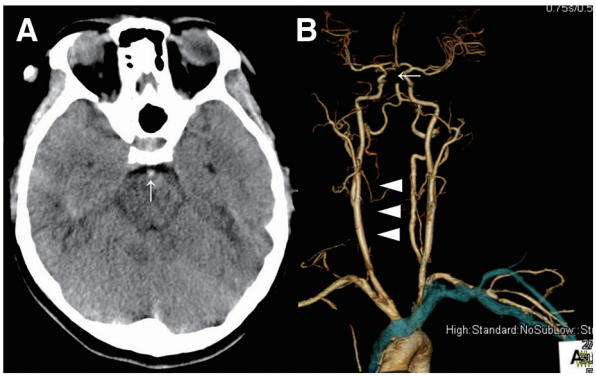

Fig. 1 Hyperdense artery sign was positive on head CT on admission, and acute BAO was suspected (arrow). (**A**) BAO from mid basilar to basilar top is shown (arrow), and V1 and V2 segments of the right VA were not well enhanced (arrowheads) on CTA (**B**). BAO: basilar artery occlusion

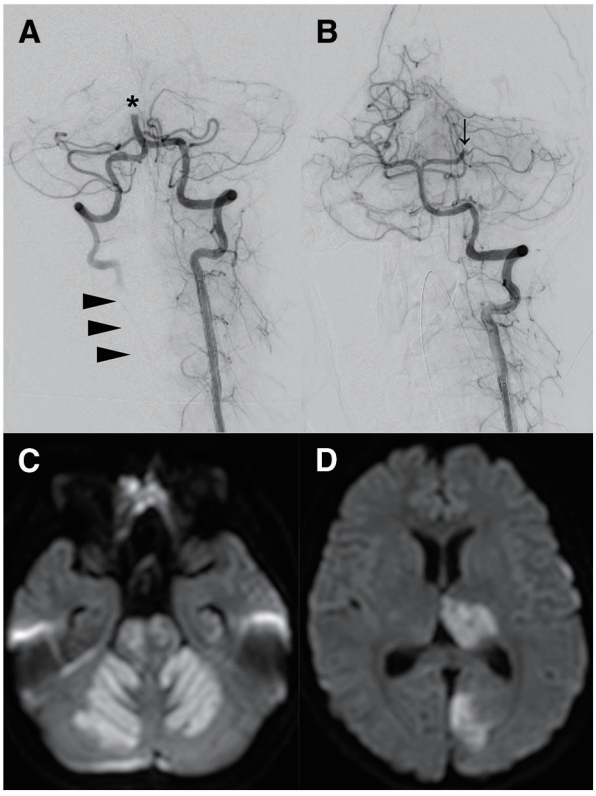

Fig. 2 In DSA, it is evident that the BA was occluded from the mid BA (asterisk), and the right VA flowed retrogradely via the union from the left VA (arrowheads). (**A**) Recanalization of the BA with thrombolysis in cerebral infarction grade 2b was achieved by MT. The P2 segment of the left posterior cerebral artery was remained to be occluded (arrow). (**B**) MRI on the next day of MT showed infarction in the territories of the bilateral superior cerebellar artery, perforators of BA, and left posterior cerebral artery (**C** and **D**). BA: basilar artery; MT: mechanical thrombectomy; VA: vertebral artery

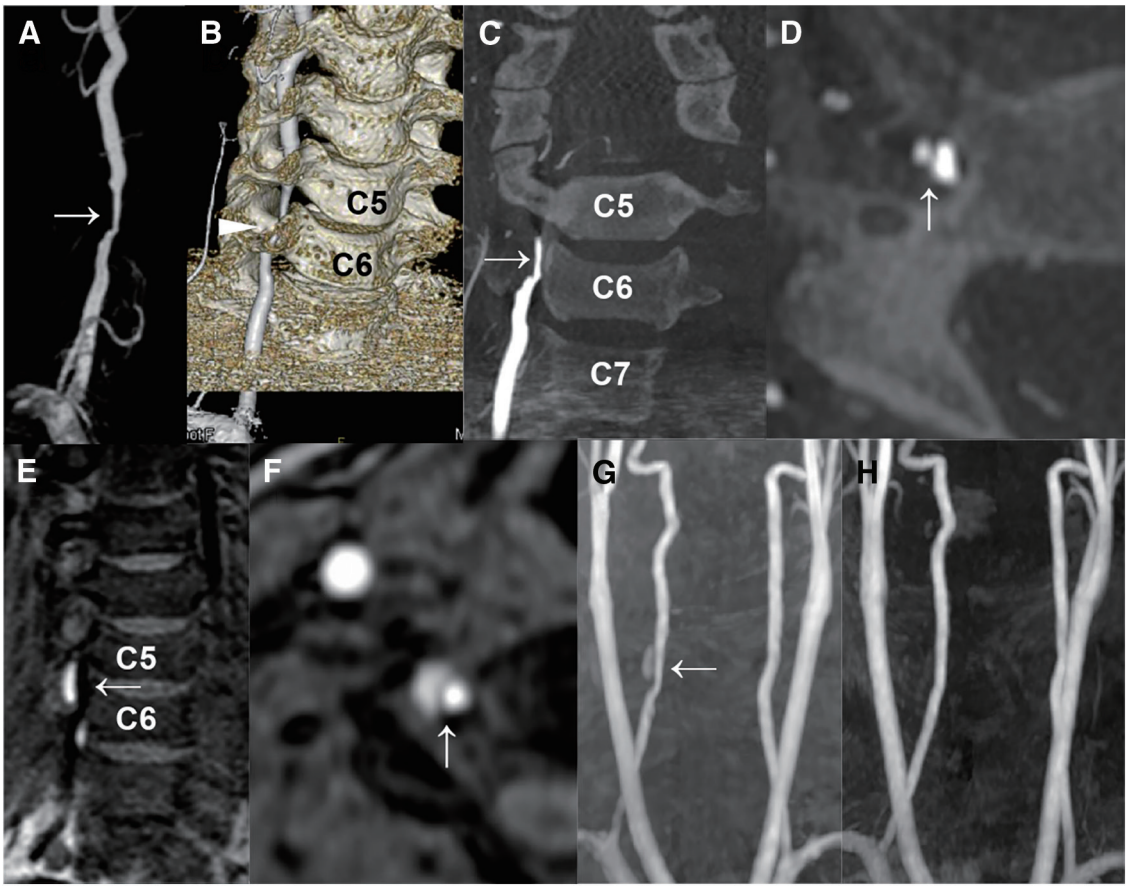

Fig. 3 Follow-up DSA on day 14 after the onset showed recanalization and anterograde flow of the right VA with stenosis due to dissection at the entry into the transverse foramen of C6 (arrow, arrowhead) (**A**–**D**). Follow-up MRI on day 30 after the onset showed that the pseudo lumen of dissection was cured with thrombus formation, and true lumen patency was preserved (arrow) (**E**: T1 black blood imaging; **F**: time of flight imaging; **G**: MRA). Follow-up MRA at four months after the onset showed that the vessel wall was modified and anterograde blood flow was preserved (**H**). VA: vertebral artery

infarction score of 2b was achieved with 2 PASS (the times of MT procedure until recanalization); the time details were as follows: onset-to-recanalization time, 349 min; door-to-puncture time, 325 min; and puncture-to-recanalization time, 44 min (**Fig. 2B**). Additionally, the right VA flowed retrogradely via the union from the left VA (**Fig. 2A**); this finding suggested the right VA occlusion. Therefore, we decided that emergent treatment for the right VAD was not necessary and aimed to manage it through thrombus formation to avoid further artery-to-artery embolism. MRI was performed on the next day of treatment and showed infarction of the bilateral superior cerebellar artery and left posterior cerebral artery territories (**Fig. 2C** and **2D**). At the 14 days after the onset follow-up DSA, the right VAD was revealed to occur in the V1–V2 junction at the transverse foramen of the C6 vertebra (**Fig. 3A**–**3D**). Antiplatelet therapy (aspirin 100 mg/day) for VA stenosis at VAD was administered to prevent further arterial embolism; as a result, no new infarction occurred. Additionally, MRI at 3 days after the onset showed that VAD was cured with thrombus formation in the pseudo lumen, and MRI at 30 days after the onset showed that recanalization and anterograde flow were achieved by modification of the vessel wall (**Fig. 3E**–**3H**). Therefore, antiplatelet therapy was quitted at 35 days after the onset, and additional treatment such as stent placement or parent artery occlusion was not required. Although partial right hemiparalysis, dysarthria, the right oculomotor nerve paralysis, the left abducens nerve paralysis, and the right homonymous hemianopia persisted, she recovered gradually to be able to eat by herself and perform ambulatory exercises. For further rehabilitation, she was transferred to a specialized facility at 38 days after the onset. Finally, she had a modified Rankin scale score of 3 at 90 days after the onset.

Evaluation of coagulopathy and autoimmune diseases related to vasculitis, or vascular fragility was performed to determine the etiology of this patient's VAD and BAO. However, no significant results were obtained.

We obtained informed consent from the patient and her family to publish this clinical report.

Discussion

BAO cases secondary to VAD in young adults and pediatric patients have been reported.[6,10] VAD occurs secondary to external force trauma at the level of C2 or C6, i.e., at the level where the VA enters or exits the foramen of the transverse process.[10–12] In addition to this, vascular fragility caused by connective tissue disorders or fibromuscular dysplasia, coagulopathy, and a history of intaking oral contraceptive pill use can be considered as possible causes of stroke in young women.[11,13,15] However, all these factors were absent in our patient. In this case, a history of preceding scapula and back pain as well as a massage was key in the diagnosis of VAD. Taking into account the possibility of vascular fragility, we performed MT by using ADAPT, avoiding vascular stress with using a stent retriever.[7,8]

For the possible etiology in this case, the influence of some external force on the C6 transverse process could be considered. However, it is doubtful that the external force of massage alone could cause dissection because there were no findings suggesting factors such as fractures or osteophytes that are commonly associated with inducing VAD. Although spontaneous VAD could be considered, it is also possible that connective tissue diseases, such as Ehlers−Danlos syndrome, which can lead to vascular fragility, may be the cause of this VAD and should be investigated genetically.[13,16] In approximately 90% of cases,[17,18] the VA runs steeply into the transverse foramen at the level of the C6 vertebra and is prone to be injured by external forces such as flexion or rotation.[12] Even in cases where it is presumed to be spontaneous VAD, there is a certain number of cases that have been preceded by minor trauma such as various sports-related injuries, stretching, or severe coughing.[16] Additionally, the connection point between the longitudinal dorsal artery and the seventh cervical segmental artery during the embryonic development process[17–19] could lead to dissection due to its potentially histological fragility.[20]

Furthermore, in this case, convulsion and apnea required intratracheal intubation, which delayed the diagnosis. BAO presents with various sudden symptoms, such as unconsciousness, hemiplegia, quadriplegia, visual impairment, dysarthria, apnea, cranial nerve palsies, and convulsions.[1,2,6] Since acute BAO can present with convulsions, it may be confused with epilepsy. Diagnosing BAO is considered difficult and may sometimes be delayed in such situations.[1,7] However, a history of preceding symptoms that is suggestive of VAD, such as neck, scapula, and back pain as well as trauma, is key in diagnosis of BAO secondary to VAD.

Conclusion

Even in the absence of structural factors such as fractures or osteophytes, external forces applied at the level of VA entry into the transverse foramen at the level of the C6 vertebra could be a cause of extracranial VAD. The occurrence of this event may be influenced, to some extent, by potential vascular fragility during the embryonic developmental process. BAO secondary to VAD should be considered in young or healthy patients who present with abrupt severe neurological deficits with the relevant medical history, which is preceded by scapula and back pain.

Informed Consent

We obtained informed consent from the patient and her family to publish this clinical report.

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Disclosure Statement

The authors declare that they have no conflicts of interest.

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