

# A Case of Traumatic Vertebral Artery Dissection Complicated by Vertebro-Basilar Artery Occlusion

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## Abstract

The vertebrobasilar artery occlusion (VBAO) is an infrequent cause of acute ischemic stroke, which invariably leads to death or long-term disability if not recanalized. The VBAO as a complication of traumatic vertebral artery dissection (VAD) is rare. We describe a 61-year-old woman with VBAO caused by traumatic VAD successfully treated by mechanical thrombectomy with intravenous thrombolysis.

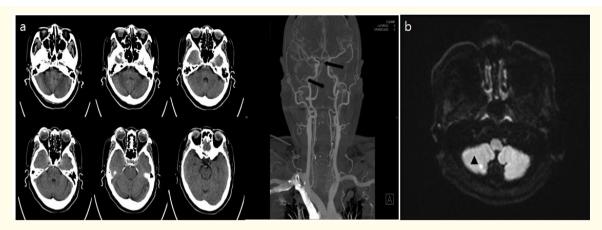
Keywords: Vertebral Artery Dissection; Stroke; Thrombolysis

# Introduction

Vertebral artery dissection (VAD) is caused by high blood pressure, trauma or connective tissue disease [1]. The annual incidence of spontaneous VAD can be estimated at 1 per 1000,1000 to 1.5 per 100,000 [2]. Spontaneous dissections of the carotid or vertebral artery account for only about 2 percent of all ischemic strokes, but they are an important cause of ischemic stroke in young and middle-aged patients [3]. The clinical manifestation of VAD is mild and the outcome is relatively good. However, the prognosis is fatal when the basilar artery is occluded by VAD. We describe the patient with vertebrobasilar artery occlusion (VBAO) caused by traffic accident related VAD.

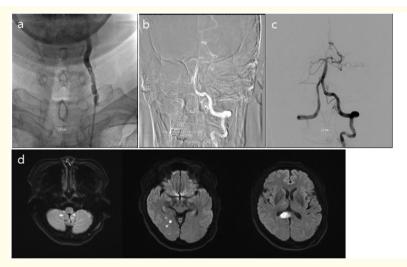
## **Case Report**

A 61-year-old woman with hypertension and dyslipidemia visited our emergency department, presenting symptoms chest pain after traffic accident. She was diagnosed sternum fracture and admitted to department of chest surgery. The 3<sup>rd</sup> day later, she abruptly complained dizziness and gait disturbance. Neurological examination showed dysarthria, left central type facial palsy with left hemiparesis, and ataxic gait. Initial national institutes of health stroke scale (NIHSS) score was 6. Brain CT angiography revealed no acute hemorrhage but the left V4 segment, distal basilar artery and both posterior cerebral artery (PCA) were invisible (Figure 1a). Magnetic resonance diffusion weighted image (MR DWI) showed acute infarction at the right cerebellar hemisphere in the posterior inferior cerebellar artery territory (Figure 1b). After neuro-imaging, her consciousness progressively deteriorated and quadriparesis proceeded; the NIHSS score of 16. She was treated with intravenous thrombolysis with rt-PA (0.9 mg/Kg for 1 hour) 110 minutes after symptom onset. Endovascular treatment (EVT) was followed after intravenous thrombolysis. Digital subtraction angiography displayed left proximal VAD (V2 segment) with right superior cerebellar artery (SCA) and both PCA occlusion (Figure 2a, 2b). After EVT with stent-retriever, right SCA was complete recanalized, but PCA was partially recanalized (Figure 2c), then we inserted the stent to portion of vertebral dissection. After EVT, her mentality and motor symptom were complete recovered. The 5<sup>th</sup> day MR DWI follow-up scan showed multiple embolic infarction at the cerebellum, right temporo-occipital cortices and splenium (Figure 2d).



*Figure 1:* Brain CT angiography (CTA) and magnetic resonance diffusion weighted image (MR-DWI) of the patient. a. Brain CTA demonstrates occlusion of the left distal vertebral artery, top of the basilar artery and both posterior cerebral artery (arrows) without hemorrhage.

b. MR DWI shows high signal intensity at the right cerebellar hemisphere, suggesting acute cerebral infarction (arrow head).



**Figure 2:** Digital subtraction angiography (DSA) shows luminal irregularities of the left V2 (a) and occlusion of right superior cerebellar artery (SCA) and both posterior cerebral arteries (PCA). Microcatheter is located at the right P2 (b), and DSA after mechanical thrombectomy displays complete recalization of right SCA and left PCA, and partial recanalization of right PCA (c). DWI follow-up scan reveals new hyper intensities in the both cerebellum, right temporo-occipital cortices and splenium that were not previously observed (d).

Laboratory test including blood counts, biochemistry, lipid panel, and urinalysis were normal. Transthoracic echocardiography and 24 hour holter monitoring showed no particular finding. Moreover, no finding was noted in blood tests for vasculitis and connective tissue diseases. She was discharged on the 7th day after thrombolysis with NIHSS score of 1, with mild dysarthria. After 3 months, the modified Rankin scale score of the patient was 1. We used dual antiplatelet agents (aspirin and clopidogrel) for the first 3 months. After that, she was prescribed clopidogrel with medications for hypertension and dyslipidemia. One year after the onset of stroke, she had no neurological symptoms and sign except paresthesia on the left limb.

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### Discussion

Arterial dissection caused by the separation of the intimal layer can induce intra-luminal stenosis or occlusion through intramural hematoma and false lumen formation. Further extension of the damage to the tunica adventitia can cause a dissecting aneurysm or cerebral hemorrhage [2]. Treatment of arterial dissection is usually conservative, with antiplatelet or anticoagulant agent [4], and there is no significant difference in efficacy reported between the two agents [5].

Recent study showed EVT is effective for acute ischemic stroke due to large artery occlusion [6]. However, EVT in patient with large vessel occlusion caused by arterial dissection has risk of additional arterial dissection, pseudo-aneurysm or vessel rupture [7]. The case reports in patient with VBAO due to trauma-induced vertebral artery dissection are rare [8].

A successful thrombolytic reversal of VBAO depends on the early initiation of treatment. Another meta-analysis of patients with largevessel ischemic stroke proved earlier treatment with EVT and medical therapy compared with medical therapy alone was associated with lower degrees of disability at 3 months [9]. In our case, EVT started 150 minutes after the symptom onset and finished 210 minutes later. Early diagnosis and treatment with thromboysis improved the prognosis of the patient with VBAO.

There is limitation in applying our patient's treatment to all patients with VAD. Conservative management with antithrombotic agents is recommended for treatment guidelines [4], because most dissection of the vertebral artery heal spontaneously. Therefore, EVT is considered once medical therapy fails. In our case, we decided intravenous thrombolysis because VBAO is caused by thromboembolic rather than hemodynamic mechanism. Despite thrombolytic treatment, the patients symptoms worsened and additional EVT was performed.

#### Conclusion

VAD should be considered in patients with cerebral infarction after trauma because rapid diagnosis and treatment determine the prognosis in VBAO. Our case confirm that the safety and efficacy of mechanical thrombectomy with intravenous thrombosis in patient with acute ischemic stroke due to VAD related VBAO. However, further studies are needed to determine the safety and efficacy of VBAO caused by VAD.

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