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Case report

Rotational vertebral artery occlusion associated with occipitoatlantal assimilation, atlantoaxial subluxation, and basilar impression

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1. Introduction

Rotational vertebral artery (VA) occlusion, also known as bow hunter's stroke, is a rare form of vertebrobasilar ischemia elicited by head rotation, and it results from transient mechanical obliteration of the VA [1]. The VA at the C1–C2 junction is involved most frequently, and hemodynamic compromise of the dominant VA is usually responsible for ischemic episodes [1]. Various pathoanatomies of the craniovertebral junction (CVJ) have been reported to be associated with rotational VA occlusion. Adult-onset rotational VA occlusion associated with a CVJ anomaly, however, is very rare. We describe a case of rotational VA occlusion associated with occipitoatlantal assimilation, atlantoaxial subluxation (AAS), and basilar impression.

2. Case report

This previously healthy 22-year-old male college student with no history of a major head/neck trauma suddenly experienced dizziness and nausea when he abruptly turned his head to the left side. The episode was painless. His symptoms persisted for several days, and he visited a local physician and was referred to our neurology department. He was alert and well oriented. Mild truncal ataxia and gait disturbance was noted on neurological examination. Neurologic tests for hearing disturbance, including the Weber and Rinne's, were negative. A mildly positive finger-to-nose test with negative Romberg's sign suggested presence of a small cerebellar lesion. He did not complain of neck pain after neck flexion/extension, and dynamic X-rays of the cervical spine to evaluate translational instability were not performed. T2- and diffusion-weighted images of the brain magnetic resonance (MR) imaging demonstrated a few small high-intensity spots in the cerebellum (Fig. 1A). He was diagnosed with a cerebellar infarction and was admitted. Based on clinical history, rotational vertebral artery occlusion was suspected. A thin-slice coronal CT image indicated that the right VA might be squeezed between the right C1–C2 joint and a bony spur of the occipital bone (Fig. 1B). Two weeks after admission, digital subtraction angiography (DSA) of the cerebral arteries was performed. The left VA was hypoplastic and barely perfused the left cerebellar hemisphere. The right VA, which was dominant and perfusing both of the cerebellar hemispheres, was markedly tortuous at the C1–C2 junction (Fig. 1C). When he was asked to rotate his head to the left for 45°, cessation of blood flow at the C1–C2 junction was noted (Fig. 1D), confirming the diagnosis of rotational VA occlusion. Precaution to ensure his safety had been taken during the procedure: both an interventional neuroradiologist and an anesthesiologist attended the angiography. As soon as cessation of the flow was confirmed, he was asked to rotate

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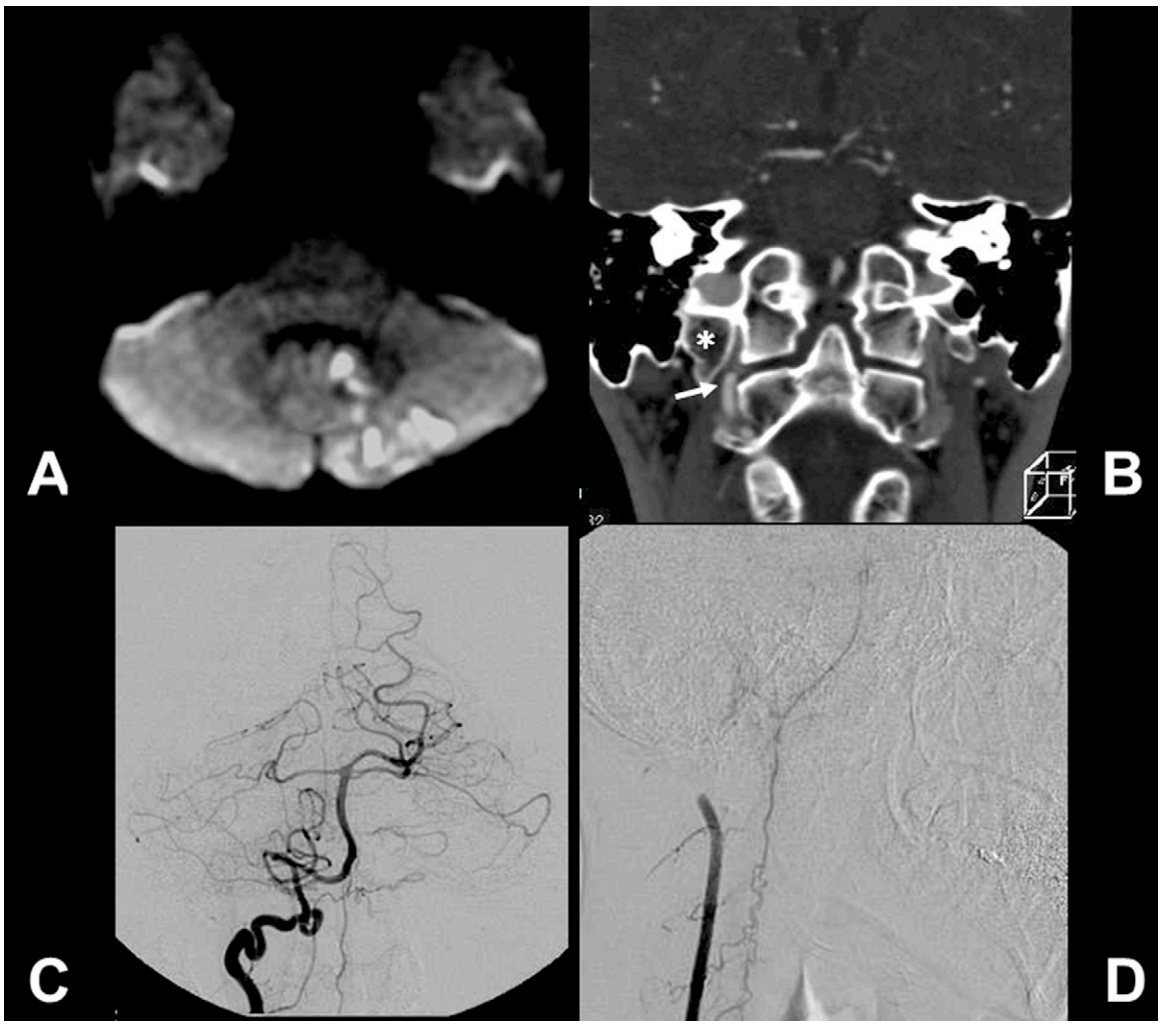

Fig. 1. (A) Diffusion-weighted MR imaging studies showing high intensity spots in the cerebellum, indicating acute cerebellar infarction. (B) A thin-slice coronal CT scan with contrast suggesting that the right VA (arrow) might be squeezed between the right C1–C2 joint and a bony spur of the occipital bone (asterisk). (C) The right VA (anteroposterior view), which was dominant and perfusing both of the cerebellar hemispheres, was markedly tortuous at the C1–C2 junction. (D) Cessation of the blood flow was confirmed when the patient rotated his head to the left side for 45°.

the neck back to neutral position, and no adverse events occurred. Furthermore, a sagittal CT image showed the atlantoaxial distance (AAD) of 9 mm (Fig. 2A), indicating the presence of atlantoaxial subluxation (AAS). The tip of the dens process was 6 mm above the McGregor's baseline (Fig. 2A), which indicated the presence of basilar impression [2]. Concomitant occipitoatlantal assimilation was also noted (Fig. 2A, arrow). T2-weighted MR imaging of the CVJ showed that basilar impression was mild, and there was only a slight distortion of the brainstem (Fig. 2B). Combination of AAS, basilar impression and tortuous right VA seemed to have resulted in rotation-induced obliteration of the artery and subsequent cerebellar infarction. He was referred to us 30 days after admission. By that time, he was neurologically intact. Because of the instability, a posterior fusion surgery was recommended, and he agreed to undergo the procedure.

An occipitocervical instrumented fusion down to C3 was planned, since the C1 posterior element was absent. A Synthes Occipital Cervical Fusion System (Synthes K.K., Tokyo, Japan) was used. He was positioned prone under general anesthesia, with his neck in neutral position. Pre- or intra-operative traction was not employed, because there were no signs of brainstem or cord dysfunction. A midline occipital plate was fixated with three bicortical titanium screws (Fig. 3A). For C2, polyaxial C2 screws were placed bilaterally into the lamina via a translaminar trajectory (Fig. 3B) to minimize the risk of VA injury [3]. A shorter polyaxial screw was placed into each lateral mass of C3. Subsequently, the screws were connected with contoured titanium rods. An iliac crest autograft was placed between the decorticated occiput and C2 spinous process. Postoperative course was uneventful, and a good alignment of the CVJ was noted. The AAD was reduced to 2 mm, which was within normal range (Fig. 3C). He was discharged 10 days after surgery. Sold bony fusion was achieved by 9 months after surgery, and he has not experienced any recurrences of ischemia. Mild inconvenience in head rotation is well tolerated.

3. Discussion

Rotational VA occlusion mostly occurs at the C1–C2 junction [1]. Although hemodynamic compromise of the dominant VA is usually responsible for the ischemic episodes, obliteration of the non-dominant VA may cause symptomatic ischemia [4], and occasionally, ischemia may be of embolic rather than of hemodynamic origin [5]. The presence of underlying pathoanatomies together with disproportion in the VA size seem to be prerequisites for development of symptomatic ischemia [1,4,5]. Our patient was unique in that he sustained a cerebellar infarction of the left posteroinferior