

## Right cerebellar infarction due to ipsilateral neck-rotation-induced right vertebral artery compression and occlusion, demonstrated by CT angiography

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Rotation-induced vertebral artery compression and occlusion with the outcome of cerebellar infarction (as opposed to the outcome of transient ischemia from hemodynamic insufficiency, known as bow hunter's stroke) is extremely rare. We report a 40-year-old male who suffered from ipsilateral neck-rotation-induced right vertebral artery compression and occlusion that resulted in right cerebellar infarction. In most reported cases, the rotation-compressed vertebral artery is located at the C1–2 level; however, our patient's compressed artery was located at the C6–7 level, as demonstrated clearly by 3-dimensional CT angiography. This case report is based on a literature review and an investigation of the likely factors of this specific incident via the patient's personal details, clinical course, and diagnostic images.

### Case report

A 40-year-old male was sent to the emergency room (ER) because of a sudden onset of dizziness and vomiting while riding a bicycle. The patient had a history of type I diabetes mellitus and hypertension as well as a 20-year smoking history while using medication. His father died from stroke. The patient reported a painful sensation in the posterior area of the brain and neck. An acute infarction in the right cerebellar area with mild fourth ventricle compression was discovered in the ER using computed tomography (CT) and magnetic resonance imaging (Fig. 1a). The right vertebral artery (RVA) from levels C7 to C1 and the right poste-

rior inferior cerebellar artery were not visualized on the magnetic resonance angiography (MRA) image (Fig. 1b). However, there was no clear evidence of RVA dissection. Neither vessel-wall atherosclerotic changes nor mural thrombus of bilateral vertebral arteries (VAs) was demonstrated on a B-mode ultrasound examination.

On the second day after hospitalization, the patient was scheduled to undergo neurosurgical decompression because a routine CT revealed that his brain edema and ventricular enlargement had become more critical.

Two weeks later, during neck angiography exploration, the RVA was perceived not to have opacified from levels C7 to C1 (Fig. 1c). However, after 1 month, no occlusion of RVA was evident from the frontal view (Fig. 2a), and only partial stenosis at the C6–7 level was revealed in a subsequent right-neck-rotation angiography image (Fig. 2b). The patient was then ordered to position his neck either rightward or leftward at different angles to evaluate the bilateral VA flow speeds and the suspicious stenotic site, according to the color Doppler ultrasound parameters. This evaluation revealed that at the C6–7 level junction, RVA flow speed changed at different ipsilateral rotation angles (Figs. 3a–c). No other RVA location nor any left vertebral artery location was found to exhibit this phenomenon. As shown on neck axial rotational contrast-enhanced CT and 3-dimensional volume-rendering CT angiography (3D-CTA)

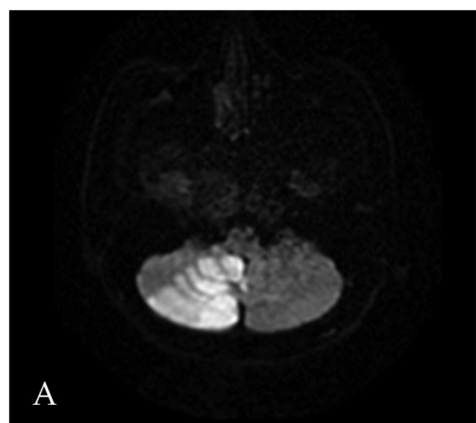
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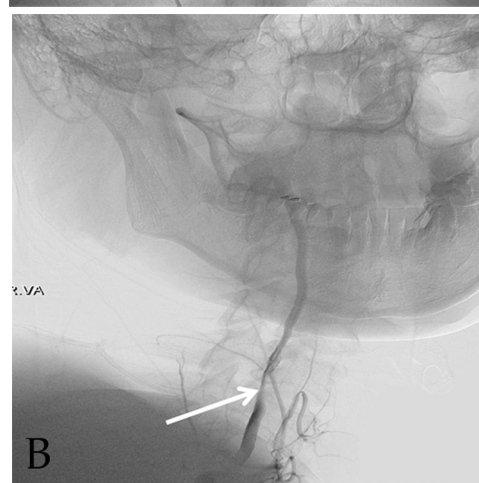
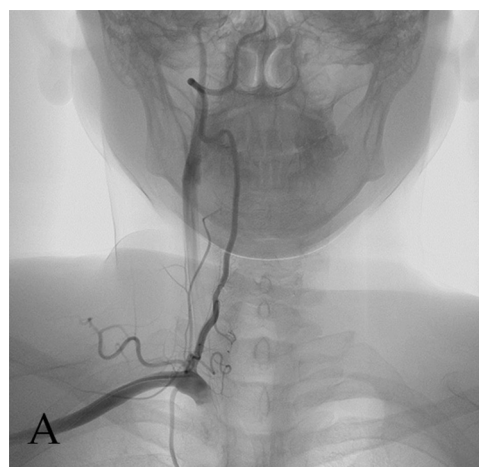
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**Figure 1.** Right cerebellar infarction changes on magnetic resonance (MR) imaging and angiography (MRA). A. Diffusion-weighted imaging revealed an acute infarction change in the right cerebellum with mild fourth ventricle compression. B (PET) and C (neck angiography) revealed that the RVA was not patent from levels C7 to C11.



**Figure 2.** Focal stenosis as shown via neck angiography. A. Neutral neck angiography showed the patent RVA. B. Neck angiography during ipsilateral neck rotation revealed focal stenosis at the C6–7 level (arrow).

examination, the RVA was found to have a narrowed area only at the C6–7 level, with an adjacent prominent osteophyte of the C6 uncinate process (Figs. 4a, b).

The patient was then discharged in a stable condition and followed up routinely at our hospital.

## Discussion

Rotation-induced VA compression and occlusion leading to cerebellar infarction is extremely rare. Some articles have reported that rotationally compressed extracranial VA leading to vertebrobasilar insufficiency (namely, bow hunter's syndrome) is reversible (1). The reasons for infarction included local stenosis, mechanical compression from the adjacent abnormal musculoskeletal structure, possible artery-to-artery (A-to-A) embolism, or even arterial dissection (2, 3).

Bow hunter's syndrome is usually accompanied by symptoms such as nystagmus, vertigo, tinnitus, fainting, blurred vision (4, 5), or presyncopal and syncopal episodes (1).

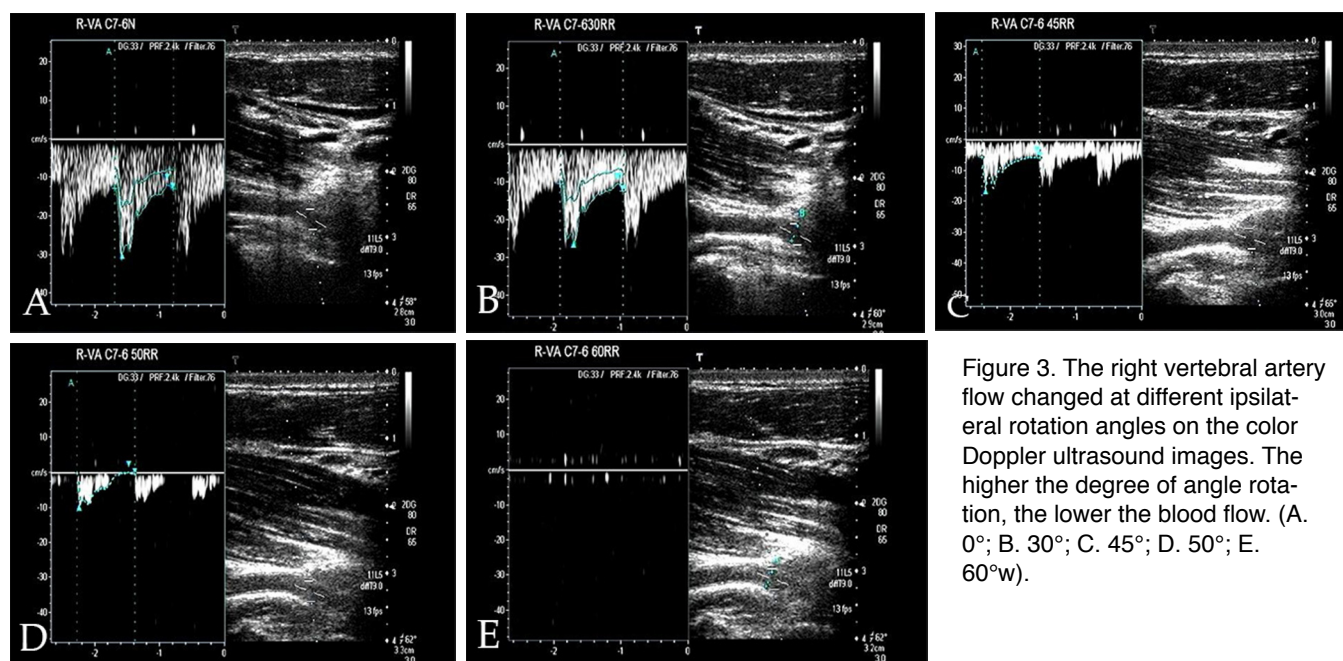


Figure 3. The right vertebral artery flow changed at different ipsilateral rotation angles on the color Doppler ultrasound images. The higher the degree of angle rotation, the lower the blood flow. (A. 0°; B. 30°; C. 45°; D. 50°; E. 60°w).

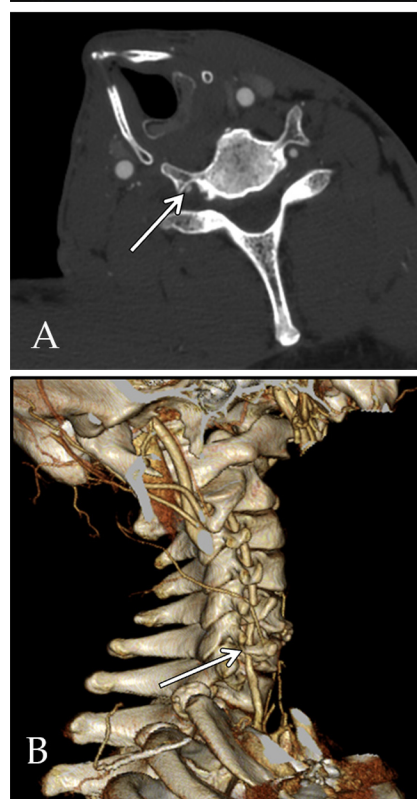


Figure 4. The right vertebral artery (RVA) at the C6–7 level was narrowed due to the impingement of the adjacent lateral osteophyte, as demonstrated on contrast-enhanced CT and 3-dimensional volume-rendering CT angiography (3D-CTA). A. Axial contrast-enhanced CT with right neck rotation showed focal stenosis of RVA with an adjacent prominent lateral osteophyte (arrow). B. 3D-CTA demonstrated the focal stenosis at the C6–7 level with rightward neck rotation (arrow). 50°; E. 60°w).

These symptoms could be attributed to asymmetrical excitation of the bilateral labyrinth induced by transient ischemia or inferior cerebellar hypoperfusion (4). In our patient, no typical symptoms occurred before the sudden onset of cerebellar infarction.

Brain infarction consequent to repetitive rotational VA occlusion has been reported in a child (2), who was found to have an A-to-A embolism with a mobile mural thrombus at the VA occlusion site upon head rotation. The A-to-A embolism due to repeated neck rotation was suggested as a mechanism of ischemic attack. However, the accurate mechanism of the underlying thrombus formation in the A-to-A embolism consequent to rotational VA occlusion remains unclear (2). This phenomenon may be associated with vascular damage following repeated VA compression (2, 6). In contrast, thrombus formation may be attributable to blood-flow stasis in VA (2).

Greiner HM et al. described a posterior circulation stroke due to the impingement of osseous and/or ligamentous structures on the VA in a child, resulting in RVA occlusion, a possible torn wall thrombus that flowed into the RVA or RICA, and/or even VA dissection (3). Similarly, we found in our patient that the RVA at the C6–7 level had narrowed along with an adjacent prominent lateral osteophyte on the rotational neck CTA image, although there was no clear evidence of mural thrombus in our patient's narrowed RVA wall. However, the conclusion of one article stated that the possibility of A-to-A embolism following repeated VA compression should be considered in the diagnostic workup of patients with vertebrobasilar stroke, despite the absence of routine MRA abnormalities (2).

Most reported cases of rotational VA compression occur at the C1–2 level during contralateral neck rotation (4). However, our patient's compression was located at the C6–7 level. There have been limited previous reports of dem-

onstrated occlusion or stenosis via 3D-CTA at this level. In this case, we performed 3D-CTA with the patient performing rightward neck rotation and could clearly depict the stenosis adjacent to the osseous component of the C6 transverse process. The most commonly compressed site associated with bow hunter's syndrome is the V2 segment, because the VA passes through the foramen transversarium of the subaxial cervical spine (1).

Several methods have been selected for the treatment of rotation-induced VA compression, including decompression surgery, cervical fusion, and conservative treatment (9). For our patient, only conservative treatment was used, and the RVA was later recanalized. However, surgical treatment may be selected if conservative therapies fail (9). Meanwhile, continued controversy surrounds the choice of first-line treatment modality for rotation-induced VA compression, particularly in younger patients (4, 9). The method for treatment must be based on the site of occlusion as well as the assessment of the patient as a surgical candidate (10).

## Conclusion

Cerebellar infarction due to ipsilateral neck-rotation-induced VA compression and occlusion is extremely rare. Repeated impingement by the adjacent prominent lateral osteophyte and embolism formation are possible causes. Decompression surgery and conservative treatment can be selected for prophylactic administration, depending on the patient's situation.

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