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Vertebral Artery Compression during Roll Tilt: Is the Edge of the Foramen Magnum a Culprit?

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Dear Editor,

Rotatory vertebral artery occlusion (RVAO) indicates a mechanical compression of the vertebral artery (VA) in certain head positions.[1] In typical RVAO, the arterial occlusion occurs mostly at the high cervical level (atlantoaxis) during contralateral head turning.[2] There is a single report of RVAO during head tilt;[3] however, the exact structure responsible for compressing the VA in that case was not established. We report here another case of tilt-induced RVAO and discuss the role of the foramen magnum in its etiology.

A 64-year-old man presented with positional vertigo of 1 month duration. The patient specifically indicated that the vertigo occurred without tinnitus and within 5−10 seconds of tilting his head to the left. Positional maneuvers including supine head roll and the Dix-Hallpike maneuver on both sides evoked neither vertigo nor nystagmus. However, a leftward head tilt while upright induced right-beating nystagmus, which changed to left-beating nystagmus after 25 seconds. There was no obvious vertical or torsional component (Fig. 1A). A rightward head tilt evoked neither vertigo nor nystagmus. MRI revealed no parenchymal lesion in the brainstem or cerebellum. However, CT angiography demonstrated occlusion of the right VA, and a severe stenosis at the intracranial segment of the VA just above the origin of the posterior inferior cerebellar artery (PICA). In addition, the stenotic segment of the left VA was spatially close to the edge of the foramen magnum, which probably compressed the stenotic segment of the left VA during ipsilesional head tilting (Fig. 1B). Proximal left VA duplex ultrasonography revealed blunting of the dynamic arterial flow and decreased peak systolic flow velocity during leftward head tilt (Fig. 1C). The right VA and PICA were visualized by a retrograde flow from the left VA on digital subtraction angiography. After balloon angioplasty and intravascular stenting of the stenotic left VA segment, the patient's symptoms completely resolved, and he did not experience recurrence during the immediate 1-year follow-up.

While confirmatory dynamic angiography was not performed, the anatomical relationship, the duplex ultrasonographic findings, and the outcome of intervention on the stenotic VA segment led to a diagnosis of RVAO during head tilt. Of particular interest was that the symptoms of RVAO disappeared after intra-arterial intervention in our patient, even though the procedure did not remove the speculated medial edge of the foramen magnum. However, given that normal subjects did not experience vertigo during head tilt and the RVAO was resolved after intra-arterial intervention in the present case, the underlying focal stenosis of the dominant VA must have contributed to the development of RVAO during head tilt.

Down-beat nystagmus with mixed horizontal and torsional components have been found in patients with RVAO in several studies, and it has been suggested that transient ischemia-induced superior labyrinth excitation is the mechanism underlying the nystagmus.[1] In the present case, the left PICA branched out before the stenotic VA segment; therefore, the ini-

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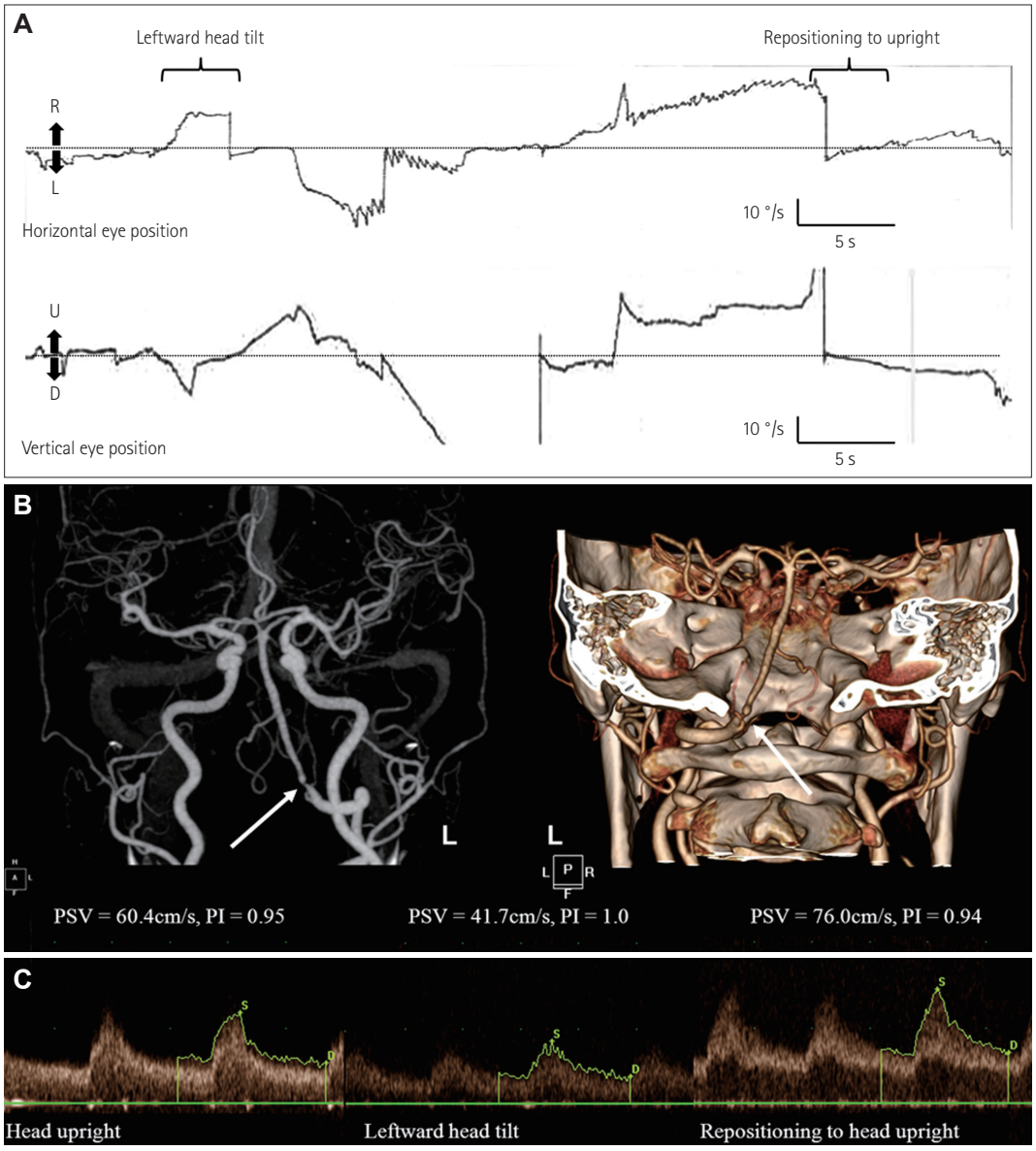

Fig. 1. Findings of rotational vertebral-artery occlusion during roll tilt. A: Electronystagmography. Leftward head tilt evokes an initial right-beating nystagmus for 10 s, followed by 15 s of a null period and then left-beating nystagmus. There is no obvious vertical or torsional component. B: 3-D CT angiography. Total occlusion of the right vertebral artery and focal stenosis of the left vertebral artery (arrow) can be seen. The medial edge of the foramen magnum is spatially close to the stenotic left vertebral artery. C: Proximal left vertebral artery duplex ultrasonography. Leftward head tilt results in a reduction in peak systolic flow velocity from 60 to 40 cm/s (a 33% reduction). Rebound hyperemia can be seen while uprighting.

tial right-beating nystagmus and subsequent left-beating nystagmus in this patient can be ascribed to transient ischemia-induced asymmetrical excitation and inhibition of the right labyrinthine. Alternatively, the initial right-beating nystagmus might have been generated by ischemia of the right cerebellum. The left medial vestibular nucleus, which is supplied directly by the left VA perforators, is also a candidate for generation of the initial nystagmus. The absence of tinnitus and the vertical and torsional components of the nystagmus might favor the latter mechanism. The evolution into left-beating nystagmus could be attributed to a compensatory mechanism.[1,2] However, the lack of evidence regarding the perfusion status leaves the exact mechanism of nystagmus uncertain.

In conclusion, arterial compression at the edge of the foramen magnum should be suspected when RVAO occurs during head tilting. Intra-arterial intervention may be considered as a treatment option.

Conflicts of Interest

The authors have no financial conflicts of interest.

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