

Letter to the Editor

A thromboembolic mechanism in bow hunter's stroke: Importance of hemodynamic evaluation by ultrasonography during head rotation



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Dear Editor,

Bow hunter's stroke (BHS) is a rare cerebrovascular disease due to vertebral artery (VA) occlusion during head rotation [1]. Its etiology has been reported to involve a hemodynamic mechanism and a thromboembolic mechanism derived from endothelial damage by repetitive VA compression, though this remains controversial [2,3]. Previously, we reported a 7-year-old patient who developed BHS with mobile mural thrombus, suggesting an artery-to-artery thromboembolic pathology [4]. Here, we describe another case of embolic BHS and consider the etiology by investigating intracranial cerebral flow using color-coded ultrasonography. Although we previously suggested the significance of detecting rotational VA occlusion for BHS with mural thrombus, suggesting an artery-to-artery embolism, ultrasonography examinations of VAs during head rotation are not performed routinely in cases of embolic stroke of undetermined source (ESUS) in the vertebrobasilar system. Additionally, this is often overlooked if hemodynamic symptoms are not observed. Even if symptoms are observed, when no obvious abnormalities are observed in the vessel wall, it may recur without intervention because of a lack of evidence for the embolic source. Here, we report cases of BHS due to an artery-to-artery embolism with mural thrombus from the viewpoint of rotational VA occlusion by echography and the importance of hemodynamic assessment.

We describe a 45-year-old patient who was admitted because of sudden left hemianopia and dysesthesia of the left side of his body. Brain magnetic resonance imaging on admission showed multiple infarctions in the right medial temporal lobe and right thalamus. Magnetic resonance angiography showed no abnormalities in the bilateral VA. Collagen diseases and thrombophilia were excluded by laboratory tests. Despite administration of antiplatelets and argatroban, infarctions recurred twice in the left occipital lobe and bilateral thalamus. Duplex color-coded ultrasonography revealed the reduction of flow with neck rotation from the neutral position to the right (Fig. 1Ai). Furthermore, end-diastolic flow was absent in the left VA (Fig. 1Aii) without any ischemic symptoms, while the contralateral VA flow was intact at the same position (Fig. 1Aiii). Computed tomography

angiography showed focal enlargement of the left VA and thrombus in that location. Additionally, the left side of the posterior atlas arch was absent, and the left VA was abnormal (Fig. 1B). Cerebral angiogram denoted rotational occlusion in the left side of the VA. The patient was managed with a cervical collar, antiplatelet therapy with clopidogrel, and anticoagulation with warfarin. After confirming the disappearance of thrombus, posterior atlantoaxial fixation was performed. After the operation, the patient had no subsequent infarctions for 1 year.

We previously reported a 7-year-old patient with posterior circulation stroke who was diagnosed with BHS due to thromboembolic etiology [4]. Color-coded duplex ultrasonography revealed cessation of end-diastolic flow in the left VA on head rotation to the right without any symptoms, meanwhile the flow of the opposite side was normal. Transcranial color-coded duplex ultrasonography (TCCS) showed normal flow in the basilar artery and to-and-fro flow in the left intracranial VA (Fig. 1C). Angiography depicted rotational occlusion of the left VA with a mobile thrombus exactly at the compression site (Fig. 1D). Computed tomography revealed atlantoaxial subluxation causing the left VA occlusion. Other etiologies of stroke were excluded. After the operation of the C1–2 posterior fusion, he had no recurrence of stroke at the 10-year follow-up.

BHS was first reported by Sorensen in a case of Wallenberg syndrome due to VA spasm associating with head rotation. In this disease, the dominant VA is often compressed, resulting in ischemia because of limited collateral flow in the contralateral VA, which can be hypoplastic, stenotic, or occlusive [5]. However, the mechanism of BHS may involve factors other than hemodynamic changes. Though both of our cases presented with complete cessation of end-diastolic VA flow during neck rotation, they didn't show any symptoms due to posterior circulation ischemia. This may be because the opposite sides of the affected VAs were not hypoplastic or stenotic, so sufficient flow was guaranteed even when the head was rotated. Furthermore, we confirmed the normal flow in the unaffected side of the VA after neck rotation in both patients. In case 2, intact flow of the basilar artery and to-and-fro pattern in the affected VA supplying the cerebellum through the posterior cerebellar artery was confirmed by TCCS, suggesting sufficient

Abbreviations: BHS, bow hunter's stroke; VA, vertebral artery; ESUS, embolic stroke of undetermined source

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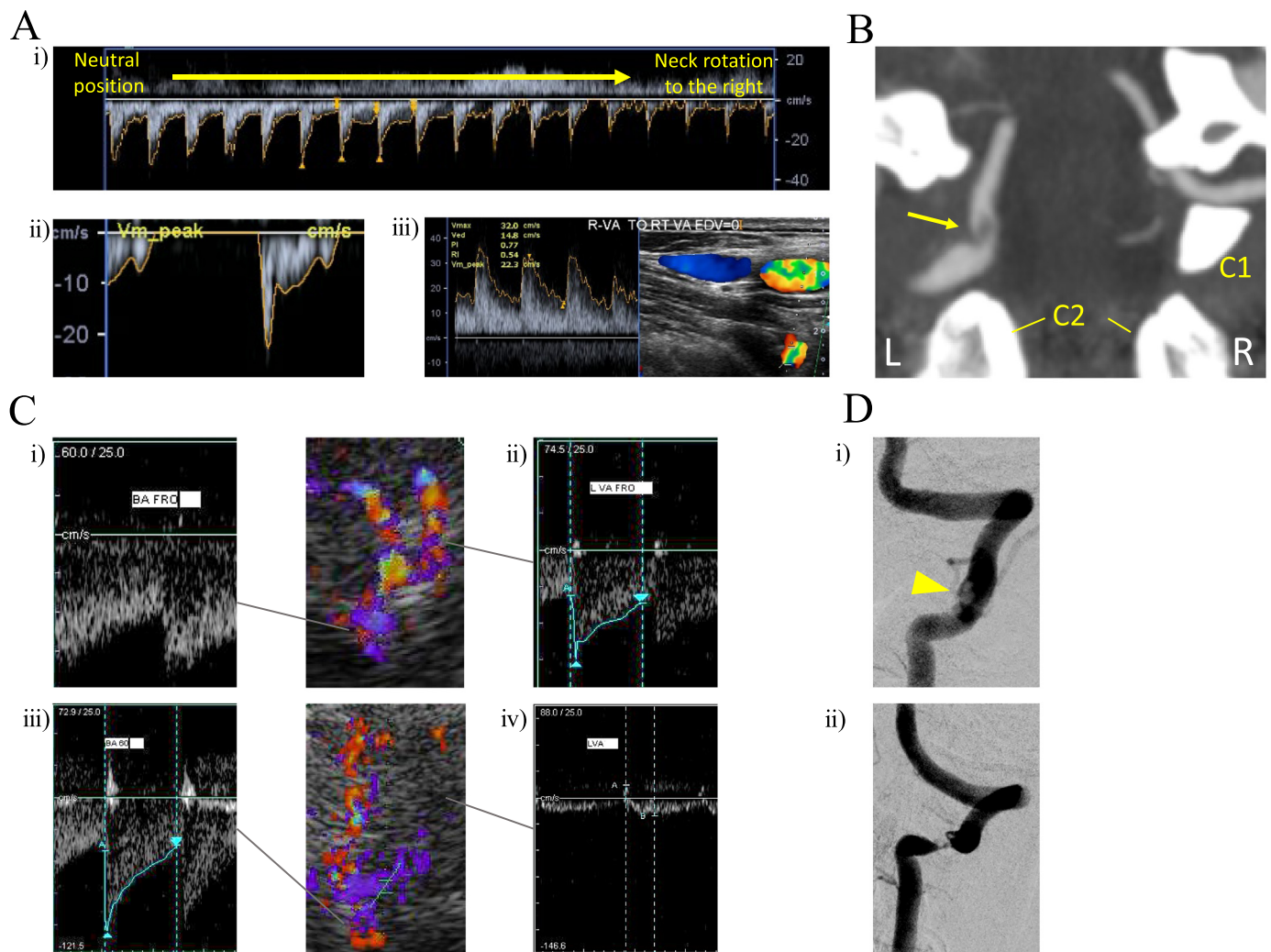


Fig. 1. In case 1, color-duplex ultrasonography of left VA showed reduction of blood flow with neck rotation from neutral position to the right (Ai). Finally, the end-diastolic flow ceased (Aii) while that of the right VA was intact (Aiii). Coronal CT angiography revealed a defect in left VA, suggesting mural thrombus (B, arrow). Also, the left side of posterior atlas arch absence and abnormal course of the left VA was also depicted. In case 2, transcranial color-coded duplex ultrasonography showed normal circulation in the basilar artery (Ci) and the left VA (Cii) at the neutral position. On neck rotation, normal flow in the basilar artery (Ciii) and reverse flow in the left VA (Civ) were seen. Angiogram depicted a clot in the left VA (Di). On neck rotation to the right, the left VA was occluded at C1-C2 level (Dii).

perfusion in the vertebrobasilar region. In a previous study of 1108 patients evaluated for flow changes in the extracranial VA during neck rotation, 55 (5.0%) had zero diastolic flow velocity, while only 28 patients presented symptoms associated with neck rotation [6]. These results indicated that BHS should not be excluded in cases of undetermined posterior circulation infarction, even when ischemic symptoms are not detected during head rotation. Recently, for ESUS, the use of an implantable loop recorder has been recommended to search for the embolic source. However, this is an invasive procedure; therefore, other factors should be fully excluded. For ESUS in the VA system, blood flow during cervical rotation, which represents a daily movement, should be evaluated using a less invasive modality with ultrasound.

Our two cases are also remarkable because mural thrombi are rarely detected in the compressed position, which is an obvious clue for artery-to-artery embolism. Though the exact pathology of BHS has not been elucidated, thrombus formation or thromboembolism has been proposed as one of the possible causes of BHS [4,7,8]. One of the speculative mechanisms of thrombus formation is that intimal injury or vascular damage by repetitive VA compression leads to platelet aggregation and activation of mediators involved in coagulation [9]. Our cases strongly supported this theory because the thrombus was detected

at the same location as the occlusion site. This indicated that temporal VA occlusion by head rotation leads not only to hemodynamic ischemia but also thrombus formation resulting in an embolic stroke.

In conclusion, we presented two cases of BHS that had mural thrombi in their VAs without any symptoms during neck rotation. These cases support the thromboembolic mechanism of BHS. In cases of posterior circulation infarction with unknown etiology, carotid ultrasound during neck rotation should be performed.

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Ethical standards

Our patient gave fully informed consent before publication.

Declaration of Competing Interest

None of the authors has any conflict of interest to disclose.

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