CASE REPORT

Juvenile Bow Hunter’s Stroke without Hemodynamic Changes

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Abstract: Bow hunter’s stroke (BHS) is a cerebrovascular disease caused by occlusion of the vertebral artery (VA) on head rotation. BHS is generally associated with hemodynamic changes, often leading to vertebrobasilar insufficiency symptoms, such as vertigo and faintness. Although artery-to-artery embolism has also been proposed as an underlying mechanism, it remains controversial. This report documents a case of BHS without hemodynamic changes. We describe a 26-year-old male patient who had VA occlusion on head rotation and repetitive infarction of thalami. He had an anomalous bypass of the VA and therefore no symptomatic hemodynamic changes. Thus, non-hemodynamic BHS should be considered in juvenile patients with vertebrobasilar stroke.

Keywords: BHS, bow hunter’s stroke, cerebrovascular disease, vertebral artery, hemodynamic changes
Introduction
Bow hunter’s stroke (BHS) is a cerebrovascular disease caused by occlusion of the vertebral artery (VA) on head rotation. BHS is associated with hemodynamic changes, often leading to vertebrobasilar insufficiency symptoms, such as vertigo and faintness. The mechanism of BHS frequently involves unilateral stenosis or hypoplasia of the VA, increasing susceptibility to hemodynamic changes on temporary occlusion of the contralateral VA. Recently, artery-to-artery (A-to-A) embolism has been suggested as another cause, which remains controversial. This report describes a young patient with BHS apparently without hemodynamic changes.

Patient
A 26-year-old man had a stroke involving the left thalamus, associated with dizziness and numbness in the right upper limb. Oral aspirin (81 mg/day) was started. Nine months later, dizziness and unconsciousness occurred without head rotation, and were found to be caused by an infarct in the right thalamus (Fig. 1A). Findings on routine MR angiography (MRA) were normal. He had congenital partial aplasia of the posterior arch of the atlas, but no apparent atlantoaxial subluxation. There was no evidence of thrombophilia or cardiac embolism. Duplex color-coded ultrasonography showed no end-diastolic flow in the right VA on head rotation to the left, unaccompanied by symptoms. Angiograms revealed rotational occlusion, associated with mild wall irregularity, and anomalous duplication of the VA. Three-dimensional CT angiography, a method that clearly visualizes both vascular lesions and surrounding structures, showed that the lower branch of the VA ran between the C1 and C2 vertebrae (Fig. 1F). The patient declined surgical treatment, but started antiplatelet therapy (cilostazol 200 mg/day) and voluntary avoidance of excessive head rotation. However, one year after the second attack MRI revealed an additional, asymptomatic infarct in the left thalamus. He then moved to another hospital, with no further clinical record available.

Discussion
To our knowledge, this is the first patient who had BHS with an anomalous bypass of the VA, suggesting the absence of apparently pathogenic hemodynamic changes. Arterial dissection was not evident on angiography, MRA, or MRI. Duplex ultrasonography on head rotation detected temporary VA occlusion associated with no symptoms. In addition, an angiographic finding suggested that the adequate vertebrobasilar flow was maintained from the anomalous bypass during head rotation. The occurrence of stroke without head rotation further discouraged a hemodynamic mechanism.

Mechanisms underlying non-hemodynamic BHS remain unclear; however, we speculate that it is associated with A-to-A embolism due to vascular damage caused by repeated VA compression. Supportive evidence for this speculation has been provided by our previous study showing that a juvenile patient with repetitive vertebrobasilar infarction and VA occlusion on head rotation had a mobile thrombus at the compression site. In our patient, the observed VA wall irregularity, a finding consistent with intimal damage, might agree with the A-to-A embolism mechanism. The sole involvement of the thalami may also support embolism in our patient. A large-scale ultrasonographic study (N = 1,108) showed that rotational VA occlusion was found in 5% of patients with possible atherosclerosis, 76% of whom had no rotation-induced symptoms. Thus, such patients may have the potential risk of stroke, if BHS is caused by A-to-A embolism.

Previous in vivo experiments showed that repeated vascular damage promotes platelet adhesion and activates various coagulation mediators. Clinically, vascular damage (thickness and sclerosis) was confirmed intraoperatively at the occlusion site in a patient with BHS. Alternatively, thrombus formation may be attributed to blood flow stasis in the VA, a well-accepted mechanism underlying hemodynamic BHS. However, thrombi form after 1 day of stasis, and our patient’s head was not fixed for such a long time. All of these facts supported the involvement of vascular damage in thrombus formation.

Various factors can cause head-rotation-induced VA occlusion in BHS. In fact, the VA is compressed by skeletal muscle or fascial bands at the transverse foramen of C6, by spondylotic osteophytes within the transverse foramina from C5 to C2, by strong membranous structures at the occipitoatlantal level, or by the atlantoaxial bones. Among them, the last
Non-hemodynamic BHS in Juvenile patients

Figure 1. A) T2-weighted MRI (Magnetom Sonata A.G., Siemens, Erlangen, Germany, TR/TE = 4000/123, ETL = 11) showed a right thalamic infarction in our patient. B), C) Angiograms showed mild wall irregularity (arrows) at the C1-2 level and the duplicated right vertebral artery (VA). D) The lower branch is compressed by head rotation to the left. E), F) CT angiograms showed normal courses of the left VA and the upper branch of the right VA (arrows). The lower branch ran between the C1 and C2 vertebrae (arrowhead). Arteries (pseudocolored red); those under other structures (light red).
one is involved in many cases of juvenile BHS, as in our patient.\textsuperscript{10} Many such patients had atlantoaxial subluxation often associated with head trauma or chiropractic manipulations,\textsuperscript{3,6} causing excessive slide on head rotation. In contrast, our patient had a rare VA duplication without atlantoaxial subluxation. The lower branch ran between the C1 and C2 vertebrae and was compressed even on moderate rotational joint movement. A similar aberrant course of the VA, but without duplication, was previously shown to cause BHS.\textsuperscript{12} Interestingly, concomitant abnormalities of arteries and cervical bones are often reported,\textsuperscript{13} as in our patient. In addition to specific factors in individual patients, greater neck mobility, generally seen in young people, may further damage the VA.

In conclusion, we demonstrated that BHS can occur without hemodynamic changes in the VA. Although the causal involvement of hemodynamic changes in BHS remains important, the possibility of non-hemodynamic change-related BHS should be considered in the diagnostic workup of patients with vertebrobasilar stroke, even in the absence of head-rotation-induced symptoms or abnormalities on routine MRA.

Disclosures
This manuscript has been read and approved by all authors. This paper is unique and is not under consideration by any other publication and has not been published elsewhere. The authors report no conflicts of interest.

References
1. Ryan GM, Cope S. Cervical vertigo. \textit{Lancet}, 1955;269:1355–8.
2. Toole JF, Tucker SH. Influence of head position upon cerebral circulation. Studies on blood flow in cadavers. \textit{Arch Neurol}, 1960;2:616–23.
3. Sorensen BF. Bow hunter’s stroke. \textit{Neurosurgery}. 1978;2:259–61.
4. Matsuyma T, Morimoto T, Sakaki T. Comparison of C1-2 posterior fusion and decompression of the vertebral artery in the treatment of bow hunter’s stroke. \textit{J Neurosurg}. 1997;86:619–23.
5. Netuka D, Benes V, Mikulik R, Kuba R. Symptomatic rotational occlusion of the vertebral artery—case report and review of the literature. \textit{Zentralbl Neurochir}. 2005;66:217–22.
6. Saito K, Hirano M, Taoka T, et al. Artery-to-Artery Embolism with a Mobile Mural Thrombus Due to Rotational Vertebral Artery Occlusion. \textit{J Neuroradiol imaging}. 2010 in press.
7. Fisher CM. The posterior cerebral artery syndrome. \textit{Can J Neurol Sci}. 1986;13:232–9.
8. Sakaguchi M, Kitagawa K, Hougaku H, et al. Mechanical compression of the extracranial vertebral artery during neck rotation. \textit{Neurology}. 2003;61:845–7.
9. Kawasaki T, Dewerchin M, Lijnens HR, et al. Mouse carotid artery ligation induces platelet-leukocyte-dependent luminal fibrin, required for neointima development. \textit{Circ Res}. 2001;88:159–66.
10. Grossmann RI, Davis KR. Positional occlusion of the vertebral artery: a rare cause of embolic stroke. \textit{Neuroradiology}. 1982;23:227–30.
11. Hanakita J, Miyake H, Nagayasu S, Nishi S, Suzuki T. Angiographic examination and surgical treatment of bow hunter’s stroke. \textit{Neurosurgery}. 1988;23:228–32.
12. Shimizu S, Yamada M, Takagi H, Fujiy K, Kan S. Bow hunter’s stroke associated with an aberrant course of the vertebral artery—case report. \textit{Neuroradiology}. 1999;39:867–9.
13. Floemer F, Magerkurth O, Jauckus C, Lutscheg J, Schneider JF. Klippel-Feil syndrome and Sprengel deformity combined with an intraspinal course of the left subclavian artery and a bovine aortic arch variant. \textit{AJNR Am J Neuroradiol}. 2008;29:306–7.

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