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Case Report

Anatomical and surgical considerations for Bow Hunter's syndrome in an elderly patient ☆,☆☆

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ABSTRACT

Bow Hunter's syndrome (BHS) is an uncommon condition characterized by impingement of one of the two vertebral arteries induced by cervical rotation, causing symptomatic vertebrobasilar insufficiency of the posterior cerebral circulation. We report a case of BHS in an 84-year-old male. Two months following a motor vehicle accident, the patient presented to an urgent care facility with subsequent transfer to the emergency department with complaints of lightheadedness upon right-lateral head movement. A cerebral angiogram demonstrated mild focal stenosis in the dominant left vertebral artery at the C2 level when in neutral position with significant worsening of the stenosis in the right-lateral head position with absent anterograde flow, consistent with BHS. Resultantly, the patient was referred for neurosurgery and successfully underwent placement of right-sided C2-C4 postero-lateral instrumentation and left-sided C2-C3 laminar screws projected towards the right side. This case highlights the importance of imaging in BHS diagnosis and guidance for treatment, as well as the need for a surgical standard of care for BHS patients.

Introduction

Bow Hunter's syndrome (BHS) is a rare neurovascular abnormality characterized by rotationally induced impingement of one of the two vertebral arteries, subsequently resulting in symptomatic vertebrobasilar insufficiency within the posterior cerebral circulation [1]. Typically, BHS patients have one dominant vertebral artery, which when compressed, leaves one artery that is insufficient to provide adequate flow [2]. Clinical symptoms arise when the head or neck is rotated or extended to resemble the head position assumed when an archer aims their bow, hence the origin of the name [2]. In

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patients with BHS, this head position results in partial to complete dynamic occlusion of the dominant vertebral artery at the atlantoaxial or subaxial level of the cervical spine (C1-C7) leading to vertebrobasilar ischemia. This pathogenesis can be attributed to a repetitive shear force and resultant hemodynamic or thromboembolic endothelial tissue damage of the arterial walls and potential stasis of blood flow within the affected vertebral artery [3,4]. BHS most commonly results from an array of factors including, but not limited to, hypertrophic osteophytes, disc herniation, fibrous bands or tumors, spondylosis of the cervical spine, head or neck trauma, and vertebral artery compromise in prior surgical interventions [5–11].

Of the commonly presenting symptoms of vision impairment, vertigo, syncope, nausea, and headache, most are self-resolved upon repositioning the head to a neutral position [3–6]. Permanent complications such as Wallenberg syndrome, characterized by nausea, vomiting, hiccups, and vertigo have been documented secondary to BHS diagnosis, though such findings are uncommon [2,12]. While BHS may present during any stage of life, existing literature demonstrates that the condition is most common among men ages 50-70, all of whom share some combination of multiple chronic conditions including hypertension, hyperlipidemia, osteoarthritis, diabetes, smoking, and coronary artery disease [4–6].

Classically, BHS is conservatively treated through neck immobilization, conscious avoidance of head rotation and extension, and anticoagulant medications [1,2]. Surgical intervention offers more permanent solutions involving osseous decompression and stent placement in the contralateral unaffected vertebral artery [13,14]. Treatment of BHS, however, is limited in its scope due to the rarity of the condition, and thus surgical intervention and its effects have not been widely explored.

We present a case of surgically treated BHS and examine the corresponding anatomical details of its presentation to further investigate the condition, evaluate treatment efficacy, and discuss possible developments in the standard of care for BHS treatment.

Case presentation

An 84-year-old male with a past medical history of type II diabetes mellitus, primary hypertension, and coronary artery stent placement presented to an urgent care facility and was subsequently put in a c-collar and sent to the emergency department for lightheadedness and near-syncope upon right-lateral head movement. The patient stated their lightheadedness and faintness began nine weeks prior, after being rear-ended in a high-speed motor vehicle accident. The patient denied headaches, vomiting, double vision, numbness, weakness in extremities, and neck pain. The patient's blood pressure was hypertensive at 182/100 and the physical exam was negative for facial droop, slurring of speech, and difficulty swallowing. Labs were unremarkable.

A CT head was performed which revealed no acute intracranial findings. A CTA neck revealed a dominant left vertebral artery with what appeared to be an intermittently occluded right vertebral artery at C1-C3 and C6-C7 (Fig. 1). The patient was subsequently referred to neurointerventional radiology and underwent a diagnostic cerebral angiogram that showed mild focal stenosis in the dominant left vertebral artery at the C2 level when in neutral position with significant worsening of the stenosis in the right-lateral head position with absent anterograde flow (Figs. 1 and 2). The right vertebral artery was hypoplastic with multi-segmental stenosis and was non-contributory to the vertebrobasilar system (Fig. 3). There was a bilateral absence of posterior communicating arteries, indicating a single dominant left vertebral

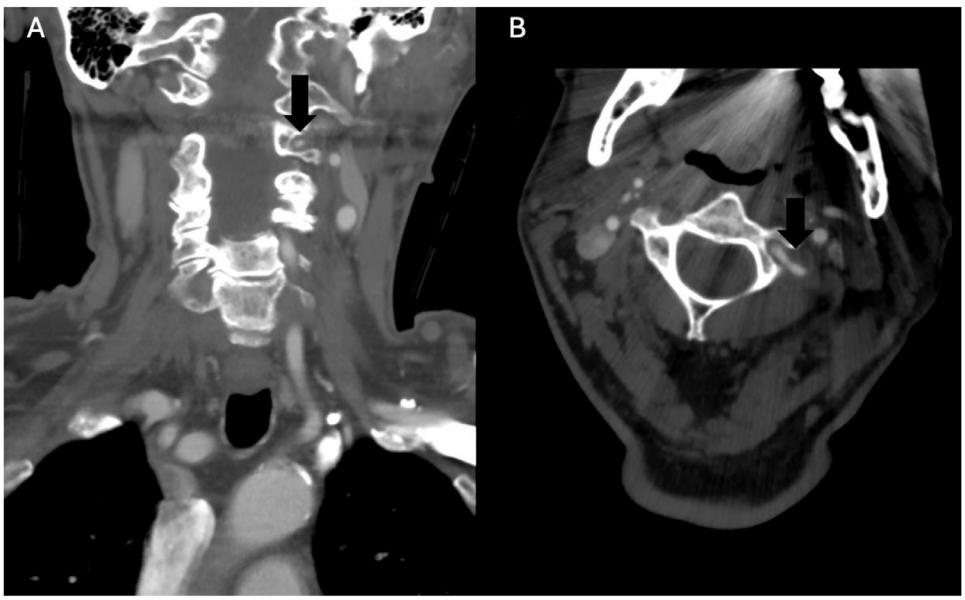

Fig. 1 – Both images show mild stenosis of the left vertebral artery. (A) Coronal CT angiogram shows mild stenosis of the left vertebral artery at the level of C1-C2. (B) Axial CT angiogram shows stenosis on the C1-2 segment.

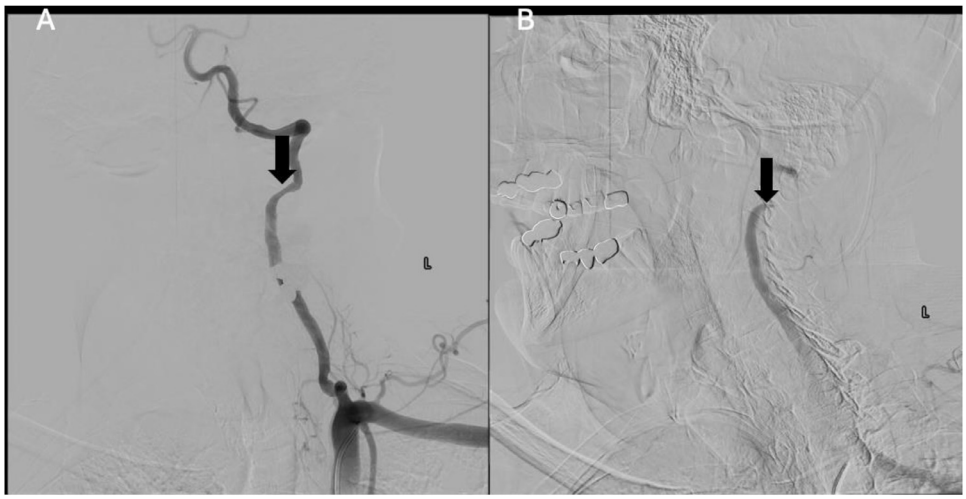

Fig. 2 – (A) The left vertebral angiogram during neutral position shows mild stenosis of the left vertebral artery at the C1-C2. (B) The left vertebral angiogram during right lateral decuditis position shows severe left vertebral stenosis at the level of C1-C2.

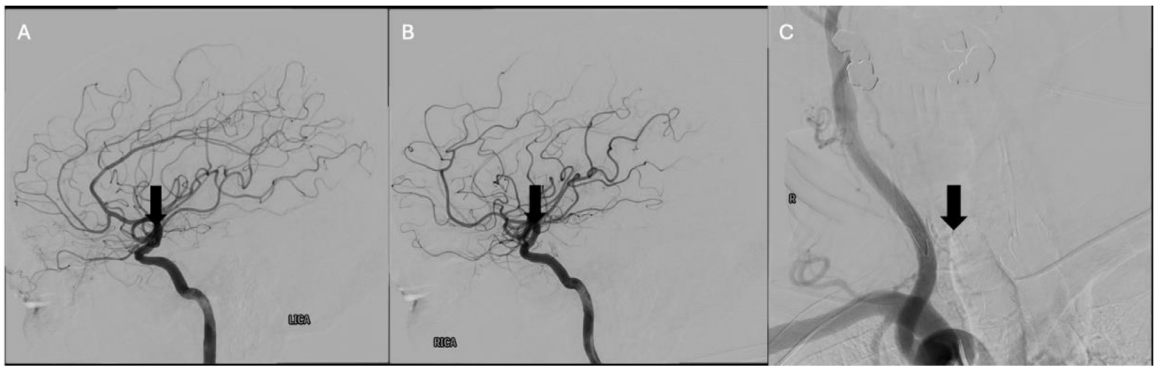

Fig. 3 – (A) A left internal carotid artery angiogram shows absence of the left posterior communicating artery. (B) The right internal carotid artery angiogram shows the absence of the right posterior communicating artery. (C) The subclavian angiogram shows absence of flow through the right vertebral artery.

blood supply to the posterior circulation that supplied both ipsilateral and contralateral arterial branches of the vertebrobasilar system (Fig. 3). The diagnostic angiogram therefore revealed occlusion of the left vertebral artery upon right-lateral head movement and was consistent with the patient's symptoms and the diagnosis of Bow Hunter's syndrome.

The patient was referred for neurosurgery and underwent placement of right-sided C2-C4 postero-lateral instrumentation and left-sided C2-C3 laminar screws projected towards the right side (Fig. 4). Placement was achieved with the use of Stealth stereotactic guidance. Postero-lateral arthrodesis using allograft and BMP at C2-C3 and C3-C4 was performed. Placement of screws was confirmed with intraoperative O-arm. Intraoperative motor and sensory evoked potentials for both pre and post positioning demonstrated no apparent complications, indicating the patient tolerated the procedure well without any neurological deficits or uncontrolled pain. The patient was discharged in stable condition to home with close follow-up.

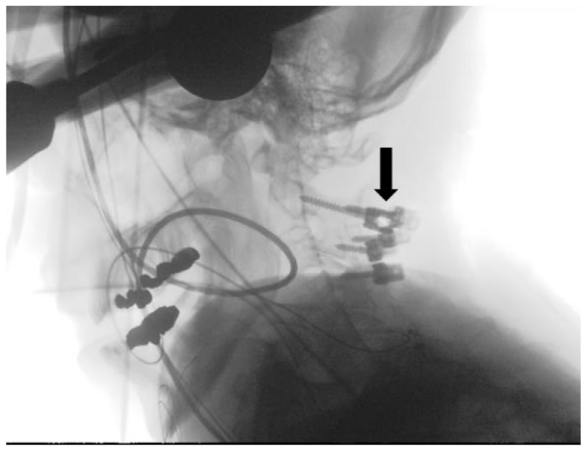

Fig. 4 – The X-ray perioperative X-ray cervical spine shows posterior fusion of the C2, C3, and C4 segments.

Discussion

The posterior cerebral circulation receives supply from two independent vessels termed the right and left vertebral arteries, each of which originates from its corresponding right or left subclavian artery. If one of these arteries is non-contributory, the contralateral artery can provide sufficient flow to the posterior inferior cerebellar artery in the majority of cases [1]. Each artery laterally navigates through the 6 transverse foramina of the cervical vertebrae, courses through the superior vertebral notch located on the surface of the arch of the atlas, and transmits both medially and laterally into the dura mater where both converge to form the basilar artery and supply the Circle of Willis. This arrangement in turn provides collateral blood flow between the anterior and posterior cerebral circulations and protects the brain from ischemia in the event of vessel damage or stenosis by allowing the brain to receive supply from the patent carotid or vertebral arteries and their branches to compensate for hypoperfusion. Due to their course through the foramina of the transverse process, the vertebral arteries are susceptible to rotational impingement as characterized in BHS [5,15].

Additionally, this anatomy allows for some collateral circulation of vertebrobasilar blood flow should a patient develop unilateral stenotic occlusion in one of the two vessels [1,15]. The presence of one or more posterior cerebral arteries allows for a collateral pathway to the basilar artery in case the vertebral arteries are occluded or stenosed. It should be noted, however, that of the two vertebral arteries, the left artery is often dominant compared to its contralateral counterpart and contributes a larger proportion of perfusion volume to the posterior cerebral circulation; BHS most often affects this larger vertebral artery, especially in cases where the contralateral counterpart is hypoplastic [5].

In this case, the right vertebral artery was completely non-contributory to vertebrobasilar flow, whereas the left vertebral artery was contributory to the vertebrobasilar system in neutral head position, but non-contributory in right-lateral head position. In most cases, collateral flow from the anterior cerebral circulation can provide flow via the posterior communicating arteries; however, this patient had a bilateral absence of these collateral pathways–a rare phenomenon. Therefore, prolonged right lateral head movement and subsequent occlusion of blood flow through the left vertebral artery is potentially devastating and can cause brainstem infarct in this patient.

The brain stem, particularly the ascending reticular activating system (ARAS) of the brain stem, plays a role in controlling consciousness. These structures go through the pontomesencephalic tegmentum. Loss of consciousness can occur for various reasons, particularly the loss of blood supply to the brain stem [16]. Loss of blood supply to the brain stem can occur as a result of several factors including dissection of a blood vessel, thrombotic stroke, external pressure to arteries due to tumors, and trauma from external sources. This case explores an interesting phenomenon where an 84-year-old male experienced near syncope and lightheadedness due to salient impingement of the right vertebral artery when turning the head. A CT scan confirmed no posterior communicating arteries and showed the left vertebral artery to be contributing the main collateral blood supply to the brain.

Common symptoms of BHS include dizziness and fainting spells. This occurs because of the rotation of the head or neck, which results in the reversible occlusion of the vertebral artery and lack of blood to the brain stem [4]. There are two types of Bow Hunter's syndrome: primary BHS and acquired BHS. Primary BHS includes abnormal bony structures, disc herniations, and ligaments leading to stenosis of the vertebral arteries and exacerbated by head rotation and resultant mechanical occlusion. Acquired BHS includes complications in previous surgeries, including issues in the vertebral arteries due to treatment for aneurysms, injuries in the head or neck, and much more [1]. The CT scan of the cervical spine does not demonstrate any bone abnormalities. The CT angiogram demonstrates mild narrowing at the vertebral artery just distal to the C1 foramina. Therefore, ligamentous cause is assumed. The transient occlusion might have occurred because of fibrous band tethering in the vertebral artery at the transverse foramina of the junction between the Cl and C2 segments [17]. The dynamic DSA demonstrates near complete occlusion of the left vertebral artery when the patient rotates the head to the right side, confirming the diagnosis of BHS.

Common treatments of BHS include posterior fusion and decompression in the C1 and C2 segments. However, a negative effect of posterior fusion is a severe reduction in atlantoaxial rotational movement. Decompression is therefore generally considered a better option as it does not limit head movement though more research needs to be conducted as it is unclear whether decompression produces significant relief for patients [14]. In this case, the patient underwent posterior fusion, which was safer than decompression.

There is no universally accepted standard of care tailored for patients with Bow Hunter's syndrome due to the rarity of cases and limited literature discussing its presentation. In many patients with this condition, it is difficult to pinpoint the cause of BHS. BHS is extremely rare, thus there is not enough research to make definitive conclusions yet [17]. Further research must be conducted to analyze the causes of BHS and effective treatments.

Patient consent

Consent was obtained from the patient for publication of this case report.

REFERENCES

[1] Duan G, Xu J, Shi J, Cao Y. Advances in the pathogenesis, diagnosis and treatment of Bow Hunter's syndrome: a comprehensive review of the literature. Intervention Neurol 2016;5(1-2):29–38. doi:10.1159/000444306.
[2] Taylor WB, Vandergriff CL, Opatowsky MJ, Layton KF. Bowhunter's syndrome diagnosed with provocative digital

subtraction cerebral angiography. Proceedings (Baylor University. Medical Center) 2012;25(1):26–7. doi:10.1080/08998280.2012.11928776.
[3] Cornelius JF, George B, N'dri Oka D, Spiriev T, Steiger HJ, Hänggi D. Bow-hunter's syndrome caused by dynamic vertebral artery stenosis at the cranio-cervical junction–a management algorithm based on a systematic review and a clinical series. Neurosurg Rev 2012;35(1):127–35. doi:10.1007/s10143-011-0343-4.
[4] Jost GF, Dailey AT. Bow hunter's syndrome revisited: 2 new cases and literature review of 124 cases. Neurosurg Focus 2015;38(4):E7. doi:10.3171/2015.1.focus14791.
[5] Rastogi V, Rawls A, Moore O, Victorica B, Khan S, Saravanapavan P, et al. Rare etiology of Bow Hunter's syndrome and systematic review of literature. J Vasc Intervent Neurol 2015;8(3):7–16.
[6] Zaidi HA, Albuquerque FC, Chowdhry SA, Zabramski JM, Ducruet AF, Spetzler RF. Diagnosis and management of bow hunter's syndrome: 15-year experience at barrow neurological institute. World Neurosurg 2014;82(5):733–8. doi:10.1016/j.wneu.2014.02.027.
[7] Ding D, Mehta GU, Medel R, Liu KC. Utility of intraoperative angiography during subaxial foramen transversarium decompression for bow hunter's syndrome. Intervent neuroradiol 2013;19(2):240–4. doi:10.1177/159101991301900215.
[8] Anaizi AN, Sayah A, Berkowitz F, McGrail K. Bow hunter's syndrome: the use of dynamic magnetic resonance angiography and intraoperative fluorescent angiography. J Neurosurg. Spine 2014;20(1):71–4. doi:10.3171/2013.9.SPINE121019.
[9] Thomas B, Barreau X, Pointillart V, Sibon I, Renou P. Endovascular embolization of a nondominant vertebral artery compressed by an osteophyte to prevent recurrence of vertebrobasilar infarctions. J Stroke Cerebrovasc Dis 2015;24(9):e257–9. doi:10.1016/j.jstrokecerebrovasdis.2015.05.033.
[10] Healy AT, Lee BS, Walsh K, Bain MD, Krishnaney AA. Bow hunter's syndrome secondary to bilateral dynamic vertebral artery compression. J Clin Neurosci 2015;22(1):209–12. doi:10.1016/j.jocn.2014.05.027.
[11] Ikeda DS, Villelli N, Shaw A, Powers C. Bow hunter's syndrome unmasked after contralateral vertebral artery sacrifice for aneurysmal subarachnoid hemorrhage. J Clin Neurosci 2014;21(6):1044–6. doi:10.1016/j.jocn.2013.10.005.
[12] Matsuyama T, Morimoto T, Sakaki T. Bow Hunter's stroke caused by a nondominant vertebral artery occlusion: case report. Neurosurgery 1997;41(6):1393–5. doi:10.1097/00006123-199712000-00030.
[13] Horowitz M, Jovin T, Balzar J, Welch W, Kassam A. Bow hunter's syndrome in the setting of contralateral vertebral artery stenosis: evaluation and treatment options. Spine 2002;27(23):E495–8. doi:10.1097/00007632-200212010-00015.
[14] Matsuyama 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. J Neurosurg 1997;86(4):619–23. doi:10.3171/jns.1997.86.4.0619.
[15] Hanakita J, Miyake H, Nagayasu S, Nishi S, Suzuki T. Angiographic examination and surgical treatment of bow hunter's stroke. Neurosurgery 1988;23(2):228–32. doi:10.1227/00006123-198808000-00018.
[16] Sandhu S, Soule E, Fiester P, Natter P, Tavanaiepour D, Rahmathulla G, et al. Brainstem diffuse axonal injury and consciousness. J Clin Imaging Sci 2019;9:32. doi:10.25259/JCIS-11-2019.
[17] Go G, Hwang SH, Park IS, Park H. Rotational vertebral artery compression : Bow Hunter's syndrome. J Korean Neurosurg Soc 2013;54(3):243–5. doi:10.3340/jkns.2013.54.3.243.