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Case Report

Uncommon cause of vertigo: Bow Hunter syndrome

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ABSTRACT

Rotational vertebral artery syndrome, recognized as Bow Hunter syndrome (BHS), was a rare but potentially debilitating condition characterized with triggered vertigo, dizziness, and syncope. It results from dynamic vertebral artery (VA) stenosis due by compression from adjacent anatomical structures during specific neck movements. It may be easily overlooked even with the use of advanced imaging techniques because its symptoms closely resemble other episodic vestibular disorders, including benign paroxysmal positional vertigo. However, BHS may cause posterior circulation stroke. The only definitive diagnostic method for BHS is provocative neck movement testing, with provocative digital subtraction angiography (DSA) as the gold standard. A 61-year-old female had vertigo, dizziness, and presyncope which was triggered by rotating her neck to the left. Angiographic head-to-neck CT showed cervical spondylosis with osteophytes at C5-C6 vertebral level without any blood vessel abnormalities. Conventional DSA discovered hypoplastic right VA. Following provocative neck movement to the left, significant stenosis of left VA was detected. She was advised to immobilized her neck and consulted to neurosurgeons for the consideration of surgical decompression. Although rare, BHS should be considered as a potential cause of vertigo. Normal vascular assessment from ancillary tests did not rule out BHS. Recognizing BHS was crucial for its comprehensive evaluation of provocative physical examination to provocative DSA. Treatment options include neck immobilization, anticoagulation therapy, and surgical intervention, with or without dynamic intraoperative angiography.

Keywords: Bow hunter syndrome, Episodic vestibular syndrome, Provocative neck movement, Rotational vertebral artery syndrome, Vertigo

INTRODUCTION

Vertigo and dizziness were some of the most common symptoms reported worldwide with lifetime prevalence of 17-30%.[1] It was mostly due to benign etiology, including the triggered episodic vestibular syndrome (EVS) of benign paroxysmal positional vertigo (BPPV), spontaneous EVS of Meniere's disease and vestibular migraine, acute vestibular syndrome (AVS) of vestibular neuritis, or due to the complication of chemicals including bilateral vestibulopathy.[1,2] However, it may also a sign of serious central nervous system (CNS) lesion including stroke, with an estimated number of 3-5% of all vertigo symptoms. The misdiagnosis of vertigo of central vertigo (cerebellar stroke) as peripheral origin may result in increased morbidity and mortality of 33% and 44%, respectively, whereas the overdiagnosis of peripheral vertigo as central origin has resulted in inappropriate hospital admission, unnecessary sophisticated imaging, and long hospitalization length of stay, which contributed to an estimated $1 billion expenses per year.[1,3] In addition, brain diffusion-weighted MRI may miss 15-20% cases of stroke with onset of less than 24-48 hours.[3] Therefore, clinical judgement with appropriate physical examination was elusive in accurately managing this symptom. Rotational vertebral artery syndrome, commonly recognized as Bow Hunter syndrome (BHS), was a vertebrobasilar insufficiency (VBI) state due to dynamic

compression of vertebral artery (VA) during rotational neck position. This syndrome was rare and firstly described in 1978 from a bow hunter during archery practice. Specific rotational movement of the neck from a person with underlying bony structure abnormalities, including spondylosis, osteophyte, herniated disk, or tumor, may mechanically compress VA. This dynamic mechanical compression therefore produces steno-occlusion of VA which manifests from the more common transient hypoperfusion syndrome until the rarer but more dangerous vessel injury with thromboembolism. Transient hypoperfusion syndrome included syncope, vertigo, dizziness, headache, or blurry vision. Thromboembolism following vessel injury may manifest as posterior circulation stroke including dysarthria, gait abnormalities, brainstem findings, Wallenberg syndrome, visual field deficits, cerebellar signs, locked in syndrome, etc.[4–8]

The key pathophysiology of dynamic mechanical compression made the diagnosis of BHS challenging and commonly missed because the pathology was discovered only following specific rotational movement.[4–8] Increased awareness and suspicion of BHS was therefore paramount for clinician as it will further direct to performing the provocative physical examination simulating the dynamic mechanical compression. This case report described elderly with BHS of left VA due to ipsilateral spondylosis of C5-C6 vertebra, which was ultimately diagnosed by provocative digital subtraction angiography (DSA).

CASE REPORT

A 61-year-old female presented to the clinic with recurrent episodic vertigo lasting three days. It was triggered by turning her neck to the left and alleviated when she returned her head to neutral or right position. She also experienced few episodes of dizziness following the similar triggering action. There was no accompanying neck pain, hypertension, diabetes, or history of head and neck trauma. Her physical and neurological examination were unremarkable. Spontaneus nystagmus was negative, Dix Hallpike maneuver on either side did not induce nystagmus, and Romberg test was negative.

Due to the suspicion of vertebrobasilar insufficiency, a head-to-neck angiographic CT scan was performed, revealing cervical spondylosis with osteophyte at C5-C6 vertebral level. Conventional DSA showed right VA hypoplasia (Figure 1), but it was not fully account for her symptoms. No vertebral stenosis was described. Provocative DSA was further performed, which demonstrated significant stenosis of V1 segment of left VA at the level of C5-C6 vertebra only following her head turning to the left. She was advised against turning her head to the left and discharged thereafter. A referral to neurosurgeon was planned for consideration of excising the corresponding osteophytes at the C5-C6 vertebral level to release the dynamic compression at the left VA. However, the patient planned to have the surgery elsewhere.

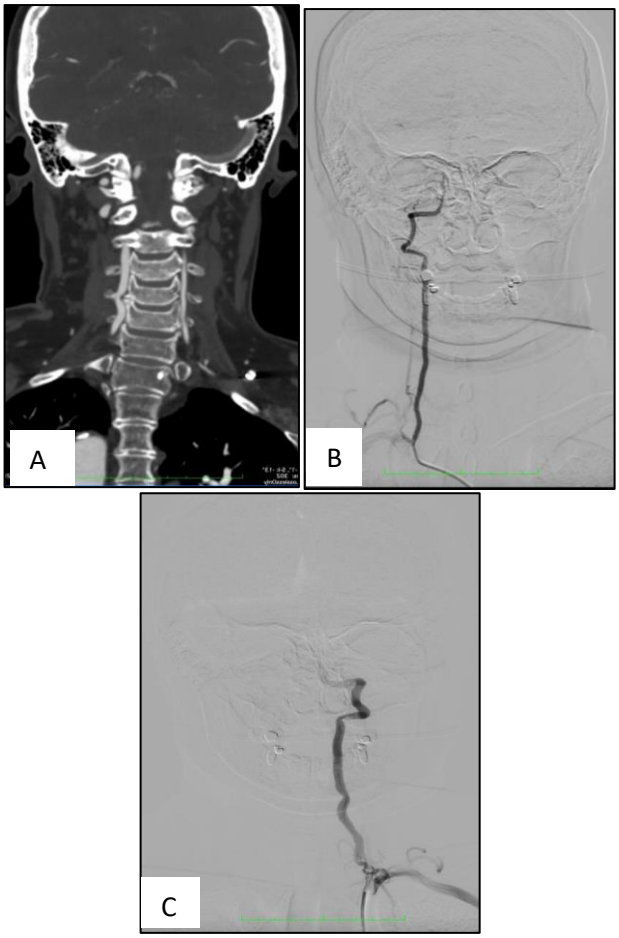

Figure 1: Ancillary examination of (A) head-to-neck angiography CT scan revealed cervical spondylosis with osteophytes left of C5-C6 vertebra, (B) digital subtraction angiography of right vertebral artery demonstrated hypoplasia, supported with the insufficient contrast filling at the basilar artery, (C) digital subtraction angiography of left vertebral artery showed normal finding.

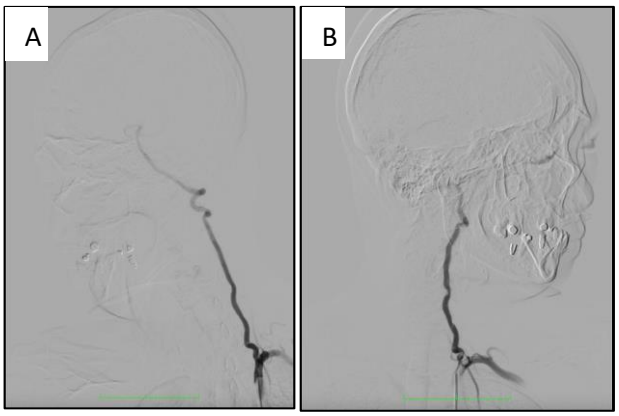

Figure 2: Provocative digital subtraction angiography of left vertebral artery when (A) rotated the head to the right, demonstrating normal finding, (B) rotated to the left, revealing significant left vertebral stenosis corresponding to the level of C6-C7 vertebra.

DISCUSSION

Balance is the "sixth sense" of human. It was the harmonious output from the combination input by vestibular, visual, and proprioceptive sensation along with the coordination system by cerebellum and brain cortices. Vertigo and dizziness occurred when there was decompensated imbalance due to different input of least one of those systems. This report presented BHS, which is a rare case of central triggered episodic vertigo resulted from dynamic VBI. Provocative physical examination and provocative DSA were not a novel technique, but the rarity of BHS and its usefulness of those technique required further awareness increase in detecting and managing BHS.

Vertigo and dizziness may be classified as AVS or EVS, which was further classified with spontaneous or triggered. The subject in this case had triggered EVS due to her vertigo characteristics of waxing when being triggered and totally waning when the trigger was removed. Triggered EVS which may due to peripheral origin including BPPV or central origin including central paroxysmal positional vertigo (CPPV). Dix Hallpike manoeuvre may aid in this diagnosis by triggering the vertigo and providing important description of vertigo. Peripheral origin of triggered EVS using Dix Hallpike was described as nystagmus with latencies before its occurrence, having horizontal unidirectional properties, lasted less than a minute, and fatigable. The subject in this case did not develop nystagmus, which therefore did not consistent with typical BPPV.[4]

The only patognomonic clinical finding raising suspicion of BHS in this case was the persistence elicitation of vertigo, dizziness, and syncope following left-head rotational movement.[4] Bow Hunter syndrome was commonly misdiagnosed due to its similar presentation with some other etiologies of triggered EVS. The commonly reported symptoms of BHS ranged from transient symptoms of vertigo, dizziness, presyncope to syncope, blurry vision, nausea, dysarthria, dysphagia, headaches, gait disturbance, until permanent symptoms of posterior circulation stroke.[8] The typical physical examination finding of BHS was horizontal nystagmus directed towards the compressed VA along with nausea, syncope, and other symptoms reported above which may appear immediately or slightly delayed. When the head was returned to normal position, there will be transitional downward nystagmus which evolved to transient horizontal nystagmus to the contralateral side for a while.[10] The finding of transitional downward nystagmus may be strong evidence to transient dysfunction of the central vestibular area due to ischemia. Besides BHS, transitional downward nystagmus may also be present in apogeotropic posterior BPPV.[10] The nystagmus in BHS may be subtle and under detected, as what thought to occur in this case.

Bow Hunter syndrome was a condition of VBI due to dynamic stenosis of VA following neck rotation of more than 30-450. Vertebral artery on each side courses from posteriorly from its origin at subclavian artery (V1 segment) through the ipsilateral transverse foramen of cervical vertebral bone at the level of C6 to C2 (V2 segment), then courses posteromedial around the atlas of C1 and atlanto-occipital junction (V3 segment) until entering the foramen magnum to intracranial (V4 segment) and joins the contralateral VA to become basilar artery.[8,9] This normal anatomical design provided collateral to ensure sufficient blood flow to posterior brain circulation while one of the VA was disrupted. It was therefore necessary for bilateral VA flow to be disrupted to observe the BHS.[7,9]

The risk factors of BHS included anatomical changes inside or adjacent of corresponding VA, including the external anatomical changes of hypertrophic osteophytes commonly from uncovertebral joints, skeletal hyperostosis, disc herniation, cervical spondylosis, neck muscle hypertrophy, fibrous bands, external compression by adjacent tumor, rheumatoid subluxation, traumatic cervical injury, chiropractic manipulation, surgical procedure (fixation, correction of subclavian aneurysm, or VA sacrifice), or internal vascular abnormalities including atherosclerosis or VA dissection.[7–9] These risk factors did not directly obstruct the VA during static position, but it produced dynamic obstruction when the neck rotation induced movement of associated pathology towards the VA.[7,9] Male of fifth to seventh decade age was slightly more susceptible to BHS presumably due to the increased potency for formation of osteophytes and the more active activities involving weightlifting. The report of elderly female in this case provided information that female may also be susceptible to BHS.[8,11]

While compression along every vertebral segments may be possible, compression at the level of C1 or C2 vertebral bone, classified as atlantoaxial type by Cornelius et al,11 was most common pathology reported in BHS.[8–10] It was associated with the relatively immobile segment of VA at the V3 segment.[9] This case reported the second possible pathology, which was the dynamic VA stenosis at C5-C6, recognized as sub axial type.[11] Some reports hypothesize that dynamic VA stenosis at higher cervical level may be induced with contralateral head movement whereas those at lower cervical level below C3 may be induced with ipsilateral head movement as observed in this case.[10-12] However, another report described that this was not entirely reliable.[9] Dynamic VA stenosis may also due to multiple-site stenosis, described as mixed type.[9,11] The left VA was more dominant in estimated half of the population, which contributed with its increased susceptibility for dynamic stenosis in BHS.[7,9] The compensation of right VA was limited due to its smaller caliber (hypoplasia), with increased collateral failure if there was stenosis or fenestration inside.[9] The combination of dominant VA dynamic stenosis and uncompensated nondominant VA was responsible for the transient symptoms of BHS during corresponding neck movement. This subject in this case had hypoplastic right VA that

could not adequately compensate the reduced blood flow during dynamic VA stenosis, which result in the development of transient vertigo, dizziness, and syncope.

In addition to transient hemodynamic disturbance, there may rarely be repetitive injury of dynamic VA stenosis, which may injure the vessel, result in endothelial injury, and predispose to local atherosclerotic event. This may ultimately form VA thromboemboli, which made BHS potentially dangerous. This pathogenesis preclude the more permanent manifestation of posterior circulation stroke including Wallenberg syndrome (lateral medullary syndrome) or VA dissection.[7–9] Common differential diagnosis included (conventional) VA occlusion, subclavian steal syndrome, vasculitis, cardiac or paradoxical embolism, reversible cerebral vasoconstriction syndrome, lactic acidosis, and mitochondrial encephalopathy.[8,9] This possibility had been excluded in this case with the finding of nonstenotic bilateral VA using conventional DSA.

The normal finding of conventional imaging technique including carotid duplex, transcranial doppler, angiographic CT or MR, and even the gold standard vascular imaging of DSA further obscures the diagnosis of BHS. The dynamic VBI in BHS can only be diagnosed by reproducing the symptoms after provocative movement of the head. It can also be objectively diagnosed by detecting the flow-loss inside VA using ultrasound or determining corresponding VA stenosis using angiographic CT, MR, or imaging solely following provocative neck rotation. Provocative DSA was the gold standard of BHS due to its ability to exclude "conventional" stenosis, detecting the anatomical and flow disturbance following provocative neck movement, and assessing the collateral flow as what performed in this case. Risks to be considered was the minimal invasive properties of DSA.[7–9,12,13]

Treatment approach of BHS included conservative with or without surgical approach. Conservative approaches included immobilizing head movement using cervical collar or brace as well as the preventing neck movement to the angle that produced BHS.[8,9] Antiplatelet and anticoagulation were also reported to be beneficial especially in those with VA dissection, but its benefit was unknown for the transient symptoms.[9] Surgical approach for the anatomical pathology, e.g. surgical decompression of VA, posterior cervical canal fusion, or corrective fusion of C1-C2, was preferred as it was more permanent to decompress VA.[8–10] Dynamic intraoperative angiography, with consideration of intraarterial remodelling balloon, during surgery may also be considered to ensure the restoration of blood flow.[9,14] Another alternative may be endovascular stenting of bilateral VA or coil embolization for symptomatic nondominant VA with patent dominant VA, but standard guideline was still not available due to its rarity.[8,9] Individualized treatment with multidisciplinary approach should be considered in managing BHS.[8–10]

This report provided further importance regarding awareness of BHS as a potential differential diagnosis for triggered EVS as it may be similarly presented as BPPV. Provocative neck movement was still pathognomonic for ultimately diagnosing BHS. Further study was needed to consider the provocative test as part of routine examination alongside Dix Hallpike in evaluating the etiology of triggered EVS.

CONCLUSION

Bow Hunter syndrome, albeit rare, must be considered as one possible etiology of triggered EVS. The specific trigger of lateral neck movement was the only key findings to suspect BHS, which opened the possibility to perform specific provocative tests. In addition, BHS may also be considered in posterior circulation stroke with specific repetitive trigger of BHS, especially if there were unknown cerebrovascular risk factors. Normal result of vascular assessment from ancillary findings did not exclude BHS, thus awareness regarding BHS was elusive as the primary step to assess the subjective vertigo complaints more thoroughly.

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REFERENCES

1. Kovacs E, Xiaoting W, Grill E. Economic burden of vertigo: a systematic review. Health Econ Rev. 2019;9:37.
2. Ghosh A, Dorasala S. Epidemiology of benign paroxysomal positional vertigo (BPPV) and risk factors for secondary BPPV: a population-based study. Egyptian J of Otolaryngol. 2023;39(1):45-8.
3. Tehrani ASS, Kattah JC, Kerber KA, Gold DR, Zee DS, Urrutia VC, et al. Diagnosing stroke in acute dizziness and vertigo pitfalls and pearls. Stroke. 2018;49(3):788–95.
4. Kühn AL, McGillicuddy GT, Singh J. Bow hunter syndrome. Can Med Assoc J. 2022;194(43):1486.
5. Hong IS, Jung EH, Kim KH, Kwon HJ, Choi SW, Kim SH, et al. Rotational Vertebral Artery Syndrome (Bow Hunter's Syndrome): A Rare Differential Diagnosis in Patients with Syncope. Korean J Neurotrauma. 2022;18(2):410–7.
6. Regenhardt RW, Kozberg MG, Dmytriw AA, Vranic JE, Stapleton CJ, Silverman SB, et al. Bow Hunter's Syndrome. Stroke. 2022;53(1):26–9.
7. Taylor WB, Vandergriff CL, Opatowsky MJ, Layton KF. Bowhunter's Syndrome Diagnosed with Provocative Digital Subtraction Cerebral Angiography. Baylor University Medical Center Proceedings. 2012;25(1):26–7.
8. Montano M, Alman K, Smith MJ, Boghosian G, Enochs WS. Bow Hunter's Syndrome: A rare cause of vertebrobasilar insufficiency. Radiol Case Rep. 2021;16(4):867–70.

9. Jadeja N, Nalleballe K. Pearls & Oy-sters: Bow hunter syndrome: A rare cause of posterior circulation stroke. Neurol. 2018;91(7):329–31.
10. Nomura Y, Toi T, Ogawa Y, Oshima T, Saito Y. Transitional nystagmus in a Bow Hunter's Syndrome case report. BMC Neurol. 2020;20(1):1–4.
11. Thomas AS, Vadakkedam SK. Bow Hunter's Syndrome with Rotational Atlantoaxial Instability: A Rare Association. Asian J Neurosurg. 2024;19(03):572–5.
12. Lee J beom, Jung G won. Bedside Approach of Vertebral Artery Insufficiency by Transcranial Doppler Sonography in Clinically Suspected Bow Hunter's Syndrome. Front Neurol. 2014;6(1):16–9.
13. Schubert MC, Carter N, Lo S fu L. Case report: bow hunter syndrome—one reason to add non-gravity dependent positional nystagmus testing to your clinical neuro-otologic exam. Front Neurol. 2021;12:1–6.
14. Ng S, Boetto J, Favier V, Thouvenot E, Costalat V, Lonjon N. Bow Hunter's Syndrome: Surgical Vertebral Artery Decompression Guided by Dynamic Intraoperative Angiography. World Neurosurg. 2018;118:290–5.

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