Human Brain (2024), 3(4)
https://doi.org/10.37819/hb..2047

Clinical Pathology Case Discussions

Bow Hunter's Syndrome: Illustrative Teaching Case with Insights and Lessons

Tianyu Jin [a], Yanfei Chen [a], Zan Chen [a,b], Wanru Duan [a,b,*]

[a]Department of Neurosurgery, Xuanwu Hospital, Capital Medical University, Beijing, China

[b]Spine Center, China International Neuroscience Institute (CHINA-INI), Beijing, China

***Corresponding Author's Email:** duanwanru@xwhosp.org

Abstract

A 63-year-old woman with a history of left basal ganglia and paraventricular infarcts, type II diabetes, and hypertension presented with recurrent dizziness when turning her head to the right. These episodes, lasting for eight months, were often accompanied by nausea and vision disturbances but resolved spontaneously. Dynamic digital subtraction angiography (DSA) revealed significant blood flow slowdown in the left vertebral artery (VA) with a 90° right neck turn, showing narrowing in the V3 segment of the left VA. The patient was diagnosed with Bow Hunter's Syndrome (BHS) and underwent atlantoaxial fusion surgery. On day three post-surgery, she developed sudden weakness in the left upper limb, visual field hemianopia, impaired sensation, and slurred speech. Emergency CT and MRI revealed acute infarcts in the basal ganglia and periventricular area. Following antiplatelet therapy and volume expansion, her symptoms improved, and by day seven, her speech and left upper limb strength had partially recovered, though residual symptoms persisted at discharge. One year post-surgery, the dizziness had resolved, but residual symptoms from the infarction remained. Cervical fusion effectively treated the symptoms of BHS; however, surgery should be considered with caution in patients with thromboembolic risk factors.

Keyword: Bow Hunter's Syndrome; Vertebral artery; Atlantoaxial fusion; Operative complication

Article History

Received: 21-Dec-2024

Revised: 27-Jan-2025

Accepted: 17-Feb-2025

Human Brain (2024), 3(4)
https://doi.org/10.37819/hb..2047

Introduction

Bow Hunter's Syndrome (BHS), also known as rotational vertebrobasilar insufficiency, was first described in a 1978 case report by Sorensen, which detailed an ischemic infarction in the posterior inferior cerebellar artery of a hunter who experienced head rotation while practicing archery[1]. This condition is primarily caused by transient stenosis or occlusion of the VA due to neck rotation or hyperextension, resulting in impaired blood flow to the vertebrobasilar system.

In rare cases, the ischemic mechanism may involve recurrent bony compression against the vessel wall, direct damage to the vascular endothelium, or complications such as entrapment, perforation, pseudoaneurysm formation, and subsequent distal embolization. Common non-traumatic causes include osteochondral hyperplasia, disc herniation, cervical spondylosis, tendon compression, fibrous tethering of the VA, atlantoaxial bony abnormalities, atlantoaxial instability, and rheumatoid arthritis. Acquired factors, such as complications from cervical spine surgery and neck injuries, also contribute to the syndrome[2, 3].

Previous studies have identified several exacerbating factors for BHS, including contralateral VA insufficiency, occlusion of the intracranial segment of the VA, and impaired collateral circulation. Additionally, vascular risk factors, including hypertension, hyperlipidemia, diabetes, smoking, and coronary artery disease, are commonly present in patients with BHS[4, 5].

Case Description

A 63-year-old woman with a history of left basal ganglia and paraventricular infarcts, type II diabetes, and hypertension presented to the neurology department with recurrent dizziness when turning her head to the right. These episodes, lasting for eight months, were often accompanied by nausea and vision disturbances but spontaneously resolved within minutes. Neurological examination revealed right-sided deficits, including tongue deviation, reduced muscle strength, hyperreflexia, and a positive Babinski sign, with no other neurological abnormalities.

CT angiography (CTA) showed severe stenosis of the right middle cerebral artery, a hypoplastic intracranial segment of the right vertebral artery with posterior inferior cerebellar artery occlusion, and moderate stenosis of the left vertebral artery's intracranial segment. DSA revealed significant blood flow slowdown in the left VA during a 90° right neck turn, with notable narrowing in the V3 segment of the left VA (**Fig. 1**). The patient was diagnosed with BHS and admitted for surgery. To avoid compromising the dominant left VA, atlantoaxial fusion was performed instead of foraminotomy decompression.

Human Brain (2024), 3(4)
https://doi.org/10.37819/hb..2047

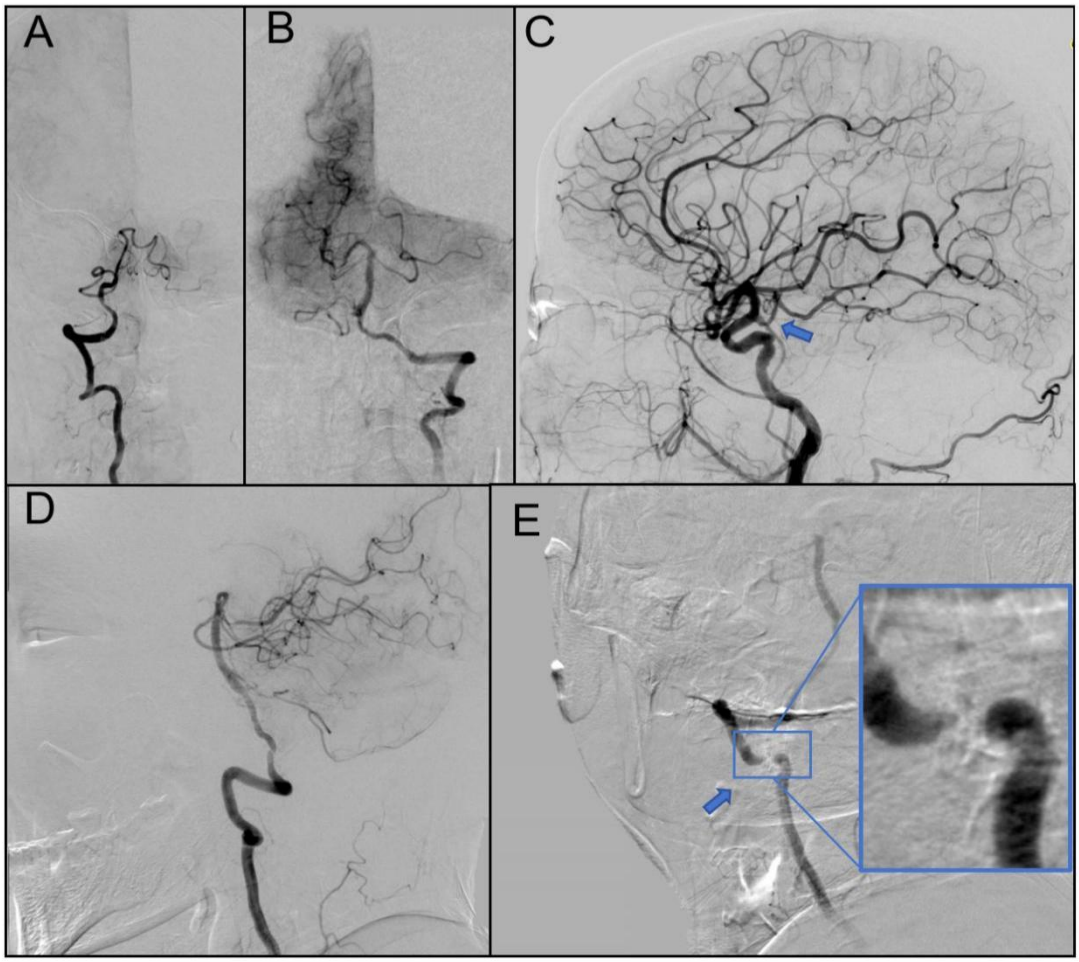

Figure 1. Preoperative dynamic DSA. In a neutral head position, the right vertebral artery V4 segment occluded beyond the origin of the posterior inferior cerebellar artery (A). The left vertebral artery moderate stenosis (60-70%) in V4 segment, the right posterior cerebral artery clearly opacified, left shows a fetal-type posterior cerebral artery (B), supplied by a well-developed left posterior communicating artery (C). Lateral view shows left vertebral artery loops at C2 level (D). With a 90-degree right head turn, severe stenosis at C2 slows blood flow in the left VA, leading to poor opacification of the distal left posterior cerebral artery (E).

On the third day post-surgery, the patient suddenly developed weakness in the left upper limb, left visual field hemianopia, impaired deep sensation in the left limbs, and slurred speech. Emergency CT and MRI indicated acute infarcts in the periventricular area (**Fig. 2**). Follow-up DSA revealed occlusion of the right posterior cerebral artery, likely caused by thromboembolism at the site of pre-existing left vertebral artery stenosis, possibly triggered by a postoperative hypercoagulable state (**Fig. 3**). After antiplatelet therapy and volume expansion, her symptoms gradually improved. By day seven post-surgery, her speech was slightly slurred, and left upper limb strength had improved to grade 4, with residual symptoms at discharge.

At 3-month and 1-year follow-ups, her dizziness had completely resolved, but residual symptoms from the cerebral infarction persisted.

Human Brain (2024), 3(4)
https://doi.org/10.37819/hb..2047

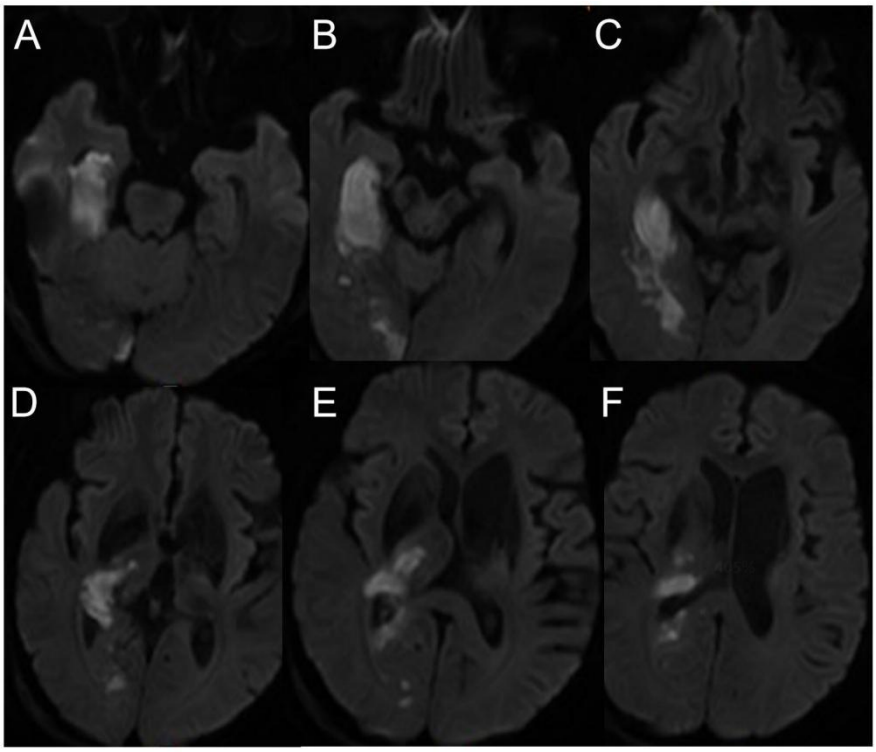

Figure 2. Postoperative brain MRI. Shows new cerebral infarcts in the right temporo-occipital lobes, right thalamus, splenium of the corpus callosum, and the corona radiata.

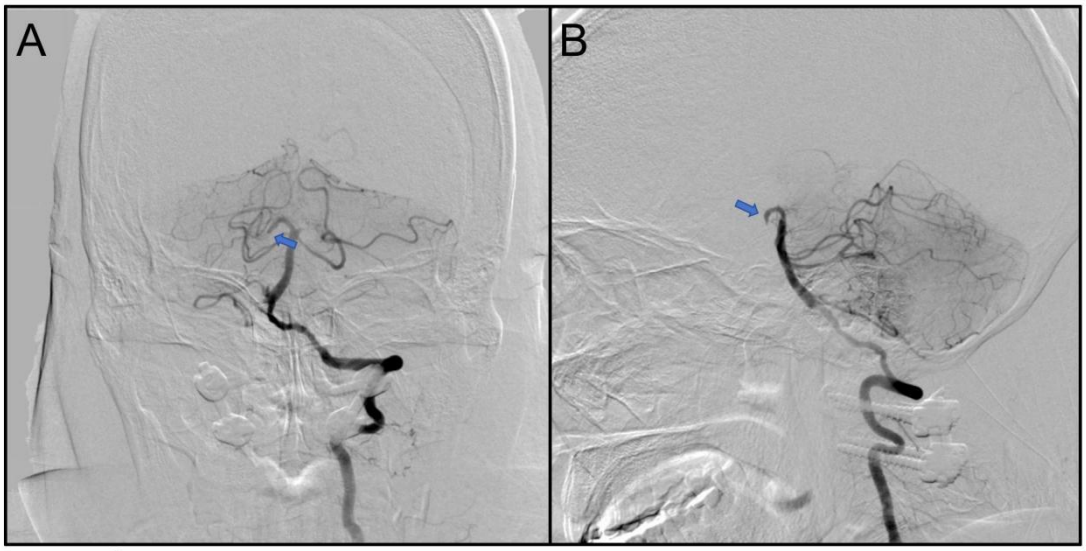

Figure 3. Postoperative DSA. Shows occlusion at the previously narrowed site of the right posterior cerebral artery P2 segment, with disappearance of distal blood flow.

Discussion

Bow Hunter's Syndrome1, also known as rotational vertebrobasilar insufficiency, derives its name from a 1978 case report by Sorensen. The condition is often associated with compression of the dominant vertebral artery (VA), along with occlusion of the intracranial segment and impaired collateral circulation, a particularly severe presentation in our patient.

Human Brain (2024), 3(4)
https://doi.org/10.37819/hb..2047

The VA, which originates from the subclavian artery, enters the bony canal formed by the cervical vertebrae's transverse processes at the C6 level. It then ascends almost vertically through the transverse foramina from C6 to C3, veering posterior-laterally at the axis vertebra, and continues through the transverse foramen of C1 to curve around the atlanto-occipital joint before penetrating the skull via the atlanto-occipital membrane. The atlantoaxial segment is the most common site of compression, traditionally attributed to the relatively fixed position of the VA at the C1 transverse foramen and atlanto-occipital membrane[6]. However, in our case, dynamic digital subtraction angiography (DSA) revealed that compression occurred primarily at the C2 level, potentially caused by the anterior edge of the superior aspect of the C2 transverse foramen. Our surgical experience suggests that when the neck is turned to the opposite side, the VA exits the C2 transverse foramen and makes a sharp forward turn, leading to compression. Between the C1 transverse foramen and the atlanto-occipital membrane, the VA forms a slack loop, providing some space for movement during neck rotation (Figure 4)[6].

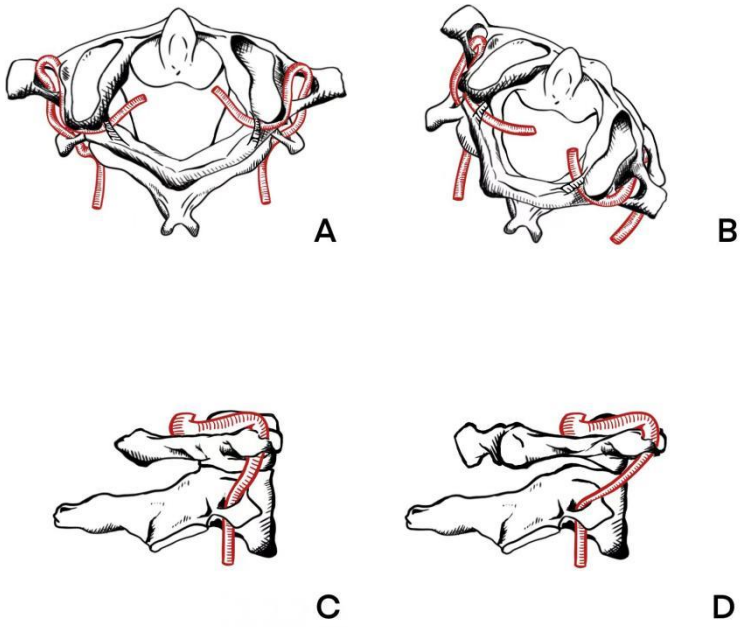

Figure 4. Illustrations showing a potential cause of BHS involving the V3 segment of the VA. The VA forms a loose loop around the lateral mass in the segment from the transverse foramen of C1 to the atlanto-occipital membrane (A), allowing for movement space during rotation (B). The transverse foramen of C2 opens posterolaterally, and the position of the vertebral artery is relatively fixed (C). During neck rotation, susceptible to compression from the anterior wall of the foramen (D).

Potential etiological factors contributing to symptoms include atlantoaxial block instability, osteophytes, developmental deformities of the atlantoaxial vertebrae, and abnormalities in VA alignment. At the C3–C6 levels, localized compression of the vertebral arteries by osteophytes growing in the intervertebral foramina during ipsilateral cervical rotation is the most common pathogenic mechanism.

The incidence and prevalence of BHS in the general population remain undefined. A retrospective analysis up to October 2022 identified 233 reported cases, with the highest prevalence observed in individuals aged 50–60 years, a male predominance, and a notable frequency of left-sided VA

involvement[7]. The syndrome typically manifests as reversible symptoms of posterior circulation ischemia during head rotation. The syndrome typically presents with reversible symptoms of posterior circulation ischemia during head rotation.

Dynamic vascular ultrasound and transcranial Doppler examinations are commonly used for screening pathophysiological changes, but they are not definitive for diagnosing BHS. In several reports, nearly half of the patients diagnosed with BHS had no evidence of dynamic vertebral artery occlusion on ultrasound[8]. Dynamic DSA remains the diagnostic gold standard, playing a crucial role in preoperative evaluation and outcome measurement. Although CTA and magnetic resonance angiography in a rotated neck position can also identify compressive structures, patient discomfort from prolonged neck rotation and the potential aggravation of symptoms pose challenges[6]. Notably, dynamic occlusions may not be visible on DSA when thromboembolic mechanisms are involved, highlighting the importance of considering BHS as a potential cause of posterior circulation strokes, especially in young adults with upper cervical anomalies and no other cerebrovascular risk factors[9].

BHS lacks a standardized treatment protocol, with surgical options, primarily cervical decompression and/or cervical fusion, being widely reported as effective definitive treatments. However, some studies suggest that decompression alone may be associated with a higher recurrence rate of stroke[4].In cases of BHS where atlantoaxial dislocation or instability compresses the vertebral artery at the V3 segment, posterior atlantoaxial fusion is required. Decompression alone in these cases could exacerbate atlantoaxial joint instability. Although there have been no previous reports of thromboembolic complications following atlantoaxial fixation in BHS patients, our experience suggests that this risk should not be overlooked, particularly in those with stroke risk factors. In our case, the patient underwent posterior atlantoaxial fixation and fusion using the Goel-Harms technique but developed a right posterior cerebral artery infarction on postoperative day 3. Given the absence of cervical vertebral artery injury or entrapment, and considering the patient's vascular risk factors, including type 2 diabetes and intracranial atherosclerosis, a postoperative hypercoagulable state-induced thromboembolism was suspected.

Following multidisciplinary consultations and postoperative analysis, we identified several key strategies to prevent postoperative thromboembolism and improve outcomes for high-risk patients:

Anticoagulation Therapy: Anticoagulation before, during, and after surgery is essential. For patients already on secondary stroke prevention therapy, bridging with low-molecular-weight heparin (LMWH) before surgery is recommended. The standard dose is 40 mg once daily, or 0.5–0.7 mg/kg every 12 hours, adjusted based on the patient's weight, to ensure adequate anticoagulation during the perioperative period[10].

Intraoperative Care: Intraoperatively, careful hemostasis must be maintained, and a paravertebral drain should be placed to minimize bleeding risks.

Postoperative Care: For patients who received LMWH bridging therapy, anticoagulation should be resumed within 24 hours post-surgery. Alternatively, novel oral anticoagulants, such as Rivaroxaban (10 mg once daily) or Dabigatran (110 mg once daily), may be used, with doses adjusted according to renal function and bleeding risk[11]. Early resumption of anticoagulation significantly reduces the risk of thromboembolism and improves postoperative outcomes.

Preoperative Evaluation and Microsurgical Technique: Preoperative CTA 3D reconstruction is essential to clearly define the course of the vertebral artery, allowing for the development of a tailored surgical plan. In cases with high-riding vertebral arteries, using fusion devices with lateral mass or transpedicular screws provides adequate fixation while minimizing vertebral artery injury risk. Before performing lateral atlantoaxial joint release, the V3 segment of the vertebral artery should be exposed without opening the arterial sheath. Fusion devices and screws should be placed under direct visualization. If the vertebral artery obstructs the joint surface, a nerve dissector can be gently used to elevate the artery. Minimizing traction or compression of the vertebral artery reduces the risk of vascular spasm, intraoperative hemodynamic changes, or endothelial damage[12].

Alternative approaches for managing BHS primarily include antithrombotic therapy, stent implantation, coil embolization, and lifestyle modifications, such as limiting neck movement or using a cervical collar. While therapeutic vertebral artery (VA) occlusion has been reported[13], it is generally not recommended as a routine treatment due to the presence of contralateral vertebral artery stenosis in the majority of patients. In older adults with refractory vertebrobasilar insufficiency caused by unilateral supra-axial BHS and flow-limiting atherosclerosis in the contralateral vertebral artery, stent angioplasty of the atherosclerotic lesion has been used effectively to restore vertebrobasilar circulation[14]. Some authors have also reported the use of direct endovascular stent implantation for treating BHS lesions[15], but this approach carries risks, including stent displacement and vascular occlusion, as the underlying conflict between the bone structure and the vertebral artery remains unresolved.

Antithrombotic therapy and restriction of neck movement are typically used as initial management strategies for BHS[6]. If BHS is suspected, we advise patients to limit excessive cervical rotation or use a rigid cervical collar to stabilize the neck. For patients with a history of stroke, antithrombotic or antiplatelet therapy should be initiated promptly for secondary stroke prevention. However, these measures do not address the underlying compressive pathology. Early reports indicated that nearly half of patients undergoing conservative treatment experienced strokes with permanent neurological deficits during the one-year follow-up[16]. A prospective study of 19 patients, despite showing good safety with conservative treatment over an average follow-up of 37.5 months, found that symptoms were not completely resolved[9]. Therefore, we advocate for definitive surgical treatment in BHS patients to prevent recurrent strokes. We recommend postponing surgery for high-risk patients until underlying conditions, such as diabetes, hypertension, and atherosclerosis, are adequately managed.

Abbreviations

DSA: Dynamic digital subtraction angiography

VA: vertebral artery

BHS: Bow Hunter's Syndrome

PCA: Posterior cerebral artery

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

The participant had signed the informed consent for clinical research at Xuanwu Hospital.

Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Competing interests

The authors declare that they have no competing interests.

Funding

National High-Level Hospital Clinical Research Funding (2022-PUMCH-D-004)
Beijing Natural Science Foundation Grant (No. L212039).

Acknowledgments

All authors declare that there is no conflict of interest.

Reference

1. Sorensen BF. Bow hunter's stroke. *Neurosurgery*. 1978; 2:259-261
2. 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. *Interventional neurology*. 2016; 5:29-38
3. Sherman DG, Hart RG, Easton JD. Abrupt change in head position and cerebral infarction. *Stroke*. 1981; 12:2-6
4. Jost GF, Dailey AT. Bow hunter's syndrome revisited: 2 new cases and literature review of 124 cases. *Neurosurgical focus*. 2015;38: E7
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. *Journal of vascular and interventional neurology*. 2015; 8:7-16
6. Ndongo Sonfack DJ, Bojanowski MW, Tarabay B, Gennari A, Shédid D, Yuh SJ. Vertebral artery stenosis from osteophyte: A systematic review and case series. *Neuro-Chirurgie*. 2024; 70:101525
7. Correia PN, Meyer IA, Michel P. Osteovascular conflicts in the neck region and cerebrovascular events: Illustrative cases and literature review. *Global spine journal*. 2023:21925682231220044
8. Choi KD, Choi JH, Kim JS, Kim HJ, Kim MJ, Lee TH, et al. Rotational vertebral artery occlusion: Mechanisms and long-term outcome. *Stroke*. 2013; 44:1817-1824
9. Jadeja N, Nalleballe K. Pearls & oy-sters: Bow hunter syndrome: A rare cause of posterior circulation stroke: Do not look the other way. *Neurology*. 2018; 91:329-331
10. Douketis JD. Perioperative management of patients who are receiving warfarin therapy: an evidence-based and practical approach. Blood. 2011 May 12;117(19):5044-5049.
11. ACCP (American College of Chest Physicians). Perioperative management of anticoagulant therapy: 2022 Update. Chest. 2022;162(2): S1-S67.
12. Chen Z, Duan W, Chou D, Guan J, Liu Z, Jian Q, Zhang B, Bo X, Jian F. A safe and effective posterior intra-articular distraction technique to treat congenital atlantoaxial dislocation associated with basilar invagination: Case series and technical nuances. Oper Neurosurg (Hagerstown). 2021;20(4):334-342. doi: 10.1093/ons/opaa391
13. Kutsuna F, Morofuji Y, Shiozaki E, et al. Endovascular coil embolization for recurrent Bow

Hunter's stroke. Intern Med. 2022;61(23):3595-3598. doi:10.2169/internalmedicine.8906-21

14. Sugiu K , Agari T , Tokunaga K , et al . Endovascular treatment for bow hunter’s syndrome: case report. Minim Invasive Neurosurg 2009; 52:193–5

15. Kageyama H, Yoshimura S , Iida T , et al . Juvenile cerebral infarction caused by bow hunter’s syndrome during sport: two case reports. Neurol Med Chir (Tokyo) 2016; 56:580–3

16. Kuether TA, Nesbit GM, Clark WM, Barnwell SL. Rotational vertebral artery occlusion: a mechanism of vertebrobasilar insufficiency. Neurosurgery. 1997; 41:427-432; discussion 432.