Open Access Original Research Article

MedPeer Publisher
Abbreviated Key Title: MedPeer
ISSN : 3066-2737
homepage: https://www.medpeerpublishers.com

BOW HUNTER'S SYNDROME CAUSING A RECENT CEREBELLAR INFARCTION: A CASE REPORT

DOI: 10.70780/medpeer

AUTHOR AND AFFILIATION

Mohamed Fadil[1], Mehdi Salmane[1], Zineb Izi[1], Zakaria Toufga[1], Rachida Saouab[1], Jamal El Fenni[1]

[1] Radiology department, Mohamed V military hospital, Rabat, Morocco.

Corresponding author: Mohamed Fadil .

ABSTRACT

Bow Hunter's Syndrome (BHS), or rotational vertebral artery occlusion syndrome, is a rare cause of symptomatic vertebrobasilar insufficiency or ischemic stroke induced by head movement. We report the case of a patient presenting with a recent cerebellar infarction who underwent supra-aortic vessel angiography. The vascular study identified predisposing structural anomalies and critical narrowing along the vertebrobasilar pathway. This case emphasizes the clinical importance of recognizing mechanical vertebral artery compromise in posterior circulation stroke, even when dynamic rotational imaging is not routinely performed.

KEYWORDS

Bow hunter's syndrome, BHS, vessel angiography, CT

MAIN ARTICLE

INTRODUCTION

Bow Hunter's Syndrome (BHS) is a rare dynamic vascular condition characterized by transient mechanical compression of the vertebral artery during head rotation or extension, leading to vertebrobasilar insufficiency or ischemic stroke. First described in an archer developing symptoms during practice, BHS typically occurs at the atlantoaxial (C1–C2) junction or along the foraminal course of the vertebral artery due to osteophytes, fibrous bands, or anatomical variations. Because dynamic rotational angiography is not routinely performed in every acute stroke evaluation, establishing the diagnosis often relies on high clinical suspicion alongside standard neurovascular imaging. Here, we present a case of recent cerebellar infarction evaluated via supra-aortic vessel angiography that demonstrated underlying vertebral artery compression consistent with BHS.

CLINICAL PRESENTATION

A 61-year-old male presented to our department with acute neurological symptoms consistent with posterior circulation ischemia, including vertigo, dizziness, and nausea. Brain computed tomography (CT) revealed a well-defined left cerebellar hypodensity within the vascular territory of the left posterior inferior cerebellar artery (PICA) (figure 1), corresponding to a recent ischemic infarction. To determine the etiology of the stroke, an angiographic evaluation of the supra-aortic vessels was performed. Angiography demonstrated an agenesis of the left transverse foramina of the sixth cervical vertebrae, with extraforaminal course of the left vertebral artery (figure 2), anterior to transverse apophysis with focal narrowing and tethering of the artery (figure 3). Correlating these anatomical and angiographic findings with the patient's recent left cerebellar infract provided strong evidence for Bow Hunter's Syndrome as the primary mechanism of the stroke.

DISCUSSION

Bow Hunter's Syndrome (BHS), or rotational vertebral artery occlusion syndrome, represents a distinct mechanical etiology of vertebrobasilar insufficiency that frequently poses a diagnostic challenge in acute stroke management [1]. First described in 1978 in an archer

who developed transient symptoms upon head rotation, the condition encompasses a broad clinical spectrum ranging from transient position-dependent dizziness and syncope to frank posterior circulation infarction [1, 2].

Pathophysiologically, BHS is driven by dynamic or static mechanical compromise of the vertebral artery (VA) along its course. While the subaxial (V2) and suboccipital (V3) segments are most susceptible due to their proximity to cervical bony structures, compression at the C6 level often involves specific anatomical variants [1, 3]. These predisposing structural abnormalities include an anomalous extraforaminal entry (where the VA enters the transverse foramen above or below its typical C6 level), focal osteophytic spurring from uncovertebral or facet joint arthrosis, fibrous bands, or congenital fibrous tethering at the entrance to the transverse foramen [1–3]. When the vessel is extraforaminal or tethered, head rotation exerts severe shear stress and focal compression at the point of dural or bony entry, leading to luminal compromise.

Classical teaching dictates that clinical BHS can only occur if the contralateral VA is hypoplastic or aplastic, on the assumption that as long as the contralateral VA remains intact, overall basilar flow remains adequate for autonomous posterior circulation perfusion [1, 3]. This case, however, highlights a common yet overlooked finding: in isolated PICA infarction, hypoplasia or aplasia of the contralateral VA is not strictly required as PICA (V4 segment) arises directly from the intracranial VA on the same side. As such, the downstream PICA is entirely reliant on an intact, open ipsilateral VA. Consequently, focal stenosis or microvascular trauma of ipsilateral VA can precipitate PICA infarction via two distinct mechanisms: direct hemodynamic flow failure proximal to the basilar junction, or local intimal damage causing microthrombus formation with subsequent arterial-to-arterial thromboembolism [1–3].

Dynamic digital subtraction angiography with provoked head movement remains the established modality through which positional arterial occlusion is best demonstrated, yet high resolution, cross-sectional CT angio of the supra-aortic vessels with neutral head position can identify underlying anatomic predispositions such as aberrant ascending artery ingress, focal extracapsular compression, bony osteophytes, or gaps in vessel axially capacity, which support the diagnosis without subjecting the recently infarcted patient to the risk of large vessel manipulation [1, 3].

Management of BHS requires balancing conservative strategies, such as antiplatelet agents, cardiovascular risk factor control, and behavioral modification to limit extreme head turns,

against surgical decompression or fusion, with surgery generally reserved for patients with established infarction, recurrent ischemic symptoms, or failed medical therapy [2, 3].

CONCLUSION

Bow Hunter's Syndrome should be strongly suspected in patients presenting with unexplained cerebellar or brainstem infarctions, particularly when symptoms are position-dependent. Supra-aortic vessel angiography is essential for unmasking vertebral artery stenosis, an anomalous arterial course, or underlying structural anomalies that compromise posterior circulation blood flow. Early identification allows for targeted conservative or surgical interventions to prevent recurrent stroke.

FIGURES:

Figure 1: Axial brain CT slice through the posterior fossa demonstrating a well-defined, wedge-shaped hypodensity in the left cerebellar hemisphere (yellow arrow), consistent with a recent infarction in the territory of the left posterior inferior cerebellar artery (PICA).

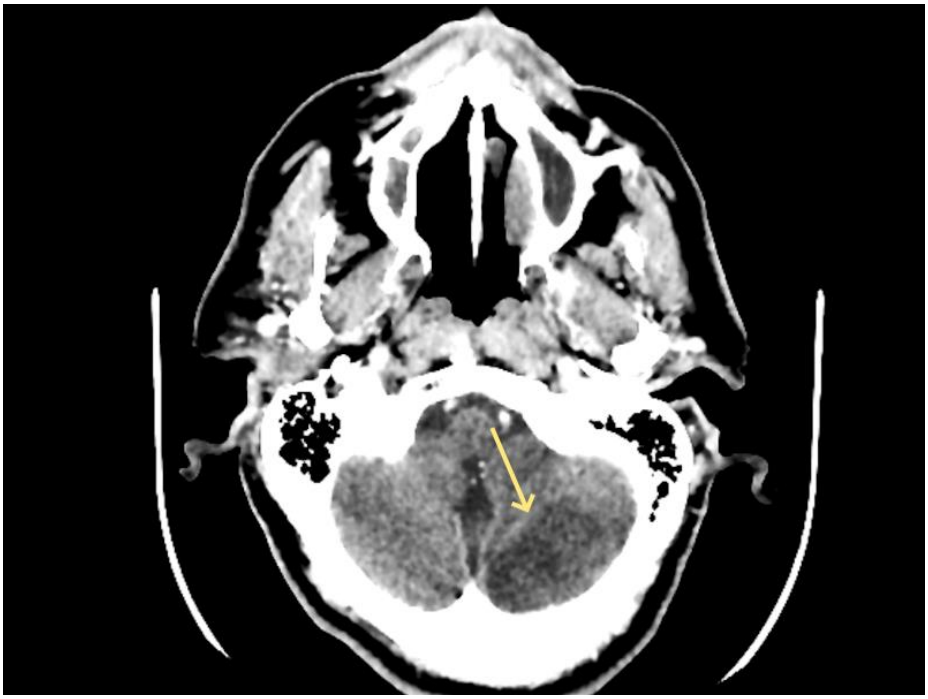


Figure 2: 3D volume-rendered reconstruction of a CT angiography of the supra-aortic vessels demonstrating an anomalous extraforaminal course of the left vertebral artery at the level of the sixth cervical vertebra (C6) (yellow arrow). Note the normal intraforaminal course of the right vertebral artery at the same level (red arrow).

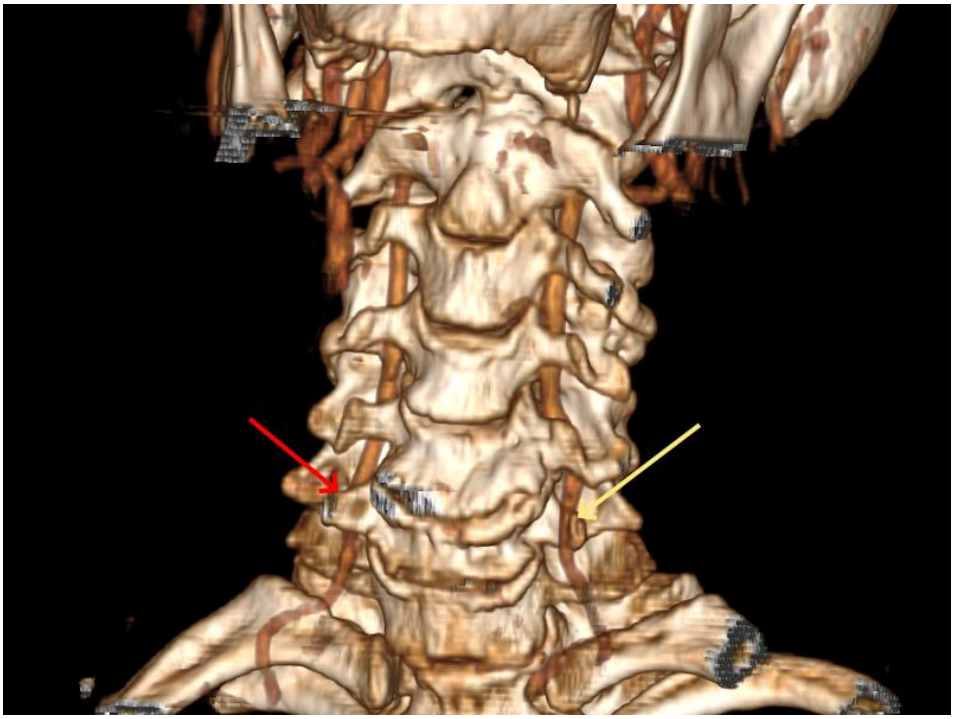
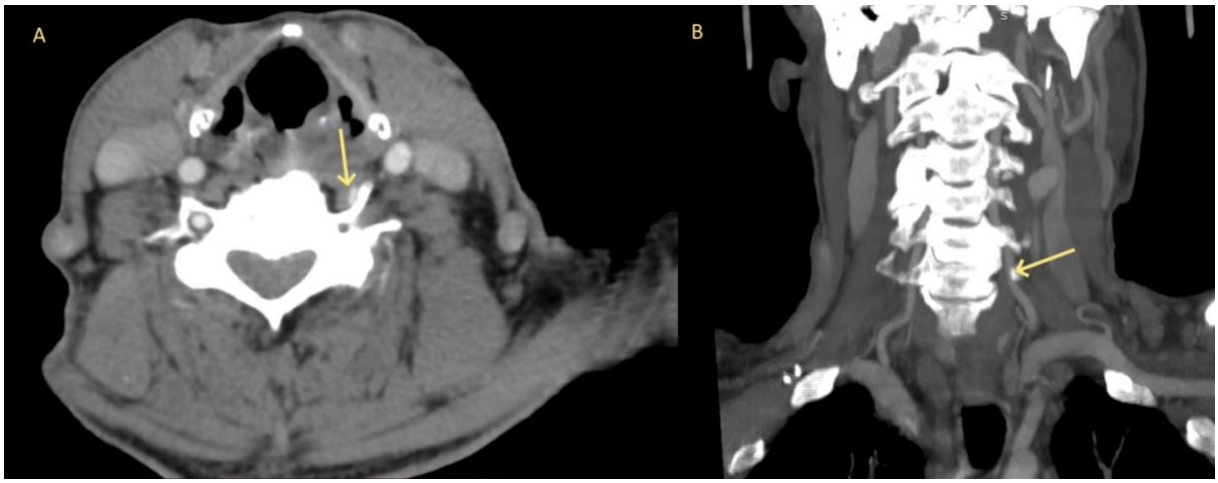


Figure 3: Axial (A) and coronal (B) maximum intensity projection (MIP) CT angiograms of the supra-aortic vessels demonstrating focal narrowing and tethering of the left vertebral artery at the C6 level (yellow arrow).



ACKNOWLEDGEMENTS

The authors have no acknowledgements to declare and report no conflicts of interest.

REFERENCES

1. Li S, Lu Y, Sun Y, et al. Bow hunter's syndrome due to an anomalous right vertebral artery origin and contralateral absence: a case report and literature review. BMC Neurology. 2024;24:375. doi:10.1186/s12883-024-03754-5.
https://doi.org/10.1186/s12883-024-03754-5
2. Elizondo-Ramirez J, Herrera-Castro JC, del Rio-Martinez CJ, et al. Bow Hunter's syndrome surgical approach and outcome: Two new cases and literature review. Surgical Neurology International. 2024;15:404. doi:10.25259/SNI_633_2024.
https://doi.org/10.25259/SNI_633_2024
3. Regenhardt RW, Kozberg MG, Dmytriw AA, et al. Bow Hunter's Syndrome. Stroke. 2022;53(1):e20-e24. doi:10.1161/STROKEAHA.121.037253.
https://doi.org/10.1161/STROKEAHA.121.037253