

# World Journal of *Clinical Cases*

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Editorial Board Member of *World Journal of Clinical Cases*, Md Moshir Rahman, MBBS, Assistant Professor, Department of Neurosurgery, Holy Family Red Crescent Medical College Hospital, Dhaka 1000, Bangladesh. dr.tutul@yahoo.com

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## Atypical case of bow hunter's syndrome linked to aberrantly coursing vertebral artery: A case report

Jun Hyong Ahn, Hyo Sub Jun, In Kyeong Kim, Choong Hyo Kim, Seung Jin Lee

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**Jun Hyong Ahn, Hyo Sub Jun, In Kyeong Kim, Choong Hyo Kim, Seung Jin Lee**, Department of Neurosurgery, Kangwon National University School of Medicine, Kangwon National University Hospital, Gangwon-do, Chuncheon-si 24289, South Korea

**Corresponding author:** Seung Jin Lee, MD, Assistant Professor, Department of Neurosurgery, Kangwon National University School of Medicine, Kangwon National University Hospital, No. 156 Baengnyeong-ro, Gangwon-do, Chuncheon-si 24289, South Korea.  
[rabbit3540@empas.com](mailto:rabbit3540@empas.com)

### Abstract

#### BACKGROUND

In bow hunter's syndrome (BHS), also known as rotational vertebral artery (VA) syndrome, there is dynamic/rotational compression of the VA producing vertebrobasilar insufficiency. Most occurrences involve atlantoaxial rather than mid-cervical VA compromise, the latter being rarely reported. Herein, we detail successful VA decompression at mid-cervical spine, given a departure from its usual course.

#### CASE SUMMARY

The patient, a 45-year-old man, presented to our hospital with occipital headache and vertigo. Computed tomography angiography showed anomalous C4 entry of right VA, with compression upon head rotation to that side. Thyroid cartilage and anterior tubercle of C5 transverse process were visibly at fault. We opted for surgery, using an anterior cervical approach to remove the anterior tubercle. Patient recovery was uneventful and brought resolution of all preoperative symptoms.

#### CONCLUSION

BHS is an important consideration where aberrant coursing of VA and neurologic symptoms coexist.

**Key Words:** Bow hunter's syndrome; Vertebral artery; Vertebrobasilar insufficiency; Case report

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**Core Tip:** Herein, we report a rare case of bow hunter's syndrome (BHS) induced by dynamic/rotational compression of the vertebral artery (VA) at the mid-cervical level associated with anomalous entry of the VA at C4. The VA was dynamically compressed between the thyroid cartilage and the anterior tubercle of the C5 transverse process upon right rotation. Complete symptom relief followed excision of the offending tubercle. BHS usually involves the atlantoaxial, rather than mid-cervical, VA. BHS linked to anomalous entry of the VA has rarely been reported. This case involves a rare etiology of BHS and a unique approach to its management.

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## INTRODUCTION

In bow hunter's syndrome (BHS), also known as rotational vertebral artery (VA) syndrome, there is dynamic compression of the VA leading to vertebrobasilar insufficiency (VBI). Vertigo, visual disturbances, and other symptoms are generally manifested upon head rotation[1,2]. Since its first description by Sorensen[3], based on complaints of practicing archers, over 40 cases have been published in the literature. To our knowledge, compression at atlantoaxial level is typical, mid-cervical VA impingement being rarely reported. Furthermore, few cases to date are attributable to inconsistencies in VA course[4,5]. Herein, we describe an instance of aberrantly coursing VA resulting in BHS and treated successfully through surgical decompression.

## CASE PRESENTATION

### Chief complaints

A 45-year-old man presented to the hospital with a 3-mo history of occipital headache, which began after a marathon race.

### History of present illness

During the prior month, he had also complained of a spinning sensation within moments of turning his head to the right.

### History of past illness

Other than admitted hypertension, his general health was good.

### Personal and family history

The patient denied any family medical history.

### Physical examination

During the neurologic examination, head-turning to the right induced nystagmus of left-sided gaze.

### Laboratory examinations

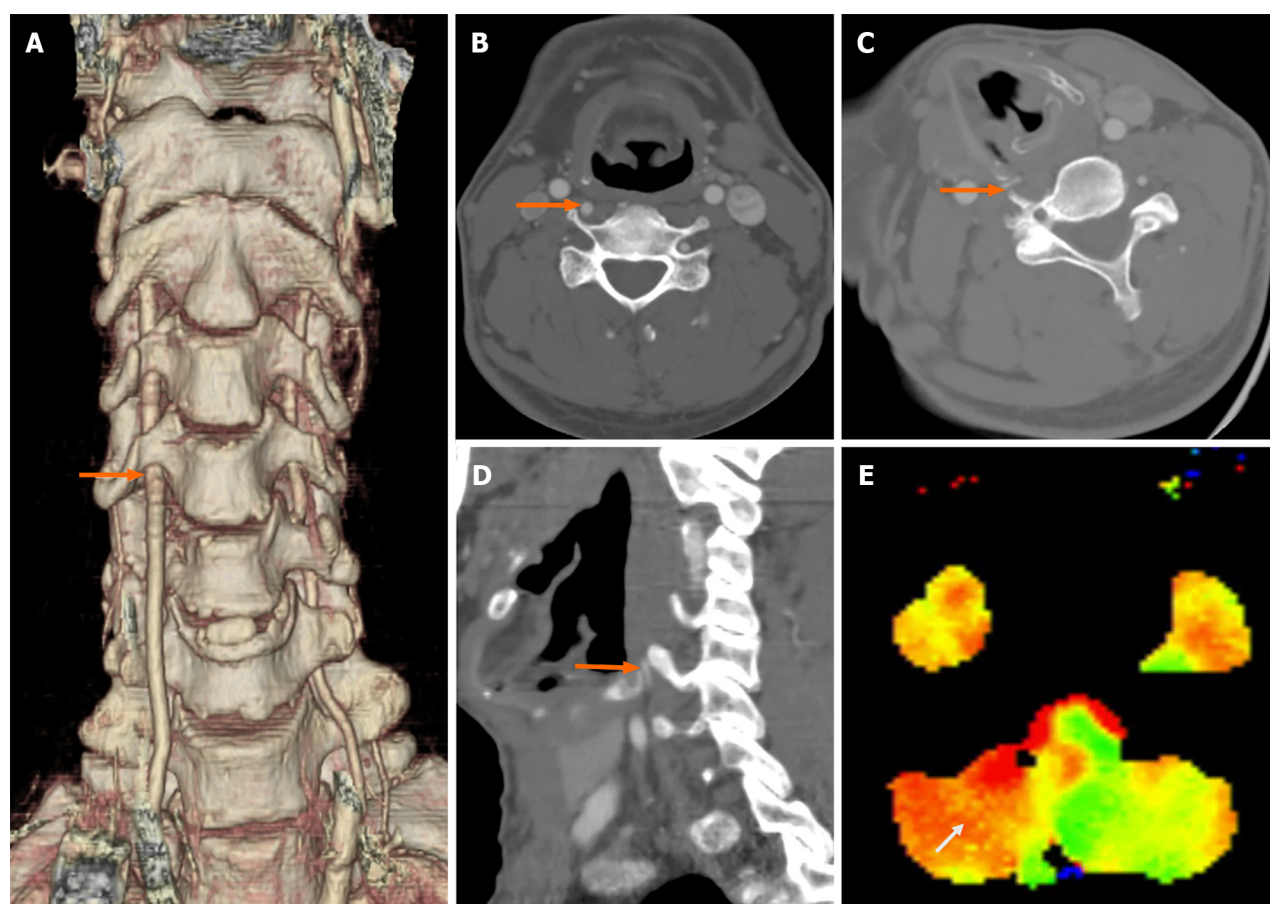
The patient had no abnormalities in blood and urine tests.

### Imaging examinations

Computed tomography angiography (CTA) and perfusion magnetic resonance imaging (MRI) were performed to assess for VBI. The CTA showed normal left VA entry at C6 vertebra, with right VA entering the C4 transverse foramen (Figure 1A and B). Both thyroid cartilage and anterior tubercle of C5 transverse process brought compression of right VA upon head-turning to the right (Figure 1C and D). Perfusion MRI acquired with right head rotation also indicated a time-to-peak delay in right cerebellar territory (Figure 1E).

## FINAL DIAGNOSIS

Base on the results of the imaging examinations, the final diagnosis was BHS linked to aberrantly coursing VA.



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**Figure 1** Preoperative computed tomography angiography and perfusion magnetic resonance imaging studies. A: Normal C6 vertebral artery (VA) entry on left and anomalous right VA entry at C4 transverse foramen (arrow); B: Axial view of patent right VA in neutral position (arrow); C and D: Rotational dynamic compression of right VA by thyroid cartilage and anterior tubercle of C5 transverse process (arrows); E: Time-to-peak delay in right cerebellar territory (arrow) during head rotation to right.

## TREATMENT

This extrinsic obstruction of VA called for surgical intervention through anterior cervical approach. A skin incision was made above medial sternocleidomastoid muscle to remove the offending C5 tubercle and achieve wide VA decompression.

## OUTCOME AND FOLLOW-UP

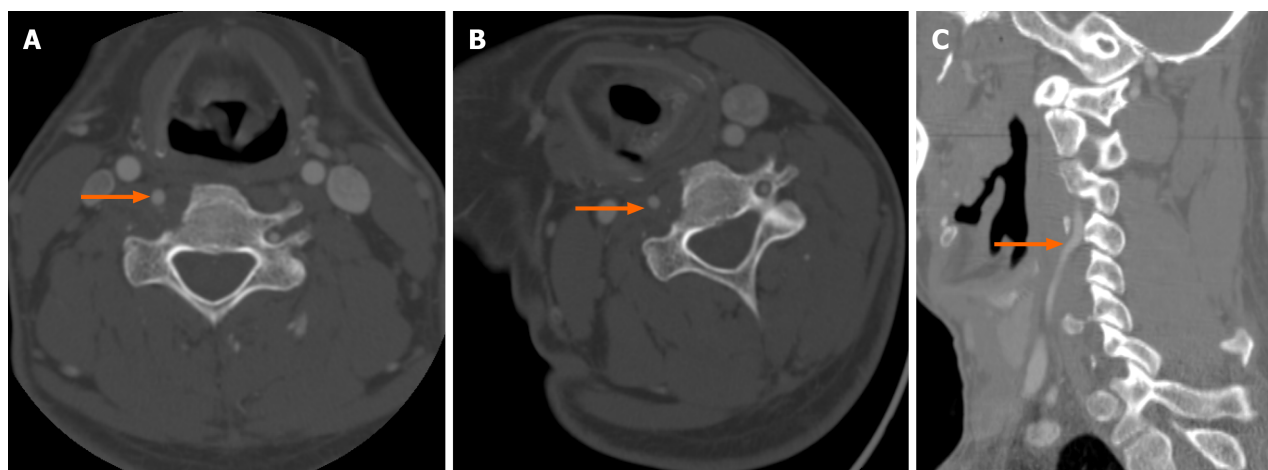
Following surgery, complete elimination of culprit bone (Figure 2A) and absence of right rotational compression were documented by CTA (Figure 2B and C). Patient recovery was uneventful, with full and lasting resolution of preoperative symptoms.

## DISCUSSION

The VA ordinarily runs cephalad within transverse foramina of the cervical spine, entering at C6 level and exiting at C1. Aberrancies in course are rare and are byproducts of anomalous embryologic development[6]. According to the literature, the compressive impact of BHS is usually borne at C2 level or above, with a left-dominant VA more likely involved[7]. Although bony malformations or regional instability are both implicated in BHS, the present case indicates that VA aberrancy may contribute as well, predisposing to dynamic mid-cervical compression. Karle *et al*[4] and Jongbloed *et al*[5] have chronicled similar VA entries at C4 and C5, respectively[4,5]. The relatively narrow space afforded and encroaching structures (*i.e.*, thyroid cartilage, anterior tubercle of cervical spine) are apt to intensify compressive insults.

Dynamic VA occlusion is expectedly signaled by VBI symptoms, namely vertigo, dizziness, syncope, paresthesia, ataxia, or headache[8]. Review of the limited available literature suggests there is also increased risk of recurrent embolic stroke[9,10]. Although the precise mechanism of our patient's occipital headache is not fully understood, it is feasible that





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**Figure 2 Postsurgical computed tomography angiography.** A: Anterior tubercle of C5 transverse process eliminated and right vertebral artery (VA) patent (arrow); B and C: Axial and sagittal images during right rotation of head showing no dynamic VA compression (arrows).

pro-inflammatory neurotransmitter release triggered by localized irritation may have elicited pain remote from points of actual injury[11]. In a patient presenting with headache and positional VBI symptoms, such as nystagmus or transient vertigo, BHS is then a valid consideration; and provocative CTA (with head rotation) is the principal means of confirming any clinical suspicions.

Once a diagnosis of BHS is established, the condition may be managed conservatively or surgically treated. Conservative measures include avoidance of head rotation and anticoagulant or antiplatelet therapy. However, these actions fail to address underlying pathologies, proving ineffective for some. Surgical treatments range from direct VA decompression to cervical spine fusion, depending on circumstances and level of VA compromise. There have been two published cases of BHS and aberrant VAs where laryngoplasties served to eliminate problematic superior cornual ossifications of thyroid cartilage[4,5]. Ultimately, direct VA decompression achieved by resecting a prominent tubercle readily sufficed for our patient.

## CONCLUSION

The middle-aged man we describe presented with BHS of unusual nature, marked by aberrant coursing of right VA and mid-cervical compressive symptoms. This predicament was successfully remedied through careful diagnostic workup and surgical decompression. BHS must always be considered in analogous situations where comparable anomalies and neurologic manifestations coexist.

## FOOTNOTES

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**Country/Territory of origin:** South Korea

**ORCID number:** Jun Hyong Ahn 0000-0002-8529-6757; Hyo Sub Jun 0000-0003-2357-0033; In Kyeong Kim 0000-0002-7761-9456; Choong Hyo

Kim 0000-0002-4665-5008; Seung Jin Lee 0000-0002-3011-7918.

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