Rotational vertebral artery occlusion secondary to adjacent-level degeneration following anterior cervical discectomy and fusion

Case report

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Rotational vertebral artery occlusion (RVAO), or bow hunter’s syndrome, most often occurs at the C1–2 level on physiological head rotation. It presents with symptoms of vertebrobasilar insufficiency (VBI). Several previously published studies have reported on subaxial sites of vertebral artery (VA) compression by head rotation. The authors report a case of subaxial spine RVAO due to adjacent-segment degeneration. A 52-year-old man presented with dizziness when rotating his head to the left. Twenty years earlier, he had undergone a C4–5 anterior cervical discectomy and fusion (ACDF) for a herniated disc. Imaging studies including a dynamic CT angiography and dynamic catheter angiography revealed occlusion of the left VA at the C3–4 level when the patient turned his head to the left, in the setting of an aberrant vertebrobasilar system. Successful treatment was achieved by surgical decompression of the left VA and C3–4 ACDF. Expedited diagnosis and treatment are dependent on the recognition of this unusual manifestation of RVAO, especially when patients present with nonspecific symptoms of VBI.

(http://thejns.org/doi/abs/10.3171/2014.3.SPINE13452)

Key Words • vertebral artery • vertebrobasilar artery insufficiency • bow hunter’s syndrome • vertebral artery compression • spinal degeneration • anterior cervical discectomy and fusion

Rotational vertebral artery occlusion (RVAO), or bow hunter’s syndrome, refers to symptomatic vertebrobasilar insufficiency (VBI) that occurs with physiological head rotation. Symptoms manifest on rotation of the head and commonly include vertigo, dizziness, ataxia, dysarthria, dysphagia, sensorimotor disturbance, diplopia, homonymous hemianopsia, nausea, and syncope. The typical mechanism is characterized by a dominant vertebral artery (VA) providing the major vertebrobasilar blood supply, which is transiently compressed at the C1–2 level with contralateral head rotation. Diagnosis is dependent on high clinical suspicion, and if left untreated, this condition may lead to strokes with permanent neurological deficits. Multiple reports of subaxial RVAO exist in the literature. The majority of these cases were second-ary to spondylotic changes. Here, we report a case of RVAO caused by nondominant VA compression at a vertebral segment adjacent to a previously performed anterior cervical discectomy and fusion (ACDF). This case is unique in both the nature of the compression and the nondominant VA that terminated in the posterior inferior cerebral artery (PICA). The patient was successfully treated by anterior cervical decompression and fusion.

Case Report

A 52-year-old man was referred to our clinic for evaluation of dizziness that occurred when he turned his head to the left. On occasion, he also noticed bilateral upper-extremity weakness when turning his head to the left. Over time, these symptoms became more frequent and occurred with a lesser degree of head turning. His history was significant for cervical disc herniation requiring C4–5 ACDF about 20 years prior to current symptom on-
Rotational vertebral artery occlusion

set. Without provocation, neurological examination was normal; however, head turning to the left side resulted in weakness of upper extremities.

Before presentation to our clinic, the patient underwent a duplex ultrasonography, which demonstrated reduction in left VA blood flow when the patient turned his head to the left side in comparison with flow in VAs when he maintained his head in a neutral position (Fig. 1A and B). Dynamic CT angiograms (CTAs) of the neck were subsequently obtained with the head neutral and turned to the left (Fig. 1C and D). This demonstrated reduced flow in the midportion of the basilar artery between the origin of the superior cerebellar artery (SCA) and anterior inferior cerebellar artery (AICA). The left VA terminated in the PICA. The caliber of the right VA decreased as it penetrated the dura at the V3–V4 junction. With head rotation toward the left, the patient’s left VA narrowed at the C3–4 level.

Dynamic catheter cerebral angiography was performed to better characterize the anomalous vertebrobasilar circulation and the hemodynamic significance of the dynamic left VA occlusion (Figs. 1E and F and 2). In the neutral position, a slight indentation was noted in the left

![Dynamic vascular imaging studies of the left VA.](image-url)

**Fig. 1.** Dynamic vascular imaging studies of the left VA. **Left-hand** panels, head neutral; **right-hand** panels, head turned to left.  
**A and B:** Duplex ultrasound images demonstrating absent diastolic wave upon head turn.  
**C and D:** Axial CTAs showing encroachment on the VA (arrow) by degenerative osteophyte and diminution of flow with head turned.  
**E and F:** Left VA catheter angiograms, anteroposterior view, showing near-complete occlusion upon head turn to the left.
VA at the C3–4 level. With head rotation to the left, there was complete occlusion of the left VA at this level (Fig. 1E and F). Additionally, a focal, approximately 40% narrowing of the right VA at its dural penetration (junction between the V₃ and V₄ segment) was again visualized. On the left, the VA was again noted to terminate in the PICA. On the right side, there was an AICA-PICA configuration. The midbasilar occlusion was again visualized just below the level of the SCAs. The basilar artery above the occlusion filled via the right posterior communicating artery (PCoA) and transiently through the left PCoA.

Cervical spine flexion-extension radiography showed solid fusion at the C4–5 level and no instability noted elsewhere. Cervical MRI demonstrated cervical disc degeneration and adjacent-segment disease at C3–4 (Fig. 3). At the C3–4 level, there was a disc-osteophyte complex causing canal stenosis and impingement on the spinal cord with associated T2 signal changes in the substance of the cord. Compression of the VA by an uncovertebral osteophyte was visualized on 3D reconstruction of dynamic CTAs (Fig. 4).

Given that symptoms persisted and became more frequent even with minimal head turning, surgery was recommended. Due to the indentation in the left VA in neutral position, VA decompression and C3–4 ACDF were chosen. A left-sided anterior approach was performed followed by C3–4 disectomy. Additionally, a high-speed drill was used to open the uncinate area on the left. The tubercle of the transverse foramen was exposed at C-3 and C-4, and the VA was completely unroofed at C-4 by microsurgical drilling with a diamond bur. A mini Doppler ultrasound probe was used intraoperatively to confirm good flow within the VA following decompression. Fusion was then performed at the C3–4 level by placing an Anatomic PEEK spacer (Medtronic Sofamor Danek), INFUSE Bone Graft (Medtronic) and recombinant human bone morphogenetic protein–2 strip (dose 0.53 mg), and a Venture cervical plate (Medtronic Sofamor Danek) with variable-angle screws. Bone morphogenetic protein–2 was used in the fusion to minimize the risk of pseudarthrosis that could lead to ongoing symptoms from rotational VA compression. Postoperatively, the patient

![Four-vessel cerebral angiograms. A: Right internal carotid artery angiogram, lateral view, demonstrating filling of the right posterior cerebral artery (PCA) and bilateral SCAs via the right PCoA. B: Left internal carotid artery angiogram, lateral view, demonstrating a fetal PCoA with transient contribution to the posterior circulation. C: Right VA angiogram, lateral view, showing narrowing of the VA at its dural penetration as well as an AICA-PICA configuration. D: Left VA angiogram, lateral view, showing the VA terminating in the PICA with no collateral supply to the left PICA territory. There is co-dominance of the right and left VAs.](image-url)
Rotational vertebral artery occlusion

recovered well and has not had any further symptoms when turning his head. The CTAs acquired 6 months after surgery showed no changes in the posterior circulation, specifically no focal stenosis, contour abnormality, or occlusion, on head turning, of the left VA (Fig. 5). The C3–4 graft was well incorporated and showed evidence of fusion across the interspace.

Discussion

History of RVAO (Bow Hunter’s Syndrome)

Sorensen first coined the term bow hunter’s stroke in 1978 to describe a patient that became symptomatic when his head turned during archery. However, descriptions of RVAO existed in the literature well before in what was referred to as cervical vertigo and characterized by vertigo, dizziness, and blurred vision with head rotation.

Mechanical compression of the VA during head rotation has been recognized to be a result of muscular or tendinous insertions, osteophytes, spondylosis, and segmental instability. The most common level of compression is atlantoaxial. This happens when the inferior facet of the atlas contralateral to the direction of rotation subluxates on the superior facet of the axis, thereby pulling the VA anteriorly and stretching it between the transverse foramen at C-1 and C-2. For such compression to be symptomatic, the contralateral VA or anterior circulation must be insufficient to provide compensatory flow. Cases of hypoplastic VA or other congenitally aberrant anatomy, dissection or previous injury, and atherosclerosis have all been associated with RVAO and symptomatic VBI.

Importance of Detailed Cervical and Cerebral Vascular Assessment

Although multiple studies can aid in the diagnosis of RVAO, the present case highlights the importance of dynamic catheter angiography to define the condition. Visualization of compression with head rotation must be evident on vertebral angiograms; however, care should be taken to ensure that stagnant contrast flow as a result of an upstream occlusion does not mischaracterize the actual level of compressive pathology. Aberrant posterior circulation anatomy can be a factor contributing to symp-

Fig. 3. Cervical MR images. Sagittal T2-weighted (left) and axial T2-weighted (right) sequences at the level of C3–4. A disc-osteophyte complex was present at C3–4, with indentation of the spinal cord and slight signal abnormality in the substance of the cord.

Fig. 4. 3D reconstruction of dynamic CTA. A C4–5 fusion is shown with adjacent C3–4 left-sided degenerative osteophyte arising from the uncovertebral joint. The image shows that with the head turned to the left, there is compression of the left VA (arrow) by the degenerative osteophyte. Also seen here is a relatively shorter distance between the C-4 transverse foramen and the uncinate process compared with other levels.
Symptoms of VBI, as is demonstrated in this case in which the left VA terminated in the PICA, and the right VA led to an AICA-PICA configuration. Full angiographic cervical and cerebral assessment therefore requires a 4-vessel examination, carefully assessing the VAs from their origin to the vertebrobasilar junction, the basilar artery, and the PCoAs. Bony imaging, ideally CT scanning, should also be performed to correlate extrinsic factors such as spondotic transverse foramina or osteophyte complexes that may cause subaxial compression.

Reported Sites of Compression

Multiple studies have reported symptomatic RVAO due to subaxial sites of compression (Table 1). In the present case, symptoms of VBI occurred about 20 years after the patient underwent C4–5 ACDF. Compression due to an uncovertebral osteophyte was found at the C3–4 level on the left side when the patient rotated his head to the left (Fig. 4). In the setting of aberrant vertebrobasilar anatomy with the left VA terminating in the PICA, contralateral narrowing at the dural penetration, and midbasilar occlusion, there was a not negligible risk of stroke with conservative therapy (cervical immobilization). Therefore, surgical treatment was indicated. A recently published treatment algorithm for bow hunter’s stroke by Cornelius et al.7 agrees with this strategy; due to a lack of evidence and risk of stroke, the authors discouraged conservative therapy with cervical collars.

Surgical Approaches for Management of RVAO

Various surgical approaches have been described as viable for decompression of the VA. These include anterior, posterior, and far-lateral routes.15 In many cases, fusion of the involved segments without decompression may be adequate for treatment.16 Such a strategy may help to minimize the risk of iatrogenic injury to a dominant, compressed VA. When decompression is deemed necessary (for example, with VA compression in a neutral position that is exacerbated on head turning), selection of the surgical approach depends on several factors: segmental level of the pathology, pathological relation to the VA in the sagittal plane (ventral or dorsal), biomechanical consequences, and surgeon preference should be considered.

At the classically described location of C1–2, fusion limits head rotation by 50%. Decompression without fusion is therefore commonly performed at this level. When fusion is needed (for example, in rheumatoid instability), instrumentation with greater risk of iatrogenic VA injury such as transarticular fixation should be avoided. Techniques for decompression all involve untethering of the VA, and both anterior and posterior approaches have been reported.10,12,25 Potential disadvantages of the anterior approach include unfamiliar soft-tissue anatomy and, more importantly, inability to address pathology in which a dominant VA is compressed posteriorly by the C-1 transverse foramen upon contralateral head rotation. Posterior approaches, therefore, are more appropriate in this typical situation. Disadvantages of posterior approaches include more extensive muscular destruction, incisional pain, higher intraoperative blood loss, and a longer recovery period. To mitigate this, a minimally invasive technique can be used for posterior VA decompression, as described by Lu et al.16 In general, we advocate for a posterior approach in cases in which the pathological entity is cephalad to C-3.

Conversely, an anterior approach for pathology caudal to C-3 can be conveniently achieved via a Smith-Robinson approach for discectomy and fusion. In cases in which compression is also present in a neutral position, unroofing the transverse foramen to further untether the VA can be concurrently performed. This is achieved by subperiosteal dissection to mobilize the longus colli laterally up to the anterior tubercle. Mobilization of the longus colli in this manner serves to protect the sympathetic ganglia lying on the lateral aspect of this muscle. Dissection should not proceed beyond the anterior tubercle posterolaterally so as to avoid injury to the cervical nerve roots. A high-speed drill with a diamond bur is then used to unroof the transverse foramen. Flow through the VA should be assessed periodically during dissection and drilling as well as after complete decompression using by a mini-Doppler ultrasound probe. Iatrogenic injury to the VA requires microvascular repair. Although ligation

Fig. 5. CTAs obtained 6 months postoperatively. **Left:** Sagittal image revealing evidence of fusion at C3–4. **Right:** Axial image at the level of C3–4; arrow denotes full caliber of the left VA after decompression.
TABLE 1: Summary of literature reporting RVAO from subaxial spondylotic compression*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Cases</th>
<th>Presenting Symptoms</th>
<th>Imaging</th>
<th>Dynamic Angiogram</th>
<th>Compressive Lesion</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bakay &amp; Leslie, 1965</td>
<td>2</td>
<td>dizziness, syncope on neck extension/rotation</td>
<td>x-ray, myelogram, DSA</td>
<td>yes</td>
<td>C5–6 osteophyte</td>
<td>ACDF, decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Nagashima, 1970</td>
<td>20</td>
<td>vertigo, dizziness, syncope on neck extension/rotation</td>
<td>x-ray, DSA</td>
<td>yes</td>
<td>C4–5, C5–6, C6–7 osteophytes, 5 cases bilateral</td>
<td>anterior decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Smith et al., 1971</td>
<td>2</td>
<td>vertigo, syncope on neck extension</td>
<td>x-ray, myelogram, DSA</td>
<td>no</td>
<td>bilateral C4–5 &amp; C5–6 osteophytes</td>
<td>ACDF, decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Sullivan et al., 1975</td>
<td>1</td>
<td>stroke, lt homonymous hemianopsia</td>
<td>x-ray, myelogram, DSA</td>
<td>no</td>
<td>C5–6 osteophyte</td>
<td>ACDF, decompression</td>
<td>unchanged</td>
</tr>
<tr>
<td>Chin, 1993</td>
<td>1</td>
<td>stroke, lt homonymous hemianopsia</td>
<td>CT, DSA</td>
<td>no</td>
<td>C4–5 osteophyte</td>
<td>anterior decompression</td>
<td>unchanged</td>
</tr>
<tr>
<td>Kawaguchi et al., 1997</td>
<td>1</td>
<td>blindness on head turning to rt</td>
<td>CTA, DSA</td>
<td>yes</td>
<td>C4–5 osteophyte</td>
<td>ACDF, decompression</td>
<td>recurrence of symptoms (angiographically misidentified level of compression at C5–6); required revision at C4–5, no further symptoms</td>
</tr>
<tr>
<td>Citow &amp; Macdonald, 1999</td>
<td>1</td>
<td>vertigo on neck extension</td>
<td>CT, DSA</td>
<td>no</td>
<td>C5–6 osteophyte</td>
<td>posterior decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Ogino et al., 2001</td>
<td>1</td>
<td>vertigo on head turning to rt</td>
<td>CT, DSA</td>
<td>yes</td>
<td>C3–4 osteophyte</td>
<td>anterior decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Vates et al., 2002</td>
<td>1</td>
<td>dizziness, syncope on head turning to lt</td>
<td>x-ray, MRI, DSA, TCD</td>
<td>yes</td>
<td>C4–5 disc herniation</td>
<td>anterior decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Nemecek et al., 2003</td>
<td>1</td>
<td>vertigo, nausea, syncope on head turning to lt</td>
<td>MRI, CTA, DSA, TCD</td>
<td>yes</td>
<td>C6–7 disc herniation</td>
<td>anterior decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Bulsara et al., 2006</td>
<td>1</td>
<td>syncope on head turning to rt</td>
<td>CT, MRI, CTA, DSA</td>
<td>yes</td>
<td>C5–6 osteophyte</td>
<td>ACDF, decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Velat et al., 2006</td>
<td>1</td>
<td>dizziness, syncope on head turning to lt</td>
<td>CTA, DSA</td>
<td>yes</td>
<td>C4–5 &amp; C5–6 osteophytes</td>
<td>anterior decompression</td>
<td>intraop angiogram confirmed decompression; no further symptoms</td>
</tr>
<tr>
<td>Miele et al., 2008</td>
<td>1</td>
<td>syncope on head turning to lt</td>
<td>CT, MRI, DSA</td>
<td>yes</td>
<td>C4–5 osteophyte</td>
<td>ACDF, decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Ujifuku et al., 2009</td>
<td>1</td>
<td>blurred vision, syncope on head turning to lt</td>
<td>CTA, DSA</td>
<td>yes</td>
<td>C4–5 disc/osteophyte</td>
<td>anterior decompression</td>
<td>intraop angiogram confirmed decompression; no further symptoms</td>
</tr>
<tr>
<td>Lu et al., 2010</td>
<td>4</td>
<td>syncope, vertigo</td>
<td>CTA, DSA</td>
<td>yes</td>
<td>C4–5 osteophytes, C5–6 disc herniation</td>
<td>anterior decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Yoshihara et al., 2011</td>
<td>1</td>
<td>dizziness on head rotation to rt</td>
<td>CT, MRI, DSA</td>
<td>yes</td>
<td>C3–4 uncovertebral instability</td>
<td>ACDF</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Fleming et al., 2013</td>
<td>1</td>
<td>dizziness, diplopia, tinnitus on head rotation</td>
<td>CTA, DSA</td>
<td>yes</td>
<td>bilateral C4–5 osteophytes</td>
<td>ACDF, decompression</td>
<td>no further symptoms</td>
</tr>
<tr>
<td>Schellfaut et al., 2013</td>
<td>1</td>
<td>transient VBI</td>
<td>CTA</td>
<td>no</td>
<td>C5–6 osteophyte</td>
<td>ACDF</td>
<td>no further symptoms</td>
</tr>
</tbody>
</table>

* DSA = digital subtraction angiogram; TCD = transcranial Doppler.
is an option for managing inadvertent VA injury during cervical spine surgery for other pathologies, this strategy should not be considered in the setting of RVOA due to inevitable stroke. If microvascular repair is not possible, a tamponade with hemostatic agents followed by immediate endovascular evaluation for stent reconstruction may be considered as a “bail-out” option.

Some consideration should be made for the direction of VA compression, which in the subaxial spine is most often due to uncovertebral osteophytes (ventral to the VA). Although infrequent, facet osteophytes (dorsal to the VA) can produce VA compression, typically upon neck extension. Unroofing the transverse foramen anteriorly in this situation allows the VA to mobilize away from the posteriory compressive osteophyte. Alternatively, direct posterior decompression may be used. Table 1 summarizes the available literature on surgical treatment of subaxial RVAO. Like most authors, we generally select an anterior approach for pathology caudal to C-3.

It is noteworthy that previous studies have indicated the vulnerability of VA to compression at C-4, explained by the relatively short distance between the uncinate process and the transverse foramen at this level. That adjacent-segment disease at C3–4 and not C5–6 resulted in RVAO in our case further supports the concept of VA vulnerability to C-4 compression. Lastly, it is important to highlight that symptomatic RVAO is typically associated with either congenital or acquired intrinsic vertebrobasilar pathology, requiring a detailed assessment of cervical and cerebral vasculature. Therefore, long-term patient management must include ongoing stroke prevention as outlined in American Heart Association/American Stroke Association guidelines.

Conclusions

Adjacent-segment degeneration can be a cause of RVAO. Recognition of such nonspecific symptoms as vertigo, dizziness, and nausea as symptoms of VBI in this scenario may lead to appropriate investigation documenting the site of compression and vertebrobasilar anatomy, expedited diagnosis, and targeted treatment. As in other reports of subaxial RVAO, successful treatment was achieved by ACDF at the affected level with concurrent microsurgical drilling of the transverse foramen for decompression of the VA.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: all authors. Acquisition of data: Buchanan, McLaughlin. Analysis and interpretation of data: Buchanan, McLaughlin. Drafting the article: Buchanan. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors.

References


Rotational vertebral artery occlusion


Accepted March 6, 2014.

Please include this information when citing this paper: published online April 18, 2014; DOI: 10.3171/2014.3.SPINE13452.
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