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CASE REPORT

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Rotational vertebral artery occlusion: Case report and review of literature

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Abstract

Rotational vertebral artery occlusion (RVAO) classically involves transient, position-dependent vertebrobasilar insufficiency (VBI) that occurs when an extra-vascular lesion (e.g. osteophyte or fibromuscular band) compresses a dominant vertebral artery with turning of the head to one side. Our patient presented with VBI associated vertigo, dizziness, and lightheadedness that occurred when her head was turned to the right. RVAO was initially suggested by transcranial Doppler ultrasound (TCD) changes that were not supported by initial catheter angiography. After her symptoms worsened over a course of two years, the diagnosis was confirmed with repeat angiography with head rotation. Further imaging with computed tomography and magnetic resonance demonstrated spondylosis at the C5-C6 vertebrae and an osteophyte near the C5 transverse foramen, which caused position-dependent extra-vascular compression. She was treated with surgical decompression and anterior discectomy and fusion at C5-C6. The unique anatomical pathology of this case combined with the diagnostic discrepancy between early TCD and angiography make it an interesting contribution to the otherwise limited body of literature on RVAO.

Keywords: *vertebral artery, vertebrobasillar artery insufficiency, vertebral artery compression, rotational vertebral artery occlusion (RVAO), bow hunter syndrome (BHS), TCD, cervical spinal degeneration*

Case report

This case involves a 50-year-old right-handed Caucasian female with a remote past medical history of subarachnoid hemorrhage and aneurysm clip ligation. She presented with worsening vertigo and dysarthria occurring when turning her head to the right. Symptoms occurred with slight head turns to the right, with frequency and severity affecting ability to drive.

She initially presented with episodes of vertigo, dizziness, lightheadedness, sensation of leaning to the left, and dysarthria when turning her head to the extreme right. Episodes began 6 years prior, initially occurring every few months. In the year leading up to presentation, frequency of episodes increased to monthly, then, in the 2 weeks before presentation, episodes were daily. Physical exam demonstrated rightward nystagmus with head turned to the right. Initial diagnostic workup included computed tomography angiogram (CTA) of the head and neck, which were without significant findings. A transcranial Doppler showed decreased signals in posterior cerebral arteries bilaterally with turning head to the right, with signal intensity normalizing after patient's head returned to neutral position. Angiography at that time demonstrated dominant left vertebral artery (LVA) with diminutive right vertebral artery (RVA) terminating in the posterior inferior cerebellar artery with 25% narrowing when head turned to the right. Her diagnosis was suspected to be Rotational Vertebral Artery Occlusion (RVAO, also known as "Bow Hunter's Syndrome"), though angiography findings were not supportive. She was initiated on aspirin and Lipitor 80 mg.

She returned to clinic for evaluation approximately two years later, during which interval her symptoms had progressed to the point that episodes could be provoked with even relatively minor head movements. Magnetic resonance imaging (MRI) was obtained, showing mild cervical spondylosis at C5-C6 with short pedicles contributing to significant spinal stenosis, but otherwise without high-grade neural effacement. A repeat catheter angiogram was performed with selective injection of the vertebral arteries bilaterally with the head straight, and then turned to the right. This demonstrated near complete occlusion of RVA at the level of C5-C6 when turning head to the right (Figure 1). These findings confirmed the diagnosis of Rotational Vertebral Artery Occlusion (RVAO). Subsequent CT of the neck demonstrated an osteophyte at the medial side of C5 transverse foramen impinging on the right vertebral artery (Figure 2).

Anterior cervical discectomy and fusion of C5-C6 was performed shortly thereafter. At 1 month follow up postoperatively, patient stated she was feeling better overall, that she could turn her head, though she was still walking with the aid of a cane and wearing a neck brace.

Literature review

PRVAO is a syndrome of transient vertebrobasilar insufficiency (VBI) that occurs with head turning due to dynamic extravascular compression of a vertebral artery as the head is turned. It was originally described in a case report by Sorenson in 1978, where a patient developed a posterior circulation infarct during archery practice (hence, it is also known as "Bow Hunter Syndrome") [1]. The position-dependent nature of this syndrome is unique in neurology. However, its underlying pathophysiologic mechanism has analogues throughout medicine. For example, in thoracic outlet syndrome, radial artery pulselessness can be produced with the patient's head extended and turned during maximal inspiration-a classic exam maneuver called Adson's test. The mechanism of

Adson's test is position-exacerbated neurovascular compression at the subclavian artery by the scalene muscles, clavicle, and/or first rib [2,3]. Functionally, the only difference between Adson's test and RVAO is that the location of the lesion and the blood vessel affected leads to an incidental exam finding in thoracic outlet syndrome and incapacitating neurologic symptoms in RVAO.

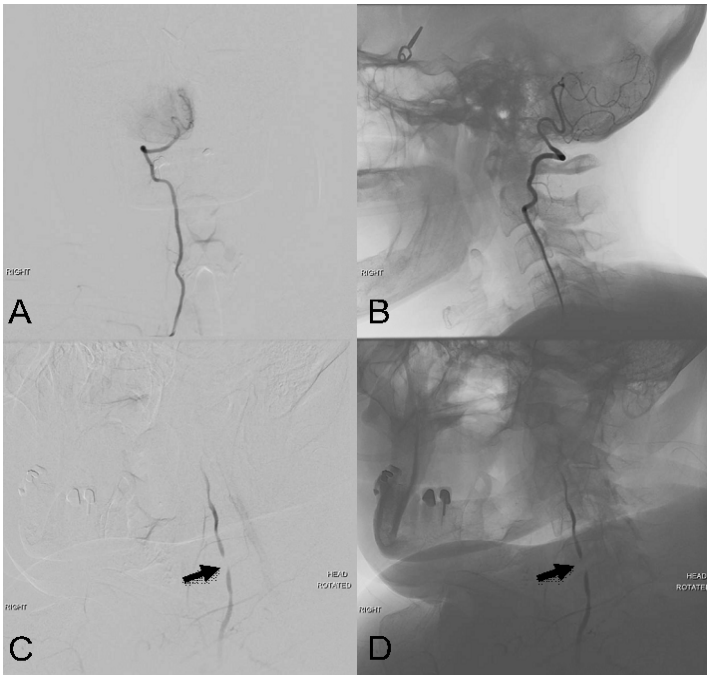

Figure 1. *Diagnostic angiogram. A) RVA angiogram with head in neutral position (anteroposterior view). B) RVA angiogram with head in neutral position (lateral). C&D) RVA angiogram with patient's head turned to the right, with near occlusion demonstrated by arrows (anteroposterior view).*

Reported cases of RVAO are limited, and so exact figures for incidence and prevalence are difficult to establish. Available case series reviews have suggested that RVAO most often occurs in the 5th-7th decade of life, with a 2:1 male to female predominance [1,4]. However, RVAO can occur at any age, with cases reported for patients as young as 15-years [5,6]. Frequently occurring comorbidities include hypertension, hyperlipidemia, obesity, osteoarthritis, diabetes, smoking, and coronary artery disease. Patients tend to have 2-3 of these comorbidities [1].

The gold standard of diagnosis for RVAO is angiography with head rotation (dynamic angiography), which will demonstrate dynamic occlusion of the affected VA [1,4,7]. Trans-cranial Doppler (TCD) ultrasonography can be used as a non-invasive method for demonstrating dynamic occlusion, as positional changes in posterior circulation perfusion patterns can be observed [1,8]. The use of CT and MR is limited in that neither modality can be used to demonstrate dynamic occlusion [1]. However, CT and/or MR are essential for characterizing the nature of the extravascular lesion causing dynamic occlusion, thereby facilitating intervention if necessary.

Treatments range from conservative medical management to surgical intervention. Conservative management employs anti-platelet medication (e.g. aspirin, clopidogrel, cilostazol) [4] as well as avoidance of head-turning, often aided with a neck brace. Anti-coagulation (e.g. warfarin) has been recommended in some cases [1,4,8]. with more frequent and worsening symptoms, surgical

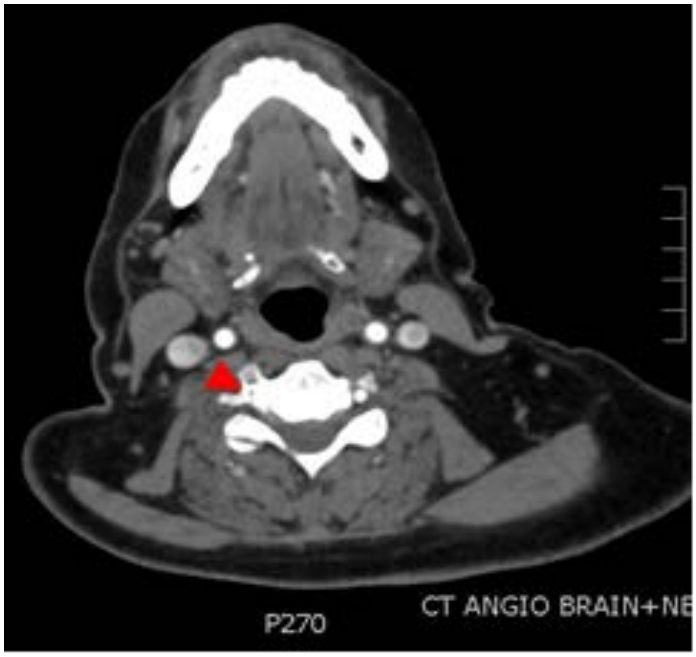

Figure 2. *CTA, head and neck, arrowhead demonstrating osteophyte near right transverse foramen of C5. This lesion was determined to be source of extra-vascular compression for our patient.*

interventions may be recommended. These interventions include decompression of the VA at the identified lesion, discectomy and fusion of the associated vertebral joint, or a combination of both procedures. Decompression with or without fusion has been associated with resolution of symptoms and more favorable outcomes than conservative management [1,7]. Endovascular stenting has also been utilized as a therapeutic intervention in a limited number of reported cases.1 Such cases have included classic RVAO with extra-vascular VA compression, as well as cases with underlying VA stenosis or vascular anomalies, such as segmental tortuosity of the VA [10,11]. While stenting in these cases was associated with clinical improvement, the literature lacks sufficient long-term data on outcomes associated this treatment modality compared to surgical intervention.

Conclusion

The case presented above represents an uncommon presentation of RVAO. The typical presentation involves extravascular occlusion of a dominant VA, thereby disrupting a significant portion of vertebrobasilar blood flow. Our patient, however, presented with a lesion compressing her non-dominant RVA. It is worth noting again that this RVA terminated in our patient's right PICA. It is likely her RVAO was facilitated by this anomaly, which may have created an intrinsic vertebrobasilar anastomotic deficiency. Additionally, our patient presented with a lesion at C5. This finding adds to the growing body of reported cases suggesting lesions affecting C3-C7 may be just as common as those affecting C1-C2. It is also interesting to note that her pathophysiology was initially suggested by TCD, though angiography at that time was not diagnostic of RVAO. Only two years later, when her symptoms had substantially worsened, was RVAO confirmed by gold standard dynamic angiography. Given early identification with TCD, it would be interesting to research the sensitivity of this imaging modality if a large enough sample of such data was available in the current

literature. This low-cost, minimally invasive imaging modality may prove to be an effective first-line test for detecting RVAO. Further, these qualities could make TCD favorable for assessing post-surgical vascular dynamics. Otherwise, our patient's workup and treatment were consistent with the standard supported by the reviewed literature. CT and MR identified her syndrome's etiology as cervical spondylosis and osteophytic compression. Surgical intervention with decompression plus a discectomy and fusion of C5-C6 led the patient to report subjective symptomatic improvement at follow-up.

Key points

- RVAO classically involves transient, position-dependent vertebrobasilar insufficiency that occurs when an extra-vascular lesion (e.g. osteophyte or fibromuscular band) compresses a dominant vertebral artery with turning the head to one side.
- The pathophysiology of RVAO is analogous to vascular thoracic outlet syndrome, where the narrowed thoracic outlet can produce extra-vascular compression on the subclavian artery with position changes.
- The epidemiology of RVAO is not well known, as there is a limited body of cases to draw from. Its incidence and prevalence are not established, though there appears to be a 2:1 male-to-female predominance.
- Our patient presents with a rare RVAO caused by compression of a non-dominant VA with termination in PICA.
- Our patient's presentation also adds to a growing body of cases with culprit lesions occurring between C3-C7.
- TCD has the potential to be a cost-effective, minimally invasive first-line screening test for RVAO.
- Treatment ranges from supportive measures to surgical decompression of the lesion with or without discectomy and fusion.
- VA stenting should be considered in patients where a vascular pathology (e.g. stenosis or tortuosity) is suspected to be the etiology. Its use as a treatment for classic extra-vascular RVAO should continue to be explored.

References

1. 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. *Interv Neurol.* 2016; 5: 29-38.
2. Braun RM, Rechnic M, Shah KN. Pulse oximetry measurements in the evaluation of patients with possible thoracic outlet syndrome. *J Hand Surg Am.* 2012; 37: 2564-2569.
3. Lee AD, Agarwal S, Sadhu D. Doppler Adson's Test: Predictor of Outcome of Surgery in Non-Specific Thoracic Outlet Syndrome. *World J Surg.* 2006; 30: 291-292.
4. Choi KD, Choi JH, Kim JS, et al. Rotational vertebral artery occlusion: Mechanisms and long-term outcome. *Stroke.* 2013; 44: 1817-1824.
5. Anene-Maidoh TI, Vega RA, Fautheree GL, Reavey-Cantwell JF. An unusual case of pediatric bow hunter's stroke. *Surg Neurol Int.* 2013; 4: 148.
6. Greiner HM, Abruzzo TA, Kabbouche M, Leach JL, Zuccarello M. Rotational vertebral artery occlusion in a child with multiple strokes: A case-based update. *Child's Nerv Syst.* 2010; 26: 1669-1674.
7. Strickland BA, Pham MH, Bakhsheshian J, Russin JJ, Mack WJ, et al. Bow Hunter's Syndrome: Surgical Management (Video) and Review of the Literature. *World Neurosurg*. 2017; 103: 953.e7-953.e12.
8. Rastogi V, Rawls A, Moore O, Victorica B, Khan S, et al. Rare Etiology of Bow Hunter's Syndrome and Systematic Review of Literature. *J Vasc Interv Neurol*. 2015; 8: 7-16.
9. Haimoto S, Nishimura Y, Hara M, Yamamoto Y, Fukuoka T, et al. Surgical Treatment of Rotational Vertebral Artery Syndrome Induced by Spinal Tumor: A Case Report and Literature Review. *NMC Case Rep J.* 2017; 4: 101-105.
10. Darkhabani MZ, Thompson MC, Lazzaro MA, Taqi MA, Zaidat OO. Vertebral artery stenting for the treatment of bow hunter's syndrome: Report of 4 cases. *J Stroke Cerebrovasc Dis.* 2012; 21: 908.e1-908.e5.
11. Motiei-Langroudi R, Griessenauer CJ, Alturki A, Adeeb N, Thomas AJ, et al. Bow Hunter's Syndrome from a Tortuous V1 Segment Vertebral Artery Treated with Stent Placement. *World Neurosurg.* 2017; 98: 878.e11-878.e15.

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