Verteobasilar insufficiency (VBI) is a condition characterized by reduced blood flow through the posterior circulation of the brain. The most common presentation of VBI is intermittent syncope or near-syncope often associated with a secondary fall (ie, a “drop attack”). Other possible presentations include nausea, visual changes, gait disturbance, dysarthria, and dysphagia. Rotational vertebral artery (VA) occlusion (RVAO) is a possible cause of VBI and is due to compression of the VA on head turning when blood flow through the posterior circulation of the brain is compromised. RVAO can be classified into three types: extraosseous segment type (type I), in which the VA is compressed by the thyrocervical trunk and medial border of the scalenus anticus muscle; subaxial spinal type (type II), in which the VA is compressed in an area between the foramen transversarium of C6 and C2; and craniovertebral junction type (type III), in which the VA is compressed by the thyroid cartilage. We present a unique case of a patient with a completely extraosseous course of the V2 segment of her right dominant VA that resulted in symptomatic rotational VA occlusion. (J Vasc Surg Cases and Innovative Techniques 2019;5:14-7.)

Keywords: Vertebral artery; Compression; Vertebrobasilar insufficiency; Syncope

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to be redundant inferiorly. It was not redundant superiorly, but
the levator scapulae was firmly adherent to it, creating signifi-
cant compression. Preoperative CTA demonstrated compres-
sion from the thyroid cartilage only, but on surgical
exploration, it was clear that compression by the levator scap-
ulae was also occurring, although the significance was unclear.
This did not appear to change with head rotation, but the
patient was in the supine position for surgery and not in an
upright functional position (Fig 2). This muscle was divided
and the artery was released, resolving the compression.

The posterior superior projection of the right thyroid cartilage
could be easily felt and was located precisely at the area on
the computed tomography scan that showed the rotationally
induced compression (Fig 3). The thyrohyoid muscle was incised,
and a 12-mm segment of the posterior superior projection of the
right thyroid cartilage was resected. The patient’s head was then
rotated to the right to test the area for residual compression.
There was now plenty of space between the thyroid cartilage
remnant and the VA. Doppler signals in the VA did not change
with head rotation and were normal in all positions.

Next, an intraoperative duplex ultrasound scan of the VA was
performed that did not identify any endoluminal fibrosis or
stenoses in the VA. The patient was awakened, found to be
neurologically intact, and taken to the recovery room. She had
an unremarkable postoperative course that was significant for
immediate and complete relief of RVAO symptoms. The patient
continues to be asymptomatic 8 months after surgery. Provoca-
tive postoperative computed tomography demonstrated no
evidence of extrinsic compression (Fig 4).

**DISCUSSION**

RVAO is a term encompassing all conditions that result
in VA stenosis or occlusion with cervical rotation. Bow
hunter’s syndrome is one of these conditions and is
characterized by symptoms of VBI on head rotation
secondary to occlusion of the contralateral VA. It is
more common for occlusion of the VA to occur above
the C2 level. VA compression is usually asymptomatic,
but if blood flow from the contralateral VA is compro-
mised by hypoplasia, stenosis, or atresia, symptoms of
VBI can develop. In this case, the patient had a
hypoplastic left VA along with compression of the right
V2 segment, which resulted in reproducible symptoms.
of VBI on head rotation to the right. Compression of the V2 segment of the VA is most commonly due to cervical osteophytes, but in this case, the narrowing of the right VA was due to extrinsic compression from the thyroid cartilage and possibly the levator scapulae muscle.

The VA is known for having variable anatomy. The most common anatomic variation of the VA is origin off the aortic arch between the left common carotid and left subclavian arteries, occurring in 6% to 8% of individuals. The VA can also vary anatomically by originating from the ipsilateral common carotid artery. In addition, the VA may terminate in the posterior inferior cerebellar artery, failing to contribute to the circle of Willis. The V2 segment of the VA, also called the transversary segment, normally ascends from the cervical vertebral foramen at C6 up through the transverse foramina to C2. This case describes a completely extraosseous V2 segment of the VA. The authors conducted a literature search using Ovid MEDLINE and PubMed databases. The following search terms were used: free text “vertebral artery,” “anatom*,” “anatom* varia*”; and subheadings “vertebral artery” and “anatomic variation.” A separate literature search was undertaken to search for papers reporting extraosseous VA, which used the additional search terms “extra-osseous,” “extra osseous,” and “V2.” This variant has been scarcely reported in patients with RVAO. On embryologic development, V2 VA variants are due to persistence of cervical intersegmental arteries. In our case, the extraosseous VA originated embryologically from the deep cervical artery.

The most common causes of compression of the V2 segment of the VA are osteophytes in the cervical spine (in the mid V2 segment) and impingement by the tendon of the longus colli muscle in the proximal V2 segment. In this case, the unprotected and unrestricted positioning of the V2 segment allowed this portion of the VA to become redundant, predisposing it to compression by the levator scapulae (in the distal V2 segment) and the adjacent segment of the thyroid cartilage. This intrinsic compression by the thyroid cartilage was only possible because of the completely extraosseous course of the V2 segment; it has not previously been reported for V2 compression syndromes.

The role of the levator scapulae is unclear. It appeared compressive at the time of surgery, but this was not evident on preoperative CTA. The compression from the levator scapulae muscle was visualized with the patient’s head in the neutral position and did not change with the patient’s head turned while in the supine position. The compression from the levator scapulae muscle on the VA with the patient in the upright position and the muscle activated for craniocervical stability and motion would conceivably be different, and for this reason we chose to divide the muscle. It is unclear why the patient remained asymptomatic for 75 years, but it is most likely the progressive elongation of the VA that led to eventual rotationally induced compression by the thyroid cartilage. Division of the levator scapulae and resection of the impinging portion of thyroid cartilage resulted in resolution of the RVAO.

The initial treatment modality of posterior circulation disease is medical; it is recommended that all patients be prescribed antiplatelet or anticoagulation therapy and have appropriate treatment of an anterior circulation (ie, carotid artery) disease. However, there is no medical therapy for rotationally induced VA occlusive disease. Endovascular repair with a stent was deemed inappropriate because of the potential for recurrent compression of the stent on head turning. Two possible surgical interventions were proposed: a carotid artery bypass to the V3 segment of the VA vs vertebral arteriosclerosis with resection of the offending structure. Extensive

Fig 4. Comparison of the preoperative (A) and postoperative (B) volume rendered computed tomography angiograms of the vertebral artery (VA; arrows) demonstrates that segmental removal of the thyroid cartilage has resolved the rotationally induced compression.
arteriolysis of the extraosseous VA with resection of the offending compressive structure was determined to be the safer, less complex, and more durable option compared with a bypass.

CONCLUSIONS

When evaluating patients for RVAO, vascular surgeons should be aware of this anatomic variant as a potential cause. Surgical correction with arteriolysis of the VA and resection of the adjacent compressive structures results in symptomatic relief.

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