Anterolateral decompression of the atlantoaxial vertebral artery for symptomatic positional occlusion of the vertebral artery

Case report

MARK W. FOX, M.D., DAVID G. PIEPGRAS, M.D., AND JOHN D. BARTLESON, M.D.

Departments of Neurosurgery and Neurology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

A case of repeated vertebrobasilar ischemic attacks related to head rotation (bow hunter’s stroke) is reported. With head rotation of 45° or more to the right, the patient would become lightheaded and feel as if she were going to lose consciousness. Angiography performed when head rotation was to the right revealed mechanical compression of the left vertebral artery at the foramen transversarium of the axis and an occluded right vertebral artery. Untethering of the vertebral artery as it passed through the foramen transversarium of the atlas in this case completely relieved the patient’s symptoms. The authors conclude that contralateral vertebral artery occlusion predisposed this patient to symptomatic vertebrobasilar insufficiency secondary to ipsilateral vertebral artery mechanical stenosis induced by head turning.

Key Words • vertebrobasilar insufficiency • vertebral artery • bow hunter’s stroke • decompression surgery

TEMPORARY occlusion or mechanical compression of the atlantoaxial portion of the vertebral artery (VA) caused by head rotation has been documented during routine cerebral angiography.\textsuperscript{1,2,5,7,10,11,15,16,19} This occlusion is usually asymptomatic due to sufficient contralateral VA or distal vertebrobasilar system collateral flow. The term “bow hunter’s stroke” has been used to describe a syndrome of hemodynamic vertebrobasilar insufficiency induced by forced or voluntary rotational head movements causing intermittent VA compression at the atlantoaxial level.\textsuperscript{9,13,17,18}

We report an additional case of a patient with recurrent vertebrobasilar ischemic episodes that occurred with voluntary head turning. In the past, neck bracing or atlantoaxial fusions have been advocated for patients with this condition to prevent axial rotation at the C1–2 level.\textsuperscript{3,16,17,19} In 1988, Shimizu, \textit{et al.},\textsuperscript{17} and Hanakita, \textit{et al.},\textsuperscript{4} described a posterior decompressive approach to the atlantoaxial VA for bow hunter’s stroke. Herein we describe an anterolateral approach to the VA at the atlantoaxial level.

Case Report

This 53-year-old woman slipped on ice and fell backward striking her upper back in December 1990. She denied head, neck, or eye discomfort at that time. During the 2nd week following her fall she developed tinnitus in her right ear and intermittent lightheadedness that occurred while turning her head to the right. For 3 months following her injury, she avoided this movement. The patient then experienced a near syncopal episode with associated nausea and blurred vision, which prompted her to seek medical attention. This event occurred within seconds after turning her head to the right approximately 45°. Returning to a neutral head position reversed her symptoms within 15 seconds.

\textit{Examination.} The patient’s neurological evaluation at our institution was completely normal. She underwent cerebral angiography in the neutral position, which revealed a right VA occlusion (Fig. 1 left). Dynamic angiography with the patient’s head rotated 45° to the right reproduced
her symptoms and documented mechanical compression of the left VA at the level of the C-2 foramen transversarium (Fig. 1 right). Surgical options for decompression of the left VA were discussed with the patient. In part because of the patient’s known cardiovascular risk factors, it was mutually agreed to continue conservative therapy consisting of limited head turning. After 6 months, however, the patient found that the restriction constituted a major impairment to her lifestyle and elected to proceed with surgical treatment.

Operation. With the patient supine and her head turned 10˚ to the right, which would not induce symptoms preoperatively, a linear longitudinal incision was made on the anterior edge of the left sternocleidomastoid muscle beginning at the mastoid process (Fig. 2 left). The sternocleidomastoid and splenius capitis muscles were retracted posteriorly. The wound was deepened into the retrojugular space, that is, posterior to the internal jugular vein and the carotid sheath. The accessory nerve was identified as it entered the posterior and medial aspect of the sternocleidomastoid (2–3 cm below the mastoid tip) and was mobilized (Fig. 2 right). The tip of the transverse process of the atlas was then palpated through the prevertebral fascia approximately 3 cm below the mastoid tip. The fascia was incised obliquely in a line paralleling the accessory nerve to expose the thick slip of levator scapulae muscle attached to the transverse process of the atlas. The levator scapulae, cervical splenius, and intertransverse muscle attachment to the atlas were then taken down and reflected laterally and posteriorly. The anterior ramus of C-1 was identified and followed into its foramen where it coursed over the VA. The foramen transversarium of the atlas was then drilled away, untethering the left VA. No unusual bone, muscular, ligamentous, or vascular pathology was observed. Following this decompression, the VA was dissected inferiorly to identify its entrance into the foramen transversarium of the axis. The ventral ramus of C-2 was encountered as it coursed across the VA between the atlas and axis. Intraoperative rotation of the patient’s head revealed no active kinking of the VA. The dense venous plexus around the VA was cautiously excised using cautery and oxidized cellulose packing. The wound was then closed in anatomical layers.

Postoperative Course. The postoperative hospital course was uncomplicated. On the 3rd day following surgery, the patient was allowed to turn her head to the right. No rep-
Anterolateral decompression of the atlantoaxial VA

Fig. 3. Postoperative computerized tomography scan through the atlas demonstrating operative removal of the foramen transversarium on the left.

etition of preoperative symptoms could be elicited. Computerized tomography scanning of the atlas revealed adequate decompression of the VA at this level (Fig. 3).

Two weeks after surgery, the patient developed left shoulder pain that proved to be caused by an accessory nerve palsy affecting both her sternocleidomastoid and trapezius muscles. She had winging of her left scapula as well. Her shoulder pain was believed to be due to both the nerve palsy and shoulder capsulitis secondary to altered shoulder mechanics from her trapezius muscle weakness. Shoulder immobilization followed by gentle physical therapy and a course of antiinflammatory agents have been helpful in providing adequate pain relief. At 6-month follow up, the patient had resolution of her pain but still had moderate weakness and immobility of her left shoulder. She continued to have freedom of neck and head movements without indication of vertebrobasilar ischemic symptoms.

Discussion

Positional temporary occlusion of one VA in the course of daily activities rarely produces any major effects on posterior circulation blood flow.1,5,10,17 However, poor or absent unilateral VA blood flow may predispose patients to vertebrobasilar ischemic attacks during head and neck rotation.1,5,17 Husni and Storer10 reported 23 patients with vertebrobasilar insufficiency induced by rotational occlusion of one VA. The opposite VA was hypoplastic or absent in 22 patients and narrowed at its origin in one.

In the case reported here, we suspect the patient’s contralateral VA became occluded at the time of her fall. This was most likely secondary to a traumatic dissection, although this could not be confirmed by her previous imaging studies. We hypothesize that the dynamic narrowing of her left VA at the C1–2 level was a premorbid condition that was rendered symptomatic with traumatic loss of the right VA blood flow. In this case, release of the induced VA compression and stenosis at C-2 was achieved by a release of the VA and rotational forces at C-1 and did not necessitate decompression of the C-2 foramen transversarium per se.

The VA at the atlantoaxial level is particularly prone to mechanical compression by head and neck rotation because of its unique relationship to the surrounding transverse foramina, paravertebral muscles, and fibrous ligaments.1,5,17 Axial rotation at the C1–2 level may cause VA occlusion within normal ranges of motion due to the relatively fixed position of the artery within the posterior arch of the atlas.3,5,17 During angiography Bauer, et al.,3 have commonly observed VA compression as it passes over the lateral mass of C-1 and consider this “within normal limits.” Head rotation to the right results in fixation of the right atlantoaxial joint, while the atlas moves forward on the axis on the left side. The segment of VA between C-1 and C-2 is stretched, and in some cases, is narrowed or occluded in the process.2 Atlantoaxial instability,5 ossification or hypertrophy of the atlantooccipital membrane,6,17 tightness of the paravertebral musculature,5,11,18 or severe changes of spondylosis4 may also contribute to VA compression.

Treatment alternatives for rotational compression of the atlantoaxial portion of the VA have included verbal warnings or braces to restrict head and neck rotation,3,18 surgical fusions to prevent atlantoaxial rotation,5,10,18 and decompression of the VA at the C1–2 level.5,17 This case indicates that simple surgical untethering of the VA at the C-1 foramen transversarium can be definitive therapy, avoiding fusion procedures associated with permanent reduction in axial rotation. An anterolateral approach to the atlantoaxial segment of the VA5,12 provided adequate exposure and decompression resulting in complete relief of the patient’s symptoms. Spinal accessory nerve palsy is an observed complication of this approach.8,12

Conclusions

Occlusion of one VA may predispose patients to vertebrobasilar ischemic attacks due to rotational obstruction of the contralateral VA at the C1–2 level. Dynamic angiography usually reveals the abnormality. Anterolateral surgical decompression, in this case untethering at C-1, provides a useful surgical option.

References


Manuscript received October 4, 1993. Accepted in final form February 2, 1995.
Address reprint requests to: David G. Piepgras, M.D., Department of Neurosurgery, Mayo Clinic, 200 First Street, SW, Rochester, Minnesota 55905.