Decompression of the vertebral artery for bow-hunter’s stroke

Case report

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A case of cerebellar infarction induced by repeated neck rotation (“bow-hunter’s stroke”) is reported. The most likely mechanism is that repeated rotational neck movement brings about thickening of the atlanto-occipital membrane, fixing the vertebral artery in the vascular groove of the atlas. The vertebral artery is thus pinched at the time of neck rotation, leading to thrombus formation. Embolization results in cerebellar infarction. Surgical decompression of the vertebral artery at the level of the atlas in this case relieved the symptoms, and postoperative angiography demonstrated good flow within the vertebral artery even when the neck was rotated. It was not necessary to restrict the patient’s neck movement postoperatively.

KEY WORDS • atlas • axis • bow-hunter’s stroke • vertebral artery • vertebrobasilar insufficiency

FORCED or voluntary rotational neck movement has been implicated in the etiology of the extracranial vertebral artery compression syndrome known as “bow-hunter’s stroke.” Occlusion or stenosis of the vertebral artery at the atlantoaxial level induced by neck rotation may be physiological when the normal anastomotic flow of the vertebrobasilar system is preserved. However, trivial vascular injury when followed by thromboembolism in the vertebrobasilar system presents potential hazards, even if voluntary rotational or extending neck movement occurs within a normal physiological range. In cases where ischemic attacks occur repeatedly following voluntary neck rotation, patients have been forbidden to rotate or extend their neck or have been treated by cervical fixation.33

This paper reports a patient with recurrent attacks of ischemia in the vertebrobasilar system after rotational neck exercise. A new method of decompression of the vertebral artery at the level of the atlas is described which does not necessitate restriction of neck movements.

Case Report

This 37-year-old man was admitted to a local hospital for evaluation of repeated attacks of unsteadiness and numbness on the left side of the body. The attacks occurred as a result of rotational neck exercises. About 3 months before, he suffered a hyperextension injury to the neck in a collision while snow sledding, causing a stiff neck for 1 week. About 2 months after the accident, he experienced the sudden onset of numbness on the left side of the body, resulting in his leaning toward the left when playing baseball. He was a catcher for a baseball team, and while playing he rotated his neck from left to right bringing on the attack. Dizziness, unsteadiness, and a left-sided numbness lasting for several minutes, then the symptoms completely disappeared. Four weeks after this event, he had a similar episode of increased severity during a rotational neck exercise as a preliminary to jogging. He suffered severe dizziness and diplopia followed by nausea and vomiting, and was admitted to a local hospital.

Neurological examination on admission demonstrated left-sided Horner’s syndrome, total sensory loss on the left side of the body, and bilateral fine horizontal nystagmus. Computerized tomography (CT) demonstrated no significant abnormal findings at that time. An enhanced CT scan 7 days later revealed diffusely enhanced lesions in the cerebellar hemispheres, predominantly on the left. A diagnosis was made of cerebellar infarction, and the patient was treated with he-
modilution and anticoagulant therapy. Over a 2-week course, he improved to almost normal, and was then transferred to our hospital for further examination.

**Examination.** Neurological examination showed only minimal left cerebellar signs. A CT scan demonstrated a large low-density lesion in the left hemisphere and a small low-density lesion in the right hemisphere. Echocardiography and thoracic aortography showed no abnormality. A vertebral angiogram demonstrated no significant abnormal findings either intracranially or extracranially when the neck was in a neutral position (Fig. 1 left). However, when the neck was rotated about 60° to the right, the left vertebral artery became stenotic at the level of the atlas (Fig. 1 center). When the neck was fully rotated to the right, the artery was completely occluded at a level between the transverse foramina of the atlas and axis (Fig. 1 right). When other types of neck movements were performed the vertebral angiograms demonstrated neither stenosis nor occlusion. Surgical decompression of the left vertebral artery was recommended because of the potential risk of recurrent embolization induced by intermittent occlusion of the left vertebral artery when the patient rotated his neck.

**Operation.** With the patient in the prone position, a midline linear skin incision was made extending from just below the occipital protuberance to the spinous process of C-3. On the left side, the posterior surfaces of the atlas and the axis were exposed subperiosteally up to the transverse foramen of the atlas. A fine linear fracture was seen at the junction of the posterior arch and the transverse process of the atlas. The thickened atlanto-occipital membrane had fixed the vertebral artery in the vascular groove of the atlas. The thickened atlanto-occipital membrane was incised, the posterior portion of the lamina of the atlas was partly drilled off, and the transverse foramen of the atlas was unroofed to liberate the left vertebral artery (Fig. 2).

**Postoperative Course.** The postoperative course was smooth and uneventful. Postoperative angiography demonstrated no rotatory obstruction of the vertebral artery. The patient was discharged without any apparent neurological deficits and returned to his previous job. When last examined 2 years after the operation, he was coping well with daily life and enjoying all kinds of sports with no impairment. His neck movement has not been limited at all, and no further incidence of stroke has occurred.

**Discussion**

The vertebral arteries are uniquely susceptible to mechanical compression induced by neck movement because of their relationship with neighboring bone structures, muscles, and fibrous ligments all along the extracranial portion. The rotational action of the atlantoaxial joint may cause compression of the vertebral artery at that level, even within a normal range of motion. Running along the transverse foramen and vascular bone groove or canal in the posterior arch of the atlas, the vertebral artery is fixed within these structures. The anatomical and physiological features of this region explain the specific vulnerability of the vertebral artery to rotational neck movement. The mechanism involved is mainly a pinching of the vertebral artery contralateral to the side of neck rotation. There are many developmental and degenerative factors contributing to this mechanism, such as the depth of

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**Fig. 1.** *Left:* Left vertebral angiogram with the neck in a neutral position showing no abnormal findings. *Center:* Left vertebral angiogram showing stenosis in the third segment of the vertebral artery when the neck is rotated about 60° to the right. *Right:* The vertebral artery is completely occluded when the neck is fully rotated to the right (about 80°).
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the vascular groove of the atlas, ossification or hypertrophy of the atlanto-occipital membrane, tightness of the paravertebral muscles, and a sliding movement or subluxation of the atlantoaxial joint [2,9,10]. Temporary occlusion of one vertebral artery may or may not have an effect on the vertebrobasilar blood flow during ordinary daily life. When the caliber of the contralateral vertebral artery and the anastomosis with the carotid arteries are inadequate, ischemic symptoms can repeatedly occur during neck rotation or extension. On the other hand, when adequate collateral circulation is preserved during transient occlusion of the vertebral artery, no symptoms may appear, in which case this compression can be considered "physiological." However, repeated occlusion of the vertebral artery may cause vascular stasis and chronic injury of the vascular wall at the occlusion site, such as intimal damage, mural dissection, vascular spasm, and subsequent thromboembolism. [17,25,28,32] Atherosclerotic change of the arterial wall may make it more prone to damage from rotational neck injuries.


Angiographic findings of rotational vertebral injury at the level of C1-2 were first described in 1974. [27,28] The usual appearance is obstruction or segmental narrowing in the vertebral artery as the vessel passes through the transverse foramen, or at the atlantoaxial joint. [27,28,39,44,46] Other authors have described rotational vertebral artery occlusion or stasis at the level of the third segment of the vertebral artery. [17,51] Grossman and Davis [17] pointed out that a vertebral artery, which is easily occluded upon head rotation, may be a source of stasis producing small thrombi and subsequent embolism. Chronic vascular injury due to rotational compression of the vertebral artery may be a factor that has been neglected in the etiology of vertebrobasilar insufficiency. When clinical symptoms are correlated with neck motion, a dynamic angiographic study should be performed, even if conventional examinations show no abnormalities. [51]

Surgical decompression of the vertebral artery has been considered to be the treatment of choice for Power's syndrome [21,22,47] or lateral bone spur compression. [3,24,38,42] Patients with dissecting aneurysms are

![Fig. 2. Schematic representation of the mechanism of vertebral artery occlusion at the level of the atlas. A: Preoperative view with the neck in the neutral position. VA = vertebral artery; AOM = atlanto-occipital membrane. B: Preoperative view with the neck rotated to the right (black arrow). The left vertebral artery is fixed by the thickened AOM, vascular groove, and transverse foramen of the atlas, and is elongated and compressed (open arrow). C: View after surgical decompression of the left vertebral artery with the neck rotated to the right (black arrow). The thickened AOM is resected, and the vascular groove and transverse foramen of the atlas are opened (arrowheads).](image-url)
treated by ligation of the proximal vertebral artery.\textsuperscript{7,11} If atlantoaxial dislocation or rotational subluxation of a facet is associated, it should be treated by fusion of the atlas and the axis.\textsuperscript{13} Yang, et al.,\textsuperscript{52} performed C1–2 fusion for the treatment of rotational occlusion of the vertebral artery to limit atlantoaxial rotational movement and prevent stretching and injury of the vertebral artery. Otherwise, the condition has been treated conservatively by forbidding the patient to perform significant neck rotation.\textsuperscript{46} Our operative technique is aimed to decompress the vertebral artery without decreasing neck mobility at the level of the atlas (Fig. 2). This method can be performed easily and safely with the aid of an operating microscope. The results in this case suggest that surgical decompression of the affected vertebral artery is the treatment of choice for preventing bow-hunter's stroke. A patient treated in this way is able to cope well with daily life and perform sports without anxiety or fear when rotating the neck.

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