Rotational occlusion of the vertebral artery caused by transverse process hyperrotation and unilateral apophyseal joint subluxation

Case report

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The authors describe transverse process hyperrotation and unilateral apophyseal joint subluxation as a novel mechanism of rotational vertebral artery (VA) occlusion. The patient, a 56-year-old man, complained of episodic bilateral blindness when rotating his head more than 90˚ to the right. Plain cervical x-ray films showed spondylotic osteophytes of the right C4–5 uncovertebral portion. Dynamic angiography revealed right VA occlusion at C4–5 and left VA occlusion at C1–2 with head rotation to the right. It was demonstrated on three-dimensional images constructed from computerized tomography scans that C-4 transverse process hyperrotation compressed the right VA against the apex of the C-5 subluxating superior articular process via the inner surface of the transverse process anterior root (processus costarius) rather than the osteophytes. It is also proposed that the true site of occlusion is different from that observed in angiographic studies.

KEY WORDS • rotational occlusion • vertebral artery • transverse foramen • apophyseal joint • subluxation

CERVICAL spondylotic osteophytes have been the only reported cause of mechanical occlusion of the vertebral artery (VA) at the second segment except for between the C-1 and C-2 levels. In a patient presenting with episodic bilateral blindness during head rotation, we demonstrate that rotational VA occlusion occurs not via the osteophytes but rather the anterior root (processus costarius) of the hyperrotated C-4 transverse process and subluxation of the C4–5 apophyseal joint.

Case Report

Examination. This 56-year-old truck driver with no history of head trauma suffered from bilateral blindness and tinnitus when rotating his head more than 90˚ to the right. There were no unusual neurological findings with the head in a neutral position and no other ancillary symptoms. Rightward head rotation was demonstrated to occlude the right VA at C-6 on angiography (Fig. 1 upper, 90˚).

First and Second Operations. During the first operation, in May of 1995, the VA was decompressed from the anterior tuberculum of the right C-6 transverse process, longus colli muscle, and anterior scalenus muscle. Following the first procedure, the patient remained free of symptoms for 2 months, after which time his symptoms reappeared. We diagnosed constriction of the VA by adhesion of the surrounding tissue. In the second operation, which was performed in July 1995, the adhesive tissue was ablated from the VA, its surrounding muscles and bone, and to prevent readhesion, subcutaneous fat tissue was taken from the abdomen and rolled around the VA. However, the patient’s symptoms did not resolve.

Third Admission. The patient was readmitted in April 1996, at which time neurosurgical examination, computerized tomography (CT) scan of the head, and magnetic resonance imaging were normal. Plain cervical x-ray films demonstrated osteophyte formation at the right C4–5 uncovertebral portion. Carotid angiography was remarkable only for bilateral hypoplastic posterior communicating arteries (PCoAs). Vertebral angiography showed no stenosis and no kinking with the head in a neutral position. The left VA originated directly from the aorta.

When the patient turned his head 90˚ to the right, at which angle he experienced blindness, the right VA was completely occluded at C-6 (Fig. 1 upper, 90˚), and the left VA was also obstructed at C1–2 due to kinking (Fig. 1 lower).
Dynamic Angiography. Using image intensification, right vertebral angiograms were obtained, with injection of contrast medium and gradual rightward head rotation from 60 to 90˚ (Fig. 1 upper). When the patient’s head was rotated to 85˚, the right VA became severely stenosed at C4–5 with a normal lumen at C-6 (Fig. 1 upper, 85˚). The obstruction thus appeared to be located at the C4–5 disc space. Apparently, the contrast medium failed to flow beyond the C-6 vertebral level secondary to complete occlusion at C4–5. Angiographic evaluation indicated that compression occurred via the osteophyte of the C4–5 uncovertebral portion. A thin-slice cervical CT scan revealed a calcified strand (the upper margin of the C-5 superior articular process) in the C-4 transverse foramen. The upper vertebra (C-4) rotated 38˚ from the horizontal line, 5˚ more than the lower vertebra (Fig. 2).

Three-Dimensional CT Angiography. Three-dimensional CT images were obtained using a Lemage scanner (GE Yokokawa Medical Systems, Tokyo, Japan). Spiral data were acquired in 35 seconds with a slice thickness and interval of 1 mm and 1.5 mm, respectively, and a table speed of 1.5 mm/second (200 mA; 80 kV). The scanning volume was 52.5 mm. Scanning was started at C-6 and continued toward the cranium following mechanical injection of 60 ml of contrast material (Iopamidol; 300 mgI/ml) into the antecubital vein at 2 ml/second after a delay of 15 to 18 seconds. Axial images were reconstructed every 0.75 mm. Reconstructed images were transferred via ethernet to a workstation (Advantage Windows; GE Yokokawa Medical Systems). The region of interest (C4–6) was selected, followed by deletion of the carotid arteries, jugular veins, and other structures, leaving the VAs and vertebrae. The VAs were cut on screen, colored red, and pasted in their original position. We were able to obtain a slice at an optimum angle on the reconstructed image.

Three-dimensional CT scanning clarified the mechanism of right VA occlusion during head rotation, revealing that the osteophytes were not involved. Rather, the VA was scissored and compressed between the anterior root of the hyperrotated C-4 transverse process and the upper margin of the subluxating C-5 superior articular process (Fig. 3).

Third Operation. In June 1996, a third operation was performed. The anterior root of the right C-4 and C-5 vertebral transverse process was exposed via the space between the carotid sheath and sternothyroid muscle, partially cutting the omohyoid muscle. After removal of the C4–5 uncovertebral osteophytes, intraoperative angiography with the patient’s head rotated to the right revealed that the stenosis remained. After removal of the thick eroded anterior root of the transverse process and dissection of the VA circumferentially from the surrounding tissue, resolution of the stenosis was demonstrated on angiographic studies (Fig. 4).

Postoperative Course. The postoperative course was uneventful except for right-sided Horner’s syndrome and transient slight weakness of the right biceps muscle. The patient had no visual symptoms on head rotation.

Discussion

It is well known that occlusion of one VA does not result in vertebrobasilar insufficiency because the PCoAs and the contralateral VA provide collateral circulation to the basilar artery (except when the VA terminates in the posterior inferior cerebellar artery30). Thus, to yield vertebrobasilar insufficiency, both hypoplasia ( aplasia) of the PCoAs and occlusion (hypoplasia30 or atherosclero-
Rotational occlusion of vertebral artery

FIG. 3. Three-dimensional reconstructed images from CT scans of the C4-6 vertebrae in the neutral position (A and C) and at 85° rightward head rotation (B and D). A and B: The uncovertebral osteophytes (arrowheads) were not related to the occlusion. C and D: Sagittal sections depicting the transverse foramen. The right VA was compressed and scissored between the anterior root of the hyperrotated C-4 transverse process and the upper margin of the subluxated superior articular process of C-5 (arrows). The left VA enters the transverse foramen at the C-5 level.

sis, or rotational occlusion of the contralateral VA are required. In the present case, there was aplasia of both PCoAs and physiological rotational occlusion of the contralateral VA at the Cl-2 level.

Classification of Segments of VA

The VA is divided anatomically into four portions. The first segment includes its origin from the subclavian artery (or rarely from the arch of the aorta) to its entry into the bone canal, usually at C-6. The second segment penetrates the transverse foramen of the cervical spine. The third segment courses around the atlas, and the fourth is the intradural portion.

Causes of Rotational VA Occlusion

Rotational occlusion of the VA has been reported at each cervical portion. At the first segment, anomalous origins of the VA, the longus colli, proximal ligament of longus colli and anterior scalene muscle, cervical sympathetic chain and thyrocervical trunk are all reported causes of occlusion. At the second segment, uncovertebral osteophytes are reported to cause positional occlusion, and between C-1 and C-2 rotational occlusion has been considered physiological except in cases of atlantoaxial dislocation. At the fourth segment, the odontoid process (atlantoaxial dislocation) causes rotational occlusion.

Previously Described Mechanisms of Rotational Occlusion at the Second Segment

In reports describing uncovertebral osteophytes as the cause of rotational occlusion at the second segment, the direction of rotation is not always ipsilateral to the site of VA occlusion. Compressive stenosis of the VA by the osteophytes of cervical spondylosis mostly occurs when the head is turned to the ipsilateral side, but occasionally when the head is rotated to the contralateral side. The question remains as to how the direction of the rotation causing obstruction of the VA can vary. Sheehan, et al. proposed that rotational occlusion of the opposite VA occurs by stretching. The fibrous connective tissue surrounding the VA may play a role in rotational occlusion of this vessel. The osteophyte causes an inflammatory reaction that produces a fibrous band around the VA; this restricts movement of the artery and increases the likelihood of compression or kinking on head rotation.

No report has described two- or three-dimensional osteophyte compression of the VA on head rotation. The
position and size of the outgrowth, as well as distance of osteophyte movement, are important factors to consider. The uncovertebral osteophyte, lying in front of the anterior root of the transverse foramen, does not reach the lateral side of the VA and features a rotational angle that is too small to compress the artery. Thus, it cannot be related to an ipsilateral rotational occlusion as seen in the present case.

A Novel Mechanism of Rotational Occlusion

We identify hyperrotation of the transverse foramen and subluxation of the apophyseal joint as a novel mechanism of ipsilateral rotational VA occlusion at the second segment. Thin-slice CT scanning demonstrated that rotational occlusion was caused not by the osteophytes but by the hyperrotating transverse foramen of the upper vertebra (C-4) with respect to the lower vertebra (C-5) (Fig. 2). Three-dimensional CT scanning revealed that the subluxated superior articular process at the apophyseal joint compressed the VA (Fig. 3). Kovács emphasized the significance of subluxation and deformation of the cervical apophyseal joints in VA compression, although he did not relate this directly to rotational occlusion. The apophyseal joint is closer to the VA than the uncovertebral portion. The distance between the VA and articular process apex is slightly more than 2 to 3 mm. Its distance from the uncovertebral portion varies between 3 and 6 mm. Alignment of the transverse processes is also an important factor in rotational occlusion. If the transverse foramen moves posteriorly by extension force or sliding, the VA is more likely to be compressed by the anterior root on ipsilateral rotation. In larger (lower) vertebrae the transverse process runs slightly anteriorly. The VA migrates posteriorly to anteriorly from C-3 to C-6 and posteriorly again at C-7.

Intervertebral disc degeneration tends to result in abnormal vertebral movements, subjecting the uncovertebral ligaments to increased strain with subsequent development of osteophytes. In our case, the osteophytes might have resulted from dynamic, hyperrotating movement.

In some previous cases of rotational occlusion of the VA caused by uncovertebral osteophytes, especially with head rotation toward the ipsilateral side, this novel mechanism may explain the VA occlusion.

Treatment Modalities

There are some reports that anticoagulation and cervical traction produced good results. Bypass surgery is useful only for constant severe vertebral stenosis. Mechanical stimulation to the arterial wall, however, causes platelet aggregation, which can be a source of emboli. A direct surgical approach is more suitable to prevent repeated mechanical compression.

Whereas the uncovertebral osteophyte has been reported to cause rotational occlusion at the second segment of the VA, there are no reports of cases in which only the osteophyte was removed. In these reports not only the osteophyte but also the anterior root of the transverse process was removed, with good surgical outcomes. This method, however, also has an effect on the transverse process hyperrotation and subluxation of the apophyseal joint. The cause of VA occlusion at the second segment appears to be hyperrotation and subluxation of the apophyseal joint; therefore, spinal fusion with a bone graft or titanium plate may be a beneficial alternative.

We considered it better to perform the fusion in the present case. The removal of the transverse process anterior root and the uncovertebral osteophyte is necessary and may be satisfactory but should not be considered the fundamental treatment, because these procedures do not correct the hyperrotation and subluxation. A fusion between the vertebrae involved can prevent hyperrotation and subluxation.

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