

Rotational vertebrobasilar insufficiency as a component of thoracic outlet syndrome resulting in transient blindness

Case report

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✓ Cases of unilateral vertebral artery compression associated with thoracic outlet syndrome infrequently result in symptoms and, of those that do, most involve the brain stem. Reports of transient blindness resulting from this condition are even more rare. The authors describe the case of a middle-aged woman who presented with transient blindness when she turned her head excessively to the left. She also exhibited other less severe brain-stem symptoms. Arteriography demonstrated occlusion of the left vertebral artery only when her head was rotated to the left. Surgical exploration revealed entrapment of the left vertebral artery by a tight anterior scalene muscle, release of which resulted in complete resolution of her symptoms. Both neurosurgeons and radiologists need to be aware that extrinsic compression of the vertebral artery precipitated by head rotation may sometimes result in transient cortical blindness.

KEY WORDS • vertebral artery • thoracic outlet syndrome • blindness • vertebrobasilar insufficiency

TRANSIENT ischemic attacks resulting from vertebrobasilar ischemia usually affect the cerebellum, brain stem, and spinal cord and result in a variety of symptoms including vertigo, ataxia, dizziness, syncope, and paresthesia.^{1,2} Supratentorial manifestations, particularly in the distribution of the posterior cerebral arteries, may occur if collateral circulation through the circle of Willis is inadequate to maintain sufficient perfusion pressure. Most frequently, the etiology involves atherosclerotic stenosis or thromboembolic events.² However, in the unusual case of symptoms arising with head rotation, compression of the vertebral artery by an extraluminal lesion should be suspected. We present a case of rotational insufficiency associated with thoracic outlet syndrome.

Case Report

This 44-year-old woman suffered a strained muscle in the left shoulder several months prior to presentation. Subsequently, she began to experience global binocular blindness on turning her head and neck to the extreme left; however, vision quickly returned to normal when she turned her head to the right. This prompted her to seek medical attention.

Examination. The patient reported some mild facial and neck pain and paresthesia along the C7-T1 dermatomes bilaterally, which had progressively worsened. She also noted circumoral numbness, left arm pain with paresthesia, and posterior neck muscle spasm. Physical examination was notable for onset of blindness and light-headedness when she rotated her head to the extreme left. Hyperabduction of the arms produced slight diminution of the left radial pulse and a weakened hand grip. Performing Adson's maneuver on the left while the patient turned her head to the left caused transient blindness and almost complete loss of the left radial pulse. The combination of these symptoms suggested vertebrobasilar circulation insufficiency.

Magnetic resonance images of the brain and cervical spine were normal. Arteriography of the aortic arch and the subclavian artery was performed with the head rotated to the right and then to the left (Fig. 1). These studies demonstrated complete occlusion of the left vertebral artery approximately 2 cm from its origin when the head was turned to the left. With the head in a neutral position, the left vertebral artery showed a normal caliber and course.

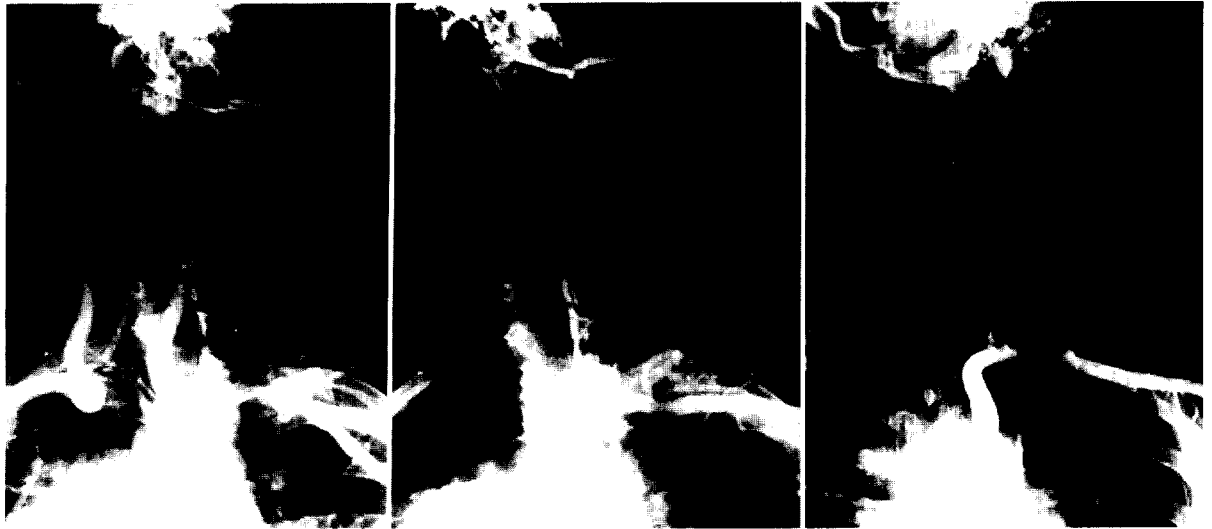


FIG. 1. Preoperative arteriograms, left anterior oblique view. *Left:* Arteriogram taken after aortic root injection of contrast material showing the right vertebral artery to be patent and of normal caliber. The blood supply provided by both vertebral arteries is nearly equal. *Center:* Arteriogram obtained after selective injection of contrast material into the left subclavian artery, with the patient's head turned to the right. The left vertebral artery is clearly patent. *Right:* Arteriogram taken after selective injection of contrast material into the left subclavian artery, with the patient's head turned to the left. The left vertebral artery is completely occluded approximately 2 to 3 cm beyond its origin.

Operation. At surgery, a tight anterior scalene muscle was discovered compressing the left vertebral artery and lower cord of the brachial plexus between the anterior and middle scalene muscles. To relieve the compression, the first rib was resected and the scalene muscles were divided and partially resected. Following surgery, all symptoms and signs resolved.

Discussion

When evaluating a patient for vertebral artery occlusion as a component of thoracic outlet syndrome, several etiologies must be considered. Among extrinsic causes, cervical spondylitic spurs are well recognized.^{2,6} These usually result in occlusion of the vertebral artery in its second portion, where it passes through the transverse foramina, at or above C-6. Other causes of rotation-induced symptoms include compression of the first portion of the vertebral artery by the anterior scalene muscle or by deep cervical fascia when the vertebral artery has an anomalous origin or kinking and redundancy.² Occlusion of the first portion of the vertebral artery just below its entry into the transverse foramina at C-6 may also be secondary to a hypertrophied ligament of the longus colli muscle and the anterior scalene muscle.²

Unilateral occlusion of the vertebral artery seldom results in neurological deficit if the collateral supply through the other vertebral and posterior inferior cerebellar arteries is sufficient. It is estimated that deficits occur in fewer than 20% of such cases.^{1,4,7} This correlates with the fact that approximately 15% of patients have hypoplasia of one vertebral artery, most often the right.⁵ The left vertebral artery is usually dominant and

is also the one that is occluded in the majority of patients presenting with deficit. In these patients, symptoms appear when the right vertebral artery does not provide sufficient collateral flow. In our patient, however, vertebral artery circulation was equally balanced; both vertebral arteries were the same size, which may account for the mild degree of posterior fossa symptoms.

The case reported here was unusual because the dominant manifestation was supratentorial (cortical blindness). For example, in a series of 15 patients with extraliminal vertebral artery occlusions reported by Hardin and Poser,³ three were described as having "blurred" vision, among other symptoms, but none exhibited overt blindness. Nagashima⁶ presented 20 patients with vertebral artery compression, only two of whom were reported to have visual disturbances; both of these suffered from multiple brain-stem symptoms.

Hypothetically, cortical blindness should occur only if collateral circulation to the posterior cerebral arteries from the anterior circulation (via the posterior communicating arteries) is poor. In such a case, the posterior communicating arteries may be hypoplastic, atretic, or diseased. However, imposing the additional risks of cerebral angiography specifically to evaluate the circle of Willis was not considered to be justified for our patient and would not have affected management.

In this unusual case, the dominant manifestation was cortical blindness (supratentorial) with mild brain-stem symptoms. We hypothesized that this was due to a combination of balanced vertebral artery supply in conjunction with poor collateral connections between the an-

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terior and posterior circulations, the result being symptomatic ischemia preferentially localized to the visual cortex when the left vertebral artery became obstructed.

If symptoms of vertebrobasilar insufficiency are seen when a patient rotates his or her head, extrinsic compression should be included as a diagnostic possibility, and subclavian arteriography should be performed in both the neutral and rotated positions to confirm the diagnosis.

References

1. Bakay L, Leslie E: Surgical treatment of vertebral artery insufficiency caused by cervical spondylosis. **J Neurosurg** **32**:596-602, 1965
2. Dadsetan MR, Skerhut HEI: Rotational vertebrobasilar insufficiency secondary to vertebral artery occlusion from fibrous band of the longus colli muscle. **J Neuroradiol** **32**: 514-515, 1990
3. Hardin CA, Poser CM: Rotational obstruction of the vertebral artery due to redundancy and extraluminal cervical fascial bands. **Ann Surg** **158**:133-137, 1963
4. Husni EA, Bell H, Storer J: Mechanical occlusion of the vertebral artery. **JAMA** **196**:101-104, 1966
5. Louw JA, Mafoyan NA, Small B, et al: Occlusion of the vertebral artery in cervical spine dislocations. **J Bone Joint Surg (Br)** **72**:679-681, 1990
6. Nagashima C: Surgical treatment of vertebral artery insufficiency caused by cervical spondylosis. **J Neurol** **32**: 512-521, 1970
7. Parent A, Harkey HL, Touchstone DA, et al: Lateral cervical spine dislocation and vertebral artery injury. **J Neurosurg** **31**:501-509, 1992

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