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Transient rotational compression of the vertebral artery caused by herniated cervical disc

Case report

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Of the many causes of vertebrobasilar insufficiency (VBI), extrinsic compression of the vertebral artery (VA) is relatively uncommon. A syndrome of VBI caused by extrinsic compression of the VA secondary to head rotation has been termed positional vertebrobasilar ischemia. The authors present a case of transient VBI caused by herniation of a cervical disc. Transcranial Doppler ultrasonography was used preoperatively to confirm the diagnosis and intraoperatively to monitor cerebral perfusion and to confirm that adequate decompression of the VA had been achieved.

KEY WORDS • vertebrobasilar insufficiency • ischemia • transcranial Doppler ultrasonography

VERTEBROBASILAR insufficiency is characterized by a constellation of symptoms including vertigo, diplopia, visual disturbances, and sensorimotor symptoms. It is most often the result of intrinsic lesions of the VA, such as arteriosclerosis.[2] Extrinsic compression of the VA has been well described and can be caused by tumor, osteophytes, or musculofascial bands.[7,9] In some cases, extrinsic compression may only manifest in symptoms of VBI when the head is rotated.[6,10] This condition usually occurs with transient compression of one or both VAs in the absence of adequate collateral circulation. The site of compression in these cases is typically at or near the entrance of the VA into the foramen transversarium at C-6, within the foramen transversarium as the artery courses cephalad from C-6 to C-2, or at the point at which the VA winds around the lateral mass of the axis.[1,6–10] In 1993 Budway and Senter[4] reported one case of a herniated cervical disc causing compression of the VA, which led to the formation of thrombus in the vessel; the vessel had embolized and caused bilateral PCA territory infarcts. We present the second case in the available literature of transient rotational occlusion of the VA due to a herniated cervical disc.

Case Report

Presentation. This 61-year-old man presented with a 3-week history of vertigo and nausea progressing to syncope, manifesting when he turned his head to the left. These events occurred several times, always with head rotation in the same direction, and also with extension of his neck. After each event, his sensorium returned to normal, and he did not notice any permanent neurological deficits.

Examination. There was no evidence of any neurological deficit, carotid artery or cranial bruits, or heart murmurs on physical examination. Magnetic resonance angiography was initially performed with the patient in the neutral position. There was absence of the right VA, and stenosis of the left VA at the C-7 level. Conventional angiography and CT angiography demonstrated a patent left VA that became focally stenosed with 10˚ of head rotation to the left, progressing to complete occlusion at 30˚ leftward (Figs. 1–3). The patient began complaining of vertigo and nausea within 15 seconds, his neck was returned to the neutral position, and the examination was stopped. Of note, neither CT nor MR imaging studies revealed a herniated cervical disc. Transcranial ultrasonography confirmed transient obstruction of intracranial flow with the head turned to the left. There were normal flow velocities in the PCA distribution bilaterally through an unpaired left VA (Fig. 4 *upper left*). Signals were lost in the bilateral PCA distribution with provocative head turning (Fig. 4 *upper left*), and a hyperemic response was observed after return to the neutral position (Fig. 4 *upper left*). Prior to surgery, the patient wore a hard cervical collar to limit head turning. He did not experience any more symptoms.

Operation. The patient underwent surgery in which a left-sided anterior approach to the cervical spine was per-

Abbreviations used in this paper: CT = computerized tomography; PCA = posterior cerebral artery; TCD = transcranial Doppler; VA = vertebral artery; VBI = vertebrobasilar insufficiency.

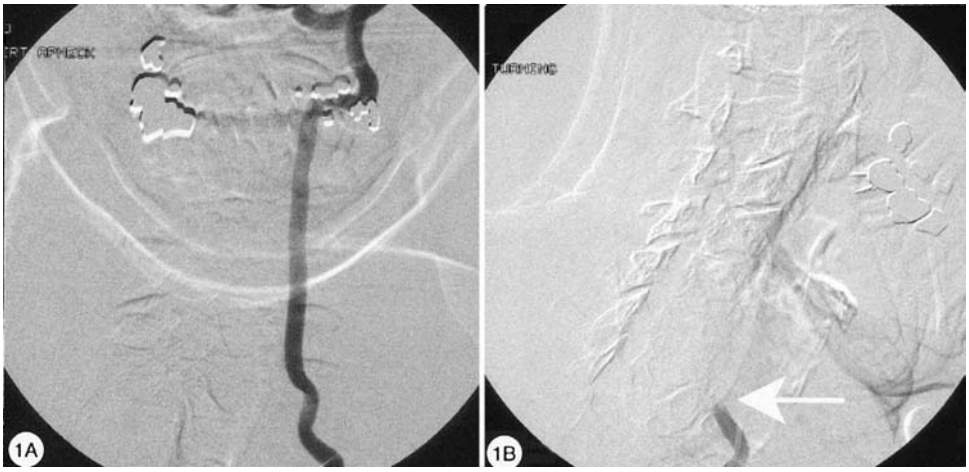

FIG. 1. Conventional angiogram of left VA with head in neutral position (A) and with head rotated to the patient's left (B). Note the complete occlusion of the left VA with rotation to the left *(large arrow)*.

formed. Bilateral TCD monitoring of the PCAs was performed intraoperatively. The anterior tubercle of C-6 was removed and the VA was identified in the foramen transversarium and distally at the C-7 level. No musculofascial bands were seen to be compressing the VA. With a Doppler probe on the VA, the patients' head was then turned approximately 15˚ to the left, at which point a small fragment of disc material was seen to enter into the foramen transversarium. This fragment was removed, and the disc space was entered laterally using a small curette, removing an additional small amount of disc material. No osteophytes were noted. The patient's head was then turned leftward, this time approximately 60˚, during TCD surveillance of the VA. No decreases in flow were noted. Additionally, TCD recordings did not demonstrate any decreases in signal with head turning after removal of the disc fragment (Fig. 4 *lower left*). At this point closure was performed in the usual fashion.

Postoperative Course. The patient was placed in a hard cervical collar postoperatively. On the 1st postoperative day, provocative testing was performed during TCD monitoring of the bilateral PCAs. No decreases in TCD signals with head turning were noted, nor did full rotation in both directions, flexion, or extension, induce neurological sequelae. The patient was discharged from the hospital, and at 6-month follow-up examination no repeated episodes have occurred; TCD ultrasonography has demonstrated normal flow velocities in the PCA distributions (Fig. 4 *right*).

Discussion

Vertebrobasilar insufficiency can be caused by either intrinsic lesions or extrinsic compression of the VA. Intrinsic lesions, such as dissecting lesions, atherosclerotic plaques, or emboli, are by far the most common cause of VBI.[7,11] Extrinsic compression of the vessel is a less common phenomenon. The VA is susceptible to extrinsic compression at several locations. Musculotendinous bands from the longus colli or anterior scalene muscles can compress the VA just proximal to its entrance into the foramen transversarium at C-6.[9] Osteophytes from C-6 to C-2 can also compress the VA from within the foramen.[7,11] Such compression within the foramen transversarium can be exacerbated with head rotation toward the compressed side. The VA can also be compressed at the C-1 level with rotation due to tension on the contralateral VA and subsequent compression of the artery against the C-1 lateral mass.[5,6,10] Similarly, the contralateral artery can be kinked as it pierces the atlantooccipital membrane.[10] Thus, extrinsic VA compression can occur with ipsilateral rotation if the site of compression is at the entrance to or within the

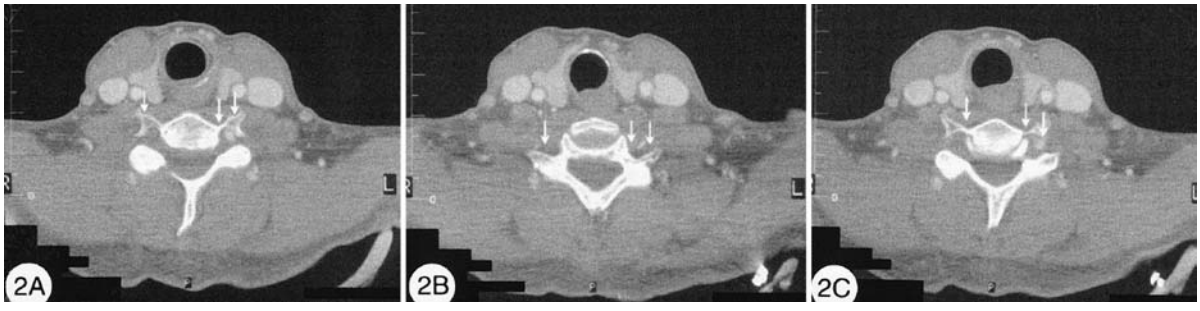

FIG. 2. A–C: Axial CT angiograms through C-6 and C-7 with head in 10˚ of leftward rotation. There is complete occlusion of the right VA at each level *(single arrows)* and focal stenosis of the left VA, which is most severe at the level of the C6–7 disc space *(double arrows)*.

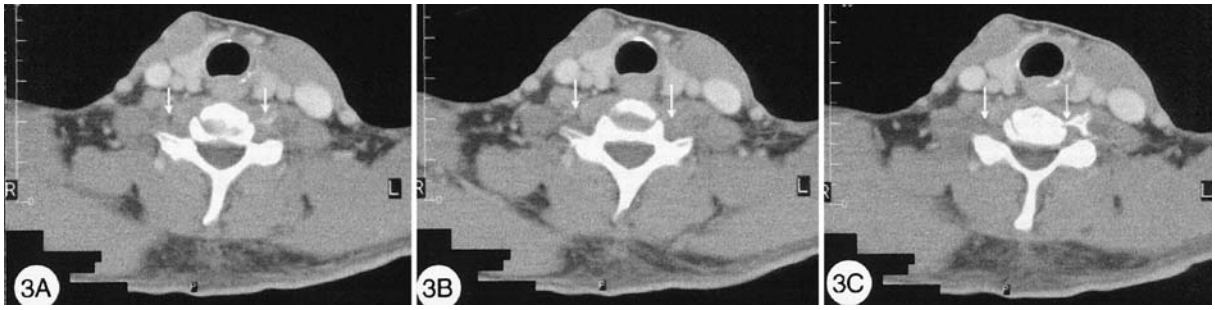

FIG. 3. A–C: Axial CT angiograms through C-6 and C-7 with head rotated 30˚ to patient's left, revealing complete occlusion of both VAs *(single arrows)*.

foramen transversarium and can occur with contralateral rotation if the site is at C-2 or above.

In 1993 Budway and Senter[4] presented the first case of VBI secondary to a herniated cervical disc. The present case differs from theirs in several ways. First, their patient presented with persistent neurological deficits consistent with VBI. The deficits were not related to head rotation, and imaging performed with the patient in the neutral position revealed an occluded ipsilateral VA. Given that the contralateral VA was patent, they surmised that the patient's deficits were the result of an embolic event. Intraoperatively, they used color-flow Doppler ultrasonography of the VA only to verify the artery's patency after decompression. In contrast, the present case involves a patient with transient symptoms clearly induced by head rotation. Imaging in the neutral position revealed a patent ipsilateral VA and absence of the contralateral VA. Intraoperative TCD monitoring of the PCA was conducted in

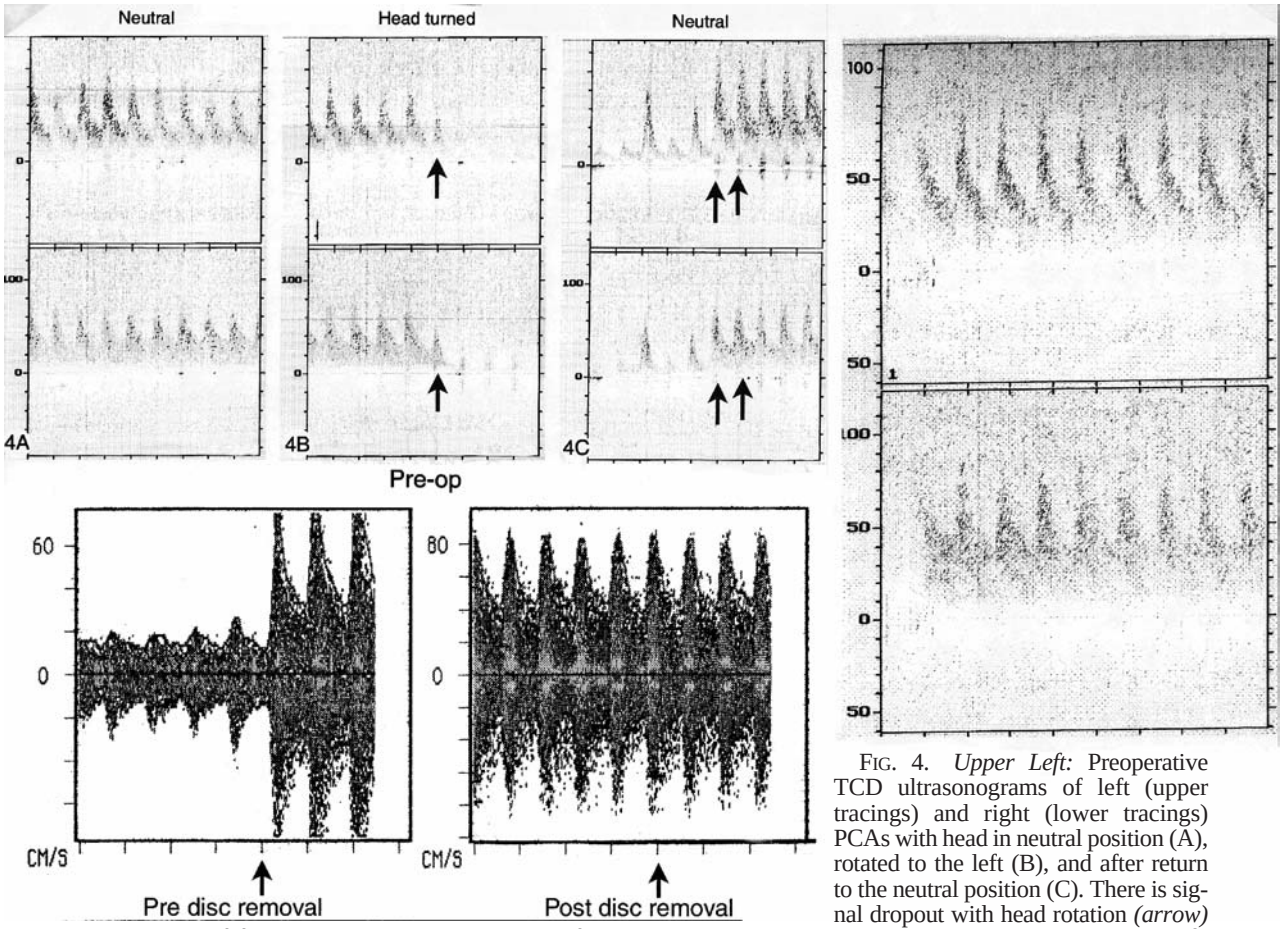

FIG. 4. *Upper Left:* Preoperative TCD ultrasonograms of left (upper tracings) and right (lower tracings) PCAs with head in neutral position (A), rotated to the left (B), and after return to the neutral position (C). There is signal dropout with head rotation *(arrow)* and the return of flow with a hyperemic response after return to the neutral position *(double arrows)*. *Lower Left:* Intraoperative TCD tracings of left PCA before and after disc removal. The head was turned to the left at the start of each tracing and returned to neutral at the time point marked by *arrow*. Note low velocities with the head turned prior to disc removal. After disc removal, there was no change in flow when the head was rotated. *Right:* Postoperative TCD tracing of bilateral PCAs with head rotated to the left obtained at 6 months. No decreases in flow are demonstrated.

addition to direct Doppler monitoring of the VA, thus providing information about the flow of blood downstream from the site of occlusion. Recently, Vates, et al.,[13] have reported the first case of bow hunter stroke secondary to a laterally herniated cervical disc similar to the present case. They concluded that TCD ultrasonography is helpful in establishing a diagnosis. We concur with this. Furthermore, the present case demonstrates the utility of intraoperative TCD ultrasonography to confirm decompression of the VA.

The diagnosis of rotational VA compression is definitively established using dynamic positional angiography. Additionally, TCD monitoring of the PCA distribution has been described as a means to assess positional compression.[3,11] Because TCD monitoring of the PCA provides a hemodynamic assessment of the ischemia induced by compression at a more proximal site, it is not useful in determining the location of the compression. Intraoperative TCD monitoring has been routinely performed in carotid endarterectomy and other cerebral revascularization procedures to assess flow in affected vascular territories. In the present case, dynamic TCD monitoring of the PCA was conducted intraoperatively to confirm adequate decompression of the VA. Follow-up TCD ultrasonography were also obtained, and in this case demonstrated good blood flow. Because of these findings, it was not necessary to subject the patient to another angiographic session.

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