Rotational vertebral artery occlusion from occipital bone anomaly: a rare cause of embolic stroke

Case report

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Vertebral artery (VA) occlusion by rotation of the head is uncommon, but can result from mechanical compression of the artery, trauma, or atlantoaxial instability. Occipital bone anomalies rarely cause rotational VA occlusion, and patients with nontraumatic intermittent occlusion of the VA usually present with compromised vertebrobasilar flow.

A 34-year-old man suffered three embolic strokes in the vertebrobasilar system within 2 months. Magnetic resonance imaging demonstrated multiple infarcts in the vertebrobasilar territory. Angiography performed immediately after the third attack displayed an embolus in the right posterior cerebral artery. Radiographic and three-dimensional computerized tomography bone images exhibited an anomalous osseous process of the occipital bone projecting to the posterior arch of the atlas. Dynamic angiography indicated complete occlusion of the left VA between the osseous process and the posterior arch while the patient’s head was turned to the right. Surgical decompression of the VA resulted in complete resolution of rotational occlusion of the artery.

An occipital bone anomaly can cause rotational VA occlusion at the craniovertebral junction in patients who present with repeated embolic strokes resulting from injury to the arterial wall.

Key words • anomaly • craniovertebral junction • embolic stroke • vertebral artery

This 34-year-old man had three occurrences of embolic stroke in the vertebrobasilar system. In the first, he experienced the sudden onset of right-sided tinnitus, vertigo, and left-limb ataxia. Magnetic resonance imaging performed 3 days after onset demonstrated small infarcts in the right thalamus and in the territory of the left superior cerebellar artery. Sudden left homonymous hemianopia occurred 16 days after the first stroke. At that time, a small infarct in the right temporal lobe was apparent on MR imaging, and subsequent DS angiography exhibited an embolus within the right posterior cerebral artery (Fig. 1). Despite anticoagulation therapy, the patient experienced a third attack and presented with headache and right-limb ataxia 1 month after the second attack. Magnetic resonance images revealed an additional infarct in the territory of the left superior cerebellar artery. Cardioembolism was excluded following a detailed cardiological evaluation. The patient was admitted to our hospital for further evaluation 2 months after the third attack. On admission, only left-sided upper quadrantanopia was noted. Radiography and three-dimensional CT scanning with bone window settings revealed an osseous process projecting from the occipital bone near the occipital condyle to the posterior arch of the atlas (Fig. 2). Dynamic DS angiography disclosed complete occlusion of the left VA between the atlas and the anomalous osseous process while the patient’s head was rotated to the right. In the neutral position, no stenosis or occlusion was found in either VA.

Surgical decompression of the left VA was performed via a posterior approach. Resection of osseous structures, including the anomalous process extending from the occipital bone as well as a portion of the lamina and transverse pro-
cess of the atlas on the left side, resulted in full exposure of the left VA between its exit from the C-2 transverse foramen and penetration of the dura mater. Intraoperative DS angiography and direct visual inspection indicated complete decompression of the artery even with passive rotation of the head. The arterial wall at the point of occlusion appeared thickened and sclerotic, in contrast with other portions of the artery (Fig. 3).

The postoperative course was uneventful. A dynamic angiogram of the VA showed no stenosis with a head rotation to the right (Fig. 4). Anticoagulation therapy was continued because of likely previous intimal injury from rotational occlusion.

Discussion

The patient in this report demonstrated a unique cause of
rotational VA occlusion with a relatively uncommon clinical presentation—that of repeated embolic stroke. Mechanisms of rotational VA occlusion can be divided into three categories. First, the VA may be compressed mechanically within the physiological range of head rotation by surrounding structures such as spondylotic osteophytes, muscles, or fascial bands. Occlusion or stenosis of the dominant VA results in hemodynamic compromise when the opposite VA is hypoplastic, stenotic, occluded, or terminates atypically at the origin of the posterior inferior cerebellar artery. Second, excessive head movement from chiropractic manipulation, surgical positioning, or sports such as swimming and archery may result in intimal injury followed by thromboembolism, which is sometimes associated with arterial dissection or pseudoaneurysm formation. Finally, instability at the atlantoaxial joint may be responsible for rotational VA occlusion.

Vertebral artery compression is less common between the atlas and dural entry than at levels below the atlas. Nonetheless, the atlantooccipital membrane and the dura mater may compress the VA. Among osseous anomalies at the craniovertebral junction, basilar impression and atlantooccipital assimilation have been reported as rare causes of rotational VA occlusion. The present report adds one more type of osseous anomaly that can result in rotational occlusion of the VA.

To our knowledge, an osseous process of the occipital bone located posterolateral to the condyle, as found in this patient, has not been described previously. Although anomalous osseous processes near the occipital condyle have been reported as paracondyloid, supratransverse, and retrocondylar processes, the osseous process in our patient had a different topography. Although its origin remains unknown, the osseous process may be a remnant of an occipital vertebra, as is suspected for other anomalous osseous processes in this region.

Artery-to-artery embolism as occurred in this patient is a rather unusual consequence of mechanical compression of the VA associated with head rotation. At operation, the arterial wall was focally thickened and sclerotic at the compression site, suggesting repeated pinching of the VA between osseous structures. Thus, pinching of the artery might have represented a different type of trauma than other compressive mechanisms associated with soft tissue or osteophytes within the transverse foramen.

Treatment options in our patient included conservative therapy with anticoagulation and antiplatelet drugs, endovascular occlusion of the left VA, surgical decompression, and atlantooccipital fusion. The patient chose to discontinue conservative therapy. Considering the patient’s relative youth, we performed surgical decompression of the VA to preserve antegrade flow in the left VA and range of motion of the atlantooccipital joint. For appropriate decompression, identification of the occlusion point is essential and requires dynamic DS angiography as reported previously. Angiography using three-dimensional CT may be an alternative. Long-term follow up is mandatory because of the possibility of repeated occlusion following posterior decompression of the VA.

References

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