Pearls & Oysters: A distinctive watershed area in the vertebrobasilar territory

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PEARL

- Hemodynamic compromise to the vertebrobasilar circulation, most often through bilateral vertebral artery disease, can cause selective ischemia of both middle cerebellar peduncles.

The middle cerebellar peduncles may represent a distinctive watershed area in the vertebrobasilar circulation between the anterior inferior and superior cerebellar arteries.

CASE REPORT A 78-year-old man experienced 2 short-lasting episodes of vertigo with unstable gait during the 5 days before admission. One week previously, he had carried large fiberboard panels, involving sustained rotation of the neck in both directions. His past medical history included arterial hypertension and dyslipidemia. The initial general and neurologic examination was normal except for high blood pressure (194/109 mm Hg). Over the next few days, the patient developed rapidly worsening bilateral dysmetria and dysidiadochokinesia, more marked on the left, cerebellar dysarthria, and unstable stance and gait.

Cerebral CT showed leukoaraiosis and lacunar sequelae of both corona radiata and of the left thalamus and caudate nucleus. CT arteriography showed narrowing and irregularities of the V2 segment of the left vertebral artery and occlusion of its V3 segment. Ultrasonography and Doppler examination confirmed the stenosis of the left V2 segment and occlusion of the left V4 segment. There was also an 80% stenosis in the V2 segment of the right vertebral artery. Cerebral MRI showed symmetric acute ischemia of both middle cerebellar peduncles (MCPs) and of the deep right cerebellar hemisphere (figure, A). Fat saturation sequences showed a wall hematoma of the V2 segment of both vertebral arteries (figure, B), extending to V3 and V4 on the left side, suggesting bilateral vertebral artery dissection.

Oral aspirin and cautious anticoagulation with IV heparin were introduced. Severe hypertension required IV labetalol and sodium nitroprusside.

Cerebral arteriography was performed. Following aortic contrast injection, brain perfusion was delayed in the posterior fossa. Carotid artery catheterization disclosed a fetal origin of the left posterior cerebral artery and a very small right posterior communicating artery. Thus, the carotid circulation provided little blood supply to the posterior fossa. Both vertebral arteries showed diffuse stenosis, suggesting atherosclerosis. The very narrow left vertebral artery received small anastomoses from the left occipital and ascending cervical arteries. However, the basilar artery showed very little opacification following injection of the left subclavian artery (figure, C). Thus the right vertebral artery was the only significant vessel contributing blood flow to the basilar artery despite severe stenosis of the V2 segment (figure, D). Angioplasty of this stenosis was deemed too risky because of the absence of alternative blood supply to the posterior fossa during balloon inflation.

Oral anticoagulation was started. Over the next 3 weeks, the patient's condition stabilized and then slowly improved. After 3 months, he was able to walk with a walker and was independent for almost all activities of daily living. Five months later, he died of a massive hemorrhage in the right cerebral hemisphere that did not appear to involve the vertebrobasilar circulation.

DISCUSSION Our patient presented with rapidly worsening bilateral cerebellar signs caused by ischemia of both MCPs. Bilateral atherosclerosis and dissection of the vertebral arteries compromised blood supply to the posterior fossa. The dissections were likely brought about by the prolonged neck rotation causing excessive strain on fragile, atherosclerotic arteries.

Blood flow to the MCP is generally supplied by the anterior inferior cerebellar artery (AICA) with a contribution from the superior cerebellar artery (SCA).^{1,2} The AICA also supplies the lateral part of the caudal two-thirds of the pons and a portion of the cerebellar hemisphere including the flocculus.

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(A) Diffusion-weighted MRI showing acute ischemia of both middle cerebellar peduncles (MCPs) and of the deep right cerebellar hemisphere (arrows). (B) Fat saturation T1-weighted MRI showing hyperintensity of the V2 segment of both vertebral arteries (arrows). (C) Left subclavian arteriography showing occlusion of the V3 segment of the left vertebral artery (arrow). (D) Right subclavian arteriography showing 80% stenosis of the V2 segment of the right vertebral artery (arrow).

Consequently, complete infarction of the AICA causes deficits of cranial nerves V, VII, and VIII in addition to cerebellar signs.¹

In our patient, the selective ischemia of the MCP, sparing the brainstem, explains the absence of cranial nerve deficits. The symmetric involvement of both MCPs in the context of severe compromise of blood supply to the posterior fossa suggests a hemodynamic mechanism.

Four patients with bilateral MCP ischemia have been reported. Only one presented with isolated bilateral cerebellar signs.³ The right vertebral artery was occluded in its V4 segment and the basilar artery, which was supplied by the left vertebral artery, was markedly stenotic just proximal to the origin of the AICAs. Two patients had unilateral or bilateral auditory dysfunction in addition to bilateral cerebellar signs, likely caused by internal auditory artery ischemia.^{4,5} In one case, both vertebral arteries were

occluded in their V3 segment due to traumatic dissection, and the basilar artery and AICAs were supplied through the posterior communicating arteries⁴; in the other, there was an occlusive dissection of segment V4 of the right vertebral artery and a hypoplastic left vertebral artery supplied the basilar artery and AICAs.⁵ The fourth patient had several cranial nerve deficits in addition to bilateral cerebellar signs. There was occlusion of the left vertebral artery in its V3 segment and of the right vertebral artery near its origin, with collateralization to the posterior fossa from the anterior spinal artery, cervical muscular branches, and circle of Willis.⁶ In all cases, both AICAs were visualized, suggesting that the MCP ischemia resulted from hemodynamic compromise rather than arterial occlusion. There was no clinical or radiologic difference between the 2 patients with intracranial vertebral or basilar artery disease and the 2 patients with extracranial vertebral artery disease.

Apart from ischemia, other diseases can involve the MCP bilaterally. These include degenerative disorders (e.g., multiple system atrophy), metabolic diseases, hypoglycemia, neoplasms, demyelinating disorders, and inflammatory diseases.⁷

The optimal management and prognosis of hemodynamic compromise to the MCPs are unknown.⁸ Patients with severe stenosis due to vertebral artery dissection often receive anticoagulation rather than antiplatelet agents. However, there is a risk of subarachnoid hemorrhage, especially when the dissection involves the intracranial V4 segment. Stenting of vertebral artery dissection or stenosis may be considered, but its efficacy remains to be better evaluated. Our patient improved functionally after several weeks of rehabilitation, but, similarly to the previously reported patients,^{3–6} did not achieve full recovery.

AUTHOR CONTRIBUTIONS

Pierre Mégevand cared for the patient, searched the literature, and drafted and revised the manuscript and figure. Maria Isabel Vargas performed the MRI, searched the literature, and revised the manuscript and figure. Hasan Yilmaz performed the arteriography and revised the manuscript and figure. Fabienne Picard cared for the patient and drafted and revised the manuscript and figure.

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DISCLOSURE

Dr. Mégevand, Dr. Vargas, and Dr. Yilmaz report no disclosures. Dr. Picard has received an educational grant from UCB for the making of a DVD on art and epilepsy.

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