ARterioVENous Malformations of the Posterior FOssa

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In recent years there has been an increasing interest in arteriovenous malformations of the posterior fossa and a number of new cases have been reported. Verbiest11 in 1961 found 108 cases in the literature since 1914.

We wish to report a case of an intradural arteriovenous malformation of the posterior fossa which was filled exclusively by a branch of the left (contralateral) external carotid artery and drained over the surface of the right cerebellum into the vein of Galen. Although this malformation was located completely within the dura mater of the posterior fossa it did not fill from the vertebral-basilar system or from the internal carotid circulation of either side.

CASE REPORT

H.K., a 58-year-old right-handed itinerant race-track manager, was admitted to the Emergency Room of the Hitchcock Hospital on Sept. 6, 1961. He had had a sudden, severe headache 3 hours previously. At the onset of the headache he became “blind and could not speak for several minutes.” There was no loss of consciousness, and he experienced no vertigo, weakness, numbness or incoordination. On regaining his vision there was no diplopia or blurred vision. He vomited three times on route to the hospital.

His past history revealed that he had an extensive physical examination with findings that were considered to be normal 1 year prior to admission. Three months before admission he noted the onset of morning headaches, occurring about three times a week and lasting several hours.

Examination. The patient was alert, oriented and cooperative. Blood pressure was 112/70, pulse rate 80, respiratory rate 12. There was no aphasia, but his speech was dysarthric. The fundi were normal. The neck was very stiff with positive Kernig and Brudzinski signs. The extraocular movements were full and the visual fields were normal to confrontation. There was no facial weakness. The corneal and gag reflexes were brisk and the tongue protruded in the midline. No bruit was heard on careful auscultation of the head. The deep tendon reflexes were slightly increased on the right side, but the plantar response was flexor bilaterally. He had no weakness of muscles, sensory loss or ataxia. Plain roentgenograms of the skull and chest were normal. A spinal tap revealed bloody cerebrospinal fluid with a pressure of 280 mm.

A serial left carotid angiogram was performed on the day of admission revealing an arteriovenous malformation on the right side of the posterior fossa (Figs. 1 and 2). The malformation was fed by the left posterior meningeal artery which came off the left occipital artery, entered the jugular foramen, and crossed to the right side as it traversed the inner aspect of the occipital bone. The vascular malformation started at the level of the torcular Herophili and extended forward to drain into the vein of Galen. Branches of the left internal carotid artery did not communicate with the malformation. To our surprise serial right carotid and vertebral angiograms failed to show the malformation.

Course. Since the patient became drowsy, more dysarthric, and lost his capacity for upward gaze, and since there appeared to be a single feeding vessel from the external carotid systems it was decided that surgical intervention would be attempted, although we were well aware that clipping a single “feeder” usually is unsuccessful.

Operation. On the 5th hospital day a posterior-fossa exploration was performed. The vascular malformation was located over the vermis and right cerebellar hemisphere extending forward into the region of the vein of Galen. The left posterior meningeal artery was a single, greatly enlarged intradural vessel. Several silver clips were needed to occlude it completely before dividing it. A postoperative serial left carotid arteriogram showed the feeding vessel occluded and the vascular malformation no longer filled (Fig. 3).

Course. The dysarthria, limitation of upward gaze and drowsiness rapidly cleared postoperatively and no
neurologic deficit could be detected at the time of discharge 3 weeks later.

DISCUSSION

The patient with an arteriovenous malformation of the posterior fossa may present clinical signs in one of several ways. In most of the cases a subarachnoid hemorrhage occurs which is followed by unequivocal signs of cerebellar or brainstem disease. These cases usually do not present a diagnostic problem. Logue and Monckton, however, pointed out that a subarachnoid hemorrhage may be the sole manifestation of a posterior-fossa malformation and that the diagnosis can be made in these cases only if vertebral arteriography is done after bilateral carotid angiograms have failed to disclose the source of the bleeding.

When the signs caused by the malformation are of slow intermittent progressive damage to the brain stem, cerebellum or cranial nerves, the condition may simulate multiple sclerosis, to which Martin, Hierons, and Teilmann have drawn attention. This progression in a stepwise fashion may be caused by a small hemorrhage, thrombosis, or by direct pressure from gradual enlargement of the malformation. In 2 of the cases reported by Logue and Monckton the disease progressed 15 years before the true nature of it became known. In these cases radiological changes such as an enlarged vascular groove, calcification in the posterior fossa, or enlargement of the foramen jugulare or transversarium may be helpful. The presence of cardiomegaly may be of great importance. The family history should not be neglected, and the presence of an intracranial

![Fig. 3. Lateral carotid angiogram identifying the malformation in the posterior fossa. Note feeding vessel from external carotid circulation just inside occipital bone.](image)

bruit should suggest the diagnosis. It has been emphasized by Verbiest that patients with intermittent and progressive signs limited to the region of the brain stem and cerebellum should be subjected to vertebral angiography.

Occasionally an arteriovenous malformation of the posterior fossa may present as a space-occupying mass which may or may not cause obstructive hydrocephalus. Characteristic of the lesions causing hydrocephalus are headache, vomiting, diplopia, and papilledema. It is difficult to determine what factors are involved in precipitating the hydrocephalus. It is of interest that Olivercrona and Ladenheim have described pseudo-encapsulation of intracranial arteriovenous malformations by the gliotic reaction of the parenchyma of the brain and by pathologic changes within the walls of the blood vessels such as hyalinization and calcification. The part played by blood in the subarachnoid space as an etiologic factor in the development of hydrocephalus as described by Kibler et al. also must be considered.

Rarely a patient with a posterior-fossa arteriovenous malformation may present trigeminal pain as the predominant symptom. This was seen as the only symptom for 4 years in a 23-year-old male reported by Verbiest.

The advent of vertebral angiography has made the precise delineation of these lesions possible during life. One of the most interesting aspects of our case is that the vascular malformation did not fill by the vertebral-basilar system. We were

![Fig. 8. Postoperative angiogram showing the feeding vessel occluded by clips and the malformation not filling.](image)