Hematoma of the quadrigeminal plate

Case report

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The authors report a case in which a hematoma of the quadrigeminal plate resulted from the rupture of a "cryptic" arteriovascular malformation (AVM) fed by the artery of the quadrigeminal plate. The AVM was symptomatic before rupturing and this clinical feature, associated with the lack of angiographic demonstration, led to the erroneous preoperative diagnosis of a tumor. In retrospect, the only finding that might have suggested the correct diagnosis was the beaded appearance on the ventriculogram of the aqueduct due to blood clots into this fluid space.

KEY WORDS • cryptic arteriovenous malformation • aqueduct • ventriculography

Intra-axial hematomas have rarely been reported in the surgical literature. Interest has been focused on the feasibility of approach to and removal of these benign lesions in the critical areas of the brain stem.

We are reporting a hematoma of the roof of the midbrain resulting from a ruptured cryptic vascular malformation fed by the artery of the quadrigeminal plate.

Case Report

This 38-year-old woman had a 2-year history of acute episodes of headache which subsided spontaneously or with mild analgesics. The headaches had increased in frequency during the last month, and diplopia developed associated with vomiting. An ophthalmologist noted bilateral papilledema and right sixth nerve palsy. Two days before admission the patient experienced acute headache, radiating to the spine and legs, followed by vomiting and drowsiness.

Examination. On admission on April 2, 1977, the patient was in distress and drowsy. Positive neurological findings included neck stiffness, bilateral sixth nerve palsy, a bilateral Babinski sign, brisk deep reflexes on the left, bilateral papilledema, and a questionable upward gaze palsy. Skull films showed a slightly demineralized dorsum sellae; a Tc\(^{99}\) radioisotope brain scan showed nothing abnormal. The cerebrospinal fluid was moderately hemorrhagic and under increased pressure. During the night the patient became comatose. Bilateral carotid angiography showed supratentorial hydrocephalus. External ventricular drainage resulted in prompt recovery of consciousness. At this point, marked impairment of upward gaze was noted. Conray\(^{60}\) ventriculography confirmed the presence of hydrocephalus, including the third ventricle. The suprapineal recess was displaced upward and forward while the fourth ventricle was normal in size.
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Fig. 1. Ventriculogram with Conray⁴⁴. Left: Early phase. The third ventricle is dilated. The fourth ventricle, which is faintly visualized, is normal in size and position. The aqueduct is pushed forward. Right: The late phase reveals a “beaded” aqueduct. This finding is due to blood clots that had penetrated the aqueduct from the quadrigeminal plate.

and position. The aqueduct was almost completely blocked and pushed forward on the early film (Fig. 1 left). On the later films the aqueductal area appeared “beaded,” suggesting an intra-aqueductal hourglass-shaped mass (Fig. 1 right). Left retrograde brachial angiography revealed stretch and depression of both pre- and lateropontine segments of the anterior superior cerebellar arteries, and backward displacement of their vermian branches (Fig. 2 left). In the venous phase, the precentral cerebellar vein was displaced backward, especially at the level of its colliculo-central point (Fig. 2 right). The preoperative diagnosis was hemorrhagic tumor of the quadrigeminal plate.

Operation. This region was exposed on April 5 through an infratentorial supracerebellar approach under the microscope. On section of the precentral cerebellar vein, a bluish-colored mass was seen occupying the central part of the cisterna ambiens. The mass was made up of blood clots which were removed piecemeal. During this procedure, an arterial vessel was found, which entered the field from the left and ran toward the brain stem. This artery of the quadrigeminal plate was occluded and followed anteriorly to expose a cryptic vascular malformation, which was completely obliterated by bipolar current. The removal of all clots caused a jet of fluid to issue from the third ventricle. A

Fig. 2. Left: Left vertebral arteriography, lateral view. The anterior superior cerebellar arteries are stretched and depressed (arrows). Right: Left vertebral phlebogram, lateral view. The precentral cerebellar vein is pushed backward, especially at the level of the colliculo-central point (arrow).
A small piece of the wall of the hematoma was removed for histological examination. At this point a thin catheter was inserted into the fourth ventricle through the foramen of Magendie, and appeared freely visible for about 2 cm before entering the third ventricle. It was believed that the vascular malformation involved the distal part of the quadrigeminal plate and that the hematoma had destroyed the anterior medullary velum and the quadrigeminal plate and had compressed the lingula and precentral lobe. The wound was closed in watertight fashion.

The histological study of the wall of the hematoma demonstrated glial tissue with small hemorrhagic areas. Around some blood vessels there were macrophages and lymphoid cells. No tumor tissue was found.

Postoperative Course. The postoperative course was uneventful and the patient was discharged on the eighth postoperative day. At that time papilledema had almost completely regressed and the only positive findings were slight impairment of upward gaze and a right fourth nerve paresis. One month after surgery, the optic discs were normal and upward gaze was full; however, the defect of the right fourth nerve persisted. Seven months later, neurological examination was normal, as was a computerized tomography scan.

Discussion

The surgical literature includes several cases of hematomas involving the brain stem. Most of them were found in the pontine or medullary areas, only one was located at the midbrain, but that extended ventrally into the aqueduct. The prevalence of hemorrhages into the pontine area is clearly related to the greater incidence in this region of “cryptic” vascular malformation which, when they rupture, probably provide the basic pathology.

The quadrigeminal plate is a well defined area, supplied by a pair of arteries and drained by branches of the precentral cerebellar vein. Our observation has shown that this area may harbor a cryptic arteriovascular malformation (AVM). When the AVM ruptured in our case, both the intraxial and cisternal hematomas resulted.

The lack of vertebral angiographic visualization of cryptic AVM’s has been emphasized. These small lesions may even escape pathological and anatomical examination, probably due to the fact that they are destroyed together with the brain substance by any massive hemorrhage. In our case the AVM was clearly seen under the microscope, closely resembling Case 1 of Margolis, et al. Spasm of the artery into the quadrigeminal plate might explain lack of angiographic visualization, and the fact that no early filling of the precentral cerebellar vein occurred might support such a hypothesis.

Due to the relationship of the precentral cerebellar vein to the inferior colliculus, the precentral lobule, and lingula, backward displacement of this vein in our patient associated with forward displacement of the aqueduct indicated a mass in the region of the quadrigeminal plate and anterior medullary velum. A pinealoma was ruled out because the suprapineal recess was patent.

In the absence of angiographic demonstration of an AVM, the beaded appearance of the aqueduct might have led to a suspicion of hematoma. This ventriculographic appearance is explained on the basis of the operative findings, which showed two contiguous blood clots embedded in the aqueduct.

In view of the operative findings, upward gaze palsy could be anticipated as a permanent defect; on the contrary, the patient is neurologically normal 7 months following surgery. As in cases with recovery of upward gaze following the removal of pinealomas or ependymal cyst, this means that the quadrigeminal plate was not destroyed by the hematoma, but simply separated along the midline and compressed, so that superior and inferior colliculi were displaced laterally on each side. This mechanism could also explain the absence of any auditory disturbances.

References

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