Posterior fossa subdural hematoma due to ruptured arteriovenous malformation

Case report


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Patients with cerebellar arteriovenous malformations (AVM) commonly present to the neurosurgical department after having suffered hemorrhages. The subarachnoid space is the usual location for these often repeating episodes of bleedings. In addition, these patients can present with parenchymal hemorrhage. Acute subdural hematoma caused by a ruptured cerebellar AVM is a rare entity and is not generally recognized. The authors present a case of acute posterior fossa subdural hematoma resulting from a ruptured cerebellar AVM.

KEY WORDS • posterior fossa • subdural hematoma • cerebellum • arteriovenous malformation

The overall incidence of acute SDH occuring in the posterior fossa is very low. Most cases are trauma related in origin. Trauma-related acute posterior fossa SDHs are believed to comprise approximately 0.3% of all cases of acute SDHs. It is difficult to estimate the exact incidence of nontrauma-related acute posterior fossa SDHs. Only a few such cases have been reported in the literature. Unusual causes of acute SDHs include bleeding and blood clotting disorders, eclampsia, disseminated intravascular coagulation, tumors, spinal anesthesia, ruptured aneurysms, iatrogenic causes such as complications related to endovascular procedures, endoscopic ventriculostomy, and "spontaneous SDHs."

Whether these lesions are traumatic or nontraumatic in origin, the morbidity and mortality rate of acute posterior fossa SDH remains very high. Recognition and prompt treatment are the key factors for the management of these cases.

CASE REPORT

Presentation. This 48-year-old man with a history of good health presented to the casualty department with a history of severe headache followed within minutes by deep coma. Although Glasgow Coma Scale score was noted to be 3 in the casualty department, there was some extensor limb response after the patient received initial care in the ward. The patient could not open his eyes or make verbal responses. Pupil size was small (2 mm) and the pupils were unresponsive.

Examination. An emergency CT scan revealed an acute posterior fossa SDH associated with hydrocephalus (Fig. 1). The patient was taken to the operating room immediately. By then his pupils had dilated bilaterally, his limbs were flaccid, and there was no response to painful stimulus.

Operation. The patient was placed in the prone position. A ventricular drainage line was established via an occipital burr hole, and a suboccipital craniectomy was performed. After opening the dura mater, it was apparent that the bleeding was still active. Removal of the clot exposed the bleeding site of the AVM on the surface of the left cerebellar hemisphere. The lesion was less than 3 cm in size (Fig. 2). An arterialized vein was draining superficially toward the transverse sinus. The main feeding vessel was thought to be a branch of the posterior inferior cerebellar artery. The bleeding vessel was coagulated first and then the whole of the AVM excised. After the excision was complete, the arterialized vein (carrying mixed blood) returned to its normal appearance.

The patient underwent elective ventilation, and the external ventricular drain was maintained for 14 days until a ventriculoperitoneal shunt was placed.

Abbreviations used in this paper: AVM = arteriovenous malformation; CT = computerized tomography; MR = magnetic resonance; SAH = subarachnoid hemorrhage; SDH = subdural hematoma.
Postoperative Course. During the initial postoperative period, it was quite clear that the patient was having difficulty swallowing and breathing. He required ventilatory support for a prolonged period (15 days). He was fed with Ryle’s tube. Although being fed, he developed aspiration pneumonia, which hampered early recovery. He was discharged from the hospital 3 months after admission. He still experienced diplopia, but his ataxia had resolved. He was examined again in the outpatient clinic 4 weeks postdischarge, at which time a full recovery was demonstrated.

No postoperative angiography was performed because he was reluctant to undergo any invasive investigation. Follow-up MR imaging and MR angiography did not demonstrate any residual AVM.

DISCUSSION

Arteriovenous malformations, considered developmental vascular malformations rather than tumors, are classified into five main groups: cavernous malformation (angioma), telangiectasia, varix, venous malformation, and AVM. Arteriovenous malformations are less common than aneurysms. According to results reported in a cooperative study, the overall ratio of AVM to aneurysm is 1:6.5 (1.4% of the patients with intracranial aneurysm harbored a coexistent AVM).

Although the incidence of these lesions is relatively rare, they are extremely demanding and often challenging to the entire neurosurgical team, including those in radiosurgery and neuroradiology services. The overall probability of a fatal hemorrhage-related outcome is 40.5%. Mortality rates tend to be higher in patients in whom additional bleeding occurs subsequent to an initial episode as compared with those suffering a first-time hemorrhage. Of patients suffering from rupture of an AVM, 16 to 19% are likely die of bleeding. In addition, there is definite risk of deterioration of neurological status with or without additional bleeding. The psychological stress experienced by an individual harboring a potentially catastrophic lesion is considerable.

Of all cases with cranial AVMs, more than half present with bleeding, fewer than half with epilepsy, and some with neurological deficit, headache, and dementia. Patients who present with bleeding tend to suffer additional episodes of bleeding more often than those presenting with epilepsy. However, some patients present with both epilepsy and hemorrhage. Surgical treatment is undertaken primarily to prevent bleeding and its consequences. It has been found that epilepsy was controlled in 14% of the patients who underwent surgery, whereas 22% of the patients in whom epilepsy was not present before surgery developed epilepsy postoperatively.

Additional presenting signs and symptoms cited in the literature include hydrocephalus, raised intracranial pressure, papilloedema, ataxia, weakness of limbs, dysfunction of cranial nerves, and various levels of impairment of consciousness.

Clinically, posterior fossa AVMs behave differently from those located in the supratentorial region. The incidence varies between 2 to 18% of all the cranial AVMs. The widely quoted incidence is 7%, which was reported in the cooperative study of cerebral AVMs and fistulas. Generally speaking, approximately 10 to 15% of all types of AVMs treated at a neurosurgical referral center will be posterior fossa AVMs.

Posterior fossa AVMs alone do not cause epilepsy. Hence the main presentation demonstrated in this group is bleeding. In some series almost all the patients with posterior fossa AVMs had suffered an episode of bleeding. According to findings reported in a cooperative study hemorrhage was found to have occurred in over 81% of patients harboring infratentorial lesions, whereas fewer than 68% of the patients with supratentorial AVMs suffered hemorrhage. Although most commonly the AVM was the source of the hemorrhage, on occasion the cause was an aneurysm associated with the AVM. In fact, it has been more often found (65% of patients) that the aneurysm, not the AVM, ruptured. Between 10 to 20% of posterior fossa AVMs are associated with aneurysms. The aneurysm may or may not be formed in the main feeding vessel. Sometimes aneurysms can be multiple.

The most common locations of these lesions are in the cerebellar hemispheres and the vermis. Other loca-
tions include the brainstem, tonsils, and cerebellopontine angle. A patient may harbor more than one discrete AVM.

The arterial supply for these malformations usually comes from the superior cerebellar artery, anterior inferior cerebellar artery, or posterior inferior cerebellar artery and occasionally from the occipital or vertebral artery.6,8,21 Venous drainage varies;5,7,32 from the deep system (straight sinus or Vein of Galen) or to the superficial channels, irrespective of whether the lesion is hemispheric or vermian in location. Brainstem lesions are more likely to drain into the deeper channels.32

As we mentioned previously, ruptured AVM is the most common presentation of posterior fossa malformations. These patients present with the signs and symptoms of SAH.21 A smaller number may suffer parenchymal hemorrhage, and in such cases cerebellar signs may be demonstrated. In one series 28 all the patients suffering from cerebellar parenchymal hemorrhage caused by the rupture of a vermis-located AVM also experienced either intraventricular bleeding or SAH. Depending on the size of cerebellar hematoma, compression on the brainstem, and secondary hydrocephalus, the patient’s neurological state can vary from simple headache, dizziness, and vertigo to nystagmus, cranial nerve palsy, and coma.

Some patients present with occipital- or mastoid-related pain or a simple headache. Others may present with hemifacial spasm, trigeminal or glossopharyngeal neuralgia, ataxia, diplopia, and other cranial nerve deficits.6,12,25,29–32 One patient presented with spasmodic torticollis. Very occasionally an AVM can present with clinical features of intracranial hypertension or multiple sclerosis.

Whereas SAH seems to be more common in younger patients, an ischemic deficit is more common in older patients.28 Although factors that correlate with increased risk of hemorrhage cannot be delineated with absolute certainty, small size,14,39 deep venous drainage, hypertension,19 and also the arterial pressure of the feeding vessel could well be of significance. Site, size, and side of the lesion and sex of the patient have not been shown to correlate with the tendency to hemorrhage.11 The presence of coexistent aneurysm(s) increases the risk of bleeding.4

Cases of ruptured cerebellar AVM presenting with SDH are extremely rare. However, a few cases of acute SDH following ruptured aneurysms have been reported.25

Clinical examination and patient history may provide insight into the diagnosis but must be verified by CT scanning, MR angiography, and digital subtraction angiography studies. In an emergency situation CT scanning is the most important tool for determining subsequent treatment. In dire cases such as the one presented here, there may be no time for further investigation before taking the patient to the operating room. However, as soon as the situation permits, one should perform cerebral angiography. For surgical planning, MR imaging in addition to MR angiography is an extremely valuable diagnostic tool.

Planning of treatment is extremely important1,10,11 in such cases in which the outcome can be so variable. Surgical- or alternative therapy–related risks must be assessed properly in light of the patient’s health, risk of bleeding, morbidity, and ability to undergo surgery. Other modes of treatment include embolization with or without surgery, radiosurgery, or a combination of these modes.8,34 Grading of AVM can provide valuable guidance in the matter.15

CONCLUSIONS

Patients with posterior fossa AVMs can present with SDH in the posterior fossa. These patients may present in critical condition, not unlike those in whom aneurysms have ruptured into the subdural space. However serious the initial pre- and postoperative deficit may be, it is possible for these patients to recover completely within a reasonable period of time. Surgical treatment should be made available even in extreme cases.

References


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