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CASE REPORT

Traumatic epidural and subdural hematomas and extensive brain infarcts in a patient with pial arteriovenous malformation: Mechanisms underlying clinical and radiological findings

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ABSTRACT

We report a rare case of a patient with a pial arteriovenous malformation (AVM) who presented in altered sensorium. He was found to have large epidural and subdural hematomas overlying a pial AVM. He underwent evacuation of these hematomas and postop computed tomography showed infarcts deep to the site of hematoma evacuation. These infarcts were postulated to be due to a steal phenomenon combined with raised intracranial pressure. The management and possible mechanisms for this rare combination are discussed.

Key words: Arteriovenous malformation, epidural hematoma, infarcts, steal phenomenon, subdural hematoma

Introduction

Arteriovenous malformations (AVMs) usually become symptomatic due to the occurrence of hemorrhage, seizures, or steal phenomena.[1] They frequently remain asymptomatic. We report a patient with an AVM that was discovered due to an unusual set of circumstances. We also comment on an unusual epiphenomenon associated with the lesion.

Case Report

A 40-year-old man was brought to the emergency department, having been found lying by the road side. He had abrasions on the left frontal and temporal regions of the scalp. On examination, his Glasgow Coma Score (GCS) was E1 M5 V2 (8/15). He had paucity of right-sided limb movements to painful stimuli; his left pupil was larger than the right and not reacting to light. This was clinically suggestive of left uncal herniation. The exact time of injury could not be ascertained. He was investigated with an urgent plain computed tomography (CT) scan of the head. The CT scan showed a large left temporoparietal epidural hematoma (EDH) causing significant mass effect on the lateral ventricles and a midline shift of 1.5 cm [Figure 1b]. There was a linear fracture of the temporal bone [Figure 1c]. There was an underlying acute subdural hematoma (acSDH) associated with severe cerebral edema with effacement of all the basal cisterns [Figure 1a].

Immediate surgery was decided on and he was shifted to the operating room within 60 min of being brought to the hospital (an analysis of the records revealed that this was the time taken for doing the CT scan, drawing blood for cross-match, and an urgent head shave). He underwent emergency left frontotemporoparietal craniotomy. During surgery, a linear fracture of the temporal bone was noted. Upon elevating the bone flap, a thick EDH was encountered and was evacuated. The source of the bleed was a laceration of the posterior branch of the middle meningeal artery (MMA), which was coagulated. The artery did not appear to be unduly enlarged. After evacuation of the EDH, the dura remained tense and had a bluish discoloration. The dura was opened and a thick subdural clot was encountered; this was evacuated. After evacuating the SDH, it was found that the posterior frontal lobe and the temporal lobe were covered by numerous distended, arterialized veins. The veins of the Sylvian fissure were grossly distended and arterialized [Figure 1d]. There was a pial laceration adjacent to the large Sylvian vein

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which was the source of the SDH. This bleeding was controlled with gelatin sponge tamponade.

Postoperatively, the pupillary asymmetry reverted. However, he remained comatose, with a GCS of E2 M5 V2 (9/15). A postoperative contrast-enhanced CT (CECT) showed complete evacuation of the EDH and SDH with reversal of the midline shift [Figure 2a]. However, cerebral edema persisted. Multiple infarcts were seen in the left medial temporal lobe and centrum semiovale [Figures 2a and b]. These were distributed in the left middle cerebral artery (MCA) and anterior cerebral artery (ACA) territories. There was a small bleed in one of the infarcts, suggestive of hemorrhagic transformation [Figure 2a]. CECT showed a nidus in the left superior frontal region [Figure 3a]. Angiogram showed a left superior frontal AVM fed by branches of the left MCA and the left ACA draining into the Sylvian vein, and superior sagittal sinus [Figure 3b]. External carotid artery (ECA) injection did not show any feeders to the AVM. The nidus measured 4.5 cm supero-inferiorly. The Spetzler–Martin grade was 3. Surgery was offered for excision of the AVM; however, since he continued to be in altered sensorium, the patient's relatives were not willing for any further treatment and he had to be discharged at request. No further follow-up data is available.

Discussion

EDHs are usually coup lesions and are caused either by seepage of blood from a calvarial fracture or injury to the dural arteries. There have been case reports of spontaneous EDH as well.[2,3] An acute SDH, on the other hand, is generally contrecoup in location and venous in origin. It is unusual, but not uncommon to find both an EDH and SDH on the same side in patients with severe head trauma.[4] The cause of the EDH is thought to be direct trauma and the SDH is probably due to brain recoil causing damage to the cortical bridging veins. In the present patient, the co-existence of an EDH and SDH on the same side was presumed preoperatively to be due to a similar mechanism. Hence, only a plain CT was done prior to surgery, as this is the standard protocol for patients brought with definite history of traumatic brain injury without any ambiguity as to the causation. It was realized in retrospect that no definite history regarding the mode of injury or the time of injury could be obtained in this case since he had been found in altered sensorium by the police patrol.

An AVM can present with parenchymal, subarachnoid, intraventricular, or subdural hemorrhage.[5] This patient had the unusual combination of coup and contrecoup hematomas on the same side. EDH is rarely caused by AVM. Spinal EDH

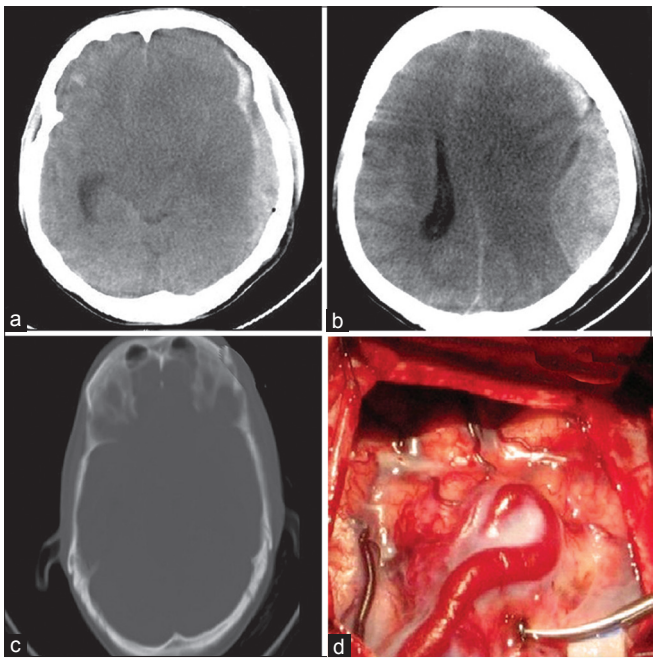

Figure 1: (a) Preop plain computed tomography axial image showing a thick left frontoparietal acute subdural hematoma. (b) Preop plain computed tomography axial image showing a left temporoparietal epidural hematoma. (c) Bone windows showing a left temporal linear fracture. (d) Intraop photo showing a lax brain after epidural hematoma and subdural hematomaevacuation and a grossly arterialized Sylvian vein

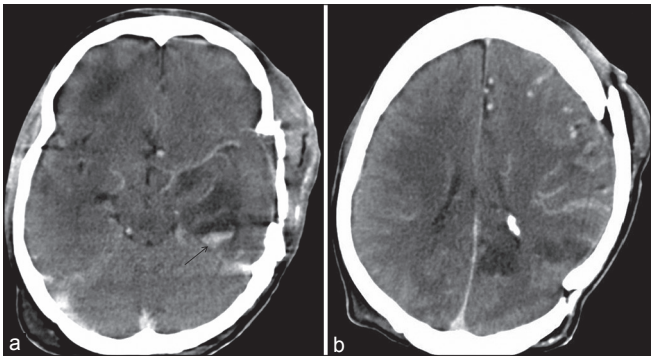

Figure 2: (a) Postop contrast-enhanced CT showing resolution of midline shift, persisting edema. There are multiple deep temporal infarcts with a small area of hemorrhage in one of the infarcts (arrow). (b) computed tomography showing infarcts in the left middle cerebral artery and anterior cerebral artery territory

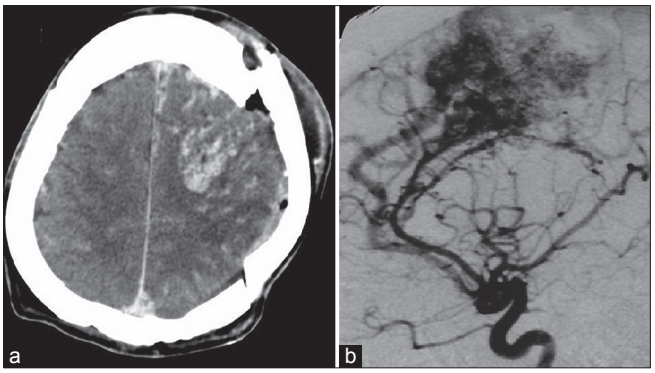

Figure 3: (a) Contrast-enhanced CT showing an enhancing nidus in the left frontal area. (b) Digital subtraction angiogram showing the arteriovenous malformation fed by branches of the anterior cerebral artery and middle cerebral artery

caused by a dural or an epidural spinal AVM has been reported.[6] Theoretically, cranial dural AV fistulae (AVF) should be able to produce an EDH. This could occur either due to direct damage to the nidus of a dural AVM/AVF or due to damage to dilated feeding dural arteries. Dilated MMA or other ECA branches supplying superficial cerebral pial AVMs are susceptible to injury due to trauma and may give rise to EDH. This appears to be an extremely rare phenomenon; in fact, there appears to be no documentation of such an EDH. In the present patient, the angiogram did not show any ECA feeders to the AVM and the MMA did not appear unduly dilated at surgery. The AVM was completely intradural. Thus, trauma to nidus of a dural AVF or dilated feeding arteries is not the cause of the EDH in this patient. Fracture of the temporal bone leading to laceration of the MMA branches (as is usual in trauma) appears to be the cause of EDH.

AVMs do not usually cause acSDH. However, there are reports of dural AVM causing acSDH.[5,7] Posterior fossa AVMs also probably give rise to acSDH more commonly than supratentorial AVMs.[8] Acute SDH can co-exist with a parenchymal hematoma as a result of AVM rupture.[9] In most of the reported cases, rupture of the AVM is the cause of the acSDH. There are no previous reports of direct impact causing damage to arterialized draining veins of an AVM, giving rise to an acSDH.

In the present case, the cause of the EDH was probably direct trauma to the head, causing a temporal fracture, and laceration of the posterior branch of the MMA. The cause of the SDH is likely to have been trauma to one of the arterialized veins draining the AVM due to the trauma. This is, we believe, very rare.[10] Thus, in this case, both the SDH and EDH are actually "coup" hematomas. In a case where direct damage to an arterialized vein (carrying blood at higher pressures) produces an acSDH, the increase in intracranial pressure (ICP) would be much more rapid than with routine traumatic acSDHs, which are venous in origin. Combined with an overlying EDH, the rapid raise in ICP would be catastrophic.

More interesting is the occurrence of numerous infarcts which were not present on the preoperative scan. Posterior cerebral artery (PCA) territory infarcts can occur due to uncal herniation. In this case, however, the infarcts were not in the PCA territory, but involved mostly the left MCA [Figure 2a] and ACA [Figure 2b] territories and were quite extensive. The small area of hemorrhage that is seen in one of the deep temporal infarcts probably represents hemorrhagic transformation of the infarct due to reperfusion injury. This would be secondary to reduced ICP leading to improved perfusion of these areas after surgery [Figure 2a]. The cause of these multiple infarcts, we postulate, could be one of two mechanisms. The first explanation is a pre-existing steal phenomenon decompensated by raised ICP.[11] Feeders to the AVM "steal" blood away from the surrounding brain, and hence regions of the brain supplied by a vessel that feeds an AVM may be just adequately perfused or even borderline hypoxic. When the patient developed two hematomas causing raised ICP and direct compression of the adjacent brain and vessels, the perfusion to this already under-perfused area along the MCA and ACA (feeder vessel) territories became grossly inadequate and infarcts appeared. This can theoretically occur even in cases where the AVM itself bleeds and the resulting hematoma produces raised ICP. Therefore, early relief of ICP would be essential in preventing infarction of otherwise healthy brain tissue in such cases.

The second mechanism could be due to the development of a new steal in those AVMs with ECA supply. Damage to ECA feeders of a pial AVM would lead to an EDH that would rapidly increase in size and tamponade the ECA feeder vessels. Since the AVM nidus is a low-resistance conduit, reduction of ECA inflow may lead to development of a new shunt leading to "stealing" of blood from the ICA circulation into the nidus, resulting in brain hypoperfusion (compounded by raised ICP) and infarcts. Since there was no ECA supply to the nidus in the present case, we believe that the first mechanism may be responsible for the infarcts.

Thus, in addition to the rarity of dual ipsilateral hematomas due to an AVM, the localized infarcts are a rare and interesting epiphenomenon.

Conclusion

Patients with dual hematomas on the same side may rarely have unusual causes for these. Raised ICP due to such hematomas or even bleed from an AVM can potentially cause infarction of brain tissue surrounding the nidus due to the steal phenomenon. This would cause significant morbidity.

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