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Acute Subdural Hemorrhage Complicating Embolization of a Cerebral Arteriovenous Malformation

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Summary: We present a case of a young adult in whom acute subdural hemorrhage developed immediately after embolization of a cerebral arteriovenous malformation with glue. Inadvertent venous outlet obstruction with glue was implicated in the production of the hemorrhage. Possible mechanisms of spread of blood to the subdural space are discussed. Awareness of the possibility of iatrogenic subdural hemorrhage is necessary before undertaking embolization procedures.

Index terms: Cerebral hemorrhage; Arteriovenous malformations, embolization; Interventional neuroradiology, complications; Iatrogenic disease or disorder

Arteriovenous malformations (AVMs) are second in importance to saccular aneurysms as a cause of nontraumatic intracranial hemorrhage. Despite significant advances in the last decade, the management of intracranial AVMs remains one of the most difficult areas of vascular neurosurgery and neuroendovascular therapy.

Endovascular embolization using microcatheters has undergone considerable technical evolution in recent years. This has allowed an increasing number of AVMs to be managed solely by this approach or, more commonly, in conjunction with surgery or radiosurgery. Embolization procedures demand meticulous anatomic, dynamic, and functional assessment of the AVM nidus, feeding arteries, draining veins, and surrounding brain in order to increase the chances of shunt reduction without complications. Despite this, perieMBOLIZATION complications sometimes occur, and can result in severe temporary or permanent neurologic deficit and death (1). The most common complications are those caused by technical mishaps (such as gluing of the microcatheter in an AVM feeder)

vasospasm, dissection or rupture of an intracranial artery, dissection of neck vessels, and intracranial emboli from coaxial catheters in the neck. Brain ischemia caused by occlusion of normal vessels is the most frequent complication after the embolization process itself (2). However, intracranial hemorrhage represents the most life-threatening of these complications. In a recent series of 283 embolized AVMs, perieMBOLIZATION subarachnoid hemorrhage and intracerebral hemorrhage occurred in 3.1% and 2.1% of patients, respectively (2). The pathogenetic mechanisms of these hemorrhages have received little attention in the literature. Other than technical mishaps, four main mechanisms have been suggested in producing rupture of AVMs or adjoining vessels: (1) pressure rises in arterial feeders, (2) venous stasis and thrombosis with delayed AVM rupture, (3) obliteration of associated arteriovenous fistulas causing immediate redistribution into an AVM nidus, and (4) venous outlet obstruction with immediate nidus rupture.

Hemorrhage from endovascular embolization of AVMs has been categorized by Purdy et al (3) into immediate and delayed. Immediate hemorrhage is more likely caused by the catheterization or embolization process, after vascular perforation or rupture. Delayed hemorrhage is more likely to be a hemorrhage into the malformation itself.

Case Report

A 17-year-old boy had a history of five convulsive episodes over the previous few years. There was no history of intracranial hemorrhage and no abnormality on physical

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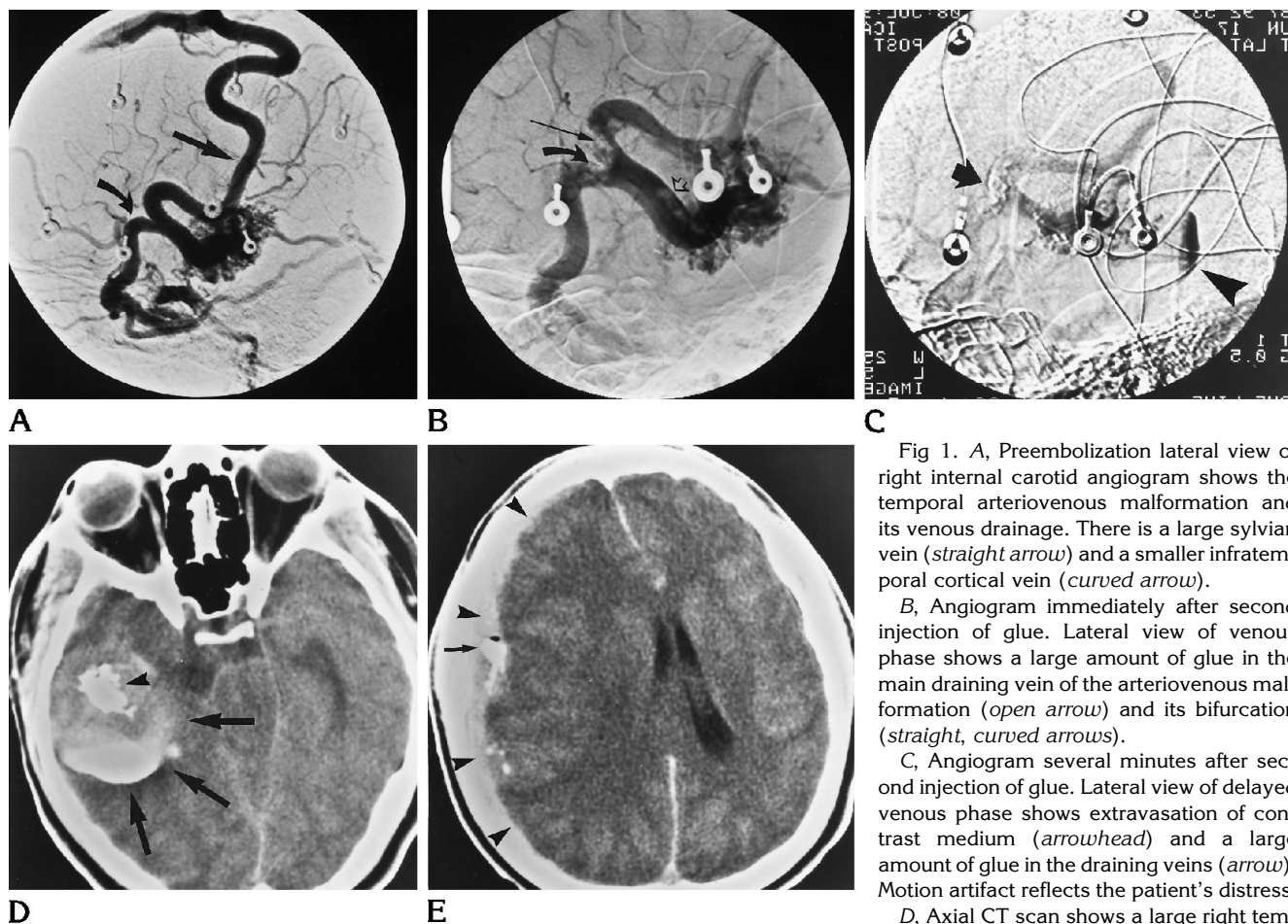


Fig 1. A, Preembolization lateral view of right internal carotid angiogram shows the temporal arteriovenous malformation and its venous drainage. There is a large sylvian vein (straight arrow) and a smaller infratemporal cortical vein (curved arrow).

B, Angiogram immediately after second injection of glue. Lateral view of venous phase shows a large amount of glue in the main draining vein of the arteriovenous malformation (open arrow) and its bifurcation (straight, curved arrows).

C, Angiogram several minutes after second injection of glue. Lateral view of delayed venous phase shows extravasation of contrast medium (arrowhead) and a large amount of glue in the draining veins (arrow). Motion artifact reflects the patient's distress.

D, Axial CT scan shows a large right tem-

poral hematoma (arrows). Opacified glue is seen in the arteriovenous malformation (arrowhead).

E, Axial CT scan shows a moderate-size subdural hematoma (arrowheads). Opacified glue and a small amount of inadvertently injected air are seen in the arteriovenous malformation (arrow).

examination. Investigations revealed a moderate-size AVM in the right anterior temporal lobe including the right superior temporal gyrus. Cerebral angiography prior to therapeutic embolization showed three to four main feeding arteries arising from the right middle cerebral artery, including anterior and middle temporal branches. A smaller feeder originated from the right posterior cerebral artery. Venous drainage was mainly via a large sylvian vein emptying into the superior sagittal sinus and a smaller infratemporal cortical vein to the right cavernous sinus (Fig 1A).

Superselective catheterization of an anterior temporal branch of the right middle cerebral artery supplying the AVM was performed with a 1.8F flow-directed catheter. Functional testing with intraarterial Amytal revealed no clinical or electroencephalographic changes. A 0.35-mL bolus of 1:5 ethiodized oil/Avacryl mixture was injected under fluoroscopy. Postembolization angiograms showed a good result. A second catheter was then superselectively placed in a second right anterior temporal branch supplying the AVM. A 0.3-mL bolus of the same glue mixture was

injected under fluoroscopy. Immediately after injection, we noted that a substantial amount of the embolic agent had reached the main draining vein of the AVM (Fig 1B), which had marked flow stagnation in its more proximal segment. Within 3 minutes of embolization, the patient had a severe headache, rapidly developing left hemiparesis, and diminishing level of consciousness. Anticoagulation was promptly reversed with protamine sulfate, and 50 g of mannitol was given to reduce intracranial hypertension. A postembolization angiogram (Fig 1C) showed nearly total occlusion of the AVM venous outlet, marked contrast stagnation within the AVM nidus, some contrast extravasation, and considerable reduction in the visualized size of the AVM suggesting compression from a surrounding intracerebral hematoma.

The patient had emergency computed tomography of the brain, during which he was intubated and hyperventilated; an emergency ventriculostomy was performed after his pupils became dilated bilaterally and he became comatose. The computed tomogram showed a large hematoma in the right temporal lobe, expanding into the right

frontal lobe (Fig 1D). A moderate-size subdural hematoma had also developed (Fig 1E). There was substantial midline shift and evidence of tentorial herniation.

The patient was transferred to the operating room for emergency surgery. The intracerebral hematoma was evacuated, the AVM excised, and the subdural blood removed. Intraoperative angiography showed complete removal of the AVM. The patient's postoperative course was uneventful. He became clinically stable with some residual but improving upper extremity weakness. He was discharged for rehabilitation on anticonvulsive medication. Follow-up at 6 months showed no residual deficit.

Discussion

The most common pattern of spontaneous AVM bleeding is intraparenchymal and subarachnoid hemorrhage (4). Subdural hemorrhage, whether isolated or in association with intracerebral hemorrhage, is an unusual consequence of spontaneous AVM rupture, with only sporadic reports in the literature (5–9). Arteriovenous malformations potentially may bleed from large feeding arteries, the nidus, or the dilated and often abnormal venous channels draining the lesion. It is generally believed that the majority of bleeding episodes with AVMs arise after rupture of a venous channel or a portion of the AVM nidus in close proximity to its draining veins, where intraluminal pressures are several times greater than in normal cerebral venous channels (4). The abnormal veins of AVMs contain no discernible elastica but show thickened, hypertrophic, and hyperplastic walls nonetheless. Some also have markedly attenuated or thinned walls, which are assumed to be the source of bleeding. However, the site of bleeding from an AVM is usually much more difficult to locate than is the bleeding site from a ruptured saccular aneurysm (10). For subdural hemorrhage to occur, one would have to assume, therefore, that the site of rupture involves abnormal bridging veins traversing the subdural space. An AVM can give rise to subdural hemorrhage by mechanisms similar to those of ruptured saccular aneurysms (9, 11). Thus, four other pathogenetic mechanisms (9, 12) have been proposed for subdural hemorrhage in the presence of an AVM: (a) successive small hemorrhages allow the AVM to develop adhesions to the arachnoid membrane, and the final rupture occurs into the subdural space; (b) the arachnoid membrane is torn by the rapid accumulation of blood under pressure from the leaking

AVM; (c) massive intracerebral bleeding ruptures the cortex and lacerates the arachnoid membrane; and (d) arterial aneurysms located on the feeding arteries of the AVM, if encroaching on the subdural space, can rupture directly, causing subdural hemorrhage. A fifth mechanism was suggested by Hashizume et al (12). Small arterial twigs that arise from the cortical vessels and traverse the subdural space to reach the dura become enlarged in the presence of an AVM to form leptomeningeal recruited feeders or collaterals and may rupture, usually in the absence of trauma, into the subdural space.

The obliteration of the AVM nidus should be the goal of successful embolization (13). Superselective intravascular pressure monitoring of arteries feeding an AVM demonstrates rising pressures as a result of embolization (14). If the malformation is incompletely embolized, residual feeders may be confronted with arterial pressures not previously encountered, resulting in their rupture (3). This local hypertensive mechanism can also occur in other circumstances and has been implicated by Beretta et al (15) in the production of subdural hemorrhage during endovascular embolization of a supratentorial meningioma. Additionally, if an AVM is nearly obliterated, there may be stasis in dilated draining veins that could lead to venous thrombosis and, again, raise the pressure confronting residual arterial feeders. This pathophysiology tends to be delayed, occurring up to 2 weeks after embolization, and may also result in subdural hemorrhage (16). Arterial feeders leading to an arteriovenous fistula in the same vascular territory as an AVM nidus is an association more frequently seen than might be expected (2). Abrupt obliteration of the fistula can cause overwhelming blood redistribution to the AVM nidus, with consequent rupture. Embolization and sudden occlusion of the draining veins can also lead to disastrous consequences. With continued inflow into the malformation but impaired venous outflow, the risk of AVM rupture immediately or in the early postprocedure period increases, as occurred in our patient.

The use of systemic heparinization and heparinized saline to flush the coaxial catheter system can in theory aggravate the resulting hemorrhage. However, the rarity of hemorrhagic complications during AVM embolization, coupled with some evidence to suggest that the use of heparin is not injurious when hemorrhage

occurs (3), are points of reassurance that justify its continued use in our practice.

The polymerization time for embolic glue must be carefully adjusted to the speed and volume of flow through the AVM (13). This requires careful dynamic assessment of preembolization angiograms, especially with regard to AVM angioarchitecture and the timing of venous filling. Precise knowledge of the polymerization characteristics of the embolic agent used are also necessary (17). Establishing the correct ratio of glue and iophendylate for use in each AVM requires timing the passage to the nidus of a bolus of radiographic contrast material filmed with rapid (three to six frames per second) digital subtraction angiography. The injection of glue is therefore carefully monitored and immediately discontinued as soon as it is seen reaching the venous drainage of the malformation. However, the in vivo polymerization time for glue is variable (17), and, despite the above precautions, glue may escape into the venous circulation with potential consequences of intracranial hemorrhage.

Several mechanisms, therefore, might be implicated in the production of subdural hemorrhage in our patient. The concomitant presence of an intracerebral hematoma, as initially suspected from the immediate postembolization angiogram and later seen on the computed tomogram, raises the strong possibility of the large intraparenchymal hemorrhage tearing through cortex and arachnoid membrane and into the subdural space, as suggested for spontaneous AVM hemorrhage. Another possible explanation, given the resultant obstruction to the venous outlet of the AVM, is rupture of the occluded draining vein as it traverses the subdural space. It is known that sudden increases in venous pressure can lead to an augmentation of tension, especially at the subdural portion of bridging veins, which can result in subdural bleeding. This can occur in physiologic (coughing and defecation) and pathologic (external cardiac massage) circumstances in the presence of histologically normal veins (18). It would not be surprising, therefore, if similar mechanisms come into play after abrupt obstruction with glue in the abnormal draining veins of an AVM. However, pathologic examination of surgical or postmortem specimens in other patients would be necessary to confirm this hypothesis.

In conclusion, subdural hemorrhage may rarely manifest as a hemorrhagic complication during embolotherapy of cerebral AVMs. In our patient, significant obstruction of the AVM venous outlet with embolic material was the most likely contributor to the development of this complication. We have speculated on several mechanisms by which blood can subsequently reach the subdural space, including rupture of the occluded AVM draining vein, which also bridges the subdural space, or rupture of a concomitant intracerebral hematoma into the subdural space. Regardless of the pathogenesis, the occurrence of subdural hemorrhage (especially in combination with an intracerebral hematoma) should be regarded as a surgical emergency. The occurrence, albeit rare, of such hemorrhagic complications in AVM embolotherapy further attests to the need for a multidisciplinary approach in the management of these lesions. In particular, awareness by neurosurgical and neuroanesthetic teams of the timing of embolization procedures in neuroangiographic suites should be ensured, lest a complication arises. Such precautionary measures should assist in successful treatment of a patient after the rare occurrence of iatrogenic subdural hemorrhage.

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