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Pseudoaneurysms within ruptured intracranial arteriovenous malformations: diagnosis and early endovascular management.

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Pseudoaneurysms within Ruptured Intracranial Arteriovenous Malformations: Diagnosis and Early Endovascular Management

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PURPOSE: To draw attention to pseudoaneurysms within ruptured arteriovenous malformations and to consider their diagnostic and therapeutic features, including pitfalls and precautions needed for safe embolization. **METHODS:** Radiologic and clinical charts of 189 patients who bled from intracranial arteriovenous malformations were retrospectively reviewed. **RESULTS:** Fifteen of the 189 (8%) were found to have pseudoaneurysms. Nine of the pseudoaneurysms were arterial, six were venous. In the early period following hemorrhage, nine patients were treated conservatively. The other six were treated with surgery (one case) or embolization (five cases) because urgent intervention was required. The clinical outcome for both conservative and interventional groups was generally favorable, but one patient in the conservative group died of a rebleed. In the patients who underwent embolization, the fragile nature of the pseudoaneurysm made it necessary to first embolize the artery feeding it. Embolization with particles was considered hazardous. Instead, free-flow (nonwedged) *N*-butyl-cyanoacrylate embolization proved safe and effective in treating both the pseudoaneurysms and arteriovenous malformations in these cases. **CONCLUSIONS:** This study highlights the importance of recognizing pseudoaneurysms in such patients and the importance of using free-flow liquid adhesive material on the artery feeding the pseudoaneurysm if embolization is required.

Index terms: Aneurysms, cerebral; Arteriovenous malformations, cerebral; Cerebral hemorrhage; Aneurysm embolization; Interventional neuroradiology, complications of

Approximately 42% to 50% of patients with cerebral arteriovenous malformations (AVMs) present with intracranial bleeding (1, 2). Previous reports have commented on the angioarchitecture of AVMs and the relation to their hemorrhagic episodes (3–6). Detailed analysis has also focused on identification of areas of fragility within the AVM and the host in an effort to predict those patients who are at risk of bleeding (7, 8). Angiography, when performed in the acute phase of a hemorrhagic episode, may show a pseudoaneurysm within the ruptured AVM. Despite little attention in the literature, a pseudoaneurysm when demonstrated is a remarkable feature of the AVM because it suggests the exact site of rupture and bleeding. In addition, because pseudoaneurysms lack true walls, embolization may be hazardous and lead to intraprocedural rupture and cerebral hemorrhage (9). In this paper, we will draw attention to pseudoaneurysms within ruptured AVMs and consider their diagnostic and therapeutic features, including pitfalls and precautions needed for safe embolization.

Material and Methods

The radiologic and clinical charts of 189 patients who bled from intracranial AVMs were retrospectively reviewed. Angiography showed a pseudoaneurysm within the AVM in 15 patients (8%). Each patient's age, gender, type of hemorrhage, type of vascular malformation, location of AVM and pseudoaneurysm, and early and subsequent treatment are summarized in Table 1. Embolization within 72 hours of cerebral hemorrhage was performed in five of the 15 patients. Table 2 summarizes the symptoms associated with the hemorrhage, territory of embolization, im-

TABLE 1: Series of 15 patients with angiographically demonstrated pseudoaneurysms within a ruptured AVM

Case	Age	Sex	Hemorrhage	Vascular Malformation	Location	Pseudo-aneurysm	Initial Treatment (within 72 hours)	Subsequent Treatment of AVM
1	6 mo	M	ICH	AVF	Paracentral	Arterial	Embolization	Further embolization
2	43 yr	F	IVH	AVM	Parasplenial	Venous	Embolization	Further embolization + radiotherapy
3	30 yr	F	IVH	AVM	Lateral ventricle	Venous	Embolization	
4	23 yr	M	ICH	AVM	Temporal	Arterial	Embolization	Radiotherapy
5	28 yr	M	SAH	AVM	Paracentral	Venous	Embolization	Further embolization
6	4 yr	M	IVH	AVM	Thalamus	Arterial	Medical	Therapeutic abstention
7	17 yr	M	ICH-IVH	AVM	Caudate nucleus	Arterial	Medical	Embolization + surgery
8	30 yr	F	IVH	AVM	Basal ganglia	Venous	Medical	Early rebleeding (died)
9	43 yr	F	ICH-IVH	AVM	Precentral gyrus	Arterial	Medical	Embolization
10	18 yr	F	ICH	AVM	Basal ganglia	Venous	Medical	Embolization
11	29 yr	F	ICH	AVM	Precuneus	Venous	Medical	Embolization + surgery
12	6 mo	F	ICH	Multiple AVMs	Temporal occipital	Venous	Medical	Therapeutic abstention
13	16 yr	M	IVH-ICH	AVM	Thalamus	Arterial	Medical	Radiotherapy
14	16 yr	F	ICH	AVM	Callosal	Arterial	Surgery	
15	34 yr	F	IVH	AVM	Insular	Arterial	Medical	Embolization

Note. M = male; F = female; ICH = intracerebral hemorrhage; IVH = intraventricular hemorrhage; SAH = subarachnoid hemorrhage; AVF = arteriovenous fistula; AVM = arteriovenous malformation.

TABLE 2: Endovascular management of our series of five patients with pseudoaneurysms within AVMs

Case	Symptoms	Vessel Targeted for Embolization[a]	Anatomic Result		Complications	Short-Term Outcome	Complementary Treatment	Long-Term Outcome	Follow-Up
			Pseudo-aneurysm	AVM					
1	Hemiparesis Seizures	Rolandic artery	Suboc-clusion	Unchanged	Transient worsening of hemiparesis	Recovery (asymptomatic)	Further embolization	Asymptomatic	11 months
2	Headaches Confusion	Posterior callosal artery	Occlusion	Reduction of nidus		Recovery (asymptomatic)	Further embolization[b] + radiotherapy	Quadranopsia	8 months
3	Somnolence Venous thrombosis of upper limb (requiring heparinization)	Posterior choroidal artery	Occlusion	Cure		Recovery (asymptomatic)		Asymptomatic	7 months
4	Somnolence Hemiparesis Hemianopsia	Posterior temporal artery	Occlusion	Reduction of nidus		Clinical improvement Residual quadranopsia	Radiotherapy	Quadranopsia	8 months
5	Hemiparesis Hemianesthesia	Internal parietal artery	Occlusion	Reduction of nidus		Recovery (asymptomatic)	Further embolization	Asymptomatic	10 months

[a] Arterial feeder of the compartment of the AVM harboring the pseudoaneurysm.
[b] After a second session of embolization, the patient developed a quadranopsia.
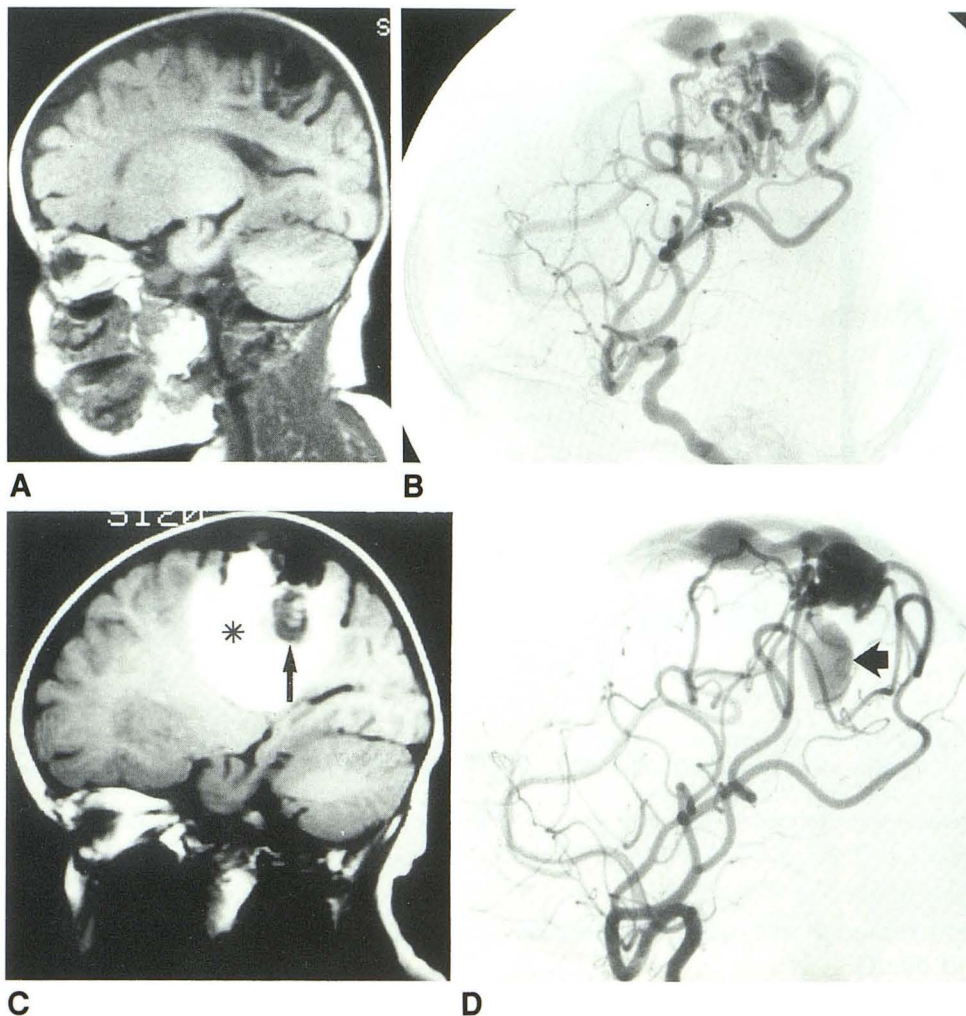
Fig. 1. Case 1.

A, Sagittal T1-weighted MR.

B, Initial internal carotid angiogram (lateral view) shows a complex arteriovenous fistula in the rolandic sulcus.

C, Sagittal T1-weighted MR done after the onset of sudden hemiparesis 1 month later shows a hematoma (*asterisk*) surrounding a pseudoaneurysm (*arrow*) at the margin of the malformation.

D, Subsequent carotid angiogram (*lateral view*) clearly shows the pseudoaneurysm (*arrow*) as a new feature of the vascular malformation.

mediate anatomic result (of both pseudoaneurysm and AVM), complications, short-term outcome, complementary treatment, long-term outcome, and follow-up of this latter group. Urgent embolization was done in these patients because of progressive clinical deterioration (cases 1 and 4), pressing need for systemic heparinization to treat an upper limb venous thrombosis (case 3), and fear of early hemorrhagic recurrence from the demonstrated bleeding point (cases 2 and 5).

Embolization was done by the transarterial route in all patients after a percutaneous femoral puncture. The procedures were done under neuroleptic analgesia or general anaesthesia, depending on the age and clinical status of the patients. Selective catheterization of the arterial pedicle supplying the compartment of the AVM harboring the pseudoaneurysm was attempted first in every case. Either a minitorquer (Nycomed-Ingenor, Paris, France) or a Tracker (Target Therapeutics, San Jose, CA) catheter with a coaxial 5-F system was used for this purpose. During catheter progression into the arterial feeder, injection of contrast material was kept to a minimum. After catheteri-

zation of the arterial pedicle at the selected point for embolization, occlusion of the nidus and the false aneurysm or its arterial feeder were done with N-butyl-cyanoacrylate (NBCA) (Bruneau, Boulogne, France) mixed with Tantalum powder (Byodine, El Cajon, CA) and Lipiodol (Guerbet, Villepinte, France).

Results

We found nine arterial and six venous pseudoaneurysms within AVMs in our series. There was no sex predominance. Pseudoaneurysms were found in both cortical (eight cases) and deep (seven cases) AVMs. There were no pseudoaneurysms located in the brain stem. Six of nine patients who were not treated immediately had pseudoaneurysms that thrombosed spontaneously. Two other patients of the same group had pseudoaneurysms that progressively annexed to the draining vein, creating a venous ectasia. All of these eight patients had a favorable
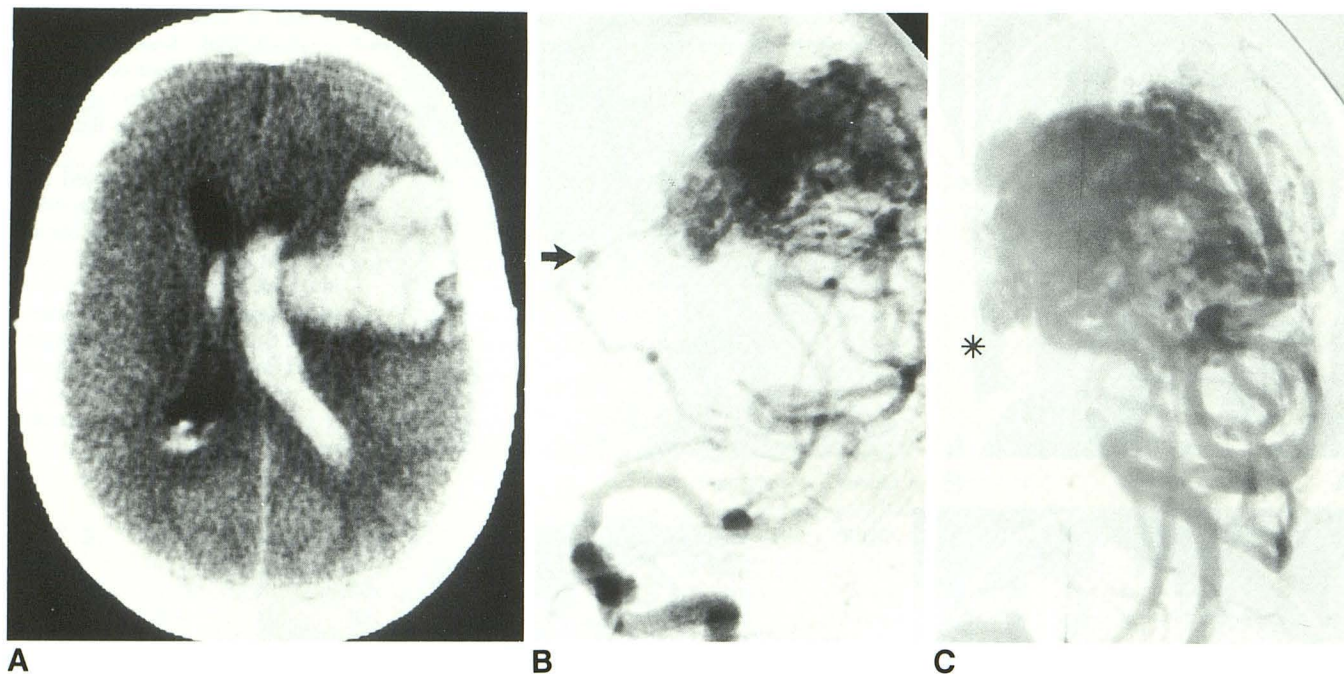
A   **B**   **C**

Fig. 2. Case 9.

A, CT scan shows intracerebral and intraventricular hemorrhage.

B, Internal carotid angiography (anteroposterior view) performed within 24 hours shows an AVM in the left precentral gyrus. An arterial pseudoaneurysm (*arrow*) is visualized on a lenticulostriate artery.

C, Follow-up carotid angiography (*lateral view*) performed 6 weeks later shows spontaneous thrombosis of the pseudoaneurysm and the distal portion of the lenticulostriate artery (*asterisk*). The AVM remains unchanged.

outcome. The remaining patient rebled 1 month after the initial hemorrhage and died.

Immediate treatment was instituted in six patients. One patient was treated by open surgery and cured; the remaining five were treated by endovascular approach. In these five patients, the presumed source of bleeding (indicated by the site of the pseudoaneurysm) was successfully embolized. There were no technical complications, although one patient had a transient worsening of his preexisting hemiparesis (case 1). He recovered fully in a few weeks. All other patients experienced clinical improvement and better response to medical treatment after embolization. In addition, endovascular treatment allowed safe systemic heparinization in case 3, with uneventful recovery from the venous thrombosis. All patients needed additional treatment except case 3, whose AVM was cured in one session. Further embolizations (cases 1, 2, and 5) and gamma knife radiotherapy (cases 2 and 4) were done. Case 2 had a quadranopsia after a second session of embolization. Complementary treatment for the other cases did not result in any complications.

Discussion

Bleeding represents the most devastating complication of intracerebral AVMs. After AVM rupture, the subsequent extravascular hemorrhage progressively clots, creating a hematoma. A pseudoaneurysm results from the unclotted portion of the hematoma still communicating with the vessel lumen. Thus, pseudoaneurysms may be visualized during angiography in patients with recent cerebral bleeding. Pseudoaneurysms can be arterial or venous depending on the site of the ruptured vessel. Arterial pseudoaneurysms are proximal to the nidus, whereas venous pseudoaneurysms are located in the nidus or distally (7, 8). Whether arterial or venous, a pseudoaneurysm can be recognized with angiography or magnetic resonance (MR) as a vascular cavity, usually of irregular shape, within or at the margin of the hematoma. Comparison with previous vascular examinations (angiography or MR), if available, confirms the pseudoaneurysm as a new angioarchitectural feature of the AVM (Fig. 1). This acquired nature secondary to a hemorrhagic episode is pathognomonic.
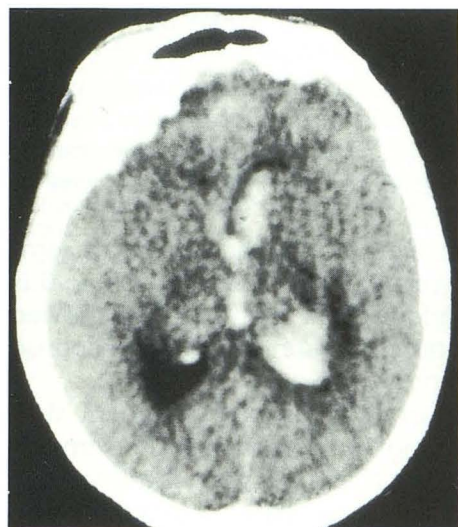
Fig. 3. Case 3.

A, CT scan shows an intraventricular and subependymal hemorrhage.

B, Vertebral angiography (*lateral view*) shows an AVM and a pseudoaneurysm (*arrow*) in the lateral ventricle.

C, Selective injection of the posterolateral choroidal artery (*thin arrow*) before embolization clearly shows the AVM and the pseudoaneurysm (*large arrow*), which has slightly increased in size during injection of contrast material. Venous drainage is through the lateral atrial vein towards the vein of Galen (*double arrows*).

D, Follow-up vertebral angiography (*lateral view*) 6 months after embolization confirms the anatomic cure.
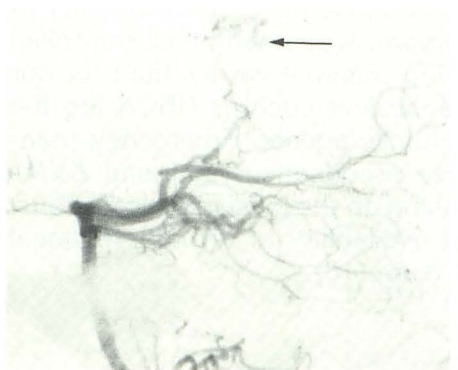
The natural history of a pseudoaneurysm and a ruptured AVM is unpredictable. However in six of eight initially untreated patients who had a favorable outcome after hemorrhage, the pseudoaneurysms showed progressive decrease in size with clotting and occlusion of the ruptured vessel in a few days (Fig. 2). In the remaining two patients, annexation of the pseudoaneurysm to the venous outlet of the AVM created a venous ectasia.

These findings correlate well with the natural history of ruptured AVMs in that the incidence of early rebleeding is not high (1, 2,10). This feature of ruptured AVMs supports the current theory that urgent treatment after bleeding from an AVM is unnecessary, unlike the treatment of subarachnoid arterial aneurysms. Although this concept is empirically true, it is founded only on cases that survive the hemorrhage. It is not possible to conclude that the natural history of a pseudoaneurysm is always unfavorable because many patients with cerebral AVMs die after bleeding,

even before angiography can be done. Evolution of pseudoaneurysms in patients with progressive neurologic deterioration after bleeding is also unknown because they are often not studied by serial angiography. It seems thus, that a "new" subgroup of patients at higher risk for rebleed (11% in this small series in comparison with the 1% to 3% noted in the overall series by Crawford (1)) can be identified; this population with pseudoaneurysms should therefore warrant earlier treatment.

When medical management of cerebral hemorrhage becomes difficult, delayed therapy of an AVM is not advisable and definitive treatment of the hematoma and/or the AVM has to be instituted early (10). Patients presenting with progressive neurologic deterioration (cases 1 and 4) or requiring formal anticoagulation therapy because of an underlying disease (case 3) are good examples of the need for this obligatory treatment. Therapeutic options are surgery (10, 11) and embolization (8, 12, 13). If the latter therapy is

indicated, it carries particular challenges among which the recognition of a pseudoaneurysm is of utmost importance. Significant increase in flow or pressure during embolization may cause intraprocedural rupture. Because pseudoaneurysms do not have vascular walls, previous authors have warned of the risks involved in embolization of pseudoaneurysms outside the central nervous system (14, 15). In the neuroradiologic literature, little attention has been given to this problem, although rupture of a pseudoaneurysm and intraventricular hemorrhage during AVM embolization have recently been reported (9).

The presence of a pseudoaneurysm, however, does not contraindicate endovascular therapy, but special precautions should be taken for safe embolization. No attempt to embolize an arterial feeder other than the one feeding the pseudoaneurysm should be carried out during the procedure even if the nonfeeding artery looks more accessible. This is because minimal changes in the hemodynamic situation of the AVM may precipitate a breakdown of the fragile demarcation between the hematoma and the patent lumen (16, 17).

In the technical setting, careful catheter manipulation and correct choice of embolic material are mandatory. During the actual endovascular approach to the lesion, catheter progression into the desired vessel (the one filling the false aneurysm) should be done with minimal injection of contrast material (Fig. 3). Overinjection of fluid may exert a significant strain on the pseudoaneurysm, increasing the risk of intraprocedural rupture. A wedged catheter represents an additional risk factor because the injecting force is transmitted entirely to the vessel and the pseudoaneurysm (15). For these reasons, flow control or balloon embolization in case of AVMs presenting with false aneurysms are felt to be extremely hazardous and should be contraindicated.

Proper choice of embolic material is another key factor for safe embolization. Pseudoaneurysm rupture and bleeding during embolization with polyvinyl alcohol particles in the central nervous system (9) and other regions of the body (15) have been reported. Particle embolization requires a substantial volume of fluid introduced at a considerable pressure to facilitate particle passage through the catheter. The pressure of each injection may exceed the compliance of the pseudoaneurysm. Thus, embolization with polyvinylalcohol particles should be discouraged in such a situation. Other embolic agents that do not require any carrier fluid would be more appropriate. NBCA proved to be efficient in embolizing both the AVMs and the pseudoaneurysms, in our hands. Embolization with microcoils can be an alternative (C.F. Dowd, personal communication) but should be regarded as a form of proximal ligature.

Following the aforementioned principles, embolization of the pseudoaneurysm was safe in this short series. In addition, all such patients showed rapid favorable outcome and improved response to medical therapy.

The real value of endovascular treatment after immediate bleeding of cerebral AVM remains uncertain. However, if embolization is considered in the treatment of a recently ruptured AVM, searching for a pseudoaneurysm within the vascular lesion is crucial. Because of the fragility of the pseudoaneurysm, we recommend controlled embolization using minimal carrier fluid. In our opinion, liquid adhesives such as NBCA are the most suitable embolic agents. Emergency management of these pseudoaneurysms and AVMs may vary depending on the local technical habits, experience, and availability of an interventional neuroradiologic team (18).

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