Endovascular Treatment of Intracranial Ruptured Aneurysms Associated with Arteriovenous Malformations: a Clinical Analysis of 14 Hemorrhagic Cases

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Key words: aneurysm, arteriovenous malformation, endovascular treatment

Summary

This study investigated and summarized endovascular therapeutic strategies for intracranial ruptured aneurysms associated with arteriovenous malformations (AVMs). Between June 2005 and June 2009, we identified 16 aneurysms in 14 hemorrhagic cases of intracranial AVM using digital subtraction angiography (DSA). Of the 16 aneurysms, 14 were ruptured and two were unruptured. Aneurysms were classified as types I to IV, and were treated. Aneurysm treatment was followed by AVM treatment via various therapies, including embolization, gamma knife radiotherapy, or follow-up and observation to reduce the risk of aneurysm rupture or intracranial hemorrhage. Over a follow-up period ranging from six months to one year, none of the patients had aneurysm ruptures or intracranial hemorrhage. Most (13/14) patients had a Glasgow Outcome Scale (GOS) score of 5, and one patient had a score of 4. Sixteen aneurysms were treated successfully, as confirmed by DSA examination, and no AVMs re-grew. Clinical therapeutic strategies for intracranial ruptured aneurysms associated with AVMs should include aneurysm treatment first to reduce the risk of rupture and intracranial hemorrhage, eventually leading to a better prognosis.

Introduction

In rare cases, intracranial aneurysms can be found with coexisting arteriovenous malformations (AVMs). These two conditions are difficult to treat simultaneously due to their complicated anatomical relationships and hemodynamics [1,2]. But when aneurysms coexisting with AVMs are ruptured, they become risk factors and should be treated prior to AVMs [2,3]. At present, therapeutic methods for intracranial ruptured aneurysms associated with AVMs have included craniotomy and endovascular treatment [4,5].

However, the relative locations of these aneurysms associated with intracranial AVMs are often obscure, making it difficult to search for and expose them during craniotomy. Moreover, brain tissue and blood vessels also suffer severe damage. In addition, not all aneurysms are saccular, so even if the field of operation is fully exposed, clipping difficulties may exist. Furthermore, aneurysms and AVMs are not always present in the same operative field, making it impossible to treat these two disorders simultaneously when necessary [3-5]. The use of digital subtraction angiography (DSA)-assisted endovascular intervention has obvious advantages. For instance, no direct damage to nervous tissue is observed with DSA. In addition, DSA-assisted endovascular intervention allows the clinician to locate aneurysms and AVMs via the relevant intracranial vasculature, allowing for the simultaneous treatment of both conditions [5,6]. But little is known regarding the efficacy of endovascular treatment for intracranial ruptured aneurysms coexisting with AVMs. Therefore, in this study we reviewed and summarized the therapeutic strategies for 14 recent hemorrhagic cases of intracranial ruptured aneurysms in coexisting AVMs.

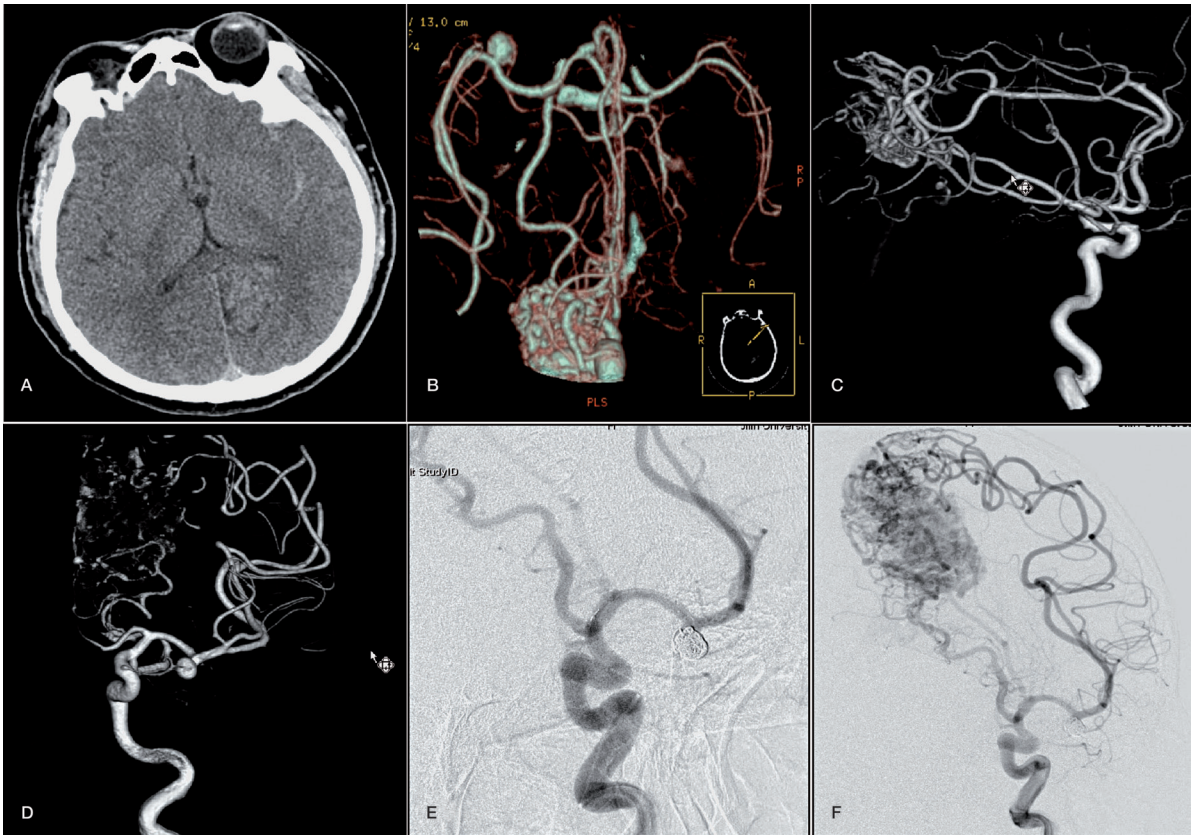

Figure 1 Intracranial type I aneurysm with coexisting AVM (case 1). A) CT showed subarachnoid hemorrhage (SAH) in the left sylvian fissure. B) CTA revealed an aneurysm of left middle cerebral artery bifurcation and an AVM in left parietal and occipital lobe. C) 3D-DSA images revealed that the bilateral anterior cerebral artery and left posterior communicating artery and left cerebral posterior artery supplied the AVM. D) 3D-DSA images revealed the aneurysm was at the left middle cerebral artery bifurcation and the middle cerebral artery was irrelevant to the AVM. E: DSA showed the aneurysm was treated with coils, and the AVM was untreated. F) The follow-up DSA showed no recanalization of the aneurysm. No changes were found in the AVM.

Materials and Methods

Clinical Information

Fourteen patients (10 males, 4 females) with ruptured aneurysms associated with intracranial AVMs presented from June 2005 to June 2009 in the department of neurosurgery of the First Hospital of Jilin University. They were between 21- and 57-year-old (median age, 42.7 year-old).

All patients presented with bleeding, including four with subarachnoid hemorrhage (SAH), five with intracerebral hemorrhage (IH), three with SAH with coexisting IH, and two with SAH with coexisting intraventricular hemorrhage (IVH). The diagnosis of ruptured aneurysm associated with intracranial AVM was confirmed by DSA. The patients' Glasgow Coma Scale (GCS) scores ranged from 13 to 15.

Imaging Data

There were 16 aneurysms in the 14 AVM cases studied, with diameters ranging from 1.2 to 8.6 mm (average 3.0 mm). A single aneurysm was found in 12 cases, while multiple aneurysms were found in two cases. Four cases were classified as pseudoaneurysms, four were classified as saccular aneurysms, and eight were classified as dissections.

Of the 14 AVM cases, three were supratentorial and 11 were infratentorial. AVM diameters ranged from 0.8 to 6.2 cm (mean 3.2 cm), of which seven cases were < 3 cm, six were from 3-6 cm, and one was > 6 cm.

The feeding artery consisted of a single branch in seven cases and multiple branches in seven cases. Using the Spetzler-Martin grading system, 12 cases were grade II and two were grade III.

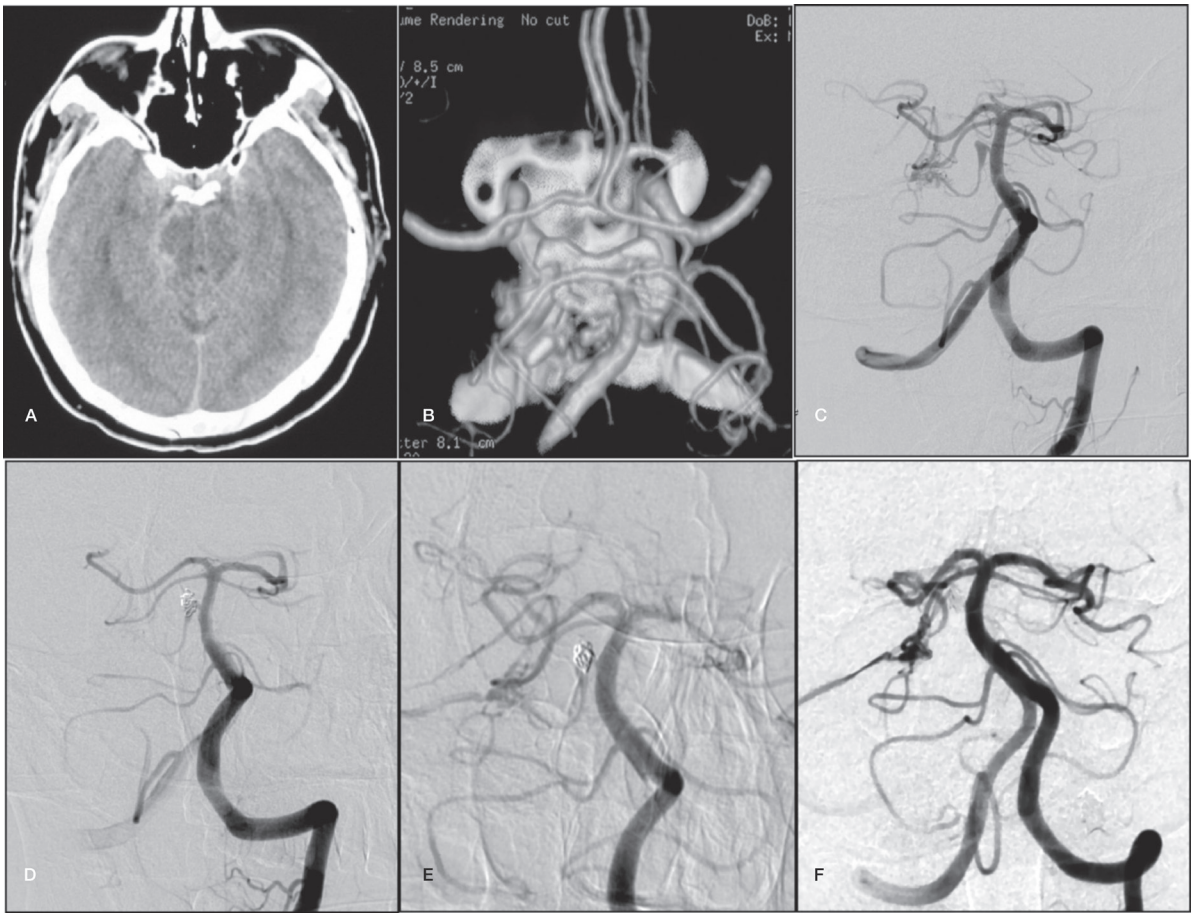

Figure 2 Intracranial type III aneurysm with coexisting AVM (case 4). A) CT showing SAH mainly in the right ambient cistern. B,C) CTA and DSA showing an aneurysm at the origin of the right pontine branch of the basilar artery. D) DSA showing the embolized aneurysm and that the patient's parent artery was preserved. E) Follow-up DSA showing the preserved pontine branch of the basilar artery and AVM. F) DSA showing AVM with Onyx embolization and complete occlusion of the parent artery with coils in the 2nd stage.

Diagnosis and Classification

Identification of the origin of hemorrhage proceeded as follows: (1) if SAH was present, the ruptured aneurysm was considered first, and the aneurysm locations were considered to be in the hemorrhage; and (2) with respect to intracerebral hematoma, the relationship between the location, shapes of the aneurysm, and the site of hemorrhage were considered. Aneurysms, and especially pseudoaneurysms, are often located in the hematoma [7,8].

Aneurysms combined with AVMs were classified into four types: Type I, or unrelated aneurysm type, in which the aneurysm is not associated with the AVM; Type II, or flow-related aneurysm type, in which the aneurysm is saccular and originates from the beginning of feeding arteries of the AVM; Type III, or dissecting aneurysm type, in which the aneurysm is on the feeding arteries of the AVM; and Type IV, or intranidal aneurysm type, in which the aneurysm is nested within the AVM.

Treatment of Aneurysm and AVM

Aneurysm treatment consisted of different methods depending on the classification. For type I aneurysms, the treatment was the same as intracranial aneurysms without associated AVMs. For type II aneurysms, a coil was used to embolize (if necessary, assisted by stent or balloon system) as extensively as possible while sparing the parent artery. For type III aneurysms, a coil or liquid embolic material (Onyx or Glubran glue) was used to occlude the aneurysm as well as the parent arteries. For type IV aneurysms, the area including the aneurysm

Table 1 **Clinical and morphological characteristics of 14 hemorrhagic cases with ruptured aneurysms associated with intracranial arteriovenous malformations.**

No.	Gender/ age	Onset form	GCS	Aneurysm				AVM				Treatment	Out-come GOS
				Type	Position	Character	Size	Position	Size	SM Grade	Feeding artery		
1	Male/ 48	SAH	15	I	At the bifurca-tion of the L MCA	Saccular (ruptured)	8.6 mm	1. parterial/oc-cipital lobe	6.2 cm	II	L PcomA-PCA/ACA	Aneurysm: CO AVM: follow-up	5
2	Male/ 57	SAH+IH	14	II	At the start of the AICA	Saccular (ruptured)	2.1 mm	R cerebellar hemisphere/ brain stem	2.3 cm	III	Multi-Branch, Main: Pica, Minor: AICA/ meningeal pituitaryb-Branch	Aneurysm: CO AVM: GKR	5
3	Female/ 40	SAH	13	II	At the start of the PICA	Saccular (ruptured)	2.3 mm	L cerebellar hemisphere	2.8 cm	II	Single: PICA	Aneurysm: CO AVM: OO in period 2	5
4	Male/ 44	SAH	13	III	On the pontine branch near the start	Dissection (ruptured)	4.0 mm	R cerebellar hemisphere	1.8 cm	II	Multi-branch, main: pontine branch; minor: SCA	Aneurysm and parent artery: CO - AVM: OO	5
5	Male/ 51	SAH+IH	13	III	On the SCA close to AVM	Dissection (ruptured)	4.5 mm	L cerebellar hemisphere	3.5 cm	II	Single: SCA	Aneurysm and parent artery CO - AVM: GKR	5
6	Male/ 39	SAH+IH	14	III	On the PICA near AVM	Dissection (ruptured)	1.2 mm	L cerebellar hemisphere	2.5 cm	II	Single: PICA	Aneurysm and parent artery CO - AVM: GKR	5
7*	Male/ 26	IH	15	III	On the MCA perforating branch	Dissection (ruptured)	2.3 mm	R temporal/ parietal lobe	4.5 cm	II	Multi-branch. main: ACA / MCA; minor: MCA perforating branches	Aneurysm and parent artery GO - AVM: GKR	4
8	Female/ 45	IH	15	III	On the MCA perforating branch	Dissection (ruptured)	1.5 mm	L frontal lobe	3.6 cm	III	Multi-branch. main: ACA / MCA; minor: MCA perforating branches	Aneurysm and parent artery GKR AVM: OO	5
9	Male/ 54	SAH+IVH	14	III×2	On the SCA near the start+ SCA close to AVM	Dissections (ruptured 1, unruptured 1)	4.0 mm; 2.5 mm	R cerebellar hemisphere	2.0 cm	II	Multi-branch. main: R of SCA; minor: Lof SCA	Aneurysm and parent artery. CO 1, GO 1 respectively AVM: GO	5
10	Female/ 52	SAH +IVH	13	IV	In the nidus	Pseudoaneurysm	1.5 mm	Cerebellar vermis	0.8 cm	II	Single: PICA	CO of one-side vertebral artery. An-eurysm/AVM: GKR	5
11	Male/ 21	IH	14	IV	In the nidus	Pseudoaneurysm	2.5 mm	Cerebellar vermis	4.3 cm	II	Single: PICA	OO of feeding artery and AVM	5
12	Male/ 38	IH	14	IV	In the nidus	Pseudoaneurysm	2.7 mm	L cerebellar hemisphere	4.0 cm	II	Single: SCA	GO of feeding artery and AVM	5
13	Female/ 41	IH	14	IV	In the nidus	Pseudoaneurysm	2.5 mm	Cerebellar vermis	4.5 cm	II	Single: AICA	GO of feeding artery and AVM	5
14	Male/ 42	SAH	13	II&IV	At the start of the PICA +in the nidus	Saccular (ruptured) + Dissection (unruptured)	3.0 mm; 1.5 mm	R cerebellar hemisphere	2.0 cm	II	Multi-branch, main: PICA, Minor: AICA	Saccular aneurysm: CO; Dissection: OO of feeding artery and AVM respectively	5

R: right, L: left, SAH: subarachnoid hemorrhage, IH: intracerebral hemorrhage, IVH: intraventricular hemorrhage, GCS: Glasgow Coma Scale, MCA: middle cerebral artery, AICA: anterior inferior cerebral artery, PICA: posterior inferior cerebral artery, SCA: superior cerebral artery, PcomA: posterior communicating artery, PCA: posterior cerebral artery. CO: coil occlusion; OO: Onyx occlusion; GO: Glubran occlusion; GKR: gamma knife radiotherapy.

**For case no. 7: an occlusion of the middle cerebral artery occurred in the type III aneurysms at the end of embolization. The artery recanalized after immediate papaverine (60 mg) and urokinase (200 000 U) treatment. However, left limb hemiparesis remained after surgery. Therefore, the GOS was 4 points.*

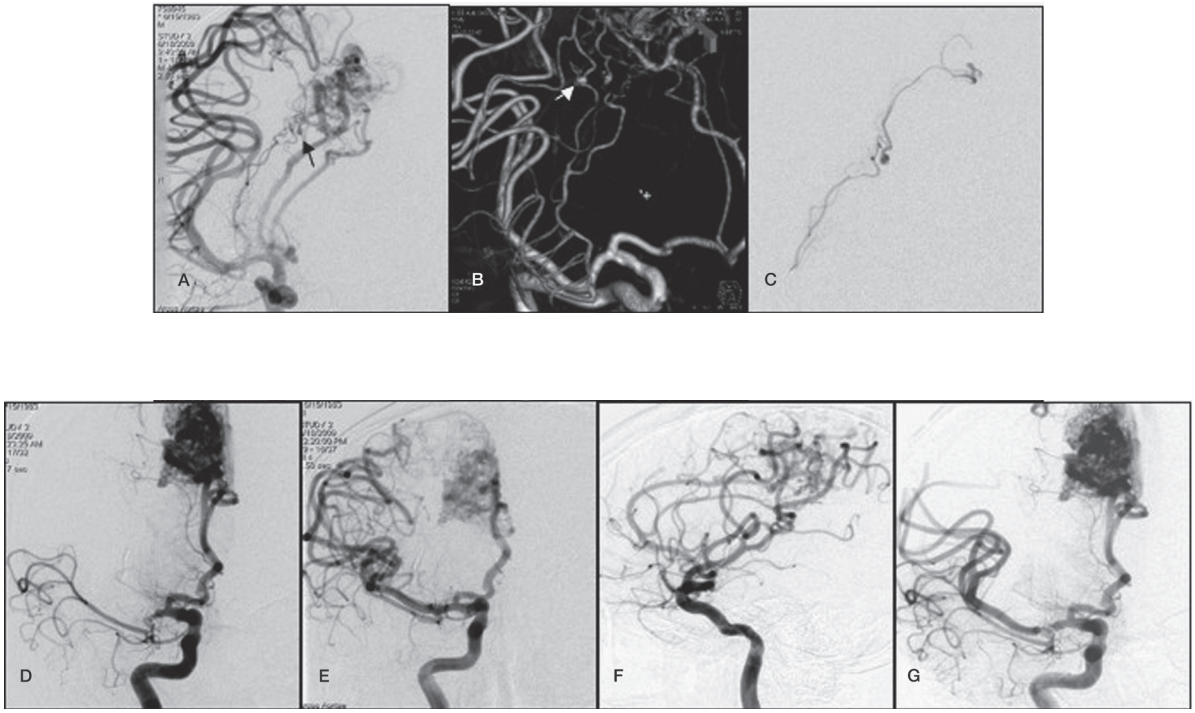

Figure 3 Intracranial type III aneurysm with coexisting AVM (case 7). A,B) DSA and 3D-DSA examinations before operative embolization showed aneurysms derived from the middle cerebral artery perforator coexisting with the AVM. C) Microcatheter angiography after entering the perforating branches. D) Onyx glue embolization of the parent arteries and aneurysms. The vasospasm on the upper stem of the cerebral middle artery was also shown. E) Vasospasm was relieved, as shown in the upper stem after treating with urokinase and nimodipine. Gamma knife was used to treat AVMs. F,G) The follow-up DSA indicated that there was no aneurysm, and the AVM did not re-grow.

and AVM were embolized using Onyx or Glubran glue. Taking the intraoperative hemodynamics and the degree of difficulty for embolization into account, AVM treatment proceeded as follows: (1) Onyx or Glubran glue was used to embolize in period I or II; (2) gamma knife radiotherapy was used; and (3) if the AVM was large, with stable hemodynamics, follow-up and observation were carried out.

Results

Treatment of Aneurysms and AVMs

Aneurysm management: Of the 16 aneurysms identified, 14 were ruptured and two were unruptured, for a total of 14 ruptured aneurysms distributed in 14 hemorrhagic cases. All aneurysms were treated. One type I aneurysm and three type II aneurysms were treated with coil embolization. Of the seven type III aneurysms, four were coil embolized with their parent arteries, and two were embolized with their parent arteries using Glubran glue. Gamma knife radiotherapy was used for one patient with failure of interventional treatment. Of the five type IV aneurysms, in two cases both the aneurysm and the AVM area were occluded via Onyx, while in two cases occlusion was achieved using Glubran glue. One patient was treated with coil occlusion on one side of the vertebral artery, and gamma knife radiotherapy was used to treat the aneurysm and AVM.

AVM management: Following aneurysm treatment, AVM treatment was performed. Of the 14 AVM cases, four were embolized using Onyx during period I, while one was embolized during period II. Three cases were embolized using Glubran glue, while five cases were treated with gamma knife radiotherapy. In one case, the hemodynamic state of the AVM appeared stable, with treatment consisting of follow-up only.

Intraoperative complications: In one type III

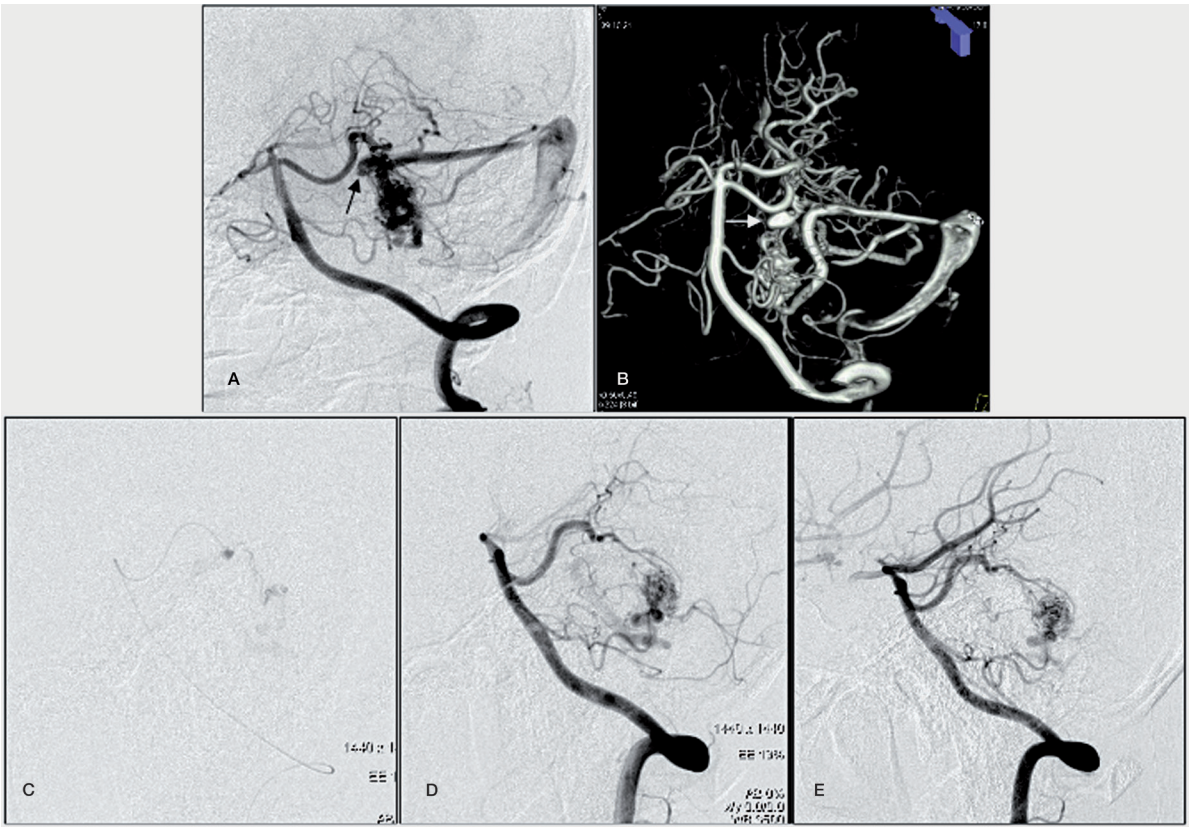

Figure 4 Intracranial coexisting type IV aneurysm with AVM (case 12). A,B) DSA and 3D-DSA examination before operative embolization showed the left superior cerebellar artery feeding the AVM, which coexisted with the aneurysm (arrow). C) Microcatheter into the AVM from the superior cerebellar artery aneurysm. D) Onyx glue embolization of the part of the AVM including the aneurysm. E) The follow-up DSA indicated that the aneurysm disappeared, and the residual AVM did not re-grow.

aneurysm case, an occlusion occurred in the upper stem of the middle cerebral artery after embolization treatment. The patient was treated with papaverine (60 mg) and urokinase (200,000 units) immediately and artery recanalization occurred, however, limb hemiparesis was present.

Clinical Outcome

Imaging results: Patients were re-examined via DSA six months or one year after treatment. Of 16 aneurysms, treatment failed in one case of type III aneurysm that was initially treated endovascularly. This aneurysm was then treated via gamma knife radiotherapy and the aneurysm disappeared. In one case a type IV aneurysm coexisting with an AVM was not obvious in the DSA image after gamma knife treatment. In the other 14 aneurysms, no recanalization or re-growth was found. After treatment, 14 AVMs did not regrow.

Prognosis: Postoperative recovery was satisfactory. Upon follow-up after six months to one year, 13 cases had a GOS score of 5 points. However, in the single case of arterial occlusion that occurred intraoperatively the GOS score was 4 points (Table 1).

Discussion

Intracranial aneurysms associated with AVMs are rarely found clinically. In fact, recent reports indicate that the incidence of aneurysm with coexisting AVM is 2.7%~16.7% [9,10]. Moreover, it is difficult to treat these two conditions simultaneously because of their complicated anatomical relationship and hemodynamics. Moreover, the etiology of aneurysms with coexisting AVMs is still unclear. Hemodynamic factors are likely to play a role in the development of this condition, in addition to congenital anomalies. AVMs are tangled anastomoses of

blood vessels in which arteriovenous shunting occurs in a central nidus. This nidus forms the area toward which multiple feeding arteries converge, and from which enlarged veins drain. Aneurysms associated with these malformations are primarily related to the feeding arteries of the AVMs. In such cases the high capacity and speed of blood flow continually damage the endothelia of the blood vessels, eventually leading to a rupture of the vascular internal elastic membrane, resulting in aneurysms [11-13]. Therefore, the risk of hemorrhage increases in AVMs associated with aneurysms to approximately 7%. In contrast, there is only a 1.7% risk of hemorrhage in AVMs without coexisting aneurysms [9,14]. Furthermore, whether aneurysms result from increased hemodynamics of the AVM (and therefore have a higher chance of rupturing) or an underlying wall weakness is debatable. In addition, not all aneurysms in coexisting AVMs are flow-related, because some aneurysms are within the nidus, some aneurysms have no relationship with AVMs, and some dissections on the feeding arteries are not clearly associated with the blood flow. Therefore the etiology of aneurysms with coexisting AVMs is unclear, and the variety of aneurysm types coexisting with AVMs further complicates the situation. Therefore, it is reasonable to treat these aneurysms according to Perata typing, which is a classification scheme for AVMs associated with aneurysms [8]. According to this scheme: type I, or remote type, is an aneurysm which is not associated with the blood flow of the AVM; in type II, or proximal type, the aneurysm originates from the feeding arteries of the AVM; in type III, or pedicled type, the aneurysm is near the feeding arteries of the AVM; and in type IV, or nest type, the artery feeding the aneurysm is nested within the AVM. However, Perata's classification scheme is currently not commonly used in the international literature and creates confusion, so in this study we simplified it to only "unrelated aneurysm"-type I, "flow-related"-type II (saccular aneurysm on the start of the feeding artery), "feeding artery aneurysm"-type III (dissecting aneurysm on the feeding artery), and "intranidal aneurysm"- type IV (dissecting or pseudoaneurysm within the AVM).

It is not clear whether all aneurysms with coexisting AVMs should be treated. There is evidence that treatment of the intracranial AVM itself can result in spontaneous regression of the aneurysm, with no further treatment necessary [15]. However, at present the consensus is that ruptured aneurysms should be treated [16]. In particular, intranidal aneurysms are known to be associated with hemorrhagic presentation of the AVM and associated with an increased rate of future hemorrhage [17]. In the present study, 14 of the 16 aneurysms were ruptured, suggesting that the aneurysm was an important hazard factor and could lead to disastrous events. Therefore, our therapeutic goal in this study was to reduce hemorrhage risk [12,18]. A large body of evidence has shown that the therapeutic strategy for aneurysms with coexisting AVMs depends on the aneurysm type and the particular clinical characteristics present [16,17,18]. When treating ruptured aneurysms with coexisting AVMs, the common idea is that associated aneurysms are high-risk factors, which should be given priority for treatment [1,9,18]. In this study the aneurysm was treated first, taking into account our typing, followed by therapy for the AVMs. Meanwhile, various stratified treatments, e.g. gamma knife radiotherapy, were adopted to reduce the risk of aneurysm rupture.

In the case of type I aneurysms, treatment should be no different from an aneurysm with no coexisting AVM. In this study there was one type I ruptured aneurysm located a considerable distance from the AVM and unaffected by AVM blood flow. In this case, routine coil embolization was performed. Type II aneurysms originate from the start of the AVM's feeding artery, and are therefore considered to be flow-related aneurysms. The feeding artery within the AVM lashes the vascular wall with high velocity blood flow, leading to saccular aneurysm formation [19]. Of this kind of aneurysm, three were treated by coil. There is no difference in outcome between saccular aneurysms associated with AVMs and those not associated with AVMs. Type III aneurysms occur on the feeding arteries without any branching around the aneurysms, and are considered dissecting aneurysms [20,21,22]. In our study, the aneurysms and parent arteries could be occluded using coils or liquid embolic material because they were near the AVMs with no emerging branches. Among the type III aneurysms, there were two special cases located in the deep perforating branches of the middle cerebral artery. Because their deep perforating branches emerged at a right angle, they possessed high transmural pressure, and were therefore prone to rupture again [23,24]. However, these perforating arteries are too tor-

tuous to import a micro-catheter. Of these two aneurysms, one could not be micro-catheterized, which lead to the abandonment of endovascular treatment in favour of gamma knife radiotherapy. The other one was embolized successfully, but required more time and was repeatedly stimulated by the micro-catheter in operation. This led to the occurrence of an occlusion in the upper stem of the middle cerebral artery. Although papaverine and urokinase were given, sequelae remained. The remaining five aneurysms were treated successfully, with good prognoses. Because type IV aneurysms are usually located in the AVM nest or hematoma, they are considered pseudoaneurysms following hemorrhage. In these cases, liquid embolism material is used to treat the region containing the aneurysms. The process of interventional treatment was successful in the five type IV aneurysms analyzed, and the prognoses were good [19,25]. Two unruptured aneurysms were included in this study. One was located on the feeding artery near the coexisting ruptured aneurysm, and when treating the ruptured one it was embolized as well. The other was in the nidus, and because intranidal aneurysms are known to be associated with an increased rate of future hemorrhage, this unruptured aneurysm was also treated [17].

Our data are consistent with previous reports indicating that, when they coexist, ruptured aneurysms should be treated prior to associated AVMs [12,16]. Treatment of the AVMs is then related to the types of aneuryms found and whether they contain risk factors for hemorrhage [17,18]. For example, for AVMs with type II aneurysms, because the aneurysm is located at the start of the AVM's feeding arteries, AVM treatment is not affected by the presence of aneurysms. Treatment in this case is the same as conventional AVM treatment, and involves occlusion via liquid embolic material. For AVMs with type III aneurysms, the aneurysms are located on the AVM's feeding arteries, which markedly affects AVM treatment. If these aneurysms are embolized using liquid embolic material, AVMs can be embolized at the same time as the aneurysm. However, when we occluded aneurysms using a coil, the parent arteries were occluded together, so AVMs were not re-embolized. In this case gamma knife treatment can be performed. For AVMs accompanied by type IV aneurysms, the aneurysms are usually located in the AVMs. Therefore, these aneurysms can be embolized along with their associated AVMs. Of the 14 AVMs studied, 13 cases were subjected to the above methods and prognoses were favorable. However, in one case the AVMs were relatively large, had blood supplied by many arterial branches, unobstructed drains and no hemorrhaging. For fear that the steady state of the hemodynamics would be disturbed by endovascular intervention, follow-up and observation were carried out in these cases, and the patients' prognoses were good.

Conclusions

In summary, the existence of ruptured aneurysms is a key risk factor for intracranial aneurysms with coexisting AVMs, and endovascular treatment should be actively carried out in these circumstances. In addition, typing can provide effective guidance in the treatment of intracranial aneurysms with coexisting AVMs. For intracranial AVMs, treatment can be performed using different treatment modalities, including embolization, gamma knife radiotherapy and follow-up, depending on the clinical characteristics encountered.

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