Angioarchitecture Related to Hemorrhage in Cerebral Arteriovenous Malformations

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Abstract

A retrospective study was conducted to determine the angioarchitecture related to hemorrhage in patients with cerebral arteriovenous malformations (AVMs), who underwent conservative treatment and long-term follow-up. The average observation period was 9.3 years, and the annual bleeding rate was estimated at 3.6%. In all cases angiographic findings were reviewed in detail. The average AVM grade by Spetzler-Martin was 3.5. Higher bleeding rate was observed in large AVM (5.4%) compared with small (2.1%) or medium AVM (2.9%). Deep venous drainage (8.6%/year) was strongly correlated to hemorrhage. Concerning location of nidus, hemorrhage was frequently found in insular, callosal, and cerebellar AVMs. Venous ectasia, feeder aneurysm, and external carotid supply were commonly demonstrated on angiograms. Comparison of annual bleeding rate revealed that AVMs with intranidal aneurysm (8.5%) and venous stenosis (5.5%) had a high propensity to hemorrhage. Therapeutic strategy should be focused on these potentially hazardous lesions by the use of endovascular embolization or stereotactic radiosurgery, even if surgical resection is not indicated.

Key words: arteriovenous malformation, hemorrhage, angioarchitecture, natural history

Introduction

In management of cerebral arteriovenous malformations (AVMs), the most serious complication is hemorrhage due to rupture of AVM. Recently, the relationship between hemorrhage and angioarchitecture has been analyzed precisely.^{9,11-13,16,20,23)} These studies, however, are not based on a long-term follow-up which would tell us what kind of angiographic characteristics actually tends to cause bleeding from the AVM. Information about the actual source of bleeding would also contribute to developing therapeutic strategies. The aim in this study is to clarify potentially dangerous structures which might initiate bleeding, from analysis of clinical observation and radiological findings.

Materials and Methods

The study was conducted retrospectively in 24 patients with cerebral AVM, who underwent conservative management. In these patients surgical intervention was avoided, because of high therapeutic risk or rejection of treatment by the patients. They were diagnosed with conventional angiograms or

digital subtraction angiograms. Some of the patients underwent superselective angiograms with microcatheters. Clinical course was investigated at the outpatient clinic or by correspondence with the patients. The follow-up period in every patient was 1 year or more. In patients who underwent subsequent therapeutic embolization or radiotherapy, the period after the treatment was excluded from this study.

All AVMs were evaluated with Spetzler-Martin grading system, which consists of size, eloquence, and pattern of venous drainage.¹⁹⁾ Furthermore, characteristics on angiograms in each AVM were listed as the following types: 1) arteriovenous fistula, 2) external carotid supply, 3) angiomatous change, 4) feeder aneurysm, 5) intranidal aneurysm, 6) venous ectasia, and 7) venous stenosis.^{9,23)} In the patients who experienced hemorrhagic episodes during the follow-up period, location of bleeding was retrospectively examined by computed tomography (CT) or magnetic resonance (MR) imaging, if available.

Table 1 Location, size, and drainage and hemorrhage during follow-up

	No. of cases	Times of bleeding	Follow-up period (months)	Bleeding rate per year (%)
Location*				
frontal	11	3	1580	2.3
parietal	9	5	1467	4.1
occipital	3	0	248	0
temporal	2	0	191	0
insular	1	1	97	12.4
callosal	2	1	164	7.3
thalamic	1	0	44	0
cerebellar	4	1	274	4.4
Size				
< 3 cm	8	1	561	2.1
3-6 cm	9	3	1245	2.9
> 6 cm	7	4	887	5.4
Drainage				
superficial	9	2	1119	2.1
deep	5	2	278	8.6
mixed	10	4	1296	3.7
Total	24	8	2693	3.6

*An arteriovenous malformation can be located in two or more regions.

Table 2 Angioarchitecture and hemorrhage during follow-up

Type*	No. of cases	Cases of initial bleeding	Times of bleeding in follow-up	Bleeding rate per year (%)
External carotid supply	4	1	3	4.4
Angiomatous change	3	0	0	0
Arteriovenous fistula	2	0	0	0
Feeder aneurysm	4	2	4	4.9
Intranidal aneurysm	2	1	2	8.5
Venous ectasia	8	2	1	1.1
Venous stenosis	2	1	1	5.5
No specific findings	13	2	2	4.1

*An arteriovenous malformation can be classified into two or more types.

Results

I. Overview of patients

The patients comprised 14 males and 10 females. Age at first onset ranged from 1 to 56 years, an average of 30.3 years. The follow-up period ranged from 12 to 508 months, 112 months (9.3 years) on average. Clinical manifestations at the first onset were hemorrhage in six cases (25%), epilepsy in five (21%), progressive neurological deficits in six (25%), and no specific symptoms in seven (29%). Two patients (8.3%) died of hemorrhage during the follow-up.

II. AVM structure and hemorrhage

The average Spetzler-Martin grade in the whole series was 3.5. With CT scan or MR imaging nine hemorrhagic events during the follow-up period were confirmed in seven cases. The annual bleeding rate in the whole population was estimated to be 3.6% from incidence of hemorrhage (8 times), divided by total follow-up years (224.4 years) (Table 1). The annual bleeding rate in relation to the location, size, and drainage was also calculated. High incidence of hemorrhage was noted in insular (12.4%/yr) and callosal (7.3%/yr) AVMs. Concerning size of nidus, large AVM relatively tended to bleed (5.4%/yr).

An AVM with deep venous drainage frequently bled (8.6%/yr).

III. Angioarchitecture and hemorrhage

Types of angioarchitecture in the series were listed in Table 2. Venous ectasia, feeder aneurysm, and external carotid supply were commonly observed. The relationship between risk of hemorrhage and angioarchitecture was analyzed. AVMs with intranidal aneurysm (8.5%/yr) and venous stenosis (5.5%/yr) frequently bled, compared with AVMs with no specific findings (4.1%/yr). In contrast, no hemorrhagic events were observed in angiomatous change or arteriovenous fistula.

Illustrative Cases

Case 1: In June 1985, a 43-year-old female experienced a headache, and was diagnosed as harboring a left frontoparietal AVM. A feeder aneurysm arising from the left middle cerebral artery and a dilated portion within the nidus, or intranidal aneurysm, were clearly seen on the angiogram (Fig. 1). In February 1996, she suffered an attack of hemiparesis and aphasia, and MR images proved a left frontal hematoma situated outside of the nidus (Fig. 2). The bleeding appeared to have been triggered not by the feeder aneurysm, but by the intranidal aneurysm, because it was adjacent to the hematoma. The intranidal aneurysm had disappeared on the second angiogram (Fig. 3). She underwent endovascular embolization twice, followed by stereotactic radiosurgery. No subsequent hemorrhage has been recognized after the treatment.

Fig. 2 Case 1. Coronal T₁-weighted (left) and axial T₂-weighted (right) magnetic resonance images demonstrating a left frontal hematoma located lateral to the nidus.

Fig. 3 Case 1. Left internal carotid angiograms, frontal (left) and lateral views (right) after the hemorrhage, showing disappearance of the intranidal aneurysm.

Fig. 1 Case 1. Left internal carotid angiograms, frontal (left) and lateral views (right) before hemorrhagic attack, showing an intranidal aneurysm (arrow) and feeder aneurysm arising from the middle cerebral artery (arrowhead).

Case 2: A 46-year-old male had undergone a therapy for epilepsy for 8 years. Carotid angiograms revealed a left frontal AVM, associated with an arterial aneurysm arising from the anterior cerebral artery, and with an intranidal aneurysm which did not directly connect to a drainer (Fig. 4). In February 1991, he experienced a mild right hemiparesis due to hemorrhage. CT scan showed a crescent high density area on the posteromedial surface of the partly calcified nidus (Fig. 5), hence the bleeding seemed to be from the intranidal aneurysm, not from the arterial aneurysm. He has preferred a conservative treatment with antiepileptic drugs.

Fig. 4 Case 2. Left internal carotid angiogram, frontal view, showing an intranidal aneurysm located lateral of the nidus (arrow), and a feeder aneurysm arising from the anterior cerebral artery near the midline (arrowhead).

Fig. 5 Case 2. Computed tomography scan demonstrating a crescent high density area on the posteromedial surface of the partly calcified nidus.

Discussion

In cerebral AVMs, the annual risk of initial hemorrhage is approximately 2% to 3%.³⁾ A mortality is about 10% from the first hemorrhage, and increases to 20% from subsequent hemorrhages.^{15,22)} Prevention of hemorrhage is, therefore, obviously essential in management of AVMs.¹⁻⁴⁾

Recent studies of angioarchitecture have provided information about hazardous structures in AVMs.^{9,10,23)} Aneurysmal vessel dilatations have been commonly stated as a risk factor of hemorrhage in these researches. Turjman *et al.*²⁰⁾ reported that intranidal aneurysm and multiple aneurysms were correlated with hemorrhage by a use of superselective angiography. Perata *et al.*¹⁶⁾ emphasized that pedicle aneurysms, arising from the midportion of feeding artery pedicles, are at risk for recurrent rupture. In our series intranidal aneurysm and venous stenosis were two main structures related to hemorrhage. Interestingly, the intranidal aneurysm was regarded as a source of hemorrhage rather than the "pedicle" aneurysm in both illustrative cases, judging from location of the hematomas on MR images or CT scans. Intranidal aneurysm may not appear on angiograms taken after the hemorrhagic episode, as shown in Case 1. Hence angiography should be repeated to analyze the latent angioarchitectures in patients with hemorrhage as an initial symptom.

Correlation between venous drainage and hemorrhage has been discussed by several authors. Viñuela *et al.*,²¹⁾ Miyasaka *et al.*,¹²⁾ and Nataf *et al.*¹³⁾ emphasized either stenosis or occlusion of venous drainage was a high risk factor of hemorrhage. There are, however, some controversies about this concept, and opposite results have been shown.^{8,20)} Such difference may be partly due to the selection bias of the patients. The previous studies based on the majority of patients with a clinical symptom of hemorrhage are apt to support a strong correlation between venous drainage and hemorrhage. Hemorrhage might have already modified venous drainage, and have led to venous stenosis or occlusion because of compression by the hematoma.

Studies concerning hemodynamics in AVMs have revealed that feeder arterial pressure is positively correlated to hemorrhage.^{6,18)} Norbash *et al.*¹⁴⁾ clearly demonstrated a significant decrease of feeding arterial pressure in patients with angiomatous change, which denotes transcortical arterial recruitment by AVM from adjoining vascular territories. No hemorrhagic events were associated with this kind of angioarchitecture during follow-up in our series, which supports the negative correlation between angiomatous change and hemorrhage.

Surgical resection of the nidus can eliminate the risk of hemorrhage.⁵⁾ Surgery is justified particularly in smaller AVMs, because mortality and morbidity are lower than natural risk.⁷⁾ From our study, however, potentially hazardous lesions such as intranidal aneurysm and venous stenosis could be differentiated from stable structures, such as angiomatous change and arteriovenous fistula. Moreover, hemorrhage did often occur in large AVMs. When these dangerous lesions are delineated on angiographically the first treatment should be focused on them. In cases of large AVMs associated with such lesions, endovascular embolization would be a treatment of choice. Stereotactic radiosurgery is also likely to be effective in patients with intranidal aneurysm.¹⁷⁾

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