Original Article

Brain Arteriovenous Malformations: Experience in the Interventional Neuroradiology Unit, Ramathibodi Hospital

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Abstract

Objective: To review patient demographics, clinical symptoms, presentation, imaging characteristics and embolization results as well as its complications of patients with brain AVMs seen in the Interventional Neuroradiology unit at Ramathibodi Hospital.

Materials and Methods: Retrospective review of all patients diagnosed with brain AVMs from cerebral angiography during January 2001 to December 2005 at the Interventional Neuroradiology Unit, Radiology Department, Ramathibodi hospital.

Results: There were a total of 189 patients. 44 patients were excluded due to incomplete medical records or loss of imaging data. Of the remaining 145 patients, 87 (60%) underwent partial-targeted embolization, 9 (6.2%) underwent curative embolization, 29 (20%) radiosurgery alone, 11 (7.6%) surgical resection alone, 1 (0.7%) spontaneously thrombosed, 2 (1.4%) received conservative treatment without any further treatment and 6 (4.1%) loss to follow up. 84 (57.9%) were male patients and 61 (42.1%) were female. The mean age at the time of angiographic diagnosis was 27.2 years +/- 15.1. The initial presentations included intracranial hemorrhage in 88 (60.7%), seizures in 29 (20%), headaches in 18 (12.4%), 2 (1.4%) with focal neurological deficit, incidental finding in 1 (0.7%) and 7 (4.8%) with other presentations. Ruptured AVM were mainly of small size (65.9%, $P=0.03$), single deep vein (27.3%, $P<0.05$) with locations in the deep gray nuclei, midline structures and corpus callosum (31.8%, $P=0.001$). Only 18 of 27 intranidal aneurysms were found in patient with ruptured AVMs, two times higher compared to patients with non-ruptured AVMs however without statistical significance ($P=0.72$). In 87 patients with goals of partial targeted embolization, the majority of the AVMs (55 of 80) had more than 50% reduced flow, which were related to small and medium sized AVMs ($P<0.05$). AVMs with single arterial feeder ($P=0.001$) and single draining vein ($P<0.05$). There was failure of partial targeted embolization in 3 of 87 patients. Success rate of curative embolization is 89% (8 of 9 patients). Clinically significant complications after embolization (ischemia or hemorrhage) were seen in 7 of 96 patients (7.3%).

Conclusion: In our experience, the presentation of brain AVMs and risk factors of hemorrhage were similar to the previous studies. The total success rate of curative embolization was 8.3%, while partial targeted embolization was able to reduce the AVM flow more than 50% in the majority of patients with less significant clinical complications.

Keywords: Brain arteriovenous malformations, cerebral arteriovenous malformations

Background

Brain arteriovenous malformations (AVMs) represent an uncommon but important source of neurological morbidity in young adults.[1] The basic morphology is of a vascular mass, called the nidus, which directly shunts blood between the arterial and venous circulations without a true capillary bed. There is usually high flow through the feeding arteries, nidus and draining veins.

Clinical presentations of brain AVMs are variable. Intracranial hemorrhage is the most common clinical presentation of AVMs, with a reported frequency ranging from 30-82%.[2,3] Others include epileptic seizures, headaches and neurological deficits with only few appearing to be asymptomatic.[4]

The risk of hemorrhage from brain AVMs is approximately 1-3% per year[27-29] and persists until the lesion is completely obliterated. Therefore, the goal of treatment is to achieve complete obliteration with the minimal neurological risk and complications.[5] There are three established treatment modalities for brain AVMs, i.e. microsurgery, endovascular embolization and radiosurgery.[6] All play a role for specific patients. The most appropriate plan for any given patient, of course, would take many factors into account, including clinical risk factors, angiographic features, age, and neurological status.

Endovascular treatment of brain AVMs aims to be either curative or partial targeted to reduce the size, weak points or flow of the AVMs, however the complications are serious with a wide range of morbidity and mortality risk (10-50% neurological deficit, 1-4% mortality) in previous reports.[7]

The purpose of this study is to review patient demographics, clinical symptoms, presentation, imaging characteristics and embolization results as well as its complications of patients with brain AVMs seen in the Interventional Neuroradiology Unit at Ramathibodi hospital.

Materials and Methods

Patients

All patients diagnosed with brain AVMs from by cerebral angiography during January 2001 to December 2005 at Interventional Neuroradiology Unit at Ramathibodi Hospital were included in this study. Medical records and imaging data were retrospectively reviewed.

The patient lists are searched from the registration records and the hospital electronic database (ICD-10 code Q28.2). Patients with incomplete medical records or radiographic studies were excluded.

Imaging analysis:

CT, MRI and angiographic images were retrospectively reviewed by board certified interventional neuroradiologists.

AVM characteristics

The cerebral angiograms were analyzed for the AVM morphological characteristics, including size, anatomic location, arterial feeder (pattern and distribution of supply), venous drainage (either into the superficial cortical veins or the deep venous system), and the presence of associated prenidal, nidal and postnidal aneurysms. The AVM size was determined from CT, MRI and cerebral angiograms.

Cerebral angiography and endovascular embolization

The purpose of cerebral angiography and embolization was classified into three groups: 1) diagnostic cerebral angiography without endovascular treatment, 2) partial-targeted embolization (i.e. pre-surgical or pre-radiosurgical procedure) and 3) curative embolization. The criteria for the last group includes small AVM size, single or two arterial feeders and probability to reach the nidus with a microcather.

Diagnostic cerebral angiography was performed via a transfemoral arterial route under local anesthesia. After placement of a 5-6-Fr Terumo sheath, a 4-5-Fr vertebral catheter was routinely used for young patients while a 5-Fr JB or Sim-2 catheter was used for older patients with tortuous aortic arches to select the carotid and vertebral arteries. Digital subtraction angiographic in antero-posterior and lateral views were performed with iodinated contrast injections of carotid or vertebral arteries, depending on AVM location.

In contrast, endovascular embolization procedures were performed with the patients under general anesthesia. Selective angiography was used in pre-embolization planning, and standard techniques of digital subtraction angiography with road mapping were used during endovascular procedures.

Flow- and wire-guided microcatheters of various sizes were used to advance the microcatheter tip as close as possible to the AVM nidus. Once in optimal position (i.e., wedged in the nidus or placed in the terminal arterial feeder proximal to the nidus), permanent vessel occlusion was obtained with injection of n-butyl cyanoacrylate (NBCA) into the target vessels. The degree of nidal penetration and target vessels varied, depending on the embolization treatment goals. If embolization was used as an adjunct to surgical resection, the specific goals of embolization were to facilitate surgical excision by 1) nidus size and flow reduction, 2) the occlusion of deep, surgically inaccessible or deep feeding arteries 3) and the occlusion of intranidal arteriovenous fistulas.[6,8] If embolization was performed before radiosurgery, then AVM compartment penetration was attempted to achieve also greater size & reduction and make the AVM more amenable to radiation therapy.[9] The flow reduction was evaluated and classified into 4 ranges depend on percentage of reduced flow after the embolization (Figure 1). If embolization was used alone, the goal of embolization was complete obliteration of the AVM nidus. All treated patients underwent both radiographic (magnetic resonance imaging, computered tomography or cerebral angiography) and clinical follow-up after embolization.

Statistical analysis

All patient data were analyses by statistical software (SPSS, version 13.0 for Windows). The continuous data such as patient's age and total number of embolization procedures were calculated for mean, SD and median (range) values. Other frequency data i.e. sex, clinical symptoms, presentation, imaging characteristics, embolization results, technical complications and clinically significant complications are described by number of cases and percentage.

Chi-square tests (X2) were done for statistical significance with a confidence interval of 95% ($P<0.05$).

Results

Patients and AVM characteristics

In a 5 year period from January 2001 to December 2005, there were 189 brain AVM patients were seen in our unit. 44 patients were excluded due to incomplete medical records in 16 patients, loss of imaging data in 25 patients and unknown causes of death in 3 patients. Remaining 145 patients

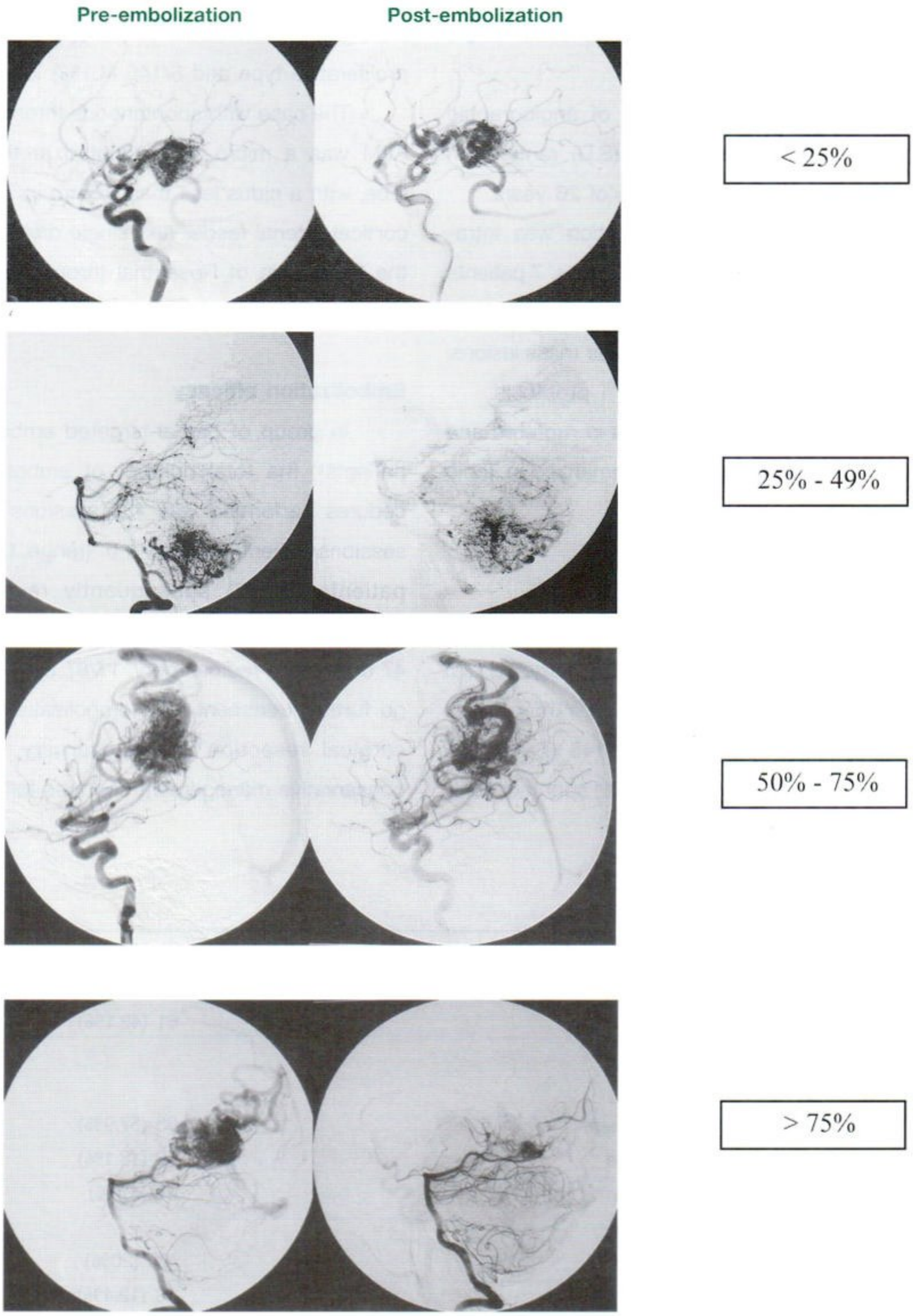

Fig.1 Percentage of flow reduction compared between pre- and post embolization

were enrolled in this study. Patient demographics and clinical presentations are summarized in Table 1.

The mean age at the time of angiographic diagnosis was 27.2 years +/- 15.1 (S.D), range from 0.8 to 16 years and a median age of 26 years.

The most common presentation was intracranial hemorrhage in 88 (60.7%) patients. 7 patients presented with other symptoms including 2 visual problems, 1 facial neuralgia, 2 vascular mass lesions, 1 alteration of consciousness and 1 proptosis.

Morphological characteristics in ruptured and non-ruptured AVM groups are summarized in Table 2.

AVM treatments

96 in 145 of the patients (66.2%) underwent endovascular treatment with a partial-targeted aim in 87 (60%) and curative in 9 (6.2%). 29/145 patients (20%) had radiosurgery alone, 11/145 (7.6%) had surgical resection alone, 1/145 (0.7%) spontaneously thrombosed, 2/145 (1.4%) received conservative management without any further treatment due to proliferative type and 6/145 (4.1%) loss follow up.

The case with spontaneous thrombosis of the AVM was a micro AVM, located at the temporal lobe, with a nidus less than 0.5 cm in size, a single cortical arterial feeder and single draining vein into the basal vein of Rosenthal thrombosed after the first episode of hemorrhage.

Embolization efficacy

In group of partial-targeted embolization (87 patients), the total number of embolization procedures performed was 133 sessions (mean =1.5 sessions/patient, median 1.0 (range 0-4)). Most patients (63/87) subsequently received other treatment; of which 16 underwent surgical resection, 47 underwent radiosurgery. 17/87 patients received no further treatment after embolization - 2 denied surgical resection or radiosurgery, 3 received conservative management and loss follow up in 12

Table 1 Patient characteristics (n=145)

Sex, n (%)	
Male	84 (57.9%)
Female	61 (42.1%)
Initial presentation, n (%)	
Rupture	
Intraparenchymal hemorrhage	63 (57.9%)
Intraventricular hemorrhage	19 (13.1%)
Subarachnoid hemorrhage	6 (4.1%)
Non-rupture	
Seizure	29 (20%)
Headache	18 (12.4%)
Focal neurological deficit	2 (1.4%)
Incidental (in patient with subdural hematoma)	1 (0.7%)
Others	7 (4.8%)

Table 2 Arteriovenous malformation characteristics

	Total AVMs (n=145)	Ruptured AVMs (n = 88)	Non-ruptured AVMs (n = 57)	P value
AVM size (Spetzler-Martin criteria), n (%)				*P=0.03*
Small (<3 cm)	73 (50.3%)	58 (65.9%)	15 (26.3%)	
Medium (3-6 cm)	44 (30.3%)	21 (23.9%)	23 (30.4%)	
Large (> 6 cm)	28 (19.3%)	9 (10.2%)	19 (33.3%)	
AVM location, n (%)				*P=0.001*
Lobar location	110 (75.9%)	60 (68.2%)	50 (87.7%)	
Frontal lobe	38 (26.2%)	18 (20.5%)	20 (35.1%)	
Parietal lobe	26 (17.9%)	15 (17%)	11 (19.3%)	
Temporal lobe	29 (20.0%)	18 (20.5%)	11 (19.3%)	
Occipital lobe	4 (2.8%)	1 (1.1%)	3 (5.3%)	
Cerebellar lobe	13 (9.0%)	8 (9.1%)	5 (8.8%)	
Deep gray nuclei and midline structures	23 (15.9%)	19 (21.6%)	4 (7.0%)	
Basal ganglia	6 (4.1%)	3 (3.4%)	3 (5.3%)	
Thalamus	13 (9.0%)	13 (14.8%)	0	
Brain stem	4 (2.8%)	3 (3.4%)	1 (1.8%)	
Others	12 (8.3%)	0	0	
Corpus callosum	11 (7.6%)	9 (10.2%)	2 (3.5%)	
Optic chiasm	1 (0.7%)	0	1 (1.8%)	
Arterial feeders, n (%)				*P=0.35*
Single	21 (14.5%)	18 (20.5%)	3 (5.3%)	
Cortical branch	13 (8.9%)	11 (12.5%)	2 (3.5%)	
Choroidal branch	6 (4.1%)	5 (5.7%)	1 (1.8%)	
Perforator branch	2 (1.4%)	2 (2.3%)	0	
Multiple	124 (85.5%)	70 (79.5%)	54 (94.7%)	
Cortical branches	78 (53.8%)	40 (45.5%)	38 (66.7%)	
Choroidal branches	3 (2.1%)	3 (3.4%)	0	
Perforator branches	6 (4.1%)	5 (5.7%)	1 (1.8%)	
Combined	37 (25.6%)	22 (25%)	15 (26.3%)	
Associated aneurysm, n (%)				
Prenidal	21 (14.5%)	10 (11.4%)	11 (19.3%)	*P=0.41*
Nidal	27 (18.6%)	18 (20.5%)	9 (15.8%)	*P=0.72*
Postnidal	18 (12.4%)	10 (11.4%)	8 (14%)	*P=0.98*
Venous drainage, n (%)				*P<0.05*
Single	54 (37.3%)	46 (52.3%)	8 (14%)	
Superficial vein	19 (13.1%)	16 (18.2%)	3 (5.3%)	
Deep vein	28 (5.5%)	24 (27.3%)	4 (7%)	
Cavernous sinus	1 (0.7%)	1 (1.1%)	0	
Posterior fossa vein	6 (4.1%)	5 (5.7%)	1 (1.8%)	

Table 2 (continue) Arteriovenous malformation characteristics

	Total AVMs (n=145)	Ruptured AVMs (n = 88)	Non-ruptured AVMs (n = 57)	P value
Venous drainage, n (%)				*P<0.05*
Multiple	91 (62.3 %)	42 (47.7%)	49 (86%)	
Presence of deep vein +/- cavernous sinus	45 (31%)	20 (22.7%)	25 (43.9%)	
Absence of deep vein or cavernous sinus	46 (31.7%)	22 (25%)	24 (42.1%)	
Spetzler-Martin grade, n (%)				*P=0.29*
I	27 (18.6%)	21 (23.9%)	6 (10.5%)	
II	43 (29.7%)	25 (28.4%)	18 (31.6%)	
III	43 (29.7%)	32 (36.4%)	11 (19.3%)	
IV	19 (13.1%)	6 (6.8%)	13 (22.8%)	
V	13 (9.0%)	4 (4.5%)	9 (15.8%)	
Presence of venous obstruction, n (%)	12 (8.3%)	6 (6.8%)	6 (10.5%)	*P=0.89*
Intranidal fistula, n (%)	12 (8.3%)	6 (6.8%)	6 (10.5%)	*P=0.34*

Table 3 Angiographic features and totally reduced flow of AVMs (n=80)

	Percentage of totally reduced flow				
	< 25% (n=5)	25-49% (n=20)	50-75% (n=33)	>75% (n=22)	P value
AVM size (Spetzler-Martin criteria), n (%)					*P<0.05*
Small (<3 cm)	0	5 (25%)	9 (27.3%)	14 (63.6%)	
Medium (3-6 cm)	3 (60%)	9 (45%)	18 (54.5%)	5 (22.7%)	
Large (> 6 cm)	2 (40%)	6 (30%)	6 (18.2%)	3 (13.6%)	
AVM location, n (%)					*P=0.59*
Lobar location	5 (100%)	16 (80%)	29 (87.8%)	16 (7.7%)	
Deep gray nuclei and midline structures	0	3 (15%)	2 (6.1%)	2 (9.1%)	
Others:					
Corpus callosum	0	0	2 (6.1%)	4 (18.2%)	
Optic chiasm	0	1 (5%)	0	0	
Arterial feeders, n (%)					*P=0.001*
Single	0	0	0	7 (31.8%)	
Multiple	5 (100%)	20 (100%)	33 (100%)	15 (68.2%)	
Venous drainage, n (%)					*P<0.05*
Single	1 (20%)	2 (10%)	7 (21.2%)	16 (72.7%)	
Multiple	4 (80%)	18 (90%)	26 (78.8%)	6 (27.3%)	

Table 3 (continue) Angiographic features and totally reduced flow of AVMs (n=80)

	Percentage of totally reduced flow				
	< 25% (n=5)	25-49% (n=20)	50-75% (n=33)	>75% (n=22)	P value
Spetzler-Martin grade, n (%)					*P=0.18*
I	0	3 (15%)	3 (15%)	4 (18.2%)	
II	2 (40%)	5 (25%)	10 (30.3%)	11 (50%)	
III	2 (40%)	4 (20%)	11 (33.3%)	3 (13.6%)	
IV	1 (20%)	6 (30%)	4 (12.1%)	3 (13.6%)	
V	0	2 (10%)	5 (15.2%)	1 (4.5%)	
Obliteration of intranidal fistula, n (%)	0	4 (20%)	4 (12.1%)	1 (22%)	*P=0.037*
Subsequently treatments, n (%)					
Radiosurgery	2 (40%)	14 (70%)	20 (60.6%)	11 (50%)	*P=0.378*
Surgical resection	1 (20%)	2 (10%)	7 (21.2%)	6 (27.3%)	*P=0.673*
No further treatment	0	0	0	3 (13.6%)	*P=0.009*
Loss follow up	1 (20%)	3 (15%)	6 (18.2%)	2 (9.1%)	*P=0.632*
Denied further treatment	1 (20%)	1 (5%)	0	0	*P=0.003*

Table 4 Patient characteristics and angiographic features of 8 completed obliterated AVM patients in curative embolization

Patient ID	Sex	Age (yrs)	Clinical presentation	AVM location	No. and type of arterial feeders	Aneurysms	No. and type of venous drainage	Emboliza-tions (n)
4	M	17	ICH*	Corpus callosum	Single, cortical br.	Intranidal	Single, cortical v.	2
77	M	32	ICH	Frontal	Single, cortical br.	-	Single, deep v.	1
99	F	22	IVH**	Cerebellar	Two, cortical br.	Intranidal	Single, deep v.	1
109	F	13	ICH	Midbrain	Single, PchA***	-	Single, deep v.	2
112	F	13	ICH	Frontal	Single, cortical br.	-	Two, cortical v.	1
114	M	20	ICH	Cerebellar	Single, cortical br.	-	Single,posterior fossa v.	1
115	F	6	ICH	Temporal	Single, cortical br.	-	Single, deep v.	1
135	F	26	ICH	Parietal	Single, cortical br.	-	Single, deep v.	1

* = Intracerebral hemorrhage

** = Intraventricular hemorrhage

*** = Posterior choroidal artery

Table 5 Technical complications and clinically significant complications

Technical Complications, n (%)	
Microperforation	7 (43.8%)
Glued vein	5 (31.2%)
Microcatheter fracture	1 (6.3%)
Dissection	2 (12.5%)
Reflux of glue into parent artery	1 (6.3%)
Clinically significant complications, n (%)	
Intraparenchymal hemorrhage	2 (28.6%)
Intraventricular hemorrhage	1 (14.3%)
Subarachnoid hemorrhage	2 (28.6%)
Arterial infarction	2 (28.6%)

patients. 3/87 patients had complete obliteration of the nidus after follow up by MRI or diagnostic angiography without further treatment. 4/87 patients in which we failed embolization - 2 patients had radiosurgery, no further management in 1 patient (large proliferative type of AVM) and loss follow up in 1 patient. Angiographic features and totally reduced flow of 80 patients are summarized in Table 3. Evaluation of the totally reduced flow after the last embolization showed: 5 patients had <25% decreased flow, 20 patients had 25 to 49% decreased flow, 33 patients had 50 to 75% and 22 patients had >75% decreased flow.

All 9 intranidal fistulas, 12 of 14 (86%) intranidal aneurysms and 6 of 8 (75%) postnidal aneurysms were successfully treated with glue.

9 patients which we aimed for curative embolization, 8 were completely obliterated within the first or second time of embolization. The details of each patient are detailed in Table 4. 1 patient lost to follow-up after diagnostic angiography.

Complications

A total of 16 technical complications occurred during embolization. There were clinically significant complications in 7/16, the most frequent being intracerebral hemorrhage. No deaths related to these complications were encountered. The technical and clinically significant complications are summarized in Table 5.

Discussion

In previously reported series,[4,10,11] the mean age of patients with brain AVM varies from 33 to 35 years with female predominance. In our study, the mean patient age is lower, about 27.2 +/- 15.2 years with slightly more male patients (52.9%).

The most common presentations of brain AVMs are intracranial hemorrhage (60.6%) and seizure (20%), which are similar to other series.4,10,11 Other presenting symptoms include headaches, focal neurological deficits, visual disturbance, facial neuralgia and vascular mass lesion. Only 1 patient had an incidentally found brain AVM during investigation of a contralateral subdural hematoma.

Ruptured and non-ruptured AVMs had statistically significant differences in angiographic features (Table 2), which were the AVM size, localization in deep gray nuclei, midline structures and corpus callosum, including number and type of venous drainage. The small size and presence of deep vein drainage were associated with a high incidence of hemorrhagic presentations.[12,13] In our study, ruptured AVMs were mainly of small size (65.9%, P=0.03) with single deep vein drainage (27.3%, P<0.05). This observation contradicts with the previously reported study[30] since small sized AVMs tend to have no other symptoms and therefore, will only present after bleeding. No statistical difference between the two groups was found in cases with multiple draining veins. Moreover, AVMs locating in the deep gray nuclei, midline structures and corpus callosum (P=0.001) were also associated with high incidence of rupture. The presence of an intranidal aneurysm with association of intracranial hemorrhage is still controversial.[14] Mansmann U, et al15 reported factors associated with intracranial hemorrhage in brain AVM. His study demonstrated no any association of intranidal aneurysm and intracranial hemorrhage. In our study, only 18 of 27 intranidal aneurysms were found in patients with ruptured AVMs, two times higher as compared to patient with non-ruptured AVM however without any statistical significance (P=0.72). The lobar AVM location, number & type of arterial feeder, Spetzler-Martin grade, presence of venous obstruction and intranidal fistula were also not statistically significant.

In many studies,[9,16-18] the aim of partial targeted embolization was to reduce the AVM size and close the intranidal aneurysm to prevent of re-rupture of the AVM, which was then followed by radiosurgery or surgical resection for further treatment. In addition, in our personal experience, that reduction of the flow through the AVM and closing of intranidal fistula are helpful to promote epithelialization of AVM, facilitating complete obliteration after radiosurgery. Pre-operative embolization before surgical resection of the AVM has been shown to improved postsurgical outcome.[19]

The factors related to percentage of total flow reduction are shown in Table 3. The AVM size, number of arterial feeder and venous drainage were statistically significant. Most of the small and medium sized AVMs had more than 50% (55 of 80) reduced flow from embolization (P<0.005). AVMs with single arterial feeder (P=0.001) and single draining vein (P<0.05) were also found to have a higher percentage of decreased flow from the embolization. There was no statistical difference in the large sized AVMs and the AVMs with intranidal fistulas. All patients which received no further treatment after the embolization (n=3) until the time of this study had >75% reduced flow (P=0.009). 2 patients are children (11 and 14 age years) with residual small AVMs, whom we decided to follow up with possible further radiosurgery, 1 patient had CAMS with a large proliferative AVM. The AVM location and Spetzler-Martin grade were not statistically significant.

The prior study[20] revealed success rate of curative embolization was 60% with no recurrence in follow up angiography. In our study, we succeeded in complete obliteration of the AVM in 8 of 9 patients (89%) whom we aimed for curative embolization. The total success rate of curative embolization was 8.3%. All of these AVMs have a small size, one or two arterial feeders and required one or two sessions of embolization, similar to the other study[20] with varying locations. However, this high success rate is probably due to a large number of patients who were excluded from the study.

The major risk of embolization is ischemic or hemorrhagic complications during and after treatment. The complication rate is influenced primarily by the technical aspects of the procedure, including vascular damage, which is related to operator skill, catheter features,[21,22] characteristics of the embolic material including hemodynamic changes after embolization[23] and inflammatory reaction of vessels.[24] Complication rates vary among studies. Guo WY, et al[25] reported 19% morbidity and Sorimachi T, et al[26] reported 14% morbidity and death in 3%. In our series, permanent deficits were seen in 7of 96 patients with embolization (7.3%).

Our study has some limitations. Due to retrospective study, first, a large number of patients were excluded from loss of medical or angiographic data. Second, evaluation of total flow reduction is subjective data, which may lead to misinterpretion.

In the future, comparison of non-embolization and partial targeted embolization with complete obliteration of AVM after radiosurgery should be studied, in order to confirm our hypothesis regarding the usefulness of decreased flow after embolization.

Conclusion

The most common presentation of brain AVMs is intracranial hemorrhage, being more frequent in AVMs localized in the deep gray nuclei, midline structures and corpus callosum with single deep vein drainage. Small sized AVM were also found to have higher hemorrhagic presentations. The presence of intranidal aneurysms were two times higher in the ruptured AVMs compared to the non-ruptured ones and may be one risk factor. In our experience, partial targeted embolization was able to reduce the AVM flow more than 50% in the majority of patients with less significant clinical complications than the previous studies.

References

1. Arteriovenous Malformation Study Group. Current concepts: arteriovenous malformations of the brain in adults. N Engl J Med. 1999 June;340:1812-18.
2. Al-Shahi R, Warlow C. A systematic review of the frequency and prognosis of arteriovenous malformations of the brain in adults. Brain. 2001 Oct;124:1900-26.
3. Fleetwood IG, Steinberg GK. Arteriovenous malformations. Lancet. 2002 Mar 9;359(9309):863-73.
4. Stapf C, Khaw AV, Sciacca RR, Hofmeister C, Schumacher HC, Pile-Spellman J, et al. Effect of age on clinical and morphological characteristics in patients with brain arteriovenous malformation. Stroke. 2003 Nov;34(11): 2664-9.
5. Lee CZ, Young WL. Management of brain arteriovenous malformations. Curr Opin Anaesthesiol. 2005 Oct;18(5): 484-9.
6. Ogilvy CS, Stieg PE, Awad I, Brown RD Jr, Kondziolka D, Rosenwasser R, et al. Recommendations for the management of intracranial arteriovenous malformations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. Circulation. 2001 May 29;103(21):2644-57.
7. Ledezma CJ, Hoh BL, Carter BS, Pryor JC, Putman CM, Ogilvy CS. Complications of cerebral arteriovenous malformation embolization: multivariate analysis of predictive factors. Neurosurgery. 2006 Apr;58(4):602-11.
8. Vinuela F, Dion JE, Duckwiler G, Martin NA, Lylyk P, Fox A, et al. Combined endovascular embolization and surgery in the management of cerebral arteriovenous malformations: experience with 101 cases. J Neurosurg. 1991 Dec; 75:856-64.

9. Gobin YP, Laurent A, Merienne L, Schlienger M, Aymard A, Houdart E, et al. Treatment of brain arteriovenous malformations by embolization and radiosurgery. J Neurosurg. 1996 Jul;85(1):19-28.

10. Halim AX, Singh V, Johnston SC, Higashida RT, Dowd CF, Halback VV, et al. Characteristics of brain arteriovenous malformations with coexisting aneuryms: a comparison of two referal centers. Stroke. 2002 Mar;33(3):675-9.

11. Hartmann A, Stapf C, Hofmeister C, Mohr JP, Sciacca RR, Stein BM, et al. Determinants of neurological outcome after surgery for brain arteriovenous malformation. Stroke. 2000 Oct;31(10):2361-4.

12. Watimo O. The relationship of size, density and localization of intracranial arteriovenous malformations to the type of initial symptom. J Neurol Sci. 1973 May;19(1):13-9.

13. Itoyoma Y, Uemura S, Ushio Y, Kuratsu J, Nonaka N, Wada H, et al. Natural course of unoperated intracranial arteriovenous malformations: study of 50 cases. J Neurosurg. 1989 Dec;71(6):805-9.

14. Kader A, Young WL, Pile-Spellman J, Mast H, Sciacca RR, Mohr JP, et al. The influence of hemodynamic and anatomic factors on hemorrhage from cerebral arteriovenous malformations. Neurosurgery. 1994 May;34(5):801-7; discussion 807-8.

15. Mansmann U, Meisel J, Brock M, Rodesch G, Alverez H, Lasjaunias P. Factors associated with intracranial hemorrhage in cases of cerebral arteriovenous malformation. Neurosurgery. 2000 Feb;46(2):272-9; discussion 279-81.

16. Liu HM, Huang YC, Wang YH. Embolization of cerebral arteriovenous malformations with n-butyl-2-cyanoacrylate. J Formos Medl Assoc. 2000 Dec;99(12):906-13.

17. Mounayer C, Hammami N, Piotin M, Spelle L, Benndorf G, Kessler I, et al. Nidal embolization of brain arteriovenous malformations using Onyx in 94 patients. AJNR Am J Neuroradiol. 2007 Mar;28(3):518-23.

18. van Rooij WJ, Sluzewski M, Beute GN. Brain AVM embolization with Onyx. AJNR Am J Neuroradiol. 2007 Jan;28(1):172-7, discussion 178.

19. DeMeritt JS, Pile-Spellman J, Mast H, Moohan N, Lu DC, Young WL, et al. Outcome analysis of preoperative embolization with N-Butyl cyanoacrylate in cerebral arteriovenous malformations. AJNR Am J Neuroradiol. 1995 Oct;16(9):1801-7.

20. Yu SC, Chan MS, Lam JM, Tam PH, Poon WS. Complete obliteration of intracranial arteriovenous malformation with endovascular cyanoacrylate embolization: initial success and rate of permanent cure. AJNR Am J Neuroradiol. 2004 Aug;25(7):1139-43.

21. Wikholm G, Lundqvist C, Svendsen P. Transarterial embolization of cerebral arteriovenous malformations: improvement of results with experience. AJNR Am J Neuroradiol. 1995 Oct;16(9):1811-7.

22. Frizzel RT, Fisher WS. Cure, morbidity, and mortality associated with embolization of brain arteriovenous malformations: a review of 1246 patients in 32 series over a 35-year period. Neurosurgery. 1995 Dec;37(6):1031-40.

23. Takeuchi S, Abe H, Nishimaki K, Minakawa T, Koike T, Kameyama S, et al. Cerebral haemodynamic changes after endovascular treatment of arteriovenous malformations: evaluation by single-photon emission CT. Acta Neurochir (Wien). 1994;127(3-4):142-50.

24. Deveikis JP, Manz HJ, Luessenhop AJ, Caputy AJ, Kobrine AI, Schellinger D, et al. A clinical and neuropathologic study of silk suture as an embolic agent for brain arteriovenous malformations. AJNR Am J Neuroradiol. 1994 Feb;15(2): 263-71.

25. Guo WY, Wikholm G, Karlsson B, Lindquist C, Snendsen P, Ericson K. Combined embolization and gamma knife radiosurgery for cerebral arteriovenous malformations. Acta Radiol. 1993 Nov;34(6):600-6.

26. Sorimachi T, Koike T, Takeuchi S, Minakawa T, Abe H, Nishimaki K, et al. Embolization of cerebral arteriovenous malformations achieved with polyvinyl alcohol particles: angiographic reappearance and complications. AJNR Am J Neuroradiol. 1999 Aug;20(7):1323-8.

27. Karlsson B, Lindquist C, Johansson A, Steiner L: Annual risk for the first hemorrhage from untreated arteriovenous malformations. Minim Invasive Neurosurg. 1997 Jun; 40(2):40-6.

28. Brown RD Jr, Wiebers DO, Forbes G, O'Fallan WM, Peipgras DG, Marsh WR, et al. The natural history of unruptured intracranial arteriovenous malformations. J Neurosurg. 1988 Mar;68(3):352-7.

29. Graf CJ, Perret GE, Torner JC. Bleeding from cerebral arteriovenous malformations as part of their natural history. J Neurosurg. 1983 Mar;58(3):331-7.

30. Stefani MA, Porter PJ, terBrugge KG, Montanera W, Willinsky RA, Wallace MC. Large and deep brain arteriovenous malformations are associated with risk of future hemorrhage. Stroke. 2002 May;33(5):1220-4.