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Treatment of brain arteriovenous malformations with high-flow arteriovenous fistulas: risk and complications associated with endovascular embolization in multimodality treatment

Clinical article

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Object. High-flow fistulas associated with brain arteriovenous malformations (AVMs) pose a significant challenge to both stereotactic radiosurgery (SRS) and surgical treatment. The purpose of this study was to examine the outcomes of multimodality treatment of AVMs in association with a large arteriovenous fistula (AVF), with a special focus on endovascular embolization and its associated complications.

Methods. One hundred ninety-two patients harboring cerebral AVMs underwent endovascular treatment in the authors' department between 1997 and 2003. Of these, the authors selected 74 patients presenting with an AVM associated with high-flow AVF(s) for a retrospective analysis based on the findings of superselective angiography. After endovascular embolization, 32 patients underwent resection, 33 underwent either SRS or hypofractionated stereotactic radiotherapy (HSRT), and 3 underwent both surgery and SRS. Six patients underwent embolization only. Immediate and midterm treatment outcomes were analyzed.

Results. Fifty-seven (77%) of the 74 patients had AVMs that were Spetzler-Martin Grade III or higher. A complete resection was achieved in all 32 patients. Of patients who underwent SRS/HSRT, 13 patients (39.3%) had either complete or > 90% obliteration of the AVM, and 2 patients (6.1%) had incomplete obliteration. Fourteen patients (42.4%) with residual AVM underwent repeated radiotherapy (and remain under observation). Of the 3 patients who underwent both SRS and resection, resection was complete in 2 and incomplete in one. No follow-up was obtained in 6 patients (8.1%). An endovascular complication was observed in 4 patients (5.4%). Fistula embolization was safely performed in every patient, whereas every endovascular complication was associated with other procedures such as nidus embolization.

Conclusions. Endovascular occlusion of the fistulous component was successfully achieved in every patient; every endovascular complication in this series was related to other procedures such as nidus embolization. The importance of the fistula treatment should be emphasized to minimize the endovascular complications and to maximize the treatment effect when a multimodality therapy is used to treat brain AVMs with large AVF.
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Key Words • **brain arteriovenous malformation** • **high-flow fistula** • **endovascular complication** • **multimodality treatment**

A combination of endovascular embolization, SRS, and resection has been used in the therapy of brain AVMs.[9,17,23,25] Recent reports have indicated that AVMs with a nonplexiform component such as a high-flow AVF appear to be more resistant to radiosurgery than other lesions.[6,8,19] It is also known that AVMs with high-flow shunts are associated with an increased incidence of perioperative complications such as intra- or postoperative bleeding.[4,18,31] Therefore, the endovascular occlusion of those high-flow fistulas plays an important role in perioperative radiosurgery management.[28,32]

Recently, Haw et al.[11] reviewed a large series of patients with brain AVMs treated with embolization and concluded that the presence of a high-flow fistula or fistulous component of the AVM increases the risk of complications. Although much has been described about the angiographic characteristics of the fistulous component of AVMs,[12,16,36] there have been very few studies that specifically addressed the treatment risk of embolizing high-flow fistulas in AVM and the complications related to this aspect of surgery.

Abbreviations used in this paper: AVF = arteriovenous fistula; AVM = AV malformation; GDC = Guglielmi detachable coil; GOS = Glasgow Outcome Scale; HSRT = hypofractionated stereotactic radiotherapy; NBCA = *N*-butyl cyanoacrylate; SRS = stereotactic radiosurgery.

In this series, we reviewed the treatment outcomes of 74 patients with brain AVMs and high-flow AVFs. All patients underwent endovascular embolization followed by resection or SRS. The treatment outcome was analyzed with a special focus on the risk of endovascular treatment.

Methods

Patient Demographics

One hundred ninety-two patients with cerebral AVMs underwent endovascular treatment in our department between January 1997 and October 2003. Of these individuals, 74 patients presenting with an AVM and an associated high-flow AVF were selected for a retrospective analysis. Thirty-two patients were male and 42 patients were female. Their average age was 40 years. The AVMs were diagnosed by either CT or MR imaging at the time of initial presentation. Preembolization digital subtraction angiography was performed in all cases to obtain a detailed angioarchitecture of the vascular malformation.

Image Evaluation

The AVMs were classified using the Spetzler-Martin classification (size of the AVM, eloquence of the adjacent brain, and the presence/absence of deep venous drainage).[27] The AVM size was determined by CT, MR imaging, or digital subtraction angiography. Cerebral digital subtraction angiography was used to determine the patterns of the angioarchitecture and to identify the presence of any intranidal aneurysms, high-flow AVFs, and compromised venous drainage associated with the AVM nidus.

Definition of "High-Flow AVF" in an AVM

In most cases, an AVM is a cluster of multiple components with different types of angioarchitecture.[16,36] Although conventional selective angiography with a guide catheter is useful to ascertain general angiographic information about the AVM, the modality is not sufficient to delineate the angioarchitecture of its structural components. Once the complete picture of an AVM is obtained, superselective injection of every major feeding artery using a microcatheter is mandatory to evaluate detailed anigostructure.[12,16,33,36] Furthermore, the tip of the microcatheter needs to be placed near the fistula so that the orifice of the fistula—that is, the transition of artery and vein—is clearly visualized (see Fig. 2B). In this study, we defined a high-flow AVF as follows: 1) An abnormally dilated feeding artery is present. 2) The abnormally dilated feeding artery is directly connected to an abnormally dilated venous component or varix. 3) There is no plexiform component between the 2 structures. 4) The diameter of the feeding artery is more than twice as large as the arteries supplying the comparable areas not supplying the AVM (for example, the corresponding contralateral cerebral artery), or the diameter of the feeding artery is > 2 mm. Any case involving a dural AVF was excluded from this study.

Endovascular Procedure

All procedures were conducted after administration of general anesthesia and systemic heparinization. Fistulous occlusion was prioritized in every endovascular procedure, whereas nidus occlusion was performed as complementary therapy to be followed by surgical/radiosurgical treatment.

Selective catheterization of the artery supplying the AVF was performed using road-mapping techniques. Superselective angiography of the AVF was performed to locate the junction between the feeding artery and the initial venous component. The primary aim of the procedure was to occlude the lesion at the fistula site. However, not infrequently, it was necessary to occlude the abnormally dilated venous component associated with the fistula. Prior to the embolization, we always confirmed that there were multiple venous outlets in the AVM and that the target fistula component was not the conjoined outlet of a plexiform AVM but an isolated arteriovenous shunt. As an initial step, oftentimes, detachable coil devices were delivered to a target site through a microcatheter that was advanced using an over-the-guidewire technique. To avoid untoward embolic migration into the venous system, cerebral veins, dural sinus, and pulmonary circulation, the liquid embolic agents were used only after a sufficient flow reduction was obtained by predeployed coil materials. Liquid embolic agents used included NBCA or the Onyx Liquid Embolic System. In most cases, the endovascular procedure was shifted to embolization of the nidus component. In some cases, however, the nidus embolization was postponed for a few days if postembolization angiography showed intense flow stagnation in large cerebral varix/varices. An immediate postembolization head CT scan was obtained in all cases to rule out the development of "silent" intracranial bleeding. On average, a total of 2 to 4 feeders were embolized per session. Multistaged embolizations were performed at intervals of 3–4 weeks.

Treatment Strategy of Radiation Therapy/Resection

Surgically approachable lesions were generally indicated for resection. Small-sized AVMs (defined as < 3 cm) and superficial AVMs were generally resected. For those considered to be surgically unapproachable, stereotactic radiotherapy was considered. There were 2 different approaches depending on the radiation method: single-dose SRS or HSRT. If an AVM was < 5 cm in maximal diameter, it was treated with SRS. If the maximal diameter was ≥ 5 cm, it was treated with HSRT. Lesions treated with HSRT had a volume of 12 ml or larger (12–155 ml). Three to 5 years after the radiation therapy, repeated SRS or HSRT was discussed with the patient if any residual AVM was observed on follow-up imaging studies. In individuals with remaining fistulous components or a significant plexiform component, additional endovascular treatment was performed prior to the second SRS/HSRT.

Resection was generally performed between 7–10 days after the final embolization. All patients underwent postoperative angiography and MR imaging. For those who underwent SRS, a Radionics BRW or a BrainLAB stereotactic frame was placed during the last endovascular procedure. The patients were then transferred to the SRS treatment room. A custom-fitted thermoplastic mask

(U-PLAST, BrainLAB AG) was used for immobilization during HSRT. The Novalis dedicated system (BrainLAB AG) with miniature multileaf collimator capability was used in this series.

Evaluation of Posttreatment Imaging Studies

For the purpose of analysis, the patients were subdivided into 4 groups: patients who underwent resection, radiation therapy, both surgery and radiosurgery, and those who only underwent embolization. For those who underwent resection, postoperative angiograms were obtained 1–2 weeks after the procedure. Posttreatment MR images were acquired at 3 and 6 months. For those who underwent radiation therapy, posttreatment MR imaging and MR angiography were performed every 12 months up to 36 months after treatment or until complete occlusion was confirmed. Magnetic resonance angiography was used as the screening tool to evaluate the occlusion rate, but conventional angiography was always performed to make the final confirmation. Regardless of the treatment modality, patients with residual AVM confirmed on follow-up angiograms were reevaluated for repeated radiation therapy or resection on the basis of their age, clinical condition, and the response to the first radiosurgical procedure.

Evaluation of Clinical Outcome

The GOS[14] was used to evaluate clinical results after surgery or SRS/HSRT. Midterm follow-up evaluations were performed in each case with office visits and telephone interviews. Just as we did with the image analysis, the patients were subdivided into 4 groups, and the clinical outcome in each group was independently evaluated.

Results

Patient Demographics

Seventy-four patients underwent 127 embolizations for an average of 1.7 embolizations per patient. Seven patients (9.5%) had Spetzler-Martin Grade I, 10 (13.5%) had Grade II, 20 (27%) had Grade III, 22 (29.7%) had Grade IV, and 15 (20.3%) had Grade V lesions (Table 1). Thirty-two patients underwent endovascular embolization followed by resection. Thirty-three patients underwent endovascular embolization followed by SRS/HSRT. In the subgroup of patients treated with SRS/HSRT, there were no Grade I lesions. Two patients (6.1%) had Grade II, 12 patients (36.4%) had Grade III, 12 patients (36.4%) had Grade IV, and 7 patients (21.2%) had Grade V. In the subgroup of patients treated by surgery, 6 patients (18.8%) had Grade I, 7 patients (21.9%) Grade II, 8 patients (25.0%) Grade III, 5 patients (15.6%) Grade IV, and 6 patients (18.8%) had Grade V lesions. Clinical presentations associated with hemorrhagic events included parenchymal hemorrhage in 20 patients (27%), subarachnoid hemorrhage in 3 patients (4%), and intraventricular hemorrhage in 5 patients (6.8%). Other clinical presentations were seizures in 27 patients (36.5%), headaches in 10 patients (13.5%), progressive neurological deficit in 5 patients (6.8%), and an incidental finding in 4 patients (5.4%). High-resolution cerebral digital angiography depicted one or more high-flow AVFs in each patient.

TABLE 1: Spetzler-Martin grading of brain AVMs

Spetzler-Martin Grade	No. of Patients (%)
I	7 (9.5)
II	10 (13.5)
III	20 (27)
IV	22 (29.7)
V	15 (20.3)
total	74 (100)

In the SRS/HSRT group, the mean AVM nidus volume was 15.1 ml (range 0.2–43.9 ml) and the median volume was 11.3 ml. The prescription dose at the first treatment varied between 7.5 and 30 Gy (mean 17.1 Gy, median 15.5 Gy), and the dose at the second treatment was between 12 and 30 Gy (mean 16.6 Gy, median 15 Gy). The prescription isodose lines varied between 85 and 90% (mean 88.3%, median 90%). Three patients underwent both surgery and SRS after embolization. Six patients received only endovascular treatment for the following reasons: 1) palliative treatment was indicated for progressive neurological symptoms associated with high-flow shunts in 2 patients; 2) 1 patient planned to have a multimodality treatment but died after an endovascular complication; 3) 1 patient died due to rupture of the AVM; and 4) 2 patients were lost to follow-up while waiting for the surgical/radiosurgical treatment. Superselective angiograms were acquired before endovascular embolization. We used NBCA for embolization in 59 patients (79.7%), either alone or in combination with coil materials. Berenstein Liquid Coils (Target Therapeutics, Inc.) were used in 21 patients (28.4%) and GDCs (Target Therapeutics, Inc.) alone were used in 19 patients (25.7%). Onyx (ev3) was used in 18 patients (24.3%).

Angiographic Outcome

In all 32 patients (100%) who underwent resection after embolization, complete AVM removal was achieved. In 33 patients who underwent postembolization radiation therapy, complete AVM obliteration was achieved in 10 patients (30.3%) and > 90% obliteration was achieved in 3 patients (9.1%), whereas in 2 patients (6.1%) obliteration was incomplete. In 14 patients (42.4%) the lesion remained incompletely obliterated and these individuals underwent repeated radiotherapy for a residual nidus component. No follow-up was obtained in 4 patients (12.1%). Of the 3 patients who underwent both SRS and resection, 2 patients had complete resection and 1 had an incomplete resection. In the 6 patients who underwent endovascular embolization alone, complete AVM obliteration was demonstrated in 2 and incomplete obliteration was shown in 2. The remaining 2 patients did not undergo follow-up evaluation (Table 2).

Immediate Morbidity/Mortality

Fifty-three patients (71.6%) exhibited a good recovery (GOS Score 5) immediately after the acute treatment.

TABLE 2: Midterm angiographic outcomes

Treatment & Extent of AVM Obliteration	No. of Patients (%)
resection	
complete	32 (100)
incomplete	0 (0)
no follow-up	0 (0)
SRS/HSRT	
complete obliteration	10 (30.3)
>90% occlusion	3 (9.1)
incomplete (stopped)	2 (6.1)
incomplete (ongoing)*	14 (42.4)
no follow-up	4 (12.1)
SRS + op	
complete	2 (66.7)
incomplete (ongoing)*	1 (33.3)
no follow-up	0 (0)
embolization only	
complete	2 (33.3)
incomplete (stopped)	2 (33.3)
incomplete (ongoing)*	0 (0)
no follow-up	2 (33.33)
total	74

* The patient is in a posttreatment observation period (under treatment) after initial or secondary radiosurgery, and a complete obliteration of treated AVM has not been confirmed.

Fourteen patients (18.9%) exhibited a moderate disability (GOS Score 4) and 5 patients (6.8%) severe disability (GOS Score 3). One patient died of initial bleeding and another died (2.7%) of a complication related to the embolization procedure (GOS Score 1).

Midterm Morbidity/Mortality

The mean follow-up duration after the last treatment was 20.2 months. Fourteen of 33 patients who underwent SRS/HSRT had follow-up of at least 3 years Fifty-four patients (73.0%) exhibited a good recovery (GOS Score 5). Ten patients (13.5%) had a moderate disability (GOS Score 4) and 6 (8.1%) had severe disability (GOS Score 3). Four patients (5.4%) died (GOS Score 1). One patient died due to a technical complication. The other 3 patients died due to hemorrhage of AVMs after treatment.

Midterm Morbidity Related to Technical Complication: Endovascular Treatment

Technical complications related to the embolization occurred in 4 patients.

Case 1 (Spetzler-Martin Grade II). A cerebral cortical infarction occurred 2 days after the initial embolization. The patient presented with transient hemiparesis that resolved over a month (GOS Score 5).

Case 2 (Spetzler-Martin Grade V). A distal migration of NBCA into the draining vein occurred during the nidus embolization, which resulted in cortical bleeding. Although we removed the AVM immediately after the endovascular procedure, the patient had mild hemiparesis (GOS 3).

Case 3 (Spetzler-Martin Grade III). A perforation of a parent artery occurred during the procedure. A postembolization CT scan revealed mild subarachnoid hemorrhage, and the patient underwent resection of the AVM immediately. The patient presented with a mild lower-extremity monoplegia (GOS Score 4).

Case 4 (Spetzler-Martin Grade III). A patient with intractable seizure caused by a large AVM in the posterior fossa was treated with 2 sessions of embolization. An arterial perforation occurred in the second procedure, and intraparenchymal bleeding was confirmed on a postoperative CT scan. This patient died in the acute postoperative period.

There was no complication related to the endovascular occlusion of high-flow AVFs.

Midterm Morbidity Related to Technical Complication: Surgical Treatment

Technical complications related to the resections occurred in 2 patients.

Case 1 (Spetzler-Martin Grade III). This patient had a mild short-term memory disturbance after removal of an AVM located at the left temporal lobe (GOS Score 4).

Case 2 (Spetzler-Martin Grade V). A postoperative hemorrhage occurred. A minor oozing from the residual AVM was confirmed during the second operation and hematoma evacuation was performed. The patient had mild cerebellar symptoms (GOS Score 4).

Midterm Morbidity Related to Technical Complication: SRS/HSRT

Case 1 (Spetzler-Martin Grade V). A radiation necrosis caused by SRS occurred in this patient 1 year after the treatment. The patient, who has an AVM located in the right temporal lobe, developed progressive left hemiparesis that exhibited a mild improvement at the last visit (GOS Score 4) (Table 3).

Discussion

High-Flow AVF Associated With AVMs: Anatomical and Hemodynamic Challenge for Radiation Therapy and Surgery

Cerebral AVMs associated with high-flow AVFs present a therapeutic challenge to SRS and frequently result in incomplete AVM obliteration.[8,19,24] Histopathological findings observed in animal experiments as well as in clinical data suggest that SRS is more successful in occluding small vessels of plexiform AVMs than in larger high-flow vessels.[13,26] Meanwhile, AVMs with high-flow AVFs are also associated with increased perioperative hemorrhagic events.[4,18,31] Therefore, endovascular embolization of large AVFs is considered to be of utmost importance in the endovascular management before the SRS/HSRT or resection.[9,32]

TABLE 3: Midterm morbidity caused by technical complications

Treatment Method	No. of Patients (%)
embolization	4 (5.4)
resection	2 (5.6)*
radiation therapy	1 (2.8)†

* Denominator is the number of patients (35) who underwent surgery.
† Denominator is the number of patients (36) who underwent radiosurgery.

Concerns About Postembolization Hyperemic Status and Postoperative Bleeding

In general, acute compromise of the venous drainage of a plexiform AVM or dural sinus in the early phases of embolization does carry a risk of hemorrhage.[1,37] Nevertheless, a fistulous occlusion does not necessarily lead to such hemorrhagic complications as long as the procedure does not compromise venous drainage of the plexiform component or dural sinuses. Moreover, despite the fact that fistula embolization was always the priority during the endovascular procedure and was often done before the occlusion of the plexiform component in this series, there was no perioperative bleeding directly associated with fistula embolization. Figure 1 shows conceptual diagrams of an AVM before and after fistulous occlusion. A high-flow fistula unit, in most cases, is observed as an independent vascular architecture from the nidus component (Fig. 1 *upper*). Before the treatment, in most cases, intracranial venous pressure is significantly increased because of the high-flow shunting. The resultant venous hypertension would therefore induce a circulation disturbance in the surrounding tissue, including nidus components. In fact, after the occlusion of a large AVF, angiograms often demonstrate a significant reduction of cortical vein reflux as well as remarkable size reduction in the draining veins of a plexiform AVM (Fig. 1 *lower*). Therefore, it is conceivable that the positive effect from the decreased venous pressure can outweigh the potential negative effect from the relatively increased flow in the plexiform AVM.

Preoperative Embolization and Perioperative Hemorrhagic Event

Spetzler and colleagues[28] have reported their clinical experience of surgically treated AVMs that were pretreated with staged embolization. They emphasized the importance of staged presurgical embolization to facilitate the intraoperative manipulation and to minimize the perioperative complications such as nonperfusion-pressure breakthrough. In every patient in our series who underwent resection, a complete AVM removal was achieved, and there was no postoperative hemorrhagic event associated with the hemodynamic change. This may indicate that the presurgical embolizations indeed played a positive role in this series.

Use of SRS/HSRT for the Treatment of Large AVMs

Thirty-one (93.9%) of 33 patients in the SRS/HSRT group had a Spetzler-Martin Grade III or higher lesion,

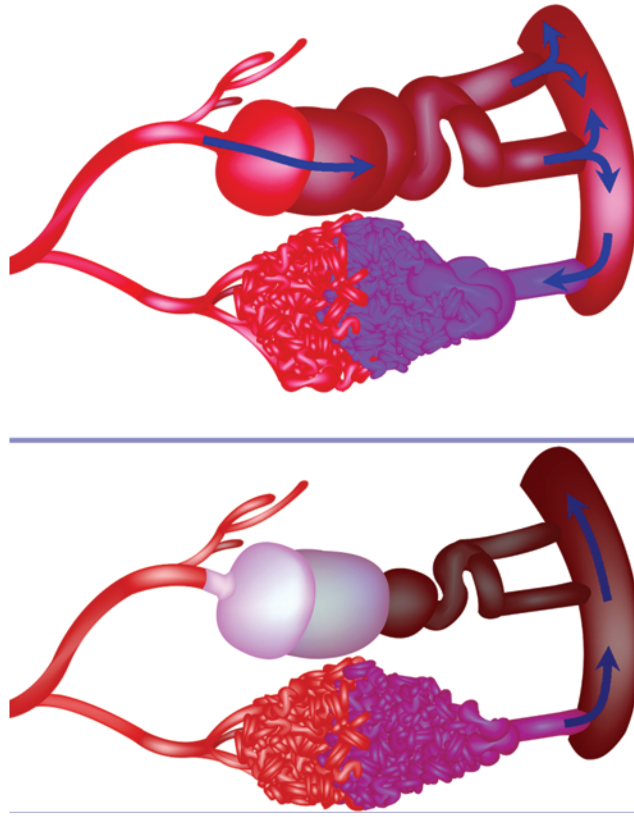

Fig. 1. Conceptual diagrams of pre- and postembolization of a high-flow AVF. ***Upper:*** A high-flow fistula unit *(upper portion)* is, in most cases, an independent vascular architecture from the plexiform component *(lower portion)*. Because of the high-flow arteriovenous shunting, the venous pressure is significantly increased *(arrows)*. Resultant venous hypertension induces circulation disturbance in the surrounding tissue including plexiform components. ***Lower:*** After occlusion of a large AVF, local circulation dramatically improves along with venous hypertension. Postoperative angiograms often demonstrate a reduction of cortical vein reflux as well as the size reduction of draining veins of plexiform AVM. The positive effect from the improved local circulation may outweigh the potential negative effect from the relatively increased flow in the plexiform AVM.

and 30 (90.1%) of 33 harbored AVMs ≥ 3 cm at the time of the first treatment.

Treatment of high-grade AVMs (Grades III–V), or so-called large/giant AVMs, has proven to be a greater challenge for SRS,[3,5,10,15] and treatment in this group of patients remains controversial.[7,22,35] However, recent reports indicate that the hemorrhage rate of large AVMs is higher than previously believed.[2,29] It is also to be noted that this patient group includes many young patients with a high risk of bleeding throughout their lives, and the consequences of a large AVM hemorrhage are usually catastrophic. In this series, the treatment indication in each patient was carefully discussed and determined in individual cases.

In our series, 13 patients (39.3%) in the SRS/HSRT group had complete or > 90% obliteration of the AVM, which was relatively suboptimal. However, given the fact that most patients in this group had large AVMs and approximately half of the entire group (48%) are still under-

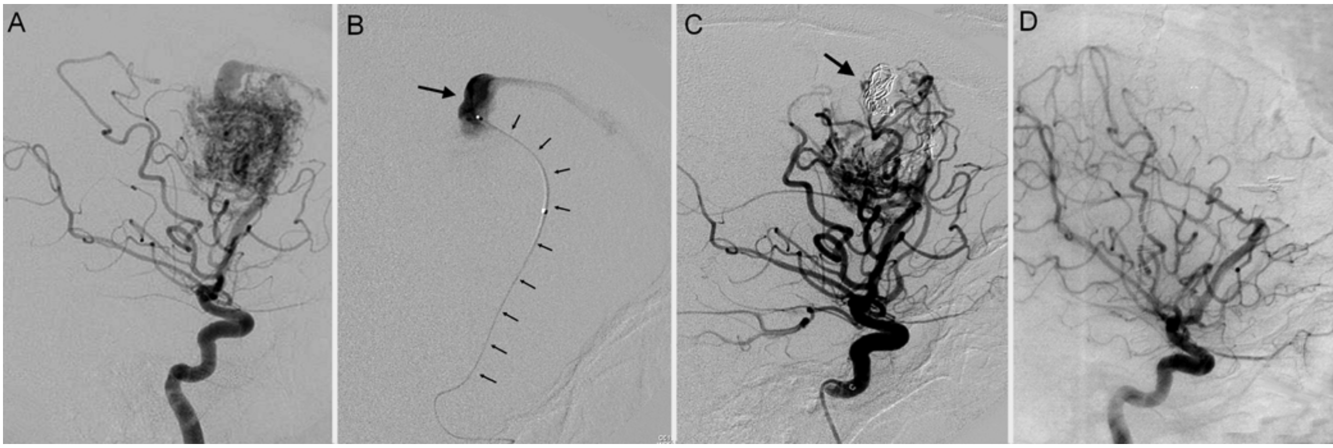

Fig. 2. **A:** An internal carotid artery (ICA) angiogram, lateral projection, demonstrating a right frontal AVM (Spetzler-Martin Grade II). A dilated venous component of AVF is observed. **B:** A superselective angiogram, lateral projection, showing a large high-flow fistula. A microcatheter navigated through abnormally dilated single feeder *(small arrows)* goes directly into the dilated venous component *(arrow)*. **C:** A postembolization ICA angiogram, lateral view, demonstrating an embolized fistulous component with GDC coils followed by NBCA *(arrow)*. A partial embolization of the nidus component was also performed. **D:** A postoperative ICA angiogram, lateral view, revealing complete removal of the AVM.

going treatment, we believe that the angiographic result in this group was still comparable to other studies of large AVMs treated by SRS.[8,20]

Endovascular Technique for Embolization of AVMs With High-Flow AVF

Recently, the technology of intracranial catheter navigation has dramatically improved due to rapid advances in microcatheter systems. In the past, flow-guided microcatheters, through which we could not deploy detachable coils, were often the only means of reaching distally located fistula sites. Today, the latest microcatheter systems allow us to use the over-the-wire technique and access distal branches so that we can deploy detachable coil materials into the AVF. Furthermore, the presence of an array of embolic materials such as coils and liquid embolic agents has made the endovascular procedure more controllable and predictable.

Unlike the technically challenging maneuvers involved in occluding a large nidus with multiple feeders and drainers, embolizing a fistulous component is relatively simple. The most important aspect is to avoid untoward migration of embolic material into draining veins or dural sinuses. Therefore, we seldom used liquid embolic agents alone to treat large high-flow AVFs. Instead, GDCs or Liquid Coils were primarily used to achieve the gradual reduction of blood flow by multistep delivery of different embolic materials.

After achieving sufficient reduction of flow rate, total occlusion was completed by injecting liquid materials such as NBCA or Onyx (Fig. 2). Occasionally, if the feeders were extremely tortuous and coil deployment was considered to be difficult, a microballoon system was placed proximal to the tip of the microcatheter to control the flow rate while delivering liquid embolic agents via the microcatheter (Fig. 3).

Risk of Endovascular Procedure as a Part of Multimodality Treatment for AVMs

A discussion of the anticipated benefit of preoperative embolization compared with the risks needs to take place before treatment. Intracranial embolization still

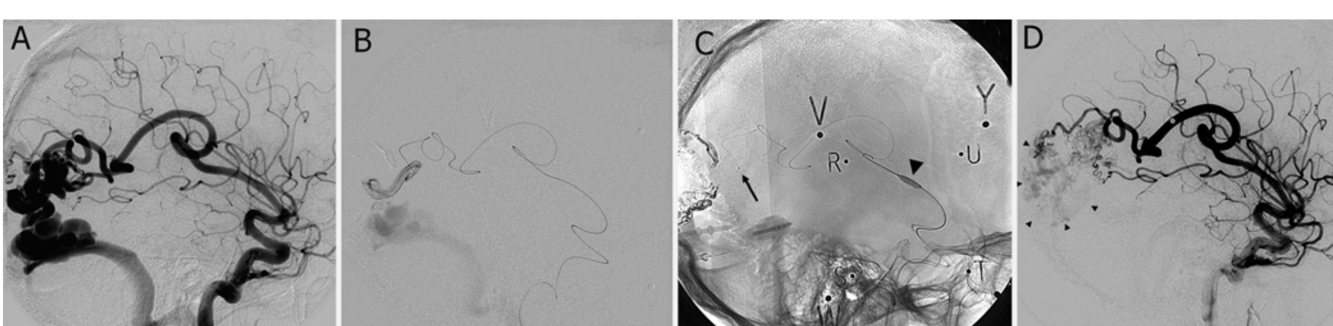

Fig. 3. **A:** An ICA angiogram, lateral view, demonstrating a left occipital AVM (Spetzler-Martin Grade III). **B:** A superselective angiogram, lateral view, obtained through the advanced microcatheter. **C:** A balloon system (Hyperglide 4 × 10) *(arrowhead)* was used to control the blood flow rate at the proximal side of the feeding artery (*arrow*; tip of the microcatheter). Onyx-34 was injected to embolize this fistulous component. **D:** A postembolization ICA angiogram, lateral view, revealing an obliterated fistulous component and significantly reduced arteriovenous shunting.

carries a risk of morbidity and death. Risks of complications leading to long-term morbidity range from 2 to 20%.[11,21,34] Taylor et al.[30] have reported their series of 201 patients treated with embolization prior to resection: death occurred in 2% and permanent neurological deficits developed in 9%. They emphasized that the complication rate of the endovascular procedure itself should be carefully evaluated prior to the treatment because the number is not negligible.

In our series, 3 patients (4.1%) had endovascular complications leading to morbidity, and 1 patient (2.5%) died. Although most of the patients (3 of 4) with technical morbidity experienced a relatively mild neurological deficit, the incidence of the complication is far from negligible and should be carefully considered.

Of particular note is the fact that there was no technical complication directly related to the fistula embolization, whereas all the endovascular complications were related to other procedures such as nidus embolization. Given the benefit from the procedure and its relatively safe technique, the importance of the fistula treatment should be emphasized to minimize the endovascular complication and to maximize the treatment effect when a multimodality therapy is used to treat brain AVMs.

Conclusions

Seventy-four patients with AVMs and associated high-flow AVFs underwent endovascular embolization followed by radiation therapy or surgical treatment. Most patients (77%) had lesions classified as Spetzler and Martin Grade III or higher. In the resection group, a total resection was achieved in each patient without a major perioperative hemorrhagic event. In the SRS/HSRT group, 39.3% of the patients had a complete or > 90% occlusion, and 6.1% had an incomplete obliteration. Patients undergoing SRS/HSRT tended to have larger lesions than individuals in the surgical group.

Fistula occlusion was prioritized in every endovascular procedure and nidus occlusion was also undertaken as a supportive therapy in light of the subsequent surgical or radiosurgical treatment. There was no technical complication directly related to the AVF embolization, whereas all the endovascular complications associated with long-term morbidity were related to other procedures such as nidus embolization. The importance of the fistula treatment should be emphasized to minimize endovascular complications and to maximize the treatment effect when a multimodality therapy is used to treat brain AVMs.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

References

1. al-Rodhan NR, Sundt TM Jr, Piepgras DG, Nichols DA, Rufenacht D, Stevens LN: Occlusive hyperemia: a theory for the hemodynamic complications following resection of intracerebral arteriovenous malformations. **J Neurosurg 78:** 167–175, 1993
2. Chang SD, Marcellus ML, Marks MP, Levy RP, Do HM, Steinberg GK: Multimodality treatment of giant intracranial arteriovenous malformations. **Neurosurgery 53:**1–13, 2003
3. Colombo F, Benedetti A, Pozza F, Marchetti C, Chierego G: Linear accelerator radiosurgery of cerebral arteriovenous malformations. **Neurosurgery 24:**833–840, 1989
4. Drake CG: Cerebral arteriovenous malformations: considerations for and experience with surgical treatment in 166 cases. **Clin Neurosurg 26:**145–208, 1979
5. Duma CM, Lunsford LD, Kondziolka D, Bissonette DJ, Somaza S, Flickinger JC: Radiosurgery for vascular malformations of the brain stem. **Acta Neurochir Suppl (Wien) 58:**92–97, 1993
6. Elisevich K, Redekop G, Munoz D, Fisher B, Wiese K, Drake C: Neuropathology of intracranial arteriovenous malformations following conventional radiation therapy. **Stereotact Funct Neurosurg 63:**250–254, 1994
7. Friedlander RM: Clinical practice. Arteriovenous malformations of the brain. **N Engl J Med 356:**2704–2712, 2007
8. Friedman WA, Bova FJ, Bollampally S, Bradshaw P: Analysis of factors predictive of success or complications in arteriovenous malformation radiosurgery. **Neurosurgery 52:**296–298, 2003
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 85:**19–28, 1996
10. Guo WY, Wikholm G, Karlsson B, Lindquist C, Svendsen P, Ericson K: Combined embolization and gamma knife radiosurgery for cerebral arteriovenous malformations. **Acta Radiol 34:**600–606, 1993
11. Haw CS, terBrugge K, Willinsky R, Tomlinson G: Complications of embolization of arteriovenous malformations of the brain. **J Neurosurg 104:**226–232, 2006
12. Houdart E, Gobin YP, Casasco A, Aymard A, Herbreteau D, Merland JJ: A proposed angiographic classification of intracranial arteriovenous fistulae and malformations. **Neuroradiology 35:**381–385, 1993
13. Jahan R, Solberg TD, Lee D, Medin P, Tateshima S, De Salles A, et al: An arteriovenous malformation model for stereotactic radiosurgery research. **Neurosurgery 61:**152–159, 2007
14. Jennett B, Bond M: Assessment of outcome after severe brain damage. **Lancet 1:**480–484, 1975
15. Kondziolka D, Lunsford LD, Kanal E, Talagala L: Stereotactic magnetic resonance angiography for targeting in arteriovenous malformation radiosurgery. **Neurosurgery 35:**585–591, 1994
16. Lasjaunias P, Manelfe C, Chiu M: Angiographic architecture of intracranial vascular malformations and fistulas—pretherapeutic aspects. **Neurosurg Rev 9:**253–263, 1986
17. Lawton MT, Hamilton MG, Spetzler RF: Multimodality treatment of deep arteriovenous malformations: thalamus, basal ganglia, and brain stem. **Neurosurgery 37:**29–36, 1995
18. Luessenhop AJ, Rosa L: Cerebral arteriovenous malformations. Indications for and results of surgery, and the role of intravascular techniques. **J Neurosurg 60:**14–22, 1984
19. Meder JF, Oppenheim C, Blustajn J, Nataf F, Merienne L, Lefkoupolos D, et al: Cerebral arteriovenous malformations: the value of radiologic parameters in predicting response to radiosurgery. **AJNR Am J Neuroradiol 18:**1473–1483, 1997
20. Miyawaki L, Dowd C, Wara W, Goldsmith B, Albright N, Gutin P, et al: Five year results of LINAC radiosurgery for arteriovenous malformations: outcome for large AVMS. **Int J Radiat Oncol Biol Phys 44:**1089–1106, 1999
21. Nakstad PH, Nornes H: Superselective angiography, embolisation and surgery in treatment of arteriovenous malformations of the brain. **Neuroradiology 36:**410–413, 1994
22. Ogilvy CS, Stieg PE, Awad I, Brown RD Jr, Kondziolka D, Rosenwasser R, et al: AHA Scientific Statement: 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. **Stroke 32:**1458–1471, 2001
23. 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 103:**2644–2657, 2001
24. Petereit D, Mehta M, Turski P, Levin A, Strother C, Mistretta C, et al: Treatment of arteriovenous malformations with stereotactic radiosurgery employing both magnetic resonance angiography and standard angiography as a database. **Int J Radiat Oncol Biol Phys 25:**309–313, 1993
25. Richling B, Killer M, Al-Schameri AR, Ritter L, Agic R, Krenn M: Therapy of brain arteriovenous malformations: multimodality treatment from a balanced standpoint. **Neurosurgery 59 (5 Suppl 3):**S148–S157, 2006
26. Schneider BF, Eberhard DA, Steiner LE: Histopathology of arteriovenous malformations after gamma knife radiosurgery. **J Neurosurg 87:**352–357, 1997
27. Spetzler RF, Martin NA: A proposed grading system for arteriovenous malformations. **J Neurosurg 65:**476–483, 1986
28. Spetzler RF, Martin NA, Carter LP, Flom RA, Raudzens PA, Wilkinson E: Surgical management of large AVM's by staged embolization and operative excision. **J Neurosurg 67:**17–28, 1987
29. 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 33:**1220–1224, 2002
30. Taylor CL, Dutton K, Rappard G, Pride GL, Replogle R, Purdy PD, et al: Complications of preoperative embolization of cerebral arteriovenous malformations. **J Neurosurg 100:** 810–812, 2004
31. Valavanis A, Yaşargil MG: The endovascular treatment of brain arteriovenous malformations. **Adv Tech Stand Neurosurg 24:**131–214, 1998
32. 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 75:**856–864, 1991
33. Vinuela F, Fox AJ, Debrun G, Pelz D: Preembolization superselective angiography: role in the treatment of brain arteriovenous malformations with isobutyl-2 cyanoacrylate. **AJNR Am J Neuroradiol 5:**765–769, 1984
34. Wallace RC, Flom RA, Khayata MH, Dean BL, McKenzie J, Rand JC, et al: The safety and effectiveness of brain arteriovenous malformation embolization using acrylic and particles: the experiences of a single institution. **Neurosurgery 37:**606–608, 1995
35. Wedderburn CJ, van Beijnum J, Bhattacharya JJ, Counsell CE, Papanastassiou V, Ritchie V, et al: Outcome after interventional or conservative management of unruptured brain arteriovenous malformations: a prospective, population-based cohort study. **Lancet Neurol 7:**223–230, 2008
36. Willinsky R, Lasjaunias P, Terbrugge K, Pruvost P: Brain arteriovenous malformations: analysis of the angio-architecture in relationship to hemorrhage (based on 152 patients explored and/or treated at the hopital de Bicêtre between 1981 and 1986). **J Neuroradiol 15:**225–237, 1988
37. Wilson CB, Hieshima G: Occlusive hyperemia: a new way to think about an old problem. **J Neurosurg 78:**165–166, 1993

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