

Management of Supratentorial Arteriovenous Malformations

Andrei F. Joaquim, M.D., and Evandro de Oliveira, M.D., Ph.D.

Management of patients with arteriovenous malformations (AVMs) remains one of the most difficult problems in neurosurgery. Treatment of AVMs requires the efforts of a multidisciplinary team, including the vascular neurosurgeon, the interventional neuroradiologist, and the radiosurgery group. Other professionals are also essential to complete the team, such as a neurologist, psychologist, neurointensivist, and neuroanesthesiologist. The ultimate treatment goal is to achieve better results than the natural history of the pathology.⁴

The main therapy for most AVMs is microsurgical resection. The challenge of AVM surgery should be reserved for major referral centers to improve clinical results because only a few neurosurgeons have the most experience in treating this pathology, which requires endovascular and radiosurgery techniques. With the development of microsurgical techniques, anatomic knowledge, and advances in neuroimaging, neuroanesthesia, and neuromonitoring, the surgical treatment of these lesions became possible.

The AVM-related risk of hemorrhage is generally assumed to be 2% to 4% per year, with a mortality rate of approximately 1% to 1.5% per year. Approximately 30% to 40% of these patients will bleed, and 10% to 15% of them will die in a 10-year period. This assumed risk is based largely on the work of Ondra et al.⁶ who followed 262 patients over a 33-year period in Finland.

The treatment goal is to achieve AVM resection without morbidity. A multidisciplinary decision-making process is the best way to offer patients better results with less morbidity. In addition to the AVM grade, the patient's occupation, age, and clinical status should be carefully considered before surgical treatment. Then, after a neuroteam decision, surgical planning should consider anatomic structures from different angles and approaches in a three-dimensional way. The fine and delicate microsurgical techniques necessary to deal with the fragile vessels of an AVM can only be obtained with diligent work with laboratory animals and surgical experience. The importance of microscopes, microsurgical instruments

and an equipped intensive care unit cannot be overstated. A team of well-trained neuroanesthetists, intensive care staff, and specialized nurses is also extremely necessary.

PATIENTS AND METHODS

We present the senior author's (EO) experience of supratentorial AVMs treated with surgical resection combined with or without adjunct treatment modalities (interventional neuroradiology and radiosurgery). The surgical results of a total of 505 patients with supratentorial AVMs in a 22-year period (1984–2006) are presented.

A modified Spetzler and Martin classification³ was used to subdivide grade III to augment the surgical resection rate (IIIA, large cortical; IIIB, small limbic lobe or callosal; and IIIC, small central core⁸ or insular block AVM, constituted externally by the insular cortex, internally by the basal ganglia and the thalamus, and with the internal capsule within). Because AVMs of the same grade can offer different difficulties for the surgeon according to their localization and size, many subgroups can be created, although the most important characteristics when considering a surgical procedure, in our opinion, are the localization and size of the AVMs.

The general treatment strategy followed was surgery for grades I and II, embolization plus surgery or radiosurgery for grade IIIA, surgery for grade IIIB, radiosurgery for grade IIIC, and conservative treatment for grades IV and V, with standard surgery for selected cases.

To evaluate surgical results, we classified patient outcome as (1) good (no additional neurological deficit), (2) fair (minor neurological deficit: cranial nerve deficits, mild aphasia, or mild ataxia), (3) poor (major neurological deficit: hemiparesis, increased aphasia, or homonymous hemianopsia), and (4) death.

General Considerations

Although AVMs are lesions with abnormal vasculature, the vessels involved in the arterial supply and venous drainage usually follow a predictable pattern. Therefore, detailed anatomic knowledge gives the surgeon the opportunity to understand the radiological images in a three-dimensional picture, choosing the most adequate route to approach the lesion.

Computed tomography, magnetic resonance imaging, and digital subtraction angiography are essential to diagnose, localize, and decide the best treatment option. According to the radiological examinations, the surgical strategy will be planned and the AVM will be classified. Angiography plays the most important role in the complementary studies. Understanding the normal vasculature as seen on the angiogram is essential for the surgeon to evaluate the arterial supply and venous drainage of an AVM to plan the best surgical approach and anticipate all the possible complications and difficulties of the case.⁴

The craniotomy must be large enough to expose all the AVM as well as normal brain around its borders, identifying cisterns and fissures that allow the surgeon to identify the best route to the lesion. Following the patterns of the normal anatomy of the cisterns, fissures, vessels, sulci, and gyri, the surgeon can access the lesion with less difficulty and perform the surgical resection in a more rational way. Some principles are extremely important: a clean operative field with minimal or no bleeding, opening of the cisterns and fissures, no damage to normal brain tissues and vessels, and minimal brain retraction. Preserving the arachnoid plane is necessary to decrease the risk of entering the nidus of the AVM before an adequate exposure of the lesion, which can result in serious bleeding and damage to the normal tissue.¹⁰

Saccular aneurysms associated with AVMs are not uncommon.¹ They generally should be treated first, and then AVM resection is performed. When the aneurysm occurs in the main feeders, we avoid preoperative embolization because it can increase the pressure inside the feeding vessel and the risk of aneurysm bleeding is higher.

Bleeding can be controlled with bipolar cauterization and saline irrigation. The blood fills the subarachnoid and subpial spaces, overshadowing references points for the surgeon.^{4,5}

Ideally, all the vessels should be exposed, achieving proximal control. Then, the vessels that belong to the AVM can be obliterated very close to the lesion, avoiding the coagulation of the en passage arteries that go to normal areas of the brain. The deep feeders are the more difficult to coagulate. These vessels are usually small and have a high flow and fragile walls that are difficult to obliterate. After coagulation of the afferent vessels, the arteriovenous shunt is interrupted. The last step before resection is cauterization of the drainage veins.^{4,5}

Good bipolar cauterization is the key to obliterating the small and fragile AVM vessels. Laser coagulation is not superior to it. Temporary miniclips can be useful sometimes, although we rarely use aneurysm clips to obliterate vessels.^{4,5}

In selected cases, as discussed below, preoperative embolization can be performed. Embolization of the afferent artery does not help the surgeon. The nidus should be obliterated, starting with occlusion of the small feeders.⁵ If a large vessel is obliterated, the flow is averted to the smaller vessels,

making the control of bleeding during surgery much more difficult. After the obliteration of an AVM, the pressure inside the vessels close to it increases abruptly. Because of the lack of autoregulation, brain swelling may occur, resulting in the breakthrough phenomenon.⁹ With previous embolization, the AVM flow becomes lower and so the vessel pressure close to the lesion grows. In our opinion, the surgical resection of the AVM in this case is less risky because it does not cause abrupt alterations in the perfusion of the normal brain areas.¹

Radiosurgery is especially useful for small lesions (<3 cm) located in deep-seated or in eloquent areas of the brain.^{2,8} The drawback of this treatment is that requires several years to work, leaving the patient untreated during this period. Also, the total obliteration rate with this modality is not superior to that achieved with surgical resection.⁷

After surgery, we generally maintain a mean blood pressure of approximately 60 to 70 mm Hg for 3 days. We prefer to extubate our patients in the operating room to avoid prolonged mechanical ventilation and sedation.

Treatment strategies for AVMs according to the modified Spetzler-Martin classification are summarized in *Table 7.1*.^{3,4}

RESULTS

A total of 550 patients were surgically treated. We present the surgical results of each grade of AVM. The distribution of the patients according to modified Spetzler-Martin classification is given in *Table 7.2*.

Grade I and II AVMs

All lesions in these groups should be operated on. For elderly patients with limited life expectancy or who are in poor clinical condition, surgery can be contraindicated. For patients who refuse surgical treatment, radiosurgery can be an alternative (*Table 7.3*).

Grade III, IV, and V AVMs

For these patients, the best treatment should be individualized and decided on by the neurovascular team.

TABLE 7.1. Treatment strategies of arteriovenous malformations according to modified Spetzler-Martin classification

Grade	Treatment	Option for Selected Patients
I	Surgery	Embolization + radiosurgery
II	Surgery	Embolization + radiosurgery
IIIA	Embolization + surgery	Embolization + radiosurgery
IIIB	Surgery	Radiosurgery
IIIC	Radiosurgery	Embolization + surgery
IV	Conservative	Embolization + surgery
V	Conservative	Embolization + surgery

TABLE 7.2. Distribution of the patients operated on between 1984 and 2006 according to grade

Grade	No.	%
I	65	12.8
II	143	28.3
IIIA	48	9.5
IIIB	104	20.5
IIIC	56	11.0
IV	62	12.2
V	27	5.3
Total	505	100

TABLE 7.3. Surgical results of grade I and II arteriovenous malformations

Grade	Results	No.	%
I	Good	65	100
II	Good	140	97.9
	Fair	3	2.1

Grade IIIA AVMs (Fig. 7.1)

Most AVMs in this group are superficial, of medium size, larger than 3 cm but smaller than 6 cm, and in close proximity to eloquent areas. Rarely, they can be large (>6 cm) and located in noneloquent areas, with superficial drainage. Deep, medium-sized AVMs without deep venous drainage are extremely rare and are usually found in the lateral hemispheric surface. Unless there are clinical limitations, we manage patients with grade IIIA AVMs with preoperative embolization followed by surgical resection. Radiosurgery can be performed after embolization if surgery is contraindicated.

Grade IIIB AVMs

These are AVMs of the limbic lobe, corpus callosum, paraolfactory gyrus, and paraterminal gyrus. They are smaller than 3 cm and usually located in the mesial aspect of the temporal lobe, with deep drainage, and in eloquent areas. The surgical approach is easier than for those AVMs located in deep-seated areas because the former are close to the cisterns, which provide natural corridors for a surgical approach with less morbidity. This group of lesions can be treated surgically. As an alternative, radiosurgery can also be performed.

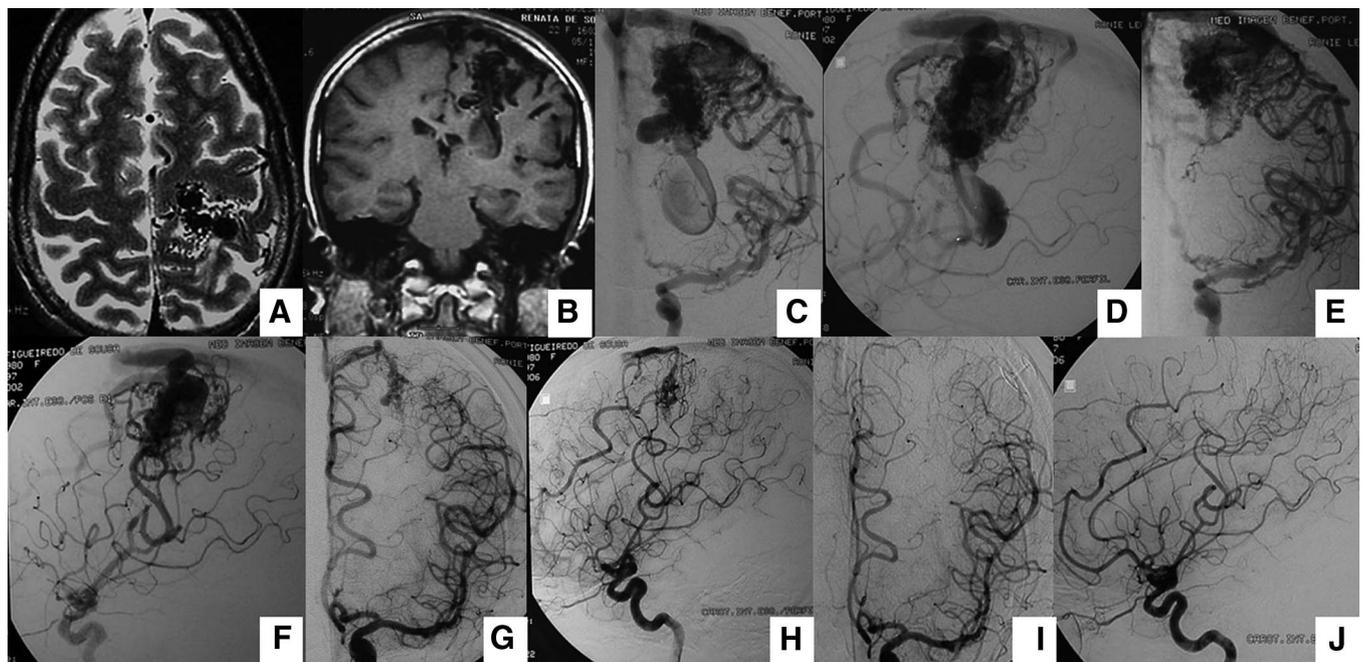


FIGURE 7.1. Management of a grade IIIA arteriovenous malformation (AVM) located in the left pre- and postcentral gyri. Axial (A) and coronal (B) magnetic resonance imaging showing the AVM in the left pre- and postcentral gyri. Also note the presence of a large venous aneurysm. Anteroposterior (C) and lateral (D) internal left side carotid angiograms showing an AVM supplied by branches of the middle cerebral artery and the venous aneurysm. The patient was initially treated with embolization (control angiograms shown in E and F), followed by radiosurgery after 3 months. Anteroposterior (G) and lateral (H) angiograms 3 years after radiosurgery treatment. Finally, the patient underwent surgical resection of residual AVM. I and J, postoperative angiogram showing complete AVM resection.

TABLE 7.4. Surgical results of grade IIIA, IIIB, and IIIC arteriovenous malformations

Grade	Results	No.	%
IIIA (n = 48)	Good	39	81.2
	Fair	5	10.4
	Poor	3	6.2
	Death	1	2.0
IIIB (n = 104)	Good	90	86.5
	Fair	12	11.5
	Poor	1	0.9
	Death	1	0.9
IIIC (n = 56)	Good	49	87.5
	Fair	6	10.7
	Poor	0	0
	Death	1	1.7

Grade IIIC AVMs

These are small AVMs located in the insular block, an eloquent area with deep venous drainage. They are often feeders from the lenticulostriate arteries or perforating vessels from the carotid, posterior cerebral, posterior communicating, anterior choroidal, and posteromedial choroidal arteries. Sur-

gery is either difficult or prohibitive, and the treatment of choice is usually radiosurgery. However, some small AVMs (usually <2 cm), deep-seated, located surrounding the insular block sparing its central region, the thalamus and the internal capsule fibers (generally in the head of the caudate nucleus, in the pulvinar, in the floor of the lateral ventricle, or outside the lenticular nucleus, in the insular cortex) can be surgically excised with minimal morbidity and similar results to grade IIIB.

The surgical results of grade III AVMs are shown in *Table 7.4*.

Grade IV AVMs

These AVMs are preferably managed with conservative treatment. The risk of causing a permanent deficit with surgical treatment in this group is very high. For patients with progressive neurological deficits, repeated hemorrhagic event, or with increased intracranial pressure syndrome, surgery after endovascular embolization can be considered.

Grade V AVMs (Fig. 7.2)

Similar to grade IV AVMs, we usually recommend conservative treatment for these lesions. However, as in patients with grade IV AVMs with progressive neurological deficits, repeated hemorrhagic events, or with increased in-

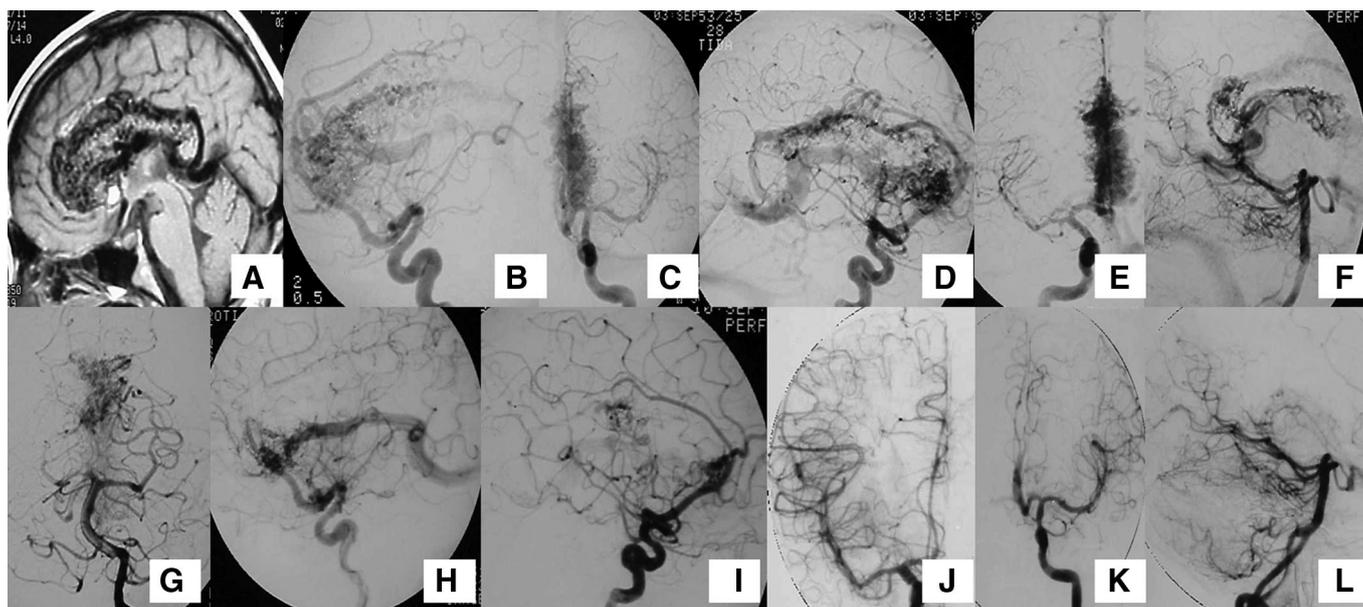


FIGURE 7.2. A Grade V arteriovenous malformation (AVM) located in the medial brain surface supplied by branches of both anterior and posterior cerebral arteries, treated with embolization followed by 2 surgical procedures. Sagittal magnetic resonance imaging of the AVM located in the medial brain surface in the region of the lamina terminalis, cingulate gyrus, and corpus callosum (A). Lateral (B) and anteroposterior (C) left- (D) and right- (E) side internal carotid digital angiograms showing the AVM in the medial brain surface supplied by branches of both anterior cerebral arteries. Lateral (F) and anteroposterior (G) angiograms of the posterior cerebral circulation showing the branches of the posterior cerebral arteries supplying the AVM. Left- (H) and right- (I) side internal carotid digital angiogram after embolization. Left- (J) and right- (K) side internal carotid and lateral posterior cerebral (L) angiograms after 2 surgical procedures showing complete AVM resection.

TABLE 7.5. Surgical results of grade IV and V arteriovenous malformations

Grade	Results	No.	%
IV (n = 62)	Good	48	77.4
	Fair	10	16.1
	Poor	2	3.2
	Death	2	3.2
V (n = 27)	Good	19	70.3
	Fair	6	22.2
	Poor	1	3.7
	Death	1	3.7

tracranial pressure syndrome, surgery after endovascular embolization can also be considered.

The surgical results of grade IV and V AVMs are shown in *Table 7.5*.

Complications

Since 2003, we have been working with a neurologist specializing in cognitive disorders. In 35% of our patients, we found some cognitive deficits, most of them not detected on a general neurological examination. Motor deficit is another common complication: we had transient deficits in 166 patients (31%) and permanent deficits in 63 patients (8%).

Residual AVMs were found in 30 patients (6%). Although we do not use intraoperative angiography, because, in our opinion, it does not change surgical management because of false-negative findings, we routinely perform a postoperative digital subtraction angiography before patient discharge. If any residual AVM is found, a second procedure is planned to achieve total resection.

Intraparenchymal cerebral hemorrhage was a severe complication found in 15 patients (3%), generally associated with poor clinical outcome. Among the six obits in this series, three deaths were secondary to intraparenchymal hemorrhage, one death was caused by ventricular bleeding and acute hydrocephalus, and the other three deaths occurred in

patients in poor clinical condition before surgery and were caused by bleeding during AVM embolization in the interventional neuroradiology suite.

CONCLUSION

Management of AVMs remains a great challenge. The interventional radiologist and radiosurgeon must work as a team led by the vascular neurosurgeon. The best treatment modality is decided for each case based on a team approach.

For the neurosurgeon, detailed anatomic knowledge of the brain structures and a refined microsurgical technique are mandatory. Experience can be achieved only in referral centers with a great number of cases, thus achieving acceptable surgical results.

Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

REFERENCES

1. Crawford PM, West CR, Chadwick DW, Shaw MD: Arteriovenous malformations of the brain: Natural history in unoperated patients. *J Neurol Neurosurg Psychiatry* 49:1–10, 1986.
2. Friedman WA, Bova FJ: Linear accelerator radiosurgery for arteriovenous malformations. *J Neurosurg* 77:832–841, 1992.
3. Mattos JP, de Oliveira E, Tedeschi H, Marengo HA: Treatment strategy for grade III arteriovenous malformation. *Contemp Neurosurg* 28:1–7, 2006.
4. Oliveira E, Tedeschi H, Raso J: Comprehensive management of arteriovenous malformations. *Neurol Res* 20:673–683, 1998.
5. Oliveira E, Tedeschi H, Siqueira MG, Ono M, Rhoton AL Jr, Peace D: Anatomic principles of cerebrovascular surgery for arteriovenous malformations. *Clin Neurosurg* 41:364–380, 1994.
6. Ondra SL, Troupp H, George ED, Schwab K: The natural history of symptomatic arteriovenous malformations of the brain: A 24-year follow-up assessment. *J Neurosurg* 73:387–391, 1990.
7. Pikus HJ, Beach ML, Harbaugh RE: Microsurgical treatment of arteriovenous malformations: Analysis and comparison with stereotactic radiosurgery. *J Neurosurg* 88:641–646, 1998.
8. Rhoton AL Jr: Cranial anatomy and surgical approaches. *Neurosurgery* 1–746, 2003.
9. Spetzler RF, Wilson CB, Weinstein P, Mehdorn M, Townsend J, Telles D: Normal perfusion pressure breakthrough theory. *Clin Neurosurg* 25:651–672, 1978.
10. Yasargil MG: *Microneurosurgery*. Stuttgart, Georg Thieme, 1984, vol. II, pp 232–295.