Arteriovenous Malformations of the Anterior Fossa

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C.D. Gandhi, C.J. Prestigiacomo (eds.), *Cerebrovascular and Endovascular Neurosurgery*, https://doi.org/10.1007/978-3-319-65206-1_15

Checklist: Hemorrhagic Complication Management in AVM Microsurgery

Equipment needed	Procedural steps
Surgical • Emergency suction • Bipolar electrocautery • Vessel microclips, aneurysm clips • Irrigation • Thrombin-soaked cotton pads • Hemostatic product (Gelfoam, Floseal, etc.) • Microscope • Retractors • Rhoton dissectors • EVD, if needed • Drill, to expand craniotomy if needed for decompression Anesthesia • Blood pressure and ICP monitoring • End-tidal CO_2 monitoring and manipulation via respiratory rate • Blood transfusion • Crash cart Pharmacologic • Mannitol/Hypertonic Saline (23.4%) • Anticonvulsant • Antihypertensive/vasopressors • Paralytic Neurointerventionalist • Femoral artery access • Guide or diagnostic catheter • Guide wire • Microcatheter, microwire • Balloon microcatheter • NBCA glue	Identification • Visible hemorrhage • Hemodynamic changes, ICP changes • Identify source: arterial, nidus, venous occlusion Initiate and Engage • Alert entire team, communicate clearly • Backup OR tech, nursing, anesthesia availability • Maintain systolic blood pressure <100 mmHg with antihypertensives • End-tidal CO_2 <30 mmHg with hyperventilation • Open EVD if available • Osmotic agents if ICP >20 mmHg • Maintain cerebral perfusion in sustained bleeding with transfusion, vasopressors • Inform neurointerventionalist • Prepare crash cart Repair • Maximize visibility with suction, irrigation, retraction, head position • Cauterization of visible bleeder with irrigating bipolar • Application of microclips for small perforating arteries or aneurysm clips for large feeding arteries • Obtain proximal exposure of thin, friable perforators that tend to retract with cauterization • Nidus or small vessel bleeding: thrombin-soaked cotton products or hemostatic agents • Avoid packing of arterial bleeding which can result in hematoma formation out of visible field • Venous occlusion: rapid resection of remainder of nidus and coagulation of remaining arterial feeders If Needed: • EVD placement • Expand craniotomy for further boney decompression • Endovascular intervention (balloon tamponade, parent vessel sacrifice, glue embolization)

Complication Avoidance Flowchart

Complication	Cause	Management	Prevention
Intraoperative hemorrhage	1. Nidus penetration 2. Venous injury 3. Residual AVM 4. Retraction injury	1. Hemostasis with bipolar cautery, clip placement, gentle tamponade, or Gelfoam 2. Reevaluation of the true plane between the malformation and brain and widening the diameter of the dissection 3. Use cell saver and blood transfusion when severe blood loss is anticipated 4. Identify the residual AVM and further microsurgical resection	1. Judicious use of preoperative embolization 2. DSA and CTA localization of residual nidus, deep perforators, and venous drainage 3. Meticulous dissection technique 4. Control of deep feeding arteries near the end of dissection 5. Preserve venous drainage until major sources of arterial flow can be isolated and occluded 6. Gradual increase of blood pressure 10–15 mmHg above preoperative BP and inspection for 15 min before dural closure to identify residual AVM 7. Confirm complete resection on intraoperative angiogram or Doppler ultrasound
Normal perfusion pressure breakthrough/ occlusive hyperemia	1. Failed autoregulation of AVM feeders due to prolonged ischemia-related steal phenomenon adjacent to AVM nidus	1. Increase cerebral perfusion pressure (CPP) by EEG-burst suppressive anesthesia with phenobarbital 2. Systemic arterial BP reduction (systolic 80–90 mmHg) with sodium nitroprusside or nicardipine 3. Hemorrhagic brain tissue should be resected along with the AVM with absolute hemostasis 4. Pharmacological reduction of ICP 24-h post-resection	1. Staged preoperative embolization 2. Complete resection should be confirmed with intraoperative angiography

(continued)

Complication	Cause	Management	Prevention
Postoperative hemorrhage	1. Residual AVM 2. Normal perfusion pressure breakthrough	Early detection and surgical decompression ± microsurgical resection	1. Gradual increase of blood pressure 10–15 mmHg above preoperative BP & inspection for 15 min before dural closure to identify residual AVM 2. Confirm complete resection on intraoperative angiogram or Doppler ultrasound 3. Pharmacological reduction of ICP 24-h post-resection

Introduction

Cerebral arteriovenous malformations (AVMs) comprise 1.5–4% of all intracranial lesions, approximately one-tenth as often as intracranial aneurysms [1]. AVMs are the second most identifiable cause of subarachnoid hemorrhage after cerebral aneurysms, accounting for 10% of all cases of subarachnoid hemorrhage [1]. The majority (80%) of the AVMs are supratentorial distribution with 65% in the lobar and 15% in the deep location [2]. They usually occur as single lesions, but as many as 9% are multiple [2]. According to the American Stroke Association, 1 in 200–500 people have an AVM, while 50% of patients suffer intracranial hemorrhage and 25% of AVM patients experience seizures at some point in their lives. Also, 5–15% of AVM patients report severe headaches because of the increased intracranial pressure and a similar percentage of patients exhibit neurological deficits.

Modern management of cerebral AVMs is centered around a multidisciplinary approach involving endovascular embolization, microsurgical resection, and stereotactic radiosurgery, which can be used as an isolated modality or more commonly as part of a multimodality treatment plan. Typically, embolization is used as an adjunct to radiosurgery or microsurgery to eliminate the risk factors for hemorrhage or to eliminate specific compartments of the AVM, facilitating subsequent treatment. The role of embolization in Spetzler-Martin grade I–II AVMs is debatable [3]. Typically, these lesions are resected with microsurgery.

Procedural Overview

The concept of complication avoidance should be applied at pre-, intra-, and post-treatment stages in the cerebral AVM management. Detailed understanding of the angiographic and functional anatomy, familiarity with the natural history, multidisciplinary evaluation, multimodality approach, realizing patient current functional status and outcome expectations, clear risk stratification, and effective communication are vital successful management of the cerebral arteriovenous malformations.

Pretreatment Evaluation

Digital subtraction angiography (DSA) is the investigation of choice in the evaluation of cerebral arteriovenous malformations (AVMs) due to its unparalleled spatial and temporal resolution in the evaluation of angioarchitectural features, nature of the nidus (compact or diffuse), high-flow fistulous communication within the nidus, and predictive risk factors for hemorrhage such as intranidal, flow-related aneurysms, venous stenosis, single draining vein, deep venous drainage, and deep cerebral location. Multiplanar magnetic resonance imaging (MRI) is helpful in precise localization, evaluating adjacent eloquent regions and surrounding parenchymal changes such as edema, gliosis, and encephalomalacia. Thorough understanding of these findings is vital in choosing the appropriate single/multimodality treatment. Functional MRI is increasingly used in patients with AVMs centered in the eloquent locations for better delineation of the speech, motor, and cognition areas, which helps in better surgical planning and risk stratification. Be aware of the differential diagnosis such as "proliferative angiopathy" mimicking large AVM with diffuse nidus.

A multidisciplinary team of cerebrovascular microsurgeons, endovascular neurosurgeons, interventional neuroradiologists, and radiosurgeons should evaluate each brain AVM patient on a case-by-case basis and determine the optimal treatment modality or combination of modalities for the goal of complete AVM obliteration while minimizing risk. When endovascular embolization is agreed upon as a treatment modality, the role of the embolization should be clearly outlined, i.e., curative, adjuvant, or palliative.

Patient Selection Based on Natural History of Cerebral AVMs

The first step in the complication avoidance is "justification of treatment risk." If the cumulative lifetime estimate of an AVM rupture based on the natural history far exceeds the immediate risks of treatment for a patient, the treatment is justified. To make this decision, a thorough knowledge of AVM natural history is mandatory. The attempts to clearly delineate the natural history of the cerebral AVMs began decades ago and continue to pose a considerable challenge owing to a remarkable degree of heterogeneity not only in study design but also in the results.

Natural History of Ruptured AVMs

Hemorrhage is the most common presentation in up to 53% of the cerebral AVMs. Estimated risk of rebleeding after hemorrhage from cerebral AVMs is approximately 6% during the first year and 2% per year up to 20 years after the initial hemorrhage [4, 5]. The average interval between the bleeding events was reported to be 7.7 years [6]. Although the ubiquitous maxim of "previously ruptured vascular malformations have a higher risk of rerupture" among cerebrovascular neurosurgery was not favored by the original AVM studies by the Ondra et al. [6] and the Toronto group [7], these were overturned by the recent studies in the same cohort and longer follow-up [8, 9]. More recent meta-analysis by Gross et al. based on the nine natural history studies from 1986 to 2009 confirmed prior AVM hemorrhage is a significant risk factor for subsequent bleeding with a hemorrhagic risk of 4.5% (3.7–5.5%), and risk of recurrent hemorrhage in the first year after initial hemorrhage ranges from 6 to 15% [10].

Natural History of Unruptured AVMs

Average risk of hemorrhage in unruptured AVMs was estimated at 2–4% [5, 6] per year based on individual series, while a 2.2% (1.7–2.7%) risk was quoted by the meta-analysis [10]. The annual incidence of de novo epilepsy in patients with cerebral AVMs is 1–4%. Population-based study with longest follow-up of 23.7 years reported AVM-related risk of mortality and morbidity per annum at 1 and 1.7%, respectively [6]. Deferring the treatment until AVM becomes symptomatic carries a significant risk because of the mortality and morbidity rates from initial hemorrhage of approximately 17 and 40%, respectively, to a significant long-term neurological morbidity and mortality of as high as 35 and 29%, respectively [11–13].

The ARUBA trial [14] is the first study comparing medical management to surgical care on patients with unruptured cerebral AVMs and a Rankin score <2. The trial states that 30.7% of patients in the interventional arm reached the primary endpoint of death or stroke, a threefold higher rate than the medical management arm, and concluded that the medical management alone is superior over the intervention in the management of the unruptured cerebral AVMs. While the trial provides important data, it has received plenty of criticism concerning its study design and the credibility of its findings. Our institutional experience involving retrospective review of all the unruptured intracranial AVMs (n = 64) with completed treatment over a 12-year period (2003–2015) with same endpoints as ARUBA showed that risk of symptomatic stroke or death in our cohort (7.8%) was significantly lower (p = 0.004) than the "interventional arm" in the ARUBA trial. Moreover, the one-time upfront risk of 7.8% with intervention in our group was not only comparable to the 10% incidence of death or stroke reported over a short time interval of <3 years among the medically managed ARUBA population but also provides a permanent curative treatment with a 97% of angiocure.

Risk of Hemorrhage

Based on the evidence available from the natural history studies, the angioarchitectural features and their association with hemorrhagic presentation were categorized in Table 15.1.

To minimize the variability in the prediction of long-term risk of hemorrhage based on available statistics among the neurosurgical community, Kondziolka et al. [15] suggested the below formula to estimate the lifetime risk of hemorrhage in patient not treated for an AVM based on multiplicative law of probability.

Table 15.1 Angioarchitectural characteristics of cerebral AVMs related to risk of hemorrhage

Consistent risk factors	Inconsistent risk factors	Potential risk factors	Potential protective factors
• Exclusive deep venous drainage (2.4%) • Single draining vein • Venous stenosis • High mean arterial pressure in the feeding artery	• Prior hemorrhage (3.2%) • Intranidal aneurysm (1.8%) • Small nidus; <3 cm (1%) • Deep location (2.4%) • Venous stasis	• Systemic hypertension • Vertebrobasilar supply • Perforator supply • Increasing age • Smoking • Pregnancy	• Arterial stenosis • Neoangiogenesis • Venous recruitment

$$\text{Risk of hemorrhage} = 1 - (\text{Annual risk of no hemorrhage})^{\text{Expected years of remaining life}}$$

This formula assumes some degree of population homogeneity and uniformity of the AVM natural history and provides a quick means of estimating lifetime risk of hemorrhage in a clinical setting.

The formula was further simplified estimating the cumulative risk of hemorrhage based on patient's age at the time of discovery of a brain AVM, which can be predicted by using a formula [15, 16]:

$$\text{Lifetime risk (\%)} = 105 - \text{Patient age in years}$$

Younger patients have longer life expectancy and therefore higher cumulative lifetime risk of rupture. As AVM in older patients are significantly associated with several high-risk angioarchitectural features of rupture, it is important to stratify older patients based on the high-risk factors in the treatment decision analysis.

Timing of AVM Treatment

Ruptured AVMs

The timing of the AVM treatment after intracranial hemorrhage requires careful consideration. A hematoma secondary to AVM rupture of significant size or precarious location may result in mortality or permanent morbidity due to primary or secondary (edema) mass effect. Although small hematomas and occasionally large (usually lobar) hematomas can be managed nonsurgically, those that produce significant obtundation or herniation syndromes should be evacuated on emergency basis. Simultaneous removal of AVM with hematoma should be considered only when the lesion is small and located immediately adjacent to the clot cavity. Complete resection of the AVM should be confirmed with intra- or postoperative angiogram (Fig. 15.1). With an aggressive approach to AVM resection after acute hemorrhage, reversible neurological deficit can be permanent, as the surrounding parenchyma is more vulnerable to injury in this setting. Jafar et al. [17] reported only 50% of the patients without significant neurological deficit following acute AVM surgery. Heros and Samson [18] reported that control of intracranial pressure (ICP) with careful hematoma evacuation and delayed AVM treatment continue to be the preferred management for ruptured cerebral AVMs. In case of small hemorrhages or neurological deficit, a delay of several weeks may be indicated prior to AVM treatment. The interval delay in the ruptured AVM treatment allows resolution of cerebral edema, peri-hematoma inflammation, plateau of recovery from neurological deficit, liquefaction, and contraction of the hematoma with reduced mass effect on the AVM nidus leading to both improved radiological evaluation and ease of surgery (Figs. 15.2 and 15.3). Once the intracranial dynamics are normalized, proper preoperative radiographic evaluation, surgical planning, and treatment can proceed in an organized fashion. Delaying treatment for 4 weeks after initial hemorrhage subjects the patient to a low (<1%) risk of rehemorrhage [19] and may result in spontaneous thrombosis of the AVM in rare instances [20].

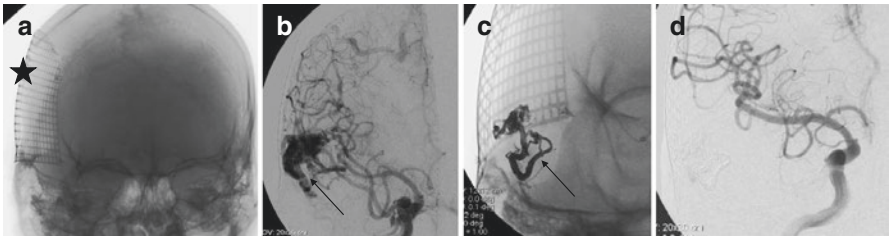

Fig. 15.1 Residual AVM following decompressive craniotomy and hematoma evacuation. Forty-eight-year-old patients with decompressive craniotomy and hematoma evacuation for spontaneous right-sided hemorrhage (**a**) in China, presented 13 years later with headaches. Based on CTA findings, catheter angiogram was performed which demonstrated right temporal AVM (**b**) just beneath the surgical site. This was successfully treated with onyx embolization (**c**) and no residual filling on the post-embolization angiogram (**d**). This highlights the importance of catheter angiogram in patients with spontaneous intracranial hemorrhage for initial or follow-up evaluation of underlying vascular abnormalities

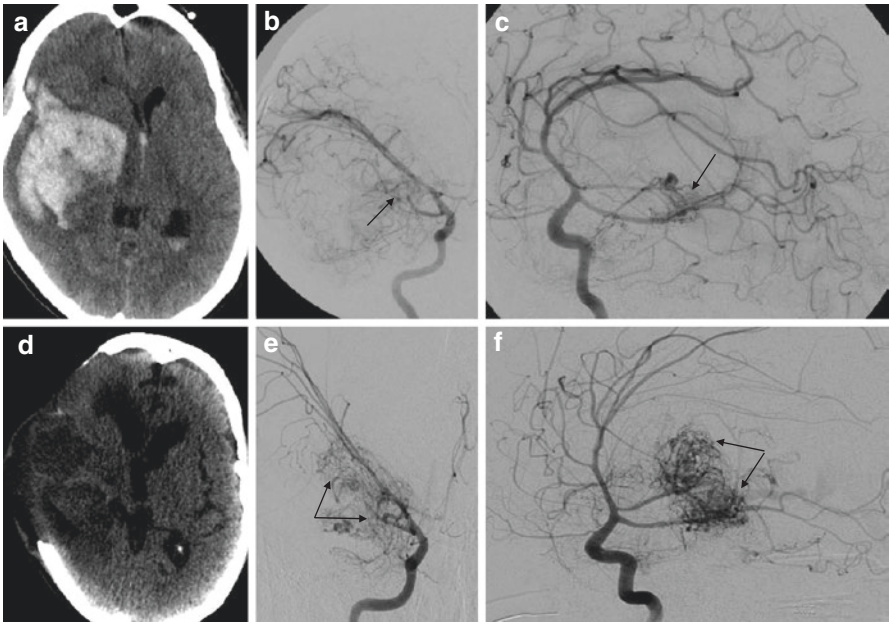

Fig. 15.2 Improved visualization of AVM following hematoma resolution. Ruptured basal ganglia AVM (**a**) with initial catheter angiogram (**b** and **c**) demonstrated significant mass effect with vascular distortion and faint visualization of small AVM nidus. Follow-up CT at 6 weeks (**d**) after decompressive craniotomy showed hematoma resolution and encephalomalacia. DSA at 6 weeks (**e** and **f**) shows marked improvement in the visualization of nidus due to reduced mass effect, which argues against embolization of ruptured AVMs in acute phase

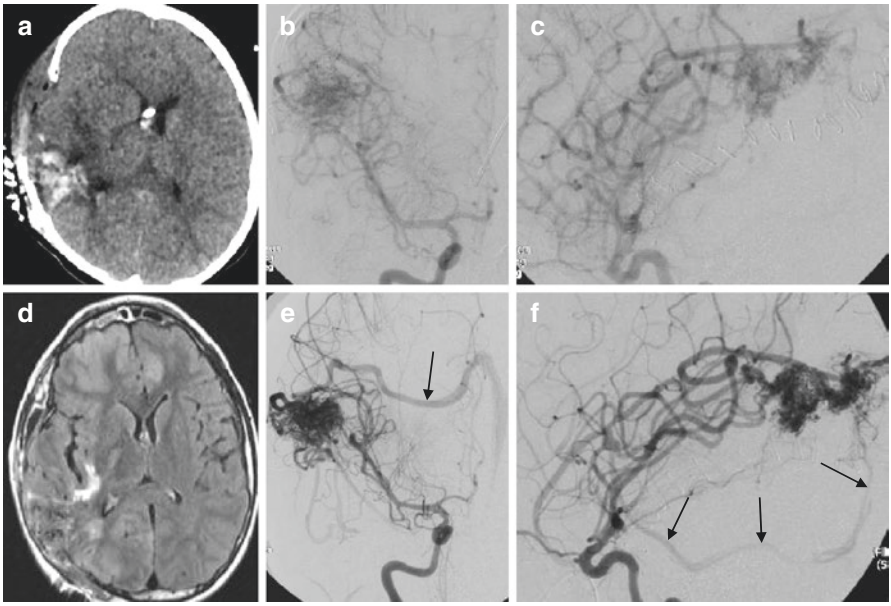

Fig. 15.3 Poor visualization of angioarchitectural features in acute phase angiogram. Ruptured right parietal AVM following decompressive craniotomy and hematoma evacuation (**a**). DSA in acute phase (**b** and **c**) shows poor opacification of nidus and nonvisualization of the draining vein. Follow-up MRI at 4 weeks (**d**) showed partial resolution of cerebral edema and mass effect. DSA at this point (**e** and **f**) shows better visualization of the AVM nidus and angioarchitectural features. Better understanding of the draining vein location minimizes the embolization risk, which again favors conservative approach in the acute phase

Unruptured AVMs

Treatment of unruptured AVMs needs careful evaluation of the complications and outcome related to the treatment against the natural history of these lesions. The angioarchitecture of the AVM should be thoroughly evaluated prior to embolization (Figs. 15.4 and 15.5) is mandatory to recognize high-risk factors flow-related or intranidal aneurysms (Fig. 15.6), venous ectasia, venous stenosis, venous hypertension and high-flow shunt with steal phenomenon.

Steal phenomenon is a known cause for progressive neurological deficit in the absence of intracranial hemorrhage or increased size of AVM. These patients benefit from more aggressive approach, especially with partial embolization. Failure to respond quickly may result in an irreversible deficit that could have been prevented. These deficits have been attributed to a steal phenomenon, as AVM theoretically recruits arterial flow away from the normal brain. Partial embolization is the preferred initial step in the management of this situation resulting in temporary amelioration of the steal symptoms and potential reduction of the post-resection perfusion problems.

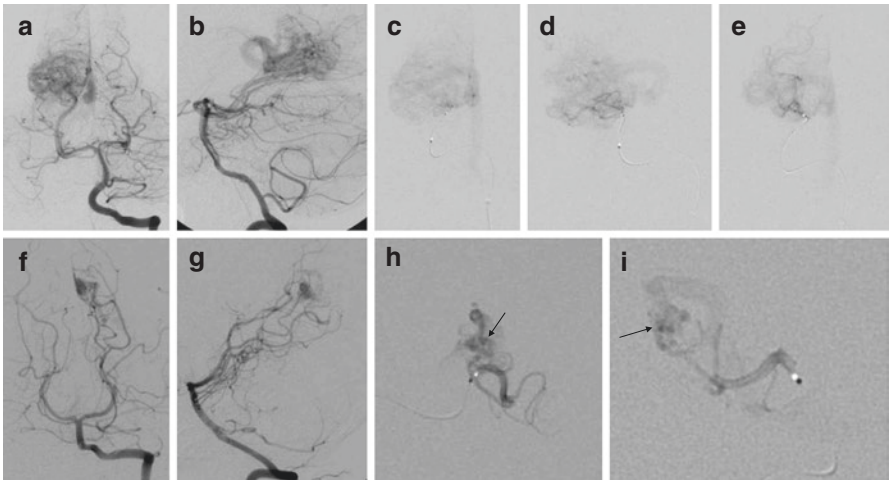

Fig. 15.4 Favorable and unfavorable angioarchitectural features of AVMs. Two different patients with unruptured occipital AVMs showing favorable and unfavorable anatomy for AVM treatment. Patient-1 (**a–e**) shows right occipital AVM with diffuse nidus (**a** and **b**). Super selective microcatheter exploration again shows diffuse nidus with intervening normal parenchymal vasculature (**c–e**), which is a high-risk factor for post-procedure neurologic deficit. In contrary, patient-2 (**f–i**) with left occipital AVM shows compact nidus, with two definite arterial feeders and draining vein, an excellent scenario for treatment. Patient-1 is currently managed conservatively while patient had stereotactic radiosurgery

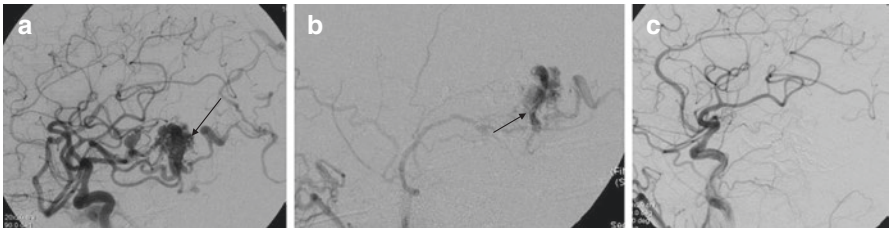

Fig. 15.5 Dural and pial contribution for temporal AVM. The angioarchitecture features of the AVM should be thoroughly evaluated prior to proceeding with embolization. In contrary to the conventional notion of pial feeders for brain AVMs, this patient with temporal AVM has both dural and pial feeders (**a**). The dural contribution from posterior division of middle meningeal artery (MMA) is better appreciated on the dedicated external carotid (ECA) angiogram (**b**). Good preoperative onyx embolization required both pial and dural approach. Good preoperative onyx embolization required both pial and dural approach (**c**)

Venous hypertension, mass effect, and ischemia caused by the high arterial flow with limited venous drainage, venous arterialization, stenosis, or occlusion are the other potential causes for progressive neurological symptoms and irreversible cerebral damage in patients with unruptured cerebral AVMs. Partial staged embolization is a preferred choice to ameliorate the risk factors associated with progressive neurological deficit or hemorrhage and convert a high-risk AVM to a more suitable microsurgical target for complete resection.

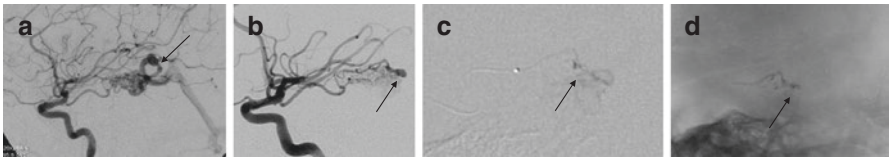

Fig. 15.6 Intranidal aneurysm—High-risk factor for hemorrhage in AVM. Ruptured thalamic AVM with intranidal aneurysm (*black arrow*) and deep venous drainage in to vein of Galen (**a** and **b**). The intranidal aneurysm being a known high-risk factor for hemorrhage was obliterated with super selective microcatheterization (**c**) and onyx embolization (**d**). Further management of this deep AVM in the eloquent region was performed with stereotactic radiosurgery

Pretreatment Functional Evaluation

As a part of complication avoidance, accurate risk analysis for treatment of individual AVMs is crucial which includes functional evaluation of the surrounding brain parenchyma. In patients with congenital AVMs, the locations of the cerebral functions may be altered through developmental plasticity. Pretreatment functional evaluation can be used to identify functionally active cortex even in unexpected locations, which helps to choose an appropriate therapeutic modality for AVM treatment. Delineation of the active tissue from the malformation allows safer placement of boundaries for radiosurgery or selection of resection planes. Information about physiologic significance can be gathered from various diagnostic tests developed to evaluate local cerebral parenchymal function and blood flow such as positron emission tomography (PET), functional magnetic resonance imaging (fMRI), magnetoencephalography, relative perfusion measurement, and invasive provocation.

Recognize the Differential Diagnosis: AVM Mimics

Cerebral Proliferative Angiopathy

Cerebral proliferative angiopathy (CPA) , previously known as “diffuse nidus type AVM” or “holohemispheric giant AVM,” is present in an estimated 2–4% of all brain AVMs [21]. Proliferative angiopathy may be confused with true cerebral AVMs and thought to represent a diffuse nidus. Seizures and disabling headaches are the most common clinical symptoms at presentation in cerebral proliferative angiopathy, occurring significantly more often than in the AVM population. Progressive neurologic deficits and transient ischemic attacks are also possible, whereas hemorrhage is exceptional [22]. The risk of hemorrhage is extremely rare at presentation; however, if a hemorrhagic episode has occurred, the risk of recurrence seems to be high compared to classic brain AVMs [22].

The most striking feature is the presence of normal-appearing neural tissue intermingled between these vascular channels, whereas perivascular gliosis is only mild, with additional capillarogenesis within the subcortical region. This implies that the

brain tissue within the "nidus" of the CPA is functional, like brain tissue found in between the abnormal vascular channels present in capillary telangiectasias. The lack of clear early venous drainage on dynamic images is the key to differentiating this disease from classic brain AVM. A multifactorial analysis by a French group evaluating angioarchitectural features in relation to hemorrhagic risk showed that the association of arterial stenoses with angiogenesis was the only factor associated with a reduced risk of hemorrhage. This typical association of proximal arterial stenoses with angioneogenesis in cerebral proliferative angiopathy explains the extremely low risk of hemorrhagic presentation in the natural history of CPA compared to classic brain AVMs. Because the pathomechanism of proliferative angiopathy is mainly due to cortical ischemia (as proved with perfusion-weighted studies), there have been reports of successful treatment with pial synangiosis (EDAS) or burr-hole therapy to enhance supply to healthy brain tissue from the external carotid artery.

Complication Avoidance and Management

Complications during the microsurgical management of cerebral AVMs are undeniable. Despite wide variability in the morbidity and mortality rates of the published microsurgical series, the microsurgical series report superior occlusion rates than the embolization. The major microsurgical series of AVM treatment were summarized in Table 15.2.

Table 15.2 Summary of complications and outcomes of the published surgical series

Series, year	Patients	Age (mean)	Morbidity and mortality (%)	Occlusion (%)	S-M grade
Abad JM, 1983	70		11.0	81.4	
Jomin M, 1985	128		21.0	92.9	
Spetzler RF, 1986	100		4.0	100.0	I–V
Andrews BT, 1987	28	34	10.7	67.9	
Heros RC, 1990	153		8.4	100.0	I–V
Deruty R, 1993	64		18.8	93.7	I–V
Sisti MB, 1993	67		1.5	94.0	I–III
Hamilton MG, 1994	120	36	8.3	100.0	I–V
O'Laorie SA, 1995	56	36	5.3	92.9	I–V
Tew JM, 1995	39	30	15.4	97.4	III–V
Malik GM, 1996	156	33	14.7	95.8	
Schaller C, 1998	150	35	13.3		I–V
Pikus HJ, 1998	72		8.3	98.6	I–III
Hassler W, 1998	191		11.0		I–V
Pik JHT, 2000	110	38	2.7	98.8	I–III
Hartmann A, 2000	124	33	6.0		I, II
Solomon RA, 2000	86		1.2	90.7	
Stapf C, 2002	240	34	1.7	93.8	
Morgan MK, 2004	220		1.4	98.6	I, II
Lawton MT, 2005	224	38	7.1	98.0	I–V
Spears J, 2006	175	40	13.5		I–IV
Bradac O, 2013	74	40	1.4	97.3	I–IV
Theofanis T, 2014	264	38	7.8	100.0	I–V

Intraoperative Hemorrhage: Venous Injury/Retraction Injury

While most AVM microsurgical resections are challenging with some form of bleeding due to nature of lesions, some of the problems are avoided with careful planning and technique. Endovascular embolization should be judiciously used in selected AVMs to reduce the risk of hemorrhagic complications and to assist in surgical removal. Injury to major venous drainage pathway of the AVM may require an attempt to repair the vein, particularly in those lesions lacking major alternate venous drainage. Hemostasis techniques like bipolar cautery, clip placement, gentle tamponade, or Gelfoam itself or in combination may be required to preserve venous drainage until major sources of arterial flow can be isolated and occluded. In cases where substantial blood loss is anticipated, use of cell saver may be helpful.

Arterialized veins are differentiated from the feeding arteries by high-magnification inspection for the thickness of the vessel wall and degree of pulsations, often higher in the arteries. The vessels can be temporarily occluded with bipolar or temporary clips to assess for distal collapse or color change (vein) versus continued pulsations (artery).

Significant retraction of the brain parenchyma in the parasagittal approaches must be avoided to minimize the injury to the draining veins. Venous infarction, hemiplegia, and parietal lobe syndromes will likely result from sacrifice of posterior draining veins. Excessive retraction of the temporal lobe near the vein of Labbe could result in venous-related parenchymal damage and serious neurological sequelae. To minimize excessive venous retraction, brain resection is recommended to access AVMs in two specific locations: (i) corpus callosum for deep parasagittal malformations and (ii) inferior temporal gyrus for deep temporal AVMs [23]. Routine-induced hypotension to control bleeding during AVM resection should be viewed with caution as the normal parenchymal vessels are already dilated to counteract the arteriovenous (AV) shunting from AVM, and hypotension would result in ischemia [24]. In extreme circumstances of high-flow, poorly controlled bleeding, hypotension may be used as a strategy.

Bleeding During AVM Resection

Arterial bleeding from the AVM is usually controllable with judicious bipolar use and gentle tamponade, either manually or with self-retaining retractor. As dissection of the AVM proceeds into deeper portions of the lesion, persistent bleeding may indicate that the nidus has been violated. Reevaluation of the true plane between the malformation and brain and widening the diameter of the dissection will permit control of this type of bleeding. Control of deep perforating vessels near the end of dissection is the most challenging task in AVM surgery [24]. Maximally dilated with very thin walls, these arteries are fragile and resistant to bipolar cautery. Tamponade is generally ineffective and may obscure deep bleeding into the parenchyma or ventricular system. Patience and clear identification of the margins of the vessels are key to hemostasis in this area, as premature vessel rupture leads to retraction into the white matter with continued bleeding. Following these vessels for some distance may reveal a portion of the vessel that is more amenable for cautery.

An array of maneuvers may be necessary for achieving control of these vessels such as the use of routine microaneurysm clips, specially designed microclips, and clip occlusion followed by cautery, bipolar cautery of an exposed length of the vessel, and tangential cautery of the exposed length of the vessel. Morgan et al. reported 24 complications in their series of 112 patients with AVM resection. Hemorrhagic complications accounted for 25% of total complications (5/24) including fatal intraoperative hemorrhage in three patients, fatal normal perfusion pressure breakthrough (NPPB) in one patient, and a nonfatal intraoperative hemorrhage in one patient. Overall 80% of the hemorrhagic complications (4/5) were fatal [25].

Yasargil et al. reported requirement of blood transfusion in 30% (124/414) of their AVM resection. Of these 124 patients, 51% required up to 500 mL transfusion, 31% required 500–1500 mL transfusion, and 19% required more than 1500 mL transfusion. Six patients were reported to have out of control bleeding requiring >5 L of transfusion; two patients died and four patients had good outcome [3].

Residual AVM

Occasionally, a daughter nidus may be disconnected from the major nidus during AVM resection. The retained malformation may be the cause of persistent intraoperative bleeding. Although intraoperative angiography is the best way to detect residual nidus, this study is not always available or feasible. Doppler sonography is helpful for identifying retained AVM [26]. Usually daughter nidus is hidden in a sulcus closely related to the main nidus and is connected by one or two vessels. Through inspection of the resection cavity after the surgeon feels the AVM has been completely resected occasionally reveals a swollen, tense or deformed margin that usually represents residual malformation. Routine elevation of the patient's blood pressure 10–15 mm Hg above preoperative levels and inspection for 15 min before dural closure may also identify retained AVM and prevent complications or need for additional AVM therapy. At our center, we routinely perform intraoperative angiography to confirm complete resection, and re-exploration will be pursued if angiogram reveals evidence of persistent arterial to venous shunting.

Brain Swelling

Brain swelling during neurosurgical procedures by no means is unique to the surgery performed for AVMs, and the specific pathophysiology of the lesions presents additional challenges. General causes such as hypercapnia from obstruction of the endotracheal tube or ventilator disconnection should be ruled out first. Venous drainage compromise must be ruled out by checking patients positioning. The head should be elevated above the heart and care should be taken not to flex, extend, or rotate the head excessively to avoid the risk of jugular venous compression resulting in excessive bleeding, brain swelling, and damage to the normal brain parenchyma.

Once these are ruled out, specific complications for AVM surgery must be explored including occult bleeding, obstructive hydrocephalous from intraventricular bleeding, and cerebral edema from dysautoregulation.

Occult Bleeding

In patients with ruptured AVM with intraparenchymal hematoma, a portion of the AVM may become isolated from the surgical exposure and bleed when its venous drainage is disconnected. This may result in significant brain swelling with no immediately apparent cause. Often, this rapidly expanding hematoma will rupture into previous plane of resection. This requires expanding the pane of resection to include the hematoma cavity, and circumferential dissection must continue after the hematoma has been evacuated. The potential consequences from such hemorrhage include parenchymal damage from compression and vascular injury during evacuation and control of bleeding. There is also potential for rupture into the ventricular system which can result in obstructive hydrocephalous.

Obstructive Hydrocephalous

The ventricular system is entered during many AVM resections; hence precautions to prevent complications of expected bleeding into the ventricular system are important. The precautions include placement of cotton sponges to block exposed entry points during resection and removal of all identifiable intraventricular clot with extensive irrigation of the exposed ventricular system before closure. Occasionally bleeding into the ventricle may be occult and present only as global or focal brain swelling, accompanied by bradycardia, sudden hypertension, or change in the vital signs. Premature entry into the ventricle secondary to deep dissection with inadequate circumferential exposure of the AVM is the likely source of such bleeding. When suspected the ventricle must be immediately exposed through the ependymal wall and the clot evacuated. The site of bleeding must be methodically identified and secured.

Normal Perfusion Pressure Breakthrough/Occlusive Hyperemia

Normal perfusion pressure breakthrough (NPPB) is a one of the known causes for intraoperative cerebral edema. This is originally described by Spetzler and Wilson [27], subsequently reported, and challenged by other experienced neurosurgeons [28, 29]. This is characterized by acute massive brain swelling with a firm, distended herniating margin of the brain around the malformation with multiple bleeding points that are resistant to coagulation. The theory states that the brain around the AVM was subjected to prolonged ischemic steal, resulting in chronic dilatation and loss of autoregulation of the brains' arteries to divert the blood from AVM. These vessels are putatively unable of autoregulation when normal perfusion is established

by resecting the AVM nidus. The adjacent brain capillary breakthrough results in edema and hemorrhage.

When NPPB occurs intraoperatively, it usually appears toward the end of the resection when the high-flow shunt has been removed. Treatment consists of immediate brain protection, elevated cerebral perfusion pressure (CPP) by EEG-burst suppressive anesthesia with phenobarbital, and systemic arterial blood pressure reduction (systolic 80–90 mm Hg) with sodium nitroprusside or nicardipine [28]. This approach usually arrests spread of cerebral edema, allowing for craniotomy closure. Hemorrhagic brain tissue should be resected along with the AVM with absolute hemostasis, and complete resection should be confirmed with intraoperative angiography. The occurrence of NPPB was reported between 1 and 2% with 0–100% mortality in various AVM series [30, 31].

The ICP should be lowered by pharmacological means over the next 24 h under barbiturate coma. Non-contrast CT head should be performed for any unexplained alteration. If the surveillance CT demonstrates no progression, the patient should be weaned from antihypertensive agent over the next 12–24 h provided the ICP remains controlled and systemic blood pressure does not rise inappropriately. Subsequently, the patient can be weaned from the barbiturate over the next 24 h.

Prevention of NPPB) is the best form of treatment. High fistula flow with a paucity of flow entering the immediately adjacent brain is the angiographic hallmark predicting this condition. Staging AVM treatment with repeat operative approaches and/or endovascular embolization technique can be effective prophylaxis. This approach theoretically allows restoration of autoregulation at a gradual pace, as high-flow shunt is gradually and methodically reduced.

Postoperative Hemorrhage/Cerebral Edema

"Residual AVM" is the most common cause of the postoperative hemorrhage, and a daughter nidus is one of the most frequent causes of the retained lesion usually left on the wall or in the adjacent sulcus to the main area of nidus resection. The residual compartments are usually resected from the bulk of the nidus during the attempt to follow the resection plane along the embolic material of the lesion. Residual AVMs were reported up to 17% on postoperative angiography after AVM resection and are responsible for immediate and delayed rebleed accounting for 40–50% of spontaneous intracranial hemorrhage following AVM resection requiring re-exploration [31].

Normal perfusion pressure breakthrough (NPPB) is a rare but potential cause of postsurgical bleeding. Mayo Clinic group reported "occlusive hyperemia" in 6.4% of their 295 AVM resections, a phenomenon of otherwise unexplained brain hemorrhage or edema occasionally seen after the resection of high-flow AVMs [32]. Post-resection angiography in these patients consistently demonstrated slow flow in formal AVM feeders, their parenchymal branches, and impaired venous drainage in the region of resection. Authors postulated that stagnant flow in the arterial feeders produces hypoperfusion significant enough to cause ischemia with resultant hemorrhage and/or edema further complicated by venous outflow obstruction. This results in a vicious cycle of hyperemia, swelling, and worsened arterial stagnation.

Early diagnosis followed by aggressive medical and surgical management of these hemorrhagic complications of AVM surgery is essential to preserve the lowest rates of morbidity and mortality that are possible with the treatment of cerebral AVMS.

Vascular Thrombosis

Retrograde thrombosis back to the point of a proximal major branch has been reported as cause for delayed postoperative neurological deficit. Old age, larger AVM size, marked dilatation, and elongation of the feeders were identified as potential risk factors for this complication.

Epilepsy

Literature review shows that 27–38% of the patients with AVMs gave epilepsy before treatment and 4–30% develop new seizures after treatment [24, 33]. Most seizure disorders associated with AVMs are effectively controlled with antiepileptic medications, and patient with malformations in epileptogenic regions should routinely be treated prophylactically with these agents. Seizures were eliminated in roughly 18% of patients in whom arterial feeders were eliminated by ligation or embolization [34]. Complete resection AVM increases seizure-free outcome to approximately 56% of patients. Directed seizure surgery with AVM resection can result in up to 75% chance of seizure-free outcome [35, 36].

Residual AVM/Regrowth

Regrowth of the AVMs after angiographic evidence of complete resection is a reported entity. Yasargil et al. [3] reported five patients of complete AVM resection and documented regrowth of the AVM requiring additional surgery 1–7 years after the initial surgery. Four of these patients had rehemorrhage before regrowth was discovered. Patterson et al. [37] and Forster et al. [38] reported one case each of delayed rebleeding after complete AVM resection in their series. Lavine et al. reported recurrent hemorrhage following complete resection of the malformation and good recovery in two patients with cocaine and methamphetamines abuse, which were presumed to have precipitated the presentation.

Conclusion

Patient selection, evaluation of comorbidities, risk stratification, treatment justification against natural history, and decision analysis for treatment are vital in avoiding the complications. Patients must be evaluated on individual basis and discussed in multidisciplinary team meeting (MDT) involving professions from neurosurgery, endovascular neurosurgery, radiosurgery, and critical care. Complications are to be expected even in the hands of most skilled cerebrovascular neurosurgeons who deal with large number of AVMs.

Familiarity with these complications, high index of suspicion, early detection, and appropriate management are essential to achieve good outcomes that are possible with microsurgical treatment of these lesions.

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