Abstract

Background: Posterior fossa arteriovenous malformations (AVMs) are rare vascular lesions, representing 7–15% of all intracranial AVMs. Although less frequent than supratentorial AVMs, they present higher rupture, morbidity, and mortality rates. Microsurgery, radiosurgery, and endovascular neurosurgery are treatment options for obliteration of those lesions. In this paper, we present a critical review of the literature about the management of posterior fossa AVM.

Methods: A MEDLINE-based search of articles published between January 1960 and January 2014 was performed. The search terms: “Posterior fossa arteriovenous malformation,” “microsurgery,” “radiosurgery,” and “endovascular” were used to identify the articles.

Results: Current data supports the role of microsurgery as the gold standard treatment for cerebellar AVMs. Brainstem AVMs are usually managed with radiotherapy and endovascular therapy; microsurgery is considered in cases of pial brainstem AVMs.

Conclusions: Successful treatment of posterior fossa AVMs depend on an integrated work of neurosurgeons, radiosurgeons, and endovascular neurosurgeons. Although the development of radiosurgery and endovascular techniques is remarkable, microsurgery remains as the gold standard treatment for most of those lesions.

Key Words: Arteriovenous malformation, endovascular, microsurgery, management, posterior fossa, radiosurgery

INTRODUCTION

Posterior fossa arteriovenous malformations (AVMs) are for a rare group of vascular lesions located in the brainstem and cerebellum. The first report of these lesions date to 1908 when Clingestein published a case report presenting the clinical manifestations of this entity.[28] In 1932 the first successful resection of a cerebellar AVM was performed by Olivercrona and Riives[20] in Sweden. Even though posterior fossa AVMs represent 7–15%[1,3,7,9] of all intracranial AVMs, they carry a higher risk of rupture than supratentorial AVMs, and are associated with considerable rates of morbidity and mortality.[2] Most cerebellar AVMs are favorable for surgical resection. Brainstem AVMs are often treated with radiosurgery and/or endovascular therapy, given the high risks of major postoperative deficits after resection of AVMs in this region.[5] Currently, optimal treatment of infratentorial
AVMs requires adequate preoperative evaluation, a cerebrovascular team composed of microneurosurgeons, endovascular surgeons, and radiosurgeons and appropriate postoperative care in a neurocritical care unit.\textsuperscript{[1],[3],[8]}

We present a review of the management of posterior fossa AVMs. The roles of surgery, radiosurgery, and endovascular treatment as well as treatment outcomes are discussed based on the analysis of the current literature.

**METHODOLOGY**

Studies included in this review were selected from a MEDLINE-based search of articles published between January 1960 and January 2014. The search terms: “Posterior fossa arteriovenous malformation,” “microsurgery,” “radiosurgery,” and “endovascular” were used to identify the articles. Analysis was restricted to articles that included patients diagnosed with posterior fossa AVM despite its location or age. Articles about the natural history, diagnostic work up, management, surgical, endovascular, and radiosurgical therapies were evaluated. For centers with multiple reports on the management of posterior fossa AVM, the most recent and/or most comprehensive study was used for analysis.

**RESULTS**

**Classification of posterior fossa AVMs**

The infratentorial space is one of the most complex regions of the nervous system. A thorough knowledge of the anatomy of this area is necessary for adequate resection of posterior fossa AVMs. Only then the surgeon is able to adequately evaluate the spatial location of the malformation (i.e. its relationships within the cerebellum and/or brainstem to the posterior fossa cranial nerves, arteries, and veins) based on the preoperative catheter digital subtraction angiography (DSA), magnetic resonance imaging (MRI) scans, and computed tomography (CT)\textsuperscript{[12]} scans.

Based on the anatomy of the posterior fossa, infratentorial AVMs can be classified in eight groups [Table 1]. Brainstem AVMs can be classified according to their location as mesencephalic (supplied by the superior cerebellar artery [SCA]), pontine (supplied by the anterior-inferior cerebellar artery [AICA]), or medullary (supplied by the posterior-inferior cerebellar artery [PICA]). They can also be classified according to the depth of the lesion as either pial (superficial) or parenchymal. Pial brainstem AVMs are usually located in the anterolateral region of the pons and are considered potentially resectable lesions in experienced hands. Surgery is only recommended in those cases if early control of the feeding arteries and venous drainage system is possible and limited manipulation of the brainstem is required. Parenchymal brainstem AVMs, however, are not favorable for surgical treatment, considering their location, closely related with important cranial nerve nuclei and major fiber tracts, and considering potential disruption of the perforating branches of the vertebrobasilar system.\textsuperscript{[4]}

According to Lawton et al.,\textsuperscript{[22]} cerebellar AVMs can be anatomically classified into five groups: Suboccipital, tentorial, petrosal, vermian, and tonsillar. Suboccipital, inferior vermian and tonsillar AVMs are mainly supplied by branches of the PICA, tentorial and superior vermian AVM are mainly fed by the SCA, and petrosal AVMs by the AICA. Most of hemispheric and tonsillar AVMs have no extension into the fourth ventricle. Vermian malformations often have intraventricular extensions.

**NATURAL HISTORY**

Posterior fossa AVMs, represent 7–15% of all intracranial AVMs.\textsuperscript{[1],[3],[7]} Cerebellar AVMs, account for 75–81.2% of all the posterior fossa AVM while brainstem AVMs are observed in only 12.5–22.7%.\textsuperscript{[3],[7]} The mean age of presentation of intracranial AVMs is 32.8 ± 15.0 years.\textsuperscript{[10]}

In the specific case of posterior fossa AVMs, the mean age is 42 years,\textsuperscript{[15]} although brainstem AVMs present even earlier, with a mean age of 32 years.\textsuperscript{[25]} Men and women seem to be equally affected.\textsuperscript{[8],[14]}

In recent years, important contributions have been made to our understanding of the prognosis and behavior of posterior fossa AVMs.\textsuperscript{[10],[17]} Posterior fossa AVMs, unlike supratentorial malformations, present more frequently with subarachnoid hemorrhages.\textsuperscript{[12]} Mortality rates of up to 66.7% have been associated with the rupture of those lesions.\textsuperscript{[27]} In one of the most comprehensive studies about the natural history of AVMs, Hernesniemi et al. performed a retrospective analysis of 238 AVM patients with a mean follow-up period of 13.5 years. The authors evaluated risk factors for AVM rupture and the annual incidence of rupture of those lesions. According to this study, an infratentorial location is one of the most important risk factors for rupture. Univariate analysis demonstrates an annual rate of rupture of 11.6% in the first 5 years after admission, with a cumulative rupture rate of 45% in the first 5 years, as compared with an annual rate of 4.3% and a cumulative 5 years rate of 19% for supratentorial AVMs. They concluded that infratentorial location was an independent risk factor for rupture during the whole follow-up period, with a relative risk of rupture of 3.07 (1.37–6.87, CI: 95%) as compared with supratentorial AVMs.\textsuperscript{[10]} Other important risk factors for AVM rupture are previous hemorrhage and anatomic variations of the lesion, such as high feeding artery pressures.\textsuperscript{[2],[9],[10],[17]}
CLINICAL PRESENTATION

Patients with posterior fossa AVMs commonly present with infratentorial hemorrhages (60‑86%).[2,3,7] According to the location of these lesions, hemorrhages may present as parenchymal, subarachnoid or intraventricular. In cases of intraventricular hemorrhages, hydrocephalus is usually one of the initial presenting symptoms. The second most common presentation is progressive neurological deficits.[2] These are often associated to ischemia, mass effect, and/or hydrocephalus. Less common presentations include headaches, cranial nerve palsies, ataxia, and/or hemiparesis.[5,14]

PREOPERATIVE EVALUATION

Adequate preoperative evaluation and patient selection are important aspects of the clinical work up. CT, MRI scan, and DSA are necessary for evaluation and treatment planning. CT scan evaluation is helpful to diagnose brain hemorrhage and to assess potential associated complications, such as hydrocephalus or mass effect. Contrast‑enhanced CT scan can detect up to 95% of all intracranial AVMs,[23] which are seen as hyperdense contrast enhanced lesions. If a diagnosis cannot be made with CT‑scan evaluation, computed tomography angiography (CTA) can be used to confirm the presence of enlarged arteries and veins, but it does not provide precise anatomical information. Therefore, DSA is recommended in all cases to complete the evaluation of such lesions.

MRI studies define with higher accuracy the anatomical location of the AVM. Additionally, it is useful for evaluation of intraventricular and brain parenchyma extensions. MRI studies can evaluate the size of the nidus and its precise location as well as demonstrates recent and old hemorrhages and the presence of intralesional and perilesional gliosis.[8]

The gold standard study for diagnosis and evaluation of posterior fossa AVMs is DSA. The study is usually performed in a 2‑step manner: First a selective angiographic evaluation of the AVM is performed. Then, a superselective evaluation of the arterial nidus with microcatheters[21] is done. High‑resolution magnification studies are required of both vertebral arteries, internal carotid arteries, and external carotid arteries, because approximately 10% of infratentorial AVMs are supplied by one or more of these vessels.[19] Angiographic identification of flow‑related and intranidal aneurysms as well as “en passage” vessels is a paramount consideration in the preoperative evaluation of posterior fossa AVMs.

TIMING OF SURGERY AND PATIENT SELECTION

Surgical intervention is performed according to the patient overall and emergency status. Although most cases can be treated conservatively during the acute hemorrhagic period, in some cases the initial treatment may require insertion of intraventricular catheters, craniectomy, and duraplasty. In cases in which partial evacuation of a hematoma is pursued, it is important to avoid resecting the AVM at this time considering the difficult visualization of normal structures, the presence of brain edema and blood products, and the loss of autoregulation of the brain vasculature after the hemorrhage. Surgical resection of the AVM should be attempted in the acute period only as a last resort in cases of uncontrolled and persistent bleeding.[24] Resection of the AVM itself should be deferred for approximately 3 months after the last hemorrhagic episode.[19,24]

Treatment selection must be performed according to the characteristics of each case and the final objective must be the complete resection or obliteration of the AVM. Microsurgical resection remains the gold standard for treatment of most of these lesions. It is associated with excellent outcomes when performed by dedicated vascular microneurosurgeons.[3,6,7,19,24] AVMs located in the periphery of the cerebellar hemispheres, lower vermis, tonsils, and pial surface of the brainstem can be surgically treated with low rates of morbidity.[24]

In contrast, patients with lesions at the deep cerebellar nuclei and brainstem parenchyma and those in poor
SURGICAL TECHNIQUE

The basic strategy for microsurgical resection of AVMs can be described in five steps: (i) Creation of a wide craniotomy centered over the nidus, (ii) adequately exposing the feeding vessels and the draining veins, (iii) gradual devascularization of the lesion by occlusion of the arterial feeders, (iv) circumferential separation of the AVM from the adjacent parenchyma, and (v) division of the draining veins and extirpation of the lesion.

In our experience, intraoperative monitoring is essential in these cases. Routinely, brainstem auditory-evoked potential and somatosensory-evoked potentials are used for surgical approaches of posterior fossa AVMs. For lesions involving the floor of the fourth ventricle, facial nerve monitoring is mandatory. Intraoperative angiography is encouraged by the authors to confirm the complete resection of the AVM before finishing the procedure. If a residual lesion is observed, the surgery is continued in order to achieve the complete obliteration of the lesion.

The lateral decubitus (“park bench”) position is recommended for the surgical treatment of posterior fossa AVMs. This position is superior to the prone position because it decreases the intrathoracic pressure and lowers intracranial pressure. The “Concorde” position is avoided as it impairs venous return to both chest cavities, requires extreme flexion of the head, and is often associated with brain edema and increased intravenous pressure intracranially.

In cases of vermian, tonsillar, and fourth ventricular AVMs, a midline incision from the occiput to about C3 is performed. A lateral “sigmoidal” incision centered over the cerebellar hemisphere is used for cerebellar hemispheric AVMs or placed slightly more laterally for petrosal surface and anterolateral brainstem lesions. A wide midline or retrosigmoid suboccipital craniectomy is usually performed for posterior fossa AVMs. The far lateral transcylindrical extension can be used in cases of laterally located lesions, such as cerebellopontine angle (CPA) AVMs and anterolateral brainstem surface AVMs. The dura overlying the draining veins might be close adherent to these structures and should be carefully dissected to avoid venous bleeding. In cases where the dura is adherent to either a draining vein or to the nidus itself, it is best to cut around this portion of dura and to leave it attached to the AVM.

Adequate exposure of the AVM, careful inspection of the lesion and detailed analysis of its anatomy with correlation to the angiogram must be performed. The surface draining veins are usually the key to orientation regarding the location of the nidus.

The approach to the malformation itself should be initiated by identification and dissection of the major feeding vessels until their point of entry into the nidus. After identification, the arterial feeders can be coagulated or ligated with the use of small hemostatic clips. Adequate occlusion is followed by reduction of the pulsation and turgidity of the nidus. However, erroneous occlusion of a draining vein often results in increased pulsation, turgidity, and, even, hemorrhages. The “en passage” vessels must be carefully identified and preserved in order to avoid vascular injuries and subsequent ischemic complications.

The superficial portion of the AVM must be dissected away from the parenchyma after division of the superficial feeding vessels, freeing the lesion at its interface with the surrounding cerebellum/brainstem. Then a circumferential dissection around the nidus down to its apex must be performed. Special attention should be given to small vascular loops (Hashimoto’s U-shaped channels) that are encountered in the periphery of the AVM. These loops emerge from the nidus and reenter the lesion, consisting of shunting vessels or draining venules that should be preserved until the arterial supply has been completely obliterated. Cottonoids can be used to mark the progression of the dissection, separating the interface between the parenchyma, and the nidus. The dissection and bleeding control in the periventricular region can present as a significant problem in this stage of the surgery. Application of multiple small temporary microaneurysm clips followed by coagulation, lower bipolar coagulation, broader bipolar tips, coating the tips with wax, and efficient entry into the ventricles to eliminate the small ventricular feeders are techniques that might help in this part of the procedure.

The final part of the AVM resection consists of coagulation of the draining veins and resection of the lesion. These vessels are ligated as close as possible to a normal vein or sinus to avoid creating a blind sac. Then the AVM is extirpated and the cavity of the lesion is inspected. Valsalva maneuvers are performed to peak pressures of 30–40 mm Hg and a 5-min period of sustained systolic blood pressure to 140 mm Hg to test the resection cavity. Copious lavage of the subarachnoid space and ventricular system is done, followed by water-tight dural closure and subsequent closure of the operative wound in the usual fashion.
POSTOPERATIVE CARE

All patients are admitted for 2–5 days to the neurosurgical intensive care unit for monitoring of the blood pressure and possible signs of acute intracranial hypertension secondary to hemorrhages or thrombosis of the venous system. Strict blood pressure control for at least the first 3 days after surgery is recommended for the management of posterior fossa AVMs. This hemodynamic regimen prevents excessive perfusion of the chronically ischemic brain tissue surrounding the resection bed and avoid reperfusion injury.[10] If significant intraventricular hemorrhage occurs intraoperatively and in cases that require surgical approach to the region of the IV ventricle, it is reasonable to perform a ventriculostomy to avoid acute hydrocephalus after surgery. Whenever the intraoperative angiogram is not sufficient to exclude residual malformations, a postoperative angiography is recommended.

COMPLICATIONS

Intra- and postoperative hemorrhages, venous and arterial ischemia, cranial nerves, and long tract deficits are potential complications associated to the surgical resection of posterior fossa AVMs.[3,7] Although ataxia is common after resection of large cerebellar AVMs, it is usually transient and improves in a few weeks after surgery. Intra- and postoperative hemorrhages are associated with poor outcomes after surgery. Compromise of venous structures early in the approach, inappropriate hemostasis of arterial feeding vessels, or rupture of the nidus during the procedure is associated with intraoperative bleeding. Postoperative hemorrhages might be secondary to three factors: Incomplete AVM resection, inadequate hemostasis, or “breakthrough” bleeding. Hydrocephalus might be present after intraventricular hemorrhages and may require a ventriculostomy.

OUTCOMES

Surgical resection remains the gold standard for treatment for posterior fossa AVMs. In one of the largest, early series of posterior fossa AVM surgery, Drake et al.[1] achieved complete resections in 92% of cases and reported excellent and good outcomes in 71% of the patients, with morbidity and mortality rates of 21% and 15%, respectively.[7] Rodriguez-Hernandez et al.[22] recently reported 60 cases of surgically treated posterior fossa AVMs at The University of California, San Francisco. Resection rate of 100%, transient morbidity rate of 20%, and mortality rate of 5% were achieved in that study. Other groups have reported similar favorable outcomes (Glasgow Outcome Scale (GOS) 4 and 5) in 80–91% of the patients[3,14,27] as well as morbidity and mortality rates of 9.0–17.0% and 4.1–8.3%, respectively[2,3,14,27] (Table 2).

ROLE OF STEREOTACTIC RADIOSURGERY

The goal of SRS is to achieve complete obliteration of the AVM nidus while avoiding adverse radiation effects. It is the main treatment modality for brainstem and deep cerebellar AVMs, given that surgery is usually associated with significant postoperative deficits.[12] Small AVMs that receive at least 20 Gy at their margins are the best candidates for such treatment. Total obliteration rates after SRS range from 44% to 75% over 3–4 years based on MRI or angiography studies.[12,16] In cases of incomplete obliteration of the AVM after 3–5 years of the SRS, another radiosurgical procedure can be performed.[12] Factors associated with a higher rate of total obliteration include: Smaller number of isocenters, smaller nidus diameter, and higher margin dose.[12,29] One of the most important drawbacks of SRS is the fact that patients remain at risk for hemorrhage until obliteration of the AVM is achieved, usually around 2–5 years. Previously, clinical series have reported an annual risk of hemorrhage of 2–4% after SRS for brainstem AVMs.[12,16,29] Complications related to SRS are described to occur in approximately 10% of the patients,[12,29] including diplopia, hemiparesis, ataxia, and sensory dysfunction.

ROLE OF ENDOVASCULAR THERAPY

Endovascular embolization of posterior fossa AVMs may be useful as an adjuvant treatment before surgical resection or radiosurgery. Cases of large hemispheric cerebellar AVMs and brainstem-cerebellar AVMs might specially benefit from a multidisciplinary treatment that includes adjuvant endovascular therapy.[14] Superselective angiography for evaluation of the nidus and “en passage vessels” is paramount for avoidance of complications.

Table 2: Treatment of posterior fossa arteriovenous malformation

| Study                        | $N$ | Total obliteration (%) | Morbidity(%) | Mortality (%) |
|------------------------------|-----|------------------------|--------------|--------------|
| Drake et al. (1986)[7]       | 66  | 92                     | 14           | 15           |
| Batjer and Samson (1986)[3]  | 32  | 93.7                   | 13           | 7            |
| Symon et al. (1995)[27]      | 28  | 82.6                   | 17           | 8            |
| Kelly et al. (2008)*[14]     | 48  | 52                     | 18           | 16           |
| Rodriguez-Hernandez et al. (2013)**[22] | 60  | 100                    | 16           | 5            |

*Patients with brainstem and cerebellar AVMs grades III-IV according to the Spetzler-Martin classification. Surgery, radiosurgery and embolization results reported. ** Only included cerebellar AVMs.
Intra- and postprocedural hemorrhages are usually related to direct nidus injury or migration of the embolic material and occlusion of drainage veins. Complications related with endovascular treatment are described in 9.4–20.8% of the cases of posterior fossa AVMs and are associated with significant morbidity in 12.5% of the patients.

**CONCLUSIONS**

Posterior fossa AVMs are among the most challenging lesions in neurosurgery. Management of these AVMs requires knowledge of their natural history, thorough clinical and radiological evaluation, and collaboration between neurosurgeons, endovascular surgeons, and radiosurgeons. Surgical resection is the optimal treatment option for most cerebellar AVMs, whereas most cases of brainstem AVMs will require radiosurgical treatment. Endovascular therapy has an important role as an adjuvant treatment in cases of large cerebellar AVMs and represents an important treatment option for occlusion of superficial brainstem lesions, particularly since the development of modern embolic agents.

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