Surgery of Posterior Fossa AVM

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21.1 Signs and Symptoms

Prior to the routine use of vertebral angiography, posterior fossa AVMs were usually found unexpectedly during the evacuation of posterior fossa hematomas [1]. In 1932 (May 5th), Olivecrona had reported their first successful radical removal of a left cerebellar AVM in a 37-year-old male who was misdiagnosed of having a posterior fossa tumor. After an 8-h-long marathon surgery under local anesthesia and with a blood transfusion of 2000 ml, the AVM was removed. The postoperative course was uneventful and the patient left the hospital 3 months later [2]. As vertebral angiography became more widely used, the preoperative diagnosis of posterior fossa AVMs became possible. Even so, at that time, neurosurgeons thought that it was possible to operate on small-to-moderate-sized AVMs in silent areas of the brain but were reluctant to touch AVMs in nonsilent areas [2], including the more demanding posterior fossa. Neurosurgeons were, however, disappointed that only very few AVMs could be removed entirely without great risk, which led to the development of other, ineffectual techniques, such as vertebral artery or feeder ligation [1].

In 1961, based on a review of literature and his personal experience, Verbiest showed that small cerebellar AVMs could be removed safely. However large posterior fossa AVMs, even if located outside the brainstem, could be extremely dangerous to operate on, while brainstem AVMs were considered inoperable lesions by their nature [3]. Since that report, the frequency of successful surgical removal of the posterior fossa AVMs has increased with the development of new microsurgical and radiological techniques, modern neuroanesthesia, and refined intensive care principles. Later, Drake with his big series reported that removal of the posterior fossa AVMs even in the more sensitive areas of the hindbrain could also be accomplished with excellent outcomes.

Over the last 30 years, around 1000 patients with AVMs have been treated in two centers in Finland (Helsinki and Kuopio); one tenth of them were posterior fossa AVMs. The accumulated experience in the treatment of these patients is documented in this chapter, as well as the philosophical and practical basis for our current management of these relatively uncommon lesions.

All types of AVMs contribute about 2% of all hemorrhagic strokes. When AVM rupture occurs, it usually affects young adults in third or fourth decade of their life, more often males than

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females. Based on the available data, the estimated rate is about 1:100,000 person-years which is about 1/10 of that of cerebral aneurysms. Posterior fossa AVMs are even more uncommon, and it can be roughly concluded that 1/10 of cerebral AVMs are located in the posterior fossa [1].

21.1.1 Location

The posterior cranial fossa is the largest and deepest of the three cranial fossae, which extends from the tentorial incisura superiorly to the foramen magnum inferiorly. The major contents of it are the cerebellum and the brainstem which consist of the midbrain, the pons, and the medulla oblongata. Posterior fossa AVMs can be generally classified into brainstem (25%), cerebellar vermian (15%), superficial cerebellar hemispheric (40%), and deep cerebellar hemispheric (20%) lesions based on the dominant location of the nidus.

Of the 631 AVM patients admitted to Helsinki University Central Hospital between 1942 and 2005, a total of 64 patients were diagnosed with infratentorial AVMs (Table 21.1).

21.1.2 Signs and Symptoms

In many studies, including ours, hemorrhage is the main clinical presentation of the posterior fossa AVMs accounting for 72–92% of the patients [1, 2, 4–7]. The type of hemorrhage depends on the location of the AVM, e.g., cerebellar AVM cases often come with intraparenchymal hemorrhage, cerebellopontine angle (CPA) AVMs are highly related to subarachnoid hemorrhage, and brainstem AVMs are more likely to cause subarachnoid and intraventricular hemorrhage (Figs. 21.1 and 21.2a). We have earlier reported that the annual rupture rate of posterior fossa AVMs within 5 years after admission is almost three times higher than in their supratentorial counterparts (11.6% vs. 4.3%) [8].

Table 21.1 The locations of the posterior fossa AVMs [4]

	Females *n* (%), 28 (44)	Males *n* (%), 36 (56)	Total *n* (%), 64 (100)
Pontomesencephalic	3 (10.7)	12 (33.3)	15 (23.4)
Medulla oblongata	1 (3.6)	0 (0)	1 (1.6)
Cerebellar vermis	5 (17.8)	4 (11.1)	9 (14)
Superficial cerebellar hemispheric	11 (39.2)	16 (44.4)	27 (42.2)
Deep cerebellar hemispheric	8 (28.6)	4 (11.1)	12 (18.8)

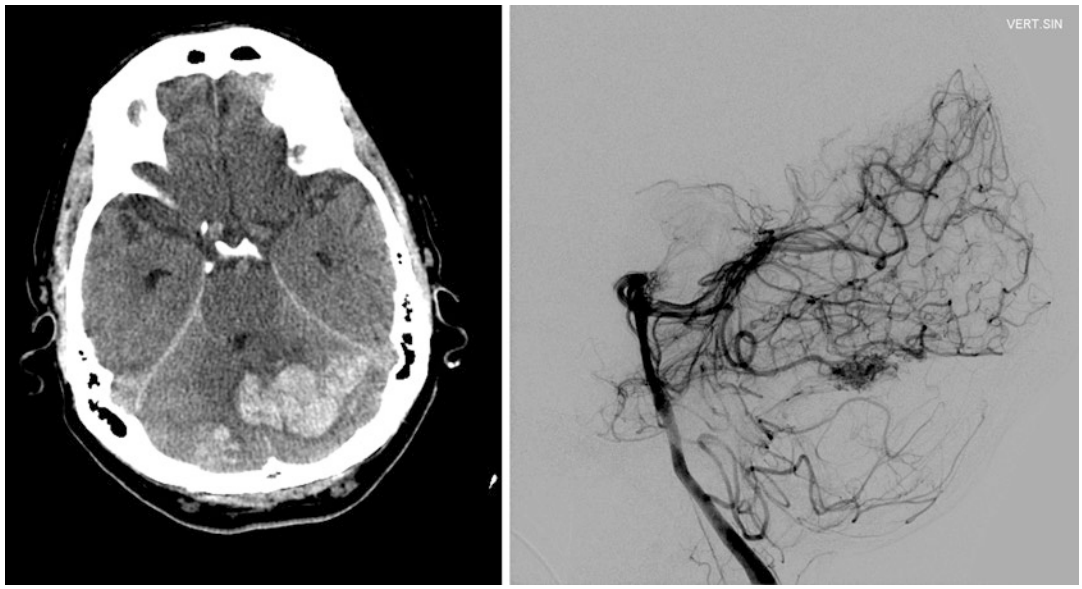

Fig. 21.1 Axial CT scan shows ICH due to AVM rupture, and posterior circulation DSA (lateral view) shows the location of the AVM

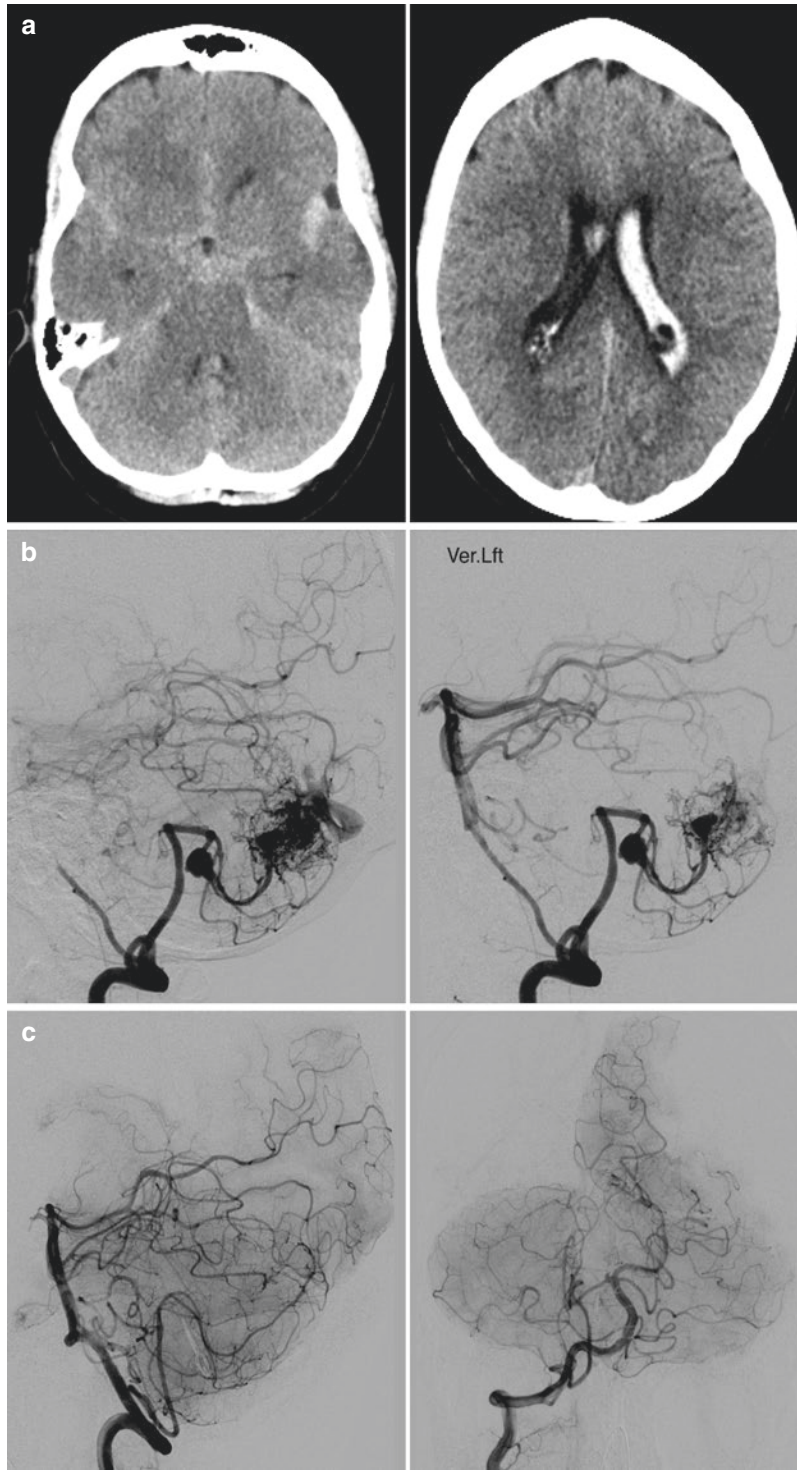

Fig. 21.2 (**a**) CT scan of the brain shows SAH and intraventricular bleeding due to posterior fossa AVM with associated aneurysm confirmed by left vertebral artery DSA (**b**). (**c**) Post-op DSA illustrates complete surgical removal of the AVM and clipping of the associated aneurysms

The risk factors for hemorrhagic presentation of AVMs in general based on studies from the Finnish population are young age, AVM, deep venous drainage, previous rupture, and infratentorial and deep location. Small AVM size tends to have a higher risk for hemorrhagic presentation in almost every series, while large AVM size is also a factor for AVM rupture in most multivariate analyses (including ours) [8, 9]. Some multivariate analyses didn't find large size as a risk factor for AVM rupture, but importantly, small AVM size has never been found as a prospective risk factor for rupture in a multivariate analysis. That is because the small AVMs will not be diagnosed unless they are ruptured, whereas many large ones become symptomatic even before the hemorrhage [10, 11]. AVM-associated aneurysms are found as risk factor for hemorrhage in many meta-analysis studies [10–12]. This risk is highest for the first few years after diagnosis, then it declines, but overall it remains significant for decades [13].

Other clinical presentations are relatively uncommon and depend on the location of the AVM. Cerebellar AVMs are more likely to cause headache, hydrocephalus, and gait disturbances. CPA AVMs may give symptoms of cranial nerve compression such as trigeminal neuralgia or hemifacial spasm. Brainstem AVMs can cause significant neurologic deficits even in the absence of hemorrhage probably due to local mass effect, vascular steal phenomenon, or venous congestion. Overall, progressive neurologic deficits, cranial nerve palsy, headache, and ataxia are possible symptoms of posterior fossa AVMs [1, 7]. In recent years the incidental finding of posterior fossa AVMs has also become more common with the increasing use of modern imaging techniques, making incidental AVMs to account for to 10% in recent studies, compared to that of less than 2% in early studies [14].

21.2 Investigation

Since the most common clinical presenting sign of posterior fossa AVM is an acute hemorrhage, a computed tomography (CT) scan is the most commonly used initial imaging modality. Location, volume, and mass effect of the hemorrhage can be detected quickly with a CT scan as well as signs of hydrocephalus. Any evidence of a previous bleeding or calcification raises the possibility/probability of an AVM as the origin of the hemorrhage (Fig. 21.1). Contrast injection may show feeding arteries and draining veins as linear, vermiform, tubular, or serpentine high-density areas.

Magnetic resonance imaging and angiography (MRI and MRA) provide exquisite detail of the anatomic relations of the AVM. This is particularly useful when the AVM lies in or near the brainstem or cerebellar peduncle, in order to determine the operability or to plan the surgical approach. MRI is superior to other investigations in defining the nidus size and its location in relation to the surrounding structures. Nidus of the AVM is seen as an area of signal void and may have a honeycombed pattern (Fig. 21.3). Tubular signal void structures converging on the nidus represent the feeding arteries and draining veins. Bony artifacts are avoided with MRI, allowing therefore better identification of superficial supply to, or superficial drainage from, the AVM. Gliosis and infarction surrounding the nidus are shown with the hyperintensity area in T2 and FLAIR on MRI. MRI is however an impractical imaging technique for critically ill patients, for example, those harboring a cerebellar clot.

Digital subtraction angiography (DSA) imaging is essential for all patients with AVMs in order to exactly delineate the vascular structures comprising the AVM and for the subsequent management. With DSA the feeding arteries and draining veins can be visualized (Fig. 21.4), but this will necessitate selective injections of contrast material into both the external and internal carotid arteries as well as both vertebral arteries and, occasionally, super-selective injections into the larger feeding arteries. DSA is also vital in the detection of anatomic variants, associated aneurysms, and normal vessels of passage (Fig. 21.2a, b).

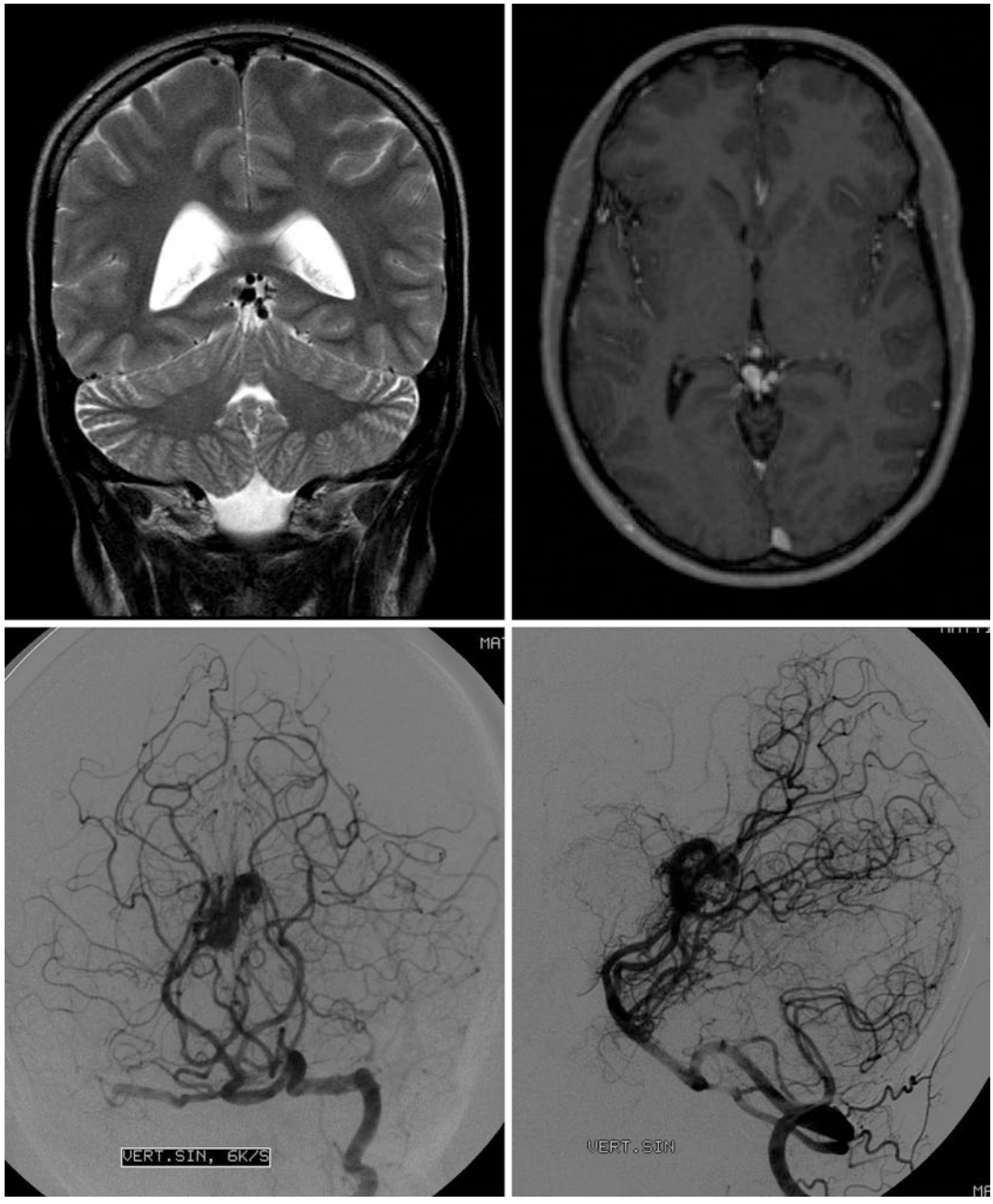

Fig. 21.3 MRI and MRA determine the precise location of the AVM in quadrigeminal cistern after DSA of posterior circulation

21.2.1 Approach

Posterior fossa AVMs are very prone to rupture, and treatment should be considered in every case, except perhaps for the elderly patients with incidental lesions. In AVM treatment, the aim is to eradicate the malformation completely without disrupting normal blood flow. Incomplete treatment provides no significant protection from further hemorrhage.

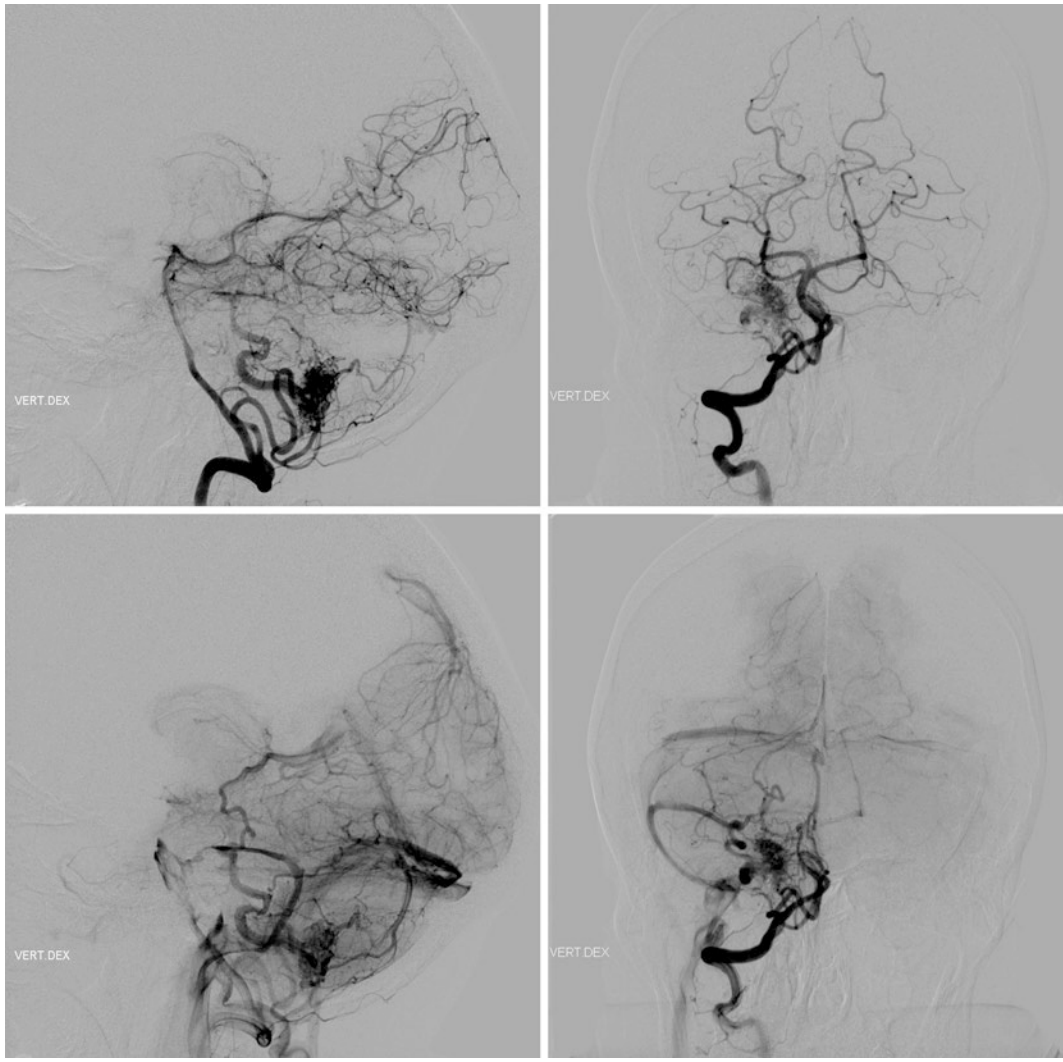

Fig. 21.4 Right vertebral artery angiogram (DSA) demonstrates suboccipital AVM, feeders from PICA, AICA, and SCA

The decision of whether to treat these lesions and the choice of the best treatment modality is difficult. A multidisciplinary team of neurosurgeons, neuroradiologists, and radiotherapists is needed when selecting the best management strategy for each individual patient.

In that process one must take many factors into consideration:

1. The presenting symptom of the lesion.
2. The age and any other comorbid condition of the patient.
3. The anatomical properties of the AVM (location [eloquent, non-eloquent area], size, feeding arteries and its type [terminal, en-passage, deep feeders], and the venous drainage).
4. AVM's relationship to the adjacent brain structures.
5. Presence of a hematoma or not.
6. The associated aneurysm with AVM and which type (intranidal, flow related).
7. Experience of the neurosurgeon.

After the evaluation of all of these factors, the most suitable modality of treatment is chosen as single or combined therapy which directs to prevent future hemorrhage and resolve the related symptoms (Fig. 21.2a–c).

21.3 Modalities of Treatment

21.3.1 Endovascular Techniques

During the last two decades, endovascular techniques have become a therapeutic option in the management of the AVMs. Preoperative embolization aims to devascularize a large AVM with particulate material, mostly by using rapidly (butyl cyanoacrylate, Histoacryl) or more recently by using a slowly setting glue (ethylene vinyl alcohol copolymer, Onyx). Complete obliteration of an AVM by a rapid embolization alone is an uncommon situation and accounts for only about 10% of all our cases, but with a slow technique of Onyx injection, it can be obtained in up to 50% of the selected cases [15].

Until now, however, none of the embolic agents, which have been used to obliterate these lesions, provide total protection against recanalization, regrowing, and rebleeding. Therefore, we use embolization as combined therapy preoperatively to diminish intraoperative bleeding, making the surgery resemble more that of extrinsic tumor removal. Endovascular techniques can also be used to obliterate the feeders that arise from the deep which are usually difficult to reach or controlled with microsurgical techniques, but such vessel may also difficult to reach by microcatheters.

Many complications may follow the embolization procedures, like occlusion of the main draining vein with risk of AVM rupture during or after the procedure, occlusion of arteries in passage, or occlusion of major cerebral arteries. In some locations in the posterior fossa, preoperative embolization may form a hard mass which later can be difficult to dissect from the cranial nerve roots or brainstem. Also, if the preoperative embolization is performed poorly, the result might be of more harm during the surgery rather than benefit; the surgery could become even more difficult especially if they embolized more draining instead of the feeder.

Recently, we started to perform both the embolization and the microsurgical removal at the same day to avoid hemorrhage resulting from the hemodynamic changes inside the nidus, caused by partial embolization of the AVM.

21.3.2 Radiotherapy

Irradiation activates the swelling and degeneration of endothelial cells and perivascular tissue, which promote thrombosis and proliferative fibroblastic repair [1]. These changes in the abnormal vessel walls lead to occlusion of the shunt and finally obliteration of the AVM. The success of this technique depends on the delivery of ionizing radiation in a precise and focused manner to the malformation and avoiding exposure of the surrounding brain. Traditionally, the indication of radiosurgery has been a deep location or a location in eloquent brain that is associated with high surgical morbidity and mortality; such locations include the basal ganglia and brainstem.

The obliteration of AV shunt by radiosurgery takes between 1 and 3 years with a risk of recurrent hemorrhage during this period; actually the rate of recurrent bleeding is 2.98%/year/patient [16], and it seems that the occurrence is the same as if the lesion had been left untreated.

21.3.3 Microsurgery

Microsurgery is considered an ideal treatment for ruptured AVMs. It is, however, controversial in unruptured AVMs, even if radical eradication allows a complete cure of the lesion. ARUBA study found that conservative treatment of unruptured AVMs is superior to the interventional ones (microsurgery, endovascular, and radiosurgery) [17]. The study has nevertheless been criticized because of the short follow-up time, the low number of patients that does not allow subgrouping of the patients according to location or size of AVM, and most patients in the interventional arm being treated by radiosurgical methods.

Laakso et al. studied retrospectively 623 AVM patients [4]. The total follow-up is 10,165 person-years, and this study provides a significant long-term follow-up. In their study, they compare the survival of AVM patients to the general population. Their results show that AVMs are associated with significant and long-lasting excess mortality that is considerably lower in actively treated patients. Even with these results, however, the decision-making regarding incidentally found unruptured AVMs is difficul [4, 5, 13, 18].

The decision of whether to operate on AVMs is based on many factors mentioned before. Size and location are the most important factors in the decision-making; major involvement of the brainstem may increase the risk above acceptable. We found superficial and vermian cerebellar AVMs generally favorable for microsurgical treatment. In ruptured AVMs, the hematoma cavity surrounding the nidus facilitates removal of even deep-seated lesions [5, 19].

Drake et al. in their series had no morbidity or mortality in small AVMs (<2.5 cm), and just over 15% morbidity and mortality in large AVMs (>5 cm) [1].

21.4 Steps of the Surgery

Posterior fossa AVMs are classified into (1) cerebellar hemispheric AVMs (tentorial, suboccipital, and petrosal); (2) vermian AVMs (suboccipital and tentorial); (3) tonsillar AVMs; and (4) brainstem AVMs (midbrain, pontine, and medulla oblongata) [1, 14].

In Helsinki we use four approaches to resect most posterior fossa AVMs: (1) supracerebellar approach for cerebellar tentorial, vermian tentorial, and posterior midbrain AVMs; (2) suboccipital approach for cerebellar suboccipital and vermian suboccipital AVMs; (3) suboccipital telovelar approach for tonsillar AVMs; and (4) retrosigmoid approach for cerebellar petrosal and some brainstem AVMs.

21.4.1 Supracerebellar Approach [20]

Unless contraindicated, e.g., due to old age and cardiac disease, we use the sitting position for the supracerebellar approach. The advantages of this approach are providing better drainage of CSF and possible less bleeding during surgery, provide a relax vein, and being able to observe the superior anatomical view of the AVM and identify the feeder. But we need to be careful with the risk of air embolism, cervical myelopathy, and hypotension.

Instead of the classical midline supracerebellar approach, we prefer paramedian approach. Its advantages are fewer veins in the surgical trajectory, less steep tentorium, and reduced risk of air embolism due to avoidance of exposure to the confluence of sinuses. The disadvantage is the difficulty to identify the best trajectory toward the center of the quadrigeminal cistern.

21.4.1.1 Positioning

The sitting position we use in Helsinki is similar with praying position. We bent the upper torso and head of the patient forward about 30° and downward. By doing that, we expect the tentorium would be almost horizontal. This position will provide a good viewing angle from the most cranial portions of the posterior fossa to the lowest part. Two fingers have to fit between the chin and sternum; it will provide enough room to prevent compression of the airways and jugular veins.

21.4.1.2 Incision and Craniotomy

We make a linear incision 2–3 cm lateral from the midline. The paramedian incision will start about 2 cm cranial to the occipital protuberance and extends until the craniocervical junction. The muscle can be easily split until the occipital bone, along the incision line, and can be held with self-retaining retractor. To spread the wound, we place a curved retractor both from cranial and caudal directions. We detach the muscle insertions with diathermia and expose the occipital bone. Bony exposure is usually enough at 3–4 cm below the transverse sinus.

We place one burr hole 3 cm lateral from the midline, slightly superior to the transverse sinus. We detach the dura from the transverse sinus, gently with blunt and curve dissector. Then we perform craniotomy with 3–4 cm bone flap. The bone flap should be above the transfer sinus to allow retraction of transfer sinus cranially. The bone opening should leave 1 cm edge of bone to the midline.

21.4.1.3 Dural Opening

Sometimes the draining vein or the AVM itself is firmly adherent to the dura. Therefore, we carefully inspect the dura under the microscope to avoid any injury to the draining vein. To avoid injury to the veins or transverse sinus, the dura is opened under microscope. We open the dura in a V-shaped form and the base at the sinus. Many veins at supracerebellar area usually located in the midline such as superior cerebellar vein and could be avoided with this approach. Once the thick arachnoid and few of the bridging veins between tentorium and cerebellum were coagulated and cut, the cerebellum will follow the gravity, allowing a good surgical view without any brain retraction.

21.4.2 Retrosigmoid Approach [20]

This approach is described in detail elsewhere in this book. We describe only our modification of this technique.

21.4.2.1 Positioning

We place the patient in lateral position (park bench), with the head elevated about 20 cm above the heart level. We rotate the upper body slightly (5–10°) backwards, in order that the shoulder is retracted with tape more easily caudally and posteriorly. The head (1) is slightly flexed forward, (2) may be tilted laterally, and (3), if necessary, may be rotated slightly toward the floor.

To gain slack brain, we use spinal drainage with 50–100 ml CSF removed.

21.4.2.2 Skin Incision and Craniotomy

We place a linear incision 2 cm behind the mastoid process. The length of incision varies depending on distance of the AVM from the foramen magnum. Of importance is to have the skin incision long enough to obtain an adequate bony exposure. We place a large-sized curved retractor at the cranial side of incision. We continue to split the subcutaneous fat and muscles with monopolar until we found the yellowish fat at the level of the foramen magnum. This should be a sign for warning of extracranial vertebral artery.

We made one burr hole just at posterior border of the incision and then continue with the craniotomy. A bone flap of 2–3 cm is usually sufficient. If necessary, the bony opening can be extended by drilling mastoid part of the temporal bone. If the mastoid air cells are exposed, they really need to be meticulously closed to prevent the postoperative CSF leak.

21.4.2.3 Dural Opening

The duramater is opened in a curvilinear shape, and the base at the mastoid. The dura edges are tackled with sutures. When the dura cut near the transverse sigmoid junction, the dura will be cut in a three-leaf fashion. One is directed exactly to the junction for better exposure.

The brain should be slack due to the spinal drainage, and once the dura is opened completely, we may close the drain temporarily. If the brain is still tight, opening the cerebellomedullary cistern (cisterna magna) helps to slack the brain. In this approach, the petrosal vein is often the prominent vein seen when approaching the tentorium or the upper cranial nerves. Although some papers mention that we may sacrifice this vein, many surgeons have observed complications after its occlusion. The best suggestion is to preserve it.

21.4.3 Suboccipital Craniotomy [20]

21.4.3.1 Positioning

We usually use the sitting position described previously in this chapter.

21.4.3.2 Incision and Craniotomy

We make the incision on the midline. The incision starts from the external occipital protuberance and extends till the C1–C2 level. We split the muscles at the midline with monopolar all the way to the occipital bone. If we split it exactly at the midline, there will be less vascular tissue. We place one large self-retaining retractor on each

end, cranial and caudal. When dissecting the muscles, care is needed to avoid injury to the vertebral artery, which is located 1–2 cm from the midline.

We place one burr hole 1 cm paramedian from the midline, below the transverse sinus. We make two cuts with the craniotomy toward the foramen magnum. At the cranial aspect, the bone flap is secured with strong and large rongeur. If needed, we use a high-speed coarse diamond drill or a small rongeur to extend the lateral exposure of the foramen magnum.

21.4.3.3 Dural Opening

We open the dura under a microscope in a reverse V-shaped form, and the base at the foramen magnum. We start dura cutting below the occipital sinus, and the dural leaf is everted caudally and sutured tightly to the muscles. At this point, usually the arachnoid membrane that covers cisterna magna is usually intact. The arachnoid is opened and attached it to dural leaf using hemoclips. Then under high magnification, we gently push apart the cerebellar tonsils, and we start to enter the fourth ventricle. By changing the table and microscope angle, we may even obtain visualization of superior part of fourth ventricle and aqueduct.

21.4.3.4 Dissection

Mostly microsurgical removal of AVMs is a great challenge. In posterior fossa AVMs, the lesion is located deep in a small compartment close to many critical structures, which makes posterior fossa AVM surgery even more difficult. Therefore, only experienced neurovascular team should operate on these lesions (Table 21.2).

After opening the dura, we inspect the AVM under less magnification to estimate the nidus and their relationships to the adjacent neurovascular structures. We discern also the superficial feeders, the pattern of draining veins, and the plane of cleavage. During this initial inspection of the AVM, we use indocyanine green (ICG) angiography to distinguish arterial feeders from arterialized draining veins. ICG provides great help in this purpose.

We perform all the AVM dissection using maximize magnification. The cleavage plane between the nidus and the neural tissue is distinguished as a yellow gliotic tissue formed by previous hemorrhage(s). The feeders enter the AVM through this plane, and they should be identified, coagulated, and cut at this point. If no clear plane surrounds the AVM, especially in critical areas, dissection must be considered on the AVM itself depending on the axiom that no normal neural tissue between the AVM aggregation.

Besides carefully studying of 3D reconstruction images preoperatively, the large superficial feeding arteries are identified intraoperatively with ICG. We often place temporary clips only on the feeders that terminate in the AVM and avoid the main trunk of en-passage arteries, which also have branches that supply the normal cerebellum and/or

Table 21.2 Characteristics of 64 patients with infratentorial arteriovenous malformation, stratified by result of treatment [4]

	Total occlusion $n = 36$ (%)	Partial occlusion $n = 12$ (%)	Conservative $n = 16$ (%)
Pontomesencephalic	4 (11.1)	3 (25)	7 (43.7)
Medulla oblongata	0 (0)	1 (8.3)	0 (0)
Cerebellar vermis	6 (16.7)	2 (16.7)	1 (6.3)
Superficial cerebellar hemispheric	17 (47.2)	4 (33.3)	6 (37.5)
Deep cerebellar hemispheric	8 (22.2)	2 (16.7)	2 (12.5)
Multiple	1(2.8)	0 (0)	0 (0)

brainstem. Unintended occlusion of these branches may cause devastating brainstem infarction.

The small feeders should be coagulated directly in the cleavage plane. The tiny and fragile feeders, or dilated capillaries around the AVM, remain the most difficult part of the procedure, particularly when these vessels are close to the ventricle. Because of their fragile nature and little tissue to act on, bleeding from these tiny vessels is difficult to control. We use continuous coagulation with nonsticking bipolar forceps, which we quickly interchange when they become soiled. Bipolar is also used to shrink the AVM and to clarify the cleavage plane.

After disconnecting all the feeders, the AVM becomes border-free, bloodless, smaller, and non-tense, and the color of the draining vein(s) changes from red into blue. At this point, the draining vein can be coagulated and divided safely. If the venous drainage is obstructed during the procedure prior to this point, the AVM may become tense and prone to massive uncontrolled bleeding, and also swelling of the cerebellum can occur. If a secondary draining vein impedes the resection, the trick is to occlude this vein by a temporary clip. If no swelling of the AVM or cerebellum is observed, dividing the vein is safe.

Once the total AVM removal is achieved, the cavity should be carefully inspected for any remnants. Our technique is to touch all walls with the cotton and bipolar tip gently. A bleeding may indicate an AVM remnant, and another resection plane should be performed in that location. Hemostasis of the resection cavity must be meticulous. We use hemostatic materials (Tachosil ®, Surgicel ®, fibrin glue) to seal the resection cavity.

The dura should be closed in watertight and few stitches are used to lift it up to the bone edges. We put back the bone flap and secure it before closing the wound in layers.

21.5 Expert Opinion and Suggestions to Avoid Complications

- You cannot "try" AVM surgery – the surgeon always need to be aware that once the AVM surgery started, it has to be carried till it finish, and the nidus should be removed completely (Figs. 21.4 and 21.5).
- Before starting the operation, preoperative study on the images is essential to have clear understanding of anatomy of the main feeders and the relationship between the AVM and the adjacent structures.
- Initiating the dissection around the AVM in the correct plane facilitates the removal of even complex AVM with minor bleeding.
- The whole AVM should be removed as one piece; piecemeal removal causes a massive bleeding which is difficult to control.
- Hemostasis of the deepest part of the AVM with thin-walled feeders is the most difficult part of the surgery. It requires a lot of patience, time, and blunt-tipped bipolar coagulation.
- Persistent bleeding from the resection bed usually indicates the presence of nidus remnant, and then another specific detection is needed.
- To be more familiar with AVM surgery, it is good to know that the small feeders surrounding gliomas are similar to those of AVMs; this helps you to sharpen your skills in AVM surgery.
- The draining veins have to be preserved until the end of the resection.
- Simple ligation of feeding vessels of the AVM is ineffectual. The fistulous connections between the abnormal arteries and veins in the nidus will rapidly change and recruit any vessel and make it even more prominent prior to ligation.

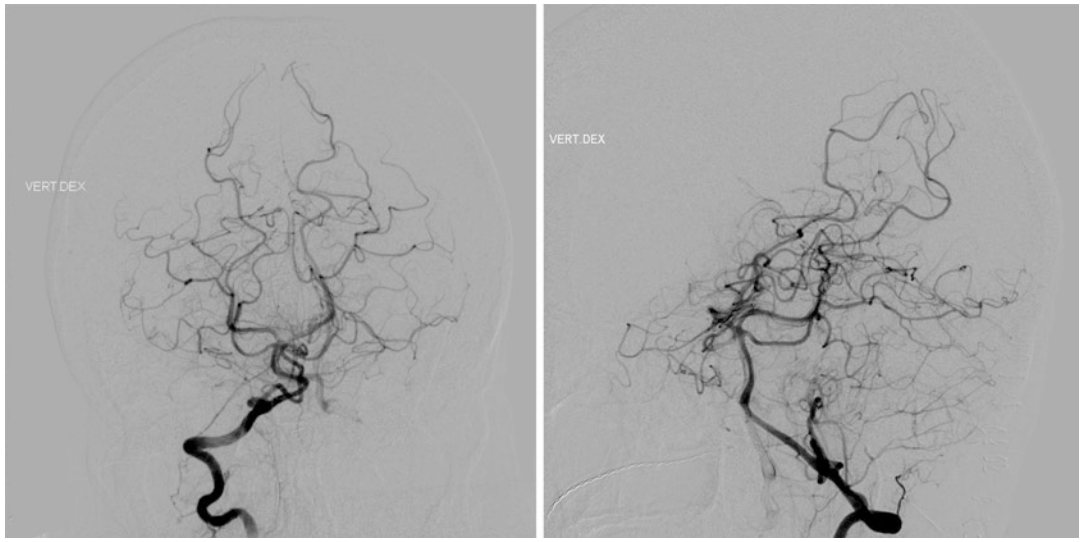

Fig. 21.5 Post-op vertebral artery angiogram demonstrates complete surgical removal of the AVM

21.6 Postoperative Care and Follow-Up

At the end of the procedure, immediate CT and CT angiography are performed directly before awaking the patient to check any postoperative hematoma and to find out any remnant in the AVM resection bed; then the patient is transferred to the neurosurgical ICU, where ECG, arterial blood pressure, central venous pressure, and other vital signs are recorded. Patients with small AVMs are woken up slowly over the period of several hours; those with large or complex AVMs are kept sedated and under moderate hypotension (mean arterial pressure 20% below the patient's normal level) by restricting fluid intake and hypotensive medications for several days. Careful repeat neurosurgical assessments are achieved.

Postoperative DSA is used to check if the AVM is completely eradicated.

Conclusion

Posterior fossa AVMs are very prone to rupture, and their treatment should be considered in almost every case. Microsurgical removal of posterior fossa AVMs is a big challenge in neurosurgical practice. Therefore, only experienced neurovascular team should operate on these lesions. The decision of whether to treat these lesions and choosing the best treatment modality is complex. A multidisciplinary team of neurosurgeons, neuroradiologists, and radiotherapists is important in the selection of the best management in each individual patient.

References

1. Peerless SJ, Hernesniemi J, Drake CG, Arteriovenous malformations of the posterior fossa. In: Wilkins RH, Rengachary SS, editors. Neurosurgery. New York: McGraw-Hill; 1996 p. 2463–76.
2. Yasargil MG. Microneurosurgery, Volume IIIA: AVM of the brain, history, embryology, pathological considerations, hemodynamics, diagnostic studies, microsurgical anatomy. New York: George Thieme Verlag; 1996. p. 11–2.
3. Verbiest H. Arterio-venous aneurysms of the posterior fossa, analysis of six cases. Acta Neurochir. 1961;9:171–95.
4. Laakso A, et al. Long-term excess mortality in 623 patients with brain arteriovenous malformations. Neurosurgery. 2008;63(2):244–53; discussion 253–5.
5. Kuhmonen J, Piippo A, Väärt K, Karatas A, Ishii K, Winkler P, Niemelä M, Porras M, Hernesniemi J. Early surgery for ruptured cerebral arteriovenous malformations. Acta Neurochir Suppl. 2005;94:111–4.

6. da Costa L, Thines L, Dehdashti AR, Wallace MC, Willinsky RA, Tymianski M, Schwartz ML, ter Brugge KG. Management and clinical outcome of posterior fossa arteriovenous malformations: report on a single centre 15-year experience. J Neurol Neurosurg Psychiatry. 2009;80:376–9.
7. Arnaout OM, Gross BA, Eddleman CS, Bendok BR, Getch CC, Batjer HH. Posterior fossa arteriovenous malformations. Neurosurg Focus. 2009;26(5):E12. 1–6.
8. Hernesniemi JA, Dashti R, Juvela S, Väärt K, Niemelä M. Natural history of brain arteriovenous malformation: a long-term follow-up study of risk of hemorrhage in 238 patients. Neurosurgery. 2008;63:823–31.
9. Laakso A, Dashti R, Seppänen J, Juvela S, Väärt K, Niemelä M, Sankila R, Hernesniemi JA. Long-term excess mortality in 623 patients with brain arteriovenous malformation. Neurosurgery. 2008;63:244–55.
10. Gross BA, Du R. Natural history of cerebral arteriovenous malformations: a meta-analysis. J Neurosurg. 2013 Feb;118(2):437–43.
11. Abecassis IJ, Xu DS, Batjer HH, Bendok BR. Natural history of brain arteriovenous malformations: a systematic review. Neurosurg Focus. 2014;37(3):E7.
12. Stapf C, Mohr JP, Pile-Spellman J, Sciacca RR, Hartmann A, Schumacher HC, Mast H. Concurrent arterial aneurysms in brain arteriovenous malformations with haemorrhagic presentation. J Neurol Neurosurg Psychiatry. 2002;73:294–8.
13. Hernesniemi JA, et al. Natural history of brain arteriovenous malformations: a long-term follow-up study of risk of hemorrhage in 238 patients. Neurosurgery. 2008;63(5):823–9; discussion 829–31.
14. Aki Laakso RD, Juvela S, Niemela¨ M, Hernesniemi J. Natural history of arteriovenous malformations: presentation, risk of hemorrhage and mortality. In: Laakso A, Hernesniemi J, Yonekawa Y, Tsukahara T, editors. Surgical management of cerebrovascular disease. Germany: Springer; 2010. p. 65–9.
15. Natarajan SK, Ghodke B, Britz GW, Born DE, Sekhar LN. Multimodality treatment of brain arteriovenous malformations with microsurgery after embolization with onyx: single-center experience and technical nuances. Neurosurgery. 2008;62:1213–25; discussion 1225–1226.
16. Nataf F, Merienne L, Schlienger M, Lefkopoulos D, Meder JF, Touboul E, Merland JJ, Devaux B, Turak B, Page P, Roux FX. Cerebral arteriovenous malformations treated by radiosurgery: a series of 705 cases. Neurochirurgie. 2001;47(2–3 Pt 2):268–82.
17. Mohr JP. A randomized trial of unruptured brain arteriovenous malformations (ARUBA). Acta Neurochir Suppl. 2008;103:3–4. Review.
18. Laakso A, et al. Risk of hemorrhage in patients with untreated Spetzler-Martin grade IV and V arteriovenous malformations: a long-term follow-up study in 63 patients. Neurosurgery. 2011;68(2):372–7; discussion 378.
19. Hernesniemi J, Keränen T. Microsurgical treatment of arteriovenous malformations of the brain in a defined population. Surg Neurol. 1990 Jun;33(6):384–90.
20. Lehecka M, Laakso A, Hernesniemi J. Common approaches. In: Laakso A, Lehecka M, Hernesniemi J, editors. Helsinki microneurosurgery basics and tricks. Helsinki: Department of Neurosurgery, Helsinki University Central Hospital; 2011. p. 111–93.