Vascular Malformations of the Central Nervous System: Case Studies

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Table of Contents

Introduction ............................................. 2
Arteriovenous Malformations ...................... 2
Cavernous Angiomas ................................. 8
Capillary Telangiectasias ............................. 11
Venous Angiomas ..................................... 11
Summary Points ........................................ 13
References ............................................. 13

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I. INTRODUCTION

Vascular malformations of the central nervous system (CNS) represent a heterogeneous group of lesions with various natural histories and treatments. CNS vascular malformations (Table 1) are commonly classified as belonging to 1 of 4 categories: (1) arteriovenous malformations (AVMs), (2) cavernous angiomas (ie, cavernomas, cavernous malformations), (3) capillary telangiectasias, and (4) venous angiomas (ie, developmental venous anomalies).1 Based on autopsy series, the incidence of venous angiomas is approximately 2%, AVMs are 1%, capillary telangiectasias are 0.7%, and cavernous angiomas are 0.4%.2 AVMs and cavernous angiomas commonly present with hemorrhage, seizures, or progressive neurologic deficit; treatment is often required. In contrast, capillary telangiectasias and venous angiomas are almost always incidental and/or asymptomatic and thus rarely require treatment. This manual describes the presentation, diagnosis, and treatment of all 4 CNS vascular malformations. Two case patients are presented to highlight specific features of the management of patients with AVMs or cavernous angiomas.

II. ARTERIOVENOUS MALFORMATIONS

CASE PATIENT 1 PRESENTATION

Patient 1 is a 40-year-old woman with a several-year history of headaches, initially believed to be menstrual migraines. Soon after her fortieth birthday, she experienced an increase in the severity and frequency of her headaches. Later, she noticed episodes in which her vision on the left became “cloudy” and she saw “spots.” On examination, a partial left visual field defect is noted. A computed tomography (CT) scan reveals a mass in the right occipital lobe, which enhances after contrast administration (Figures 1A and 1B). A subsequent magnetic resonance image (MRI) reveals a lesion comprised of multiple flow-voids (Figure 1C). Patient 1 is referred for an arteriogram, which demonstrates a 3- to 4-cm high-flow AVM in the right parieto-occipital region (Figure 2A).

GENERAL PRINCIPLES

Presentation

- AVMs commonly present with which of the following signs or symptoms?
A) Headache  
B) Seizures  
C) Hemorrhage  
D) Progressive neurologic deficit  
E) All of the above

The correct answer is E. Hemorrhage is the most common presentation in patients with cerebral AVMs, occurring in 50% to 60% of patients.\textsuperscript{3,4} Seizures are the next most frequent presentation, occurring in 11% to 33% of patients.\textsuperscript{5} About 5% to 35% of patients may experience the onset of a new type of headache before the discovery of their AVM.\textsuperscript{3} A small percentage of patients develop a progressive neurologic deficit. Neonates may occasionally present with high-output heart failure secondary to shunting through the AVM; vein of Galen malformations commonly present in this manner.

AVMs, or more specifically pial high-flow AVMs, are congenital lesions, thought to arise during the first trimester of fetal development. They consist of primitive arteries directly connected to veins without an intervening capillary network. Dysplastic brain is present between the vessels in the core or nidus of the AVM (Figure 3). AVMs are usually fed by multiple arterial branches, although outflow is commonly provided by only 1 or 2 dilated veins. The high-pressure and high-flow nature of these lesions makes them prone to hemorrhage. In addition, the shunting of arterial blood directly into the venous system can lead to poor perfusion and ischemia in the surrounding brain.

Natural History

- When compared with intracranial aneurysms, the yearly rate of rupture from a high-flow AVM:
  A) Is less than that of an aneurysm  
  B) Is equal to that of an aneurysm  
  C) Is greater than that of an aneurysm  
  D) Cannot be compared because AVMs never rupture  
  E) Cannot be compared because aneurysms never rupture

The correct answer is C. Unfortunately, the natural history of AVMs—in particular the rate of hemorrhage—remains poorly understood. Of the 4 categories
of vascular malformations discussed in this manual, perhaps the most is known about high-flow AVMs. In a long-term follow-up study, Ondra and colleagues found an annual rate of hemorrhage for high-flow AVMs of 2% for asymptomatic lesions and 4% for symptomatic malformations; this study has been quoted frequently. This same group placed the mortality rate at 1% per year, with a combined major morbidity and mortality of 2.7% per year.

Other studies have suggested a 10% to 15% risk of death and a 20% to 30% risk of major morbidity with each hemorrhage. In comparison, the risk of hemorrhage from an intracranial aneurysm is reported to be 1% to 2% per year, with a 30-day mortality rate after aneurysmal subarachnoid hemorrhage of almost 50%.

A history of seizures does not appear to adversely affect the chances of AVM re-hemorrhage. However,
various other factors, including small size of the AVM, venous stenosis or ectasia, periventricular location, history of previous hemorrhage, and the presence of intranidal or associated aneurysms have all been linked to an increased risk of hemorrhage.\textsuperscript{14–18} The relationship between aneurysms and AVMs appears to be more than sporadic, with several studies reporting a 2.7% to 14% incidence of aneurysms in patients with AVMs.\textsuperscript{19–23} In some situations, because of the high risk of mortality associated with aneurysm rupture, appropriate management of these patients may require definitive treatment of the aneurysm before AVM obliteration.\textsuperscript{19}

**Diagnosis**

- If treatment is contemplated in a symptomatic patient with an apparent AVM visible on CT and MRI, what would be the next most appropriate diagnostic study?
  - A) Xenon CT scan
  - B) Angiography
  - C) Perfusion or diffusion MRI
  - D) Lumbar puncture
  - E) MRI spectroscopy

  The correct answer is B. AVMs usually present during the third or fourth decades of life. A CT or MRI scan is usually the initial diagnostic study of choice. On CT scan, high-flow AVMs appear as a cluster of densely enhancing vascular structures (Figure 1). A noncontrast CT is the study of choice to detect hemorrhage from an AVM (Figure 4) because MRI scans will only show blood in the AVM and not the hemorrhage itself. However, MRI provides improved resolution when compared with CT and allows viewing of the AVM in multiple planes. On MRI, AVMs appear as a collection of flow voids, visible even on noncontrast images (Figure 1C). MRI is particularly useful for planning approaches for microsurgical resection and treatment with stereotactic radiosurgery.

  After a lesion suspicious for AVM is found on CT or MRI, an angiogram is necessary if any treatment is required. High-resolution, 4-vessel, digital subtraction angiography is the gold standard for AVM evaluation. Angiography continues to provide the best means of evaluating an AVM’s structure, including assessing the character, type, and location of its feeding arteries and draining veins. The dynamic nature of angiography also enables an assessment of the flow characteristics of the malformation. The hallmark of a high-flow AVM on angiography is the direct arteriovenous connection, giving rise to the rapid filling of the draining vein(s) (Figures 2A, 2B, and 2C). This rapid venous filling, referred to as early venous drainage, is readily seen during the arterial phase when normal veins are not visible. Because of the small risk of complications related to angiography, many advocate the performance of angiography only when treatment is contemplated.

**CASE PATIENT 1 FOLLOW-UP**

Patient 1 appears to have a symptomatic high-flow AVM. Given her relatively young age, she is felt to be at significant risk for morbidity secondary to the AVM during the remainder of her life; therefore, treatment is recommended. A multidisciplinary approach using endovascular embolization followed by microsurgical resection is selected based on the patient’s clinical status as well as the location and size of the AVM. Follow-up arteriograms obtained after embolization and surgery demonstrate a decrease in the size of the AVM followed by complete obliteration of the lesion (Figures 2B and 2C). Surgical pathology confirms the diagnosis of a high-flow AVM (Figure 3). Postoperative clinical examination reveals a persistent left visual field defect.

**GENERAL PRINCIPLES**

**Treatment**

- Clinical management of high-flow AVMs may include all of the following EXCEPT:
  - A) Microsurgical resection
  - B) Stereotactic radiosurgery
  - C) Observational follow-up
  - D) Needle biopsy and aspiration
  - E) Endovascular embolization

  The correct answer is D. Three major modalities are available for the treatment of AVMs: microsurgical resection, endovascular embolization, and stereotactic radiosurgery. Needle biopsy and aspiration are strongly contraindicated when a vascular malformation is suspected because of the high risk of inducing hemorrhage. In some situations, the risks of treatment may outweigh the benefits, and simple observational follow-up may be recommended.

  Microsurgical resection can provide an immediate cure and eliminate the future risk of hemorrhage. However, surgical resection may be limited by the size of the lesion or by the AVM’s location within deep or eloquent brain structures. In a retrospective series of 100 surgically cured AVMs, Spetzler and Martin\textsuperscript{24} reported minor postoperative deficits in 5% to 19% of patients and major deficits in 4% to 12%. The incidence of postoperative deficits depended on the location, size, and...
Figure 2. Lateral views of ICA angiograms obtained during the diagnosis and treatment of patient 1. (A) Pretreatment angiogram shows an AVM in the occipital lobe fed by dilated branches of the middle cerebral artery (solid arrows) and drained by a single superficial vein (open arrow). During this arterial phase image, minimal contrast remains in the ICA, suggesting extremely high flow. The draining vein is visible at this time, confirming “early venous drainage.” (B) After embolization, flow is slowed and the size of the nidus (arrow) has been greatly reduced. (C) Postoperatively, the nidus is no longer visible and early venous drainage is not apparent; however, some dilated arteries remain (arrow). AVM = arteriovenous malformation; ICA = internal carotid artery.

Figure 3. An H&E-stained histologic section of an arteriovenous malformation from patient 1. Note the thick-walled abnormal arteries (solid arrow) and the intervening dysplastic brain (open arrow). H&E = hematoxylin and eosin.
type of venous drainage of the AVM. Based on their results, the authors proposed an AVM grading system, which remains in widespread use.

Although endovascular embolization alone may cure some small AVMs, this technique is usually used as an adjuvant therapy to reduce the size of large AVMs before microsurgical resection or stereotactic radiosurgery. Various materials have been used to endovascularly occlude AVMs; the most popular methods currently in use include cyanoacrylate-based glues, polyvinyl alcohol particles, and detachable platinum coils. A large AVM may require multiple sessions of embolization before surgery or radiation.

Stereotactic radiosurgery is often used to treat AVMs in deep locations or those situated within eloquent brain structures. However, radiosurgery is limited by size, with the best results seen in lesions smaller than 3 cm in maximal dimension. The 3 major delivery systems for focused beam radiosurgery include linear accelerator–based (LINAC) programs, gamma radiation units (gamma knife), and heavy particle (helium ion or proton beam) systems. Various clinical series have demonstrated efficacy for all of these techniques. Three years after treatment, cure rates of 80% to 95% have been reported for lesions smaller than 3 cm that were treated with 20 to 25 GyE. These series also reported low complication rates of 2.5% to 4.5% for permanent neurologic deficits and 2.5% to 4.5% for transient deficits. Most complications arose from radiation toxicity and necrosis in the surrounding brain. Aside from the toxicity issues, the major disadvantage of radiosurgery is that during AVM obliteration, the risk of hemorrhage persists and may even be increased.

Regardless of the treatment modality used, once complete obliteration of the AVM has been documented by a post-treatment angiogram, the patient is generally considered cured. The use of routine delayed follow-up angiography is controversial. Certainly, late follow-up angiography may be required as part of a clinical research protocol or in cases of clinical uncertainty. Neurologic deficits present after AVM treatment tend to improve over time, but full resolution is not typical. Long-term neurologic follow-up may be necessary in selected patients, particularly those with persistent seizures or severe neurologic deficits.

The decision about whether and how to treat a patient with an AVM must be based on various factors. The patient’s clinical situation and the natural history of the lesion are perhaps the most important considerations when a physician decides whether to treat. The exact method of treatment can then be planned after a careful analysis of the AVM’s radiographic characteristics. Except

Figure 4. Hemorrhage from an arteriovenous malformation. (A) Noncontrast axial CT scan of a young woman showing an acute hemorrhage in the left thalamus. (B) Lateral view of a vertebral artery injection from her subsequent angiogram confirms the presence of an arteriovenous malformation. Note the associated aneurysm (arrow). CT = computed tomography.
for very small AVMs, optimal treatment today often uses a combination of modalities; the best results are typically achieved at specialized centers where all 3 major treatment modalities are available. Regardless of the modality used, the AVM must be completely obliterated (as documented by angiography) to eliminate any future risk of hemorrhage. Partial treatment is not an effective means of preventing future hemorrhage.

Spinal AVMs

AVMs can also occur in the spinal cord and represent about 4% of intraspinal masses. Although these lesions may be purely intradural and intramedullary, most are located within the dura rather than the CNS itself. Dural AVMs, especially the intracranial variety, are widely regarded as predominantly acquired lesions, with a natural history very different from the pial AVMs previously discussed. In general, patients with spinal AVMs present with back pain associated with a progressive neurologic deficit. However, about 10% to 20% of younger patients present with the sudden onset of myelopathy secondary to hemorrhage. Spinal angiography is challenging and relatively high risk but is required to confirm the diagnosis. At angiography, many spinal AVMs are actually found to be an arteriovenous fistula, with only a single arteriovenous connection. Successful treatment may then be accomplished by either endovascular embolization or microsurgical resection of the nidus or fistula.

III. Cavernous Angiomas

CASE PATIENT 2 PRESENTATION

Patient 2 is a 31-year-old, right-handed graduate student with a known cavernous angioma, who notes the sudden onset of partial left visual field loss while working on his thesis. Seven years previously, patient 2 was found to have a cavernous angioma when he presented with a dense left homonymous hemianopsia, which had subsequently resolved. Patient 2’s family history is remarkable for an uncle with an intracranial hemorrhage and a sister with a known cavernous angioma.

On examination of patient 2, a partial left visual field defect is noted to confrontation and is confirmed by formal visual field testing. An MRI scan demonstrates an irregularly enhancing mass in the medial right temporal lobe. The T1-weighted images reveal that the mass is composed of various signal intensities; on the long TR-weighted images, a rim of hypointense signal surrounding the lesion is apparent (Figures 5A and 5B).

GENERAL PRINCIPLES

Presentation

- Cavernous angiomas (cavernomas) may present with which of the following signs, symptoms, or both?
  A) Hemorrhage
  B) Progressive neurologic deficit
  C) Seizures
  D) Headache
  E) All of the above

The correct answer is E. Patients with supratentorial cavernous malformations most commonly present with seizures. The second and third most common presentations are hemorrhage and headaches, respectively. Most infratentorial cavernomas present with progressive neurologic deficits, possibly as a result of repeated minor hemorrhages. Although most cavernomas are sporadic in nature, about 15% of patients have a familial form, which localizes to chromosome 7q and is inherited in an autosomal dominant manner. Most patients with the familial form have multiple malformations.

Diagnosis

- The radiographic appearance of cavernous angiomas may include all of the following EXCEPT:
  A) Calcification on CT scan
  B) Hypointense periphery on T2-weighted MRI
  C) Feeding arteries and draining veins on angiography
  D) MRI evidence of multiple hemorrhages of different ages
  E) Multiplicity on CT or MRI

The correct answer is C. Cavernous angiomas or cavernomas are part of a group of vascular malformations collectively referred to as angiographically occult vascular malformations (AOVMs) or cryptic malformations. In other words, these vascular malformations are not visible on angiography. Also included in this category are capillary telangiectasias and some histologically true AVMs, which may have “burned out” through thrombosis or previous hemorrhage.

On histologic examination, cavernous angiomas appear as a collection of low-flow venous sinusoids without any intervening neural parenchyma (Figure 6). There are no large feeding arteries or draining veins. The brain immediately surrounding the malformation is usually gliotic and stained with hemosiderin. Like high-flow AVMs, cavernomas may occur anywhere in the CNS. However, lesions in the spinal cord are rare.
On CT scan, these malformations appear as a heterogeneous collection of various densities. Calcification is frequently present, as is evidence of hemorrhage. On routine MRI, the hallmark of a cavernous angioma is evidence of multiple hemorrhages that occurred over time (ie, at different ages), giving the lesion a “popcorn” like appearance. On long TR images, a hypointense rim is seen, resulting from hemosiderin deposition in the brain surrounding the malformation (Figures 5A and 5B). A gradient-recalled echo (GRE) sequence further accentuates this hemosiderin blos-soming and is therefore ideal for screening for even the smallest of malformations (Figures 7A, 7B, and 7C).

Natural History

Like vascular malformations in general, the natural history of cavernous malformations is poorly understood. Estimates of yearly hemorrhage rates range anywhere from less than 1% to almost 30%. However, when only prospectively documented symptomatic hemorrhages are recorded, a narrower range of 2.6% to 4.2% is apparent. Female sex, pregnancy, previous symptomatic hemorrhage, association of the cavernoma with a venous angioma, and deeply located (thalamus, basal ganglia, and brainstem) malformations have all been associated with an increased risk of hemorrhage. In addition, some authors have found a higher rate of bleeding for familial cavernous angiomas, suggesting that their natural history may be worse than that of their sporadic counterparts. Many hemorrhages from cavernous malformations are clinically silent and fatal hemorrhages are rare, yielding a low hemorrhagic risk in contrast to the high risk associated with high-flow AVMs.

Aside from hemorrhage, the major cause of morbidity from cavernous angioma is seizures. One recent study put the yearly risk of developing new seizures at 2.4%, with an overall yearly risk of seizures in patients with cavernous angiomas of 4.8%. CASE PATIENT 2 FOLLOW-UP

Given the natural history of this suspected cavernous angioma, as well as patient 2’s young age and his history...
of 2 symptomatic hemorrhages, treatment is recommended. Patient 2 undergoes a gross total microsurgical resection of the lesion (Figure 5C). Final pathology confirms the diagnosis of a cavernous angioma (Figure 6). Postoperative examination reveals a persistent left visual field defect. Serial visual field tests that are performed during the next several months show slight improvement in the field loss.

GENERAL PRINCIPLES

Treatment

- Which of the following treatments is most effective for a superficially located symptomatic cavernous angioma?
  A) Endovascular embolization
  B) Stereotactic radiosurgery
  C) Needle biopsy and aspiration
  D) Microsurgical resection
  E) Whole brain irradiation

The correct answer is D. As with high-flow AVMs, decisions regarding the appropriate management of cavernous malformations are made more difficult by the limited understanding of their natural history. In addition, the angiographically occult nature of these lesions eliminates endovascular therapy as a treatment option. The low risk of hemorrhage from asymptomatic cavernous malformations suggests that these lesions do not need to be treated. In the absence of definitive treatment, the avoidance of anticoagulants and antiplatelet agents is often recommended.

In symptomatic patients, microsurgical resection is the only definitive treatment and is therefore reserved mainly for those with progressive neurologic deficits, medically intractable seizures, and/or severe headaches. For surgically accessible lesions, resection can be quite straightforward, given the lack of large feeding arteries and the presence of a gliotic plane around the malformation. Surgical results are generally good, with most patients (75% to 95%) demonstrating clinical improvement or at least stable deficits. For deeply located lesions, surgical resection is more challenging and stereotactic localization techniques may be useful. However, even high-risk lesions in the brainstem, thalamus, or basal ganglia can be surgically resected with acceptable morbidity. In the brainstem, it may be necessary to wait until the lesion presents itself to the pial surface in order to establish a safe pathway for resection.

Treatment of cavernous malformations with stereotactic radiosurgery is controversial. Some studies have reported an increase in the rate of hemorrhage after radiosurgery, although others claim an apparent decrease in the bleeding rate, especially at longer follow-up intervals. Current treatment is probably best reserved for patients with symptomatic deep-seated AVMs that do not present to a pial or ventricular surface and are therefore considered inoperable.

Cure after surgical resection is best documented by serial MRI scanning. However, proof of cure after radiosurgery may be difficult to ascertain because the MRI appearance of the lesion may change only minimally. As such, careful long-term clinical and radiographic follow-up is probably warranted after any form of treatment.
IV. CAPILLARY TELANGIECTASIAS

Capillary telangiectasias consist of a network of abnormally dilated capillaries with intervening brain parenchyma. There are no abnormal feeding arteries, although drainage into a central enlarged vein has been reported.67 These lesions are thought to be clinically silent and are usually discovered incidentally at autopsy.5,68,69 On gross examination, the lesions appear similar to a cluster of petechial hemorrhages (Figure 8A). On microscopic sections, hemorrhage or gliosis in the brain parenchyma are not apparent; however, dilated capillary vessels devoid of muscle or elastic fibers can be seen (Figure 8B). Most capillary telangiectasias are found in the pons, and multiple lesions are common, similar to cavernous malformations.

Capillary telangiectasias are not visible on angiography, constituting about 3.8% of AOVMs.70 Occasionally, they may show up as incidental findings on MRI, where the malformations appear as punctate areas of decreased signal intensity on T2-weighted images and may demonstrate contrast enhancement with gadolinium administration.71 Because these lesions are thought to be clinically silent, no treatment is required. Capillary telangiectasias are of interest mainly because they may represent a potential developmental link with cavernous angiomas.72,73

V. VENOUS ANGIOMAS

Venous angiomas, also known as developmental venous anomalies (DVAs), are anomalous collections of normally functioning draining veins. On histologic examination, the veins themselves are normal and they are surrounded by normal brain parenchyma. Most are located in the deep white matter of the cerebral hemispheres or the cerebellum.75 No abnormal arteries are associated with these lesions; hemorrhage or calcification is unusual.75 The malformations eventually empty into the deep or superficial venous system.76

Venous angiomas are best visualized during the venous phase of angiography. They appear as a collection of smaller veins converging into a larger vein in a radial or “spoked-wheel” pattern, giving rise to the classic angiographic term of caput medusae (Figures 9A and 9B). Venous angiomas are easily distinguished from high-flow AVMs by their angiographic features. DVAs lack feeding arteries and, unlike AVM draining veins, these are seen during the venous phase of the angiogram instead of “early” in the arterial phase. Venous angiomas are also well visualized on contrast-enhanced CT scans and show up as contrast-enhancing flow-voids on MRI (Figures 10A and 10B).

Like capillary telangiectasias, venous angiomas are thought to be clinically silent.56,69 In fact, obliteration or removal of a DVA by any modality is likely to result in a venous infarction because the anomaly is providing venous drainage for a normal brain.77,78 The situation is somewhat complicated by the high association of venous angiomas with cavernous angiomas.79 Various symptoms—including headache, seizures, hemorrhage, and progressive neurologic deficits—have all been previously ascribed to venous angiomas.78–80 However, after more rigorous examination with CT and MRI, these symptoms have in general been attributed to associated cavernous angiomas, rather than the
Figure 9. Lateral views of a left ICA injection of a patient with an incidentally found venous angioma. (A) The arterial phase appears normal. (B) During the venous phase, a complex of radially converging abnormal veins (caput medusae) is seen (arrow), consistent with a developmental venous anomaly or venous angioma. ICA = internal carotid artery.

Figure 10. Axial CT and MRI scans of an incidentally found developmental venous anomaly. (A) On contrast-enhanced CT scan, the malformation appears as an anomalous area of linear enhancement (arrow). (B) A T2-weighted MRI scan of the same patient shows a linear flow void (arrow). CT = computed tomography; MRI = magnetic resonance imaging.
venous anomaly itself. As mentioned previously, symptomatic cavernous angiomas may be successfully treated with microsurgical resection. However, care must be taken to avoid resection of any associated venous anomaly to prevent postoperative venous infarction.

VI. SUMMARY POINTS

- CNS vascular malformations include AVMs, cavernous angiomas, capillary telangiectasias, and venous angiomas.
- AVMs and cavernous angiomas most commonly present with hemorrhage, seizures, progressive neurologic deficit, or headaches. Capillary telangiectasias and venous angiomas are usually clinically silent.
- AVMs are visible on CT, MRI, and angiography. Cavernous malformations are visible on CT and MRI but are angiographically occult. Capillary telangiectasias are occasionally visible on MRI but are most commonly found incidentally at autopsy. Venous angiomas can be seen on MRI, contrast-CT, and angiography.
- On a yearly basis, AVMs hemorrhage more frequently than do intracranial aneurysms. However, the morbidity and mortality associated with aneurysm rupture is considerably greater than that associated with AVM hemorrhage.
- AVMs may be treated with microsurgical resection, endovascular embolization, stereotactic radiosurgery, or a combination of these modalities. Microsurgical resection remains the treatment of choice for most symptomatic cavernous angiomas. Capillary telangiectasias and venous angiomas do not require treatment. Resection of a venous angioma may actually lead to venous infarction.

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