Vascular Malformations of the Brain—Overview and Classification

W. Caleb Rutledge, Kurtis I. Auguste, and Michael T. Lawton

1 Introduction

Vascular malformations of the brain are a heterogeneous group of disorders, including arteriovenous malformations (AVMs), arteriovenous fistulas (AVFs), cavernous malformations (CMs), telangiectasias and developmental venous anomalies. AVMs and AVFs shunt blood from the arterial to the venous circulation, while CMs, telangiectasias and developmental venous anomalies are non-shunting. While adults and older children with vascular malformations commonly present with headaches, hemorrhage, seizures or focal neurologic deficits, neonates, infants and young children may present with congestive heart failure (CHF) and multiorgan failure, intracranial hemorrhage, seizures, macrocephaly, hydrocephalus and developmental delay.

2 Arteriovenous Malformation

An AVM is a tangle of abnormal vessels or nidus, which is composed of dilated feeding arteries and arterialized draining veins without intervening capillaries that form a high-flow, low-resistance shunt (Fig. 1). AVMs have long been considered congenital lesions, arising from errors during embryogenesis when arteries and veins are without intervening capillaries, but their pathogenesis is not well understood. Most are solitary and occur sporadically without a clear genetic basis. In

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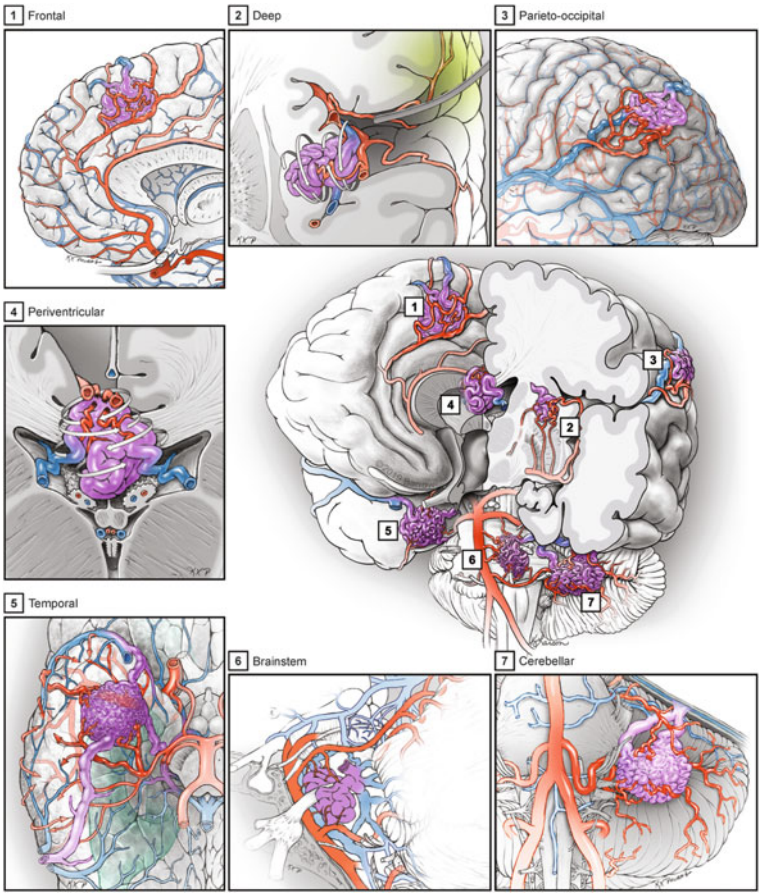

Fig. 1 AVM taxonomy. Although size, shape, and location make AVMs infinitely variable and unique, 32 anatomical categories exist based on their 7 locations: (1) frontal lobe; (2) deep (basal ganglia and thalamus); (3) parieto-occipital lobes; (4) periventricular/ventricular; (5) temporal lobes; (6) brainstem; and (7) cerebellum and the brain surface on which they are based (e.g., lateral, medial, basal, sylvian, and paramedian surfaces in the frontal lobe). Like genus and species that characterize animals, AVM type (location) and subtype (surface) characterize AVMs in a taxonomy that deciphers pathological anatomy and defines surgical strategy. Center image used with permission from Barrow Neurological Institute, Phoenix, Arizona. Parts 1-7 are from Lawton MT: *Seven AVMs: Tenets and Techniques for Resection*, Thieme, 2014 (reprinted with permission)

contrast, multiple AVMs usually signal an underlying genetic syndrome, such as Hereditary Hemorrhagic Telangiectasia (HHT), an autosomal dominant disorder characterized by mucocutaneous telangiectases and AVMs of major organs like lung, liver, and brain. Brain AVMs frequently occur in HHT type 1 from mutation in endoglin, a transforming growth factor- β (*TGF- β*) co-receptor.

The overall incidence of ruptured and unruptured AVMs is about 1 per 100,000 person-years. Peak presentation is in young adulthood during the second and third decade of life, but as many as 25% present during childhood [1]. Patients present

with headaches, hemorrhage, seizures or focal neurologic deficits from mass effect. Children may be more likely to present with hemorrhage, despite absence of high-risk features including venous ectasia and feeding artery aneurysms [2]. The presence of a single draining vein or deep venous drainage, particularly in smaller AVMs, increases the risk of hemorrhage in children [3, 4]. Once ruptured, the risk of another hemorrhage increases significantly [5, 6]. However, children with AVM hemorrhage often have better outcomes and are more likely to make a functional recovery than adult patients [7].

AVMs are classified using the Spetzler-Martin and Lawton-Young grading systems. Both grading systems estimate the risk of surgery. The Spetzler-Martin system includes size, eloquence of surrounding brain and venous drainage patterns [8]. The Lawton-Young system supplements the traditional Spetzler-Martin system by incorporating additional factors important to surgical selection and outcome, including patient age, hemorrhagic presentation and compactness [9, 10]. More recently, Lawton described 7 types of AVMs depending on their location in the brain—frontal, temporal and parietal-occipital lobes, ventricles, deep central core, brainstem and cerebellum (Fig. 1). Within each type of AVM are subtypes defined by the brain surface on which the AVM is based, with each subtype characterized by unique arterial supply, draining veins, eloquent surrounding structures, surgical approach, and management strategy. As examples, temporal lobe AVMs are subtyped into lateral, medial, basal and Sylvian subtypes, and cerebellar AVMs are subtyped into subocciptal, tentorial, vermian, petrosal, and tonsillar subtypes. With this new classification, the broad spectrum of brain AVMs is simplified to 32 different AVM subtypes, which is meant to inform the learning and descriptive processes and guide surgical planning.

Management of unruptured AVMs is controversial as the risk of treatment-associated morbidity and mortality must be weighed against the risk of spontaneous hemorrhage [11]. Conservative management includes anticonvulsants for patients with seizures, but currently there are no medications to prevent hemorrhage. Initial hemorrhage is the strongest predictor of future hemorrhage, but associated aneurysms, deep location, exclusively deep venous drainage and increasing patient age also increase the risk of hemorrhage [6, 12–15].

Modern management of AVMs is multimodal and includes microsurgical resection, endovascular embolization and stereotactic radiosurgery in addition to conservative observation [16–21]. Microsurgery remains the mainstay of treatment, as outcomes in patients with favorable grades are excellent [22–24]. Children may have better outcomes than adults after microsurgical resection of AVMs [25]. Endovascular embolization is most commonly used as preoperative adjunct to microsurgical resection. Stereotactic radiosurgery is an alternative to microsurgical resection, particularly for small AVMs with a single draining vein [26], but patients remain at risk of hemorrhage during the latency period and may experience delayed adverse radiation effects [16, 27]. Volume-staged radiosurgery is an option for high-grade AVMs not amenable to microsurgical resection or conventional single-session stereotactic radiosurgery [28]. Regardless of treatment, the goal is obliteration of the AVM and elimination of the risk of future hemorrhage.

In adults, complete angiographic obliteration is curative and eliminates the risk of future hemorrhage. However, in pediatric patients there is a small risk of AVM recurrence, even after angiographic obliteration [29–32]. Diffuse AVMs may be more likely to recur [30, 33]. Most recurrences are detected within a year of treatment [30, 33]. Thus, follow-up angiography is crucial to detect recurrences in children.

3 Dural Arteriovenous Fistulas

Dural arteriovenous fistulas (dAVFs) are less common than AVMs and a rare cause of intraparenchymal hemorrhage. Like AVMs, there is an abnormal connection between meningeal arteries and dural venous sinuses or cortical veins, but dAVFs lack a true nidus. Feeding arteries are derived from either pia or dura. The most common location is the junction of the transverse and sigmoid sinus, but dAVFs also occur at other locations including the cavernous sinus, tentorium, superior sagittal sinus and anterior cranial fossa.

dAVFs are classified according to pattern of venous drainage in the Borden and Cognard classifications [34, 35]. The key feature is direction of venous drainage. Borden type I dAVFs drain directly into dural venous sinuses or meningeal veins with no cortical venous drainage. Similarly type II lesions drain into dural venous sinuses or meningeal veins, but also have retrograde drainage into cortical subarachnoid veins. Borden type III dAVFs drain directly into cortical subarachnoid veins. Cognard described five types of dAVFs based on the direction of venous drainage, but also type of venous outflow (nonectatic vs ectactic). Cognard type I dAVFs have anterograde flow only. Type II dAVFs reflux into the sinus (IIa), cortical veins (IIb) or both (IIa + b), while type III fistulas have direct cortical venous drainage without venous ectasia and type IV dAVFs have direct cortical venous drainage and ectasia. Finally type V dAVFs have spinal venous drainage.

Most dAVFs have a benign clinical course with some even spontaneously regressing, but others cause symptoms such as an audible bruit or intraparenchymal hemorrhage from rupture, although patients with hemorrhage from rupture of dAVF may have a lower morbidity and mortality than patients with intraparenchymal hemorrhage from other causes [36]. Patients with high-grade lesions with cortical venous drainage are at highest risk of hemorrhage [37]. Symptomatic patients and those with anterior cranial fossa or tentorial dAVFs often have cortical venous drainage and are at higher risk for hemorrhage or progressive neurologic deficits, while transverse-sigmoid and cavernous sinus dAVFs are most often benign [38]. dAVFs may result in high-output heart failure, developmental delay and obstructive hydrocephalus in children. Children with dAVFs may be more likely to have progressive enlarging or recurrent fistulas [39].

Treatment of AVFs is also multimodal and includes microsurgical resection, endovascular embolization with liquid embolic agents such as Onyx, and stereotactic radiosurgery for lesions refractory to surgical or endovascular treatment. The

goal of treatment is occlusion of the fistula or disconnection of the feeding arteries and draining veins. Endovascular embolization is safe, effective and curative for the majority of dAVFs and is usually considered as the first-line treatment, with surgery reserved for those that are not completely obliterated endovascularly [40–44]. Recurrence following angiographic cure in patients treated with Onyx embolization can occur, and long-term follow-up is recommended [45]. Clinical outcomes in children with dAVFs undergoing endovascular or combined endovascular and surgical treatment are excellent [46–49]. Workup for underlying genetic syndromes or hypercoagulability is indicated.

4 Cavernous Malformations

Along with AVMs, CMs are common vascular malformations of the brain, and up to 25% of CMs occur in children (Fig. 2). They are discrete lesions, composed of thin-walled, endothelial-lined caverns filled with blood, but devoid of smooth muscle or intervening brain parenchyma. Like AVMs, they often occur sporadically as solitary lesions, but may be multiple and inherited. Familial cases are inherited in an autosomal dominant manner due to mutations in *CCM1*, *CCM2* and *CCM3* genes. Acquired CMs are far less common, but may develop in children after whole brain radiation therapy.

Microsurgical resection is the treatment of choice for symptomatic patients. Resection of pediatric CMs is safe and effective with low rates of recurrence and excellent seizure control, particularly for supratentorial lobar lesions [50–53]. Resection of brainstem CMs is associated with higher surgical morbidity and mortality; however, new deficits from recurrent hemorrhage arise in children managed conservatively [54–57].

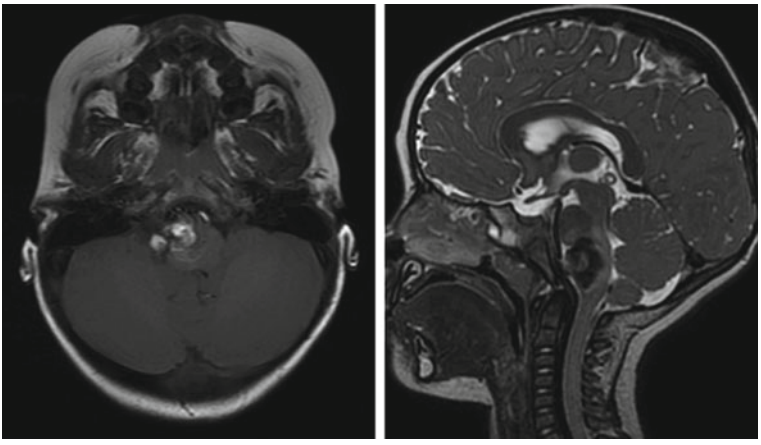

Fig. 2 Axial T1 (left) and Sagittal T2 (right) weighted magnetic resonance images showing 2.5 cm pontomedullary cavernous malformation in a two year-old female who presented with left hemiparesis and cranial neuropathies

5 Vein of Galen Malformation

Veins of Galen malformations are rare congenital vascular malformations occurring in the choroidal fissure. They are characterized by multiple arteriovenous shunts draining into a dilated median prosencephalic vein of Markowski, the embryonic precursor of the vein of Galen. They are most frequently diagnosed in neonates and infants with high-output CHF or seizures, or in young children with macrocephaly, hydrocephalus or developmental delay. Endovascular embolization using n-butyl cyanoacrylate (NBCA) or liquid embolic agents such as Onyx is the primary treatment modality [58, 59]. Treatment is generally delayed until 6 months of age. Many treated children now survive with normal neurologic development.

6 Conclusions

Vascular malformations of the brain are an important cause of stroke and early morbidity and mortality in children and young adults. AVMs and CMs are the most common vascular malformations in children. While adults commonly present with headache or focal neurologic deficits from hemorrhage or mass effect, infants and young children more commonly present with CHF, macrocephaly, hydrocephalus or seizures. Screening for underlying genetic syndromes or hypercoagulability is indicated. While many vascular malformations are benign, some patients may be symptomatic or at high risk of hemorrhage. Treatment is safe, effective and curative in appropriately selected patients.

Pearls.

- Vascular malformations of the brain are an important cause of stroke and early morbidity and mortality in children and adults.
- Their pathogenesis is not well understood and they may be congenital or acquired. Workup for underlying genetic syndromes or hypercoagulability is indicated in infants and children.
- AVMs and AVFs are characterized by abnormal connections between arteries and veins and shunt blood from the arterial to venous circulation.
- Infants with vascular malformations of the brain causing arteriovenous shunting may present with congestive heart failure, macrocephaly, hydrocephalus or seizures.
- AVMs are classified using the Spetzler-Martin or Lawton-Young Supplemental grading scale.
- Microsurgical resection is the treatment of choice for appropriately selected patients with low-grade AVMs and is safe and effective in children.
- dAVFs are classified using the Borden or Cognard classifications according to pattern of venous drainage. dAVFs with cortical venous reflux are at higher risk of hemorrhage.

- Endovascular embolization with Onyx is safe, effective, and may be curative in children with dAVFs, but surgery should be considered when endovascular occlusion is incomplete.
- Cavernous malformations are non-shunting lesions that nonetheless have associated hemorrhagic risk, making microsurgical resection the treatment of choice in symptomatic patients.

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