10. Arteriovascular Malformations

Arteriovascular malformations (AVMs) are the most common cerebrovascular malformation, consisting of direct communication between the arterial and venous circulations without intervening capillaries. Their most serious complication is hemorrhage (4% annual rate) with a yearly mortality of 1%. Symptoms of AVMs include headache, seizures, and neurologic defects. AVMs are associated with a variety of inherited conditions such as Sturge-Weber (port-wine stain, mental retardation, glaucoma, and seizures), hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu - mucocutaneous telangiectasias and AVMs), and Wyburn-Mason (midbrain AVM, facial nevus in distribution of the trigeminal nerve, and retinal angioma ipsilateral to the facial nevus).
The most common major feeding vessel for a brain AVM is the middle cerebral artery, and 80% of lesions are supratentorial. Classically, an AVM is visualized on MR (arrow, Fig. 10.1 A) as a tangled nidus of dilated vessels supplied by enlarged feeding arteries, with multiple enlarged, tortuous draining veins. AVM arterial supply is most frequently pial but may be dural, especially in infratentorial malformations. Feeding arteries are identified by location and dilatation. As shown in Figure 10.1 B where the anterior cerebral artery is feeding an AVM, TOF MRA may be useful in localizing the feeding artery. Aneurysms within feeding arteries occur in approximately 10% of patients and often regress after AVM treatment. Draining veins (Fig. 10.1 C, D black arrow) appear even larger than feeding arteries, and drain into deep or cortical veins. Because of rapid shunting, draining veins

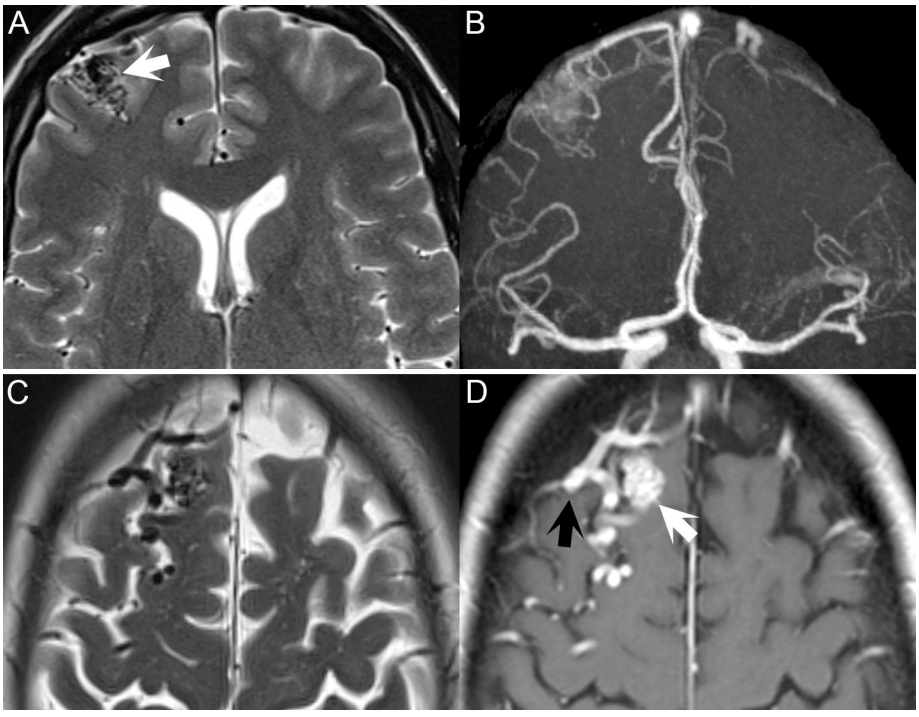

Fig. 10.1

appear as serpentine flow voids on MRI. This differs from the intermediate or high SI typically seen with slow-flowing veins. Contrast administration demonstrates prominent enhancement of the nidus (Fig. 10.1 D, white arrow) and slower flowing venous blood. Rapidly moving arterial blood may still not enhance. Pulsation artifacts, which often appear on non-enhanced images, may be more prominent with contrast administration. With smaller, untreated lesions, the typical appearance is that of a nidus and associated large draining veins, without gliosis or edema, and little mass effect. MRI is also very sensitive for the detection of superficial siderosis, related to repeated subarachnoid hemorrhage, and hemosiderin – from prior parenchymal hemorrhage with an AVM. On non-enhanced FSE images, AVMs may be difficult to distinguish from hemorrhagic components (i.e. hemosiderin) or areas of dense calcification as the low SI of both of these may be confused for flow voids. These entities may be distinguished on GRE where flowing blood appears of high SI, in contrast to the low SI of dense calcification or hemosiderin.

MRA is comparable to invasive angiography in assessing AVM nidus size and may also demonstrate feeding arteries (Fig 10.1 B) and allow visualization of associated aneurysms. Nidus size dictates course of therapy and inversely correlates with prognosis. MRA has several technical drawbacks that must be understood for proper image interpretation. Complex or tortuous arterial flow may appear as signal voids in feeding vessels and some draining veins may not be visualized. Detection of AVM-associated thrombus on TOF MRA may also be difficult as methemoglobin clot is of high SI—indistinguishable from flow. PC MRA avoids this latter problem, allowing for differentiation of thrombus from flow. Catheter angiography, for now, remains the standard for pre-operative evaluation of AVMs due to its more accurate detection of feeding and draining vessels. However, MRI and MRA are extremely useful, non-invasive pre-operative adjuncts and are heavily relied upon for post-operative followup.

Dural-based AVFs, unlike the congenital AVMs described above, are usually acquired lesions, often secondary to trauma. These lesions may originate in areas of venous or sinus thrombosis where collateralization has occurred. The most common site is the skull base, specifically involving the transverse and/or sigmoid sinus. Dural AVFs in this location are usually fed by the external carotid artery. Complications include venous infarction, parenchymal hemorrhage and subdural hematoma. MRA is essential for detection of these lesions when they are adjacent to the inner skull table as both vascular flow and cortical bone appear as signal voids on conventional MRI. While MRA is better for direct evaluation of dural vessels, clues to the diagnosis of a dural AVF on MRI include sinus thrombosis and dilated cortical veins.