

40. Vascular Abnormalities

Vascular pathology within the thecal sac includes structural abnormalities such as arteriovenous malformations (AVM) and dural arteriovenous fistulas (dAVF), in addition to ischemic and hemorrhagic disease resulting from these and other etiologies. Within the category of spinal vascular malformations, there are actually 4 types, type 1 being the dAVF, type 2 the intramedullary glomus-type AVM – similar to a brain AVM, type 3 being the juvenile AVM, and type 4 being the perimedullary/pial AVF.

Arteriovenous malformations are congenital lesions (associated with Osler-Weber-Rendu) that consist of a nidus of pathologic vessels between enlarged feeding arteries and draining veins. The nidus is typically located within the cord, and high flow into the nidus may result in aneurysms within the feeding spinal arteries. This increased flow may similarly result in ischemia of adjacent cord parenchyma through a steal phenomenon or venous hypertension. On MRI AVMs appear as multiple flow voids representing the nidus along with enlarged, extramedullary (often anterior) feeding vessels. T1 and T2WI best identify abnormalities within the cord and CSF, respectively. CSF pulsation artifacts on T2WI may occasionally mimic an AVM, but since the enlarged draining veins of an AVM brightly enhance, contrast administration may aid in this distinction and also in the detection of smaller lesions. High cord SI on T2WI adjacent to an AVM on MRI may represent gliosis or edema resulting from the mechanisms above. Intramedullary hemorrhage may also be present, its MRI appearance varying with the stage of blood products. As opposed to the glomus AVM (intramedullary) discussed above, juvenile AVMs (extradural-intradural) are significantly larger, occupying the entirety of the canal at a given level. These are also the rarest of spinal AVMs.

In comparison to cord AVMs, dAVFs are much more common and are acquired lesions favoring the lower cord. They consist of feeding arteries draining directly into enlarged veins. Two types of AVFs are defined in the spine: dural (most common) and pial AVFs (type 4). Dural AVFs lead to venous stasis and in some cases infarction. They occur along the dorsal aspect of the lower cord and conus and are fed by a single radicular artery through a dural branch. Secondary to increased venous flow, dural veins dilate, transmitting elevated venous pressures to cord veins, resulting in myelopathy and edema which may both be visualized as high cord SI on T2WI (Fig. 40.1 A). Typically these SI changes involve the length of the cord from the conus to lower thoracic spine and are thus more extensive than the SI changes present in demyelinating or inflammatory disorders. Even more suggestive of a dAVF is the presence of dilated pial veins on T2WI - seen as low SI flow voids against high SI CSF. In Figure 40.1 A, these serpiginous flow voids are present



along both the ventral and dorsal aspects of the cord in the lower thoracic spine and even within the cauda equina. CSF pulsation artifacts may mimic, in rare cases, such flow voids. Furthermore, the low SI appearance of the abnormal vasculature may be lost in areas of particularly slow venous flow as stagnant protons remain in-plane for the duration of the spin echo (see Chapter 12). In any case, contrast-enhanced T1WI as well as contrast enhanced MRA reliably demonstrate enhancing, engorged pial veins. DSA provides the definitive diagnosis with identification of feeding and draining vessels: the dural AVF in Figure 40.1 was found on (B) DSA to be fed by a branch of the right T11 intercostal artery. In distinction to dural AVFs, the anomalous fistula of a pial AVF is fed

Fig. 40.1

directly by a spinal artery (which can be either the anterior or posterior spinal artery). Cavernous malformations of the spinal cord—another important vascular abnormality—are discussed in Chapter 36.

Cord ischemia and infarction result from many causes, including atherosclerosis, vasculitis, embolism, infection, radiation, trauma, surgery, and spontaneous dissection. DWI is infrequently used in imaging of the cord due to marked susceptibility artifacts. T2WI demonstrate high SI within the cord (Fig. 40.2 A) often confined to the metabolically active gray matter, and correlating with vasogenic edema. Differential considerations for this appearance are broad and include multiple sclerosis, transverse myelitis, neoplasia, and the venous hypertensive changes described the preceding paragraph. Furthermore, on sagittal images, ischemic changes may not be apparent unless the cord is completely in-plane (that is, not oblique to the imaging plane) for its entire length. T1WI may only demonstrate focal cord enlargement (Fig. 40.2 B), although enhancement may occur post-contrast. The key to diagnosing cord ischemia thus lies in the recognition of the longitudinal and cross-sectional distribution of the spinal arteries. The dorsal columns, in which isolated ischemia is rarely seen, are supplied by the paired posterior spinal arteries, however the remainder of the cord is supplied by the single, midline anterior spinal artery (ASA). Although it is continuous, various radiculomedullary arteries feed the ASA at its major portions (cervicothoracic,

midthoracic, and thoracolumbar), and full collateral flow between these portions does not occur. Because the midthoracic ASA and its portions from the low thoracic cord to the conus are fed by single radicular vessels, these areas of the cord, along with watershed areas at the junctions of the major cord regions, are particularly prone to infarction. For example, the artery of Adamkiewicz (which typically arises from a left posterior intercostal artery) alone feeds the lower thoracic cord and conus. Paraplegia due to spinal cord infarction is a rare but devastating complication of open abdominal aortic aneurysm repair, with one cause being occlusion of the artery of Adamkiewicz. This has been reported as well with endovascular repair.



Fig. 40.2