4. Meningiomas

Meningiomas are far and away the most common extra-axial tumor. They occur most often in females. Risk factors including ionizing radiation, head trauma, and likely high exposure to estrogen and progesterone. Meningiomas are most commonly located over the convexity near the midline, but may occur, in decreasing order of frequency: along the lateral convexity, the sphenoid ridge, olfactory groove, suprasellar-parasellar region, and in the posterior fossa.

Meningiomas may be relatively difficult to visualize on non-enhanced MRI as they tend to be relatively isointense to brain on T1WI, T2WI, and FLAIR (Fig. 4.1 A). The presence of adjacent vasogenic edema, demonstrating increased SI on T2WI and decreased SI on T1WI, may aid in lesion recognition on unenhanced scans. Approximately half of meningiomas, however, do not demonstrate significant associated edema (Fig. 4.1 A). Thus, contrast administration (as in Figure 4.1 B) greatly aids in the detection of these lesions. Due to their lack of a BBB, meningiomas demonstrate prominent (and typically homogeneous) enhancement—a finding useful for lesion identification, characterization and distinguishing surrounding edema (Fig. 4.1 C, asterisk) from the tumor itself (Fig. 4.1 D). The dura adjacent to a meningioma often enhances as well (Fig. 4.1 D), with the presence of a “dural tail” characteristic, although this is occasionally seen with other lesion types. Meningiomas may have mixed SI on T2WI secondary to cystic changes or areas of dense calcification.

Although MR does not demonstrate calcifications well, when extremely dense (similar to cortical bone) these are visualized as areas of low SI on both T1 and T2WI. These findings can be more obvious on gradient echo imaging (GRE) and on imaging at higher-field (3 T). Hypointensity on GRE, however, is not specific for calcification as products of hemorrhage
can appear similar (although these would not be low SI on T1WI). MR is vastly superior to CT in localizing meningiomas as extra-axial. A broad margin along the dura (Fig. 4.1 B, D) and the presence of an enhancing dural tail (Fig. 4.1 D) strongly support the diagnosis of meningioma. Even more specific is the so-called cleft sign (Fig. 4.1 C) whereby CSF is visualized, usually best as a hyperintense cleft on T2WI, intervening between the tumor and brain parenchyma. As in Figure 4.1 C, an additional rim of hypointense parenchyma may separate the CSF cleft from surrounding parenchymal edema. Meningiomas within the cavernous sinus may displace the dura, which is then seen as a lateral hypointense line between the tumor and temporal lobes on post-contrast scans. Intra-axial tumors may grow outward to involve the dura, but only extra-axial tumors demonstrate interposed structures between the tumor and brain parenchyma. Another clue to the extra-axial location of these tumors is the bowing of adjacent white matter, due to compression of otherwise normal brain by the lesion. Meningiomas may also involve the vasculature or venous sinuses. Within the cavernous sinus, they can displace or encase the carotid artery—the latter finding may be confused for atherosclerosis on CT or digital subtraction angiography but is easily visualized with MRI. A tumor displacing the carotid artery from its normal position is most likely a meningioma, as similarly located pituitary macroadenomas tend to encase it. Meningiomas may invade the venous sinuses as well, such invasion being demonstrated on enhanced T1WI (Fig. 4.2, black arrow showing transverse sinus invasion) but may also be seen, prior to contrast administration, on 2D time-of-flight MR venography (TOF MRV). Postcontrast T1WI show high SI venous sinuses abutted by the enhancing soft tissue of the meningioma. 2D TOF MRV depicts venous flow as high SI, which with an adjacent meningioma, demonstrates irregular contour or, if the obstruction is complete, absence of flow within the sinus.

Meningiomas are typically benign (WHO grade I) and slow-growing. Atypical (grade II) and anaplastic (malignant, grade III) meningiomas are less common and exhibit restricted diffusion, likely from a combination of necrosis, decreased cytoplasmic space, and
decreased extracellular space (from tumor proliferation). En plaque meningiomas pose a particular diagnostic and therapeutic challenge. These tumors grow in a "carpet-like" fashion along the surface of the brain and frequently infiltrate through the dura and adjacent bone, thus rendering total resection impossible. Osseous invasion of meningiomas may be seen as bone thickening, and the tumors may also be visualized within the diploic space. Because en plaque lesions may simply appear as dural thickening, and are obscured due to beam hardening artifact from the adjacent skull, they are not seen on CT unless osseous invasion occurs. Even then, MRI comparatively easily makes the diagnosis because of the marked enhancement and extra-axial location of the lesion. When multiple or occurring in childhood, meningiomas (Fig. 4.2, black arrow) are often associated with neurofibromatosis type 2—an inherited condition for which presentation with bilateral vestibular schwannomas is pathognomonic (Fig 4.2, white arrows).