19. Parenchymal Infections

Pyogenic abscesses most frequently arise hematogenously, seeding the brain at the gray-white matter junction. They may also occur as sequelae of contiguously spreading sinus infections (see Ch. 20) or trauma. *Streptococcus* is the most common etiologic organism, followed by *staphylococcus* and *pneumococcus*. Abscesses favor the frontal and parietal lobes within the MCA distribution, and their early appearance is one of a diffuse cerebritis. The lesion at this stage enhances heterogenously, and demonstrates high SI on T2WI with low to moderate SI on T1WI. Within the first week, a central necrotic area forms, an area subsequently enveloped by collagen in the second week. This collagen wall appears as a rim of low SI on T2WI and may be surrounded by a disproportionate amount of edema (Fig. 19.1 A). The central area is necrotic and typically demonstrates restricted diffusion (Fig. 19.1 B) due to its pustulant contents. Central ADC values rise with treatment, and any return in diffusion restriction foretells a recurrence in infection. Similar DWI findings may be seen, although much less common, in necrotic primary or metastatic cancers, which can also demonstrate ring-enhancement and thus make differentiation from a pyogenic abscess difficult. In pyogenic abscesses, the enhancement (Fig. 19.1 C) is distinguished by the

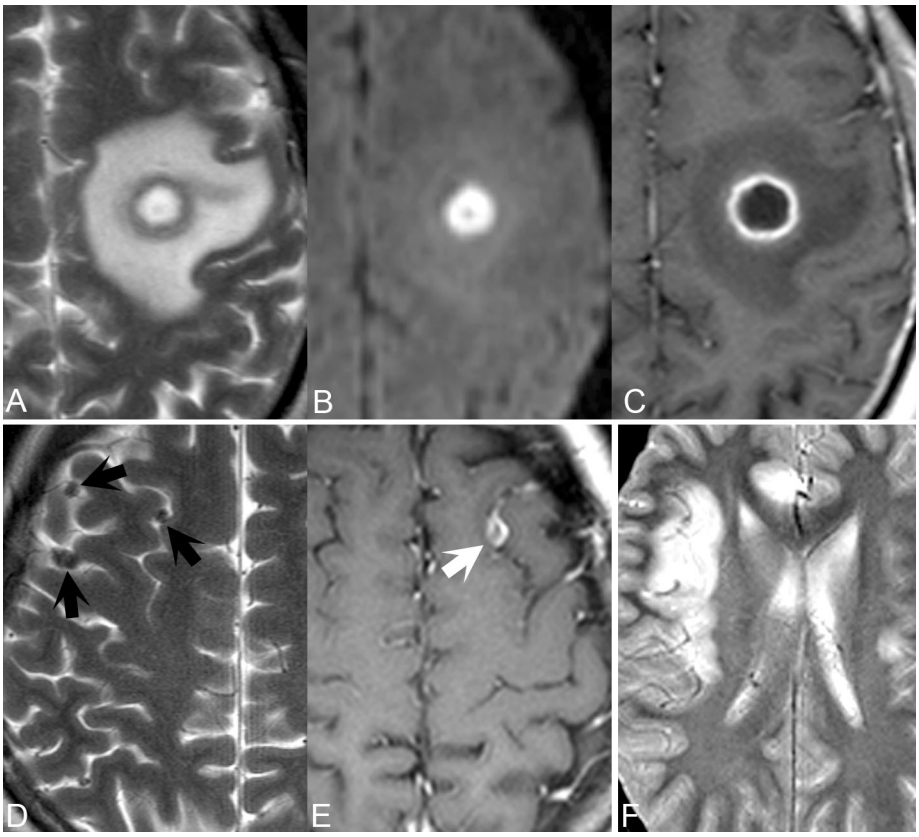

Fig. 19.1

completeness, uniformity, and lack of nodularity of the ring, although daughter abscesses may mimic nodularity. Ring-enhancement may persist for months after treatment and is a less reliable marker than ADC values for monitoring treatment response. Rupture into a ventricle with subsequent ependymitis can occur, although this is rare. Imaging findings in this instance include increased intraventricular SI on FLAIR scans along with ventricular margin enhancement on post-contrast T1WI.

Neurocysticercosis—a common cause of adult-onset seizures in the developing world—is caused by the pork tapeworm *Taenia Solium*. Within the CNS, neurocysticercosis can involve the spinal cord, ventricles, brain parenchyma, and the subarachnoid space (Fig. 19.1 D-E, arrows). Within these structures, the larvae progress through several stages. Larvae in the vesicular stage rarely enhance, and the cysts are typically seen as CSF-like SI. The colloidal vesicular phase is associated with larval death and degeneration, demonstrating a thick, enhancing capsule with surrounding edema on MRI. Fluid within the cyst, which appears as high SI on T2WI, is hyperintense to CSF on FLAIR and T1WI due to its high protein content. With particularly mucinous fluid, high SI is seen on all conventional sequences. Progression to the granular nodular stage is marked by diminished edema with continued enhancement, often in a rim pattern (Fig. 19.1 E, white arrow). By this time, the cyst itself has shrunk into a nodule. The chronic lesions of neurocysticercosis are typified by an almost complete absence of edema and moderate SI on T2WI with areas of low SI representing dense calcifications (Fig. 19.1 D, black arrows) which are best visualized on GRE.

Encephalitis involves the brain parenchyma more diffusely than the lesions above and is most frequently viral in origin. In adults, Herpes Simplex (HSV) type 1 is a common culprit, presenting in a typical pattern initially affecting one of the temporal lobes. As seen in Figure 19.1 F, lesions may eventually extend to involve the bilateral temporal and inferior frontal lobes. Increased edema in these areas leads to high SI on FLAIR and T2WI (Fig. 19.1 F), although the presence of hemorrhage—a frequent finding—may interfere with the expected low SI appearance of edema on T1WI (due to the presence of high signal intensity methemoglobin). Enhancement, particularly of the adjacent meninges, is frequently present in the acute phase of the disease. Diffusion is initially restricted in HSV encephalitis (and may precede changes on FLAIR), but then may recover or even increase over baseline in later, more edematous lesions. Late in the disease, atrophy and parenchymal destruction can be seen. DWI abnormalities and edema are seen early in neonatal HSV infection – which is usually caused by HSV type 2 – with variable brain involvement (no specific anatomic predilection). MRI characteristics of the developing brain complicate the detection of neonatal encephalitis. The use of T2-weighted sequences

optimized for adult imaging leads to low sensitivity to brain edema in neonatal HSV encephalitis – with specific neonate optimized scan protocols advocated. Late imaging findings following neonatal infection include microphthalmia, hydrocephalus, atrophy, microcephaly and brain calcifications, with mental retardation seen clinically.