18. Multiple Sclerosis

MR is the most sensitive test for multiple sclerosis (MS), although imaging findings may be confused with those of other diseases, in particular small vessel ischemia. Characteristic imaging findings in a younger, female patient favors MS, but given the high prevalence of small vessel disease, older patients should not be diagnosed with MS solely on the basis of imaging. According to the last (2010) revision of the McDonald Criteria, the diagnosis of MS can be based on objective demonstration of dissemination of lesions in both space and time on clinical grounds alone or by integration of clinical and MR findings. Dissemination of lesions in space is defined specifically for MR by demonstration of one or more T2-weighted high SI lesions in at least 2 of the following 4 CNS areas – periventricular, juxtacortical, infratentorial and spinal cord. Dissemination of lesions in time is defined on MR by a new T2-weighted high SI lesion or an enhancing lesion on follow-up MR (with reference to a baseline exam) or by the simultaneous presence of asymptomatic enhancing and non-enhancing lesions.

Figure 18.1 A demonstrates the typical pattern of disease on FLAIR scans, with small focal areas of abnormal high SI—correlating pathologically with edema and gliosis—scattered throughout the periventricular white matter. Periventricular lesions are nonspecific for MS, as opposed to callosal lesions (which are specific)—with the latter best seen on sagittal FLAIR images as in Figure 18.1 B (white arrow). These are typically oval-shaped with a flat inferior border, line the ependymal surface, and are oriented perpendicular to the lateral ventricle. Characteristic callosal lesions are sometimes overlooked on axial scans (black arrow, Fig. 18.1 A), with the clue being that they are medial to the lateral ventricles. Other typical areas for plaques include the centrum semiovale, the major and minor forceps, and the white matter surrounding the atrial trigones, temporal, and occipital horns of the lateral ventricles. In the posterior fossa, lesions of the colliculi, middle cerebellar peduncles, and pons are characteristic. Gray matter is less frequently involved. The contrast enhanced T1WI in Figure 18.1 C displays a non-enhancing plaque with low SI (black arrow). Such “black hole” lesions represent areas of axon loss and signify a poor prognosis. Since small vessel ischemic disease is not well-seen on T1WI, the presence of these lesions favors MS. With contrast administration, plaques may enhance in a uniform, punctuate, or ringed pattern (white arrow), often with progression to the latter over time. Enhancement signifies an active lesion and is thus not commonly seen (unless the patient is symptomatic), and persists for less than a month when present. Triple dose contrast improves detection of active lesions, while steroid therapy decreases enhancement. The concurrent presence of enhancing and non-enhancing lesions is fairly unique to MS and may help rule out

otherwise similar appearing conditions.

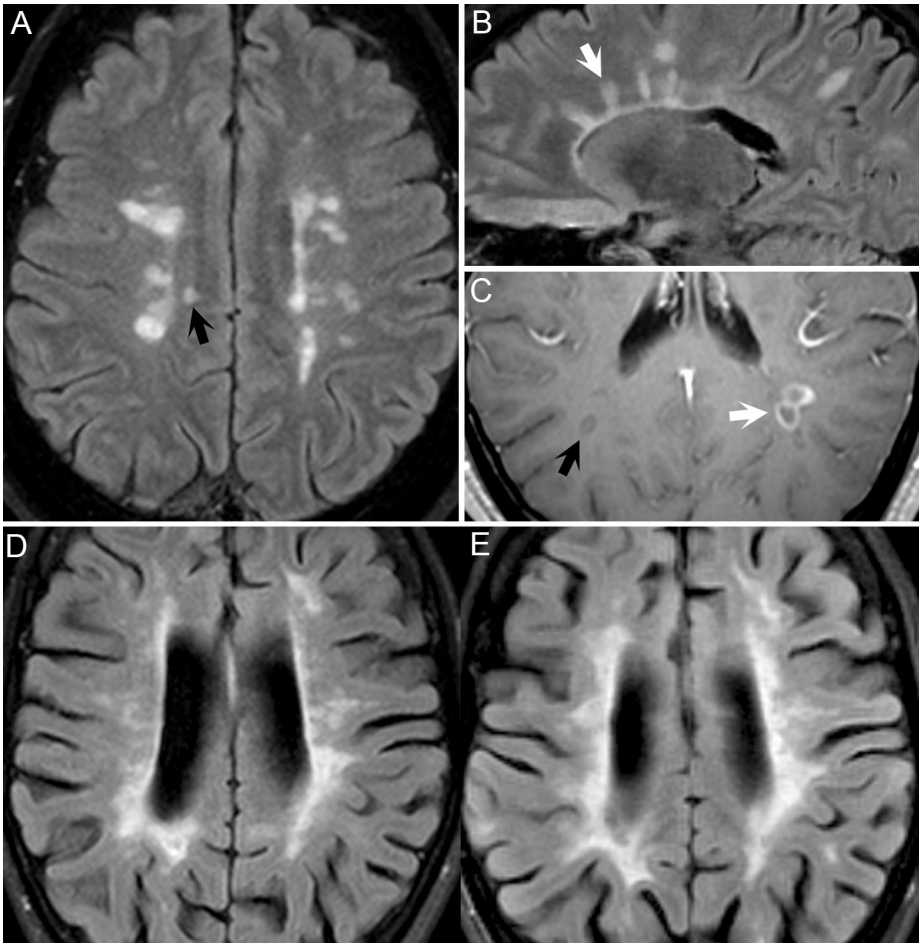

Fig. 18.1

The progression of MS over time is marked by increasingly confluent lesions with a resultant loss of their punctate appearance, rendering them even less distinct from the appearance of small vessel ischemia. The FLAIR images of Figure 18.1 D, E display the progression of high SI (correlating with gliosis in chronic MS) periventricular lesions in scans performed on the same patient but three years apart. Lesion confluence lends the periventricular lesions a “lumpy bumpy” outer margin, distinct from the often smooth outer margins of small vessel ischemia. Ventricular prominence, widening of the sulci (seen progressively from Fig. 18.1 D to E), and callosal thinning (from Wallerian degeneration) may also occur in chronic MS. Tumefactive MS is rare, but an important entity to know – in which a large intracranial lesion > 2 cm in diameter is present with mass effect, edema, and an open ring of enhancement. In most instances such a lesion is accompanied by smaller, more characteristic disseminated lesions. Unlike abscesses and ischemic lesions, ADC values in tumefactive MS are elevated, while ischemic disease may be further

differentiated by areas of low SI on GRE correlating with hemorrhage. Cord and optic nerve lesions comprise the remaining spectrum of MS. Optic neuritis is a common initial clinical manifestation with findings including abnormal high SI on T2WI within the optic nerve, best seen on images with fat suppression. Lesions of optic neuritis may also enhance. Acute disseminated encephalomyelitis (ADEM) is an inflammatory, demyelinating disorder of white matter that may mimic MS on MR. Unlike MS, it occurs predominantly in children (following viral infections and immunizations), and is typically associated with a monophasic course, with near-complete clinical recovery in 50% of patients. Multiple foci of demyelination (often larger than the typical lesions in MS) may be seen on MR, fewer in number than those of MS, with deep/juxtacortical white matter lesions more common than those periventricular in location. Asymmetric involvement of the hemispheres along with the brainstem and cerebellum are frequent. With clinical improvement, MR changes may completely resolve.