21. Meningitis

Clinically meningitis presents as a combination of headache, nuchal rigidity, fever, and altered mental status. Infections of the meninges are most commonly of hematogenous origin but may result from trauma, surgery, or the extension of local infections. Bacterial etiologies vary by age: neonates are affected most commonly by *E. coli* and Group B Streptococcus, older children by *N. meningitis, S. pneumonia*, and Type B *H. influenza*, and adults by *Streptococcus* and *N. meningitis*. MRI and CT may be performed prior to lumbar puncture to rule out masses and subarachnoid hemorrhage. Non-enhanced MRI is relatively insensitive to the detection of meningitis, particularly aseptic (usually viral) forms, but due to the necessity of early antibiotic treatment, when present, these features must be recognized. FLAIR is the most sensitive non-enhanced sequence, the higher protein content of the subarachnoid space manifesting as high SI compared the normally low SI CSF. Figure 21.1 A demonstrates posterior cortical sulci with normal low SI on FLAIR along with the high SI sulci (black arrow) that typify meningitis. Enhanced scans should be performed immediately after contrast administration, as delays result in the dilution of contrast agent within the CSF. The degree of enhancement is dose-dependent, and occurs in pachymeningeal—along the contours of the inner skull and dura—and leptomeningeal

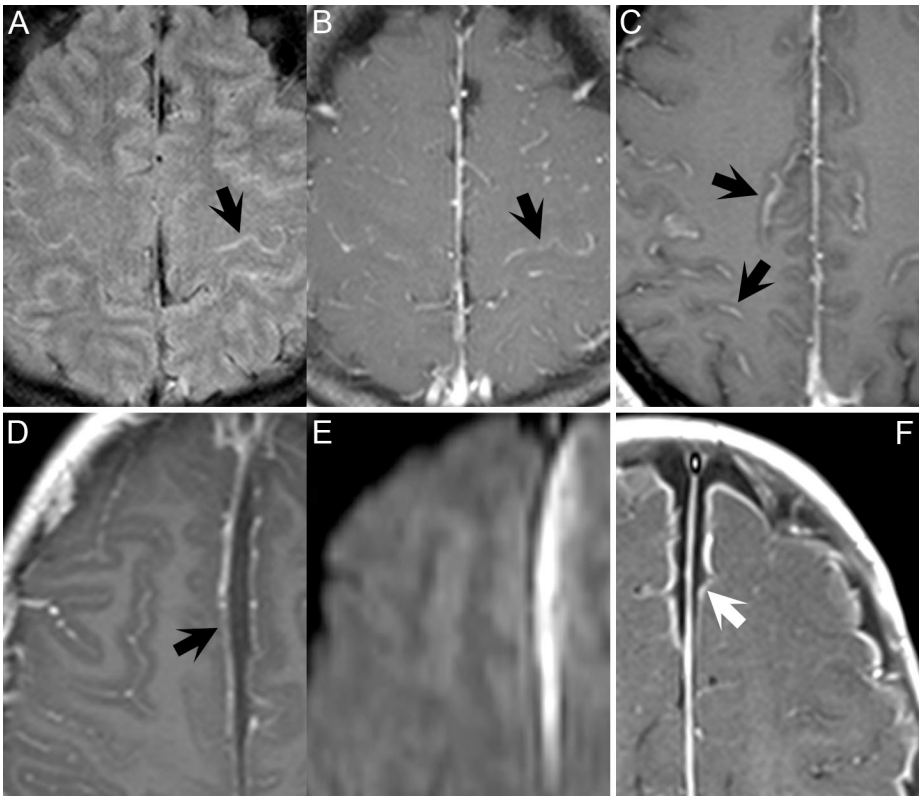

Fig. 21.1

patterns—extending to the sulci, fissures, and basilar cisterns. The degree of contrast enhancement does not correlate well with the extent of disease (in general underestimating the severity of involvement), as exemplified by Figure 21.1 B (black arrow) where mild leptomeningeal enhancement is seen in a patient who died of herniation secondary to meningitis the following day. Contrast enhanced MRI is similar in sensitivity to FLAIR but may be positive when former is negative and vice-versa, as in Figure 21.1 C which displays a case of meningitis with clear sulcal enhancement (black arrows) in which FLAIR images were normal. Findings of meningitis on FLAIR and contrast enhanced studies are nonspecific with differential considerations including subarachnoid hemorrhage, meningeal carcinomatosis, post-traumatic, and post-surgical changes. The blood due to acute subarachnoid hemorrhage is hyperdense on CT, and the enhancement of meningeal carcinomatosis on MRI tends to be either thin or nodular. Trauma or surgery may result in indefinitely persisting dural enhancement, whereas leptomeningeal enhancement is more suggestive of acute disease. Bacterial and viral causes of meningitis are most common, although fungal, tubercular, and chemical (post-surgical) etiologies are possible. In the appropriate clinical situation, neurosarcoidosis must be considered as a cause of non-infectious meningitis. This presents as a granulomatous leptomeningitis involving the skull base in a focal or diffuse pattern with possible extension along cranial nerves. Parenchymal involvement results from the spread of disease via the Virchow-Robin spaces, presenting clinically as a mass lesion and with a SI pattern on MRI that may be similar to MS. Complications of meningitis include empyema, hydrocephalus, and infarction. Figure 21.1 D demonstrates a low SI subdural fluid collection on post-contrast T1WI extending along the interhemispheric fissure that could represent either a subdural effusion or empyema. The leptomeningeal enhancement flanking the lesion (black arrow) and the marked hyperintensity (restricted diffusion) on DWI (Fig. 21.1 E) suggest the presence of meningitis complicated by a subdural empyema. Pediatric meningitides caused by *S. Pneumonia*, on the other hand, produce sterile fluid collections. Figure 21.1 F demonstrates such a subdural collection—extending laterally across cranial sutures of the attached dura but not medially across the midline—with prominent associated leptomeningeal (white arrow) but not dural enhancement. This lesion lacked any restriction in diffusion, consistent with a sterile subdural fluid collection associated with *S. Pneumonia* meningitis. Hydrocephalus may result from meningitis due to adhesions or loculations impairing CSF resorption at the arachnoid villi. MRI findings include dilated ventricles and transependymal resorption (also termed interstitial edema). The high SI of ependymitis on FLAIR and T2WI may mimic transependymal resorption, but the former usually enhances. The most common complication of adult meningitis is infarction, resulting from the

inflammatory involvement of the pial vasculature, leading to thrombosis and subsequent infarction. The temporal changes of MRI SI in infarction have been previously described in Chapters 13 & 14.