20. Sinus Disease

Incidental sinus disease is a common finding on MRI, with active sinusitis identified much less often. Most sinus infections are viral but may result in the obstruction of outflow tracts predisposing the sinus to bacterial infection, frequently by *S. pneumonia*, *H. influenza*, or *M. catarrhalis*. MRI findings of retention cysts (with a CSF-like SI) and mucosal thickening are often seen in asymptomatic patients and are not specific for active sinusitis. True active disease is typified by the presence of both mucosal thickening and an air-fluid level. Increased SI within the fluid on T2-weighted scans is usually seen (Fig. 20.1 A), which decreases with increasing protein content and inspissation. Although typically low SI on T1-weighted scans, fluid within the sinus can have high SI due to elevated protein content. Chronic desiccation of the sinus contents leads to low SI on T1 and T2WI that may be confused for the SI of a normal, patent sinus. In these cases, the characteristic peripheral enhancement of sinusitis serves as a clue to diagnosis, mandating closer image inspection. The major role of MRI in the evaluation of acute sinusitis is in delineating the spread of infection to surrounding structures. Such extension is demonstrated in Figure 20.1 A as an area of isointensity compressing the parenchyma adjacent to the frontal sinus on the right. This area is lined posteriorly by low SI dura and demonstrates restricted diffusion (Fig. 20.1 B) consistent with the diagnosis of an epidural abscess. Contrast administration confirms the confinement of this abscess to the epidural space (Fig. 20.1 C, white arrow) but also reveals more extensive enhancement of the dura, consistent with spreading meningitis (black arrow). Figure 20.1 C also demonstrates an infected left frontal sinus showing the pattern of peripheral enhancement typical of sinusitis. Infections may also breach the meninges to involve brain parenchyma. Figure 20.1 D demonstrates the high SI of acute sinusitis on a T2WI of the mastoid sinus (note however that this is characteristically slightly lower in SI than simple fluid), while the T2WI slightly superior to this plane reveals abnormal high SI in the posterior temporal lobe consistent with cerebritis (Fig. 20.1 E, white arrow). As in Figure 20.1 F, cerebritis often enhances (black arrow) and is accompanied by peripheral edema, the latter appearing as low peripheral SI on this T1WI. Cerebritis may progress to an intracranial abscess, the classic appearance of which—a ring-enhancing lesion (asterisk) with surrounding prominent edema—is shown on the contrast-enhanced T1WI in Figure 20.1 G.

The anatomic proximity of the mastoid sinus to the transverse venous sinus is evident in Figure 20.1 D, E. Spread of infection to the venous sinuses may result in their thrombosis. Prior to the antibiotic era, this mechanism was the most frequent etiology of venous thrombosis, but today noninflammatory causes—pregnancy, oral contraceptives, trauma,

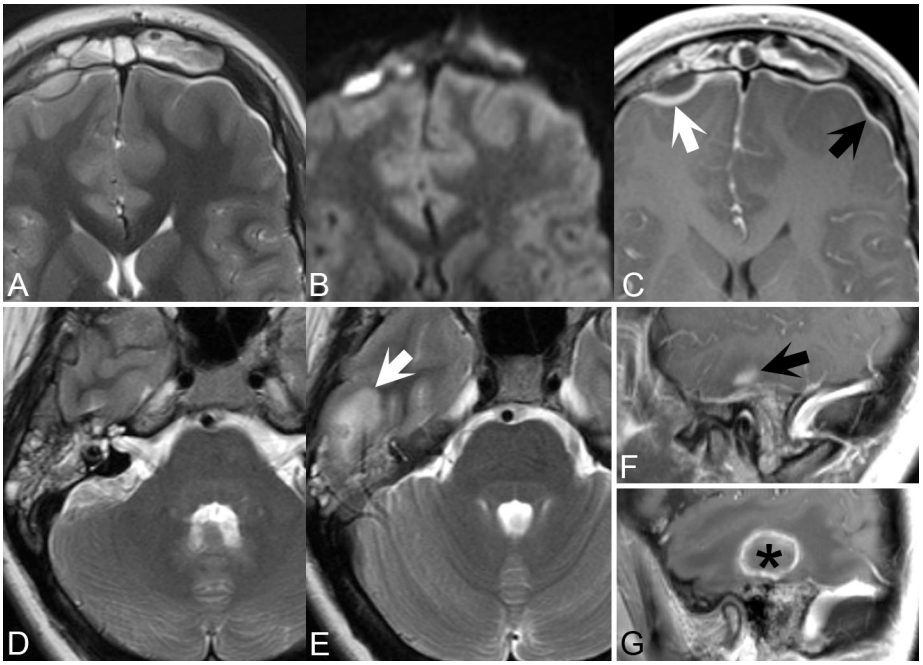

Fig. 20.1

dehydration, neoplasm, and L-asparaginase therapy (for treatment of acute lymphocytic leukemia)—predominate. MRI findings of venous thrombosis progress in an orderly manner, beginning with the initial absence of a normal flow void seen on T1WI. At this early stage, the deoxyhemoglobin content of the thrombus results in a moderate SI on T1WI and low SI on T2WI. Venous collaterals may be seen bypassing the obstructed area. With reduction to methemoglobin, the clot appears as high SI on T1 and eventually T2WI. Upon vessel recanalization, flow voids are again visualized. Diagnostic pitfalls of venous thrombosis include slow flow in a patent vein masquerading as a high SI thrombus. Thrombus does not enhance and maintains a consistent SI in every plane (when imaged with the same scan technique). MR venography is not strictly necessary for the diagnosis and assessment of venous thrombosis, although commonly employed and often making diagnosis simpler. Ancillary findings include suggestions within the venous system of increased intracranial pressure, including visible emissary veins, prominent flow in the deep medullary veins, and hemorrhage. A thrombosed sinus generally lacks the hyperintensity seen on T2WI with normal venous flow and may demonstrate a frayed appearance if recanalized. Congenitally hypoplastic or asymmetric sinuses may also be confused with thrombosis, as may the signal voids of in-plane venous flow.

Venous thrombosis may result in parenchymal infarction. Venous infarctions tend to affect a younger age group than their arterial counterparts and do not follow the patterns of arterial distributions described in Chapter 13 and 14. Instead they classically consist of parasagittal, high-convexity lesions, unilateral temporal lobe infarctions, or lesions of the deep gray

nuclei. Venous infarctions are also more frequently hemorrhagic than their arterial counterparts. Acute hyperintensity on DWI may be more heterogenous in venous, as compared to arterial, infarcts. Venous thrombosis is often readily identified accompanying a venous infarct.