ELSEVIER

Seminars in
ROENTGENOLOGY

Infections of the Brain and Meninges

Behroze Vachha, MD, PhD,[*,†] Gul Moonis, MD,[‡] and Andrei Holodny, MD[*]

Bacterial Infections

Imaging findings for the various typical bacteria infecting the central nervous system (CNS) are nonspecific and require correlation with laboratory tests to establish a definitive diagnosis. Mycobacterial and spirochetal infections have a clinical presentation and imaging appearance different from those of typical bacteria. Therefore, the following sections first discuss the various stages of pyogenic infections that occur with most bacteria, followed by a description of mycobacterial (tuberculosis) and spirochetal (Lyme disease) infections.

Pyogenic Infections of the Brain

Bacterial infections of the brain parenchyma occur most frequently through hematogenous seeding, although there may be spread from direct extension following trauma, surgery, sinusitis, otomastoiditis, or dental infections.[1-3] Anaerobic bacteria are the most common organisms overall, whereas *Staphylococcus aureus* frequently is the causative agent following surgery or trauma.[3,4] Hematogenous seeding commonly involves the frontal and parietal lobes (middle cerebral artery territory), sinus infections typically spread to the frontal lobes, and otomastoiditis most commonly involves the temporal lobe.[2]

Pyogenic infection evolves predictably from early cerebritis to frank abscess. Early cerebritis is seen in the first few days of infection and is characterized by an edematous brain.[1,2] On computed tomography (CT), the brain appears normal or may show low attenuation corresponding to vasogenic edema that is associated with mild mass effect and patchy ill-defined enhancement. On magnetic resonance imaging (MRI), the lesion shows hyperintense signal on T2-weighted (T2W) and fluid-attenuated inversion recovery (FLAIR) images and hypointense signal on T1-weighted (T1W) imaging, with variable enhancement. If the diagnosis is made at this stage, the patient can be treated effectively with antibiotics.

Late cerebritis develops within 1-2 weeks of infection. On CT, there is now thick, irregular contrast enhancement at the edges of the lesion. On MRI, FLAIR and T2W images demonstrate increased signal centrally within the lesion, which is surrounded by a thick, irregular enhancing rim and vasogenic edema. Diffusion-weighted imaging (DWI) may show increased signal within the center of the lesion. There is no discrete low-signal capsule on T2W images at this stage, unlike the appearance in mature abscesses. Although an infection at this stage also can be treated with antibiotics, surgery is often performed because the imaging appearance of late cerebritis frequently is difficult to distinguish from an abscess.

Within 2 weeks of infection, the lesion becomes an abscess, which is walled off by a capsule that characteristically appears on CT and MRI as a well-defined rim of enhancement that has low signal on T2W images. The lesion contains central necrosis, which has low attenuation on CT, is hypointense on T1W images, is hyperintense on T2W and FLAIR images, and has increased central signal on DWI. At a later stage, the lesion shows markedly increased signal on DWI and rim enhancement that is often thinner along the ventricular side (Fig. 1).[3,5,6] A solitary abscess is often treated with stereotactic needle aspiration followed by antibiotic therapy, whereas multiple abscesses are managed with antibiotics alone.[2]

The thin capsular rim along the ventricular side of an abscess predisposes it to rupture into the ventricle. This results in ependymitis and ventriculitis, which are characterized on CT and MRI as enhancement of the ependymal lining of the ventricles and abnormal density or signal intensity within the ventricle (Fig. 2).[7]

The differential diagnosis of pyogenic abscess includes such rim-enhancing lesions as primary neoplasms (eg, glioblastoma multiforme), solitary metastasis, toxoplasmosis, demyelinating lesions, and resolving hematoma. Clinical features, homogeneous restricted diffusion centrally within the lesion (compared to more peripheral restricted diffusion in tumors and demyelinating disease), and the characteristic appearance of a smooth enhancing rim (compared to more nodular and irregular rim enhancement in tumors and asymmetric comma-shaped peripheral enhancement in demyelinating lesions) usually help distinguish an abscess from other ring-enhancing lesions.[8] MR

*Neuroradiology, Memorial Sloan Kettering Cancer Center, New York, NY.
†Neuroradiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA.
‡Neuroradiology, Columbia University Medical Center, New York, NY.
Address reprint requests to Behroze Vachha, MD, PhD, Neuroradiology, Memorial Sloan Kettering Cancer Center, 1275 York Ave, New York, NY 10065. E-mail: vachhab@mskcc.org

http://dx.doi.org/10.1053/j.ro.2016.07.003

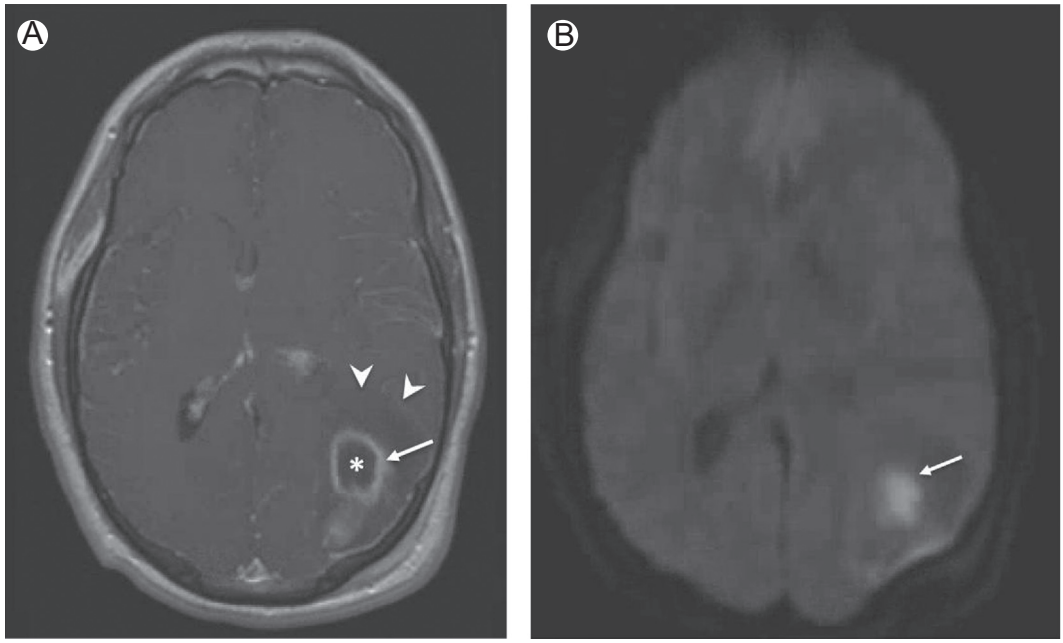

Figure 1 Cerebral abscess. (A) Axial contrast-enhanced image demonstrates a ring-enhancing abscess (arrow) with a central necrotic core (asterisk) and surrounding edema (arrowhead) in the left parietal lobe. (B) Axial DWI demonstrates restricted diffusion within the necrotic core of the abscess (arrow).

spectroscopy, although not performed routinely, can demonstrate decreased n-acetyl aspartate (NAA), presence of amino acids (0.9 ppm), lactate (1.3 ppm), acetate (1.9 ppm), and succinate (2.4 ppm) in the necrotic center.[9]

Bacterial Meningitis

Streptococcus pneumoniae (in older adults) and *Neisseria meningitidis* account for most bacterial meningitis in the United States.[10] The diagnosis is usually made clinically and by lumbar puncture. Noncontrast head CT often has a normal appearance and only occasionally demonstrates hyperdensity in peripheral sulci. FLAIR imaging may show prominence of subarachnoid spaces and hyperintense signal within sulci (Fig. 3).[11] Postcontrast T1W imaging may demonstrate leptomeningeal enhancement.[11] CT and MRI play a role in evaluating complications of bacterial meningitis, which include

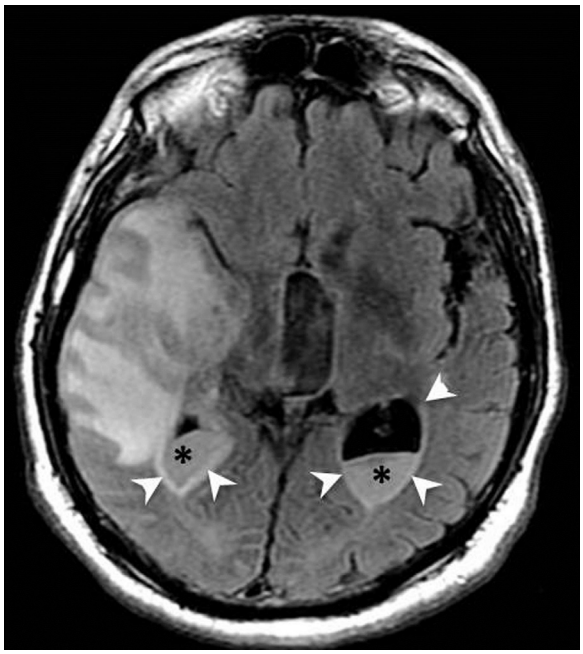

Figure 2 Ventriculitis. Axial FLAIR image shows hyperintensity along the ventricular ependyma (arrowhead) with hyperintense debris filling both lateral ventricles (asterisk) in a patient with temporal abscess (not shown) that ruptured into the ventricular system.

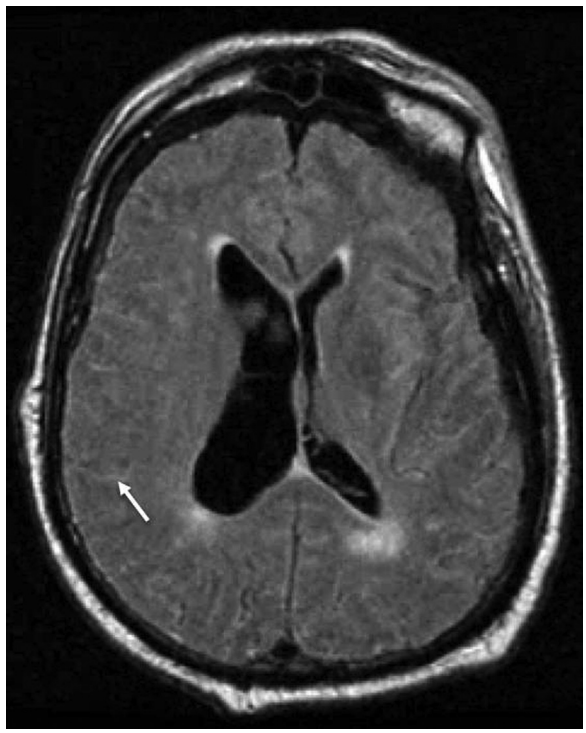

Figure 3 Bacterial meningitis. Axial FLAIR image shows diffuse abnormal hyperintensity within sulci (arrow).