
Imaging of intracranial infectious diseases in adults

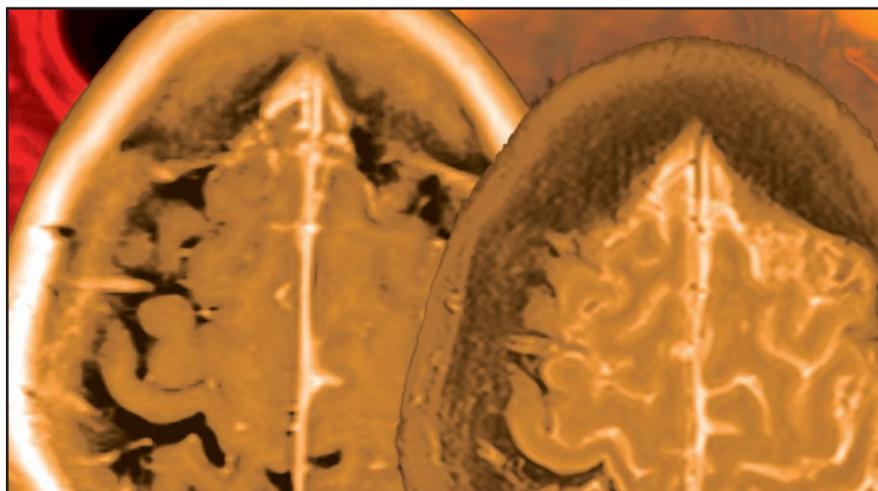
Bradford C. Mitchell, MD, and Seena Dehkharghani, MD

Infectious diseases affecting the central nervous system (CNS) range from acute to chronic, from indolent to life-threatening, and from self-limited to relentlessly progressive. Prompt detection of intracranial infections can have a significant effect on patient outcomes with regard to morbidity and mortality. Accurate characterization with medical imaging can allow appropriate empiric therapy to be initiated, complications to be managed appropriately and prognosis to be offered. In some cases, a specific infectious pathogen can be suggested based on typical imaging findings and clinical scenarios; other times, the broad category of infection can only be offered among a range of possible etiologies. In this article, the authors present an overview of intracranial infections, organized by category of pathogen, with an emphasis on the degree of specificity afforded by various imaging findings.¹

Bacterial infections

The various typical bacteria infecting the CNS usually cannot be distinguished by imaging; rather they are grouped according to site of infection, which directs

Dr. Mitchell and Dr. Dehkharghani are at the Department of Radiology and Imaging Sciences, Division of Neuroradiology, at Emory University School of Medicine, Atlanta, GA.



therapy. Exceptions include mycobacterial and spirochetal pathogens, which are discussed separately.

Epidural infection

Epidural infections typically result from direct extension of an adjacent infection (commonly mastoiditis or sinusitis) or from direct seeding following neurosurgical intervention; hematogenous seeding is possible, but less common. Epidural phlegmon appears as an ill-defined, typically enhancing collection with associated dural thickening within the epidural space on postcontrast computed tomography (CT) or magnetic resonance (MR) images (Figure 1). If adjacent to a site of infection, these findings indicate extension into the epidural space.

If adjacent to a site of surgery, the findings may not be reliably differentiated from sterile reactive and postsurgical changes. Progression to epidural abscess is evidenced by a well-defined lentiform fluid collection with an enhancing rim. The internal contents often demonstrate restricted diffusion; however, this can be variable given the often indolent development and commonly subacute evolution of the suppurative contents.^{2,3} Again, in the postoperative period, an evolving hematoma, complicated seroma, or pseudomeningocele could have a similar appearance; the presence of restricted diffusion suggests infection, but even this finding is less sensitive and specific in the postoperative state largely due to intermixed blood products.

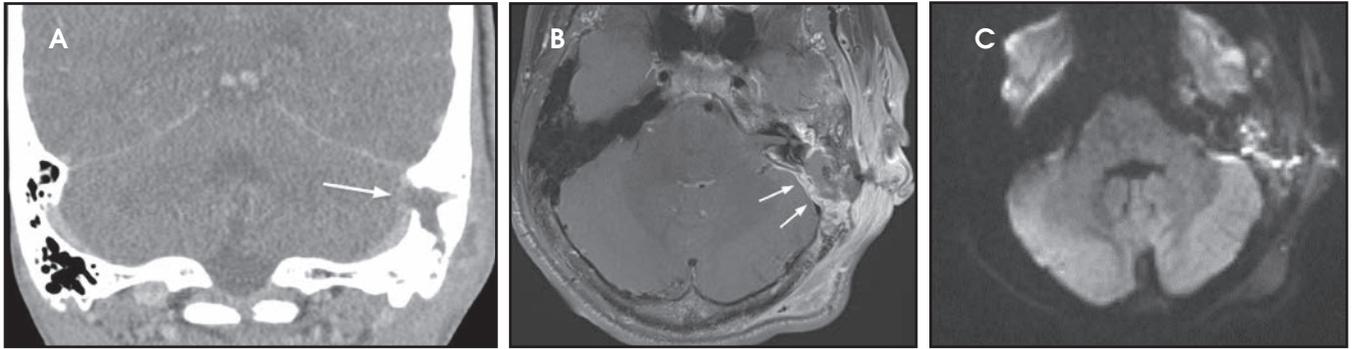


FIGURE 1. Epidural phlegmon. Coronal CT with contrast (A) demonstrates coalescence and opacification of left mastoid air cells with dehiscence of the sigmoid plate and extension of infection intracranially into the epidural space (arrow), compressing the sigmoid sinus. A lateral subperiosteal abscess is also noted. Axial post-contrast T1-weighted MR image (B) better demonstrates the extent of epidural enhancing phlegmon (arrows). Diffusion-weighted image (C) shows some signal change within the mastoid bone but no defined epidural abscess.

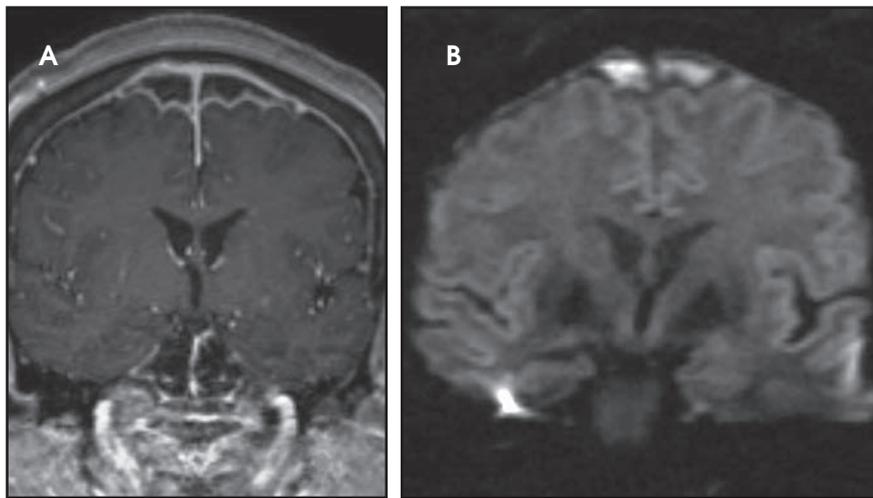


FIGURE 2. Subdural empyema. Coronal post-contrast T1-weighted MR image (A) demonstrates bilateral parafalcine subdural fluid collections with thick dural and arachnoid enhancement. The fluid contents demonstrate diffusion restriction (B, diffusion-weighted image), confirming empyema in this patient with bacterial meningitis.

Subdural infection

Subdural empyema refers to an infected fluid collection in the subdural space. Etiology may be related to an extradural infection (again including mastoiditis and sinusitis) that has breached the dura or may be secondary to leptomeningitis with spread to the subdural space through arachnoid villi or bridging veins; primary hematogenous seeding is also possible. The appearance on imaging is of a crescent-shaped collection with rim enhancement and internal restricted diffusion (Figure 2). In some cases, evolving subdural hematoma can have a similar appearance, though again the combination of restricted diffusion and thick rim enhancement strongly suggests infection in the appropriate setting.²⁻⁴ As with epidural abscess, diffusion restriction is less sensitive and specific in the postoperative period.

Thrombophlebitis

As a complication of either epidural or subdural infection, the dural venous sinuses may become involved, resulting in thrombosis with the possibility of resultant venous hypertensive edema or hemorrhage. The cavernous sinus may similarly be involved, and the cavernous segment of the internal carotid artery (ICA) may be affected with resultant vasospasm, occlusion, and/or mycotic pseudoaneurysm. Thrombophlebitis appears as a change in signal and loss of flow-void of the venous structure on spin echo MRI, often with expansion (Figure 3).⁵ Contrast-enhanced MR

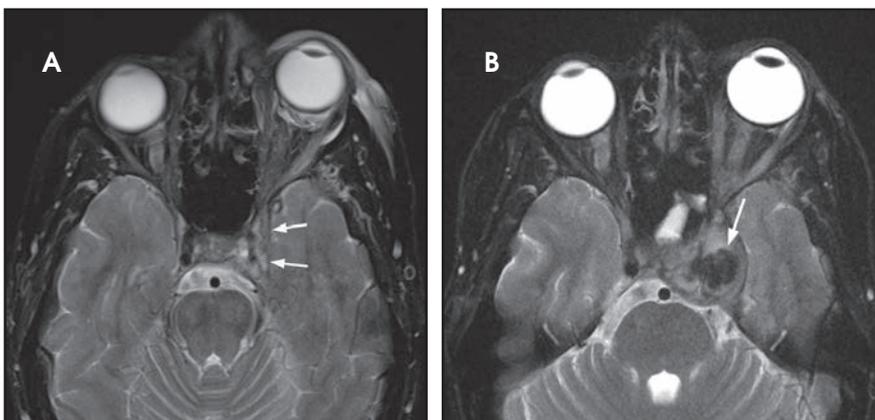


FIGURE 3. Cavernous sinus thrombophlebitis and carotid pseudoaneurysm. Axial fat-saturated T2-weighted image (A) demonstrates left ocular proptosis and orbital fat infiltration. The left cavernous sinus is mildly expanded with heterogeneous signal (arrows), suggesting thrombosis, and the ICA flow-void is narrowed. Repeat MRI 10 days later (B) reveals further expansion of the cavernous sinus and development of a lobular heterogeneous and somewhat lamellated mass of low signal (arrow) contiguous with the ICA flow-void, consistent with a mycotic pseudoaneurysm.

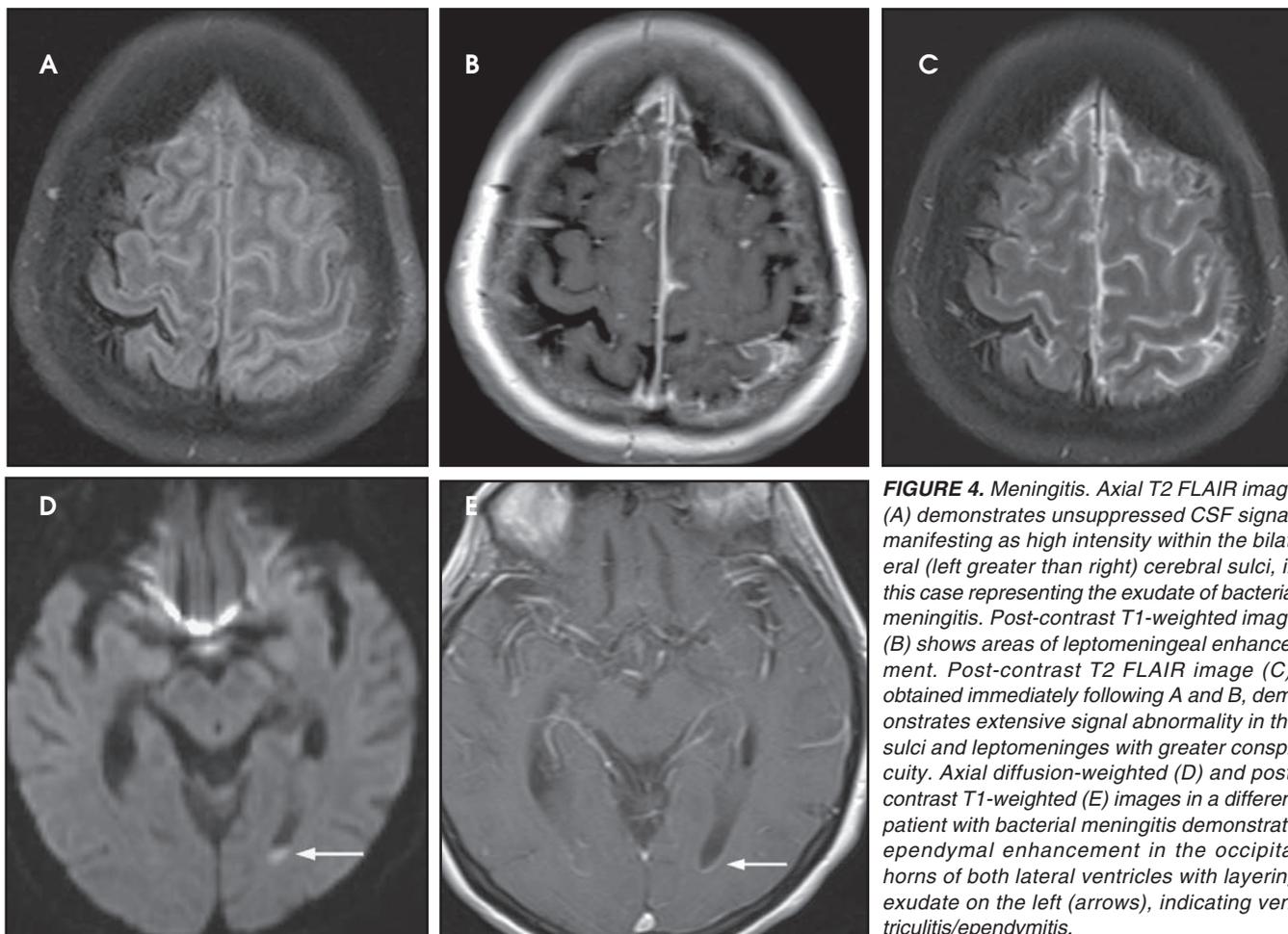


FIGURE 4. Meningitis. Axial T2 FLAIR image (A) demonstrates unsuppressed CSF signal, manifesting as high intensity within the bilateral (left greater than right) cerebral sulci, in this case representing the exudate of bacterial meningitis. Post-contrast T1-weighted image (B) shows areas of leptomeningeal enhancement. Post-contrast T2 FLAIR image (C), obtained immediately following A and B, demonstrates extensive signal abnormality in the sulci and leptomeninges with greater conspicuity. Axial diffusion-weighted (D) and post-contrast T1-weighted (E) images in a different patient with bacterial meningitis demonstrate ependymal enhancement in the occipital horns of both lateral ventricles with layering exudate on the left (arrows), indicating ventriculitis/ependymitis.

venography is highly sensitive and specific for thrombosis.⁶ Vascular imaging (eg, MR angiography) should be considered in cases of cavernous sinus involvement and may show narrowing or occlusion of the artery or a saccular pseudoaneurysm.⁷

Meningitis

Bacterial meningitis is primarily a clinical and laboratory diagnosis. Imaging may be employed to exclude other diseases when the diagnosis is unclear or to assess for complications when the patient fails to respond to empiric therapy. CT has low sensitivity for uncomplicated meningitis; increased density within the subarachnoid spaces or leptomeningeal enhancement may be seen in some cases. MRI is more sensitive than CT.⁸ T2-weighted fluid-attenuated inversion-recovery (FLAIR) images may demonstrate hyperintense signal

within the cerebral sulci as inflammatory exudates change the inversion time of cerebrospinal fluid (CSF). Post-contrast T1-weighted imaging may be more sensitive, demonstrating leptomeningeal enhancement.⁹ Recently, post-contrast T2 FLAIR images have been shown to have even higher sensitivity, demonstrating both leptomeningeal enhancement and seepage of gadolinium contrast material into the CSF (Figure 4).¹⁰ Pre- and postcontrast magnetization transfer imaging has been shown in limited investigations to provide up to 100% sensitivity for bacterial meningitis, though it remains of uncertain utility and thus not in widespread use.¹¹

Complicated meningitis

Complications of bacterial meningitis are readily detected by CT and MR, the latter being more sensitive for certain complications. Hydrocephalus is

the most common complication, caused by inflammation of arachnoid villi and decreased CSF resorption; typically the entire ventricular system is enlarged (“nonobstructive” or “communicating” hydrocephalus). Infectious processes can extend into the ventricles (ventriculitis/ependymitis), evidenced by enhancement of the ependymal lining with occasional layering purulent debris (Figure 4), and can cause adhesions and loculations, resulting in focal entrapment or obstructive hydrocephalus. Chronic hydrocephalus may result from either case, requiring long-term CSF shunting.⁴ Inflammation of the membranous labyrinth may occur through the cochlear aqueduct or internal auditory canal and can cause hearing loss and/or vestibular dysfunction. In the acute phase, MRI may demonstrate increased signal of labyrinthine fluid on FLAIR or enhancement of the membranous labyrinth fol-

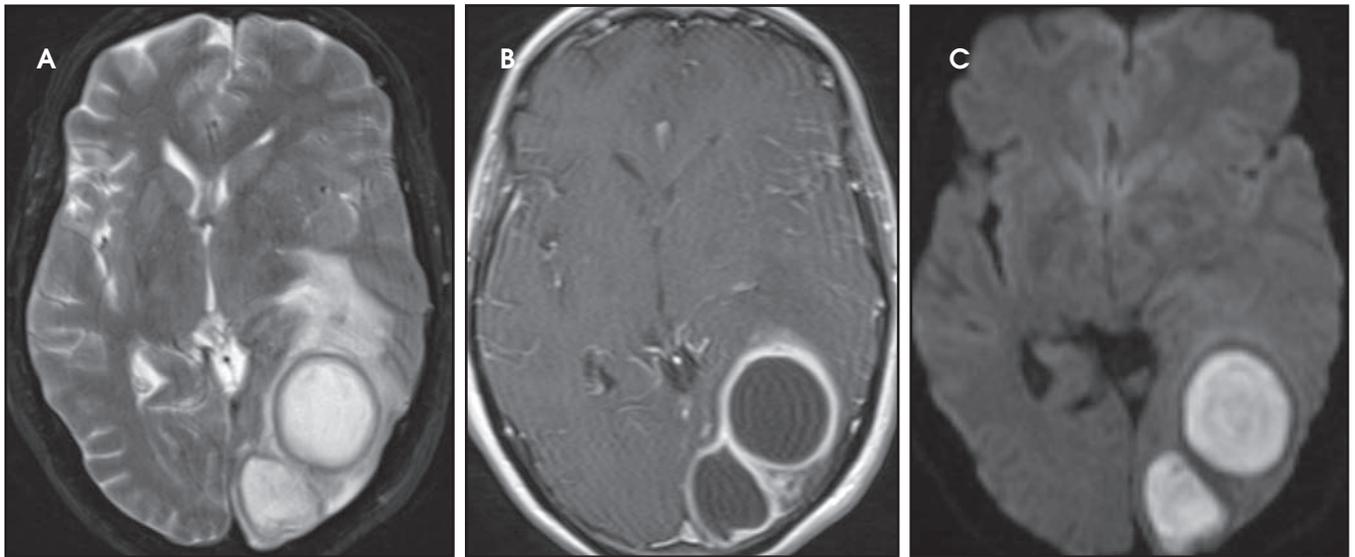


FIGURE 5. Pyogenic abscess. Axial T2-weighted image (A) demonstrates two adjacent fluid collections in the left occipital lobe with hypointense rims and vasogenic edema. There is smooth rim enhancement on the post-contrast T1-weighted image (B). The fluid contents exhibit restricted diffusion (C), confirmed on ADC map (not shown), highly suggestive of pyogenic abscess (confirmed at surgery).

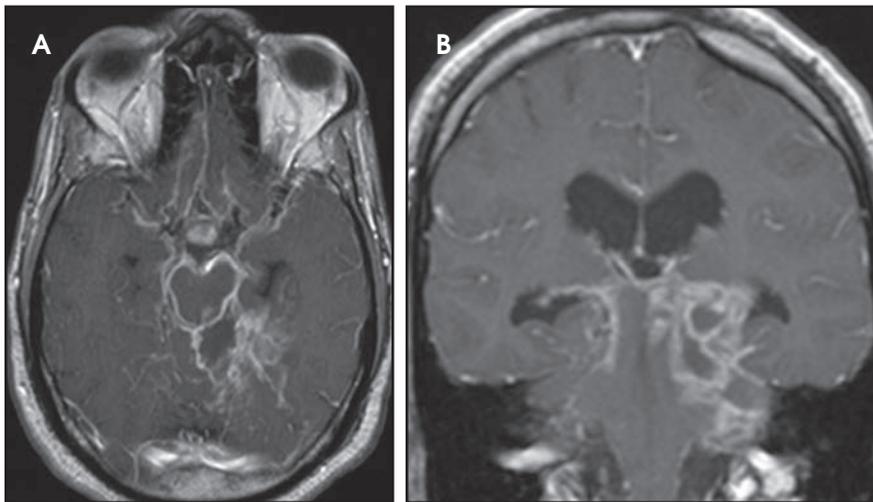


FIGURE 6. Basal meningitis. Axial (A) and coronal (B) post-contrast T1-weighted images demonstrate thick leptomeningeal enhancement primarily involving the basal meninges and cerebellar fissures. Areas of hypointense signal in the left superior cerebellum and medial temporal lobe suggest parenchymal involvement (confirmed on other sequences). The appearance is nonspecific but suggests an atypical infection (mycobacterial or fungal), other granulomatous process, or neoplasm. In this patient with AIDS and lymphocytic meningitis and insidious clinical course, tuberculous meningitis was the presumptive diagnosis, though the patient expired without laboratory confirmation.

lowing contrast administration. In some cases, the labyrinth may become ossified (labyrinthitis ossificans). This will be apparent on CT as increased attenuation within the cochlea, vestibule, and/or semicircular canals. MR findings include decreased signal or obliteration of fluid spaces on T2-weighted images.¹² The major arteries and branches supplying the brain course in the subarachnoid spaces, and meningitis can lead to sec-

ondary arteritis. Vasospasm may be seen on angiographic studies (catheter, CT, or MR angiography), and parenchymal infarctions or septic emboli may occur. Finally, infection may extend into the subdural or epidural spaces or into the brain parenchyma.⁴

Parenchymal infections

Infections of the brain parenchyma most commonly occur through hema-

togenous seeding, though spread from adjacent sources or seeding from trauma or surgery is also possible. Infection evolves predictably from cerebritis to abscess. Early cerebritis is characterized by vasogenic edema (low attenuation on CT; hyperintense signal on T2-weighted and FLAIR MR images) and mass effect with patchy, ill-defined enhancement. In late cerebritis, irregular rim enhancement develops. Cerebral abscess (early capsule stage) appears as a fluid-filled cavity with well-defined rim enhancement (Figure 5). The abscess capsule is typically seen on CT as higher in attenuation than the abscess core and surrounding parenchymal edema. On MRI the capsule is hypointense on T2-weighted and precontrast T1-weighted images. Avid rim enhancement following contrast administration is expected on CT or MRI. The abscess cavity contains purulent material of low attenuation on CT, hypointense signal on precontrast T1-weighted images, and hyperintense signal on T2-weighted and FLAIR images. Characteristic diffusion restriction within the cavity reliably distinguishes pyogenic abscess from toxoplasmosis, most neoplastic conditions, and other rim-enhancing lesions.¹³⁻¹⁶ MR spectroscopy will demonstrate a prominent lactate peak due to anaerobic metabolism and decreased NAA due to neuronal loss.¹⁴

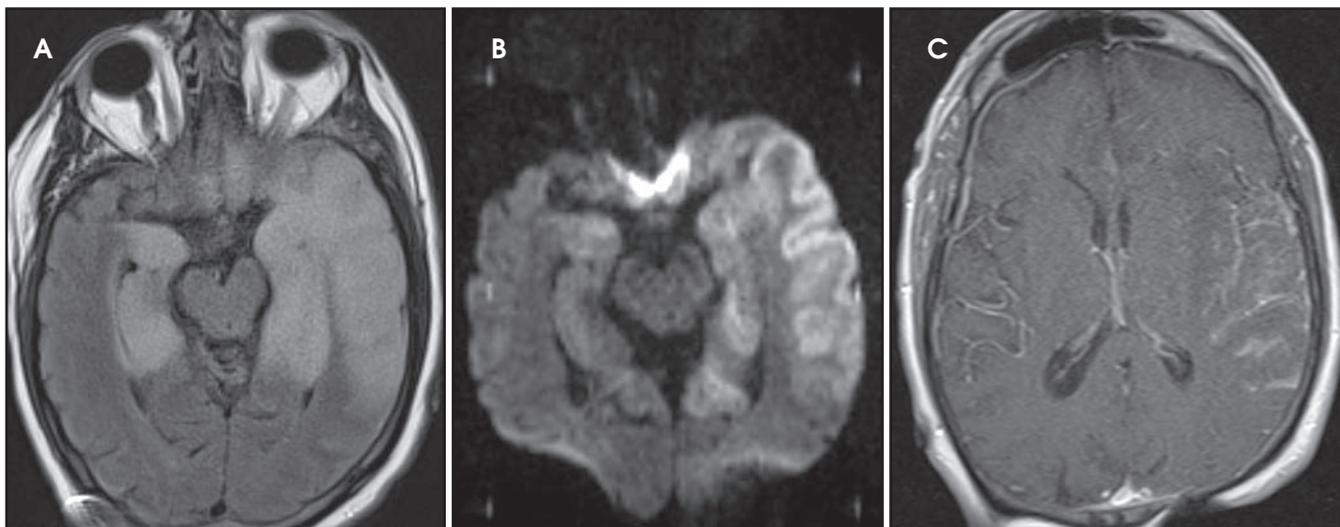


FIGURE 7. Herpes simplex virus (HSV) encephalitis. Axial FLAIR image (A) demonstrates gyral swelling and signal abnormality involving both grey and white matter of bilateral mesial temporal lobes and frontal straight gyri as well as the lateral left temporal lobe. Diffusion-weighted image (B) shows cortical restricted diffusion in the same areas. Post-contrast T1-weighted image (C) shows leptomeningeal and parenchymal enhancement in the left perisylvian region suggesting meningoencephalitis. The distribution is typical of HSV encephalitis, which was confirmed by CSF analysis.

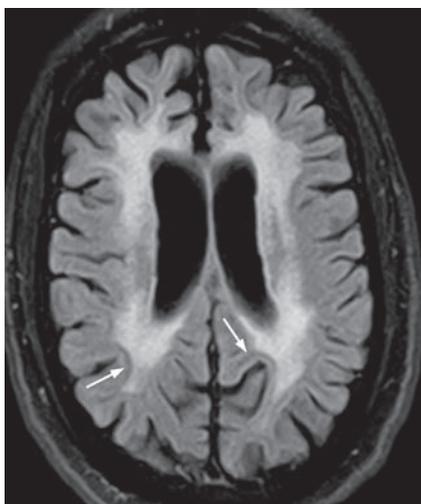


FIGURE 8. Human immunodeficiency virus (HIV) encephalopathy. Axial FLAIR image demonstrates diffuse cerebral volume loss and confluent, symmetric periventricular and subcortical white matter signal abnormality sparing the subcortical U-fibers (arrows) and without associated mass effect, diffusion restriction (not shown), or postcontrast enhancement (not shown). The appearance is nonspecific but, in a patient with AIDS and cognitive decline, supportive of HIV encephalopathy.

Perfusion imaging will demonstrate normal to mildly decreased relative cerebral blood volume (rCBV) in the periphery and absent perfusion centrally.¹⁷ However, MR spectroscopy and perfusion are less specific than diffusion-weighted

imaging and typically add little information. The final stage, late capsule, is defined by cavity collapse and decreasing parenchymal edema. Septic embolization is a special case of hematogenous dissemination occurring typically in the setting of valvular vegetation in bacterial endocarditis. Infected small particles embolize to small intracranial arteries and produce a combination of infarction and parenchymal infection. The finding of enhancement in what appears otherwise as an acute infarct should suggest the possibility of infection. Individual foci may progress to frank abscess if untreated.

CNS tuberculosis

CNS infection caused by *Mycobacterium tuberculosis* (TB) generally has a clinical presentation and imaging appearance different from that caused by typical bacteria. The diagnosis is made by CSF sampling, but imaging findings may be suggestive. The most common presentation is basal meningitis with leptomeningeal enhancement and signal hyperintensity on T2 FLAIR images within and along the basal cisterns (Figure 6). The appearance is not specific for TB, as other infectious processes (eg, from fungi and other bacteria) as well as noninfectious inflammatory (eg, sarcoid-

osis) and neoplastic (eg, metastases) processes can have similar features; findings of tuberculosis in other organs (most commonly the lungs) increase specificity. Complications of TB meningitis are similar to those of bacterial meningitis, most commonly including hydrocephalus and vasculitis with infarctions. Parenchymal involvement is seen in a minority of cases, but is more common in patients infected with the human immunodeficiency virus (HIV). Many cases co-exist with basal meningitis, with the combination of findings more specific for TB than either alone. The most common parenchymal finding is a tuberculoma (tuberculous granuloma). Tuberculoma can be T2-hyperintense with solid enhancement (non-caseating granuloma), T2-hypointense/isointense with rim enhancement (solid caseating granuloma), or T2-hyperintense with rim enhancement (liquid caseating granuloma). The latter appearance may also represent a true tuberculous abscess, the two being indistinguishable by imaging. Restricted diffusion is not typically seen, differentiating TB from pyogenic abscess. Spectroscopy may demonstrate an elevated lactate peak and decreased NAA. Other less common findings in CNS TB are focal cerebritis (appearing similar to bacterial cerebritis) and leuko-

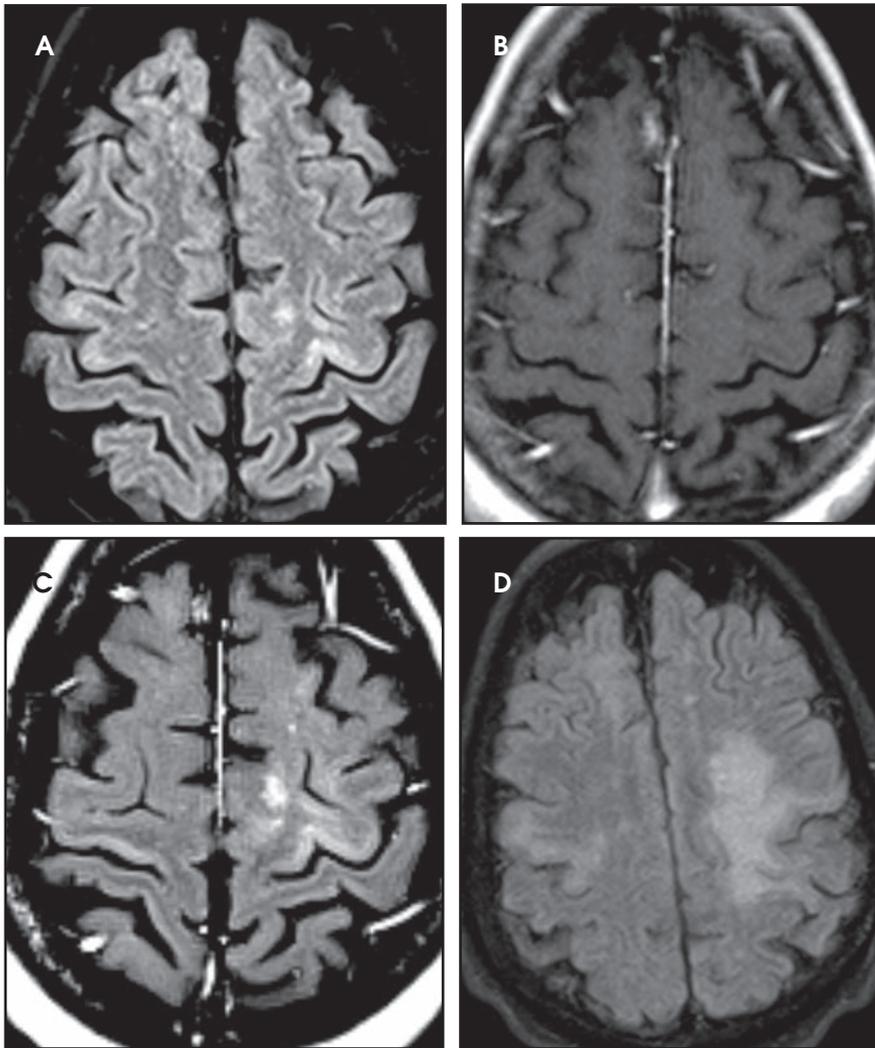


FIGURE 9. Progressive multifocal leukoencephalopathy (PML). Axial fat-suppressed T2 FLAIR image (A) demonstrates patchy areas of signal abnormal in the subcortical white matter with involvement of subcortical U-fibers (in contrast to HIV encephalopathy; see figure 8). No enhancement is seen on this initial study (B). PML was confirmed by isolation of JC virus from the CSF. Repeat imaging 2 weeks after initiation of antiretroviral therapy (C, postcontrast T1) shows new enhancement in the affected areas. In the setting of worsening clinical status, this suggests immune reconstitution inflammatory syndrome (IRIS). Five months later, axial FLAIR image (D) shows progression of white matter disease.

encephalopathy (appearing as a diffuse demyelinating process). Treated lesions often become calcified.^{18,19}

Neurosyphilis

Like tuberculosis, neurosyphilis tends to involve the basal meninges. Vasculitis and infarctions are commonly seen. Parenchymal involvement may also be seen and manifest as nonenhancing white matter lesions or, rarely, enhancing cerebral gummas. Cerebral atrophy may be seen in the chronic phase in patients with dementia. In the current

age, the majority of patients with neurosyphilis are also HIV-positive (largely due to shared risk factors), therefore, intracranial manifestations of HIV/AIDS and opportunistic infections may coexist with neurosyphilis, potentially confounding the imaging findings.^{20,21}

Lyme disease

Intracranial manifestations of Lyme disease (*Lyme neuroborreliosis*) assume two primary forms which, when seen together, allow this disease to be suggested with relative specificity. The first

includes parenchymal lesions (low attenuation on CT and hyperintense on FLAIR with variable enhancement) affecting the subcortical (primarily frontal) and periventricular white matter in a pattern that mimics multiple sclerosis or other demyelinating diseases. The second pattern is a cranial neuritis—enlargement and enhancement of cranial nerves; the facial nerves are most commonly affected (rendering Lyme disease a differential consideration in cases of Bell's palsy), with oculomotor and trigeminal nerve involvement also reported.^{21,22}

Viral infections

Viral infections of the CNS typically manifest as meningitis or encephalitis (or combined meningoencephalitis). A few pathogens with characteristic imaging features are highlighted here.

Herpes simplex virus (HSV)

Herpes simplex virus (HSV) is notable for its typical imaging appearance and because it is readily treatable when diagnosed early, but potentially devastating in cases of delayed diagnosis. The typical appearance of HSV encephalitis includes parenchymal edema (hypoattenuating on CT; hyperintense on FLAIR) with variable patchy or gyriform enhancement and variable degrees of restricted diffusion (Figure 7); parenchymal hemorrhage and necrosis may occur in severe or untreated cases. MRI findings are seen earlier in the course of infection than CT findings, with DWI changes most sensitive among routine sequences. The classic distribution includes bilateral (though asymmetric) involvement of the limbic system (most notably the mesial temporal lobes). Other cortical regions may also be involved, while the basal ganglia are typically spared.^{23,24}

Cytomegalovirus (CMV)

Cytomegalovirus (CMV) meningoencephalitis is typically seen in immunocompromised patients. Ventriculitis is a classic feature, seen as ependymal enhancement, ventriculomegaly, and occasionally layering debris. CMV encephalitis has a predilection for the

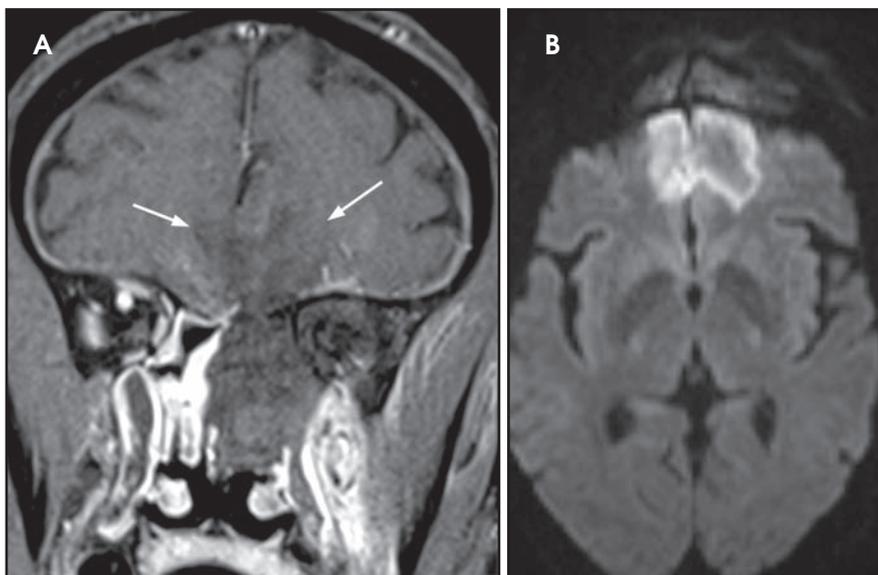


FIGURE 10. Invasive fungal sinusitis. A coronal postcontrast T1-weighted image (A) demonstrates nonenhancing tissue filling the left nasal cavity, ethmoid sinus, and orbit. There is extension through the ethmoid roof to involve both frontal lobes (arrows) with some faint peripheral enhancement noted. Mucormycosis was confirmed at surgery in this diabetic patient. Diffusion-weighted image (B) shows restricted diffusion in both frontal lobes, likely reflecting a combination of fungal cerebritis and infarction from angioinvasion. Of note, the cavernous sinuses were normal on other images, despite prominence of the right superior ophthalmic vein in (A).



FIGURE 11. Cryptococcal meningitis. An axial CT image demonstrates a round focus of low attenuation in the region of the left lentiform nucleus in a patient with AIDS and cryptococcal meningitis. The lesion was not present on initial CT at admission 2 weeks prior, militating against a dilated perivascular space, and is consistent with a gelatinous pseudocyst.

periventricular/subependymal regions. Parenchymal lesions may be mass-like, but tend not to enhance except in the late necrotic stage.^{23,24}

Human immunodeficiency virus (HIV)

Human immunodeficiency virus (HIV), besides opening the door for opportunistic infections, can infect the CNS directly. In fact, HIV encephalopathy is the most common neurologic manifestation of HIV infection. This encompasses both an acute meningoencephalitis sometimes seen at the time of seroconversion and a spectrum of chronic diseases known as HIV-associated neurocognitive disorder (HAND). The latter is typically progressive and includes HIV-associated dementia (formerly AIDS dementia complex) as its most severe form. Imaging features tend to progress with the disease, but do not strictly correlate with clinical severity. These include cerebral atrophy and symmetric, typically confluent periventricular and deep white matter lesions without enhancement (Figure 8).^{24,25}

Progressive multifocal leukoencephalopathy (PML)

Progressive multifocal leukoencephalopathy (PML) is an opportunistic infection caused by the JC virus and seen almost exclusively in immunocompromised patients. The typical appearance includes asymmetric white matter lesions, predominantly subcortical with characteristic involvement of U-fibers, without mass effect or enhancement (Figure 9). Gray matter structures may also be involved. Longstanding infections may begin to show faint peripheral enhancement. If a patient with AIDS and PML is started on anti-retroviral therapy, the lesions may begin to enhance, and symptoms may worsen temporarily, a condition known as immune reconstitution inflammatory syndrome (IRIS).^{24,26,27}

Other viral infections

Varicella zoster virus (VZV) can cause acute meningitis, notable for its propensity to cause arteritis and infarcts, or reactivation infection of the facial nerve (herpes zoster oticus; Ram-

say Hunt syndrome), appearing as abnormal facial nerve enhancement.²³ A group of arthropod-borne viruses (arboviruses, of which West Nile virus is an example) can cause encephalitis with patchy parenchymal signal changes and variable enhancement; involvement of the deep gray nuclei is common. Cerebellitis and rhombencephalitis are terms referring to encephalitis of the cerebellum and/or brain stem, typically caused by a variety of viral pathogens. A host of other viral pathogens may have variable nonspecific imaging findings.²⁴

Fungal infections

Invasive fungal sinusitis

Seen primarily in neutropenic and diabetic patients, invasive fungal sinusitis is caused by *Aspergillus* or *Zygomycetes* (typically *Mucorales*) species and leads to rapid involvement of the orbits, cavernous sinus, and intracranial structures with a high risk of death or serious disability. Variable amounts of sinus disease may be seen, but extension into the soft tissues and orbits differentiates this seri-

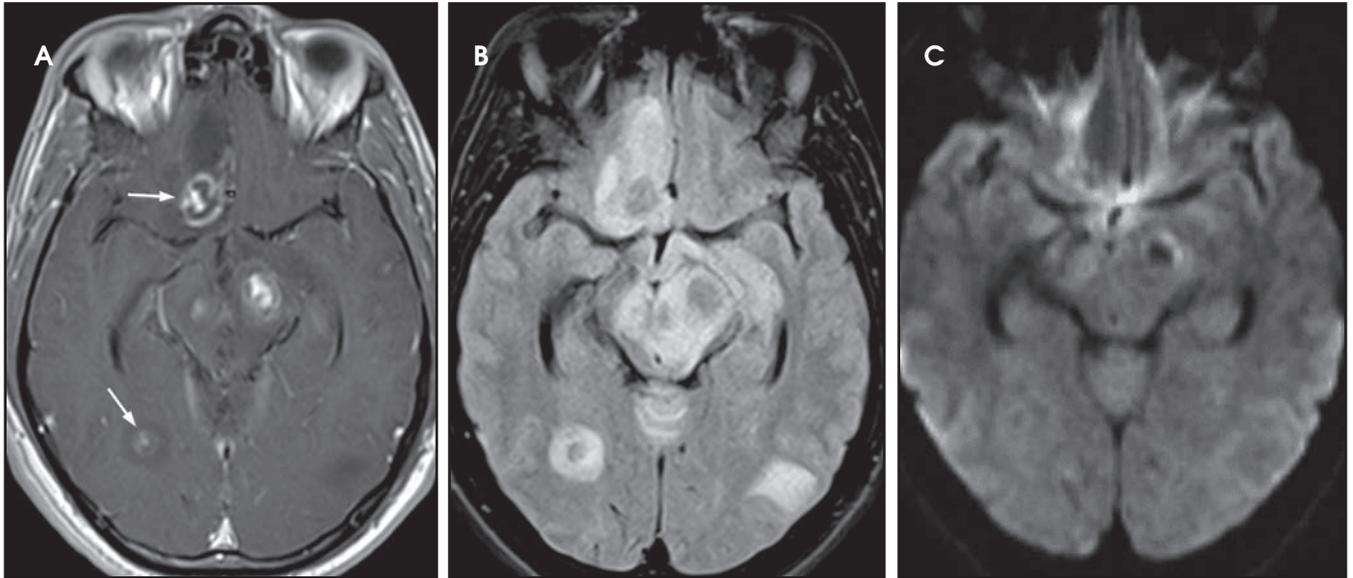


FIGURE 12. Toxoplasmosis. Postcontrast T1-weighted image (A) demonstrates multiple nodular and rim-enhancing lesions, some demonstrating the classic “target” appearance (arrows) typical of toxoplasmosis. FLAIR image (B) exhibits vasogenic edema, and diffusion-weighted image (C) shows normal to facilitated (rather than restricted) diffusion. This patient with AIDS was treated for toxoplasmosis and showed clinical and radiologic improvement over 2 weeks.

ous disease from more benign forms of sinusitis. Epidural phlegmon and leptomeningeal enhancement are indicators of intracranial spread. Direct parenchymal infection or infarcts from arterial invasion are the most severe complications (Figure 10).²⁸

Cryptococcus

Cryptococcal meningitis is seen most commonly in immunocompromised patients. Nonspecific leptomeningeal enhancement may be seen. The fungi may produce a mucinous material that fills and dilates perivascular spaces within the deep-gray and white-matter structures; these are known as gelatinous pseudocysts and tend to mirror CSF in attenuation and signal characteristics without enhancement or diffusion restriction (Figure 11). Rarely, parenchymal involvement may occur, either with one or more rim-enhancing or solidly-enhancing masses (cryptococcomas) or with a miliary pattern of small enhancing nodules.²⁸ Rare reports in immunocompetent hosts have underscored the potential for atypical imaging appearances in such cases, including robust enhancement, diffusion restriction, and inflammatory changes.²⁹

Other fungal infections

Coccidiomycosis, histoplasmosis, and other fungal infections may involve the meninges and/or parenchyma to varying degrees and may be more common and more severe in immunocompromised patients. Basal meningitis is an especially prominent feature in many fungal infections.²⁸

Parasitic infections

Toxoplasmosis

CNS toxoplasmosis is among the most common CNS manifestations of AIDS.²¹ Parenchymal lesions are typically multiple (though occasionally solitary) and most often located in the deep gray nuclei or at the gray-white junctions in the cerebral hemispheres. Larger lesions are rim-enhancing, sometimes with a central dot creating a classic “target” appearance; surrounding parenchymal edema and mass effect are variable, while increased diffusivity (rather than restricted diffusion) of the T2-hyperintense center differentiates these from pyogenic abscesses (Figure 12).¹³ Small lesions may demonstrate nodular enhancement. When differentiation from CNS lymphoma is necessary, thallium-201 scintigraphy will be negative for toxoplasma lesions, while lymphoma reliably demonstrates intense

uptake. Further specificity may sometimes be provided by relative apparent diffusion coefficient (ADC) values, tending towards greater ratios versus normal white matter in toxoplasmosis. Proton MR spectroscopy may occasionally function to differentiate the two processes, demonstrating elevations in the lipid-lactate resonance frequencies in toxoplasmosis, without the elevations in choline resonance sometimes encountered in CNS lymphoma. Perfusion imaging in toxoplasmosis does not commonly demonstrate the perfusion elevations characteristic of lymphoma. Notwithstanding the above considerations, it is the authors’ opinion that imaging features alone are commonly insufficient to conclusively narrow the differential diagnosis between these entities, and therapeutic response monitoring remains common. Treated lesions may develop calcification.

Cysticercosis

Neurocysticercosis is characterized by lesions exhibiting characteristic evolution through stages with unique imaging features.³⁰ Lesions may be parenchymal (though often on the surface), subarachnoid/cisternal, or intraventricular. The first stage (vesicular) is cystic in nature, with internal fluid of CSF attenuation

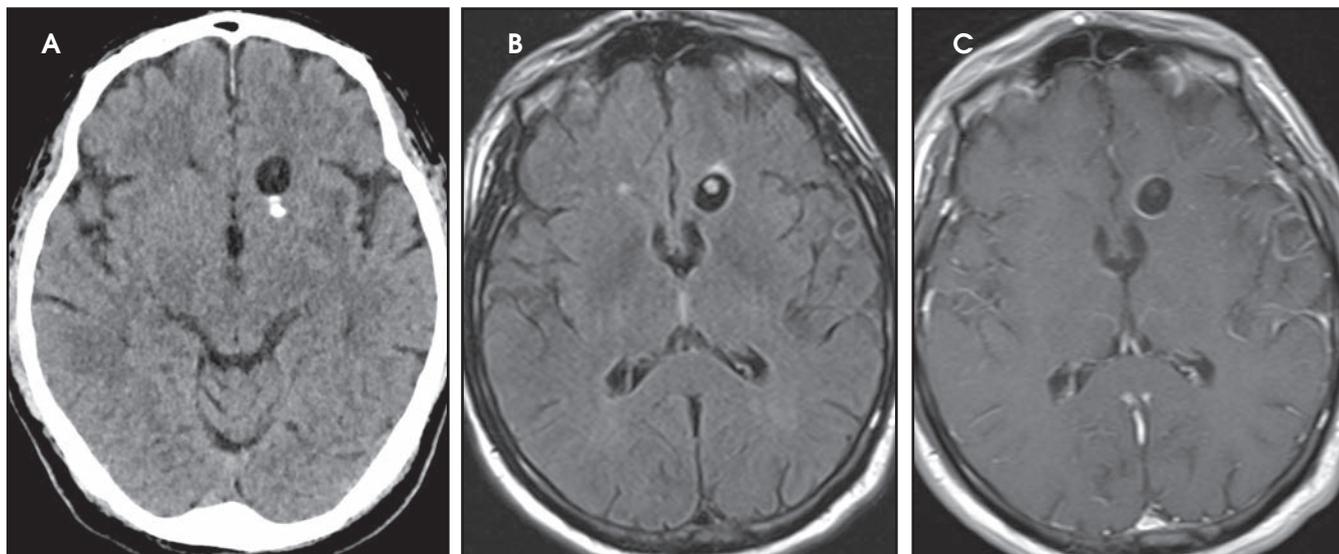


FIGURE 13. Neurocysticercosis. Axial CT image (A) demonstrates a periventricular cystic lesion with faint internal nodule (scolex). Axial FLAIR image (B) shows the cyst fluid to follow CSF signal, and the scolex is more conspicuous; minimal parenchymal edema is apparent. Post-contrast T1-weighted image (C) shows only mild thin rim enhancement. Findings are consistent with the vesicular stage of neurocysticercosis. Adjacent calcification on CT (A) suggests a co-existent lesion in the nodular calcified stage. The patient improved with albendazole therapy.

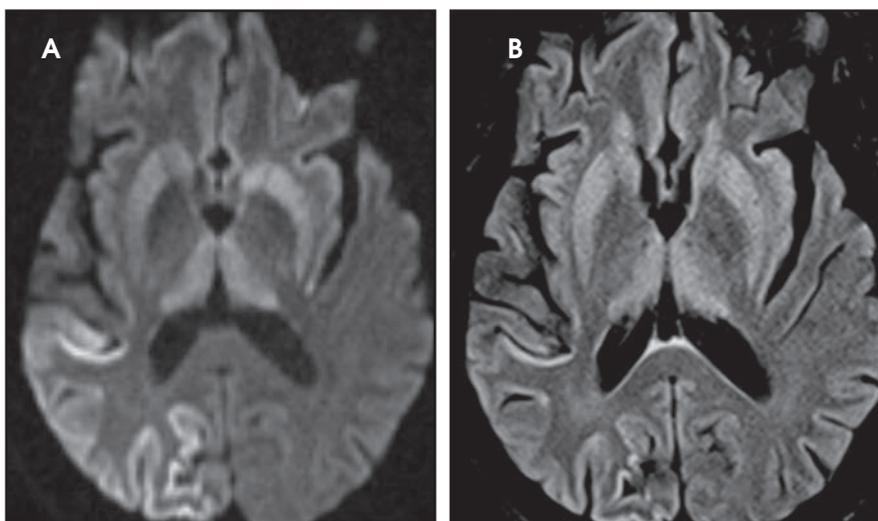


FIGURE 14. Creutzfeldt-Jakob Disease (CJD). Axial diffusion-weighted image (A) demonstrates restricted diffusion in the bilateral caudate nuclei, putamina, and thalami as well as the right temporal and occipital cortex. The findings are also apparent on FLAIR image (B), although cortical ribboning may be less apparent than with diffusion weighting. The pattern is typical of sporadic CJD in this patient with rapidly progressive dementia and myoclonus.

and signal characteristics, sometimes with thin rim enhancement, and without significant parenchymal edema. An intracystic nodule (best seen as hyperintense to fluid on FLAIR or DWI) representing the scolex (head of the tapeworm) may be seen (Figure 13). In the second stage (colloid vesicular), the larva degenerates, the fluid becomes complex (increased attenuation on CT; increased signal on FLAIR; occasional diffusion restriction³¹), perilesional

edema develops, the cyst wall thickens and enhances, and the scolex enhances. Finding a lesion with a cyst and scolex (vesicular or colloid vesicular stage) is sufficient to diagnose neurocysticercosis according to accepted diagnostic criteria.³² In the third stage (granular nodular), the cyst retracts and edema decreases, leaving only an enhancing nodule. Once healing is complete, a nonenhancing calcified nodule remains and may persist indefinitely (nodular

calcified stage); this is best seen on CT, while GRE and SWI are most sensitive among widely available MR sequences. In a given patient, lesions in different stages may coexist. A “racemose” variety involves clustered cysts in the subarachnoid spaces, often without a visible scolex, and may not proceed through the typical stages.³³

Other parasitic infections

Various amebic parasites can infect the CNS, often causing a severe and relentless meningoencephalitis. Imaging findings are varied and nonspecific, typically including enhancing meninges and/or parenchymal mass lesions.²¹

Prion disease

While often categorized as infectious because they can be transmitted from one host to another, prions are not microorganisms but rather native proteins folded in an abnormal configuration. By far the most common prion disease in humans is Creutzfeldt-Jakob disease (CJD), clinically manifested as a rapidly progressive dementia and myoclonus. Imaging findings are rather specific in the appropriate clinical setting, often preceding the full clinical syndrome or characteristic electroencephalographic findings. Findings include nonenhanc-

ing signal abnormalities involving the supratentorial gray matter structures. Increased signal on DWI typically precedes or exceeds that seen on FLAIR (Figure 14). The cerebral cortices, caudate nuclei, and putamina are commonly involved in sporadic CJD, which is typically bilateral though perhaps asymmetric. Involvement of the dorsal thalamus (“pulvinar sign”) and medial and posterior thalamus (“hockey stick sign”) were originally described as being specific for variant CJD but can be seen in the more common sporadic form as well.³⁴ It has been suggested that defining the “pulvinar sign” as signal intensity (on FLAIR images) in the pulvinar greater than that in the anterior putamen affords a high degree of specificity for variant CJD³⁵, though this has not yet been fully validated.

Conclusion

This article describes typical features of various intracranial infectious diseases. While some cases may be pathognomonic in imaging appearance, most will require a combination of clinical, laboratory, and imaging data to arrive at a diagnosis.

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