4 Inflammatory Diseases

Brain Abscess, Bacterial

- **Frequency:** a common computed tomography (CT) diagnosis.
- **Suggestive morphologic findings:** ring-enhancing lesion with smooth margins, often located near the frontal sinus or petrous temporal bone.
- **Procedure:** antibiotic therapy, surgery if required.
- **Other studies:** magnetic resonance imaging (MRI) can detect contrast enhancement with higher sensitivity. It is especially useful for detecting or excluding accompanying meningeal and ependymal reactions. Also better than CT for detection of complicating dural sinus thrombosis.

**Checklist for scan interpretation:**
- Number and location of enhancing lesions?
- Proximity to frontal sinus or petrous bone?
- Inflammatory changes in frontal sinus or petrous bone (bone window)?
- Meningeal enhancement?
- Mass effect (midline shift, ventricular compression, etc.)?

**Pathogenesis**

Bacterial brain abscesses can develop in a variety of clinical settings. Many brain abscesses result from an infection of the frontal sinus or petrous bone that has extended intracranially (Fig. 4.1). In both cases, the normally air-filled portions of these bones become filled with fluid, providing a clue to the etiology of the intraparenchymal changes.

Multiple brain abscesses may develop as a complication of sepsis (Fig. 4.2) or endocarditis (Fig. 4.3). In the latter case, infected thrombi are shed from the diseased valves and disseminate to the brain by the hematogenous route. The blood currents tend to carry the septic emboli through the internal carotid artery and

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**Fig. 4.1 Otogenic brain abscess.** Postcontrast CT shows a digitate pattern of hypodensity in the left temporal lobe, representing perifocal edema. The abscess itself appears as a ring-enhancing structure bordering the calvarium above the petrous bone.

**Fig. 4.2 Septic brain embolism.** Postcontrast CT shows conspicuous enhancing lesions in the right occipital and left frontoparietal areas, and a less prominent lesion in the left occipital area. The gyral changes show the ring-enhancing pattern typical of brain abscess.
into the middle cerebral arteries. Once septic emboli have been seeded into the territory of the middle cerebral artery (MCA), a brain abscess begins to form. This occurs more or less independently of the specific etiology. The initial stage is a focal cerebritis, which soon develops central necrosis and is surrounded by perifocal edema. With time, the lesion forms a capsule and “ripen” into an established brain abscess.

The bacterial inflammatory process can have various central nervous system (CNS) manifestations. The brain abscess may be accompanied by localized or more extensive meningeal inflammation (Fig. 4.4), subdural empyema formation, ventriculitis (Fig. 4.5), or dural sinus thrombosis. Bacterial contamination from a penetrating head injury can also result in a brain abscess (Fig. 4.6).

■ Frequency

The incidence of brain abscess is increased in patients with pulmonary disease (especially pulmonary arteriovenous fistulae or bronchiectasis), patients with endocarditis, and immunosuppressed patients. Males predominate by about a 2:1 ratio.

■ Clinical Manifestations

While purulent forms of meningitis typically present with nuchal rigidity, headache, and hypersensitivity to light and sound, the clinical picture of brain abscess is dominated by focal neurologic symptoms. These may be accompanied by signs and symptoms of meningeal irritation.

■ CT Morphology

The typical CT appearance of brain abscess is that of a ring-enhancing lesion with central hypodense areas on the plain scan did not enhance after contrast administration.
necrosis and perifocal edema (see Fig. 4.1). The ring is usually thin and uniform, without scalloping. As noted above, abscesses due to sinusitis are located either in the frontal lobe or in the middle or posterior cranial fossa in close proximity to the petrous bone. Brain abscesses secondary to endocarditis tend to be small, multiple, and located at the gray−white matter junction in the territory of the MCA.

The early cerebritis stage may not enhance after contrast administration. These patients require serial examinations at close intervals. If the initial examination is delayed, it can be difficult to distinguish brain abscess from other lesions that show a ring pattern of contrast enhancement. Metastases, glioblastoma, and other etiologies should be considered. CT may direct attention to complications that require surgical intervention, such as extensive mass effect. As stated earlier, it is important to recognize subdural fluid collections with accompanying meningeal enhancement that would signify a subdural empyema. Dural sinus thrombosis is not uncommon. A deep abscess may rupture into the ventricular system, giving rise to ventriculitis, with contrast enhancement of the ventricular wall (Fig. 4.5).
**Differential Diagnosis**

Among the various diseases that can produce a ring-shaped enhancement pattern, the features of brain abscess are often the most characteristic. Apart from the typical location of sinogenic abscesses, the ring itself tends to have distinctive features: it is usually thin, circular, and of uniform diameter. By contrast, glioblastoma usually has a scalloped rim, while small tumors tend to have an enhancing rim of varying thickness.

### Tuberculosis

**Frequency:** once very rare, but the incidence has been increasing in recent years.

**Suggestive diagnostic findings:** basal meningitis.

**Procedure:** cerebrospinal fluid (CSF) examination.

**Other studies:** MRI is much more sensitive than CT for detecting meningitis (the most common pattern of CNS involvement).

**Checklist for scan interpretation:**
- Hydrocephalus?
- Meningeal enhancement? Enhancement in the basal cisterns?
- Enhancement of the ventricular wall?
- Enhancing lesions in the brain parenchyma?
- Evidence of ischemic infarcts?

**Pathogenesis**

Cerebral or meningeal involvement by tuberculosis is almost always a result of hematogenous spread. The causative organism in nearly all cases is *Mycobacterium tuberculosis.* Most cases start as a pulmonary infection that spreads swiftly to other organs by the hematogenous or lymphogenous route.

The most common CNS manifestation of the disease is meningitis (Fig. 4.7) or meningoencephalitis.

Other manifestations of intracranial infection are abscess formation and the development of multiple tuberculomas. Involvement of the cranium itself is extremely rare.

Involvement of the meninges, ependyma, and choroid plexus is characterized histologically by typical inflammatory reactions and, in severe cases, by caseating necrosis. Cranial nerve involvement is very common, and even the optic nerve may be affected. When meningitis develops, the associated vasculitis can result in areas of infarction. A frequent complication is hydrocephalus, with its associated clinical manifestations.