Computed tomography and magnetic resonance imaging of tuberculous meningitis

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Tuberculous meningitis usually results from hematogenous seeding of the central nervous system from a primary pulmonary source of infection. Initially, the meningitic process can mimic a flu-like syndrome, followed rapidly by the development of profound neurologic deficits. Computed tomography scanning or magnetic resonance imaging of the head often demonstrates a characteristic pattern of basal cistern involvement with *Mycobacterium tuberculosis*. The incidence of tuberculous meningitis in the more developed countries has increased during the past 5 years because of the growing number of cases in patients with acquired immunodeficiency syndrome, the spread among the homeless, and the expanding immigrant population. The significant morbidity and mortality rates associated with tuberculous meningitis—generally considered to be a disease of the past—emphasize the need for greater clinical awareness, early diagnosis, and prompt treatment.

(Key words: Tuberculosis, meningitis, computed tomography, magnetic resonance imaging)

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Tuberculous involvement of the central nervous system (CNS) continues to be a significant health problem in many underdeveloped countries. Recent reports have indicated an increasing incidence of CNS tuberculosis in the more industrialized nations. This can be attributed to the growing number of cases in patients with acquired immunodeficiency syndrome (AIDS), the expanding immigrant population, and the crowded living conditions in shelters for the homeless.[1-3] Considered by many to be a disease of the past, CNS tuberculosis presents the general practitioner with a disease that may be unfamiliar to him.

Central nervous system involvement usually manifests as a meningitic process; evidence of extracranial tuberculosis (TB) need not be present. The prodromal phase of the disease often mimics a flu-like syndrome with fever, headache, and malaise, quickly followed by the development of meningitic signs and profound neurologic deficits.[4] Although the diagnosis of meningitis is based primarily on the clinical presentation, computed tomography (CT) and magnetic resonance imaging (MRI) exclude any related abnormalities such as intraparenchymal abscess, subdural empysema, hydrocephalus, and cerebral infarction.[4,5]

The cases reported here illustrate the clinical and neuroradiologic features of tuberculous meningitis. In light of the significant morbidity and mortality associated with this condi-

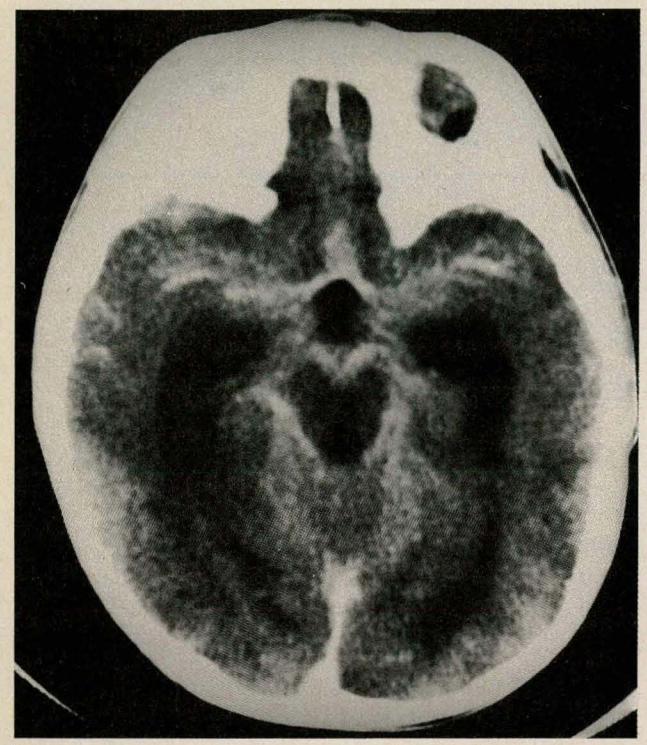

Figure 1. *Tuberculous meningitis in a 13-month-old girl. After administration of contrast, axial CT scan demonstrates marked enhancement of basal cisterns and sylvian fissures.*

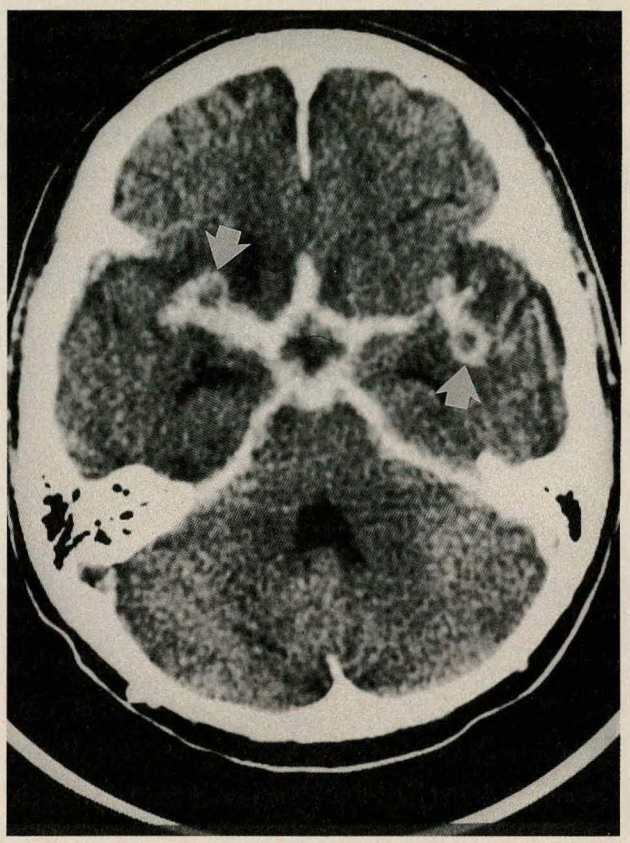

Figure 2. *Postcontrast CT scan from a 28-year-old woman with tuberculous meningitis shows marked enhancement of basal cisterns, extending into sylvian fissures. Tuberculomas (*arrows*) showing ring-enhancing pattern are present in anterior portion of left temporal lobe and right sylvian fissure.*

tion—even in the current era of antibiotics—increased awareness and prompt clinical and radiographic recognition are necessary to avoid devastating neurologic sequelae.

Report of cases

Case 1

A 13-month-old boy was transferred to Tripler Army Medical Center with a 4-week history of intermittent fevers to 104°F (40°C). His condition was originally diagnosed as viral gastroenteritis and otitis media, for which he was treated with oral antibiotics. The fever persisted and neck stiffness and increasing somnolence developed. One day before admission, the patient had become responsive only to painful stimuli and displayed generalized seizure activity.

A CT scan of the head (*Figure 1*) showed marked enhancement of the leptomeninges within the basal cisterns, findings consistent with meningitis. Dilation of the ventricular system was also identified, indicating communicating hydrocephalus. The patient's chest radiograph showed a diffuse miliary pattern suggesting TB. Four-drug antituberculosis therapy was administered. Cultures of cerebrospinal fluid (CSF) and tracheal aspirates subsequently grew *Mycobacterium tuberculosis*. The infant's neurologic status did not improve and he remained comatose.

Case 2

A 28-year-old woman was seen with a 2-week history of fever, headache, malaise, and disorientation. The findings of the physical examination were remarkable for nuchal rigidity, and lumbar puncture revealed pleocytosis, elevated protein levels, and a normal glucose value. A CT scan of the head (*Figure 2*) showed diffuse enhancement of the basal cisterns, ring-enhancing lesions in the right frontal and left temporal lobes, infarction within the right basal ganglia, and hydrocephalus—findings suggestive of tuberculous meningitis.

The patient's neurologic status rapidly deteriorated into a comatose state and did not improve despite the antituberculosis therapy. Her CSF cultures were positive for *M tuberculosis*. No known history of pulmonary, genitourinary, or gastrointestinal TB was present.

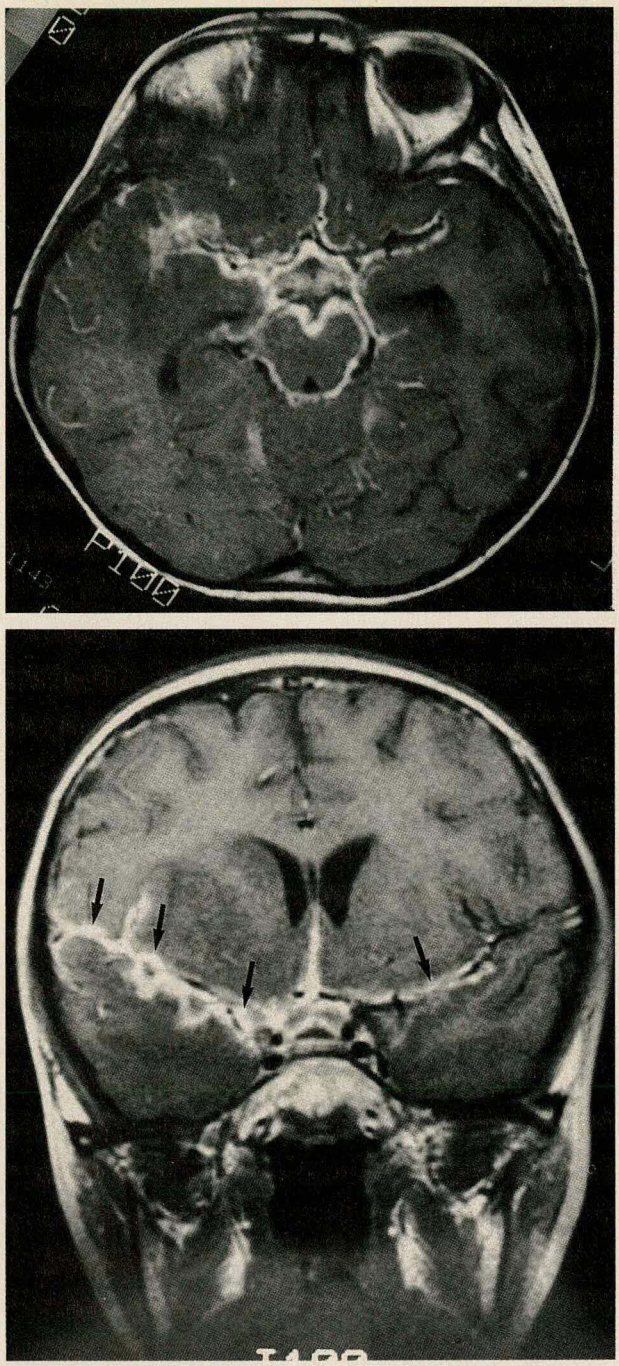

Figure 3. *Tuberculous meningitis in 9-year-old girl.* Top frame: *Axial T1-weighted (750/20) MRI with contrast shows marked enhancement of basal cisterns. Decreased signal in white matter of right temporal lobe is caused by presence of edema.* Bottom frame: *Coronal T1-weighted (750/20) MRI with contrast shows inflammatory enhancement of the leptomeninges within basal cisterns and extending into sylvian fissures* (arrows).

Case 3

A 9-year-old girl was seen with a several-day history of fevers, headache, and new onset of seizure activity. A CT scan of the head showed enhancement of the basal cisterns consistent with meningitis and edematous changes in the right temporal lobe. An MRI examination of the head (*Figure 3*) also showed marked enhancement of the basal cisterns, reflecting leptomeningeal involvement by the inflammatory process. Cerebrospinal fluid cultures subsequently grew *M tuberculosis*. The patient's chest radiograph revealed a calcified azygous lymph node, suggesting previous pulmonary involvement with TB.

Discussion

Tuberculous meningitis is the most common form of intracranial TB and is usually the result of hematogenous spread from a primary pulmonary, gastrointestinal, or genitourinary focus of infection. Approximately 40% of adult cases are secondary to pulmonary disease, whereas the majority of pediatric cases are the result of primary disease in the chest.[4] The mycobacteria are deposited in the cerebral tissue where they incite a granulomatous reaction. The granulomas may remain asymptomatic or rupture into the subarachnoid space, leading to the formation of a purulent, gelatinous exudate filling predominantly the basal cisterns.[4,5]

Inflammatory involvement of the cisternal spaces, leptomeninges, intracranial vessels, cranial nerves, and other cerebral structures can result in a number of clinical manifestations. Irritability, nausea and vomiting, fever, and generalized seizures are common presenting symptoms in the pediatric population. Adults more commonly present with altered mentation, lethargy, headache, fever, and cranial nerve palsies.[4,5] Cranial nerves 6, 3, and 4 are typically involved by the inflammatory process as the result of their course through the cisternal spaces at the base of the brain. Neck stiffness and signs of meningeal irritation (Kernig's sign and Brudzinski's sign) are usually present as well.

Tuberculous meningitis is usually fatal if left untreated, and prompt clinical recognition

of this condition and early therapeutic intervention are necessary to avoid significant neurologic compromise. Approximately 25% of patients who survive will have residual neurologic deficits including hemiparesis, blindness, deafness, and seizure disorders.[4] Mortality rates of 20% for children younger than 5 years of age and 50% for patients older than 50 years have been reported despite antibiotic therapy. These figures emphasize the clinical significance of tuberculous meningitis and the need for early diagnosis.[4]

Neuroimaging of tuberculous meningitis is performed with either CT or MR imaging, and both types of examination can be diagnostic in the appropriate clinical setting. Precontrast CT scans often show obliteration of the basal cisterns because of the presence of the inflammatory exudate. Postcontrast CT scans typically demonstrate intense enhancement of the basal cisterns and sylvian fissures secondary to inflammatory involvement of the leptomeninges.[6] Meningeal enhancement over the cerebral convexities is less common.

Communicating hydrocephalus is a frequent finding on CT and is caused by blockage of CSF reabsorptive pathways by the gelatinous exudate. Inflammatory involvement of the intracranial circulation may lead to arterial or venous occlusion and cerebral infarction. The major vessels of the circle of Willis are usually involved and demonstrate a narrowed and beaded appearance on angiography. Inflammation and subsequent occlusion of the smaller lenticulostriate and thalamoperforate arteries result in basal ganglia infarcts, a common CT finding in severe cases of tuberculous meningitis.[4,7]

Tuberculomas may be present intracranially in the setting of tuberculous meningitis and are typically identified on postcontrast scans as ring- or nodular-enhancing lesions. The ring-enhancing pattern is nonspecific, and correlation with other clinical and radiographic findings is necessary. The tuberculomas commonly calcify with healing and are easily demonstrated by CT. Intracranial calcifications are a frequent finding in patients who survive the meningitis.

The MRI manifestations of active tuberculous meningitis are almost identical to the findings demonstrated on CT. Similar to noncontrast CT studies, unenhanced MRI scans are relatively insensitive for detecting leptomeningeal inflammation.[8] T2-weighted sequences are also insensitive for detecting inflammation caused by the increased signal of CSF within the cisternal spaces.

Striking enhancement of the leptomeninges is readily identified on gadolinium-enhanced T1-weighted images, and enhanced MR imaging has proved to be more sensitive than CT for detecting meningeal involvement along the cerebral convexities because of the multiplanar capabilities of MRI. Communicating hydrocephalus and basal ganglia infarcts are also common MRI findings in tuberculous meningitis.

Magnetic resonance imaging is more sensitive than CT for the detection of ischemic and hemorrhagic infarcts associated with this condition. Edema secondary to inflammation or vascular infarction is easily demonstrated with MRI and is visualized as areas of increased signal on T2-weighted images. Tuberculomas seen on MRI demonstrate variable signal characteristics, depending on the size and maturity of the lesion and the presence or absence of necrosis and edema. Magnetic resonance imaging is less sensitive than CT for detection of calcifications associated with healed tuberculomas.

The leptomeningeal abnormalities associated with tuberculous involvement of the CNS can also be seen with other infectious, granulomatous, and neoplastic conditions. Bacterial, viral, or fungal meningitis can mimic the clinical presentation of tuberculous meningitis and should be considered in the differential diagnosis.[4,5]

Clinical history—such as an antecedent episode of bacterial sinusitis or otitis media, a previous viral respiratory tract infection, or an impaired immune system—helps to narrow the diagnostic possibilities. Additionally, patients with a known malignancy—such as breast cancer, leukemia, or lymphoma—may also present with similar leptomeningeal abnormalities. Central nervous system involvement secondary to syphilis or sarcoidosis

should be included in the differential diagnosis in the appropriate clinical setting.

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