PERSPECTIVE

Overview of Meningococcal Tuberculosis

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Tuberculosis of the central nervous system (CNS) is a potentially life-threatening illness that can be cured if diagnosed early enough. Its clinical and radiologic signs and symptoms may resemble those of other infectious and noninfectious brain diseases. As a result, familiarity with the imaging presentations of various kinds of CNS TB is critical for earlier identification and thus lowering the disease's morbidity and mortality. Although Mycobacterium tuberculosis can affect any organ, the most frequent being the lung, tuberculosis of the central nervous system (CNS) is the most dangerous. CNS involvement affects approximately 5–10% of all tuberculosis patients, and up to 20% of individuals with AIDS-related tuberculosis. Hematogenous spread is the most common cause of CNS tuberculosis, but direct dissemination from an intracranial or extracranial center is uncommon. CNS tuberculosis can mirror other infectious and noninfectious neurological disorders, such as brain tumors, in terms of clinical and radiologic symptoms. As a result, infectious disease specialists must be conversant with the imaging manifestations of CNS tuberculosis to make an accurate and timely diagnosis of this condition. The most prevalent manifestation of CNS tuberculosis is meningitis, which is most encountered in children and adolescents. Tuberculous meningitis is caused by Mycobacterium TB spreading through the bloodstream; however, it can also be caused by the extension and/or rupture of a subpial or subependymal focus (i.e., Rich focus) into the subarachnoid spaces or the ventricular system. Tuberculous meningitis, especially in youngsters, has an insidious course with a vague clinical appearance in the early stages. As a result, imaging is critical in ensuring quick diagnosis and lowering morbidity and death.

On computed tomography (CT) and magnetic resonance (MR) scans, the most prevalent and a rather specific symptom of leptomeningeal tuberculosis is increased exudate in the basal cisterns. In the basal parts of the brain, the exudate contains neutrophils, mononuclear cells, erythrocytes, and bacilli. Meningeal enlargement has been detected in up to 90% of tubercular meningitis cases and is thought to be the most sensitive sign of the disease. The inferomedial surface of the frontal lobes, the anteromedial surface of the temporal lobes, the superior aspect of the cerebellum, and the floor of the third ventricle are the most common sites for subpial exudate. These major sites may also extend to suprasellar, interpeduncular, and pontomesencephalic cisterns. Meningeal involvement can be detected in the sulci over the cerebral convexities, the sylvian fissures, and the ependymal surfaces of the ventricles in most patients; the latter is frequently seen in the latter stages of the disease. Parenchymal illness can occur on its own or in conjunction with tuberculous meningitis. Tuberculoma is the most common manifestation of parenchymal involvement. Cerebritis, cerebral abscess, miliary tuberculosis, and tuberculosis encephalopathy are all possible symptoms. Meningitis may or may not be present with parenchymal tuberculosis. On neuroimaging tests, tuberculosis cerebritis or an abscess may appear like a pyogenic bacterial illness.

Focal tuberculous cerebritis is extremely rare, causing small areas of patchy enhancement on postcontrast images and hypo- and hypersignal intensities on T1- and T2-weighted images, respectively. Tuberculoma is the most common parenchymal lesion in CNS tuberculosis, and it can appear anywhere inside the intracranial region. The lesion could be single or numerous, and it could be present with or without meningitis. The adult tuberculoma is histologically formed of a necrotic caseous center surrounded by a capsule of fibroblasts, epithelioid cells, Langhans giant cells, and lymphocyte. Miliary tuberculosis is particularly common in critically immunocompromised patients and is generally linked with meningitis or extracranial source sites. The lesions are generally found at the corticomedullary connections because the spread is hematogenous. The lesions are small (2-3 mm in diameter) and may be undetectable on non-contrast MR sequences. In visible lesions, MRI indicates tiny lesions that are hypointense on T2-weighted sequences. On a CT scan, these lesions can occasionally be observed as modest hypodensities.

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