Radiologic Diagnosis / *Radyolojik Tanı*

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What is Your Radiologic Diagnosis?

Radyolojik Tanınız Nedir?

Ekim Gümeler(İD), Gökçen Çoban Çifçi(İD)

Department of Radiology, Hacettepe University Faculty of Medicine, Ankara, Türkiye

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A 17-year-old girl is admitted to the pediatric emergency outpatient clinic with nausea-vomiting and accompanying headache. It was stated that she had suffered a short-term loss of consciousness before she came to the hospital, and then she had meaningless conversations. It was also learned that the patient has a diagnosis of combined immunodeficiency due to the absence of Cernunnos.

Non-contrast computed tomography (CT) examination reveals very mild ventricular dilatation, which is more prominent in the third ventricle (Figure 1). Contrast-enhanced cranial magnetic resonance imaging (MRI) is then performed. In fluid attenuated inversion recovery (FLAIR) imaging, diffuse hyperintensities are observed in the interpeduncular-suprasellar cistern, around the M1 segment of the middle cerebral artery (Figure 2A). Diffuse leptomeningeal enhancement is seen in the suprasellar, interpeduncular, ambient and prepontine cisterns on post-contrast T1-weighted images (Figure 2B). Leptomeningeal enhancement is also observed in the M1 segment of the middle cerebral artery, around the base of the frontal lobe, and in the sylvian sulci, but no markedly increased leptomeningeal enhancement is observed at the cerebral convexity level. In addition, multi-

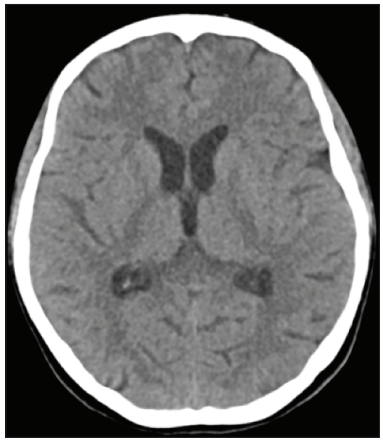

Figure 1. There is mild ventricular dilatation in the non-contrast CT scan.

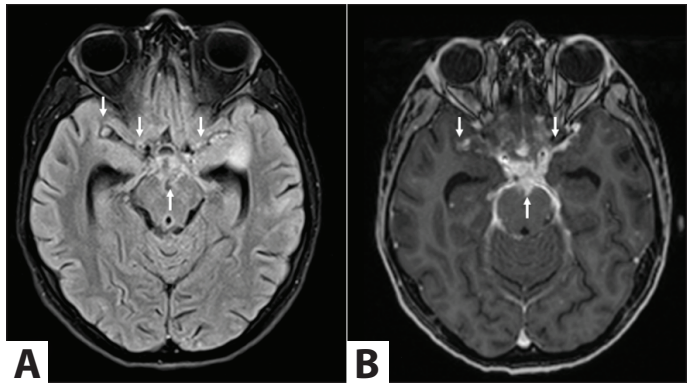

Figure 2. A. In FLAIR imaging, there is hyperintensity in the interpeduncular-suprasellar cistern and around the middle cerebral artery (arrows). **B.** Postcontrast T1W imaging shows marked leptomeningeal enhancement in the basal cisterns and at the base of the frontal lobe (arrows).

Correspondence Address/Yazışma Adresi

Ekim Gümeler

Hacettepe Üniversitesi Tıp Fakültesi,
Radyoloji Anabilim Dalı,
Pediyatrik Radyoloji Bilim Dalı
Ankara-Türkiye
E-mail: ekimgumeler@gmail.com

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ple diffuse nodular foci are detected in the brain parenchyma, including the cerebellum, brainstem, and central gray matter, some of which are accompanied by mild edema (Figure 3A-D). In spinal MRI, diffuse leptomeningeal enhancement is observed along the spinal cord (Figure 3E). What is your diagnosis in the presence of these findings in the patient whose CT and MRI sections are given?

Diagnosis: Tuberculous Meningitis and Miliary Tuberculosis of the Brain

Short Discussion

Central nervous system (CNS) tuberculosis is a very severe form of extrapulmonary tuberculosis and is seen in approximately 5% of the patients (1). Dissemination to the central nervous system occurs hematogenously during the bacillary phase of primary disease or late reactivation. A focus called "Rich focus" develops on meningeal, subpial and/or subependymal surfaces, and rupture or enlargement of this focus results in CNS infection (1).

The most common form of central nervous system infection is meningitis (70-80%) (1). The distinctive feature of tuberculous meningitis is the involvement of the basal cisterns (2). The exudate fills the basal cisterns, most commonly interpeduncular, suprasellar, ambient, and pontine. Cerebral convexity involvement is not common. While the exudate can be seen as iso-hyperdense on unenhanced CT, it shows strong enhancement after contrast. MRI is more sensitive in evaluation. While the exudate in the basal cisterns is hyperintense on FLAIR images, it is heavily contrasted in post-contrast T1W. The most common complications of tuberculous meningitis are hydrocephalus (due to cerebrospinal fluid circulation impairment), vasculitis and infarcts, cranial neuropathies.

Brain parenchymal involvement of tuberculosis is most commonly seen as tuberculoma. Tuberculoma can be single or multiple, usually located at the corticomedullary junction or periventricular area. There are three types of tuberculoma: noncaseating (T1 hypointense, T2 hyperintense, homogeneous nodular enhancement), caseified (T1 iso-hypointense, T2 hypointense, annular enhancement), caseified with central liquefaction (T1 hypointense, T2 central hyperintense accompanied by a peripheral hypointense border) central hyperintense, annular enhancement) (2).

A rarer form of parenchymal tuberculosis is miliary tuberculosis. Miliary tuberculosis presents as numerous, small (<10 mm), contrast-enhancing nodules scattered throughout the brain parenchyma. There is no or mild edema. These lesions are usually located along the perforating vascular structures at the corticomedullary junction. Miliary tuberculosis is often associated with tuberculous meningitis, pulmonary miliary tuberculosis, and/or another primary focus in immunocompromised patients. Although rare, a study conducted in South Africa has reported that miliary tuberculosis is seen in 18% of children with CNS tuberculosis (3).

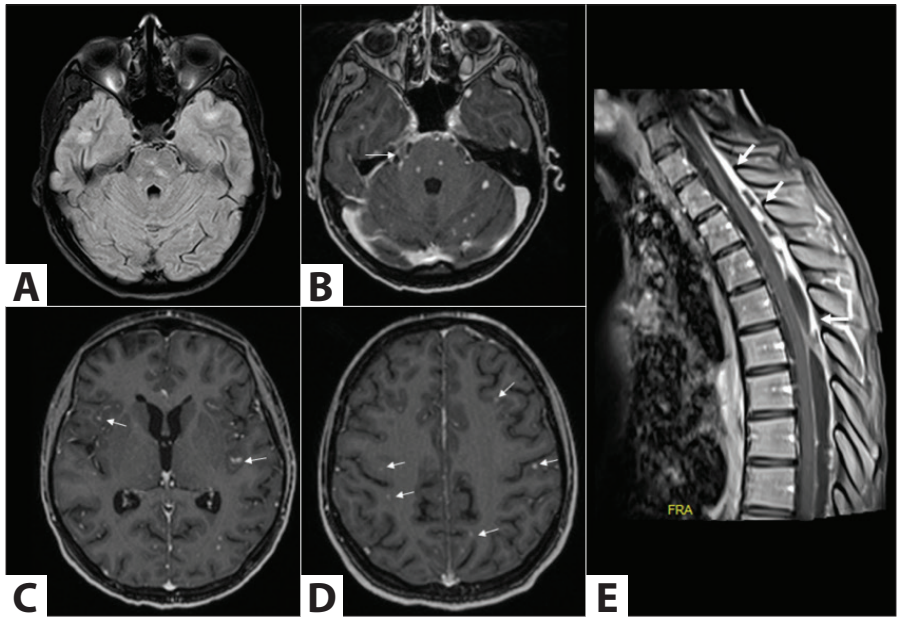

Figure 3. A. FLAIR imaging shows hyperintense nodular foci in the temporal lobes, brain stem and cerebellum. **B.** Intense contrast enhancement in post-contrast T1W images. There is also enhancement in the cisternal part of the left trigeminal nerve (arrow). **C** and **D.** Postcontrast T1W images passing through the superior brain parenchyma show nodular leptomeningeal (arrows) in both sylvian sulci and small nodular foci (arrows) with parenchymal contrast enhancement in both cerebral hemispheres. **E.** Thoracic spinal postcontrast T1W images show intense leptomeningeal enhancement around the spinal cord (arrows).

Spinal meningitis/arachnoiditis due to tuberculosis may be the result of tuberculous meningitis or vertebral osteomyelitis. Spinal tuberculous meningitis is seen as nodular or liner intense contrast enhancement and may completely fill the subarachnoid space. CNS involvement of tuberculosis may also manifest as abscess, cerebritis, encephalopathy, spinal tuberculoma, and myelitis more rarely (2).

In conclusion, CNS tuberculosis has many different involvement patterns, and imaging findings are very diverse. Being aware of the imaging spectrum can aid in early diagnosis, and early diagnosis is vital to the treatment and recovery of patients.

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