Intraventricular Tuberculoma: Single Case Report from Tertiary Care Centre in Eastern India

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ABSTRACT

Intraventricular tuberculomas as a manifestation of CNS tuberculosis is a rare finding. Suspicion for tuberculosis in these lesions is by the constitutional symptoms like low grade fever, weight loss, presence of tuberculosis elsewhere, contact with a TB patient, low socioeconomic status, occurrence in an endemic region, raised ESR, abnormal Chest X Ray findings, positive mantoux test. It is difficult to make a differential diagnosis from lesions if no systemic tuberculosis is present. Medical treatment is the preferred management method of this disease, and surgical intervention should be considered in certain situations. We report a case of an intraventricular tuberculoma in a 15-year-old girl and we discuss the pathogenesis and the radiological findings according this location.

Keywords: Intraventricular Tuberculoma, Case Report, Tertiary Care Centre, Eastern India

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INTRODUCTION

Tuberculomas represent a common neurological disorder in developing countries, forming 12-30% of all intracranial masses (1,2). Central-nervous-system infection with Mycobacterium tuberculosis is seen either in a diffuse form as basal exudative leptomeningitis or in a localized form as tuberculoma, abscess or cerebritis [3]. Intraventricular tuberculomas are not very common and only a few cases have been reported regarding their presence. [4, 5,6,7]. They are usually located in the frontoparietal region and in the basal ganglia, and rarely in the corpus callosum, quadrigeminal cistern, cerebellopontine angle or suprasellar region [4]. No separate diagnostic criterion have been present for intraventricular tuberculomas and they are suspected on imaging studies and confirmed by other noninvasive and invasive investigations. In this report we will describe another case of these rare location of tuberculosis.

CASE REPORT

A 15 year old girl presenting with low grade fever, dull aching headache since 1 month and complex partial seizures with secondary generalization for which she received anticonvulsants and improved. The patient had no history of tuberculosis. Physical examination was normal. Lumbar puncture of the cerebrospinal fluid (CSF) revealed 30 cells/mm³ with 95% lymphocytes. The protein level was 104 mg/dl and the sugar level was 56 mg/dl. CSF Gram stainig and fungal staining were normal. Mantoux was negative. ELISA for HIV was negative. Chest X Ray and EEG was normal.

CECT Brain revealed Rim like enhancing hypodense elliptical lesion with intralesional septae seen in posterior horn of lateral ventricle. Rim enhancing hypodense lesion in right CP angle cistern with rotation of brain stem and dilated temporal horn of lateral ventricle.

MRI of brain revealed lesions hypointense in T1WI, heterogeneously hyperintense in T2WI and heterogeneously hyperintense with peripheral thick rim like hyperintensity on FLAIR in right cerebellopontine angle cistern and extending upto perimesencephalic cistern. After gadolinium administration thick plaque like enhancement is seen. Compression over brainstem is noticed. Similar enhancing lesion also seen in right posterior horn of lateral ventricle. Lesions show peripheral diffusion restriction and GRE reveals calcification foci in the lesions. Pachy meningeal enhancement is seen. MRS revealed increased choline peaks and multiple lipid lactate peaks suggestive of a non neoplastic lesion.
T1, T2, DWI & GRE Axial MRI
CONTRAST MRI

Patient was given antitubercular therapy and became asymptomatic by second week of therapy.

DISCUSSION

Intraventricular tuberculomas as a manifestation of CNS tuberculosis is a rare finding. The most likely mechanism is hematogenous spread of the tubercular bacilli through choroid plexus (4).

During the acute stage non-contrast enhanced CT may show only a hypodense area caused by cerebritis or it may be normal. At the established inflammatory granulomatous stage, the lesion is either isodense or more commonly hypodense with a poorly defined outline on pre-contrast images and has marked enhancement following contrast. At the stage of central caseation, the tuberculoma is either hypodense, or less commonly, isodense or slightly hypodense on pre-contrast images, rarely small central calcifications may be seen. Post contrast images at this stage may show ring-like appearance (9)

Parenchymal tuberculomas demonstrate various patterns. They are typically hypointense on T2-weighted images, but they may be hyperintense as well. Tuberculomas, like bacterial cerebral abscesses, have hypointense walls or rims on T2-weighted MRIs. The cause is unknown, but free oxygen radicals released by the inflammatory process are believed to decrease T2 values. Noncaseating granulomas are homogeneously enhancing lesions. Caseating granulomas are rim enhancing. Granulomas may also form a miliary pattern with multiple tiny, enhancing nodules scattered throughout the brain. Lesions are typically surrounded by hyperintense edema on T2-weighted images. The differential diagnoses include fungal infections, bacterial infections, neurocysticercosis, and cerebral metastases.

MR spectroscopy with a single-voxel proton technique can be used to characterize tuberculomas and differentiate them from neoplasms. Tuberculomas show elevated fatty-acid spectra that are best seen by using the stimulated-echo acquisition mode technique and a short echo time. The necrosis of the waxy walls of mycobacteria within the granuloma is believed to cause the elevation of fatty-acid peaks. The lactate peak is caused by anaerobic glycolysis and is found in inflammatory, ischemic, and neoplastic lesions of the brain; this finding is nonspecific (8).

As antitubercular therapy is very effective in the managent of intracranial tuberculomas and total cure rates are very high (10), and the various imaging studies were suggestive of a likely tubercular pathology so this patient was started on chemotherapy and showed significant improvement of symptoms.

References

upon 107 case records of each. Brain 88: 375-396.


