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Case Report

An interesting case of disseminated tuberculoma of brain and spinal cord.

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Abstract
Tuberculosis is a public health problem worldwide causing significant morbidity and mortality. Manifestations of tuberculosis are widely classified as pulmonary and extra pulmonary. Tuberculoma is one of the CNS manifestations, which on imaging shows ring enhancing lesions. Although intracranial tuberculomas are common, spinal tuberculomas are rare and constitute only 2% of CNS tuberculomas. We present a case of both intra-axial and spinal tuberculoma in the same patient.

Keywords: Tuberculosis, CNS, Tuberculoma, Ring enhancing lesion, MRI.

Introduction

Tuberculosis (TB) is a great threat in developing countries with 8.6 million new cases and 1.3 million deaths worldwide in 2012. It is a disease of poverty affecting most of the underdeveloped and developing countires[1]. In India prevalence of tuberculosis is approximately 1.8 million cases per year[2]. Tuberculoma of brain is an important manifestation of CNS tuberculosis. It constitutes approximately 5% of overall extra pulmonary manifestation of tuberculosis[3]. Spinal tuberculoma on other hand is extremely rare occurring in one out of 50,000 individuals and constitutes only 2% of CNS tuberculosis[4].

Case Report

A 30-year-old male patient presented with history of weakness of both the lower limbs. He was diagnosed with pulmonary tuberculosis three months ago and was put on anti-tuberculosis treatment (ATT). Sputum test for acid fast bacilli (AFB) was positive. Lumbar puncture was not performed due to cerebral edema. The patient was sent for magnetic resonance imaging (MRI) brain contrast study, which was performed using 1.5 Tesla MRI scanner (SIEMENS® MAGNETOM AVANTO) was used for diagnosis. MRI brain plain study showed multifocal, well-defined, variable sized, rounded and ovoid, thick walled solid and cystic lesions with diffuse perilesional edema in bilateral cerebral and cerebellar hemispheres, predominantly at grey-white matter junctions, left pons, cerebellar vermis. Few of the lesions revealed internal T2 hyperintense signal indicating liquefaction and few lesions revealed central T2 hypointense signal indicative of caseation (Figure 1A-1C). There was diffuse cerebral edema with effacement of sylvian fissure and cortical sulci. Additionally, T2 whole spine screening in sagittal sections was performed to rule out possibility of spinal tuberculomas as patient had weakness of both the legs. MRI spine showed similar lesions in cervical and thoracic segment of spinal cord at the level of C4 and D11 vertebral levels (Figure 2A, 3A). On post-contrast study, the liquefactive lesions demonstrated smooth ring enhancement and solid lesions demonstrated homogeneous enhancement (Figures 1D and 2B). On MR spectroscopy elevated lipid peak (1.3 ppm), mildly reduced NAA (3.2 ppm), and reduced NAA/Cho ratio were demonstrated. All vertebrae revealed normal signal, ruling out possibility of vertebral Koch's. Additionally, T2 coronal BLADE sequence of thorax was performed to look for pulmonary manifestations. MRI showed loculated pleural effusion with multiple 'tree-in-bud' appeareance in bilteral lung parenchyma (Figure 3B). The patient was diagnosed with disseminated central nervous tuberculomas with pulmonary Koch's.

The patient was put on ATT treatment, which included isoniazid (INH) 300 mg/day, rifampicin (RF) 450 mg/day, pyrazinamide 1500 mg/day, and ethambutol 800 mg/day daily for two months, followed by INH and RF for four months. Pyridoxine at 40 mg/day was given for all six months. Prednisolone at 1 mg/kg body weight was given for one month and then subsequently tapered over a period of one month. The patient reported improvement in his symptoms after one month of therapy and did not have weakness in both the legs. He also reported improvement in his pulmonary symptoms. The patient was advised for regular follow-up and recommended for repeat MRI of brain and spine to look for regression of lesions.

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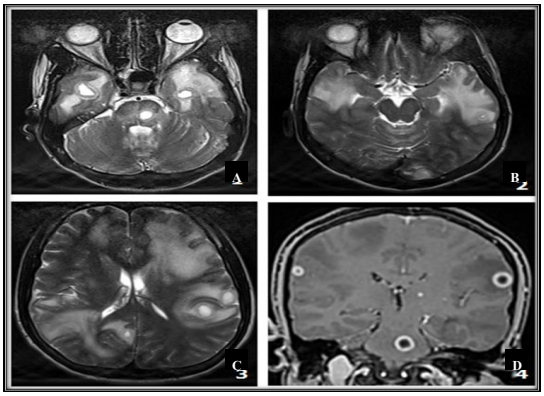

Figure 1. A - Multiple T2 hyperintese with peripheral hypointense liquefactive lesions in left pons (curved white arrow) and bilateral temporal lobes (thick white arrows) with moderate perilesional edema. B – T2 hypointense caeseating lesions in left occipital lobe (thick white arrow) and liquefactive lesion in left temporal lobe (thin white arrow). C – Multifocal liquefactive lesions with moderate perilesional edema in left parietal (thick white arrow) and right occipital lobes (curved white arrow). D – T1-FS Post contrast image showing smooth, thick ring ehancing lesions with moderate perilesional edema in left pons (thick white arrow) and bilateral parietal regions (thick black arrows).

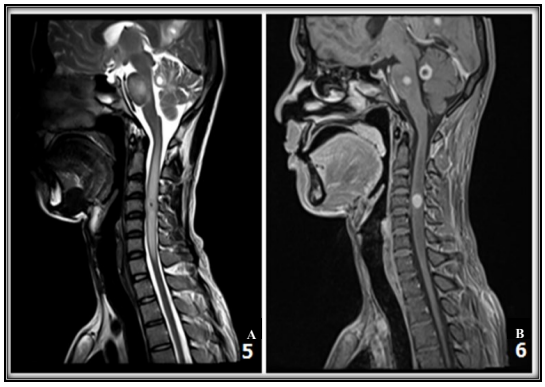

Figure 2. A - T2 sagittal image of cervical spine showing hypointense caeseatig lesion with moderate perilesional edema in pons (thin white arrow) and spinal cord (thick white arrow). Liquefactive lesions with mild perilesional edema in cerebellar hemisphere (thick black arrow) and occipital lobe (thin black arrow). B – T1-FS post contarst image demonstartes homogeneous enhancement of spinal cord (thick white arrow) and pontine lesion (thin white arrow) and thick, smooth ring enhancement in cerebellar lesion (black arrows).

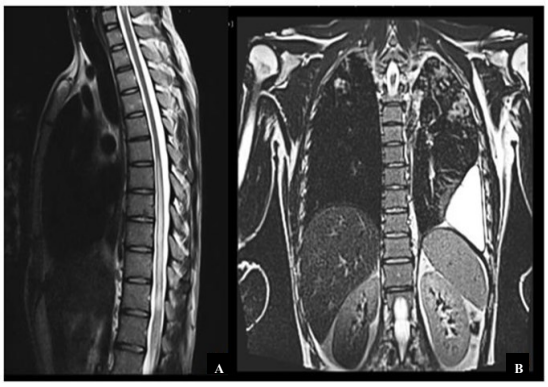

Figure 3. A– T2 sagittal image of dorso-lumbar spine showing T2 hyperintense liquefactive lesion with moderate perilesional edema in the spinal cord (thin white arrow). B – T2 coronal BLADE sequence of thorax showing left oculated pleural effusion (thick black arrow) with multiple 'tree-in-bud' appeareance in bilteral lung parenchyma (thick white arrows).

Discussion:

Central nervous system (CNS) tuberculosis is a life threatening and devastating condition, which is curable when diagnosed in early stages. Imaging manifestations of CNS tuberculosis are meningitis, tuberculoma, miliary tuberculosis, abscess, cerebritis, and encephalopathy. Tuberculoma is the most common parenchymal lesion in CNS tuberculosis which can be found in anywhere in intracranial space. Tuberculomas may be solitary or multiple and may be seen with or without meningitis[5]. Most common clinical findings in CNS tuberculosis encountered is seizure followed by meningitis. Other manifestations include focal neurological deficits, behavioural changes, and altered sensorium[6].

Mode of spread for spinal tuberculoma is through CSF, haematogenous route or local spread from spinal tuberculosis. In our case spinal tuberculoma was associated with pulmonary tuberculosis with associated intracranial tuberculomas. It could be possible that spinal tuberculomas could be due to either spread from CSF or haematogenous route. It is therefore important to look for pulmonary tuberculosis in patients with spinal intramedullary tuberculomas. Lu M et al showed a positive association between spinal tuberculoma and pulmonary tuberculosis[7].

On MRI, the lesions are classified as non caseating granuloma, caseating granuloma, caseating granuloma with central liquefaction and calcified granuloma. Non caseating granulomas are iso-to-hypointense on T1, hyperintense on T2 weighted image (WI) with homogeneous enhancement on post contrast images. Caseating granulomas are hypointense on both T1 and T2 WI with hyperintense rim on T1 WI and homogeneous or ring enhancement on post contrast study. Caseating granulomas with liquefaction are iso-to-hypointense lesion with peripheral T1 hyperintense and

T2 hypointense rim, central hyperintensity on T2 WI and ring enhancement on post contrast image. Calcified granulomas are hypointense on T1 and T2 weighted image with no enhancement on post contrast study.

Conclusion

Spinal tuberculomas are rare CNS manifestations of tuberculosis and should be suspected in any patient who presents with CNS symptoms. It is also critical to look for pulmonary manifestations in a patient presenting with spinal tuberculomas. Prompt treatment and regular follow-up is essential in the management of these patients.

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