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Case Report

Re-Print: An Interesting Case of Disseminated Tuberculoma of Brain and Spinal Cord Type of Study: Case Report

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Received Date: 24 August 2021 | **Accepted Date:** 22 September 2021 | **Published Date:** 01 October 2021

Citation: Parameshwar Keerthi B H, Anil K Sakalecha, Varun S, Shivaprasad G Savagave. (2021). Re-Print: An Interesting Case of Disseminated Tuberculoma of Brain and Spinal Cord Type of Study: Case Report. Biomedical Research and Clinical Reviews. 5(1); DOI: 10.31579/2692-9406/083

Abstract:

Tuberculosis is an important public problem worldwide from 19th century. Manifestations of tuberculosis widely classified as pulmonary and extra pulmonary manifestations. Central nervous system (CNS) tuberculosis is a serious condition where patients most often present with seizure. Tuberculoma is one of the CNS manifestations of tuberculosis. The imaging feature of tuberculoma is ring enhancing lesions. Tuberculoma should be differentiated from other diseases with ring enhancing lesions like neurocysticercosis, coccidiomycosis, toxoplasmosis and malignancies.

Keywords: tuberculosis; CNS; tuberculoma; ring enhancing lesion; MRI

Introduction:

Tuberculoma of brain is an important manifestation of CNS tuberculosis. It constitutes approximately 5% of overall extra pulmonary manifestation of tuberculosis [1]. Spinal tuberculoma on other hand is extremely a rare manifestation occurring at the frequency of 1 in 50,000 individuals. It constitutes only 2% of CNS tuberculosis [2].

Epidemiology:

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 [3]. In India prevalence of tuberculosis is approximately 1.8 million cases per year [4].

Case report:

30 Y/M, a known case of tuberculosis on ATT for 3 months presented with history of weakness of both lower limbs. 1.5 Tesla MRI scanner (SIEMENS® MAGNETOM AVANTO) was used for diagnosis.

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, cervical and thoracic segment of spinal cord at the level of C4 and D11 vertebral levels. Few of the lesions revealed internal T2 hyperintense signal– suggestive of liquefaction. Few of the lesions revealed central T2 hypointense signal– suggestive of caseation. On post-contrast study, the liquefactive lesions demonstrated smooth ring enhancement . Solid lesions demonstrated homogeneous enhancement. Diffuse cerebral edema noted in the form of effacement of sylvian fissure and cortical sulci. On MR-Spectroscopy elevated lipid peak (1.3 ppm), mildly reduced NAA (3.2 ppm), reduced NAA/Cho ratio were seen. All vertebrae were normal. \

Above imaging features were suggestive of disseminated central nervous tuberculoma.

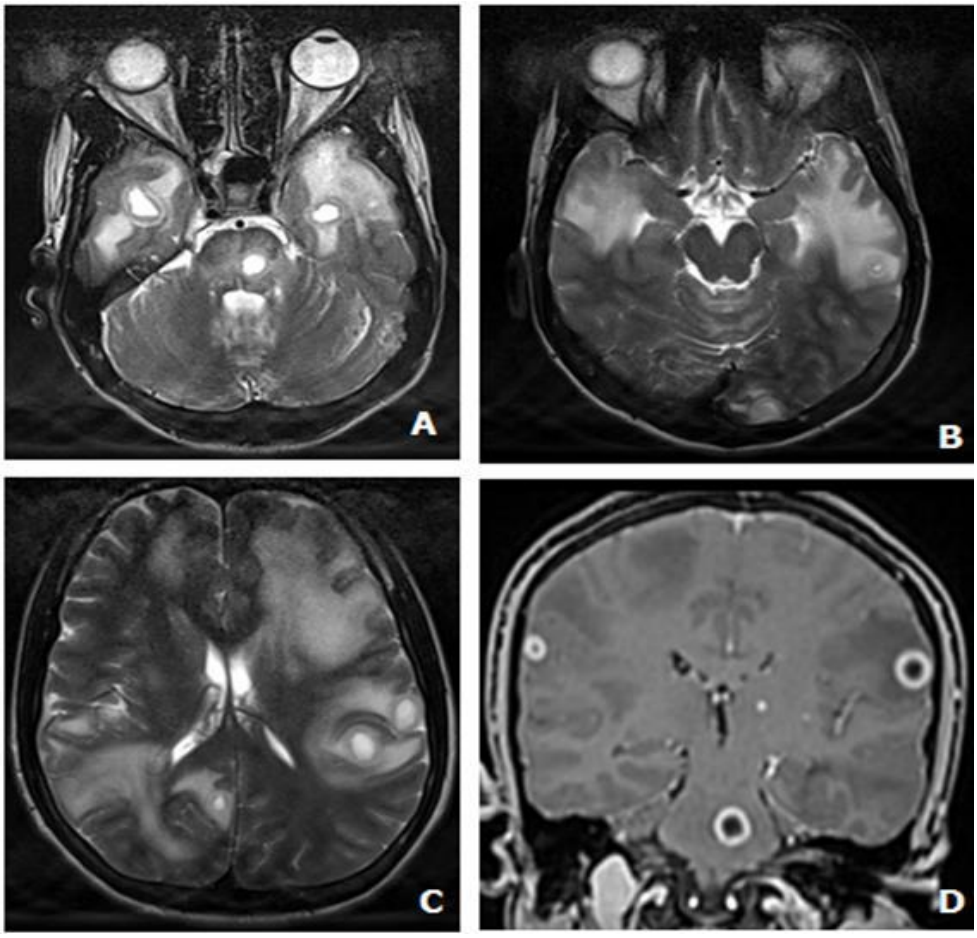

Figure 1: A-*Multiple T2 hyperintese with peripheral hypointense liquefactive lesions in left pons and bilateral temporal lobes with moderate perilesional edema.* **B –** *T2 hypointense caeseating lesions in left occipital lobe and liquefactive lesion in left temporal lobe.* **C –** *Multifocal liquefactive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

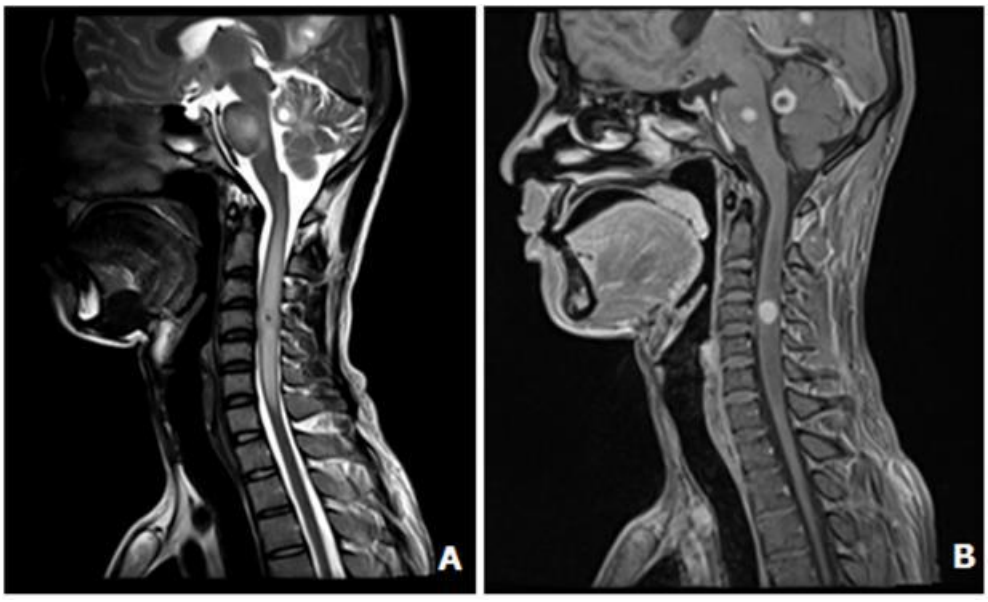

Figure 2: A *T2 sagittal image of cervical spine showing hypointense caeseatig lesion with moderate perilesional edema in pons and spinal cord. Liquefactive lesions with mild perilesional edema in cerebellar hemisphere and occipital lobe.* **B –** *T1-FS post contarst image demonstartes homogeneous enhancement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

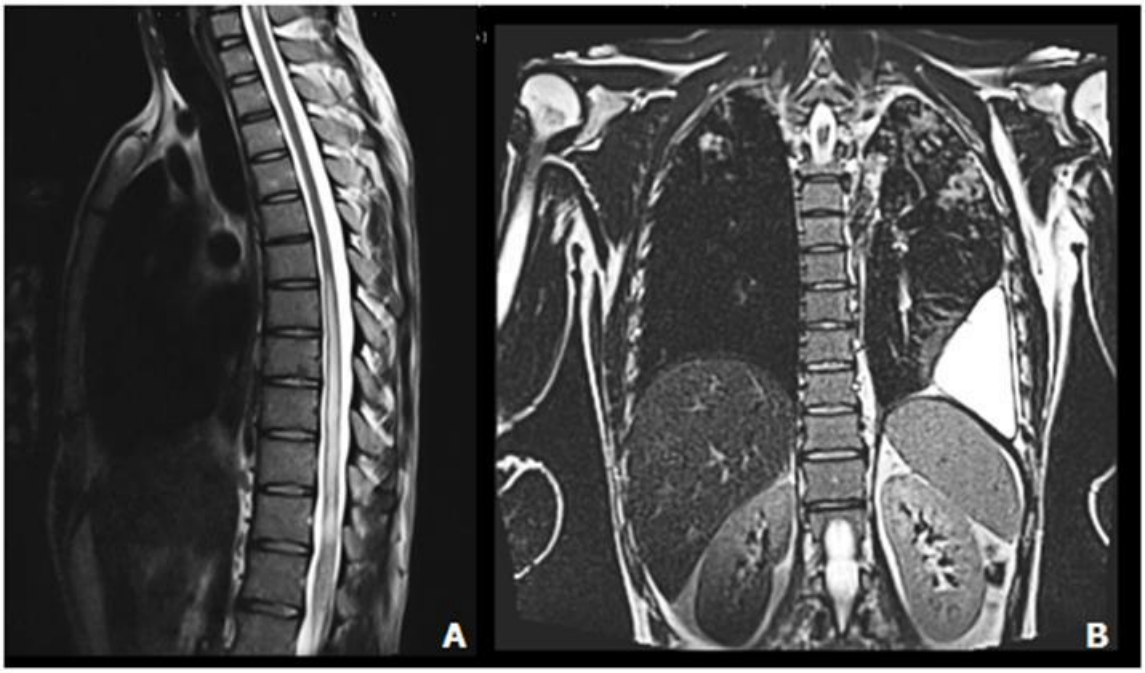

Figure 3: A *T2 sagittal image of dorso-lumbar spine showing T2 hyperintense liquefactive lesion with moderate perilesional edema in the spinal cord.* **B –** *T2 coronal BLADE sequence of thorax showing left oculated pleural effusion with multiple 'tree-in-bud' appeareance in bilteral lung parenchyma.*

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 could be found in any portion of the intracranial space. The lesion 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 CSF spread, haematogenous spread or local spread from spinal tuberculosis. In our case spinal tuberculoma was associated with pulmonary tuberculosis. It is important to look for pulmonary tuberculosis in patients with spinal intramedullary tuberculomas. Lu M et al in his study also 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 granuloma are iso-to-hypointense on T1, hyperintense on T2 weighted image with homogeneous enhancement on post contrast image. Caseating granuloma are hypointense on both T1 and T2 weighted image with hyperintense rim on T1 weighted image and homogeneous or ring enhancement on post contrast enhancement. Caseating granuloma with liquefaction are iso-to-hypointense lesion with peripheral hyperintense rim and hypointense with central hyperintensity on T2 weighted image with ring enhancement on post contrast image. Calcified granuloma are hypointense on T1 and T2 weighted image with no significant enhancement on post contrast study.

Treatment:

ATT treatment include isoniazid (INH) 300 mg/day, rifampicin (RF) 450 mg/day, pyrazinamide 1500 mg/day, and ethambutol 800 mg/day daily for 2 months, followed by INH and RF for 4 months. Pyridoxine at 40 mg/day was given for all 6 months. Prednisolone at 1 mg/kg body weight was given for 1 month and then subsequently tapered over a period of 1 month. Follow up MRI imaging should be performed to look for the reduction in the size of the lesion. Pulmonary CT should also be performed to look for the regression of the lesions.

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DOI: 10.31579/2692-9406/083