

# TUBERCULAR MENINGITIS WITH CONCURRENT INTRACRANIAL AND INTRA-SPINAL TUBERCULOMAS

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Central nervous system (CNS) tuberculosis commonly manifests as tubercular meningitis CNS tuberculomas are more common intracranially and less frequently involve the spinal cord. We report an unusual case of CNS tuberculosis presented with predominant features of tubercular meningitis with concurrent intra-cranial and intra-medullary tuberculomas in any evidence of pulmonary disease. Most noticeable feature of this patient was lower motor neuron type of bladder involvement in the beginning of clinical deterioration that led to the discovery of intra-spinal tuberculoma.

**Key words:** Tuberculosis, tubercular meningitis, intracranial tuberculoma, intra-spinal tuberculoma, tuberculoma

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## INTRODUCTION

The most frequent manifestations of CNS (Central nervous system) tuberculosis are tuberculous meningitis and intra-cranial tuberculomas. Intra-dural spinal tuberculosis commonly manifests as spinal meningitis. Intra-medullary tuberculomas are rare and constitute only 0.2 to 5% of all CNS tuberculomas (1,2). The combination of intra-medullary and intra-cranial tuberculomas is extremely rare and only few cases have been reported in the literature so far (3-7).

We report a case of tubercular meningitis, intracranial and intra-spinal tuberculoma without any evidence of pulmonary disease.

## CASE

A 28 year gentleman presented with high grade fever without chills and rigors, bifrontal headache, vomiting and photophobia of 2 days duration. At the time of admission (At 4.30 PM) he was conscious, alert and oriented. There were no focal neurological deficits. At 3.30 AM he developed urinary retention for which he was catheterized. At about 5.30 AM he noticed weakness of right upper and lower limbs and also weakness of right side of face. Over next 2 hours he became progressively drowsy and lapsed into altered sensorium. On neurological examination he was opening

eyes to deep pain, localizing to pain and talking irrelevantly. He had paucity of the movement on the right sided limbs including right facial weakness. He had neck rigidity and Kernig's sign was positive. Fundus did not show papilloedema. Other physical examination, including the examination of the chest, revealed no abnormality. There was no history of tuberculosis or contact with tuberculosis. On haematological examination, the leucocyte count was 9,500/mm<sup>3</sup> and the ESR at the end of one hour was 40 mm. Other biochemical parameters were normal. Plain CT scan study was normal. On lumbar puncture, clear CSF was drained under normal pressure. CSF protein was raised (149mg/dl) and sugar was normal (68 mg/dl). CSF cytology showed 50 cells (all lymphocytes). Dark-ground microscopy of CSF and staining by Ziehl Nielsen's/Grams' method were negative. CSF culture did not show any growth. The Mantoux test, serology for HIV, Brucella, Syphilis and Toxoplasma were negative. X-Rays of the chest and dorso-lumbar spine were normal and sputum stain for AFB was negative. A diagnosis of tuberculous meningitis was made and he was treated with anti-tuberculous drugs (isoniazid, rifampicin, pyrazinamide and ethambutol), mannitol and dexamethasone. After three days, oral

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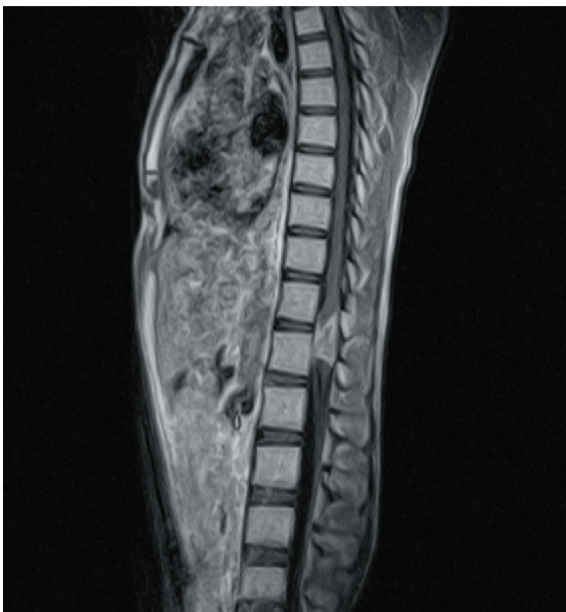
**Figure 1. MRI T1 weighted sagittal section showing iso-intense dilation of conus medullaris**



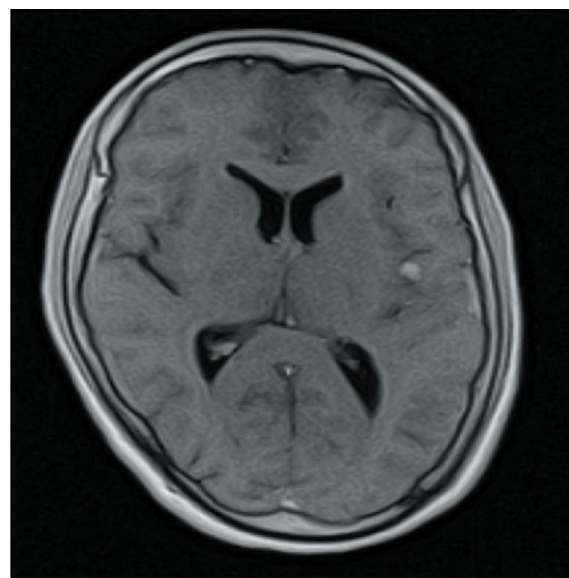
**Figure 2. MRI T2 weighted sagittal section showing that lesion is becoming heterogeneously hyper-intense**

prednisolone in the dose of 2 mg/kg/day was substituted for dexamethasone. Fourth day after admission he recovered in sensorium and he was conscious, alert and oriented. After a week of treatment he recovered in motor weakness but lower motor neuron (LMN) type of bladder dysfunction was persisting. There was no evidence of cerebellar dysfunction or cranial nerve involvement. In view of LMN bladder dysfunction he underwent MRI of the dorso-lumbar spine and screening of the

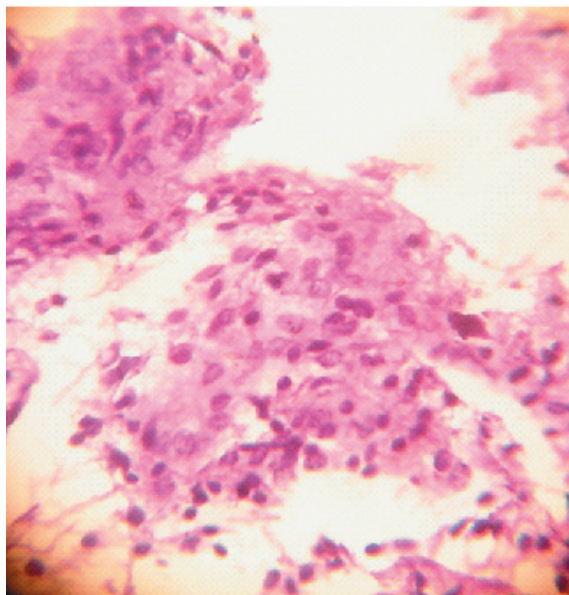
brain. The sagittal T1-weighted MRI images showed an iso-intense fusiform dilatation of the spinal cord, at the level of conus (Figure 1). T2-weighted images showed heterogenous hyperintensity at the conus level (Figure-2). On administration of Gadolinium-DTPA contrast, the lesion demonstrated irregular enhancement at the periphery (Figure 3). There was no abnormality in the vertebral bodies or the paraspinal soft tissues. This lesion, due to its characteristic location,



**Figure 3. On post contrast MRI T1 weighted sagittal section lesion is enhancing at periphery and involving the conus**



**Figure 4. Post contrast MRI T1 weighted axial section of brain showing solid enhancing ring involving medial part of left temporal lobe**



**Figure 5. Histopathological section of thickened arachnoid showing presence of giant cell, epitheloid cell and inflammatory cells (H & E 40x)**

size and classical enhancement was thought to be typical of a tuberculoma. MRI of the brain showed iso-intense lesion on T1, becoming hyper-intense on T2 and enhancing after contrast administration in medial part of the left temporal lobe (Figure 4). Because of large size of lesion and bladder involvement patient was planned for surgery. He underwent D-11, 12 and L-1 laminectomy. After opening the dura there was thickening of arachnoid with adhesions, conus was dilated and congested and nerve roots were adhered to each other. As the conus and roots were densely adhered to each other it was left undisturbed and biopsy was taken from the arachnoid. Histopathological examination of the arachnoid revealed a granulomatous lesion containing multinucleated giant cells, inflammatory cells and epithelioid cells (Figure 5). At follow up patient is doing well however bladder dysfunction is persisting for that he is advised intermittent clean catheterization.

## DISCUSSION

CNS involvement is a less frequent manifestation of extra-pulmonary tuberculosis as compared to the involvement of other systems. Neuro-tuberculosis is seen in up to 10% of patients with systemic tuberculosis and occurs as a result of haematogenous spread from a primary focus, usually the lung (3,5). There may not be any evidence of extra-neural tuberculosis in up to a third cases of neuro-tuberculosis (3). As seen in the

present case, the absence of an extra-neural source, should not rule out the possibility of tubercular aetiology particularly in developing countries. The MRI is a sensitive and non-invasive tool for diagnosing and localizing intra-medullary as well as brain tuberculomas. The lesion appears as an iso-intense or hyper-intense ring on the T1-weighted images and as an iso-intense or hypo-intense lesion on the T2-weighted images. MRI will also delineate the extent of surrounding oedema. MRI also helps in determining the stage of tuberculoma formation. Presence of a bright central spot in the granuloma (target sign) is indicative of central caseation (3,7,8).

Gd-DTPA enhancement MRI is more sensitive than MRI without enhancement in demonstrating the lesions of tuberculoma and arachnoiditis. In early stages of brain tuberculoma contrast MRI will show homogeneous enhancement representing the early tuberculoma stage, which may later evolve to ring enhancement with hypointense center (3,7,8). In present case MRI after contrast administration showed characteristic peripheral irregular ring enhancement of conus lesion suggestive of tuberculoma. However MRI of brain showed solid ring like enhancement of lesion suggestive of early stage of disease without caseating necrosis.

The differential diagnosis of intramedullary tuberculomas includes other granulomas such as cysticercosis, and neoplastic lesions such as astrocytoma, metastasis or lymphoma (3).

In present case, the clinical and imaging features combined with the classical CSF findings were suggestive of tuberculous aetiology as this was later confirmed by histopathology also. Recently polymerase chain reaction (for Mycobacterial protein) combined with contrast MRI has been used with a reasonable degree of certainty, without resorting to an invasive biopsy (3). As this facility is not available with us, we had to rely on the clinical picture for management and on histopathological examination for confirmation of diagnosis.

There is no unanimity regarding the management protocol. Anti-tuberculous therapy with anti-oedema measures is the mainstay of treatment for these patients (7). Conservative treatment with anti-tuberculous medications and a short course of injectable steroids offers an effective, inexpensive, safe and feasible option for treating intra-medullary tuberculoma, especially in developing countries (3). Role of steroid is largely unproven. However in patients with

peri-lesional oedema short-term steroids may be helpful (9). Usually the conservative treatment is successful in achieving complete clinical neurological recovery over a period of one year, which is also accompanied by resolution of the tuberculomas (3). With skilled microsurgical techniques it is possible to safely excise the spinal tuberculomas as these lesions are well circumscribed. Surgery is reserved for the patients with large lesions causing significant compression, patients who do not respond to or deteriorates during conservative treatment (1,2,5,6,8-12). However in present case there was presence of dense adhesion which hampered the surgical decompression. As with any surgical procedure there are attendant risks of anesthesia and probability of developing post-surgical tuberculous meningitis, sinus formation and residual deficit (11,12). Patient is doing well however long term follow up is awaited.

In conclusion, this case emphasizes that tubercular meningitis, intra-cranial and intramedullary tuberculomas can co-exist in a patient. In acute stage, clinical features of tubercular meningitis may predominate (as in present case). However bladder involvement (Particularly LMN type) that persists after recovery in sensorium needs further evaluation to rule out spinal involvement.

#### REFERENCES

1. Suzer T, Coskun E, Tahta K, Bayramoglu H, Duzcan E. Intramedullary spinal tuberculoma presenting as a conus tumour: A case report and review of literature. *Eur Spine J* 1998;7:167-71
2. Citow JS, Ammirati M. Intramedullary tuberculoma of the spinal cord: Case report. *Neurosurgery* 1994;35:327-30
3. Thacker MM, Puri AI. Concurrent intramedullary and intra-cranial tuberculomas. *J Postgrad Med* 2004;50:107-9
4. Huang CR, Lui CC, Chang WN, Wu HS, Chen HJ. Neuroimages of disseminated neurotuberculosis: Report of one case. *Clinical Imaging* 1999;23:218-22
5. Yen HL, Lee RJ, Lin JW, Chen HJ. Multiple tuberculomas in the brain and spinal cord: A case report. *Spine* 2003;28:E499-502
6. Shen WC, Cheng TY, Lee SK, Ho YJ, Lee KR. Disseminated tuberculomas in spinal cord and brain demonstrated by MRI with Gadolinium-DTPA. *Neuroradiology* 1993;35:213-5
7. Lin SK, Wu T, Wai YY. Intramedullary spinal tuberculomas during treatment of tuberculous meningitis. *Clin Neurol Neurosurg* 1994;96(1):71-8
8. Shaharao VB, Pawar M, Agarwal R, Bavdekar SB. Intra-medullary tuberculoma occurring during treatment of tuberculous meningitis. *Indian J Pediatr* 2004;71:107-8
9. Kayaoglu CR, Tuzun Y, Boga Z, Erdogan F, Gorguner M, Aydin IH. Intramedullary spinal tuberculoma. A case report. *Spine* 2000;25:2265-8
10. Kumar R, Jain R, Kaur A, Chhabra DK. Brainstem tuberculosis in children. *Br J Neurosurg* 2000;14:356-61
11. Gupta VK, Sharma BS, Khosla VK. Intramedullary tuberculoma: Report of two cases with MRI findings. *Surg Neurol* 1995;44:241-4
12. Rhoton EL, Ballinger WE Jr., Quisling R, Sybert GW. Intramedullary Spinal Tuberculoma. *Neurosurgery* 1988;22:733-5