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Case Report

Paraplegia due to Tubercular Meningomyelitis: A Case Report

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Abstract

Paraplegia due to Pott's disease or radiculomyelitis are common in developing countries. Though tubercular meningomyelitis presenting as paraplegia is rare. We are reporting a case of tubercular meningomyelitis in a 23-year-old male who had been treated for tubercular meningitis for two months, later presented with sudden onset paraplegia. MRI Brain revealed basal meningitis and MRI of Dorso-lumbar spine showed hyperintense signal changes in long segment of spinal cord at the level of D7 to L1 vertebrae. He was treated with prednisolone and gradually recovered.

Keywords: *Paraplegia; Tubercular meningitis; Radiculomyelitis; Meningomyeliti*

Abbreviations

TB: Tuberculosis; CNS: Central Nervous System; INH: Isoniazid; RMP: Rifampicin; PZA: Pyrazinamide; EMB: Ethambutol; ANA: Anti-Nuclear Antibody; Gd: DTPA: Gadolinium Diethylenetriamine Penta-Acetic Acid; STIR: Short Tau Inversion Recovery; T1WI: T1 Weighted Image; T2WI: T2 Weighted Image

Introduction

CNS TB has a wide and protean manifestations. A delay in the diagnosis contributes to significant neurologic sequalae. Despite optimum treatment, mortality of patients with CNS TB remains significant. Paraplegia due to TB is often secondary to Pott's disease, a common cause of compressive myelopathy in developing countries [1]. TB also can cause spinal tuberculoma and granulomatous arachnoiditis, which may lead to paraplegia or paraparesis [2,3]. Long segment myelitis along with meningitis may cause paraparesis or paraplegia which is very rare [4].

Case Report

A 23-year-old male, who had been treated for one and half months for TB meningitis, was admitted for sudden onset weakness of both lower limbs and bladder incontinence. He was in intensive phase of his anti-tubercular therapy with INH, RMP, PZA and EMB.

Physical examination on admission revealed spastic paraparesis, bilateral extensor plantar responses, and brisk deep tendon reflexes. Asymmetric sensory loss and urinary sphincter dyssynergia were also noted. An MRI of spine revealed hyperintense signal on T2WI, STIR sequence (iso intense on T1WI) involving long segment of spinal cord at the level of D7 to D12 vertebra. Post contrast T1 weighted

image revealed intense enhancement of posterior meninges over a long segment of dorso-lumbar spine at the level of D7 to L1 vertebra, suggestive of meningomyelitis (Figure 1 and 2). MRI brain showed features of tubercular meningitis with multiple granuloma. VEP study was normal. Anti-aquaporin 4 antibody was negative. ANA profile was normal. A lumbar puncture revealed xanthochromic fluid with 300 cell/cm and 6026 mg/100 ml protein. HIV serology was negative and the chest X-ray was normal. Nerve conduction studies were normal.

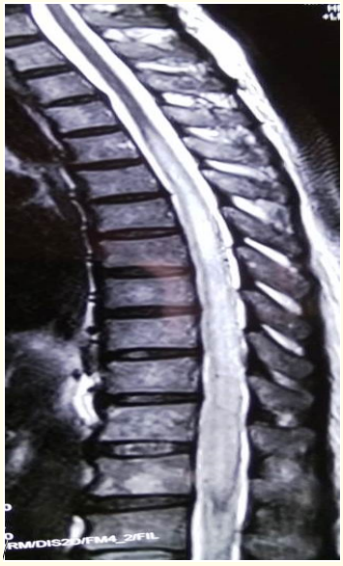

Figure 1

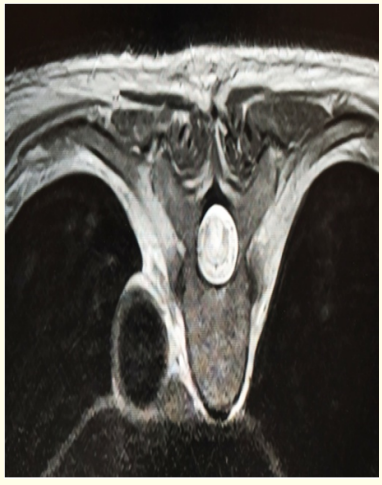

Figure 2

His regime was modified to INH, RMP, PZA, EMB daily and inj streptomycin on alternate day, as well as oral prednisolone 40 mg/day. He started improving after one week. Repeat CSF study after three weeks revealed decreased cells and protein. After one month, he was able to walk without support, had mild sensory loss, mild spasticity, and no sphincter disturbances.

Discussion

Tubercular myelopathy is a rare form of CNS TB. It has frequently been reported in Indian literature because of the high prevalence of active TB. The largest series of patients was reported by Wadia and Dastur, in which 70 patients with tubercular arachnoiditis and radiculomyelitis were treated between 1958 and 1967.

The clinical distinction between the various forms of spinal TB with paraplegia is often very difficult, requires help of newer investigation like MRI. Pott's disease being the most common cause of myelopathy, caused by spinal cord compression due to abscess, granulomatous tissue, or bony displacement. Other forms of myelopathy includes encasing granulomatous arachnoiditis with cord compression, vasculitis of spinal cord and meningomyelitis. Among these, meningomyelitis is rarest. Meningomyelitis may occur as primary lesion, or secondary to intracranial tubercular meningitis, or due to vertebral osteomyelitis. In our patient, MRI of spine showed long segment meningomyelitis involving dorsal cord; MRI brain revealed features of tubercular meningitis and tuberculoma.

Wadia and Dastur [5-7] proposed four mechanisms: (1) border zone edema due to venous impediment, (2) ischemic myelomalacia due to vasculitis, (3) infarction of cord and (4) intramedullary tuberculoma with pericentral necrosis.

Clinical signs usually begin within two months and are followed by a more rapid progressing stage that develops over two to five days. Patient typically presents with paresthesia, weakness, and bladder dysfunction. Radicular pain may be there if radicular involvement. Our patient presented with paraparesis and bladder dysfunction, after one and half month of initiation of anti-tubercular therapy.

Chang., *et al.* [8] have stated that conventional myelography remains the primary radiographic method for spinal radiculomyelitis. Nowadays Gd-DTPA-enhanced MRI is the investigation of choice. It can reveal tuberculoma, enhancement of dura-arachnoid complex around the cord, and segmental enhancement or swelling of cord.

In our patient addition of corticosteroid resulted in improved outcome. Despite the high mortality rate in the range of 30% of exudative tubercular meningitis with myelopathy, our patient responded to medical therapy alone.

Conclusion

We report a rare case of tubercular meningomyelitis with intracranial tubercular meningitis. Paraplegia due to spinal TB is a common entity in developing country, but the mechanism of spinal involvement varies from case to case. Technologies such as MRI, which are not always available to most of the patients, are of significant importance to characterize the nature of spinal cord involvement. The therapy for spinal TB should be conservative as the neurological deficits is mainly secondary to inflammatory process.

Source of Support

Nil.

Conflict of Interest

Nil.

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