Short Communication

Intramedullary cervical tuberculoma

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Introduction

Tuberculosis continues to be one of the most important causes of paraplegia in developing countries.¹ Intramedullary tuberculoma is uncommon and has been reported in six out of 74 autopsy cases with CNS tuberculosis.² In an MRI study, however, intramedullary tuberculoma was found in eight out of 24 patients with intraspinal tuberculosis.³ We recently managed a patient with intramedullary tuberculoma who had disseminated miliary lesions in the brain and a solitary spinal lesion and responded to a combination of methyl prednisolone and antitubercular therapy. In this communication, we report the clinical, radiological and neurophysiological changes in this patient.

Case report

A 15-year-old boy was admitted with 4 days history of rapidly progressive asymmetric leg weakness with retention of urine. He complained of severe backache extending from cervical to mid-dorsal region. Two months earlier he suffered from low grade fever and headache, and had a generalised tonic clonic seizure for which he was prescribed antitubercular treatment. After 3 weeks, his fever and headache subsided. Four years earlier he had suffered from cervical lymphadenopathy, diagnosed to be tubercular on the basis of fine needle aspiration and cytology, for which he received antitubercular therapy (HRE) for 6 months.

On examination, the patient was pale, temperature of 38°C, pulse 80 beats.min⁻¹, BP 130/80 mmHg. There was no sign of meningeal irritation. He had tenderness over the upper dorsal region. The lower limbs were spastic with grade 0 power, knee and ankle reflexes were brisk and with bilateral extensor plantar responses. Joint position and vibration sensation were impaired in the legs. Pinprick sensation was reduced below the tenth thoracic spinal level. He required intermittent catheterisation for urinary retention.

His haemoglobin was 11 gm%, blood counts were normal and ESR 25 mm. Blood chemistry, serum electrolytes and liver function tests were normal. Blood tests for syphilis and HIV were negative. Radiograph of chest (PA) was normal. Mantoux test was positive, ELISA for Myco TB was positive in CSF. Cerebro spinal fluid under normal pressure protein 496 mg.dl⁻¹; glucose 20 mg.dl⁻¹, 300 lymphocytes.mm³, and CSF was negative for acid fast bacilli. Cranial CT scan was normal. Gadolinium enhanced MRI scan revealed multiple small enhancing lesions in the brain parenchyma and a solitary lesion in spinal cord at the level of fifth cervical vertebra (Figure 1). Plain MRI scan in T1, T2 and PD sequences, however, was normal. There was mild meningeal enhancement of cervical spinal meninges. Electroencephalogram revealed background activity of theta range with superimposed delta activity. Central motor conduction time to abductor digiti minimi (CMCT-ADM) was normal bilaterally but to tibialis anterior (TA) was not recordable. Tibial somatosensory evoked potentials were also unrecordable. His visual evoked potential and Brainstem auditory evoked potential studies were normal.

The patient was prescribed rifampicin 450 mg, isoniazide 300 mg, pyrazinamide 1500 mg and ethambutol 800 mg with methyl prednisolone (MPS) 500 mg iv for 5 days followed by oral tapering dose of prednisolone over 2 months. On seventh day of MPS, his backache disappeared, he became continent and lower limb power improved to grade III. At the 3 month follow-up the patient was able to walk with minimal support and was partially dependent for his daily activities. His central motor conduction time (CMCT) to tibialis anterior; improved 22.0 ms on right and 34 ms on left side. Tibial somatosensory evoked potential however remained unrecordable. At the 8 month follow-up the patient had full functional recovery but with lower limb spasticity and knee and ankle hyperreflexia. He had impaired joint position in the right leg. Tibial SEPs were still unrecordable but CMCT-TA on the left side had further improved to 29.0 ms.

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Figure 1 (a) Gadolinium enhanced spinal MRI revealing a disc enhancing lesion at C₅ vertebral level. There is meningeal enhancement as well. (b) Gadolinium enhanced cranial MRI in axial cut revealing multiple miliary enhancing lesions. There is mild meningeal enhancement as well

This patient with intramedullary tuberculoma developed acute paraplegia and retention of urine which responded dramatically to MPS and antitubercular therapy. This improvement is more likely to be due to MPS rather than ATT. Megadose corticosteroid therapy restores the blood brain barrier, reduces oedema and prevents circulatory toxins or immunoreactive cells from entering the CNS.⁴ Cranial MRI of our patient showed multiple granuloma but the paraplegia was probably due to a spinal granuloma because of the horizontal levels of sensory loss. Cervical spinal cord pathology usually results in quadriplegia; partial or small lesions, however, may manifest with paraplegia and a dropped sensory level due to laminar arrangement of motor and sensory fibres. The diagnosis of tuberculosis in our patient was based on positive IgM ELISA in CSF and lymph node biopsy consistent with tuberculosis. Tuberculoma develop following haematogenous dissemination of bacilli from an infection elsewhere in the body, usually lung. The tuberculoma range from 2 and 40 mm in size and may enlarge and coalesce. In our patient, the lesions may be of immature form, because of their small size, lack of visualisation on plain MRI and rapid response to therapy. Spinal tuberculoma are commonly associated with vertebral involvement. Out of 74 patients of tubercular paraplegia, without apparent Pott's spine, 48 had extradural granuloma, 19 had spinal meningitis, one with meningitis secondary to an extradural infection and only six had intraspinal tuberculoma.² The clinical picture of our patient simulated acute transverse myelitis. Such presentation may occur due to associated vasculitis leading to infarction but it was unlikely because MRI was normal in T1, T2 and PD sequence and the lesions could be detected only in contrast MRI and the morphology of cranial and spinal lesions were similar. It is possible that these lesions were detected early in the course of tubercular dissemination and could only be diagnosed by contrast MRI studies. Our patient improved following MPS adjunctive therapy but further studies, are needed to clarify the role of MPS therapy in addition to ATT especially in patients presenting with acute spinal cord syndrome.

References

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