Case Report

Spinal cord compression due to primary intramedullary tuberculoma of the spinal cord presenting as paraplegia: A case report and literature review

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Abstract

Background: Spinal cord compression can be due to various causes but spinal intramedullary tuberculoma is a rare cause. We report a case that had an intramedullary spinal cord tuberculomas in which the diagnosis was made histologically, without evidence of symptoms of systemic tuberculosis. This lesion, located in the thoracic region, mimicked as an intramedullary tumor radiologically.

Case Description: The patient was a 25-year-old male who presented with a history of progressive paraparesis. Initial diagnosis was made as an intramedullary tumor by magnetic resonance imaging (MRI). The treatment of the patient involved is complete surgical excision of intramedullary lesion followed by appropriate antituberculous therapy. Postoperatively, his neurological symptoms were dramatically improved. With combination of both surgical and medical treatments, excellent clinical outcome was obtained.

Conclusion: This case illustrates the risk of misdiagnosis and the importance of histological confirmation of a pathological lesion as spinal cord tuberculoma prior to surgical therapy, which should be kept in mind as a differential diagnosis of the intramedullary spinal cord tumors.



Key Words: Intramedullary, spinal cord tumor, tuberculomas

INTRODUCTION

Tuberculosis (TB) of the central nervous system (CNS) is rare, with an incidence of 0.5–2% in patients with systemic TB^[12] of which the majority are intracranial, with intradural and intramedullary spinal tuberculomas (IMSTs) comprising only a small percentage of cases. Involvement of vertebral column is common in TB but intramedullary tuberculomas are rare.^[17] Intramedullary spinal TB was first described by Abercrombie in 1828.^[1] The reported incidence of intramedullary tuberculomas first described by Serra in 1840, is 2 per 100,000 cases of TB and 2 per 1000 cases of CNS TB.^[13] The common age is 18–45 years and is often associated with tuberculous foci elsewhere but is also reported in as early as 4–6 years of age without any tubercular focus elsewhere in the body.^[5] Spinal TB presents commonly as tuberculous spondylitis or arachnoiditis. IMSTs are rare, even in geographic areas where TB is endemic.^[19] IMST should be considered in the differential diagnosis of spinal cord masses. Here, we report a rare case of IMST, where a diagnosis of intramedullary spinal cord tumor was made preoperatively on radiological basis. We describe the diagnosis, treatment, and pathological features of this lesion, which was sparsely reported in literature.

CASE REPORT

A 25-year-old male, a worker in a garment factory, presented with complaints of band like feeling in the upper abdomen, not associated with any abdominal or back pain for 3 months duration. Simultaneously he had urinary hesitancy, a feeling of incomplete voiding of urine along with sense of inadequate evacuation of stool. Fifteen days later he developed descending paresthesia from the upper abdomen up to the both feet followed by weakness of trunk muscles, weakness and tightness of both lower limbs over a period of 2 months, which initially started in left lower limb and subsequently involved the right lower limb. There was no loss of perianal sensation. On examination, his higher mental functions and cranial nerves were normal. His upper limb power was 5 on both sides with normal tone and deep tendon reflexes. His lower limb power was 3 with hypertonia, exaggerated reflexes and ill-sustained clonus on the both side. He had sensory impairment below T5 corresponding to vertebral level D3. General physical examination and other system examinations were normal. A provisional diagnosis of thoracic myelopathy was made and patient was investigated. His complete blood count, renal profile, liver function tests, human immunodeficiency virus (HIV) and hepatitis B surface antigen were negative. His chest X-ray was normal. Erythrocyte sedimentation rate was moderately high and Mantoux was nonreactive. Magnetic resonance imaging (MRI) of whole cord revealed an iso- to hypointense lesion at D3 level on T1-weighted imaging (T1WI). The lesion was iso- to subtle hyperintense with central flow void onT2-weighted imaging (T2WI) [Figure 1], with cord edema rostral to the mass. Contrast-enhanced MRI showed a brilliantly enhancing lesion with hypointense centre at D3 with sharp margins [Figure 2]. The oval-shaped lesion measured 16 × 10 mm. The diagnosis was intramedullary spinal cord tumor by MRI. Because of worsening of the patient's neurological examination, surgical removal of the lesion was undertaken. At D3-4, laminectomy was performed, posterior longidutinal myelotomy was executed, and a well-circumscribed pinkish fleshy mass [Figure 3] was found to be located 2 mm anterior to posterior aspect of the cord. The lesion was dissected along a readily definable plane and was removed totally by use of the operating microscope. The histopathology showed multiple granulomas [Figure 4] comprising of lymphocytes plasma cells, neutrophils, and large number of epitheloid cells [Figure 5] in clusters with demonstration of acid fast bacilli (AFB) [Figure 6] typical of Mycobacterium tuberculosis. Postoperatively the patient was given antituberculus treatment (ATT), started with 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 10 months. Pyridoxine at 40 mg/day was given for all 10 months. Postoperatively, the patient's neurological examination gradually improved and he could sit erect on the bed and able to walk over a period of 3 weeks without support. The follow-up time is 1 1/2 year.

DISCUSSION

Tuberculomas involving the CNS are not uncommon in developing countries. Most of these lesions are intracranial, with the ratio of cranial and spinal lesions averaging 30:1.^[2] Though these lesions are seen at any level of cord, the thoracic cord is the most commonly involved segment.^[11] Sharma *et al.*^[17] reviewed 10 cases of spinal intramedullary tuberculomas and in their series, the most common site of involvement was dorsal cord



Figure 1:T2-weighted sagittal imaging (T2WI) of spine showing oval shaped iso- to subtle hyperintense lesion with indistinct central flow voids at D3 level

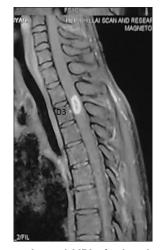


Figure 2: Contrast-enhanced MRI of spine showing a brilliantly enhancing lesion with hypointense centre at D3 with sharp margins

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Figure 3: Operated specimen showing the pinkish fleshy mass

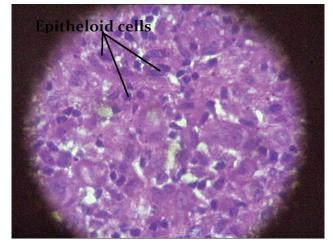


Figure 5: Histopathology section in high power view showing epitheloid cells

followed by cervical, cervicodorsal, and dorsolumbar regions.

Spinal TB can present in various forms, which include Pott's spine (64%), arachnoiditis (20%), intramedullary involvement (8%), and other rarer forms such as subdural and extramedullary involvement (8%).^[1] Dastur reviewed 74 cases of spinal tuberculomas with paraplegia without evidence of Pott's disease and the lesion locations were: Extradural in 64%, arachnoidal in 20%, intramedullary in 8%, and intadural extramedullary in 8%.^[4] The various types of intramedullary tuberculous lesions include tuberculoma, spinal cord edema, and cavitation. IMST is a rare form of CNS TB and an uncommon cause of spinal cord compression.^[2]

Clinically, patients with intramedullary tuberculomas present with signs and symptoms depending on the location of tuberculoma in the spinal cord.^[12] The most common symptoms were those of sub acute spinal cord compression with progressive lower limbs weakness, paresthesia, bladder and bowel dysfunction. The major



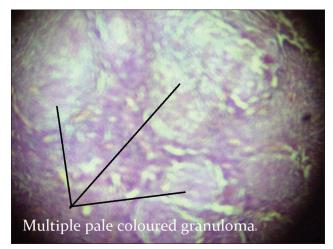


Figure 4: Histopathology section in scanner view showing multiple granulomas

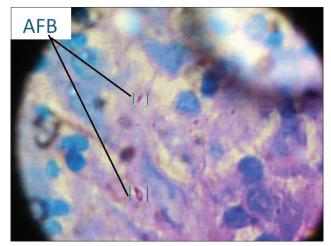


Figure 6: Histopathology section with special stain demonstrating the AFBs

physical findings were paraplegia, either spastic or flaccid. The majority of patients usually have a thoracic sensory level.^[9] Our case also presented with similar picture of sub acute spinal cord compression along with a definite sensory level as described in literature. Most of the reported cases of intramedullary tuberculomas are associated with foci of TB elsewhere in the body but in our case there was no clinical or radiological evidence of TB elsewhere in the body, and the laboratory parameters were not corroborative. Cerebrospinal fluid (CSF) examination is helpful to identify CNS TB, which was not done in our case. CNS TB is increasingly common in immunocompromised patients. In our case, HIV examination was negative.

The traditional investigative modality of myelography has been replaced by the more accurate modality of MRI. The first MRI documented description of tuberculoma was given by Rhoton *et al.* in 1988.^[16] Intramedullary tuberculomas are characterized by ring enhancement,

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with or without accompanying central hyperintensity on T2-weighted MR images.^[8] Contrast MRI shows central hypointensity with rim enhancement and with the development of caseation, T2WI shows a typical "target sign."^[19] Radiologically, the lesions that required to be differentiated from intramedullary tuberculomas include neoplastic (astrocytoma, ependymoma, hemangioblastoma, metastasis. lymphoma). inflammatory, demyelinating (multiple sclerosis), vascular (malformations, infarctions), and granulomatous lesions (syphillis, pyogenic, mycotic, parasitic).^[6] If the lesion has a typical appearance with MRI, and if the patient has systemic TB, the first thing to be thought must be tuberculoma. But, if the patient does not have systemic TB and MRI is not characteristic for tuberculoma, as it is in our case, the diagnosis is difficult to establish. Similar highly confusing clinico-radiological picture described by Arora et al.^[2] in cervicomedullary junction tuberculoma of a patient co-existing with spinal myxopapillary ependymoma and Kadir et al.^[9] in a patient with thoracic spinal intramedullary tuberculoma. Thacker et al.^[18] have reported a rare case of concurrent intracranial and IMST. Jain et al.^[7] have reported 17 cases of intraspinal tubercular granuloma (15 extradural and 2 intramedullary) with neurological deficit and only 3 cases having past history of TB. Ozek et al.^[14] have reported an interesting case of intradural extramedullary tuberculomas mimicking as en plaque meningioma. A case of pulmonary TB presenting acutely as paraplegia (MRI spine revealed a paravertebral abscess extending from T7 to L2 vertebra) without any preexistent complaints of cough with sputum, fever, night sweats, or weight loss (but subsequent sputum examination was positive for AFB and computed tomography chest revealed bronchiectatic changes in the right lower lobe) reported by Apurva Pande.^[15] Intramedullary spinal cord tubercular abscess with involvement of whole cord with syrinx formation following disseminated meningitis were also reported by Khalid et al.^[10]

The importance of surgical treatment for intramedullary tuberculoma extends to the diagnostic procedure, curative procedure, and palliation. The palliative surgical procedure was effective against the lesion responsible for the neurological deficits among intramedullary tuberculoma.^[9] Results of combined surgery and medical treatment are excellent^[17] as it is in our case. In the series reported by Mac Donnel *et al.*,^[13] 65% of the patients had recovery after surgical resection. Safe excision of intramedullary tuberculoma has been advised when: (i) Neurological deficits are present; (ii) severe

compression of cord parenchyma is evident; (iii) diagnosis is uncertain; or (iv) there is clinical deterioration in spite of adequate antituberculous drug therapy.^[3]

CONCLUSION

IMST, although a rare entity, must be considered in the differential diagnosis of spinal cord tumors to avoid unnecessary surgery. If the lesion is known, medical therapy with ATT is the mainstay of the treatment. But timely surgical decompression in selected cases, followed by ATT provide excellent long-term outcome when diagnosis is uncertain or there is clinical deterioration.

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