Spinal Cord (2010) 48, 80–82

www.nature.com/sc

CASE REPORT

Intramedullary thoracic tuberculoma

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Study design: Case report.
Objective: To report a rare case of intramedullary thoracic tuberculoma.
Setting: China Rehabilitation Research Center, Beijing, China.
Case report: A 42-year-old Chinese man presented with the complaints of weakness of the lower extremities and urinary retention. Neurological examinations confirmed the motor dysfunction of the lower extremities and hypesthesia in areas below the T8 dermatome. Results from laboratory studies were normal and X-ray over the chest and thoracolumbar spine showed no signs of tuberculosis. However, the magnetic resonance imaging (MRI) scan detected a ring-enhancing intramedullary lesion at the T6–7 intervertebral level. The condition was diagnosed as intramedullary glioma. A neurosurgical resection was carried out, and the pathological examination following the surgery confirmed the tubercular lesion. The patient was treated with antituberculous therapy after the surgery and resulted in a satisfactory recovery.
Conclusion: Intramedullary spinal tuberculoma (IMT) is rare, and the diagnosis for IMT is often difficult and inconclusive, especially in patients without symptoms of systemic tuberculosis. Although the management of IMT remains challenging, our report suggests that the combination of microsurgical resection and antituberculous chemotherapy may represent an effective treatment strategy for IMT.
Spinal Cord (2010) **48,** 80–82; doi:10.1038/sc.2009.58; published online 9 June 2009

Keywords: tuberculoma; intramedullary lesion

Introduction

Intramedullary spinal tuberculoma (IMT) is a rare form of spinal tuberculosis, which can be easily misdiagnosed.[1] Here, we report a case of intramedullary thoracic tuberculoma in a patient who had no history of previous systemic disease.

Case report

A 42-year-old Chinese male complained about the weakness in his lower extremities and urinary retention that lasted for two weeks. T2-weighted magnetic resonance imaging (MRI) indicated an intramedullary schistic hyperintense lesion at T6–7 intervertebral level. Being suspected for myelitis infection, the patient was treated with dexamethasone and γ-globulin. However, the symptoms worsened after six weeks of treatment. Neurological examinations at this time showed a decreased muscle power in the lower limbs and hypesthesia in areas below the T8 dermatome. Hyper-reflexia was elicited in the lower limbs, and plantar response was extensor on both sides. Laboratory tests, including complete blood count, erythrocyte sedimentation rate, the examination of cerebrospinal fluid and cerebrospinal fluid culture were all normal. X-ray film showed no abnormality over the chest and thoracolumbar spine, and the result of tuberculin skin test was negative. However, T2-weighted MRI of the thoracic spine detected isointense signals at T6–7 intervertebral level and edema signals surrounding the lesion from T6 to T7. There were isointense signals on the T1-weighted MRI with a ring-shape enhancement of the intramedullary lesion at the T6–7 intervertebral level after gadolinium-diethylene triaminepentaacetic acid administration (Figure 1). Accordingly, the patient was diagnosed for intramedullary inflamed granuloma or glioma. After being treated with anti-inflammatory and antivirus medications and receiving rehabilitation training, the patient achieved a moderate recovery of motor functions in the affected lower extremities. However, the weakness in his left leg began to worsen at one month later, and he was unable to stand. The MRI at this time point showed an enlargement of the intramedullary lesion and edema in spinal cord (Figure 2). A provisional diagnosis of

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Received 4 November 2008; revised 31 March 2009; accepted 6 May 2009; published online 9 June 2009

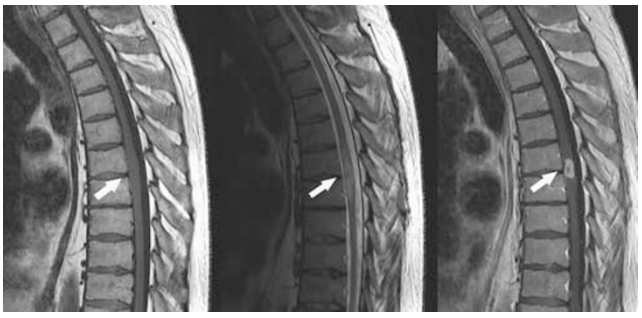

Figure 1 Magnetic resonance imaging (MRI) of the thoracic spine showed an enlargement of the spinal cord between T-6 and T-7 level, isointensive on T1-weighted imaging (left, arrow), ring isointense lesion surrounding the edema on T2-weighted imaging (middle, arrow) and ring enhancement (right, arrow) at the T6–7 intervertebral level.

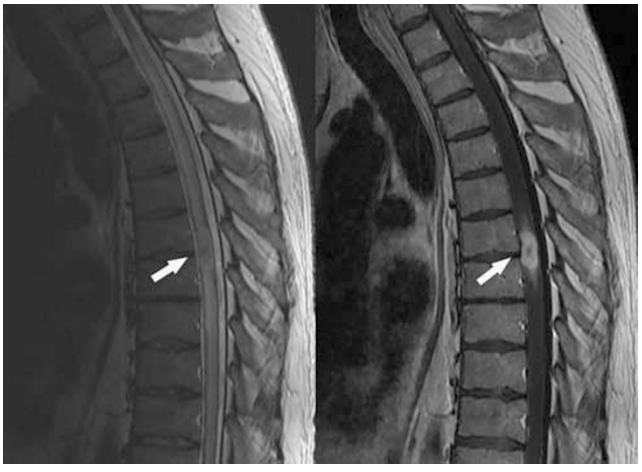

Figure 2 Magnetic resonance imaging (MRI) of the thoracic spine carried out at a later time point showed an extension of the intramedullary lesion (left and right, arrow) and the enlarged edema of spinal cord between T2–T10 on T2-weighted imaging.

intramedullary glioma was made and a resection surgery was carried out on this patient. Unexpectedly, post-operational histopathology test showed caseating epithelioid-cell granulomas, which suggests tuberculosis (Figure 3). Accordingly, the patient started the one-year-long chemotherapy for tuberculosis after the surgery. Six months after the surgery and chemotherapy, the patient was able to walk with support, and regained bladder control.

Discussion

Neurotuberculosis merely represents 0.5–2% of extrapulmonary tuberculous infection in the general tuberculosis prevalence.[2] The incidence of IMT is extremely low and is seen in 2 out of 1000 cases of central nervous system tuberculosis. It seems that IMT occurs more often in young people (mean age: 29.7 years), and thoracic cord is the most commonly affected site.[3] IMT is commonly developed from hematogenous seeding originated from an extraneural focus (usually pulmonary), or it can be a part of miliary

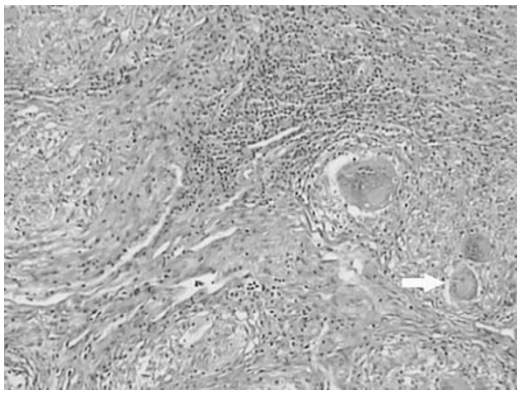

Figure 3 Histopathology of the specimen from the patient showed granuloma with peripheral Langhans' giant cells (arrow) and epithelioid cells (hematoxylin and eosin, original magnification $\times$ 200).

tuberculosis. In other cases, it may present as an isolated extrapulmonary form. The case of isolated intramedullary tuberculosis was rare, and current report suggests that silent hematogenous dissemination of primary tuberculosis could be responsible for such an isolated lesion.

Some characteristic features of IMT can be shown using MRI scans, including isointense ring on T1-weighted imaging with cord expansion, isointense or hypointense ring on T2-weighted imaging and ring enhancement with hypointense centre on gadolinium–diethylene triaminepentaacetic acid MRI. In addition, the extent of cord edema may range from one to nine vertebral levels. However, MRI features vary with the stages of the tuberculoma formation. Therefore, it is difficult to differentiate IMT from other spinal-cord lesions (for example, myelitis, glioma). In this case, the lesion identified by T1-weighted imaging is isointense with ring enhancement, and hyperintense in the outer layer with an isointense center on T2-weighted imaging. In this circumstance, the physician should always consider IMT as one potential diagnosis, regardless of the absence of systemic tuberculoma. The subsequent antituberculous chemotherapy will be helpful for differentiating IMT from other diseases. The pharmacological intervention will also be critical for a better outcome on patient diagnosed at the early phase of the disease.

The antitubercular therapy remains the mainstay treatment for IMT. Although surgery will be considered if the lesion is large and associated with a rapid deterioration of the neurological status, or there is a paradoxical increase in the size of the lesion following antituberculous therapy. Recent study suggested that an early surgical intervention is also important for improving the treatment of IMT, particularly in patient with profound neurological deficits.[4]

One-third of patients with IMT may show only signs of spinal-cord compression, but no symptoms of systemic tuberculosis.[5] As the diagnosis of IMT is usually difficult and inconclusive, the needed surgical and pharmacological treatment can be significantly delayed or neglected. Accordingly, we suggest that the diagnostic and therapeutic surgical resection of the intramedullary lesion should be carried out without delay in patients with deteriorated neurological

deficit or those who are irresponsive to antituberculous chemotherapy. Although the outcome of IMT treatment is unpredictable, we suggest that the combined treatment with microsurgical resection and antituberculous chemotherapy may yield a better result.

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