Intramedullary Spinal Tuberculoma
—Case Report—

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Abstract

A healthy 33-year-old man presented with an intramedullary tuberculoma of the thoracic spinal cord manifesting as a 2-month history of progressive paraparesis and sphincter dysfunction. Magnetic resonance imaging showed ring enhancement of the intramedullary thoracic lesion with perifocal edema. General physical examination was unremarkable with no signs of inflammation except for a positive finding by the tuberculin skin test. Total resection of the intramedullary mass was performed through a posterior myelotomy following T11–12 laminectomy. Histological examination revealed a granulomatous lesion that contained Langhans giant cells, inflammatory cells, and caseating necrosis. Acid-fast bacilli staining of the specimens was positive, and cultures grew Mycobacterium tuberculosis. Postoperatively, the paraparesis and sphincter dysfunction improved sufficiently for the patient to return to his ordinary activities. Intramedullary spinal tuberculoma is rare, but must be considered in the differential diagnosis of spinal cord compression.

Key words: intramedullary lesion, spinal cord, surgery, tuberculoma

Introduction

Tuberculosis of the central nervous system (CNS) is rare, with an incidence of only 0.5% to 2% of patients with systemic tuberculosis.1) The most common form of CNS tuberculosis is meningitis. CNS tuberculoma is unusual, and generally presents as an intracranial lesion. Spinal cord involvement is extremely rare.8,13–15) The ratio of intramedullary spinal to intracranial tuberculomas is 1:42.9) We present a rare case of intramedullary spinal tuberculoma in an otherwise healthy, human immunodeficiency virus-negative Japanese man who showed no other evidence of systemic disease.

Case Report

A 33-year-old man was admitted with a 2-month history of progressive weakness and hypesthesia of the legs. The patient reported urinary urgency and difficulty walking during the 2 weeks prior to admission. The patient had previously been healthy and had no identifiable recent contact with tuberculosis, although the patient had traveled to Southeast Asia 10 years before.

Physical examination, including examination of the chest, revealed no abnormalities. Neurological examination found motor weakness (grade 3/5) in the lower extremities and hypesthesia below the L-1 dermatome. Hyperreflexia and pathologic reflexes were elicited in the lower extremities. Laboratory studies revealed normal complete blood count and no evidence of inflammation. The human immunodeficiency virus serologic test was negative. Examination of the cerebrospinal fluid revealed no abnormality, and culture was sterile. Spinal and chest roentgenography also found no abnormality. The tuberculin skin test, with 100 units of purified protein derivative, was positive. Magnetic resonance (MR) imaging demonstrated an intramedullary hyperintense lesion with a mixed intensity center and enlargement of the spinal cord between the T-8 and L-1 levels on the T2-weighted images, and an isointense lesion on the T1-weighted images with ring enhancement of the intramedullary lesion at the 11-12 intervertebral level after administration of gadolinium-diethylenetriaminepenta-acetic acid (Fig. 1). The diagnosis was an intramedullary
mass, possibly tuberculoma of the spinal cord.

A T11–12 laminectomy was performed, and the dura was opened. There was no evidence of extramedullary abnormality except for spinal cord expansion. Intraoperative ultrasonography was used to localize the lesion. Posterior longitudinal myelotomy was performed and a well-circumscribed grayish mass was totally excised. Histological examination of the specimen revealed a granulomatous lesion containing multinucleated giant cells, inflammatory cells, and caseating necrosis (Fig. 2). Tissue stain for acid-fast bacilli was positive, and tissue cultures grew *Mycobacterium tuberculosis*.

Postoperatively, the patient’s paraparesis gradually improved. Two weeks after the operation, the patient was able to walk without assistance and had completely recovered normal sphincter function. Chemotherapy for tuberculosis was administered for 6 months.

**Discussion**

Intramedullary tuberculosis of the spinal cord is very rare, occurring in only 2 of 100,000 cases of tuberculosis and in 2 of 1000 cases of CNS tubercular disease. The clinical characteristics of intramedullary tuberculoma have been clearly described. Intramedullary spinal tuberculomas are most common in relatively young patients, usually in their 20s or 30s. This review found 94% of patients presented with progressive myelopathy aggravating in several months, 67% with bowel or bladder dysfunction, and 33% with back pain. Intramedullary tuberculoma developed most often in the thoracic spinal cord, but also occurred at other levels. No evidence of active or distant tubercular disease was found in 38% of patients, but the tuberculoma was thought to represent hematogenous dissemination. Clinical signs and cerebrospinal fluid examinations usually show no evidence of meningitis. Therefore, the clinical presentation of patients with intramedullary spinal tuberculoma is principally that of spinal cord compression syndrome.

MR imaging provides detailed, valuable information about intramedullary lesions, and therefore is
useful in determining the pathology. The MR imaging appearance of intramedullary tuberculomas changes according to the stage of tuberculoma formation. In the early phase, inflammatory reaction with variable amounts of peripheral edema is present without the development of a collagenous capsule. At this stage, the tuberculoma appears isointense on both T1-weighted and T2-weighted images, and is homogeneously enhanced. Subsequently, the tuberculoma capsule becomes richer in collagen and the surrounding inflammatory reaction may decrease in intensity or disappear, resulting in an isointense lesion on T1-weighted images and an isointense to hypointense lesion on T2-weighted images, with ring enhancement with a hypointense center. The center of an intramedullary tuberculoma becomes hyperintense on T2-weighted images with the development of liquefied caseation. The solid parts of granulomas may appear as hypo- to hyperintense on T2-weighted images, corresponding to the phase and condition. Cord edema shown as surrounding hyperintensity on T2-weighted images can extend from one to almost nine vertebral levels. MR imaging of our case showed the lesion as isointense on T1-weighted images with ring enhancement, and hyperintense in the outer layer with a mixed intensity center without hyperintensity of the core on T2-weighted images. These findings may correspond to a transitional phase, in which the lesion contains both granuloma with little collagen and organized caseation.

Medical therapy is now curative for intramedullary tuberculoma. Medical therapy without surgical resection may be appropriate for selected individuals under an established diagnosis of tuberculosis. However, the patient’s neurological condition has occasionally worsened while receiving adequate antitubercular therapy. Furthermore, one-third of patients with intramedullary tuberculoma present with only signs of spinal cord compression and no evidence of systemic tuberculosis. The diagnosis and treatment of intramedullary tuberculoma should be undertaken immediately to minimize morbidity, although it is benign and curable. Therefore, if the diagnosis is unclear or the patient does not respond to medical management, surgery to resect the intramedullary lesion should be performed without delay for a definitive histological diagnosis. Intramedullary tuberculoma is usually well-circumscribed after the early stage, so can be removed without neurological embarrassment. In addition to surgery, chemotherapy with antituberculous drugs should be instituted as soon as the diagnosis is established. Optimal medical treatment, even if after surgery, is necessary for 6 months or more to achieve the best neurological outcome.

References

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