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CASE REPORT

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Intramedullary spinal tuberculoma presenting as a conus tumor: a case report and review of the literature

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Abstract Intramedullary spinal tuberculoma is a rare form of central nervous system tuberculosis. Although tuberculosis is unusual in the west, it is still prevalent in Asia and Africa. We report a case in which the diagnosis was made histologically without evidence of symptoms of systemic tuberculosis. The lesion, located in the conus medullaris, mimicked a conus tumor. The patient was a 20-year-old man who presented with a history of progressive leg weakness, urinary urgency, and impotence. There was no history of, or recent contact with, tuberculosis. A diagnosis of an intramedullary tumor in the conus medullaris was made by MRI. The patient underwent a T11-L1 laminectomy and total resection of the lesion with microsurgical technique. Histologic examination revealed a granulomatous lesion containing Langhans' giant cells, inflammatory cells, and caseating necrosis. Antituberculous medication was instituted as soon as the diagnosis was made. Neurologic symptoms and signs slowly improved postoperatively. A combination of microsurgical resection and antituberculous chemotherapy should be the choice of treatment for intramedullary tuberculomas.

Key words Intramedullary spinal tuberculoma · MRI · Spinal cord · Tuberculoma · Tuberculosis

Introduction

Tuberculosis of the central nervous system (CNS) is unusual in the west; however, it continues to be a common problem in third world countries [4, 5, 7, 22]. The most common form of spinal intradural tuberculosis is meningitis. The occurrence of intramedullary tuberculoma is rare. Lin et al., in 1960, reviewed 105 cases of intramedullary spinal tuberculomas reported in the literature from the time of the first case report by Aberrombie in 1828 [13, 15]. In 17 of these cases the diagnosis was made surgically. A total of 148 cases of intramedullary tuberculomas were reported in 1990 by MacDonell et al. [15]. Only a few more cases have been reported since that date. We report a case in which intramedullary tuberculoma of the conus medullaris mimicked a conus tumor.

Case report

A previously healthy 20-year-old man was admitted with a history of low back pain, progressive weakness, and paresthesia in the legs. He reported urinary urgency, impotence, and difficulty with walking in the previous 10 days. There was no history of, or recent contact with, tuberculosis.

General systemic examination, including clinical examination of the chest, revealed no abnormalities. Motor testing revealed complete loss of dorsiflexion and severe weakness of plantar flexion on the right and mild weakness of plantar flexion on the left. Sensory testing revealed hypoesthesia in the sacral dermatomas, ascending to the L4 dermatoma on the right. Ankle jerks were depressed and both plantar responses were extensor.

Laboratory studies revealed a normal complete blood count. The erythrocyte sedimentation rate was 18 mm in the 1st h. A chest plain radiograph showed left apical scarring. Roentgenograms of the spine were normal. MRI revealed an intramedullary hyperintense lesion and enlargement of the spinal cord between the T11 and L1 levels on T2-weighted images. Gd-

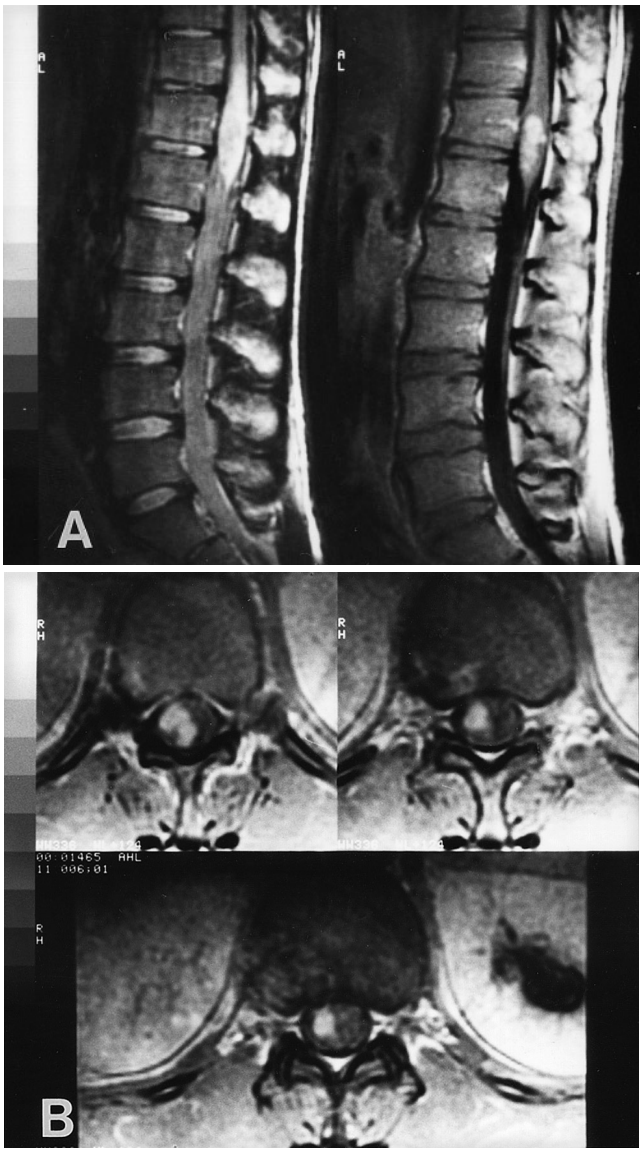

Fig. 1 **A** Sagittal images revealing hyperintense enlargement of the spinal cord between the T11 and L1 levels (*left*) and enhancement of the intramedullary lesion within the expanded cord after administration of Gd-DTPA (*right*). **B** Axial images with contrast enhancement showing enhanced lesion located within the right side of the spinal cord at the level of T12

DTPA-enhanced MRI showed enhancement of the intramedullary lesion within the expanded spinal cord (Fig. 1).

The patient was operated on without delay, with the diagnosis of an intramedullary mass in the conus medullaris. A T11-L1 laminectomy was performed and the dura was opened. There was no evidence of an extramedullary lesion. The conus was found to be swollen. Posterior longitudinal myelotomy was executed and a well-circumscribed grayish mass was totally excised using the operating microscope. Histologic examination of the specimen revealed a granulomatous lesion containing multinucleated Langhans' giant cells, inflammatory cells, and caseating necrosis (Fig. 2). Tissue stain for acid-fast bacilli was negative and tissue cultures were sterile.

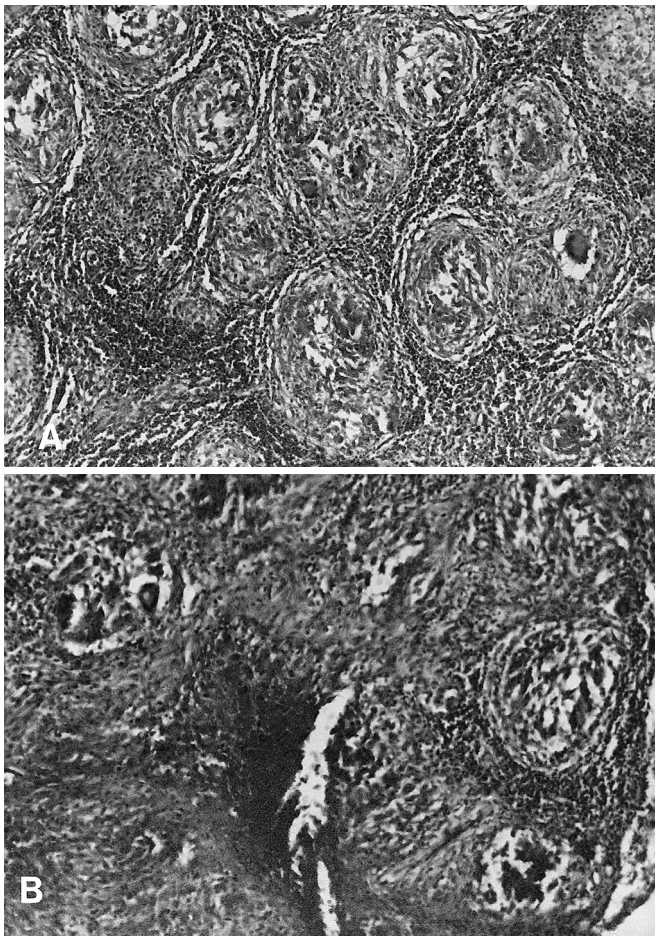

Fig. 2 **A** Photomicrograph showing granulomas with Langhans' giant cells surrounded by inflammatory cells (H & E × 100). **B** Central caseating necrosis and chronic inflammatory cells are seen (H & E × 100)

Other laboratory studies were performed to confirm the diagnosis of tuberculosis. Tuberculin skin test, with 100 units of purified protein derivative, was positive. Sputum and cerebrospinal fluid (CSF) acid-fast bacilli stainings were negative. CSF and sputum cultures were sterile.

Postoperatively, the patient's paraparesis slowly improved. Quadruple therapy with ethambutol, rifampin, isoniazid, and pyrazinamide was started. After 8 weeks his impotence had disappeared and the bladder disturbances were nearly resolved. At follow-up 4 months later, he was able to walk unsupported without any sexual or urinary dysfunction.

Discussion

CNS tuberculosis occurs as a result of hematogenous spread from a primary focus. Only 0.5–2% of patients with systemic tuberculosis has CNS involvement [1, 3, 17]. Meningitis, Pott's disease, and intracranial tuberculomas are the common forms of CNS tuberculosis. The most common form of spinal tuberculosis is tuberculous

spondylitis (Pott's disease). Meningitis is the most commen form of spinal intradural tuberculosis. Tuberculomas are mainly located intracranially. Spinal tuberculomas comprise 2–5% of the all CNS tuberculomas [1, 13, 14]. Arseni and Samitca [1] found 210 cases of CNS tuberculoma, of which only 9 were spinal masses. Intramedullary location was found in only five of these spinal tuberculomas. The rarity of intramedullary spinal tuberculomas was also emphasized by Citow and Ammirati in 1994 [5]. They mentioned that intramedullary tuberculomas were seen in 2 of 100,000 cases of tuberculosis and 2 of 1000 cases of CNS tuberculosis disease. The ratio of intramedullary spinal to intracranial tuberculomas was found to be 1:42 in the major reported series [15]. This ratio parallels the ratio of spinal cord to brain weight. Local factors such as blood flow may explain the infrequency of intramedullary spinal tuberculoma.

Lin [13] compiled 105 cases of intramedullary tuberculomas in 1960. In 17 of these cases the diagnosis was made surgically but 88 cases were discovered at postmortem examinations. MacDonell et al. [15] reviewed the literature and reported an additional 43 cases. Thus, they found a total of 148 intramedullary tuberculoma cases in 1990. Most of these cases were reported from the developing countries. There have been only two reports of intramedullary tuberculoma in the North American literature since Lin's review in 1960. Rhoton et al. [19] reported a case in 1988 and Citow and Ammirati [5] reported the second in 1994. Although spinal tuberculosis is unusual in the west, it is still prevalent in the developing countries.

Infection with HIV increases the risk of tuberculosis [2, 8, 21, 23]. The first bacteriologically proven case of an intramedullary tuberculoma in a patient with HIV infection was reported by Gallant et al. in 1992 [6].

Fever, weight loss, night sweats and cough may occur with the symptoms of spinal cord compression. Neurologic complaints and signs of intramedullary tuberculomas depend on the location of the lesion.

Chest radiographs, tuberculin skin testing, erythrocyte sedimentation rate, white blood cell count, protein and glucose level examination of the CSF are the supportive laboratory studies. Acid-fast stains and cultures of the CSF confirm the diagnosis of tuberculosis.

Myelography and CT reveal the intramedullary lesion and an expanded spinal cord. MRI is the best diagnostic tool to show the location, size and number of the lesions [9, 10, 12, 19]. Jena et al. [11], in 1991, demonstrated the MRI characteristics of intramedullary tuberculomas as low intensity rings with or without central hyperintensity (because of the varying amount of caseous necrosis) on T2-weighted images and low to isointense rings on T1-weighted images. Rhoton et al. [19], in 1988, presented the first known description of MRI of intramedullary spinal cord tuberculomas. They mentioned that hypointensity of the nodules were due to paucity of mobile protons within the fibrotic lesion. Kioumehr et al. [12] reviewed the MRI findings of CNS tuberculosis and commented that different appearances of tuberculomas on T2-weighted images reflected different stages of evolution of the lesion. Gupta et al. [9] studied 20 patients with intraspinal tuberculosis (8 of them had intramedullary tuberculomas) and found that the lesions were iso- or hypointense on T1-weighted images, iso- or hyperintense on T2-weighted images, and showed rim or nodular enhancement with contrast medium.

Surgical intervention is necessary to confirm the diagnosis and extirpate the intramedullary lesion. Histologic examination of the specimen reveals a granulomatous lesion containing Langhans' giant cells, caseating necrosis, and inflammatory cells. It is not always possible to demonstrate acid-fast bacilli in the specimens [16, 20]. Tissue culture for tuberculosis is important, but sometimes these cultures remain sterile. In MacDonell's review, in 5 of the 12 surgical specimens acid-fast bacilli were recognized, and only 1 specimen was culture positive.

In addition to surgical treatment, chemotherapy with antituberculous drugs should be instituted as soon as the diagnosis is made to achieve the best neurologic outcome. Drugs that penetrate the CNS (isoniazid, rifampin, ethambutol, and pyrazinamide) should be used. Courses of antituberculous medications shorter than 6 months may cause recurrence [18].

In conclusion, intramedullary tuberculoma, although a rare neurosurgical disorder, should be considered in the differential diagnosis of spinal cord compression in patients with evidence of symptoms of extramural tuberculosis, an immunocompromized state, or recent contact with tuberculosis. MRI is the best diagnostic tool to determine the location, number, and size of the lesions. Optimum neurologic outcome is possible with microsurgical technique and postoperative administration of antituberculous chemotherapy.

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