ABSTRACT

Introduction: Intramedullary spinal tuberculoma (IMT) combined with abscess is rare. Because of such rarity, there is no standardized treatment protocol for this condition. We present a case of intramedullary tuberculoma combined with abscess in a female child who was successfully treated with surgery and antituberculosis medicine.

Case report: We present a case of spinal intramedullary tuberculoma in a 2-year-old female child who was receiving antituberculous treatment for tuberculous meningitis presented with weakness in both lower limbs. Magnetic resonance imaging (MRI) lumbar spine showed heterogeneously enhancing intramedullary lesion in the lumbosacral region which was hypointense on T1-weighted images and hyperintense on T2-weighted imaging. Lesion was explored in view of neurological deficit. Pus containing solid lesion was removed. Histopathology confirmed intramedullary tuberculoma with abscess.

Conclusion: Intramedullary tuberculoma with abscess is a rare cause of paraparesis. In spite of antituberculous treatment, patients are susceptible for development of intramedullary tuberculoma with abscess. One has to keep this pathology in mind when patients present with intramedullary space occupying lesions with tuberculous lesions elsewhere.

Keywords: Abscess, Spinal cord, Tuberculoma.

INTRODUCTION

Intradural spinal tuberculomas comprise about 2 to 5% of spinal tuberculosis, while IMTs are still rare. Intramedullary tuberculosis is almost always secondary to pulmonary tuberculosis, but some cases may present only with isolated extrapulmonary forms. Concurrent occurrence of intracranial tuberculomas along with IMT, though possible, is rare. Intramedullary spinal tuberculoma combined with intramedullary spinal abscess, which has lower incidence could easily be misdiagnosed. We present a case of IMT combined with pyogenic abscess who was on antituberculous treatment for tuberculous meningitis and review the related literature.

CASE REPORT

A 2-year-old female child presented with history of weakness in both lower limbs for 2 weeks with urinary retention. Earlier, the child had an episode of fever, headache, and vomiting. Her lumbar cerebrospinal fluid study suggested the evidence of tuberculous meningitis. Her computed tomography (CT) brain was performed which was suggestive of tuberculous meningitis with mild hydrocephalus. The child was treated with four-drug antituberculous treatment along with steroids. Four months later, the child developed progressive weakness in both lower limbs that was noticed by the mother as the child needed support for walking and standing. Neurological examination revealed hypotonia in both lower limbs with power grade 3/5 with exaggerated lower limb reflexes. Magnetic resonance imaging of dorsolumbar spine showed peripheral enhancing mass lesion in the spinal canal in the intradural space involving conus, cauda equina nerve roots, and filum terminale extending from upper end of L1 body up to S3 vertebral body for a length of 10.5 cm with a maximum thickness being 1.4 cm in the region of L1 body which was heterogeneously hyperintense on T2-weighted images and hypointense on T1-weighted images (Figs 1 and 2). These features were suggestive of infective etiology. Magnetic resonance imaging brain revealed mild dilatation of bilateral lateral, third, and fourth ventricle with minimal periventricular ooze. In view of significant neurological deficit, the child underwent lumbosacral laminotomy. Spinal cord was full and edematous; nerve roots were weakly adherent to the lesions. Lower end of the lesion was entered and gush of brownish-colored pus drained out. Grayish yellow firm almost avascular solid lesion was excised by microsurgical dissection (Fig. 3). Almost complete excision of the lesion was achieved. Pus was positive for Gram stain but negative for Ziehl–Neelsen
stain. Culture was suggestive of *Escherichia coli*, that grew after 24 hours of incubation, with a colony count >1,00,000 colony-forming units/mL. Histopathological examination of the specimen revealed tuberculous etiology (Fig. 4). The child received standard antituberculous drug in addition to antibiotics for Gram-negative *E. coli*. Postoperative course was uneventful. Power in lower limb improved over a period of 3 to 4 weeks. At 3 months follow-up, the child was able to walk without support with good urinary continence.

**DISCUSSION**

Spinal intramedullary tuberculoma was first reported by Dibble and Cascino. Compared with intracranial disease, intramedullary occurrence is rare and mainly involves the thoracic cord. It is unknown whether the disease
specifically targets a particular sex or age. Spinal intramedullary tuberculoma can occur following hematogenous dissemination, cerebrospinal fluid infection, and very rarely caused by local spreading of spinal tuberculosis. In our case, already existing intracranial tuberculosis in the form of tuberculous meningitis led to intraspinal tuberculoma. Spinal intramedullary tuberculous abscess is still rare.7

Though chest radiographs and CT scanning are effective measures for the detection of pulmonary tuberculosis and extrapulmonary tuberculosis, MRI is the imaging modality of choice in the diagnosis of spinal intramedullary tuberculosis.8 Intramedullary tuberculoma appears hypointense on T1-weighted images and hyperintense on T2-weighted images. Lesion enhances on contrast administration. However, MRI findings may vary.9

Intramedullary tuberculoma should be differentiated from other contrast-enhancing intramedullary mass lesions like ependymoma, astrocytoma, hemangioblastoma, and metastatic tumors.9-11 No consensus has been made on the standard therapy of intramedullary tuberculoma combined with abscess. Published literatures suggest that most patients recovered well after antituberculosis therapy. Specific antituberculous chemotherapy is the primary modality of management. Though the response to antituberculous chemotherapy is usually good, paradoxical increase in the size of the lesion with treatment may occur necessitating surgical intervention. Surgery in the treatment of spinal intramedullary tuberculomas may be indicated in the presence of significant neurological deficit, no response to antituberculous medicine, or whenever there is diagnostic dilemma.12,13

CONCLUSION

Intramedullary tuberculoma with abscess is a rare cause of paraparesis. In spite of antituberculous treatment, patients are susceptible for the development of intramedullary tuberculoma with abscess. One has to keep this pathology in mind when patients present with intramedullary space-occupying lesions with tuberculous lesions elsewhere. Most patients respond well to the antituberculous therapy, while for patients with severe spinal cord compression or who are not responsive to drug therapy, surgical intervention could facilitate neurologic recovery and improve the prognosis.

REFERENCES