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CASE REPORT

Spinal Intramedullary Tubercular Abscess

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ABSTRACT

Introduction: Spinal intramedullary tubercular abscess is an extremely rare entity. Only few cases are reported in published literature.

Case report: A 30-year-old female with known case of pulmonary tuberculosis taking antitubercular treatment (ATT) for the last 6 months presented with back pain and progressive paraparesis. Her magnetic resonance imaging (MRI) dorsolumbar spine was suggestive of intramedullary abscess at D3 to D7 vertebrae level; she underwent D2 to D7 laminectomy and evacuation of pus and, subsequently, acid-fast bacilli (AFB) staining and pus culture. *Mycobacterium tuberculosis* was seen in both staining and culture of pus. Patient had shown signs of improvement and was discharged on 7th postoperative day.

Conclusion: In tuberculosis abscess, if the patient does not respond to medical management, and if progressive neurological deficit is present, surgery should be done for a definite histological diagnosis and decompression.

Keywords: Intramedullary tuberculosis, Spinal cord tuberculosis, Tubercular abscess.

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INTRODUCTION

Intramedullary spinal tubercular abscess is a very rare entity among various forms of intramedullary spinal abscess, since very few cases have been reported worldwide now.[1] The first case of spinal intramedullary abscess was reported by Hart.[2] Various organisms have been isolated but *M. tuberculosis* was found in five cases only.[1,3-6] We are reporting about a 30-year-old female with progressive paraparesis secondary to intramedullary dorsal spine abscess and the definitive diagnosis of tubercular abscess was finally based on histopathological and pus culture examination.

CASE REPORT

A 30-year-year old female patient presented with pain in the upper part of back for last 1 month, with gradual progressive weakness of both lower limbs for the last 7 days and urinary and bowel incontinence for the last 3 days. The patient is a known case of pulmonary tuberculosis with tubercular meningitis and is taking ATT for the last 6 months. In general physical examination, there was generalized weakness and her body weight was 42 kg. Neurological examination revealed power grade 1/5 in both lower limbs and she had 40 to 50% sensory loss for all modalities below the D4 level on both sides, plantar bilateral extensor. There was no spinal deformity or tenderness. Both upper limbs were normal and there were no signs of meningeal irritation, and fever was absent. Haemogram was within normal limits while chest X-ray showed lobulated homogeneous opacity rt. lung field, suggesting pulmonary Koch's (Fig. 1). Contrast MRI dorsolumbar spine revealed ring enhancing lesion, extending from the D3 to D7 vertebra level with obliteration of cerebrospinal (CSF) space (Figs 2 and 3A) and the lesion appeared slightly hyperintense to cord on

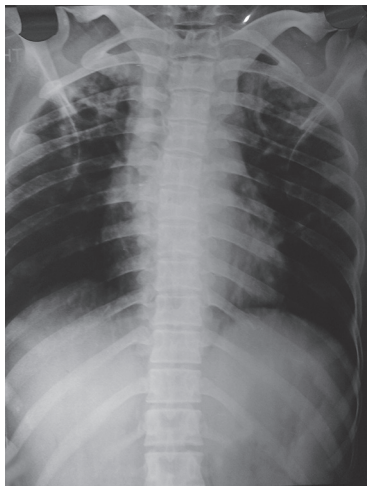

Fig. 1: Chest X-ray posteroanterior view showing tubercular cavity in the right upper lobe of lung

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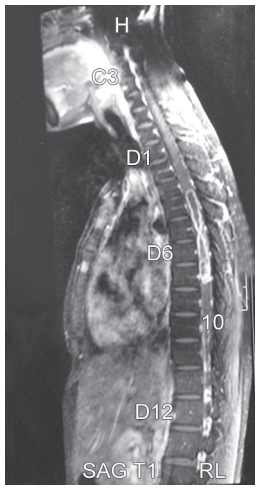


Fig. 2: T1-weighted contrast MRI sagittal section showing the ring enhancing intramedullary lesion

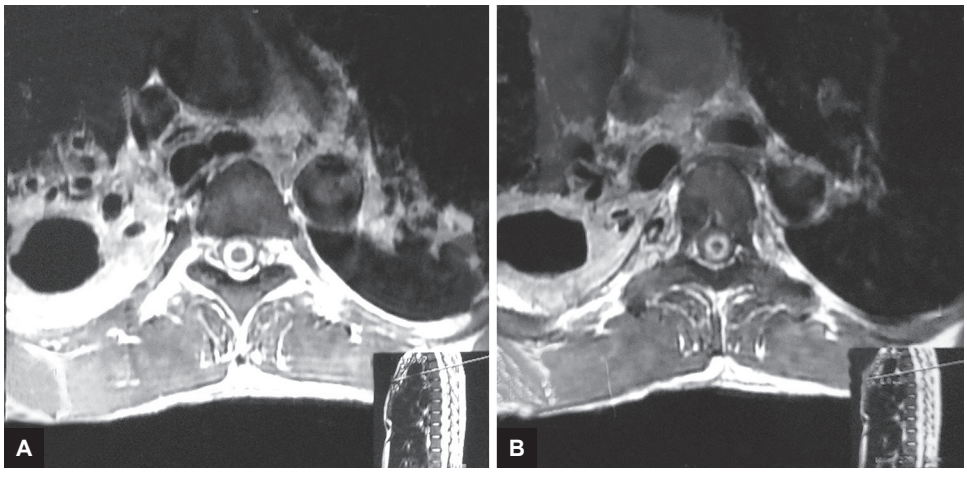
T2-weighted and short tau inversion recovery images, while hypointense on T1-weighted image She underwent posterior thoracic laminectomy extending from D3 to D7 vertebral level. Preoperatively, the epidural space was normal with increased vascularity over the dura and cord with loss of epidural fat. Dura was opened and midline myelotomy was done, frank thin creamish yellow-colored pus came out, and no caseation or granulation tissue was seen. After complete evacuation of pus, the cord became decompressed and the CSF circulation was established. Pus was sent for Gram staining, AFB staining, culture, and sensitivity for both pyogenic and tubercular infection which revealed *M. tuberculosis* on both staining and culture examination (Fig. 3B). She was continued on ATT (H,R). Immediate postoperative period, the patient was the same as previously, but after the seventh postoperative day, the patient was showing signs of improvement. After 2 months of follow-up, her urinary incontinence subsided and her power improved to 2/5 in both lower limbs.

DISCUSSION

Tuberculosis is endemic in developing countries like India.[7] With increasing incidence of human immunodeficiency virus worldwide, the incidence of tuberculosis is also on the rise.[7,8] The central nervous system is affected in about 10% of patients with tuberculosis.[7] Pachymeningitis, leptomeningitis, tuberculomas, and abscesses are the various pathological presentations of spinal cord tuberculosis.[9] The commonest form of central nervous system tuberculosis is meningitis, while tuberculoma is unusual.[1] The spinal cord involvement is extremely rare and the ratio reported of intramedullary spinal to intracranial tuberculoma is 1:42.[10-14] Intramedullary tuberculosis is almost always secondary to pulmonary tuberculosis with a rare exception as an extrapulmonary form.[10,15] Tubercular abscess in intramedullary region is much rarer; only less than 11 cases have been reported in the literature.[1,3-6,15-20] Since the first case of intramedullary abscess was documented by Hart, *M. tuberculosis* has been demonstrated in only five cases.[1,3-6] The spinal intramedullary tubercular abscess is defined as an encapsulated collection of pus, containing tubercular bacilli without evidence of tubercular granulomatous reaction.[21,22] The spinal intramedullary tubercular abscess may be diagnosed by the presence of ACB within the tissue or by a positive culture. Whitner's[23] criteria to define tubercular abscess are:

- Evidence of true abscess formation, as confirmed by surgery or autopsy
- Histological proof of presence of inflammatory cells in the abscess wall and absence of granuloma
- Demonstration of AFB in pus or abscess wall

Tubercular abscess should be differentiated from cystic tuberculoma and in the latter, pus cyst containing yellowish fluid and cyst wall has typical tuberculous



Figs 3A and B: T1-weighted contrast MRI axial section showing the intramedullary lesion at the D4-D5 level

pathology.[22,24] Constitutional symptoms like fever, weight loss may not be conspicuous in central nervous system tuberculosis[25] as are serological markers.[16] Diagnosis is made only after the tubercular colonies are grown on culture media and this can cause a considerable delay in initiating antitubercular chemotherapy.[26] In our case, *M. tuberculosis* was seen on AFB staining as well as grown on culture media. The MRI is the best and most sensitive investigation to detect mass lesions, cord changes, and defining its extent. The MRI findings are indistinguishable from intramedullary tuberculoma in many cases;[27] as in the present case, majority of the patients present with signs and symptoms of spinal cord compression with minimal symptoms of tubercular toxemia, and hence, the high index of suspicion is must to diagnose these cases as tubercular abscess preoperatively, and MRI along with other supportive investigations for primary lesion in the chest may be helpful. Biopsy, along with microscopic examination and culture, is essential to confirm the diagnosis. Medical therapy is now curative for intramedullary tuberculosis.[28,29] Presently, specific antitubercular chemotherapy is the primary modality of management,[30] although the previous authors advised surgery for all symptomatic cases.[10,14] Though the response to antituberculous chemotherapy is usually good, paradoxical increase in the size of the lesion with treatment may occur, necessitating surgical intervention.[31] Surgery is indicated for

- Large lesions with a rapid deterioration of the neurological status
- Nonspecific neuroimaging features
- Paradoxical increase in the size of the lesion following antituberculous therapy

Early diagnosis and prompt management may offer a favorable prognosis, even in cases having severe neurological deficits.[25] As with the present case, the diagnosis was doubtful even after MRI and the patient was having progressive neurological deficit with bladder involvement, and urgent surgical decompression was done to establish the diagnosis as well as to decompress the tumor. Our patient showed signs of improvement after 1 week, probably because of early surgery and decompression with continued antitubercular drugs.

CONCLUSION

In countries where tuberculosis is endemic, if the diagnosis is doubtful or the patient does not respond to medical management, and if progressive neurological deficit is present, surgery should be done for a definite histological diagnosis and decompression.

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