

Intramedullary spinal cord abscess

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Abstract

Background: Intramedullary tubercular abscess is a very rare condition, however it needs to be considered in the differential diagnosis of neurological deterioration in a patient of tuberculosis as timely intervention often gives good neurological recovery.

Case description: The authors report a rare case of intramedullary tuberculous abscess of dorsal spinal cord in a 35-year-old female patient who presented with paraplegia and bladder involvement. Magnetic resonance imaging scans revealed D9-D10 arachnoiditis with intramedullary lesion with extradural compression locally. A dorsal laminectomy with decompression of intramedullary abscess with biopsy of extradural granulation tissue was carried out and patient was started on antituberculous therapy and gradually improved neurologically postoperatively.

Conclusion: Intramedullary tuberculous abscess is a rare condition and surgical intervention is helpful even in the presence of profound neurological deficits. (p89-91)

Key words: Intramedullary, tubercular and spinal cord abscess

Introduction

Intramedullary spinal cord abscess is a rare disease entity. Only 6 cases have been reported to be associated with tuberculosis.^{1,2,5,13,14} We report a seventh case of intramedullary tuberculous spinal cord abscess with histopathological confirmation and review the literature of tuberculous intramedullary spinal cord abscess.

Case Report

A 35-year-old female patient was referred to our hospital with a 6-month history of low backache and numbness of both lower limbs and progressively increasing paraparesis with history of urinary incontinence for 4 months. Patient was bedridden and paraplegic for 1 month and was on indwelling urinary catheter. She also had low-grade fever

of 5 months duration. She was diagnosed as a case of Pott's spine on the basis of positive CSF PCR by a physician and was started on 4-drug (RHEZ) antituberculous treatment 6 months prior. She also had history of tuberculous meningitis prior to being started on antituberculous treatment. Despite this, she continued to deteriorate and became gradually paraplegic. On examination, she had paraplegia (power 0/5 both lower limbs MRC grade) and 50 - 60% sensory loss to all modalities of sensations below D4 dermatomal segment. There was marked spasticity of both lower limbs and deep tendon jerks were brisk with bilateral extensor plantars. She had no bladder sensations.

Laboratory investigations demonstrated haemoglobin of 12.6 gm% and ESR of 26 mm. She was seronegative for HIV. Chest x-ray was within normal limits. Magnetic resonance imaging (MRI) of spine revealed expansion of entire dorsal spinal cord. Enhancing soft tissue was seen intradurally at D9-D10 level posterolaterally on left side displacing the cord anteriorly and to the right. An area of ring enhancement was also noted within thecal sac adjacent to the compressed cord (Figs. 1 and 2). Dorsal cord showed gray matter hyper-intensity, on T2 image, suggestive of myelopathy. Yellow marrow changes at L4 and L5 level suggestive of healed infection were seen. Only a small area of marrow oedema/active caries was seen at L3 level. Areas of osseous destruction /altered signal were seen in the posterior bodies and both halves of the sacrum at S1 to S3

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level. A minimal effusion was seen in right sacroiliac joint. Multiple granulomas were seen intracranially in midbrain, left occipital lobe and in right cerebellar hemisphere extending into peduncle locally (Fig. 3). Radiological findings were suggestive of D9-D10 level arachnoiditis with intrathecal granuloma, tuberculous spondylitis with epidural spread L5-S3 level suggestive of healed tuberculosis with intracranial healed tuberculosis.

In view of profound neurological deficit and failure to respond to antituberculous therapy, patient was scheduled for surgery and D8-D10 laminectomy was done. The laminae were normal. There was no extradural collection or granulation tissue. After opening the dura the spinal cord was seen to be bulging out and a yellowish granulomatous lesion was seen on the surface. Biopsy of subpial caseous granulation tissue was carried out and as soon as it was opened, approximately 5 ml of thick non-foul smelling yellowish pus was drained. Injection methylprednisolone

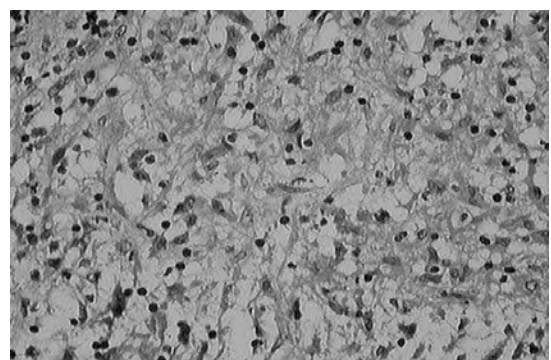
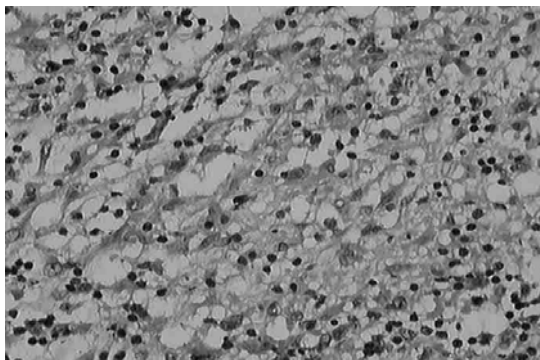
was started intraoperatively and continued postoperatively for 24 hours. Culture studies of pus obtained were negative for aerobic, anaerobic bacteria and for fungi. Histopathology of the lesion showed chronic granulation tissue suggestive of tuberculosis characterised by epithelioid cell granulomatous reaction composed of irregularly dispersed epithelioid cells surrounded by a cuff of lymphocytes and central area of hyaline necrosis (Figs. 4 and 5). Pus culture of intramedullary abscess obtained intraoperatively was sterile. Stain for acid fast bacilli were positive.

Patient's neurological status was unchanged postoperatively at the time of discharge except for minimal toe movement and some return of bladder sensation. Patient received anti-tuberculous treatment for 18 months. At 48 months follow-up, patient improved neurologically significantly (power in both hip/knee - 5/5 and foot dorsiflexors/ plantiflexors and external hallucis longus being 4/5 MRC grade respectively) and has regained bladder sensations.



Figures 1 ↑ and 2 → T1 axial and sagittal MRI (post gadolinium) showing enhancing intramedullary lesion with hypointense centre (? abscess/ ? necrosis) at D9-D10 cord level.

Figure 3 ↑ MRI showing intracranial tuberculoma in the right cerebellar hemisphere extending into cerebellar peduncle.



Figures 4 and 5 - Photomicrograph showing epithelioid cell granulomatous reaction composed of irregularly dispersed epithelioid cells surrounded by a cuff of lymphocytes (right) and central area of caseous necrosis (left) (H&E x 200).

Discussion

Intraspinal cord abscesses are very rare.² Only 83 cases of intramedullary spinal cord abscesses have been reported since Hart's original description of such an entity in 1830.^{1-5,8} Intradural spinal tuberculomas comprise about 2 - 5% of spinal tuberculosis, while intramedullary spinal tuberculomas are still rare.^{7,11} The incidence quoted being 2 in 100,000 cases of all tuberculosis.¹⁰ Spinal intramedullary tuberculous abscess is still rare even in geographical areas where tuberculosis is endemic and only 6 cases of intramedullary spinal cord abscesses caused by mycobacterium tuberculosis have been reported in the literature.^{3,4,9,12,13}

Spinal tuberculosis is known to be associated mainly with pulmonary tuberculosis and may originate in three ways: 1) by haematogenous spread from an origin outside the CNS, 2) via secondary extension caudally from cranial tuberculous meningoencephalitis and 3) by secondary intraspinal extension from osteoarticular discal tuberculosis. In the present case, there was no evidence of pulmonary tuberculosis but co-existent CNS tuberculomas with osteoarticular tuberculosis were observed. In the early stages of spinal tuberculosis, variable degrees of congestion and inflammatory exudates may be demonstrated in the meninges of the cord. The spinal cord and the nerve roots may become oedematous and surrounded by gelatinous exudates similar to our intraoperative findings. Abscess formation becomes manifest by the accumulation of necrotic tissue, debris and caseous material as the disease progresses. In the present case, intramedullary tubercular abscess developed despite patient receiving appropriate and adequate antituberculous treatment for over 6 months. Suppurative intramedullary processes, although reportedly rare are likely to be increasingly encountered because of a rise in drug resistant organisms, the resurgence of tuberculous and other opportunistic pathogens, and an increased prevalence of immunodeficiency disorders. The delay in diagnosis, presence of cord changes at the time of presentation and poor neurological status at presentation contributed to poor neurological outcome in our case. The present case highlights the importance of early diagnosis of such lesions by early MRI study and early timely intervention in suspected/diagnosed cases of spinal tuberculosis for better neurological outcome. This case also highlights the fact that surgical intervention is helpful in tuberculosis even in the presence of profound neurological deficits. Most patients usually have pulmonary tuberculosis and recover well with the combination of microsurgical resection and antituberculous chemotherapy. Intramedullary tuberculosis manifesting as Brown-Sequard syndrome has been reported in a patient of systemic lupus erythematosus.¹⁴ Kumar observed improvement in 15 of 19 cases of intraspinal tuberculomas and opined that

prognosis is poor if cord involvement is complete, if there is longer duration of neural complications, late onset cord involvement, neural complications that developed rapidly, if the patient is older, and if his/her general condition is poor.⁶ Focal spinal cord enlargement in a patient with rapidly progressive myelopathy is the usual presentation of an intramedullary abscess and should be considered for urgent neurosurgical intervention. These cases ought to be distinguished from transverse myelitis which progresses much more rapidly and do not induce significant spinal cord enlargement. The present case should serve as a reminder to instantly elevate our index of suspicion regarding this treatable but potentially catastrophic clinical entity.

Conclusion

Intramedullary tuberculosis, though a very rare disease, should be considered as a cause of neurological manifestations in a patient already suffering from tuberculosis. Even in the presence of profound neurological deficits, surgical intervention leads to neurological improvement.

References

1. Babu R, Jafar JJ, Huang PP, Bud Zilovich GN, Ransohoff J: Intramedullary cord abscesses associated with spinal cord ependymoma. *Neurosurg* 1992, 30: 121-124
2. Cheng KM, Ma MW, Chan CM, et al: Tuberculous intramedullary spinal cord abscesses. *Acta Neurochir (Wien)* 1997, 139(12): 1189-90
3. Ford PR: Myelitis and abscess of the spinal cord. *Disease of the Nervous System in Infancy and Adolescence*. Springfield, Charles C Thomas 1961, Vol 5, pp 464-466
4. Hanci M, Sarioglu AC, Uzan M et al: Intramedullary tuberculous abscess: A case report. *Spine* 1996, 15: 766-769
5. Indira Devi B, Chandra S, Mongia S, et al: Spinal intramedullary tuberculoma and abscess: A rare cause of paraparesis. *Neurol India* 2002, 50: 494-496
6. Kumar R: Spinal tuberculosis: with reference to the children of Northern India. *Childs Nerv Syst*. 2005, 21(1): 19-26
7. Lin TH: Intramedullary tuberculoma of the spinal cord. *J Neurosurg* 1960, 17: 497-499
8. Menezes AH, Van Gilder JC, Perret GE: Spinal cord abscess a review. *Surg Neurol* 1977, 8: 461-467
9. Murphy KJ, Brunberg JA, Quint DJ, Kazanjian PH: Spinal Cord infection: myelitis and abscess formation. *Am J Neuroradiol* 1998, 19: 341-348
10. Nussbaum ES, Rockswold GL, Bergman TA, et al: Spinal tuberculosis: A diagnostic and management challenge. *J Neurosurg* 1995, 83: 243-247
11. Rhoton EL, Ballinger WE, Quisling R, et al: Intramedullary spinal tuberculoma. *Neurosurg* 1988, 22: 733-766
12. Tacconi L, Arulampalam T, Johnston FG, et al: Intramedullary spinal cord abscess: Case report. *Neurosurg* 1995, 37(4): 817-819
13. Tanriverdi T, Kizilkilic O, Hanci M, Kaynar MY, Unalan H, Oz B: Atypical intradural spinal tuberculosis: report of three cases. *Spinal Cord*. 2003, 41(7): 403-9
14. Wan-UK Kim, Sang-Heon Lee, Byoung Young Shim, et al: Intramedullary tuberculosis manifested as Brown-Sequard syndrome in a patient with systemic lupus erythematosus. *Lupus* 2000, 19: 147-150