CLINICAL ORTHOPAEDICS AND RELATED RESEARCH
Number 460, pp. 62–66

Treatment of Intraspinal Tuberculoma

Sudhir Kumar, MCh; Anil K. Jain, MS, MAMS; Ish K. Dhammi, MS; and Aditya N. Aggarwal, MS

Neurological manifestations in spinal tuberculosis often occur secondary to vertebral involvement. However, tuberculoma of the spinal cord or extradural granuloma without radiological evidence of vertebral involvement may be responsible for neurological complications. We report 22 patients with intraspinal tuberculoma (19 extradural, three intramedullary) ranging in age from 17 to 70 years. Three patients had a history of paraplegia of acute onset (within 12 hours of the appearance of neural deficit). Only four patients had spinal tenderness, and bone involvement could be seen on plain radiographs in only three patients. Extradural tubercular granuloma without vertebral involvement is uncommon. Of the 15 extradural tuberculoma patients who had a CT scan and/or MRI, only five had no osseous involvement. Laminectomy and surgical decompression was performed in all 19 patients with extradural granuloma. Thirteen patients showed complete neural recovery within 2 years. Of the three patients with intramedullary involvement, one underwent myelotomy and decompression and died within 2 months of surgery. The other two patients were treated nonoperatively with antitubercular therapy and showed complete neural recovery. All patients received antitubercular therapy for a minimum of 1 year. Intraspinal tubercular granuloma should be considered in the differential diagnosis when a case of spinal tumor syndrome is encountered in an endemic zone of tuberculosis.

Level of Evidence: Level IV, therapeutic study. See the Guidelines for Authors for a complete description of levels of evidence.

Neurological manifestations in spinal tuberculosis often occur secondary to vertebral affection. However, neural deficits may also be caused by intraspinal granulomata of neural and perineural tissues (ie, the epidural space, subdural space, meninges, or cord tissue). These are distinctly less common than intracranial tuberculoma. The approximate ratio of intraspinal to intracranial tuberculoma is 1:20, and the ratio between tubercular and nontubercular expanding intraspinal lesion is 1:43.[12]

Despite the advancement of imaging and surgical facilities, intraspinal granuloma is infrequently reported.[14] Our search of the English literature revealed isolated case reports and series of few cases.[1,4,5,7–9,12–16] A tuberculoma of the epidural, intradural, or intramedullary space is liable to be misdiagnosed or mismanaged initially because the lesions are rare and clinicians are unfamiliar with their presentation. Intraspinal tuberculoma present with compressive myelopathy. If these patients are diagnosed early and treated adequately, they are likely to show excellent neural recovery.

We describe the course and outcome of treatment of intraspinal tuberculoma in 22 patients.

MATERIALS AND METHODS

We retrospectively reviewed 22 patients with compressive myelopathy and neural deficits treated for intraspinal tuberculoma based on clinical, radiographic, and operative findings (Table 1). Intraspinal tuberculoma constitutes approximately 1% of all cases of spinal tuberculosis treated in our hospital. The patients ranged in age from 17 to 70 years (mean, 29.6 years). Three patients had a history of paraplegia of acute onset (complete paraplegia within 12 hours of appearance of the first sign of neural deficit), and 19 patients developed paraplegia/paraparesis of gradual onset (over 3 months). One patient had a history of treatment for tubercular pleurisy. Two patients were already on antitubercular treatment for tubercular cervical lymphadenitis and abdominal lymphadenitis (detected on laparotomy) at the time of initial presentation. None of the patients had a history of constitutional symptoms suggestive of tuberculosis. The minimum followup was 2 years in 20 patients (average 2.6 years; range 2–5 years).

On examination, spinal tenderness could be elicited in four patients. One patient had tenderness at L5–S1 despite having upper motor neuron signs. The spinal level could be ascertained clinically in all patients. The dorsal spine was involved in 17 patients, and the lumbar spine was involved in five; none involved the cervical spine.

From the Department of Orthopaedics, University College of Medical Sciences and Guru Teg Bahadur Hospital, Delhi, India.
Each author certifies that he or she has no commercial associations (eg, consultancies, stock ownership, equity interest, patent/licensing arrangements, etc) that might pose a conflict of interest in connection with the submitted article.
Each author certifies that his institution has waived or does not require approval for the reporting of this case and that all investigations were conducted in conformity with ethical principals of research.
Correspondence to: Sudhir Kumar, MCh, Department of Orthopaedics, University College of Medical Sciences, Shahdara, Delhi-110095, India. Phone: 91-11-22586262 (ext. 520, 519); Fax: 91-11-22590495; E-mail: sudhirkumar25@hotmail.com.
DOI: 10.1097/BLO.0b013e318065b73c

TABLE 1. Radiologic Presentation and Posttreatment Results of Patients with Intraspinal Tuberculoma

Patient Age/Gender	Radiograph	Myelogram	CT/MRI	Outcome/ASIA Motor Score
17/M	Radiodense L2–3 fuzzy disc margin, decreased disc space	Not performed	On MRI EDG with L2–3 involvement	Good neural recovery/100
33/M	Involvement of D11–12	Not performed	On MRI EDG with D11–12 involvement	No neural recovery/50
31/M	No vertebral lesion	Block at L1	On CT-myelo No vertebral involvement	No neural recovery/50
27/M	No vertebral lesion	Block at D9	On MRI Intramedullary granuloma	Died 2 months postoperatively
23/F	No vertebral lesion	Block at D10	On CT Destruction of superior end plate D9 and left rib head	Good neural recovery/100
30/M	No vertebral lesion	Block at L4	Not performed	No neural recovery/50
18/M	No vertebral lesion	Block at D6	Not performed	Good neural recovery/100
40/M	No vertebral lesion	Block at L4	Not performed	Good neural recovery/100
40/M	Involvement of L5–S1	Block at L4	Not performed	Good neural recovery/100
25/M	No vertebral lesion	Block at D11	On CT No bony involvement	No neural recovery/50
70/M	No vertebral lesion	Not performed	On MRI EDG with D8–9 involvement	Partial neural recovery/78
40/F	No vertebral lesion	Not performed	On MRI EDG with D8–10 involvement	Good neural recovery/100
24/F	No vertebral lesion	Not performed	On MRI Intramedullary granuloma	Good neural recovery/100
16/M	No vertebral lesion	Not performed	On MRI EDG with D4–10 involvement	Good neural recovery/100
35/M	No vertebral lesion	Not performed	On MRI EDG with D4–10 involvement	Good neural recovery/100
25/F	No vertebral lesion	Not performed	On MRI EDG with D6–8 involvement	Good neural recovery/100
27/F	No vertebral lesion	Not performed	On MRI EDG with D9–10 involvement	No neural recovery/50
20/F	No vertebral lesion	Not performed	On MRI Intramedullary granuloma	Good neural recovery/100
25/M	No vertebral lesion	Not performed	On MRI EDG with D7–8 involvement	Good neural recovery/100
31/M	No vertebral lesion	Not performed	On MRI EDG with no osseous involvement	Good neural recovery/100
35/M	No vertebral lesion	Not performed	On MRI EDG with D8–9 involvement	Good neural recovery/100
20/M	No vertebral lesion	Not performed	On MRI EDG from D9–L3 with vertebral involvement at distant site L5–S1	Started showing neural recovery

EDG = Extradural granuloma

Seventeen of the 22 patients had upper motor neuron paraplegia while five had lower motor neuron presentation. Sixteen patients had Grade IV paraplegia (Tuli's classification, Table 2),[10,17] and six patients had Grade III paraplegia. All patients had ASIA[2] motor scores of 50.

We (SK, AKJ) did not observe any bone involvement in 19 of the 22 patients on plain radiographs (Fig 1A). Early in the series eight patients underwent myelogram, which showed complete extradural block in seven patients (Fig 1B). CT scan was also performed along with myelography in three of these patients. Of these, two patients showed extradural encroachment without vertebral destruction. The lesions were thus labeled extradural extraosseous tubercular granuloma. The third patient had destruction of the superior end plate and the medial end of the rib attached to the vertebra (Fig 1C).

MRI was performed in 15 patients. We observed intramedullary granuloma in three patients. Extradural granuloma with vertebral involvement was found in nine patients and without

TABLE 2. Tuli's Classification of Tubercular Paraplegia/Tetraplegia

Grade	Clinical Features
I	Patient unaware of neural deficit Physician detects plantar extensor and/or ankle clonus
II	Patient aware of deficit but manages to walk with support
III	Patient nonambulatory because of paralysis (in extension) Sensory deficit less than 50%
IV	All features of grade III plus flexor spasms/paralysis in flexion; flaccidity Sensory deficit more than 50% Sphincters involved

osseous involvement in three patients. One MRI revealed extradural granuloma from D9–L3 with a distant focus of vertebral involvement at L5 and S1 (Figs 2A–B).

We performed laminectomy and surgical decompression in all 19 patients with extradural granuloma and in one of the three patients with intramedullary lesion. Destruction of the lamina, which was not evident on plain radiographs, was found intraoperatively in one patient. The dura was covered with a sheet of granulation tissue in all patients with extradural granuloma (Fig 3A). The granuloma was dorsal to the cord in 18 patients and anterior to it in one. The granulation tissue was peeled off as a layer in 15 patients (Fig 3B) and was removed piecemeal in four patients. Most patients had the granuloma spanning two to three vertebral levels. It spanned six vertebral levels in three patients. Histopathology confirmed the diagnosis of tuberculosis in all patients.

Two of the three patients with intramedullary involvement were treated nonoperatively with antitubercular therapy for a minimum of 1 year. The 20 patients (19 with extramedullary and one with intramedullary disease) who underwent surgery had antitubercular therapy for a minimum of 1 year.

RESULTS

Of the 19 patients with extradural granuloma, 13 showed neural recovery within 2 years. They regained an ASIA motor score of 100 but continued to have exaggerated deep tendon reflexes distal to the lesion for up to 2 years. One patient achieved an ASIA motor score of 78 but did not experience complete neural recovery even after 2 years. The patient was, however, able to walk with support. Five of the 19 patients with extradural granuloma had no neural recovery after surgery. Postoperative myelogram showed no block to the dye column in these patients, suggesting adequate surgical decompression. One patient began to show neural recovery after decompression but the followup was less than 1 year.

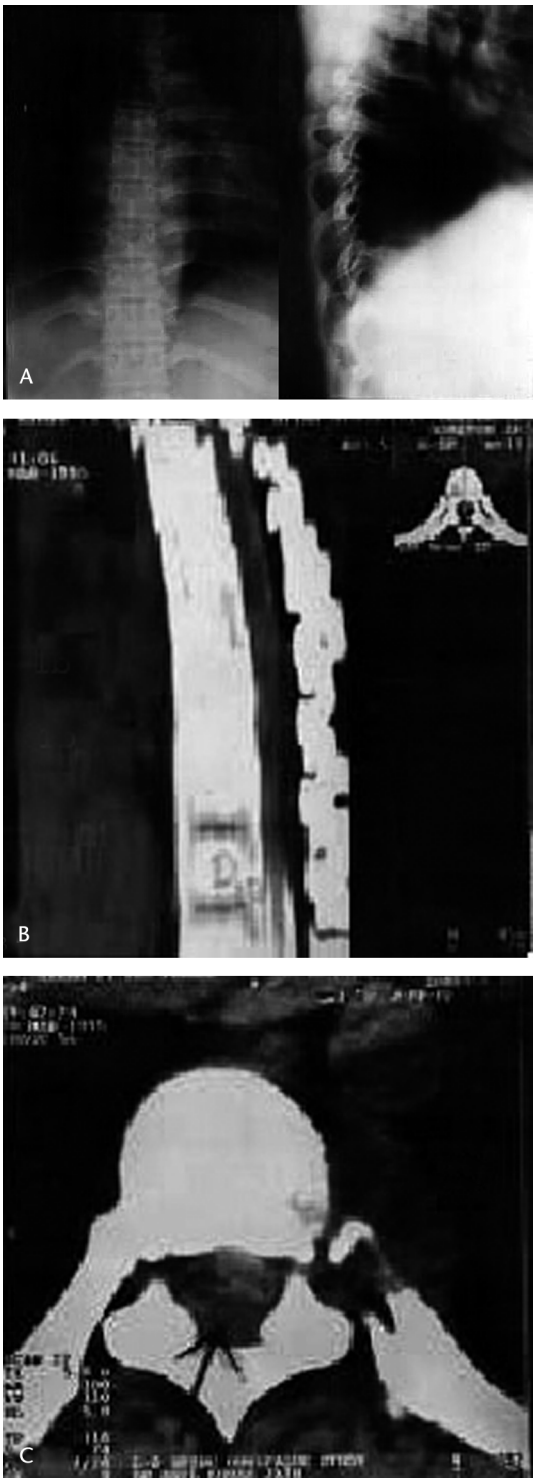

Fig 1A–C. Pictured are plain radiographs (AP and lateral view) of the dorsal spine in a 23-year-old woman presenting as compressive myelopathy with upper motor neuron type of paraplegia (showing normal radiographs). (B) Ascending myelogram shows a block at D10 level. (C) Computerized tomography scan shows the destruction of superior end plate of D9 vertebra with involvement of adjacent left rib head.

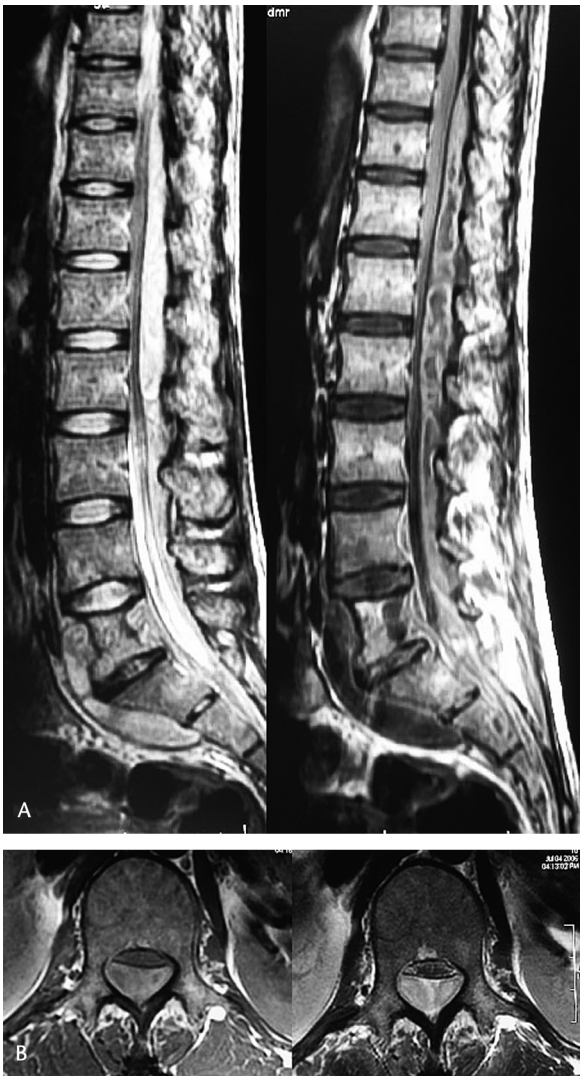

Fig 2A–B. (A) Midsagittal sections of MRI scan (T1 and T2W images) of a 20-year-old man show a long segment of extradural compression extending from the D9 to L3 vertebral level. There is also vertebral involvement at a distant site at L5 and S1. (B) Axial views of the same patient show dorsally placed extradural compression.

Of the three patients with intramedullary involvement, one underwent myelotomy and decompression. He died within 2 months of surgery. The two patients treated nonoperatively with antitubercular therapy started to show neural recovery within 3 weeks of beginning the therapy. Complete neural recovery was noted in 6 months. In one of these patients, a repeat MRI at 6 months followup also confirmed the resolution of the lesion.

DISCUSSION

Granulomatous lesions of the spine presenting as spinal tumor syndrome are variously called extradural tuberculoma, extradural extraosseous granuloma, intradural extramedullary tuberculosis, spinal arachnoiditis, and chronic adhesive arachnoiditis.[16] Because all of these present as compressive myelopathy (spinal tumor syndrome) without obvious radiological lesions, we suggest they be classified together as intraspinal tubercular granuloma.

The study was limited by nonavailability of preoperative MRI and CT scans in four cases initially. The patients who failed to show complete neural recovery could not get followup MRI scans due to financial constraints.

Arseni and Samitca[1] classified intraspinal tubercular granulomata as extradural, subdural, and intramedullary. Dastur's[5] classifications in five groups include all possible types of intraspinal tubercular granuloma: (1) extradural (a) with vertebral body involvement, (b) with vertebral arch involvement, (c) without osseous involvement, (d) without osseous or dural involvement but within the epidural fatty tissue; (2) subdural (a) localized, (b) diffused; (3) subdural and extradural; (4) arachnoiditis without dural involvement; and (5) intramedullary tuberculous granuloma.

Twenty-one of our patients were under the age of 40. The age/gender distribution of our patients is similar to the series of Arseni and Samatica.[1] Past history of tuberculosis could be elicited in three of our patients. Arseni and Samatica[1] reported past history of tubercular affection in seven of nine patients with intraspinal tuberculosis.

Sixteen patients with intraspinal tubercular granuloma had involvement in the dorsal spine indicating a prediction for the thoracic region. However, it may occur at any level. Extradural tubercular granuloma without vertebral in-

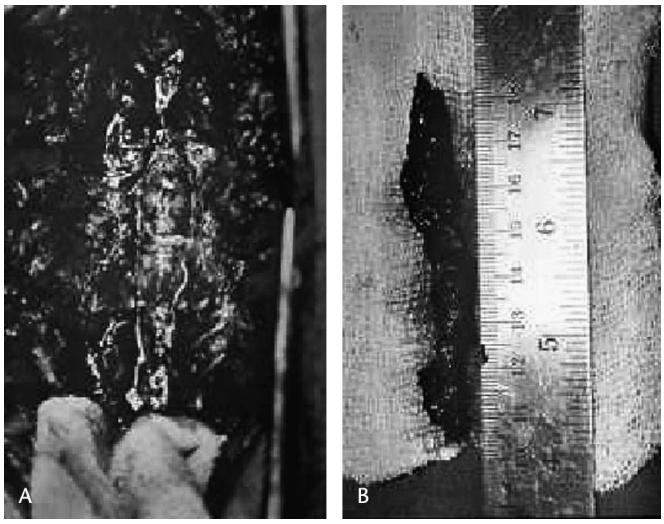

Fig 3A–B. (A) An intraoperative photograph of a patient shows the extradural granuloma compressing the cord. (B) Photograph of the removed specimen.

volvement is uncommon. Of the 15 patients with extradural tuberculoma who had a CT scan or MRI, only five had no osseous involvement. Many patients previously documented in the literature in the pre-CT/MRI era as having purely extradural extraosseous tubercular granuloma might have had a coexisting small osseous lesion not apparent on plain radiographs,[14] and not observed intraoperatively.

Three patients underwent surgery based on myelographic findings only. These patients had no evidence of bony involvement on plain radiographs and could not afford CT or MRI. In one of these patients, we found destruction of the lamina intraoperatively. Therefore, based on this surgical finding, the patient was shifted from Dastur's 1(c) group (extradural granuloma without osseous involvement) to the 1(b) group (extradural granuloma with vertebral arch involvement). It is well documented in the literature that small lesions confined to the laminae or pedicles can be missed on plain radiographs.[9] The presence of a small focus of destruction in the vertebra does not alter the surgical plan and laminectomy can be performed safely without causing instability.[15]

Intradural tuberculosis can occur as a direct extension from the vertebrae, as a downward extension of intracranial tubercular meningitis and, less commonly, as tubercular lesions primarily developing in spinal meninges.[16] Chang et al[3] compared various imaging techniques and concluded conventional myelographic findings were diagnostic of intradural tuberculosis in patients with antecedent or coexisting tubercular meningitis. However, gadolinium-enhanced MRI was considered the diagnostic tool of choice in patients with ongoing inflammation. MRI findings of intramedullary tuberculoma are well documented.[11] We had three patients with intramedullary granuloma. One patient with intramedullary tuberculoma underwent surgery (myelotomy and decompression) and died within 2 months thereafter. Arseni and Samatica[1] also reported disappointing surgical results in intramedullary lesions. Complete dissection of the mass should be avoided to reduce iatrogenic injury and devascularization of the cord.[16] There have been no randomized studies to recommend or refute the use of steroids along with antituberculous therapy in such patients.

The literature suggests intramedullary tuberculoma, if suspected on MRI scan, should be treated with supervised antitubercular chemotherapy with a close check on the neurological status.[6,8,9] Usually the lesion resolves with antituberculous therapy, and the patient starts showing good neural recovery. However, if neural status deteriorates, surgical decompression and myelotomy should be performed to decompress the cord and ascertain the diagnosis.

In patients with extradural granuloma that do not show neurological improvement after adequate surgical decompression, we suspect recovery failure occurred because of vascular thrombosis and end arteritis of radicular arteries causing infarction of the cord and cauda equina. The chances of neural recovery in extradural granuloma are promising after adequate surgical decompression and antitubercular therapy.

Tubercular granuloma should be considered in the differential diagnosis when a case of spinal tumor syndrome is encountered in an endemic zone for tuberculosis. Intraspinal tumors, secondary deposits, multiple sclerosis, and prolapsed intervertebral discs should also be considered.[9] A myelogram, CT myelogram, or MRI is required to determine the level and the possible nature of the lesion. The operative findings in extradural granuloma may be confused with an epidural tumor. The final diagnosis is established by histological examination.

References

1. Arseni C, Samitca DC. Intraspinal tuberculous granuloma. *Brain*. 1960;83:285–295.
2. ASIA. *Standards for neurological classification of spinal injury.* Chicago, IL: American Spinal Injury Association; 1996.
3. Chang KH, Han MH, Choi YW, Kim IO, Han MC, Kim CW. Tuberculous arachnoiditis of the spine findings on myelography, CT, and MR imaging. *AJNR Am J Neuroradiol*. 1989;10: 1255–1262.
4. Compton JS, Compton JS, Nicholas WC. Intradural extramedullary tuberculoma of the cervical spine. *J Neurosurg*. 1984;60:200–203.
5. Dastur HM. A tuberculoma—review with some personal experience. *Neurol India*. 1972;20:127–131.
6. Garg M, Singh S. Intramedullary spinal tuberculoma. *Br J Neurosurg*. 2002;16:75–76.
7. Hamada J, Sato K, Seto H, Ushio Y. Epidural tuberculoma of the spine: a case report. *J Neurosurg*. 1991;28:161–163.
8. Jain AK, Dhammi IK. Non-operative treatment of intramedullary tuberculoma. *Trop Doct*. 2002;32:2–3.
9. Jain AK, Singh S, Sinha S, Dhammi IK, Kumar S. Intraspinal tubercular granuloma—an analysis of 17 cases. *Ind J Orthop*. 2003; 37:182–185.
10. Jain AK, Sinha S. Evaluation of systems of grading of neurological deficits in tuberculosis of spine. *Spinal Cord*. 2005;43:375–380.
11. Jena A, Banerji AK, Tripathi RP, Gulati PK, Jain RK, Khushu S, Sapra ML. Demonstration of intramedullary tuberculomas by magnetic resonance imaging: a report of two cases. *Br J Radiol*. 1991; 64:555–557.
12. Johnston JD, Ashbell TS, Rosomoff HL. Isolated intraspinal extradural tuberculosis. *N Engl J Med*. 1962;266:703–705.
13. Lifeso RM, Weaver P, Harder EH. Tuberculous spondylitis in adults. *J Bone Joint Surg Am*. 1985;67:1405–1413.
14. Mantzoro CS, Brow PD, Dembry L. Extraosseous epidural tuberculoma: a case report and review. *Clin Infect Dis*. 1993;17: 1032–1036.
15. Nussbaum ES, Rockswold GL, Bergman TA, Erickson DL, Seljeskog EL. Spinal tuberculosis: a diagnostic and management challenge. *J Neurosurg*. 1995;83:243–247.
16. Sreeharsha CK, Shetty AP, Rajasekaran S. Intradural spinal tuberculosis in the absence of vertebral or meningeal tuberculosis: a case report. *J Orthop Surg (Hong Kong)*. 2006;14:71–75.
17. Tuli SM. *Tuberculosis of the Skeletal System,* 2nd ed. New Delhi, India: Jaypee Brothers Medical Publishers; 1997.