Neuroradiology (1994) 36: 39–43

Neuro radiology

MRI in intraspinal tuberculosis

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Abstract. We studied 20 patients with intraspinal tuberculosis (TB), to characterise the MRI features of tuberculous meningitis and myelitis. MRI leptomeningitis and intramedullary involvement in 11 patients, intramedullary lesions alone in 5, leptomeningitis alone in 2, and isolated extradural disease in 2. TB leptomeningitis was characterised by loculation of the cerebrospinal fluid (CSF), nerve root thickening and clumping (seen only in the lumbar region) or complete obliteration of the subarachnoid space on unenhanced images. Gd-DTPA-enhanced images proved useful in 6 cases, revealing linear enhancement of the surface of the spinal cord and nerve roots or plaque-like enhancement of the dura-arachnoid mater complex. Intramedullary lesions included tuberculomas (8), cord oedema (5) and cavitation (3). In seven cases of intramedullary tuberculoma multiple lesions with skip areas were seen, without significant cord swelling. One patient had an isolated lesion in the conus medullaris. The lesions were iso- or hypointense on T1-weighted images, iso-, hypo- or hyperintense on T2-weighted images and showed rim or nodular enhancement with contrast medium.

Key words: Spine – Spinal cord – Tuberculosis – MRI

Intraspinal tuberculosis (TB), though rare in the West, is a common cause of morbidity in tropical countries, including India [1–4]; radiculomyelopathy with meningitis is most common [2, 3]. Diagnosis is usually based on the clinical manifestations, cerebrospinal fluid (CSF) findings, typical myelographic appearances and response to specific treatment. Isolation of *Mycobacterium tuberculosis* is only occasionally possible [2, 3, 5–7]. Myelographic features of the different types of spinal tuberculosis have been described [8–9]. Recently, a few reports have concerned MRI [10–12]. We report the MRI features in 20 patients with different types of intraspinal tuberculosis with emphasis on characteristic appearances and the value of gadolinium(Gd)-DTPA injection.

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Materials and methods

MR images of 20 consecutive patients with intraspinal tuberculosis, 17 men and 3 women, 18–52 years old (mean age 28.6 years), were reviewed. The diagnosis was established by pathological proof through open biopsy (2 extradural, 2 intradural, and one intramedullary), coexisting intracranial TB meningitis at the time of diagnosis (2), treated intracranial TB meningitis (3), and response to antituberculous treatment on follow-up images (10). CSF analysis revealed an active inflammatory response (lymphocytosis, elevated protein, decreased glucose) in 14 patients, elevated protein without lymphocytosis in 4 and was normal in 2. No patient had tuberculous spondylitis. Chest radiographs revealed active TB in 2 patients.

Eleven patients had undergone water-soluble myelography. The time between myelography and MRI ranged from 2 days to 4 weeks.

MRI was performed on a 2.0 Tesla superconducting system operating at 1.5 T using on oval surface coil. All images were obtained using multislice, spin-echo (SE) sequences. T1- (500/ 15/3-TR/TE/excitations), proton-density- (2000/20/1) and T2-weighted (2000/80/1) SE images were obtained in the sagittal plane, with slice thickness 3 mm, interslice gap 0.3 mm, and 195 × 256 matrix. Axial T1-weighted images were acquired in 5 patients, using a slice thickness of 5 mm at the appropriate levels. After these sequences were completed, 0.1 mmol/kg body weight of Gd-DTPA was injected intravenously and T1-weighted sagittal (in 16 patients) and axial images (in 5) were obtained. Postinjection studies were not performed in 4 patients.

Results

Eleven patients had both leptomeningeal and intramedullary involvement, 5 had intramedullary disease alone and 2 only leptomeningeal involvement, while 2 had isolated extradural disease.

MRI in the 2 patients with extradural disease revealed a mass in the extradural space, isointense with the spinal cord on T1-weighted images and of mixed high and low signal intensity on T2-weighted images (Fig. 1); contrast-

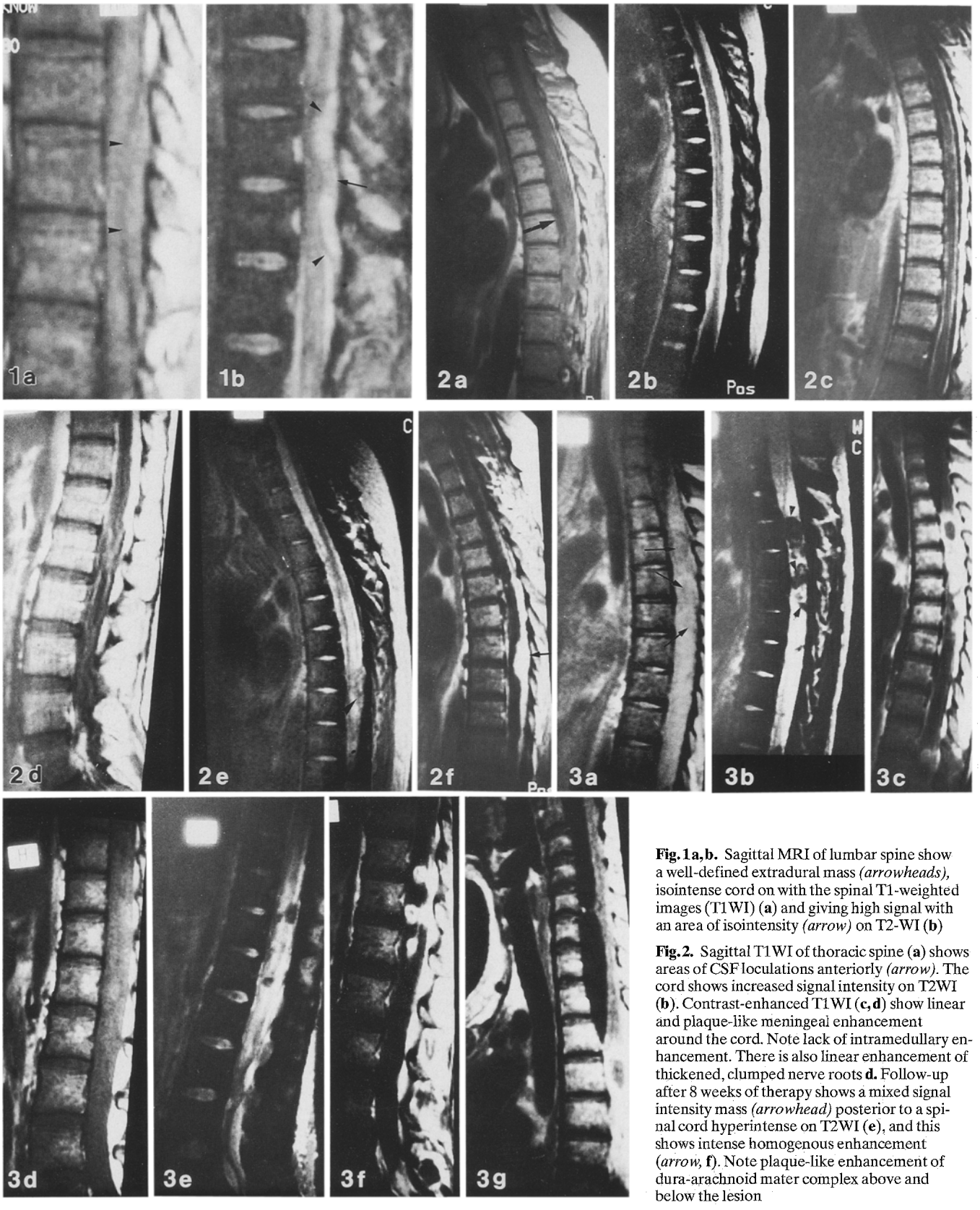

Fig. 1a, b. Sagittal MRI of lumbar spine show a well-defined extradural mass *(arrowheads),* isointense cord on with the spinal T1-weighted images (T1WI) (**a**) and giving high signal with an area of isointensity *(arrow)* on T2-WI (**b**)

Fig. 2. Sagittal T1WI of thoracic spine (**a**) shows areas of CSF loculations anteriorly *(arrow).* The cord shows increased signal intensity on T2WI (**b**). Contrast-enhanced T1WI (**c, d**) show linear and plaque-like meningeal enhancement around the cord. Note lack of intramedullary enhancement. There is also linear enhancement of thickened, clumped nerve roots **d.** Follow-up after 8 weeks of therapy shows a mixed signal intensity mass *(arrowhead)* posterior to a spinal cord hyperintense on T2WI (**e**), and this shows intense homogenous enhancement (*arrow,* **f**). Note plaque-like enhancement of dura-arachnoid mater complex above and below the lesion

Fig. 3. Sagittal SE images of thoracic region show multiple large lesions, giving mildly low signal on T1WI (*arrows,* **a**), low signal on T2WI (*arrowheads,* **b**), and ring and nodular contrast enhancement (**c**). There is loss of distinction between spinal cord, CSF and meninges. Images of lumbar spine demonstrate obliteration of subarachnoid space on T1WI **d,** and low signal lesions on T2WI (**e**), which show thick rim contrast enhancement WI (**f**). Contrastenhanced T1WI of cervical spine (**g**) shows arachnoid loculations displacing the cord posteriorly

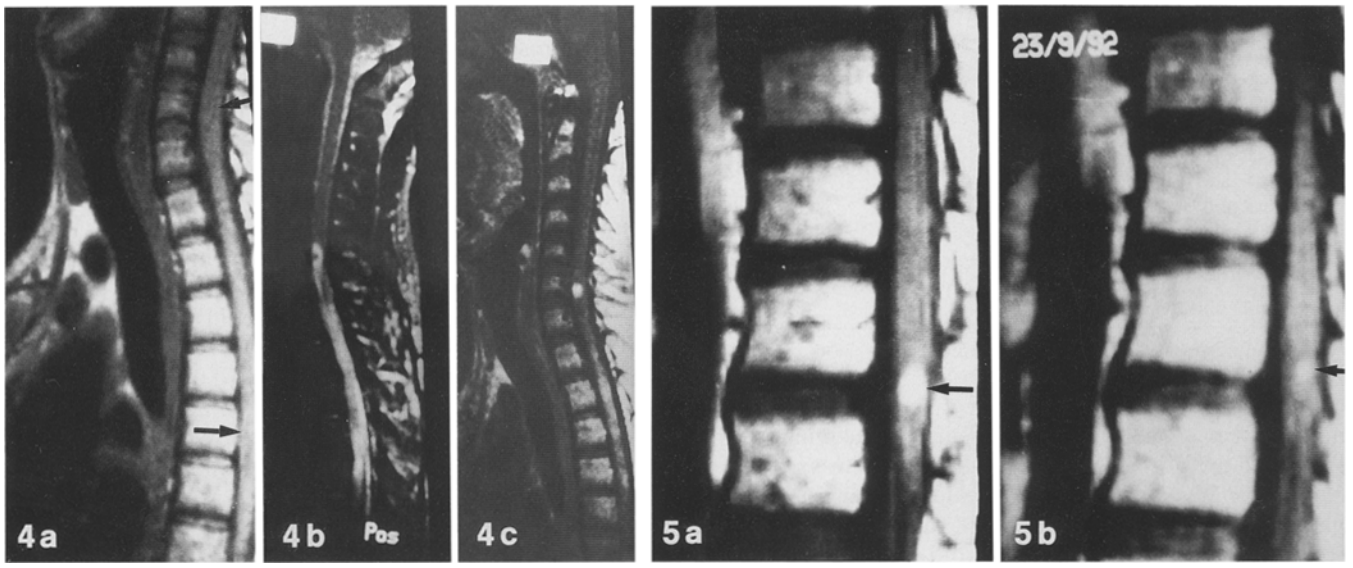

Fig. 4. Sagittal T1WI (**a**) shows two areas of fusiform swelling of the spinal cord *(arrows)* separated by a normal "skip" segment. T2WI (**b**) shows a focal isointense lesion with perifocal high signal oedema in the lower cervical cord; another area of diffuse high hyperintense signal is seen in the upper thoracic cord. Contrast-enhanced T1WI (**c**) shows multiple small areas of enhancement with intervening skip areas

Fig. 5. Contrast-enhanced sagittal T1WI (**a**) shows a nodular enhancing lesion *(arrow)* in the conus medullaris; it gave the same signal as the spinal cord on unenhanced T1- and T2WI. Contrast enhanced T1WI (**b**) following 8 weeks of antituberculous therapy shows marked reduction in the size of the enhancing nodule *(arrow)*

enhanced studies were not performed. Surgical decompression was carried out and biopsy was consistent with tuberculous granuloma.

Table 1 summarises the MRI features in the 16 patients with intramedullary involvement; 3 types of abnormality were observed. Five patients showed diffuse, patchy or continuous areas of altered signal intensity in multiple spinal segments. These gave high signal on T2-weighted images and low or normal signal on T1-weighted images and did not show contrast enhancement (Fig. 2 a–c). Mild swelling of the involved segments was present in 3 patients. All these patients had evidence of associated meningitis. Follow-up MRI in 3 of the 5 showed disappearance of the abnormal signal, together with resolution of the meningitis. Cavitation of the cord, central or eccenteric was identified in 3 patients, associated in every case with leptomeningits.

Intramedullary tuberculomas were seen in 8 patients, 3 of whom had leptomeningitis. They gave low or normal signal on T1-weighted images and high, low or normal signal on T2-weighted images (Fig. 3 and 4). The lesions were multiple in 7 patients. They were associated with perifocal oedema, with or without normal intervening "skip" areas. The tuberculomas showed rim enhancement in 7 patients, with nodular enhancement as well in 5. One patient showed single nodule in the conus medullaris which regressed on specific treatment (Fig. 5). The lesions measured less than 5 mm in all but one patient, who showed multiple large (2–3 cm) lesions involving the entire thoracolumbar cord. Most of the tuberculomas lay at the periphery of the spinal cord, and which was swollen in only 2 patients.

MRI findings in the 13 patients with meningeal disease are summarised in Table 2. Unenhanced MRI suggested the diagnosis of arachnoiditis in 9, seen as clumping of nerve roots (in the lumbar region only) (3), CSF loculations (9) (Fig. 2 a) or complete obliteration of the normal subarachnoid space with loss of the outline of the spinal cord (2). The surface of the cord appeared irregular in 12 patients.

Enhanced images were available for 11 patients. Administration of contrast medium resulted in visualisation of meningeal disease in 4 patients in whom unenhanced images were normal. In 2 patients, contrast enhanced images gave better delineation and localisation of abnormalities seen on the unenhanced images. No enhancement was observed in 5 patients. Two patterns of enhance-

Table 1. MRI findings in 16 patients with intramedullary lesions

	Cord oedema (5)	Tuberculomas (8)	Syringomyelia (3)
Signal intensity			
T1WI			
high signal	–	–	–
isointense	2	3	–
low signal	3	5	3
T2WI			
high signal	5	3	3
isointense	–	3	–
low signal	–	–	–
Contrast enhancement			
none	4[a]	–	2[a]
rim	–	2	–
nodular	–	1	–
rim and nodular	–	5	–
Associated lepto-meningitis	5	3	3

[a] One patient in each group not given contrast medium
T1WI, T2WI, T1- and T2-weighted images

Table 2. Patients with meningeal disease

Location	
Cervical only	1
Cervical and thoracic	1
Cervical, thoracic and lumbar	3
Thoracic only	4
Thoracic and lumbar	1
Lumbar only	1
Cerebrospinal fluid loculation	9
Obliteration of subarachnoid space	2
Adherent nerve roots	3
Meningeal enhancement	6[a]

[a] Two patients did not have contrast medium

ment were noted: thin, smooth linear, enhancement on the surface of the spinal cord and/or nerve roots was seen in all 6 patients (Fig. 2c); 2 showed in addition plaque-like enhancement of the dura-arachnoid mater complex, completely obliterating the subarachnoid space (Fig. 2d, g). In one patient initial studies revealed linear and plaque-like meningeal enhancement (Fig. 2c, d); MRI after 2 months of antituberculous treatment revealed an intradural soft tissue mass (Fig. 2f) which showed intense enhancement (Fig. 2g). The patient's condition improved after surgical excision. Histology was consistent with a tuberculous granuloma.

Myelograms, available for 11 patients, were normal in 5, and showed arachnoiditis in 2, thoracolumbar arachnoiditis in 2 and extradural block in 2. MRI imaging revealed arachnoiditis in 3 and intramedullary tuberculomas in 2 of the 5 patients with normal myelograms. The two with thoracic arachnoiditis also had intramedullary cavities on MRI. In the 2 showing thoracolumbar arachnoiditis MRI showed panspinal involvement, with multiple intramedullary tuberculomas in one. In the remaining 2 patients with extradural myelographic block, an extradural mass demonstrated on MRI was subsequently confirmed to be of tuberculous aetiology.

Discussion

Intraspinal TB can originate in 3 ways: (1) a primary tuberculous lesion, the first expression of TB of the central nervous system, (2) secondary extension downwards of intracranial tuberculous meningitis and (3) secondary extension from vertebral TB [2]. Wadia and Dastur [2] in a series of 70 cases found the first of these to be the most frequent cause of radiculomyelopathy associated with spinal meningitis. Chang et al. [10] recently reported 13 patients with tuberculous radiculomyelitis, of whom 11 had intracranial meningitis and 1 vertebral infection. In our series, the primary variety was seen in 75 % of the patients (15/20), the other 25 % being secondary to intracranial tuberculous meningitis. No patient had vertebral involvement.

Tuberculous leptomeningitis is thought to be frequently associated with involvement of the spinal cord and nerve roots, and has been designated radiculomyelitis [2, 4, 10]. In our series, although most (11/13) patients with leptomeningitis had associated spinal cord changes on MRI, intramedullary TB was seen in only 3, the other 8 showing an intramedullary cavity (in 3) or cord oedema (in 5), presumably secondary to the arachnoiditis. In the report of Gero et al. [11] concerning 8 patients with intradural TB. 5 had tuberculous meningitis without cord involvement while 3 showed intramedullary tuberculomas without leptomeningitis. The presence in our series of 5 patients with intramedullary tuberculomas without leptomeningeal disease indicates that tuberculous arachnoiditis is not always associated with intramedullary pathology.

The 5 patients showing diffuse high signal on T2-weighted images without any intramedullary contrast enhancement were thought to have cord oedema. Chang et al. [10] described a case of intense enhancement of the cord and attributed it to cord infarction or myelitis. However, the absence of contrast enhancement in our patients makes myelitis less likely. Also, the resolution of signal changes on follow-up MRI, observed in 3 of the 5 patients excludes myelomalacia secondary to ischaemia. The presence of leptomeningitis in all these patients suggests that the intensity changes in the cord are probably due to congestion secondary to arachnoid adhesions.

In one report of 3 intramedullary tuberculomas [11], the lesions gave high signal on T1-weighted images and high signal with hypointense centre on T2 weighting; they showed thick rim enhancement. The two cases reported by Jena et al. [13] had intramedullary lesions that gave low signal, sometimes with central high signal on T2-weighted images. These appearances have been described in intracranial tuberculomas [14–16]. In our series tuberculomas gave normal or low signal on T1 weighting and were of varied intensity on T2-weighted images. We have seen similar signal changes in intracranial tuberculomas and found a correlation between the number of macrophages and the degree of fibrosis on histopathology and increased accumulation of lipids, as detected with localised in-vivo proton spectroscopy [17]. Smaller lesions tend to give high signal and as they get bigger and better defined they become isointense and finally give low signal on T2-weighted images. In this series low signal lesions tended to be larger.

All our patients showed rim or nodular contrast enhancement, similar to that described in intracranial tuberculomas [14–16].

An interesting observation was the lack of significant cord swelling in our patients; those reported by Gero et al. [11] and Jena et al. [13] showed focal cord swelling in the region of tuberculoma.

Chang et al. [10], reported that Gd-DTPA-enhanced MRI revealed enhancing nodules or enhancement of the dura-arachnoid mater complex around the spinal cord in patients whose unenhanced images were normal. In the 5 patients studied by Gero et al. [11], unenhanced images were normal or revealed thickened dura mater (on T2-weighted images) or increased signal intensity within the thecal sac (on T1-weighted images), but contrast-enhanced images were superior for visualising and localising lesions; three patterns of enhancement were described linear or nodular enhancement coating the nerve roots and spinal cord and thick intradural enhancement completely filling the subarachnoid space. In our series, un-

enhanced MRI suggested the diagnosis of arachnoiditis in 9 of 13 patients (69%), given the findings of CSF loculation and obliteration of the subarachnoid space with loss of the outline of the spinal cord in the thoracocervical region and CSF loculation and irregularly thickened, clumped nerve roots in the lumbar region. In keeping with the findings of previous reports [10–12], Gd-DTPA-enhanced images revealed leptomeningitis in four patients with normal unenhanced studies and better delineated the extent of disease in two others. However, unlike previous studies, five patients thought to have leptomeningitis on unenhanced studies failed to show any enhancement, suggesting that absence of enhancement does not rule out meningeal involvement. Also, the pattern of enhancement is entirely nonspecific and does not differentiate tuberculous leptomeningitis from other infections or from leptomeningeal spread of tumour [10, 12].

Chang et al. [10] compared conventional myelograms, myelo-CT and MRI with and without contrast medium and concluded that conventional myelography remains the primary radiological method for diagnosing suspected spinal tuberculous radiculomyelitis, particularly chronic adhesive changes. They felt, however, that in patients with an active inflammatory process within the thecal sac or myelopathy, Gd-DTPA-enhanced MRI may be the optimal primary imaging technique, obviating invasive myelography. In our series, MRI revealed leptomeningitis in three and intramedullary tuberculomas in two of the five patients with normal myelograms. In the patients with positive myelograms, MRI better delineated the extent of leptomeningeal disease, and revealed associated intramedullary pathology. We therefore think that MRI should be the primary imaging modality in the screening of patients with suspected intraspinal TB irrespective of the stage of the disease.

The majority of extradural tuberculous granulomas are believed to be secondary to extension from adjacent vertebral disease, which may not be detectable on plain radiographs [13]. MRI is very sensitive to early tuberculous spondylitis in patients with normal radiographs, due to its ability to detect marrow abnormalities before the development of bony destruction [18, 19]. Two of our patients showed extradural tuberculous granulation tissue with normal signal from vertebral bodies, suggesting that it is possible to have extradural TB in the absence of spondylitis; the route of spread is most likely to be haematogenous in such cases.

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