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Review article

MRI findings in tubercular radiculomyelitis

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ABSTRACT

This article aims to familiarize the reader with the MR imaging findings of tubercular radiculomyelitis (TBRM) and to identify the sources of infection. We evaluated 29 patients on a 1.5 T GE MRI in a cross-sectional study. MRI of the spine with contrast and lumbar puncture were performed in all patients. MRI brain was performed for 13 patients. The typical and atypical manifestations enlisted in this article, will enable early detection of TBRM when the clinical history is ambiguous, as TBRM can present with low backache in both retrovirus positive and negative patients.

1. Introduction

Tuberculosis, caused by the bacillus Mycobacterium had a global incidence of 9.6 million in the year 2014 and thus presents a challenging public health problem [1]. Tuberculosis is one of the leading cause of mortality and morbidity both in developed and developing nations with 10 million incident cases and 1.6 million deaths in 2017 [2]. With the increasing incidence of tuberculosis especially in developing countries, meningeal and spinal vertebral column disease is becoming more common [3,4]. Intraspinal tuberculosis refers to spinal meningitis, tubercular myelitis, spinal arachnoiditis or spinal tubercular granuloma formation [5,6]. Tubercular radiculomyelitis is generic term applied to spinal arachnoiditis, myelitis and spinal cord tuberculoma, intradural tuberculomas and spinal cord edema, infarction [7]. The terms arachnoiditis, leptomeningitis and radiculomyelitis are often used interchangeably [8].

Patients with tubercular radiculomyelitis commonly present with gradual onset neurological deficits in lower limbs and bladder disturbance. The initial diagnosis is made on imaging but is confirmed by cerebrospinal fluid (CSF) analysis, AFB (Acid fast bacillus) staining with or without culture and polymerase chain reaction (PCR) gene amplification. It is imperative to achieve an early diagnosis so that therapy can prevent adverse neurological outcomes [9].

The hallmark of tuberculosis is caseating granulomas on biopsy, which are diagnostic. A response to a course of Anti-Tubercular Treatment (ATT) in a clinically suspected case is also confirmatory [10,11].

In a review of the clinical and neuroimaging features of tubercular myelitis, Wasay M et al. reported that while the cervical or thoracic segment of the spinal cord was most commonly involved, the most consistent MRI findings were hyperintense signal on T2-weighted MRI and post-contrast enhancement of the lesions, with epidural enhancement and cord swelling seen in some patients. Further, cavitation and syrinx formation portend worse outcomes [12].

In the current study the authors aim to describe the distribution of findings in tubercular radiculomyelitis on MRI, as well as identify the source of Tubercular infection. Knowledge of these imaging features will permit a higher degree of confidence in the diagnosis of this condition.

2. Materials and methods

This cross-sectional study was done at our institution after obtaining the Institutional Ethics Committee approval. Informed consent was obtained from all patients. The study included 29 patients with CSF microbiology/ PCR proven tuberculosis, who were evaluated on a 1.5 T MRI (GE, India). MRI of the spine was performed in all patients with sagittal T1W (TR 740 ms, TE 16.90 ms, 4 mm thickness, ET 4, Matrix size - 512 × 224, Nex - 4), sagittal T2W (TR 3800 ms, TE 110.5 ms, 4 mm thickness, ET 25, Matrix size - 512 × 224, Nex - 4), axial T2W (TR 2960 ms, TE 116 ms, 4 mm thickness, ET 18, Matrix size 256 × 192), and coronal STIR (TR 3200 ms, TE 49.4 ms, TI 150 ms, 6 mm thickness, ET 16, Nex 4) sequences. Gd contrast enhanced multiplanar fat sat T1W images were obtained in all patients using Magnevist. MRI brain was done for 13 patients, of whom 7 received contrast, while the remaining 6 patients did not undergo contrast MRI because of poor renal function. Confirmation of the diagnosis was achieved by CSF analysis including

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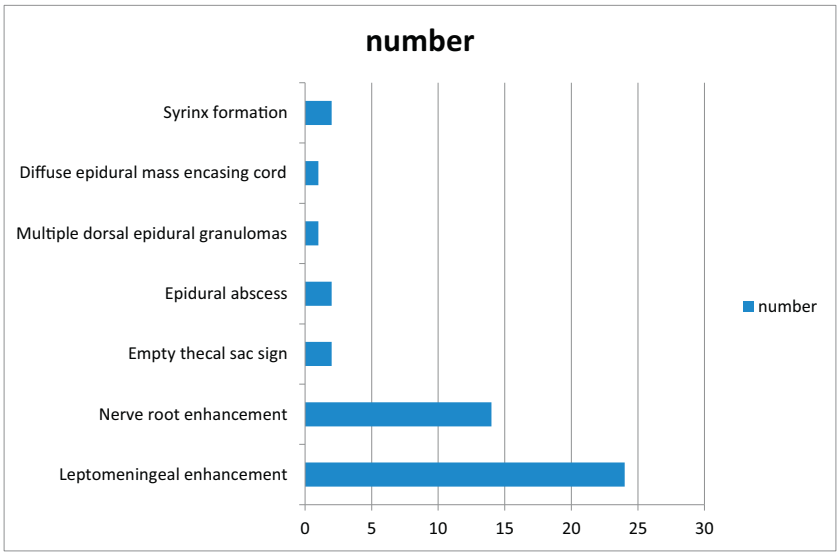


Fig. 1. Chart showing the MRI findings in patients with radiculomyelitis.

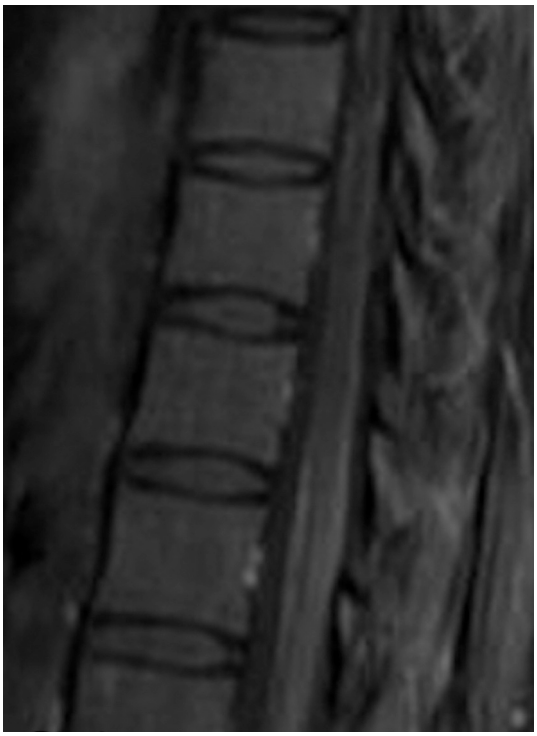


Fig. 2. Sagittal fat-suppressed contrast-enhanced T1-weighted MR image of spine shows diffuse enhancement along the surface of cord, indicating leptomeningitis.

AFB staining and CSF culture with some patients undergoing testing with PCR.

3. Results

Of the 29 patients (Fig. 1), 24 (82.8%) patients had leptomeningitis and 14 (62.1%) had arachnoiditis (Figs. 2, 3). Three patients showed the characteristic empty thecal sac sign (10.3%) (Fig. 4). One patient had intramedullary abscesses (3.5%) (Fig. 5), two (6.9%) patients had an epidural abscess (Fig. 6) and one patient (3.5%) had multiple granulomas in the dorsal intradural space (Fig. 7). One patient presented as a diffuse mass in the epidural space, encasing the cord (Fig. 8). 8 (27.6%) patients had features of myelitis, 2 patients (6.9%) had syrinx formation and 2 patients had CSF loculation (6.9%) (Fig. 9). None of our patients showed CSF hyperintense signal on T1 weighted images. (See Table 1)

8 patients suffered from myelitis (Figs. 10, 11). The most common presenting symptoms of myelitis were bowel and/or bladder incontinence or retention in 75% (6 out of 8 patients), motor deficits in 50% (4 out of 8 patients) and sensory deficits in 25% (2 out of 8 patients) patients.

Leptomeningeal enhancement (89%) and nerve root enhancement (66.6%) were the most common MRI findings noted in the subset of patients with non-CNS primary source of active infection. Myelitis (22.2%), empty thecal sac sign (22.2%), syrinx formation and CSF loculation (11.1% each) being the other findings.Table 2

Of these 9 patients in whom no active focus of tuberculosis was found, two patients gave past history of tuberculosis with one patient suffering from post-tubercular bony ankylosis and one patient with old pulmonary tuberculosis. In this subset of patients, 6 out of 9 patients (66.7%) suffered from sensory disturbances as the predominant presenting symptom.

4. Discussion

Tuberculosis is a global health problem known to commonly affect the respiratory system, the lymph nodes, gastro-intestinal tract, central nervous system, musculoskeletal system, genitourinary system and endocrine glands (such as adrenal glands). The spinal cord and nerve roots are a relatively rare site of involvement [7,13–16].

Patients suffering from tubercular radiculomyelitis present with gradually progressive paraparesis, radicular pain, bladder involvement, and paralysis in the due course [7].

In our study, bladder and bowel disturbances were seen in 75% of patients suffering from myelitis. The lower percentage of patients suffering from tubercular radiculomyelitis is probably because tuberculosis is a chronic infection where the neurological deficits are secondary to inflammatory process [17]. Further, patients would have responded to therapy initiated for the treatment of co-existing systemic infection.

Patients may not give a past medical history of tuberculosis, unless specifically questioned, or unless sequelae like apical fibrosis are detected on a chest radiograph as observed in 2 patients in our study. An important point to remember is that patients can develop TBRM at any point in their life following treated TBM [7,18]. The duration between

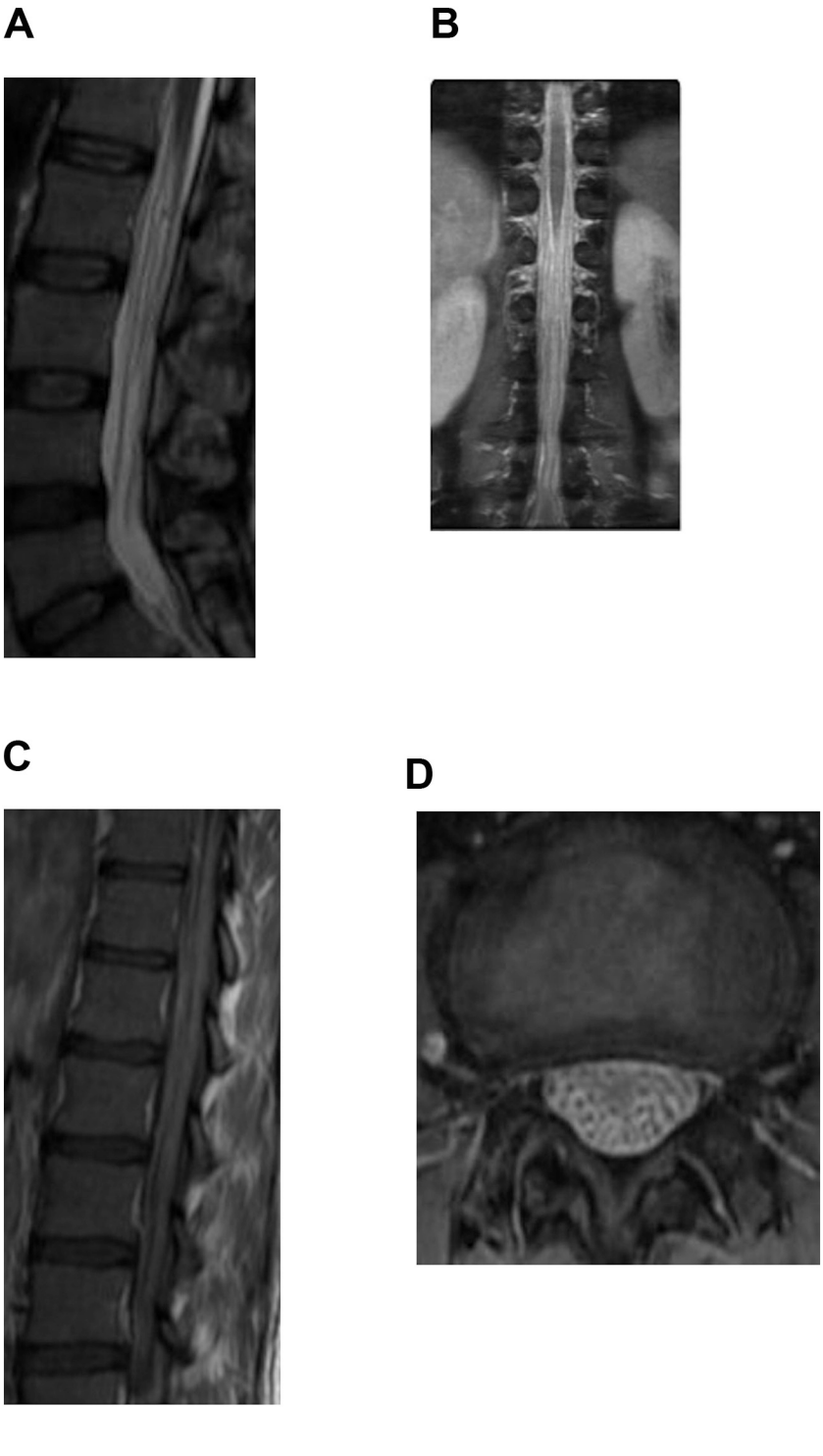


Fig. 3. A patient with arachnoiditis.
a. Sagittal T2-weighted MR image shows thickened nerve roots in the lumbar spinal canal.
b. Coronal fat-suppressed contrast-enhanced T1 MR image shows thickened enhancing fixed nerve roots.
c. Para-sagittal fat-suppressed contrast-enhanced T1 MR image shows thick enhancing meninges and nerve roots. Diffuse leptomeningitis is noted.
d. Axial fat-suppressed contrast-enhanced T1 MR image shows arachnoiditis, with thick enhancing nerve roots.

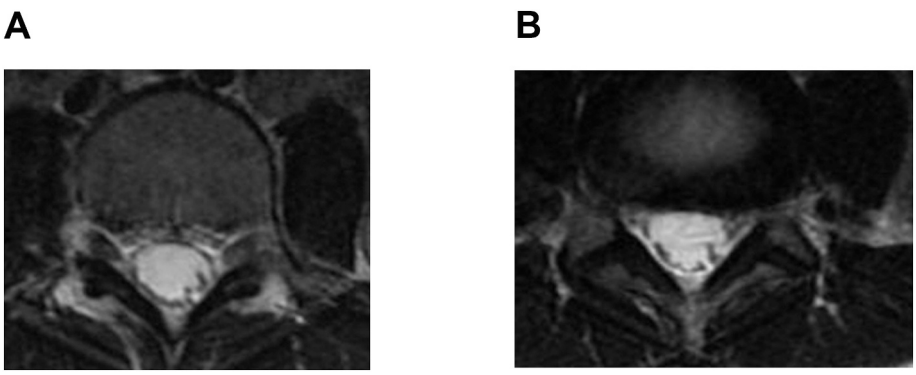


Fig. 4. 45-year-old female with constipation and urinary retention for 3 days and bilateral lower limb weakness for 3 months.
a,b. Axial T2-weighted MR images of the lumbar spine demonstrating the 'empty thecal sac sign', with clumping and peripheral adherence of nerve roots to dural sac.

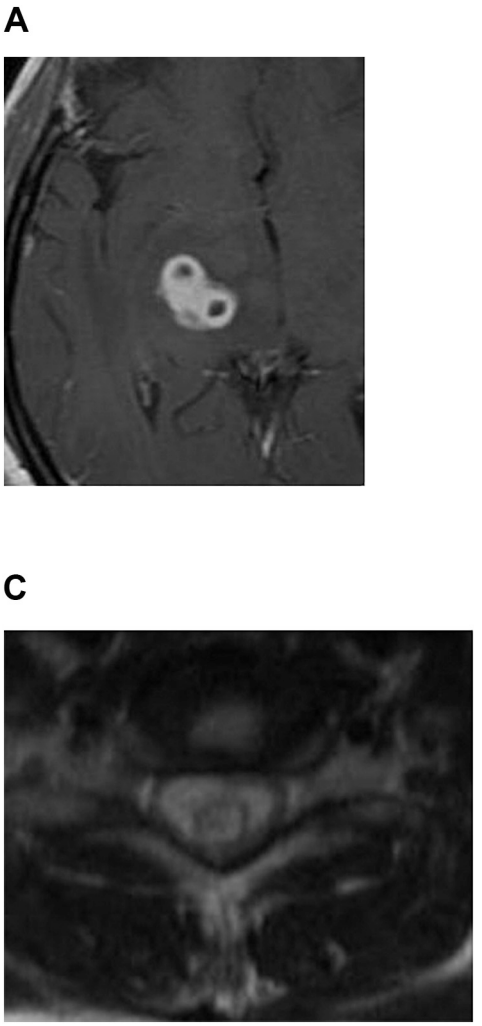


Fig. 5. 25-year-old female with clinical signs of meningitis and paraparesis.
a. Axial contrast-enhanced T1-weighted MR image through brain shows conglomerate ring enhancing tuberculomas in right basal ganglia and brainstem (not shown).
b. Sagittal fat-suppressed contrast-enhanced T1 MR image shows long segment conglomerate intramedullary ring enhancing tuberculomas. Note the enhancement along the surface of cervical and dorsal cord.
c. Axial T2-weighted MR image shows centrally hyperintense tuberculoma with a hypointense rim and a central hypointense focus, with associated cord edema and expansion, in the same patient.

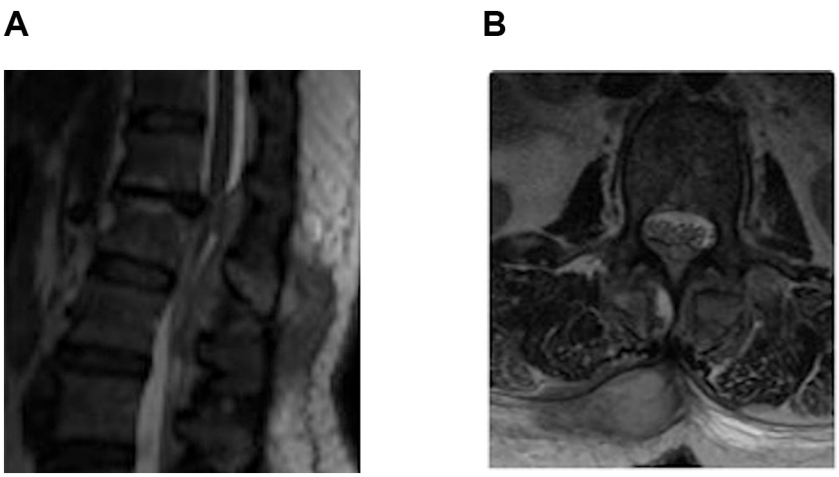


Fig. 6. 52-year-old-female with low back pain for 25 days, and history of fever 25 days back.
a. Sagittal T2-weighted MRI shows anterior and posterior epidural T2 hyperintense collection causing compression of conus medullaris and cauda equina. T2 signal alterations noted at D12 and L1 contiguous vertebral end plates, suggesting infective spondylitis.
b. Axial T2-weighted MRI shows posterior epidural collection with compression of cauda equina. Note the paraspinal collection.

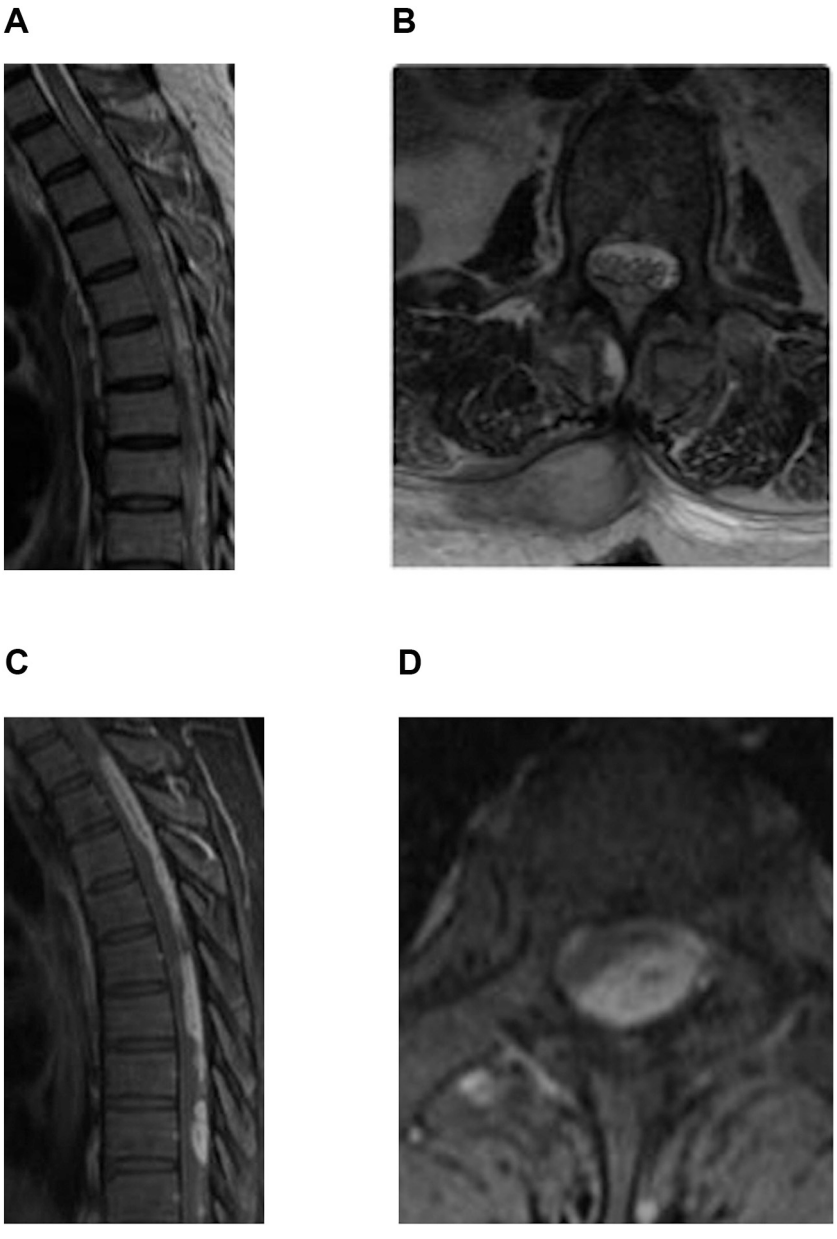


Fig. 7. 50-year-old retrovirus positive male with bilateral lower limb weakness for 10 days. Patient had history of laryngeal tuberculosis, and recent treated tubercular meningitis with cerebellar tuberculoma.
a. Sagittal T2-weighted MRI shows long segment T2 hyperintense posterior intradural lesion distorting the cord contour, with associated T2 hyperintensity in spinal cord (edema, ischemia or myelitis).
b. Axial T2-weighted MRI shows a tuberculoma with a hypointense rim.
c. Sagittal fat-suppressed contrast-enhanced T1-weighted MRI image shows long segment posterior intradural conglomerate thick ring enhancing tuberculomas causing cord compression. Associated leptomeningitis is observed.
d. Axial fat-suppressed contrast-enhanced T1-weighted MRI image shows posterior intradural tuberculoma compressing the cord.

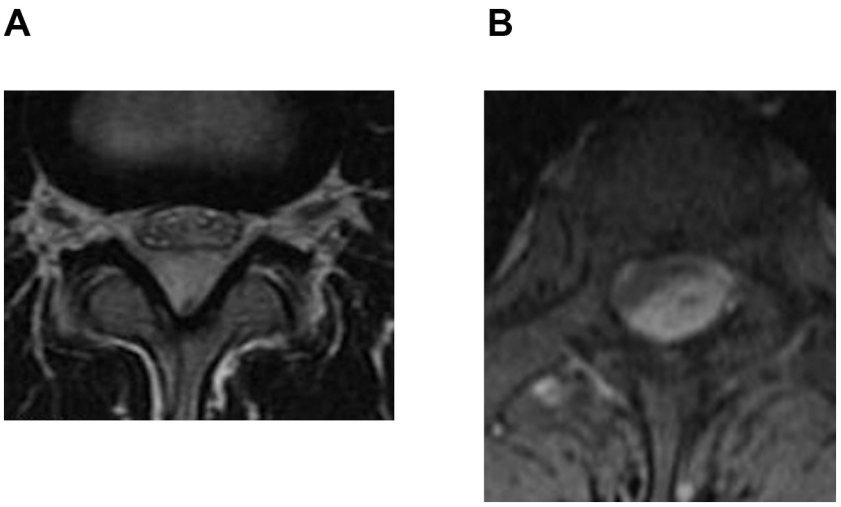


Fig. 8. 28-year-old male presented with low back ache of insiduous onset.
a,b. Axial T2-weighted MR images show a hyperintense lumbo-sacral epidural mass encasing the nerve roots of cauda equina and sacral spinal nerve roots, without infiltrating the nerve roots.
c. Sagittal T2-weighted MRI shows posterior epidural mass in lumbo-sacral spinal canal, with mixed hyperintense- hypo-intense signal. The nerve roots are encased and anteriorly displaced.
d. Sagittal fat-suppressed contrast-enhanced T1-weighted MRI shows diffuse intense enhancement in the same mass.
e. Axial fat-suppressed contrast-enhanced T1-weighted MRI shows diffuse mass like enhancing soft tissue encasing the lumbar roots. However, no nerve root infiltration is noted.

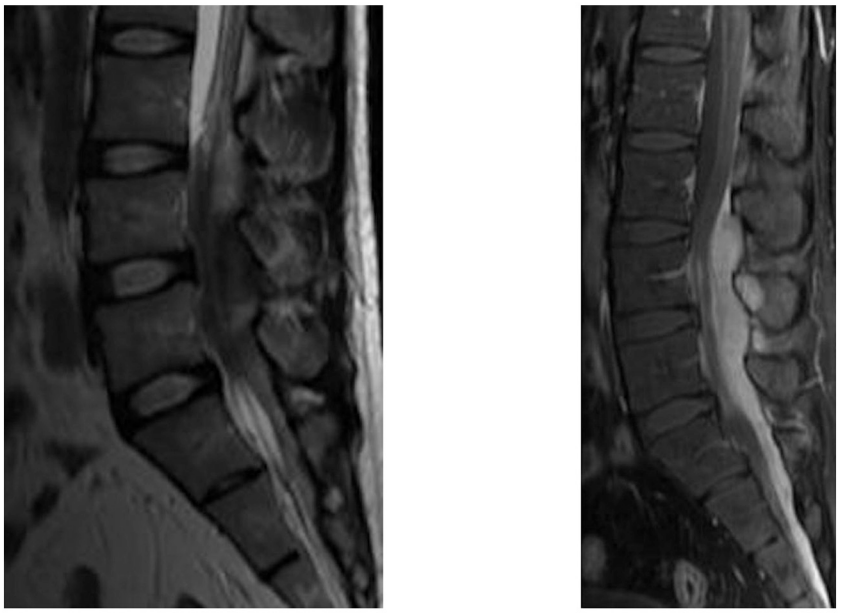
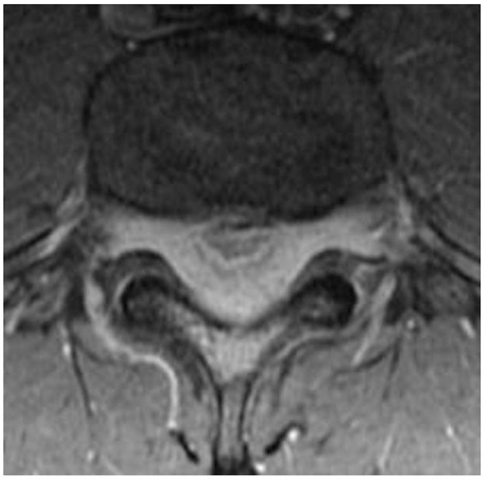


Fig. 8. (*continued*).



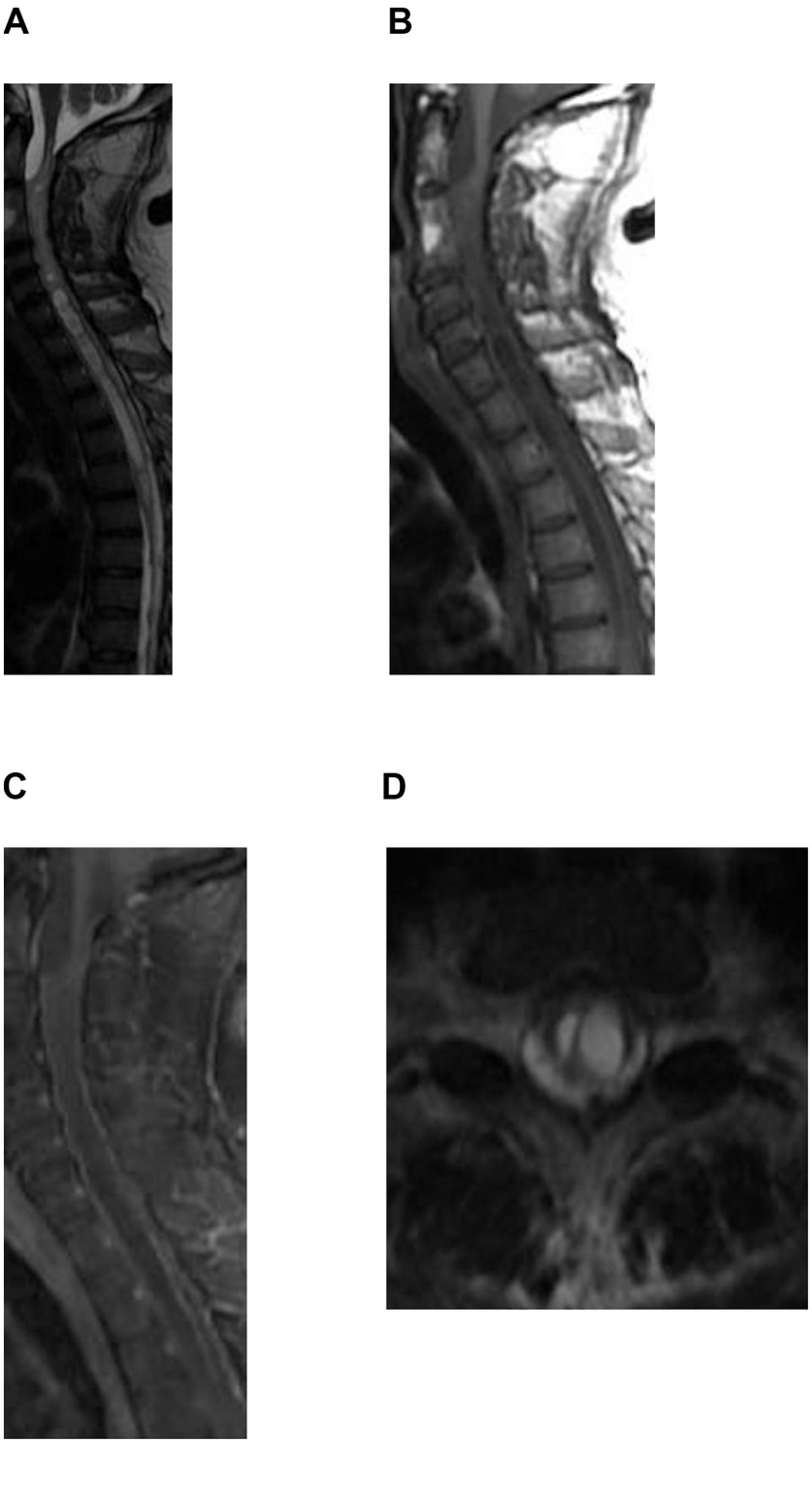


Fig. 9. 46-year-old-male with worsening ataxia and difficulty in walking for 2 months. Post- tubercular bony ankylosis of C3 and C4 vertebral bodies six years ago.
a. Sagittal T2-weighted MRI shows long segment cord cavitation of cervico-dorsal cord. There is alteration in contour of cervical cord and anterior medulla, with anterior CSF signal, suggesting CSF loculation. There is bony ankylosis of C3 and C4 vertebral bodies, a sequelae of old Tubercular C3-C4 spondylodiscitis.
b. Sagittal T1 weighted MRI showing the hypointense signal in the cord cavitation and CSF loculation, similar to CSF.
c. Sagittal fat-suppressed contrast-enhanced T1-weighted MRI. There is no enhancement of the CSF loculation. There are features of diffuse leptomeningitis.
d. Axial T2-weighted MRI shows multiseptated syrinx in the same patient.

Table 1
Table showing the primarily affected organs in patients suffering from Tubercular radiculomyelitis.

Primarily infected organ ($n = 20$)	Number	Percentage
Tubercular meningitis/tuberculoma	10	34.5
Pulmonary tuberculosis	6	20.7
Tubercular spondylodiscitis	5	17.2
Left iliacus hyperintensity/infection	2	7
Mediastinal nodes	1	3.5
Laryngeal tuberculosis	1	3.5
No primary organ identified	9	37.9

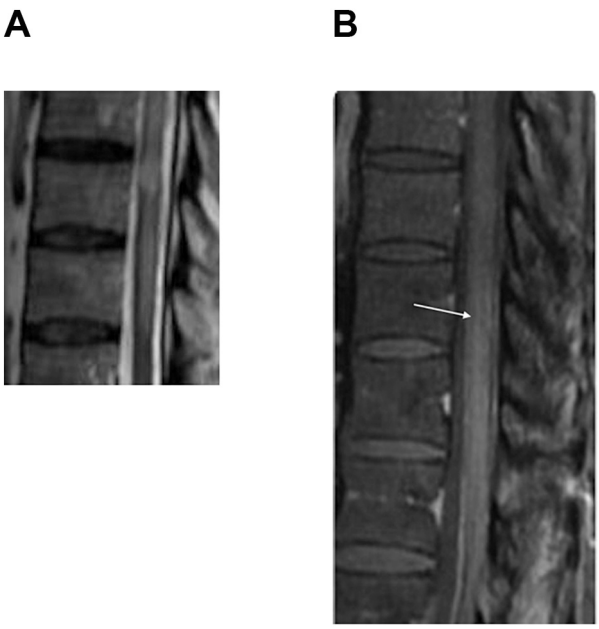


Fig. 10. a. Sagittal T2-weighted MR image shows long segment lower dorsal and lumbar cord central hyperintensity, representing edema, myelitis or ischemia. b. Sagittal fat-suppressed contrast-enhanced T1-weighted MR image shows linear enhancement (arrow) in central cord, representing myelitis or ischemia. This patient had leptomeningitis and arachnoiditis. The enhancement is subtle, as patient was started on steroids.

past history of solid organ or systemic Tuberculosis and the presentation of suspected radiculomyelitis may vary from concurrent disease to upto 5 decades after preliminary infection, as was observed in a patient in our study. These patients classically present with abnormal gait with progressive difficulty in walking over years. The chronic course of the disease, initial unsuspected or missed diagnosis, low socioeconomic status, patient ignorance, lack of accessible facilities all contribute to patient's long term suffering with psycho-social implications.

In evaluating the source of TBRM, three routes have to be considered. Tubercular meningitis (Fig. 12 and 13), hematogenous (Fig. 14) and contiguous from spondylodiscitis (Fig. 15). Fig. 12 and 13

The patterns of spread observed by us are in agreement with previously stated pathways of spread. We observed that despite the many cases of spondylodiscitis in our set up, the occurrence of TBRM in these patients is rare. In our series, only one patient developed TBRM as a complication of low CD4 counts in HIV. In our opinion, the term 'contiguous' spread from spondylodiscitis can be misleading, as one tends to think of focal infection. However, in many of these cases there is affliction of several vertebrae or a disseminated infection.

Hematogenous dissemination was the most common in patients evaluated for leptomeningitis. Wadia and Dastur reported 24 cases of presumed hematogenous spread, 14 cases of secondary extension from intracranial focus and 3 cases of contiguous spread from spondylodiscitis [8].

The pathophysiology of TBRM involves exuberant ooze of dense, viscous exudate that encases the spinal cord and duramater, or may occupy the spinal subarachnoid space and coat the nerve roots. The nerve roots tend to stick together and are described as matted. As the tenacious exudate settles into chronicity, it may become organized to produce adhesions and CSF blocks [9,19–22]. This translates to the MRI finding of CSF loculations or blocks.

Many studies have reported a loss of cord outline on T1 weighted images due to increased CSF proteins [8,11,23–27]. However, this finding was not well seen in our cases. The most common finding observed in our study was subtle to striking smooth, linear leptomeningeal enhancement, or in a few cases causing subtle surface irregularity possibly due to mild nodularity. The fibrin coated nerve roots and cauda equina may enhance as well. These findings have been previously reported in literature [11,23–25].

On T2 weighted images, the nerve roots may appear 'fixed' peripherally in the thecal sac, within the spinal CSF, producing the classic 'empty thecal sac' sign [28]. Plain MRI may show CSF loculation, loss of

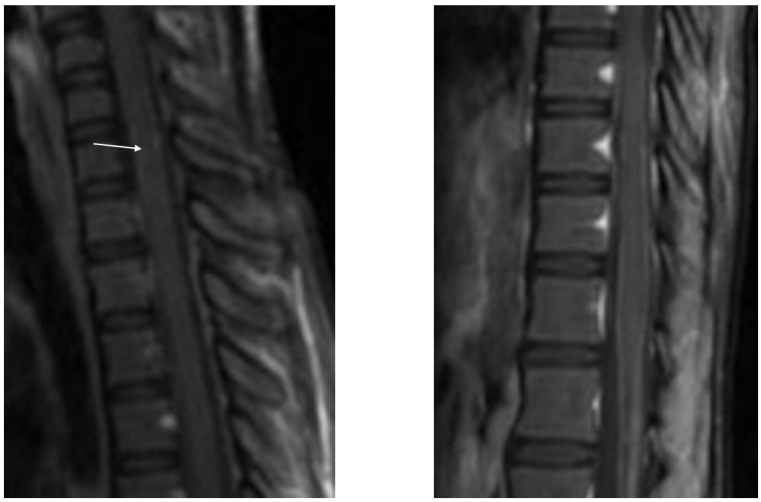


Fig. 11. 17-year-old female with Tubercular meningitis and vasculitic infarcts. Past history of treated spinal tuberculosis and TBM at age 5 years, with residual abnormal gait.
a. Sagittal fat-suppressed contrast-enhanced T1 MRI image shows linear central cord enhancement (arrow) at level of C6 and C7 vertebral bodies, consistent with myelitis. Meningeal enhancement is also noted.
b. Sagittal fat-suppressed contrast-enhanced T1 MRI image shows features of leptomeningitis and myelitis in dorsal cord.

Table 2
The distribution of findings among the 9 patients in whom no active primary infection was identified was as described below.

No.	Leptomen-ingeal enhancem-ent	Nerve root enhancement	Empty thecal sac sign	Myelitis	Syrinx	CSF Locul-ation	Any significant clinical history
1	1	1	1				Sensory disturbance in the lower limbs with weakness
2	1	1					ADPKD
3	1	1	1				Sudden fall, shock like sensation over right foot
4	1			1			Asymmetric sensory deficit in legs
5	1				1	1	Past history of tubercular bony ankylosis C3-C4. Now has paraesthesias (all limbs) and worsening ataxia with weakness
6	1			1			Burning sensation in the lower and upper limbs Urinary retention
7		1					Old case of pulmonary Koch's, Bilateral lower limb weakness
8	1	1					Fever, back pain, incontinence
9	1	1					Burning sensation, depressed reflexes and weakness right lower limb, gait disabilities

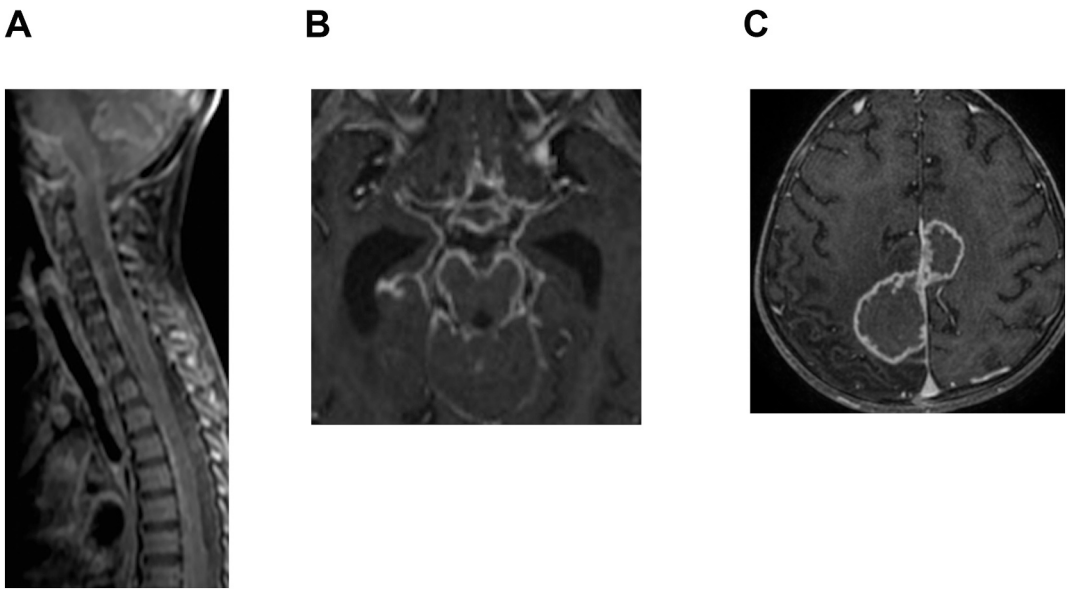


Fig. 12. 10-year-old child with fever and vomitings for 10 days, positive Kernig and Brudzinski signs, and paraparesis.
a. Sagittal fat-suppressed contrast-enhanced T1-weighted MR image of whole spine screening shows diffuse meningeal enhancement.
b. Axial fat-suppressed contrast-enhanced T1-weighted MR image shows enhancing exudates covering the brainstem and the basal cisterns.
c. Axial fat-suppressed contrast-enhanced T1-weighted MR image shows ring enhancing tuberculomas in brain.

subarachnoid spaces and cervico-dorsal spinal cord outline with clumping and thickening of lumbar nerve roots as imaging findings in spinal tubercular radiculomyelitis [8,11,24–27]. This finding, may be explained due to an increase in the level of CSF proteins, inflammatory exudates on the surface of the cord, or the formation of microadhesions between the cord and the theca [29]. Enhancing, matted nerve roots can be identified most easily on axial and para-sagittal sections of lumbar spine on contrast enhanced MRI.

Further, the inflammatory arachnoiditis results in affliction of the vessels of the cord and produces an inflammation of the end arteries and veins. The ensuing thrombosis and obliterative endarteritis, results in ischemia or even infarctions of the cord parenchyma [21,22,30]. Gradually it may produce ischemic myelomalacia and/or syrinx [22]. It is very important to keep in mind that development of syringomyelic cavities may be responsible for the late neurological deterioration in these patients.

The spinal cord may show central T2 hyperintensity representing edema, myelitis, ischemia and infarction [20]. Intramedullary spinal tuberculomas show varied T2 signal depending on stage and size of lesions, with progression of T2 hyperintense signal in smaller lesions to isointense and hypointense signal in larger lesions [24]. Linear or patchy central enhancement may be observed in case of myelitis [31], enhancement is also observed in ischemia and intramedullary tubercular granulomas [20], while no enhancement is observed in edema [24]. Initiation of treatment results in better defined peripheral or nodular enhancement on contrast enhanced T1W MRI, following similar course as does brain cerebritis when evolving into early abscess. Once established, intramedullary abscess has centrally necrotic T2 hyperintense area with surrounding T2 hypointense enhancing rim. Enhancement may take months to subside following treatment [31]. Edema surrounding the abscess, on the other hand is greater than the size of the abscess, but subsides in few weeks, following treatment [31].

Myelitis or infarction of these may progress to T1 hypointense and T2 hyperintense non-enhancing cord cavitations or syrinx [21,24]. The development of Syrinx or CSF block herald bad prognosis, and are responsible for the late functional decline and worsening of paraparesis. [21,32,33]. Conversely, preventing cavitation/ syrinx formation and CSF blocks is of utmost importance, and plausible when appropriate treatment is instituted early in the disease course. This is possible only by maintaining high clinical suspicion and early disease detection by MR Imaging.

Intradural lesions may appear as isointense T1 and isointense on T2 weighted images may demonstrate thick enhancing phlegmonous lesions filling the intradural space or nodular enhancing lesions

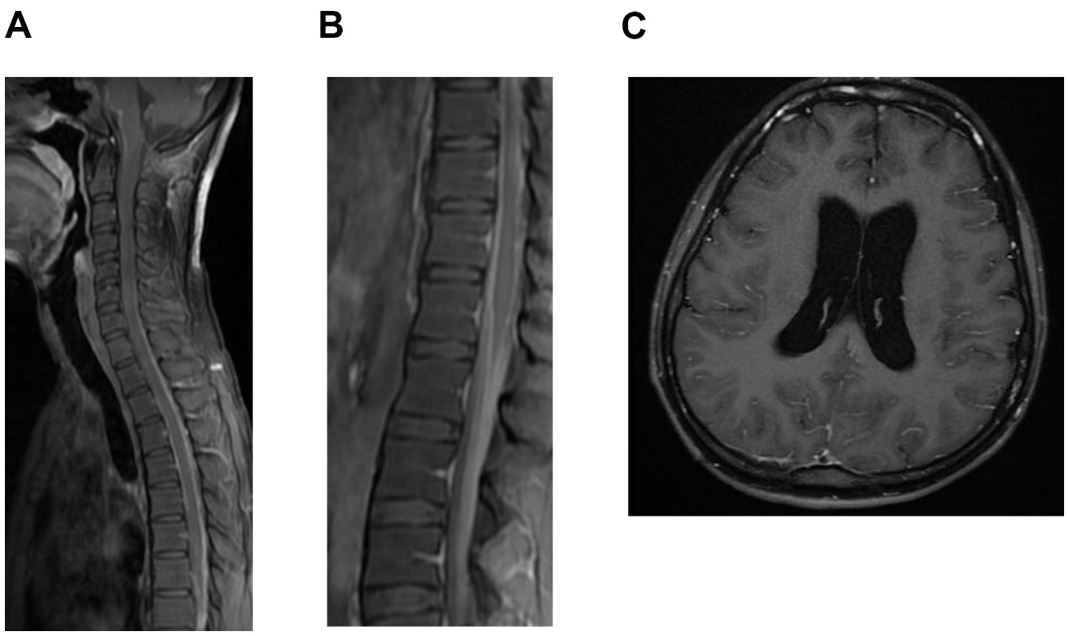


Fig. 13. 28 year-old-male presented with fever and chills for 2 weeks and multiple vomitings for 2 days.
a,b. Fat-suppressed contrast-enhanced Sagittal T1 MR images of whole spine show thick meningeal enhancement along the surface of entire cord, suggestive of leptomeningitis (arrow). Fig. 13a also demonstrates brainstem exudates.
c. Axial contrast-enhanced T1 MRI of Brain shows diffuse meningitis with hydrocephalus.

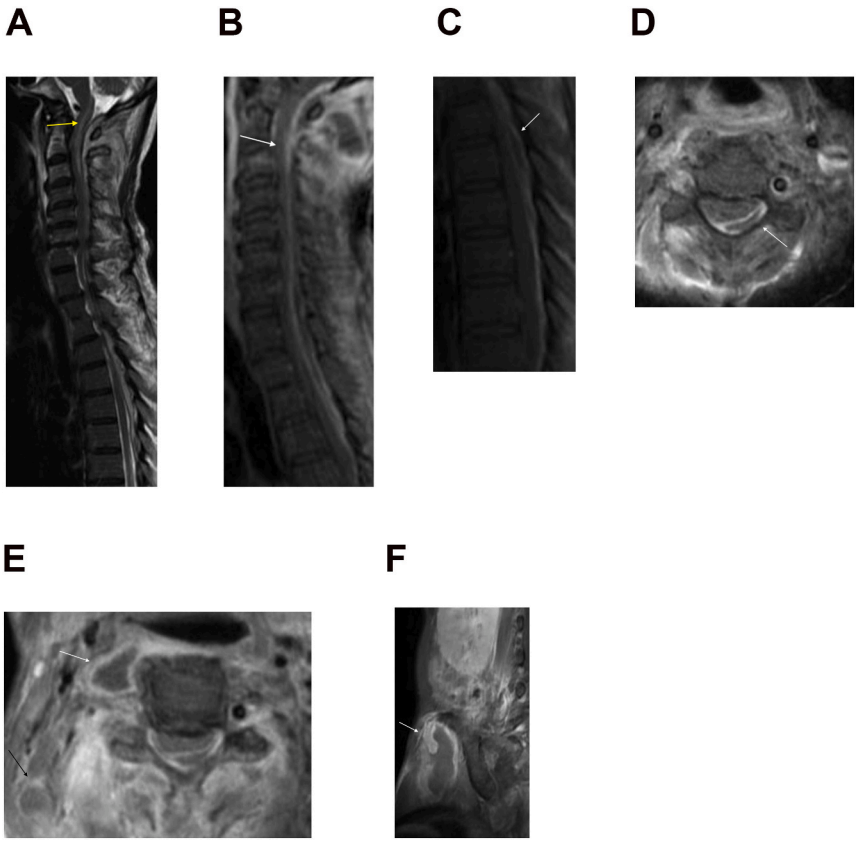


Fig. 14. 57-year-old male with disseminated tuberculosis, and past history of pulmonary tuberculosis.
a. Sagittal T2 MR image shows multisegmental cervicodorsal posterior epidural collection, with anterior displacement of the dura, and cord compression and anterior displacement. There is cervical cord enlargement and linear T2 hyperintensity representing edema or myelitis (arrow). There is congenital fusion of D2 and D3 vertebral bodies with rudimentary disc.
b. Sagittal fat-suppressed contrast-enhanced T1 MR image shows enhancement of central cord, suggesting myelitis (arrow). Note the enhancing dura in the cervical spinal canal forming posterior epidural abscess.
c. Sagittal fat-suppressed contrast-enhanced T1 MR image. The posterior epidural abscess (arrow) is better shown in this image, in the dorsal spinal canal.
d. Axial fat-suppressed contrast-enhanced T1 MR images showing the posterior epidural abscess (arrow), causing cord compression.
e. Axial fat-suppressed contrast-enhanced T1 MR images shows a right parapharyngeal (white arrow) and a paravertebral abscess (black arrow). The posterior epidural abscess compressing the cord, is again noted.
f. Coronal fat-suppressed contrast-enhanced T1 MR image shows a peripherally enhancing right gluteal abscess (arrow) in this patient with disseminated tuberculosis.

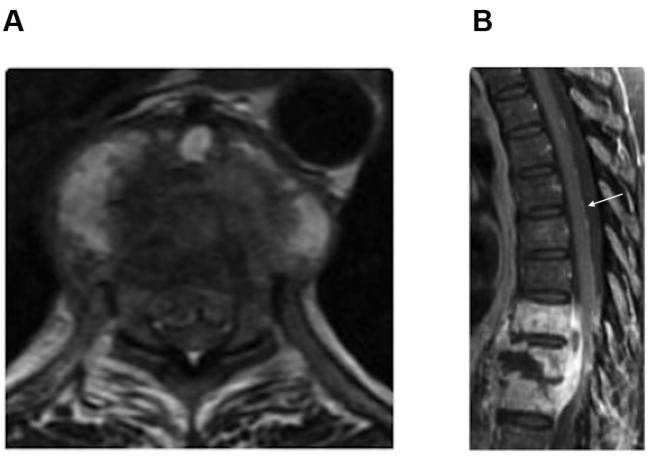


Fig. 15. 48-year-old female with low backache for 4 months, bilateral lower limb pain for 1 month and inability to walk due to pain. Patient had pulmonary tuberculosis (not shown).
a. Axial T2 MRI shows destruction of vertebral body with prevertebral and paravertebral collections. There is anterior spinal epidural collection compressing the cord with cord signal changes.
b. Fat-suppressed contrast-enhanced Sagittal T1MR images show destruction and enhancement of D8 to D10 vertebral bodies with anterior epidural collection. There is meningeal enhancement along the posterior surface of dorsal cord (arrow).

[11,23–25]. However, in the chronic phase there is no intradural enhancement, [24]. One patient in our study showed thick enhancing exudates in intradural space completely filling the thecal sac and encasing the cord.

Epidural lesions may be nodular (Tuberculomas), phlegmonous or ring enhancing abscesses. Epidural lesions may be isointense or hyperintense on T2 and with difficulty may be distinguished from CSF, by the inward displacement of the dura on T2 weighted images [24,31]. Uniform enhancement is noted if the tubercular inflammation is phlegmonous in nature, while a peripheral or ring enhancement may be seen if the lesion was a true epidural abscess or a caseating granuloma [24,30].

Other diagnostic possibilities must also be considered, in case of T2 hyperintense lesions of the spinal cord with or without enhancement, like demyelination, ADEM (Acute demyelinating encephalomyelopathy), hemangioblastoma, peripherally enhancing tumors with central necrosis or a subacute cord infarct which could mimic intramedullary abscesses. However, lumbar puncture for CSF analysis with culture or PCR, CSF adenosine de-aminase, histopathology and follow up imaging, serology of blood, tissue and CSF could be useful in differentiating these lesions, [34–36]. History of past infection with tuberculosis may have adjunctive value in arriving at the diagnosis.

In conclusion, the most sensitive method for the radiological evaluation for TBRM is an MRI using T1-weighted pre- and post-contrast images. Because of frequent long-term involvement, extended FOV images should be acquired for the whole spine in all patients. Study of the brain should also be routinely carried out to detect the frequently associated intracranial abnormalities. MRI features of TBRM include leptomeningitis, arachnoiditis with empty thecal sac sign, intramedullary abscesses, epidural abscess, multiple granulomas in the dorsal intradural space, diffuse mass in the epidural space, encasing the cord, myelitis, syrinx formation and CSF loculation.

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