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MAGNETIC RESONANCE IMAGING IN EARLY DIAGNOSIS OF SPINAL TUBERCULOSIS

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ABSTRACT

Introduction: MRI is a highly sensitive and specific, noninvasive investigation for early diagnosis of spinal tuberculosis without hazards of radiation. However, a clear consensus is lacking, as to what defines a tubercular lesion on a spine MRI. This study was aimed at establishing the commonest diagnostic features of spinal tuberculosis on MRI. **Methods:** Thirteen consecutive spinal tuberculosis patients (5 males, 8 females), diagnosed either bacteriologically by culture or clinico-radiologically, were studied retrospectively for the pathological features of tuberculosis on their pre-treatment MRI. **Results:** Thoracic (76.9%, most common), lumbar (38.5%) and cervical (7.7%) regions were involved. 40 affected vertebrae were noted, mean 3.1 vertebrae, with 92.3% patients having multiple vertebral involvement. All pathological lesions were T1W hypointense and T2W hyperintense. Central body lesions (84.6%) were more common than paradiscal lesions (69.2%). Kyphotic collapse (76.9%; greater in paradiscal lesions), vertebra plana (23.1%), disc signal changes (84.6%, with 61.5% having reduced space), end plate erosion (69.2%), end plate destruction (46.1%) and cord edema (61.5%) were seen, without myelomalacia or syrinx formation. Canal stenosis was seen in 100% patients, only 61.5% patients had neurodeficit. Abscess collection (76.9%), with spread in subligamentous space (70%), epidural space (60%) and along psoas (40%) were seen. Marrow edema was noted in 100% cases. Central vertebral lesion, disc intensity changes, marrow edema and canal stenosis were observed in combination in 84.6% patients. **Conclusion**: MRI is an indispensible modality of primary investigation in suspected cases of spinal tuberculosis. Canal stenosis, marrow edema, central vertebral body involvement, disc intensity changes and abscess collection are the most consistent MRI features of TB spine.

KEYWORDS: Tuberculosis; Magnetic resonance imaging; MRI; Diagnosis; Early diagnosis.

INTRODUCTION

Since Sir Percival Pott's first description of spinal tuberculosis (TB) in 1779,[1] our understanding of the disease, its natural history, diagnostic modalities and treatment approaches has evolved greatly. Today, spinal TB constitutes 50% of skeletal TB, 50-60% of extra-pulmonary TB and 1-5% of all TB.[2] In an endemic country like India, which has a very high prevalence of pulmonary TB, about 1.7% patients with pulmonary TB have spinal TB.[3] In spite of the massive burden of the disease, TB still remains a medically curable disease; provided there is rapid diagnosis and prompt initiation of antituberculous chemotherapy. Treatment delay is associated with increased morbidity and debilitating consequences like spinal deformity and neurological deficit. The long duration of antitubercular drug therapy, emergence of drug resistance, the plethora of investigative modalities, the necessity of ruling out other differential diagnoses whilst avoiding medico-legal discrepancies; all these challenges lead the treating clinician to look out for one single, rapid, reliable, cost-effective and reproducible modality of investigation, to definitively diagnose spinal TB.

Conventional tissue-based diagnostic tests like histopathology, bacteriology and cytology; are time consuming, invasive and observer dependent. Sometimes, it may not even be possible to obtain a sample for testing, due to the deep seated location of the lesion.[4] Conventional radiological imaging is noninvasive but it takes nearly three to four months for spinal TB lesion to be evident on plain radiographs.[5,6] The advent of magnetic resonance imaging (MRI), with a reported sensitivity and specificity of 100% and 88.2%, respectively, for spinal TB,[7] has revolutionized the diagnosis of the condition; by its ability identify the

pathological lesions in the early stages of the disease, correctly demonstrate the extent of the disease involvement and monitor the response to treatment. However, the usefulness of MRI in diagnosis depends on accurate interpretations of the findings seen. With a broad spectrum of MRI features of spinal TB outlined in literature,[4] there is no consensus at to what defines a tubercular lesion on a spine MRI. Hence, this study was conducted to outline the various pathological changes seen on MRI in established cases of spinal TB.

MATERIALS AND METHODS

Eighteen successive patients diagnosed with spinal TB, presenting at a tertiary care centre between 2013 – 2014, were included in the study. The criterion for inclusion was establishment of a definitive diagnosis of spinal TB; either by histopathogical/ cytological/ culture growth of mycobacterium tuberculosis (in 11 patients) or by classic clinico-radiological diagnosis with definite improvement on antituberculous chemotherapy (in 7 patients). The patients with an inconclusive or doubtful diagnosis of spinal TB and those with infection due to non-tuberculous mycobacteria were excluded from the study. All the patients eligible for the study had a pre-treatment MRI done at the time of diagnosis, but five patients' MRI images could not be accessed for assessment and were eventually excluded from the analysis. Thus, 13 patients were finally included in the study design. A retrospective assessment of the pre-treatment MRI of the eligible patients was done and the findings were noted. The institutional ethics committee approved the plan for the study and a written informed consent was obtained from each patient after proper explanation of the study protocol.

The baseline data collected from each patient included anthropometric data, clinical examination and a detailed neurological evaluation. Anteroposterior and lateral radiographs of the spine were obtained for all patients, and following parameters were noted to be suggestive of tuberculous infection: vertebral destruction, loss of vertebral height, wedging, kyphotic deformity, end plate destruction, intervertebral disc space obliteration and paravertebral soft tissue shadows. The MRI's were performed on systems with field strengths ranging from 1 .5 to 3 Tesla. Noncontrast T1-weighted (T1W), T2-weighted (T2W) and short tau inversion recovery (STIR) sequences in axial, sagittal, and coronal planes were obtained. The images were examined for configuration and intensity of the signal from the affected vertebral bodies and the intervertebral discs. The signal intensity in the normal vertebral bodies and discs in the field of view served for comparison. The MRI assessment was broadly divided into: vertebral body involvement, disc involvement, spinal cord changes, cold abscess and bone marrow changes.

Vertebral bodies were assessed for the nature and extent of involvement, location of vertebral lesion, degree of collapse and kyphosis. Degree of collapse was measured as midvertebral body height of the affected vertebra as compared to the average midvertebral body height of the adjacent unaffected vertebrae. Wedging and kyphosis was measured using Cobb's method. The intervertebral disc space was assessed for its intactness, signal intensity changes, loss of height and the extent of adjacent vertebral endplate involvement; the findings being compared with the disc spaces above and below, to assess deviation from the normal. Direct cord involvement, as evidenced by signal changes in the cord parenchyma, was noted, and correlated with the presence of absence of neurological deficit. The degree of canal stenosis was calculated by subtracting the anteroposterior canal diameter at the affected vertebral level from the mean canal diameter of the adjacent levels, expressed as a percentage. Canal stenosis was then correlated with the degree of neurological deficit. Presence of soft tissue collection, in and around the vertebral column, was noted and the extent of the abscess was defined. Subligamentous spread was defined as anteriorly under the anterior longitudinal ligament; epidural spread was defined as spread in the epidural space, posterior to the vertebral body; and psoas spread was defined as spread along the psoas muscle extending into the abdomen and pelvis.

RESULTS

Five males (38.5%) and eight females (61.5%), with ages ranging from 7 to 55 years (mean 28.3 years) were included in the study. Backache was the most common presenting symptom (n-10, 76.9%) followed by neurological deficit (n-8, 61.5%) and persistent low-grade fever (n-5, 38.4%). Complete neurological deficit with paraplegia (6/8 patients, 75%) was more common than incomplete neurological deficit (2/8 patients, 25%). Spinal tenderness was the most consistent examination finding seen in patients (n-11, 92.3%).

On MRI, thirteen patients had 40 involved vertebrae (range 1 – 5), with a mean of 3.1 vertebrae affected per patient. Thoracic spine was most frequently involved (10/13, 76.9%), followed by lumbar spine (5/13, 38.5%) and cervical spine (1/13, 7.7%). One patient (7.7%) had single vertebral involvement, two patients (15.4%) had 2 contiguous vertebral involvement, six patients (46.1%) had 3 contiguous vertebral involvement, three patients (23%) had 4 contiguous vertebral involvement and one patient (7.7%) had 5 non-contiguous vertebrae involved, suggesting a skip lesion (Figure 1).

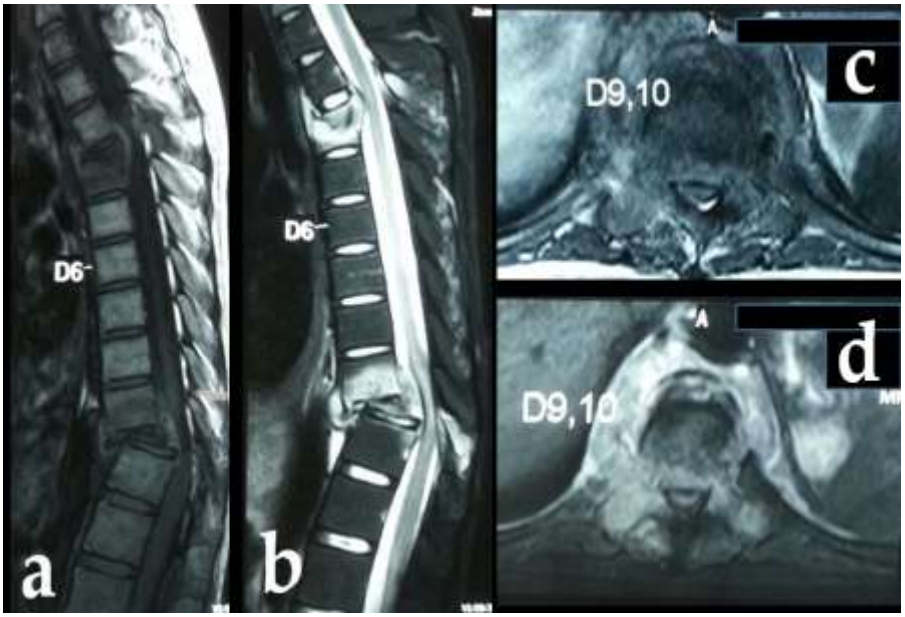

Figure 1: a, b. Sagittal section T1W and T2W showing multifocal lesion involving D3-D4 and D9-D10, central body destruction, concentric collapse at D3-D4, toppling collapse at D9-D10 intact intervertebral disc spaces, epidural abscess, marrow edema and cord edema. c, d. Axial section T1W and T2W showing vertebral body destruction, anterior subligamentous abscess, cord compression with granulation tissue causing 70% canal stenosis.

All pathological vertebral lesions showed hypointense signal on T1W images and hyperintense signal on T2W images. Marrow oedema was noted in all 13/13 patients (100%) and in all the 40/40 involved vertebral bodies; seen on MRI as hypointensity on T1W image and hyperintensity on T2W images. Anatomically, vertebral body involvement was classified as being central, paradiscal and posterior elements involvement. Central vertebral lesion was noted in 11 patients (84.6%), paradiscal lesion in nine patients (69.2%) and posterior element involvement in eight patients (61.5%). Kyphotic collapse of vertebral body was seen in 10 patients (76.9%) with mean Cobb's angle of 18.2^{o} (range 0^{o} to 65^{o}) with vertebra plana noted in three patients (23%). Paradiscal lesions were associated with a greater Cobb's angle (mean 37.6^{o}) than central vertebral body lesions (mean 12.6^{o}) (Figure 2).

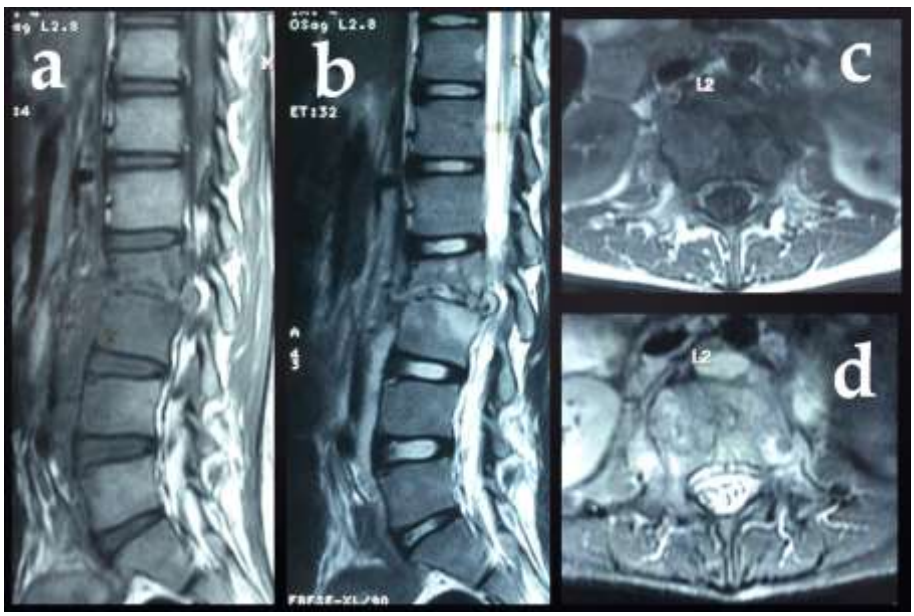

Figure 2: a, b. Sagittal section T1W and T2W showing L2-L3 paradiscal lesion with intervertebral disc space destruction, L2 inferior end plate destruction, L3 superior end plate erosion, kyphotic wedge collapse, retropulsed epidural granulation tissue with L2 and L3 marrow edema. c, d. Axial section T1W and T2W showing caseating body destruction, minimal canal stenosis (20%), anterior subligamentous abscess with intact posterior elements.

Intervertebral disc was involved in 11 patients (84.6%) as evidenced by signal changes with hypointensity on T1W images, hyperintensity on T2W images and postgadolinium enhancement on T1W images. Two patients (15.4%) had intact preserved disc spaces, with an isolated vertebral body involvement in one and isolated posterior elements involvement in the other patient. 8/11 patients (61.5%) with disc affection, had a concomitant reduction in disc height, while three patients (38.5%) maintained the disc interval with only alteration in the normal signal intensity, suggestive of discitis. Vertebral endplate erosion was seen in nine patients (69.2%); with complete endplate destruction seen in six patients (46.1%). Erosion was seen as fuzzy irregular margins on MRI, whereas endplate destruction was associated with variable degree of intervertebral disc space obliteration. Marrow hyperintensity was noted in all (100%) patients and in all the affected vertebral levels.

Cord involvement was observed in the form of intraparenchymal edema noted in eight patients (61.5%), seen as hyperintensity on T2W images. There was no evidence of myelomalacia, syringomelia or cord atrophy. A variable degree of canal stenosis was seen in all patients (100%), which varied from minimal canal encroachment to critical canal stenosis. One patient (7.7%) showed an intra-canal tuberculous granuloma compressing the spinal cord. Neurological deficit was noted in eight patients (61.5%). The mean canal stenosis was 29.6% (range 10% to 60%) in patients without neurological deficit, as compared to 41.6% (20%to 90%) in patients with neurological deficit. However, the degree of canal stenosis did not significantly correlate with the occurrence of neurological deficit.

An abscess was noted in 10 patients (76.9%). The abscess was seen tracking anterior to vertebral body in the anterior subligamentous space in 7/10 patients (70%), posterior to vertebral body in anterior epidural space in 6/10 patients (60%) and along the psoas muscle into the pelvis in 4/10 patients (40%). The vertical extent of the abscesses was greater than the extent of vertebral body involvement. One patient (10%) presented with a discharging sinus in the lateral lumbar region from a ruptured tuberculous abscess at presentation.

DISCUSSION

Early diagnosis is the keystone in the management of spinal TB. Delay in diagnosis leads to extensive bony destruction, progressive deformity and permanent neurological deficit. Traditionally, bacterial culture of mycobacterium tuberculosis has been the gold standard for diagnosis of any tuberculous focus in the body. However, culture takes a long incubation period for growth (atleast 6 weeks) on conventional growth media. Additionally, due to the smaller number of tubercle bacilli present in spinal lesions, bacteriologic studies may often be negative.[8,9,10] The histopathological diagnosis of the tuberculous lesion as well as the bacterial culture, require sampling of the lesion, which would entail an invasive procedure to be performed on the patient. Conventionally, radiographs are used as a primary tool for the assessment of any suspected tuberculous lesion in the spine. However, atleast 50% vertebral destruction should have occurred, before the lesion is evident on a plain radiograph.[5] The sensitivity, specificity and accuracy of plain X-rays are 82%, 57% and 73%, respectively, for diagnosis of spinal TB.[11] The X-rays are unable to visualise accurately the "blind areas" (cranio-vertebral junction, cervico-dorsal region, sacrum, sacro-iliac joints, posterior elements) due to overlapping of shoulder shadows.[11, 12] Advantages of MRI over conventional radiographic techniques is higher sensitivity & specificity, better visualization of blind areas, clear delineation of vertebral body, disc and cord changes.[4] It has excellent soft tissue resolution without the danger of radiation. Pathological changes appearing on MRI precede those appearing on conventional radiographs by several months, thereby hastening the diagnosis of the disease, in the early pathological stages. MRI offers excellent visualization of the bone and soft tissue components of spinal TB and helps to identify disease at distant asymptomatic sites.[13] MRI clearly demonstrated the extent of soft tissue disease and its effect on the theca/cord and foramen in cases with doubtful CT findings.[13] MRI provides a reliable guide to prognostication of the outcome of therapeutic measures in spinal TB.[14] The assessment and interpretation of the MRI findings is the primary criterion that determines the accuracy in diagnosis of spinal TB. Various studies have been conducted delineating the common features seen in MRI in spinal TB.[5,6,15,16]

With a mean patient age of 28.3 years, the finding outlines the predominant prevalence of TB in the younger, active & outgoing population group, having a higher incident exposure to the airborne tubercle bacilli. The average age of occurrence of spinal TB is below 20 years in 52% patients and below 30 years in 73% patients, with the incidence decreasing with rise in the age group.[11] Backache was the most common complaint at first presentation to the clinic, followed by neurological deficit. 90 – 100% incidence of backpain at presentation has been reported in literature.[17,18,19,20] 61.5% patients presented with neurological deficit. Frequency of neurologic involvement varies across studies, ranging from 23% – 76%, with differences in severity.[17,18]

Dorsal spine is most common site of involvement of TB spine, followed by lumbar spine.[11,18,20,22] In Cleveland's series the peak incidence was at D11, while in Hodgson's series it was observed at L1 level with a uniform fall proximally and distally.[11] He suggested that peak incidence at that level was due to contiguous spread from the kidneys.[11] A similar finding was noted in our study with thoracic vertebral involvement being most common with a peak incidence at D11, D12 & L1.

Mean number of vertebrae involved per patient, in the study, was 3.1, with 92.3% patients showed greater than one vertebral involvement. Average vertebral involvement in spinal TB is reported to be between 2.5 to 3.8.[11] Embryologically, vertebral segmental arteries bifurcate to supply two adjacent vertebrae developing from a single sclerotome (lower half of upper vertebra and upper half of lower vertebra). Hematological dissemination of the tubercle bacilli thus leads to seeding and involvement of adjacent vertebrae. Additionally, spread of the disease in the form of pus and granulation tissue, beneath the anterior or posterior longitudinal ligaments, also leads to involvement of multiple contiguous vertebrae.[23] Multiple skeletal and spinal foci of TB are commonly seen in patients from developing countries.[18] Smith et al noted that 50%-60% patients with spinal TB have three or more vertebral body involvement seen on MRI.[24]

Anatomically, the vertebral body involvement was most commonly central and paradiscal. Central body involvement occurs due to spread from the intraosseous Bateson's venous plexus, with resultant concentric collapse of the vertebral body. Complete collapse results in vertebra plana (seen in 23.1% patients). Paradiscal involvement occurs due to spread from the arterial plexus, with the initial focus of infection being adjacent to the end plate in the anterior half of the vertebral body, with further involvement of disc space. Anterior vertebral destruction leads to anterior wedging and resultant kyphosis of the vertebra (seen in 76.9% patients). Thus, paradiscal lesions were associated with a higher mean Cobb's angle than central vertebral body lesions. Involvement of posterior elements was noted as destructive lesion of pedicles, laminae with or without a posterior paraspinal abscess. The lamina is most common site of involvement in posterior element spinal TB, followed by pedicles, articular processes, spinous processes, and transverse processes.[25,26,27,28] Isolated involvement of posterior elements was seen only in one patient. Pertuiset et al had noted that neural arch involvement is fairly common and can occur either alone or in combination with vertebral body lesion.[18] Accurate diagnosis of isolated posterior element TB is essential, since similar picture is seen in spinal tumors and treatment strategy would encompass a laminectomy in addition to chemotherapy.[24]

Based on involvement of disc interval, Pertuiset et al identified two patterns of spinal TB, namely tuberculous spondylodiscitis (SPD) or classic Potts disease, and tuberculous spondylitis without disc involvement (SPwD).[18] Involvement of the disc space was a common finding observed in the study (84.6% incidence), which varied from pure signal intensity changes suggesting inflammatory edema (SPwD), to partial reduction of disc height, to complete destruction by granulation tissue (SPD). A high incidence of disc involvement has been otherwise noted, ranging from 72.4% - 100%.[16,29,30] Demographically, since TB affects the younger population more frequently, disc involvement is seen frequently in spinal TB because it is more vascularized at the younger age.[23] In older age group, disc involvement occurs secondarily following vertebral body destruction.

End plate erosion (69.2% incidence) was more common than complete end plate destruction (46.1% incidence). The relative avascularity of the end plates offers resistance to invasion and destruction by tuberculous granulation tissue. Secondarily, inability of the tuberculous bacilli to produce proteolyic enzymes confers an additional barrier to end plate penetration.[31] Liu et al reported a similar high incidence of end plate erosion in 75.8% patients,[29] whereas Smith et al reported end plate involvement in one out of the four patients in their study.[24]

'Cold abscess' of TB, with spread into pre and paravertebral space, commonly accompanies the vertebral lesion. Our study and earlier studies, demonstrate an incidence of paraspinal abscesses, on MRI, in 55%-100% patients[4,24,32,33,34] with Jain et al reporting a 100% incidence.[4] The abscess was seen tracking along one of three planes namely anterior subligamentous, epidural and along the psoas muscle. Subligamentous abscess, with spread anterior to the vertebral body below the anterior longitudinal ligament, was the most commonly seen (70% patients). This is described as a characteristic MRI finding of tuberculous spine, with various authors reporting the incidence of 58%-93%.[29,35] Epidural spread of the abscess posterior to vertebral body in the neural canal, has been demonstrated by MRI in 45%-93% patients.[4,18,30,36] Psoas abscess was seen in the patients with dorsolumbar junction and lumbar vertebral involvement (most commonly D12, L1 & L2 vertebrae) corresponding to the site of origin of the psoas muscle from the transeverse process of D12 to L5. Jain et al observed psoas abscess in 18.4% patients.[4]

Canal stenosis ranging from 10% - 90% was noted in all patients. However, only 61.5% patients manifested with neurological impairment; with patients with neurodeficit having greater mean canal stenosis as compared to those without (41.6% vs 29.6%). Jain et al reported canal stenosis in 75.5% patients,[4] while Liu et al noted the same in 93.3% patients.[29] Neural compression beyond a critical level is necessary for impairment of spinal cord function. The critical level is determined by the status of the cord, its vascularity and tolerability to hypoxia, neuronal plasticity, surrounding bony stiffness and the rate of canal occlusion. Neurological deficit is a late presentation of spinal TB, usually presented only once canal encroachment exceeds 60%-76%.[17,21]

Marrow hyperintensity was noted in all (100%) patients and in all the affected vertebral levels. Sharif et al[37] and Jain et al[21] have reported 100% marrow edema in patients of spinal TB. Marrow signal changes represent either a reactive edema due to the surrounding

inflammatory process or a primary osteitis of the vertebral body. The differentiation between the two can definitively be established by biopsy sampling of the affected vertebral body. The reactive edema would effectively subside on treatment of the primary focus, whereas the primary osteitis, being pathologically involved in the tuberculous process would entail definitive treatment at that vertebral level. Altered cord intensity with hyperintensity on T2W images was noted in 61.5% patients, suggestive of reactive cord edema. Jain et al[4] reported cord changes in 22.4% patients. Cord edema is a predictor of good neurological recovery in patients with chemotherapy, whilst severe cord changes like cord thinning, myelomalacia, arachnoiditis and syringomyelia are poor prognostic factors for neurological recovery.[4,14]

All (100%) patients had marrow edema and canal stenosis of varying degree. Central vertebral body involvement, intervertebral disc signal intensity changes, marrow edema and canal stenosis were together observed in 84.6% patients. Liu et al. concluded that a combination of contiguous involvement of ten vertebral bodies, subligamentous spread, endplate erosions, hypointense T1W lesion and hyperintense T2W lesion, were suggestive of TB.[29] Jain et al noted that marrow oedema, pre and paravertebral septate loculated collections, subligamentous collections and endplate erosions with epidural extension are among the most consistent findings in tubercular spondylodiscitis seen on MRI.[4]

CONCLUSION

MRI is an indispensible modality of primary investigation in suspected patients of spinal TB, which aids in early, rapid, noninvasive and accurate confirmation of the diagnosis of spinal TB. Amidst the multitude findings seen, canal stenosis (100%), marrow edema (100%), central vertebral body involvement (84.6%), intervertebral disc intensity changes (84.6%) and presence of a soft tissue abscess (76.9%) are the most consistent findings seen on MRI in spinal TB. The constellation of these findings on an MRI would serve as a clinician's tool for rapid diagnosis of spinal TB.

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