Atypical Imaging Features of Tuberculous Spondylitis: Case Report with Literature Review

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ABSTRACT

Spinal tuberculosis in its typical form that shows destruction of two adjacent vertebral bodies and opposing end plates, destruction of the intervening intervertebral disc and a paravertebral or psoas abscess, is easily recognized and readily treated. Atypical tuberculous spondylitis without the above mentioned imaging features, although seen infrequently, has been well documented. We present, in this report, a case of atypical tuberculous spondylitis showing involvement of contiguous lower dorsal vertebral bodies and posterior elements with paravertebral and epidural abscess but with preserved intervertebral discs. The patient presented in advanced stage with progressive severe neurological symptoms due to spinal cord compression. Non-enhanced magnetic resonance imaging led to misdiagnosis of the lesion as a neoplastic process. It was followed by contrast enhanced computed tomography of the chest and abdomen that raised the possibility of an infectious process and, post-operatively, histopathological examination of the operative specimen confirmed tuberculosis. This case indicates the difficulty in differentiating atypical spinal tuberculosis from other diseases causing spinal cord compression. The different forms of atypical tuberculous spondylitis reported in the literature are reviewed. The role of the radiologist in tuberculous spondylitis is not only to recognize the imaging characteristics of the disease by best imaging modality, which is contrast enhanced magnetic resonance imaging, but also to be alert to the more atypical presentations to ensure early diagnosis and prompt treatment to prevent complications. However, when neither clinical examination nor magnetic resonance imaging findings are reliable in differentiating spinal infection from one another and from neoplasm, adequate biopsy, either imaging guided or surgical biopsy is essential for early diagnosis.

CASE REPORT

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Presentation and Examination

A 30 year old female presented to the emergency department with low back pain and bilateral lower limb numbness for 1 month duration. She was pregnant at 37 weeks of gestation. She had no history of trauma, no complaint of fever or weakness and no bladder or bowel dysfunction. On examination, the patient's vital signs were stable, power grade 5/5 in the right lower limb and 4+/5 in the left lower limb, sensation decreased bilaterally, and normal reflexes on both sides. She was admitted for investigations and further evaluation by magnetic resonance imaging (MRI) of the thoraco-lumbar spine. The patient left the hospital against

medical advice and was re-admitted after about 2 weeks while in labour. During that period she developed progressive weakness of the lower limbs and was unable to walk without support with bowel and bladder dysfunction. Examination showed brisk reflexes bilaterally with sensory loss in the lower limbs (sensory level of T10).

Imaging Findings

After normal vaginal delivery of a healthy baby, MRI of the thoraco-lumbar spine was done and revealed lobulated pre- and para- vertebral lesion extending from T7 level down to T12 level with involvement of the vertebral bodies and the posterior elements, associated with posterior epidural extension through involved vertebral body as well as bilateral intraspinal extensions through the intervertebral foramina, forming epidural lesions causing severe degree of spinal cord compression, most severe at T10 level. The lesion showed heterogeneous low signal intensity in T1 weighted images and heterogeneous mixed high and low signal intensity in T2 weighted images. The intervertebral discs at the involved levels showed normal signal intensity. The spinal cord showed mild increase in T2 signal intensity in the compressed levels as well as in the segments proximal and distal to the compressed levels, consistent with cord oedema with no evident myelomalacia. There was also bilateral pleural effusion and bilateral pleural based rounded soft tissue lesions (Figures 1, 2).

The MRI scan was incomplete as the patient was uncomfortable during the scanning partly because of mild claustrophobia and partly due to her clinical condition. She refused to receive contrast injection and refused to continue the scan. According to the non-enhanced MRI findings, a neoplastic lesion was suggested, most likely lymphoma.

The MRI was followed by contrast enhanced computed tomography (CT) of the chest and abdomen that revealed rim enhancement of the vertebral and paravertebral lesions which raised the possibility of an infectious process (Figure 3). CT scan confirmed bony destruction involving the posterior elements of the involved vertebrae (Figures 4, 5). CT of the chest showed bilateral pleural effusion with irregular lobulated outline suggesting empyema (Figure 6). There was no sign of an abdominal lesion.

Laboratory investigations

Blood investigations showed hypochromic microcytic anaemia with high erythrocyte sedimentation rate (ESR) (90mm/hr), high C-reactive protein (69.5mg/l), normal renal and liver function tests. Negative for human immune deficiency viral infection (HIV).

Management

After reviewing the MRI and CT findings by the spine team in the Orthopaedic department, and considering the progressive neurological symptoms, surgery was decided through posterolateral approach for transfacetal excision of the vertebral lesions, with decompression laminectomy and stabilization of the spine from T5 down to L1 level with bone grafting (Figure 7).

Pathological Examination: Post operative histopathology of the resected tissues revealed fibro-collagenous tissue with numerous epitheloid granulomata mixed with Langhan's giant cells and lymphoplasmacytes. Extensive areas of caseation necrosis were seen. Ziehl-Neelsen stain demonstrated acid fast bacilli (AFB). The final diagnosis was caseating granulomatous tuberculous lesion (Figure 8).

Post-operative Medication and Follow up: The patient was put on a combination of anti-tuberculous medication and discharged in an improved state to continue medication for a total of 1 year. The anti-tuberculous medication included; a combination of Isoniazid (INH) 300mg/ day, Ethambutol (ETB)1600mg/ day, Rifampicin (RMP) 600mg/ day, Pyrazinamide (PZN) 2000mg/ day for 2 months. However, tuberculosis (TB) culture with antibiotic susceptibility showed bacterial resistance to PZN and sensitivity to other drugs. The treatment protocol was modified accordingly and a combination of INH, RMP and ETB was continued for 4 months (instead of the usual protocol of continuing medication by RMP and INH). During the remaining 6 months the patient received a combination of INH and RMP. During this year the patient was followed up regularly and she was symptom free.

DISCUSSION

TB involves both pulmonary and extrapulmonary sites. Skeletal involvement is reported in 1-5% of all TB patients [1-3]. TB is the most common cause of vertebral body infection [4].

Although the first documented spinal TB cases date back to 5000-year-old Egyptian mummies, the first modern case of spinal TB was described in 1779 by an English surgeon Percival Pott (1714-1788) [1, 3, 5, 6].

Spinal TB (Pott's disease) is the most common as well as the most dangerous form of musculo-skeletal TB and accounts for 50% of all cases of skeletal TB [1-3, 5, 7-13]. Any part of the spine can be affected but the thoraco-lumbar junction is the most common site of involvement [1-3, 5, 7-11, 14].

Tuberculous spondylitis usually has an insidious onset of back pain and stiffness with local tenderness. In later stages of the disease, severe spinal deformity due to acute kyphotic angulation occurs. Neurological symptoms and complications may occur in the acute and late stages of the disease. Males are affected more commonly than females with reported male to female ratio of 4:3, and reported average age of 50 years [2, 3, 11]. The form of the tuberculous spondylitis without discitis, described in this case report, is reported to occur in younger patients with an average age of 40 years and predilection to occur in the countries of the sub-Saharan Africa [2, 3].

Etiology

Spinal involvement is usually a result of haematogeneous spread of *Mycobacterium tuberculosis* into the dense vasculature of the cancellous bone of the vertebral bodies. The primary infection site is either a pulmonary focus or other

extraosseous foci such as lymph nodes, gastrointestinal, or any other viscera [5, 9]. Pulmonary TB is not detected in about 50% of spinal TB cases [3].

Predisposing Factors: Conditions and factors predisposing for spinal TB include; poverty, overcrowding, illiteracy, malnutrition, alcoholism, drug abuse, diabetes mellitus, immunosuppressive treatment, chronic peritoneal dialysis, previous tuberculous infection and HIV infection [5].

Tuberculosis and Pregnancy: It is reported that pregnancy associated biological and immunological changes can affect TB epidemiology. In 2012, Mathad et al [15] and Zenner et al [16] reported that the incidence of TB diagnosis is significantly increased postpartum, while there was no similar increase during pregnancy. However, the postpartum incidence may reflect an increase during pregnancy, given the diagnostic and immunological delays. In consistence with our reported case of a tuberculous pregnant patient, who showed no pregnancy related complications, Tripathy (2003) stated that there were no statistical differences in duration of gestation, preterm labor, and other complications of pregnancy, labor, and puerperium between pregnant women with TB and pregnant women without TB [17], but other studies reported that pregnant women with pulmonary or extrapulmonary TB, other than lymphadenitis, have increased risk of complications including antenatal hospitalization and miscarriages [15,18].

Imaging Findings

Classical Spinal TB

Spinal TB in its classical and most common form is easily recognized and diagnosed. The classical form is a vertebral body disease with intervertebral disc involvement (spondylodiscitis). Typically more than one (up to 10) adjacent vertebral bodies are involved with predilection for the anterior subchondral bone followed by involvement and destruction of the intervening intervertebral discs. Reactive sclerosis is typically absent. Vertebral body collapse and progressive kyphosis then follows. Extension of the tuberculous process posteriorly into the spinal canal forms epidural abscess resulting in neurological complications. Subligamentous extension of a tuberculous abscess can be seen as erosions of the anterior surface of the vertebral bodies distant from the primary infection site. More frequently detected extension is anterolaterally, forming para-vertebral and/ or psoas abscess. Paravertebral abscesses form early and are easily seen in the thoracic region as posterior mediastinal masses. Paravertebral psoas abscess can extend to the groin and thigh. A healed psoas abscess may calcify [1-3, 5, 7-11, 14, 19, 20].

Atypical spinal TB

Any tuberculous vertebral lesion, which does not have the classical/ typical features mentioned above, is referred to as atypical spinal TB [2, 8].

Increasingly more common atypical spinal TB is in the form of spondylitis without discal involvement, showing multifocal vertebral involvement without associated disc destruction. The infection typically commences at the superior or inferior vertebral end-plates anteriorly then extends by subligamentous extension over multiple vertebral segments [1-3, 8, 9]. MRI shows subligamentous abscess and abnormal signal involving multiple vertebral segments with preserved discs [2, 8], these features are consistent with our presented case. Similar case finding is reported by Sarangapani et al (2008) in a young patient that was misdiagnosed as a metastatic disease [14]. This form of atypical spinal TB is associated with more frequent extraspinal skeletal involvement [3].

Review of Literature

Different forms of atypical spinal TB are described in the literature, and the importance of these lesions is that they are difficult to differentiate clinically and radiographically from a neoplastic process [8, 14].

A central vertebral body lesion is the other atypical spinal TB form [2, 8, 20], in which vertebral collapse can occur producing a vertebra plana appearance [5, 8, 9], seen mostly in children [3]. MRI shows signal abnormalities within the vertebral body with preserved disc, and this lesion is indistinguishable from lymphoma or metastasis [8, 9, 20].

This central form of atypical spinal TB can be as a single or multiple vertebral involvement. Single vertebral TB is exceedingly rare. Zhen et al (2012) reported 5 cases in young patients showing solitary local osteolytic vertebral lesion in the lumbar spine [20]. Differentiation of a focal single tuberculous lesion from other causes of focal vertebral destruction, as focal infection, osteoblastoma and eosinophilic granuloma, is usually difficult on the basis of imaging alone, and a definite diagnosis can be achieved by CT guided biopsy or surgical intervention [8, 20].

Multi-vertebral lesions may be in continuity or may affect different levels in different regions of the spine (skip lesions) [5, 8, 9]. Skip lesions are mostly reported in an incidence of about 10% of spinal TB cases [5, 8, 11]. However, Polley et al (2009) reported a higher incidence (16.3%) of atypical skip spinal TB lesions, and stated that those patients are more prone to develop neurological complications than the typical cases [21].

It is, therefore, extremely important when investigating a spinal TB patient, that whole spine sagittal MRI sequence is done to identify non-contiguous (skip) lesions prior to surgical planning [10, 21]. This form of spinal TB shows higher incidence in areas with high prevalence of TB and it is reported to be a manifestation of HIV infection, multi-drug resistant TB (MDR-TB) or chronicity [21].

Khattry et al (2007) reported a case of spinal TB in an adolescent misdiagnosed as malignancy, lymphoma or metastatic disease, in view of skip lesions of the vertebral bodies and pedicles, contiguous and non-contiguous, with sparing of the discs. They stated that the skip multifocal spine lesions with disc sparing and absence of paravertebral lesion favors a neoplastic lesion while the presence of paravertebral lesion/ collection favors the possibility of a tuberculous pathology [8].

Another rare atypical spinal TB pattern is the isolated involvement of the posterior elements of the vertebrae [2, 8]. It

is reported that this occurs characteristically in TB and is not found in pyogenic spondylitis [2, 19]. Involvement of the posterior elements can be isolated or in combination with vertebral body lesions [2], as described in our reported case. Sinan et al (2004) reported 10% of their spinal TB cases having isolated posterior arch involvement [11].

A rare case of tuberculous vertebral infection is described in the literature involving the vertebral transverse process (spondylitis tuberculosa lateralis). The rarity of involvement of the vertebral posterior elements, which is best detected by CT, can cause delayed diagnosis and can lead to neurological deficit in high proportion of cases [13].

Ahmadi et al (1993) reported multiple cases with atypical MRI features of spinal TB that were suggestive of a neoplasm rather than infection, including; a single vertebral body lesion, a single spinous process lesion and a sacral lesion with multiple lower lumbar vertebral body lesions with preserved discs. They concluded that neither clinical examination nor MRI findings may be reliable in helping to differentiate spinal infections from one another or from neoplasm, and adequate biopsy is essential for early diagnosis and prompt treatment [19].

Yalniz et al (2000) reported low incidence of atypical spinal TB cases (2.1%), including; skip vertebral lesions, isolated spinous process lesion and neural arch lesions [13].

Furthermore, atypical intra-spinal TB has been documented in the literature. Tanriverdi et al (2003) reported 3 cases; intramedullary abscess, tuberculous arachnoiditis and tuberculous arachnoiditis with syrinx [4]. Yu et al (2013) reported a case of an extradural mass lesion (granulomatous mass with caseation necrosis) causing spinal cord compression that was mistaken for neoplasia. Therefore, in cases presenting with an intraspinal mass lesion, the possibility of a tuberculous abscess and/ or a granuolma should be considered in the differential diagnosis [22].

Recently, Thammaroj et al (2014) reported 5 MRI findings of unusual/atypical spinal TB including; multiple skip lesions, isolated vertebral body lesion, lamina and spinous process lesions, single vertebral collapse and intramedullary tuberculoma. The skip lesions type was the most common atypical pattern in their series with an incidence of 11.5% [12].

Diagnosis

Advanced imaging techniques, especially MRI, make the early diagnosis of spinal TB easier and the patients are treated earlier and more effectively before significant neurological deficit develop. However, patients can still present late with considerable spine deformity and neurological complications [1, 6, 7, 10, 11], as in the case in this report.

Both CT and MRI are helpful in the diagnosis of the tuberculous spondylitis. CT remains the preferred imaging modality for the assessment of osteomyelitis, showing bony destruction earlier. Granulation tissue is seen as a high attenuation lesion and an abscess or caseous tissue is seen as low attenuation lesion with rim enhancement. Chronicity with slowly progressive bone destruction, absence of sclerosis, presence of paravertebral abscess with calcification are considered to be signs of TB, with preserved discs and isolated posterior element lesions in the atypical cases. CT is less accurate than MRI in defining the epidural extension of the disease and can underestimate compression of the neural structures [1, 2, 5, 11, 23].

On MRI, the vertebral changes (intraosseous abscesses), paravertebral abscesses, discitis, scoliosis and kyphosis, skip lesions, spinal canal encroachment, and nerve root distortion are all readily detected on routine sagittal, axial and coronal images using T1- and T2- weighted sequences in addition to short tau inversion recovery (STIR) sequence, followed by contrast-enhanced sequences after intravenous administration of gadolinium contrast agent. The MRI features can be non-specific; T1-weighted images showing decreased signal within the affected vertebral marrow and T2-weighted images showing relative increase in signal within the diseased tissues [1, 5, 7, 11, 23]. However, it is reported that the characteristic MRI finding in spinal TB is increased signal in T2-weighted images in the diseased disc, if involved, and areas of decreased signal in the caseous granulomatous vertebral lesions with adjacent areas of high signal due to marrow oedema or intraosseous abscess resulting in mixed heterogeneous T2 signal intensity [1, 2, 20].

The high contrast resolution of MRI greatly improves the detection of intraosseous abscesses, subligamental spread and epidural extension of infection, with evaluation of the extent of the cord compression and the presence of an intramedullary lesion. Enhanced MRI studies are particularly useful for demonstrating heterogeneous vertebral body enhancement and enhancement of the inflamed meninges, as well as rim enhancement around intraosseous, epidural and paravertebral abscesses [1, 2, 5, 7, 9, 11, 23, 24]. Smooth rim of enhancement of the paravertebral and intraosseous abscesses is considered diagnostic for TB [10].

Although our presented case showed many MRI features of spinal TB, but the absence of contrast enhanced MRI sequences led to misdiagnosis. Atypical cases can show disc preservation with lack of enhancement, and posterior element lesions [1, 2]. Generally, the sensitivity and specificity of MRI for the diagnosis of spinal TB is 100% and 88.2% respectively [23].

Differential Diagnosis

A high degree of clinical suspicion and familiarity with the various radiological manifestations of spinal TB, both the typical and the atypical cases, allows early diagnosis and timely initiation of appropriate therapy, thereby reducing patient morbidity [7].

The differential diagnosis of the presented case of tuberculous spondylitis without discitis includes; pyogenic spondylitis, brucellar spondylitis, metastatic and primary bony tumors (as lymphoma) and osteoporotic vertebral fractures [5, 7, 9, 23].

Pyogenic spondylo-discitis can be distinguished from spinal TB by rapid bone and disc destruction. The earliest radiographic sign is decreased disc height with indistinct vertebral end-plates, then vertebral body destruction followed by sclerosis. CT will show additional small paravertebral inflammatory mass and epidural extension [3]. MRI shows high T2 signal in the involved disc and homogeneous alteration in signal in adjacent vertebral bodies; high T2 signal and low T1 signal with homogeneous enhancement. There is irregular ill-defined intense enhancement of the paravertebral inflammatory lesion/ abscess. Unlike spinal TB, there is preserved posterior elements, absent kyphotic deformity and absent calcification in the paravertebral abscesses. Pyogenic spondylitis can be found at any age and usually the lumbar or the cervical spine is affected as a single segmental lesion with no skip lesions [1, 3, 5, 23-25].

Brucellar spondylitis is commonly found in middle-age and elderly patients. The lumbar spine is most frequently involved but skip lesions are reported. Bone destruction occurs at the disco-vertebral junction, especially at the anterior aspect of the superior end-plate with disc involvement that shows small amount of gas. Whole vertebral end-plate and body can be destructed with sclerosis and anterior osteophyte formation (parrot beak osteophytes). Posterior vertebral elements are usually not involved. The paraspinal soft tissue planes are obliterated due to small paraspinal lesion with no or minimal epidural extension. Unlike spinal TB, vertebral collapse, kyphotic deformity, large paraspinal/ psoas abscess, and neurological deficits are rarely found in brucellar spondylitis [3, 5, 25-28]. MRI shows signal changes in the involved vertebral bodies and discs; high T2 signal and low T1 signal with enhancement. Enhancement of the facet joints is also considered a specific sign of brucellar spondylitis [27, 28].

In neoplastic involvement of the spine, the disc spaces are usually spared and paravertebral masses are not seen except when solid extraosseous soft tissue component is associated with destructed vertebral bodies [8]. The presence of an intraosseous and paravertebral abscess with bone fragments differentiates atypical spinal TB cases, with normal disc height, from neoplasia [1, 23]. Skip or multifocal contiguous involvement of the spine favors neoplastic lesion [8]. In Lymphoma, spinal involvement may manifest as paraspinal, vertebral, and epidural involvement, either in isolation or in combination. As in spinal TB, the posterior vertebral elements can be involved, compressed vertebrae can occur with epidural extension causing cord compression. The radiographic appearance is variable, most commonly are osteolytic lesions while mixed osteolytic-osteosclerotic or purely osteosclerotic lesions are rare. On MRI, lymphomatous infiltration appears as focal or diffuse areas of variable signal, mostly high T2 signal, high signal on STIR sequence and low T1 signal with diffuse heterogeneous enhancement. The appearance of vertebral lymphoma at CT and MR imaging can be non-specific, however, lesions showing bone marrow replacement and a surrounding soft-tissue mass without large areas of cortical bone destruction suggest lymphoma [29-31].

In metastatic disease, the thoracic region is most commonly involved. The posterior wall of the vertebral bodies, pedicles and lamina are involved in metastatic disease, however, the intervertebral discs are preserved. In elderly patients with vertebral collapse, metastatic disease of the spine should always be considered [5, 32]. Radiographs can detect lytic vertebral lesions in most cases with eroded pedicles. Some lesions can be mixed lytic-sclerotic or sclerotic. CT detects cortical destruction with an epidural mass presenting as amorphous soft tissue displacing the thecal sac or filling the neural foramen. Metastatic lesions usually show low T1 signal and high signal in T2 and STIR sequences and are brightly diffusely enhancing in post-contrast T1 fat saturation sequences. Sclerotic lesions are of low signal in all sequences. Metastases often, but not consistently, have a rim of bright T2 signal around the lesions; a halo sign [32].

In osteoporotic spinal fractures, the mid thoracic and thoraco-lumbar regions are frequently involved. Radiographically, there is reduced bone density in osteoporotic spine. Multiple vertebrae can be involved in osteoporotic compression fractures, showing decreased height and wedging with progressive kyphosis. CT will show associated posterior vertebral wall fracture, presence of retropulsed fragment and suspected pedicle fracture. MRI will show low T1 and high STIR signal within fractured vertebrae with normal signal within the posterior elements and no associated encasing epidural or paraspinal mass. Compressed vertebrae may show mild enhancement. MR-fluid sign is reported to be a characteristic of an acute/ subacute osteoporotic vertebral fracture, which is seen as a high T2 linear signal due to fracture related oedema [32, 33].

Recently, diffusion-weighted MRI (DW-MRI) and apparent diffusion coefficient (ADC) values are being used in musculoskeletal diseases, as for differentiating spinal TB from other lesions [5, 34]. It is reported that in the acute stage of spinal infection, the involved bone shows a hyperintense signal as compared with normal surrounding bone marrow on DW-MRI, followed by decreased signal in the chronic stages [35]. The ADC value of an area with spinal infection is reported to be higher than the ADC value of a normal area of bone marrow. Bone marrow edema causes a local increase in water movement, resulting in increased local diffusion (no restriction), that is expressed by high ADC values of the lesions [5, 34].

As in chronic spinal infections, benign vertebral compression fractures are hypo- to isointense in DW-MRI, relative to adjacent normal vertebral bodies, with increased ADC values in traumatized bone marrow compared with ADC values of normal bone [32, 34, 36].

Malignant spinal lesions, due to increased cellularity and replacement of the fatty marrow by tumor cells, show restricted diffusion of free water molecules, and accordingly high signal in DW-MRI and low ADC values [36]. But it is reported that the utility of DW-MRI and ADC values to differentiate tuberculous vertebral body involvement from malignant spinal lesions is controversial [32, 36]. The ADC values in TB can show an overlap with those of malignant/ metastatic disease. Therefore, DW-MRI and ADC values should always be interpreted in association with clinical history and conventional MRI findings [5, 36].

Differentiating spinal TB from pyogenic spondylitis as well as from primary and metastatic spinal tumors may be difficult when only clinical and radiographic findings are considered. A history of TB, a positive skin test, and an elevated ESR may be useful in the diagnosis of spinal TB [1, 23, 37]. Bone tissue or abscess samples are obtained to stain for AFB and isolate organisms for culture, antibiotic sensitivity, and histopathology. The method widely used is CT guided needle biopsy and/ or aspiration or surgical biopsy [1, 5].

Complications

Neurological deficit and deformity are the worst complications of spinal TB [23], with an incidence of 10-43% [1, 6]. Paraplegia and sometimes quadriplegia are serious complications of spinal TB, seen in approximately 10% of patients. Copious epidural pus and granulation tissue alone or in combination with vertebral collapse, subluxation, or dislocation produce cord compression. Rarely, the pus penetrates the dura resulting in severe meningomyelitis [3, 5, 9].

It is reported that patients with atypical spinal TB, as having skip lesions or posterior element involvement, are more prone to develop neurological complications and can be attributed to delayed diagnosis [13, 21]. Early recognition and prompt treatment are therefore necessary to minimize residual spinal deformity and/ or permanent neurological deficit [1, 11].

Management

For decision making and management of spinal TB, patients can be broadly classified as two groups; those with neurologic complications and those without [23]. In patients without neurologic deficit, medical therapy is the treatment of choice and surgical intervention may be needed in relatively few cases. In cases with neurologic complications, medical therapy is the first choice again but, when indicated, combination of medical and surgical treatments yield the best results [38].

The treatment principles for patients with atypical spinal TB are similar to typical cases. It has been suggested that these lesions can have the same outcome and prognosis as typical spinal TB if they are diagnosed and treated in the early stages [1].

Indications for medical treatment: The organism identified, antibiotic sensitivity, single disc space involvement without significant vertebral body destruction, minimal or no spinal instability, minimal or no neurologic deficit and the presence of medical co-morbidities such as sepsis or coagulopathy [1].

Indications for surgery: Tissue sampling when the diagnosis is doubtful, persistent or deteriorating neurologic deficits in spite of anti-tuberculous treatment, recurrent neurologic complications, drainage of an abscess in the cervical region, drainage of a large paravertebral abscess, spinal instability, and severe kyphotic deformity and MDR-TB. Children may need earlier surgical intervention compared to adults due to their growth potential in order to prevent kyphotic deformity [1, 5, 23, 39-41].

If surgical treatment is indicated, delay can cause severe kyphosis, leading to respiratory system dysfunction, painful costopelvic impingement, and paraplegia [1, 39].

The posterolateral or transpedicular approach has been used extensively for the management of spinal TB. This approach is a viable and importantly safe surgical option for ventral decompression in the thoracic spinal TB when followed by anti-tuberculous treatment [1, 23]. This type of surgical approach was done for the presented case.

Prognosis and Outcome of Spinal TB

Effective medical and surgical management of spinal TB has improved the outcome of these patients significantly, even in the presence of neurologic deficits and spinal deformities. Neurologic complications due to spinal TB seem to be "relatively benign" if early adequate medical and surgical managements are employed. A significant proportion of patients with severe motor deficits experience remarkable improvement after surgical decompression, therefore, those patients should undergo surgery even though they may be suffering from paraplegia for a considerable duration [5, 6].

Younger age and radical surgery in conjunction with anti-tuberculous therapy have been suggested as favorable prognostic factors [1].

It is reported that if diagnosis is made at pre-destructive stage and the patient is treated by standard drugs, the infection would heal in about 95 % patients without significant deformities and complications. Diagnosis and treatment at early stages would resolve the neurological deficits without operation in about 40 % of cases. Nearly 60 % of patients would require to be operatively decompressed without jeopardizing mechanical stability [42].

Using multidrug therapy, the recurrence rate for skeletal TB is approximately 2%. Long-term multidrug anti-tuberculous regimens will likely reduce the relapse rate of spinal TB [1].

TEACHING POINT

Atypical spinal TB is best evaluated with MRI, and its reported forms include; spondylitis without discitis, central single vertebral body lesion, non-contiguous (skip) vertebral lesions, isolated involvement of the posterior vertebral elements and isolated intraspinal lesions. If the clinical and imaging findings are not reliable in helping differentiate spinal infections from one another or from neoplasm, especially with the atypical presentations, then image guided biopsy is essential for early diagnosis and prompt treatment.

REFERENCES

1. Rasouli MR, Mirkoohi M, Vaccaro AR, Yarandi KK, Rahimi-Movaghar V. Spinal tuberculosis: Diagnosis and management. Asian Spine Journal 2012; 6(4): 294-308. PMCID: PMC 3530707. PMID: 23275816.

2. DeBacker AI, Mortele KJ, Vanschoubroeck IJ, Deeren D, Vanhoenacker FM, De Keulenaer BL, et al. Tuberculosis of the spine. CT and MRI imaging features. JBR-BTR 2005; 88(2): 92-97. PMID: 15906583.

3. Dahnert W(ed). Radiology Review Manual, 6th ed. Philadelphia: Lippincott Williams and Wilkins 2007. ISBN-13: 978-0-7817-6620-3. ISBN-10: 0-7817-6620-6.

4. Tanriverdi T, Kizilkilic O, Hanci M, Kaynar MY, Unalan H, Oz B. Atypical Intradural spinal tuberculosis: Report of three cases. Spinal Cord 2003; 41(7): 403-409. PMID: 12815372.

5. Ansari S, Amanullah MF, Ahmad K, Rauniyar RK. Pott's spine: Diagnostic imaging modalities and technology advancements. NAmJ Med Sci 2013; 5(7): 404-411. PMCID: PMC 3759066. PMID: 24020048.

6. Sai Kiran NA, Vaishya S, Kale SS, Sharma BS, Mahapatra AK. Surgical results in patients with tuberculosis of the spine and severe lower extremity motor deficits: A retrospective study of 48 patients. J Neurosurg Spine 2007; 6(4): 320-326. PMID: 17436920.

7. Harisinghani MG, McLoud TC, Shepard JO, Ko JP, Shroff MM, Mueller PR. Tuberculosis from head to toe. Radiographics 2000; 20(2): 449-470. PMID: 10715343.

8. Khattry N, Thulkar S, Das A, Alan Khan S, Bakhshi S. Spinal tuberculosis mimicking malignancy: Atypical imaging features. Indian J Paediatr 2007; 74(3): 297-298. PMID: 17401273.

9. Moorthy S, Prabhu NK. Spectrum of MR imaging findings in spinal tuberculosis. AJR 2002; 179 (4): 979-983. PMID: 12239050.

10. Shanley DJ. Tuberculosis of the spine: Imaging features. AJR 1995; 164(3): 659-664. PMID: 7863889.

11. Sinan T, Al-Khawari H, Ismail M, Ben-Nakhi A, Sheikh M. Spinal tuberculosis: CT and MRI features. Ann Saudi Med 2004; 24(6): 437-441. PMID: 15646161.

12. Thammaroj J, Kitkhuandee A, Sawanyawisuth K, Chowchuan P, Promon K. MRI findings in spinal tuberculosis in an endemic country. J Med Imaging Radiat Oncol 2014; 58(3): 267-276. PMID: 24529160.

13. Yalniz E, Pekindil G, Aktas S. Atypical tuberculosis of the spine. Yonsei Med J 2000; 41(5): 657-661. PMID: 11079627.

14. Sarangapani A, Fallah A, Provias J, Jha NK. Atypical presentation of spinal tuberculosis. Can J Surg 2008; 51(6): E121. PMCID: PMC 2592567. PMID: 19057720.

15. Mathad JS, Gupta A. Tuberculosis in pregnancy and postpartum women: Epidemiology, management and research gaps. Clin Infect Dis 2012; 55(11):1532-1549. PMCID: PMC 3491857. PMID: 22942202.

16. Zenner D, Kruijshaar ME, Andrews N, Abubakar I. Risk of tuberculosis in pregnancy: A national, primary care-based cohort and self controlled case series study. Am j Respir Crit Care Med 2012; 185(7): 779-784. PMID: 22161161.

17. Tripathy SN. Tuberculosis and pregnancy. Int J Gynaecol Obstet 2003; 80(3): 247-253. PMID: 12628525.

18. Ormerod P. Tuberculosis in pregnancy and the puerperium. Thorax 2001; 56: 494-499. PMC: 1746062. PMID: 11359968.

19. Ahmadi J, Bajaj A, Destian B, Segall HD, Zee CS. Spinal tuberculosis: Atypical observations at MRI imaging. Radiology 1993; 189(2): 489-493. PMID: 8210378.

20. Zhen P, Li XS, Lu H. Tuberculosis of a single lumbar vertebra presenting with a solitary local osteolytic lesion in young adults: A report of 5 cases. J Spine 2012; 1(2): 112. DOI: 10.4172/2165-7939.1000112.

21. Polley P, Dunn R. Non-contiguous spinal tuberculosis: Incidence and management. Eur Spine J 2009; 18(8):1096-1101. PMCID: PMC 2899513. PMID: 19357878.

22. Yu Y, Wang X, Du B, Yuan W, Ni B, Chen D. Isolated atypical spinal tuberculosis mistaken for neoplasia: Case Report and literature review. Eur Spine J 2013; 22(3): S302-305. PMCID: PMC 3641247. PMID: 22531896.

23. Jain AK. Tuberculosis of the spine. A fresh look at an old disease. J Bone Joint Surg Br 2010; 92-b (7): 905-913. PMID: 20595106.

24. Conzaga de Souza C, Gasparetto EL, Marchiori E, Valle Bahia PR. Pyogenic and tuberculous discitis: MRI findings for differential diagnosis. Radiol Bras 2013; 46(3): 173-177. DOI: 10.1590/S0100-39842013000300012.

25. Gouliouris T, Aliyu SH, Brown NM. Spondylodiscitis: update on diagnosis and management. J Antimicrob Chemother 2010; 65(supp3): iii11-iii24. PMID: 20876624.

26. Gokhale YA, Ambardekar AG, Bhasin A, patil M,TilluA,Kamath J. Brucella spondylitis and sacroiliitis in the general population in Mumbai. J Assoc Physicians India 2003; 51: 659-666. PMID: 14621032.

27. Pourbagher A, Pourbagher MA, Savas L, Turunc T, DemirogluYZ, Erol L,Yalcintas D. Epidemiologic, clinical and imaging findings in Brucellosis patients with osteoarticular involvement. AJR 2006; 187(4): 873-880. PMID: 16985128.

28. Ozaksoy D,Yucesoy K, Yucesoy M, Kovanlikaya I,Yuce A, Naderi S. Brucellar spondylitis: MRI findings. Eur Spine J 2001; 10(6): 529-533. PMCID: PMC 3611537. PMID: 11806395.

29. Rodallec MH, Feydy A, Larousserie F, Anract P, Campagna R, Babinet A, et al. Diagnostic imaging of solitary tumors of the spine: What to do and say. Radiographics 2008; 28(4): 1019-1041. PMID: 18635627.

30. Abdel Razek AAK, Castillo M. Imaging of primary bone tumors and pseudotumors of the spine. J Neuroradiol 2010; 37(1): 37-50. PMID: 19781780.

31. Rahmat K, Wastie ML, Abdullah BJJ. Primary bone lymphoma: report of a case with multifocal skeletal involvement. Biomed Imaging Interv J 2007; 3(4): e52. PMID: 21614300.

32. Shah LM, Salzman KL. Imaging of spinal metastatic disease. Int J Surg Oncol 2011; 2011: 769753. PMCID: PMC 3263660. PMID: 22312523.

33. Kondo KL. Osteoporotic vertebral compression fractures and vertebral augmentation. Semin Intervent radiol 2008; 25(4): 413-424. PMCID: PMC 3036532. PMID: 21326583.

34. Bozgeyik Z, Ozgocmen S, Kocakoc E. Role of diffusion-weighted MRI in the detection of early active sacroiliitis. AJR 2008;191(4): 980-986. PMID: 18806131.

35. Oztekin O, Calli C, Adibelli Z, Kitis O, Eren C, Altinok T. Brucellar spondylodiscitis: magnetic resonance imaging features with conventional sequences and diffusion weighted imaging. La radiologia medica 2010; 115(5): 794-803. PMID: 20182812.

36. Taskin G, Incesu L, Aslan K. The value of apparent diffusion coefficient measurements in the differential diagnosis of vertebral bone marrow lesions. Turk J Med Sci 2013; 43: 379-387. DOI: 10.3906/sag-1204-85.

37. Nussbaum ES, Rockswold GL, Bergman TA, Erickson DL, Saljeskog EL. Spinal tuberculosis: A diagnostic and management challenge. J Neurosurg 1995; 83(2): 243-247. PMID: 7616269.

38. Jain AK. Treatment of tuberculosis of the spine with neurologic complication. Clin Orthop Relat Res 2002; 398:75-84. PMID: 11964634.

39. Okada Y, Miyamoto H, Uno K, Sumi M. Clinical and radiological outcome of surgery for pyogenic and tuberculous spondylitis: Comparisons of surgical techniques and disease types. J Neurosurg Spine 2009; 11: 620-627. PMID: 19929368.

40. Rigotti S, Boriani L, Luzi CA, Marocco S, Angheben A, Gasbarrini A, et al. Minimally Invasive posterior stabilization for treating spinal tuberculosis. J Orthop Traumatol 2013; 14 (2); 143-145. PMCID: PMC 3667377. PMID: 22358780.

41. Tuli SM. Tuberculosis of the spine: A historical review. Clin Orthop Relat Res 2007; 460: 29-38. PMID: 17483734.

42. Tuli SM. Historical aspects of Pott's disease (spinal tuberculosis) management. Eur Spine J 2013; 22(4): S529-538. PMID: 22802129.

FIGURES

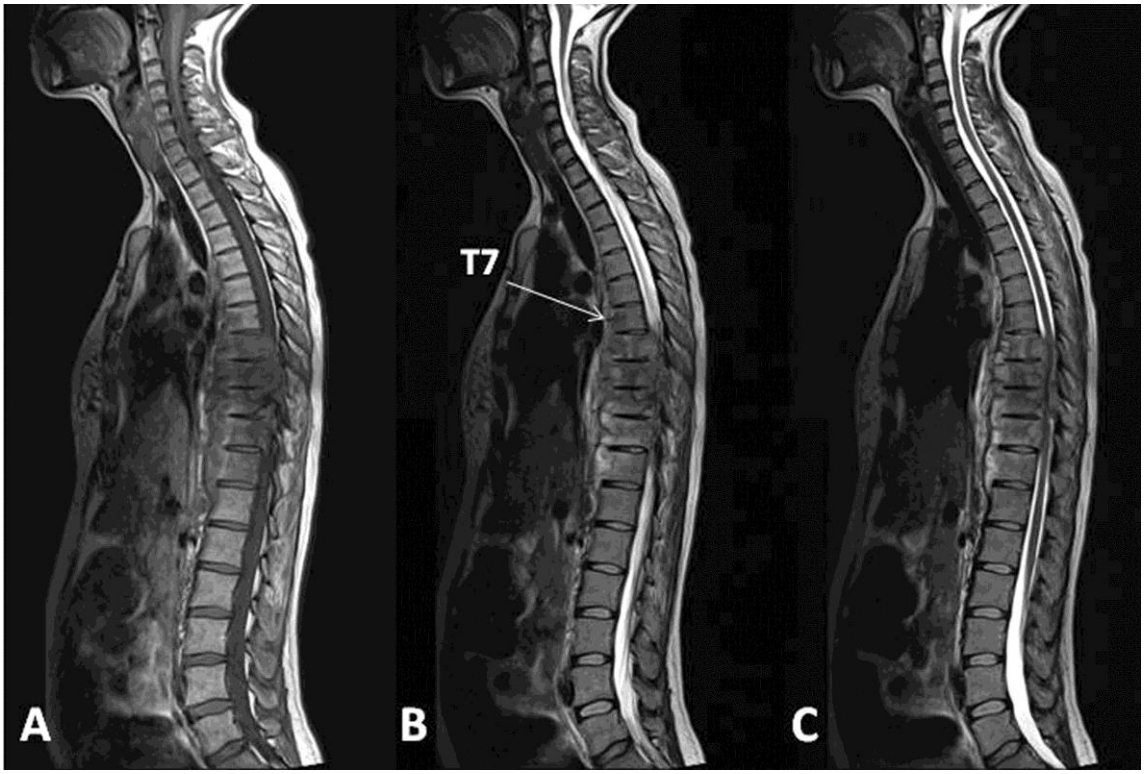

Figure 1: A 30 year old female with atypical spinal tuberculosis. MRI of the thoraco-lumbar spine. A- Sagittal T1 weighted image. B, C- Sagittal T2 weighted images, showing contiguous vertebral body involvement from T7 to T12 level with preserved intervertebral discs associated with prevertebral lobulated lesion and spinal cord compression due to epidural extension of the lesion. Technique: 3Tesla MR scanner, sagittal T1- TSE sequence (TE 9.3, TR 600), sagittal T2- TSE sequence (TE 104, TR 4000).

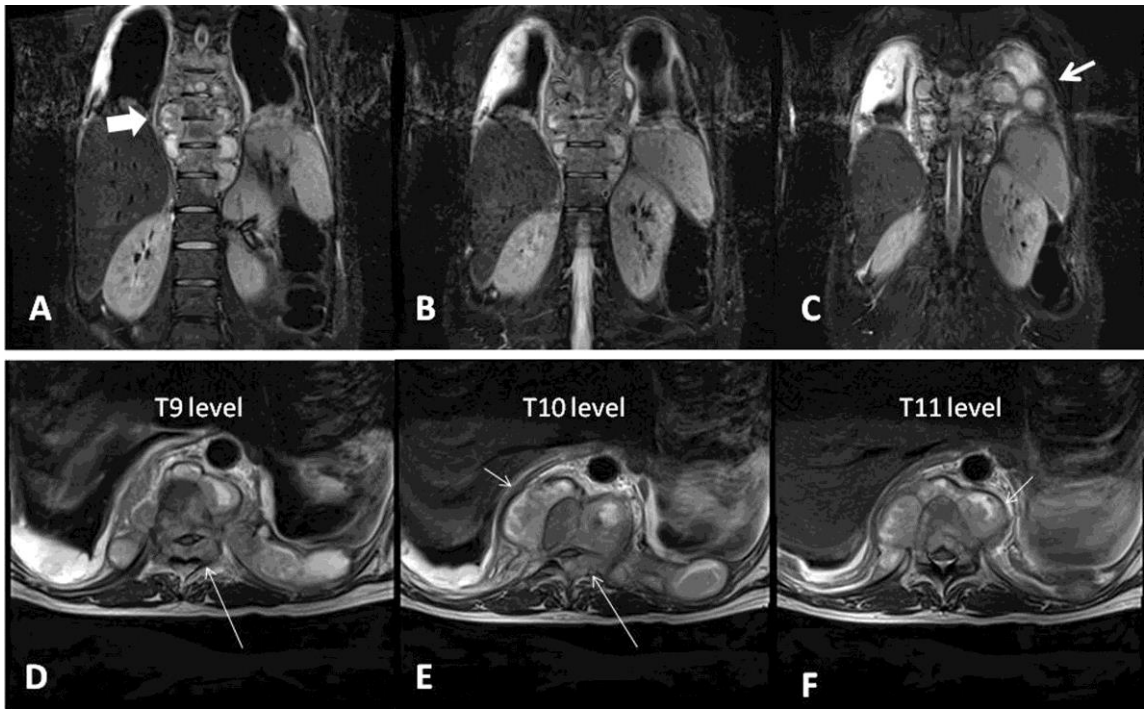

Figure 2: A 30 year old female with atypical spinal tuberculosis. MRI of the thoraco-lumbar spine. A, B, C- Coronal STIR sequences showing a multi-level contiguous lower thoracic vertebral lesions with large lobulated paravertebral lesions (wide arrow) associated with bilateral pleural based lesions and effusion (arrow). D, E, F-Axial T2 weighted images at T9, T10 and T11 levels respectively showing pre- and para-vertebral lobulated lesion involving vertebral bodies (short arrows) and posterior elements with epidural extension compressing the dural sac and the spinal cord (long arrows). Technique: 3Tesla MR scanner, coronal STIR sequence (TE 52, TR4000), axial T2-TSE sequence (TE 83, TR 7140).

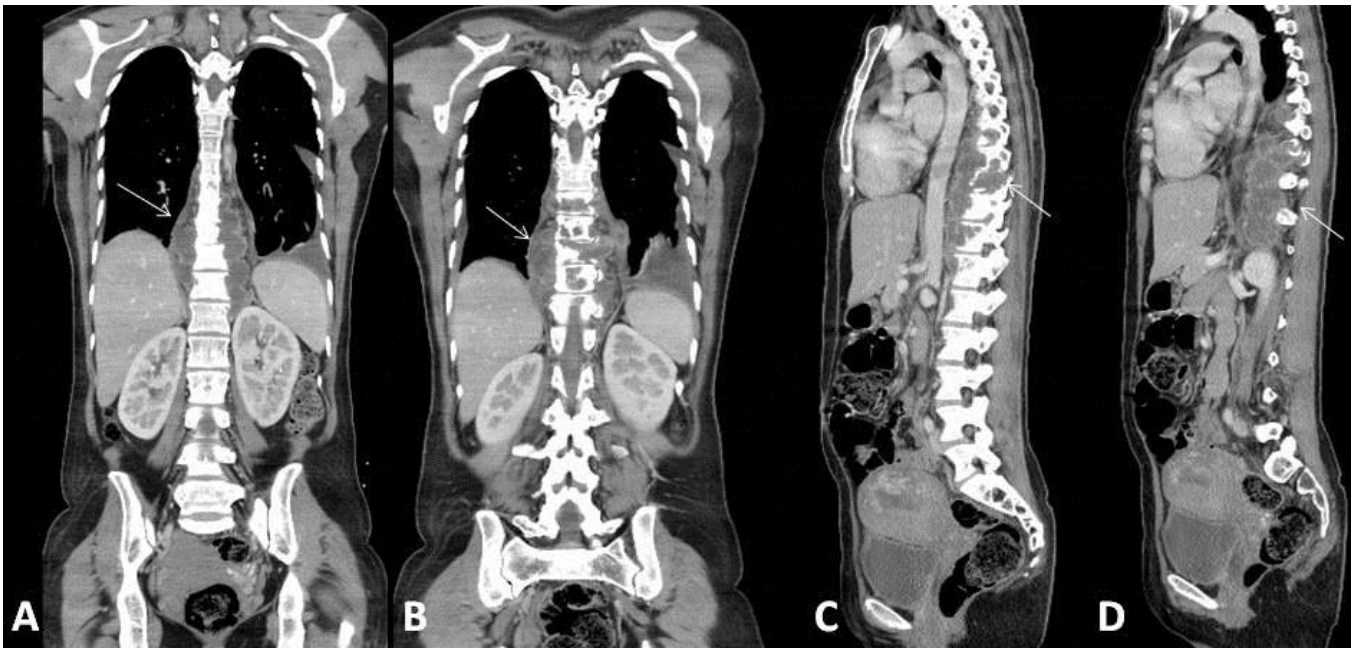

Figure 3: A 30 year old female with atypical spinal tuberculosis. Contrast enhanced CT of the chest and abdomen. A, B- Reconstructed coronal images. C, D- Reconstructed sagittal images showing contiguous multi-level vertebral involvement in the lower thoracic levels with destruction and associated large paravertebral lobulated abscess (arrows). Technique: Multi detector CT scanner (64 slice), mAs 91.7, kV 120, 3mm slice thickness. Porto-venous phase of contrast study using 100 ml Omnipaque (300mg I/ ml).

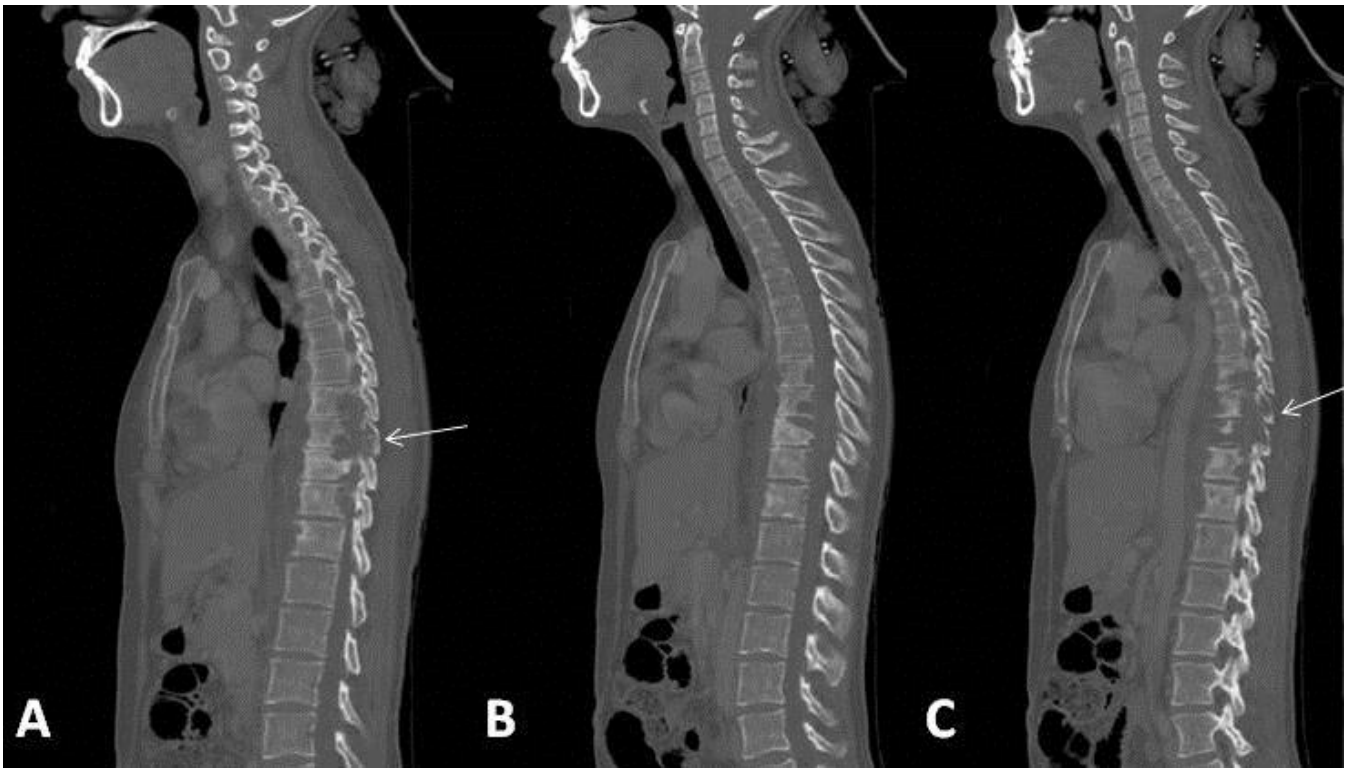

Figure 4: A 30 year old female with atypical spinal tuberculosis. CT of the chest and abdomen. Reconstructed sagittal CT images of the thoraco-lumbar spine in bone window showing multi-level vertebral destructive lesions involving the vertebral bodies and the posterior elements, from T7 to T12 levels (arrows). Technique: Multi detector CT scanner (64 slice), mAs 91.7, kV 120, 2.5 mm slice thickness.

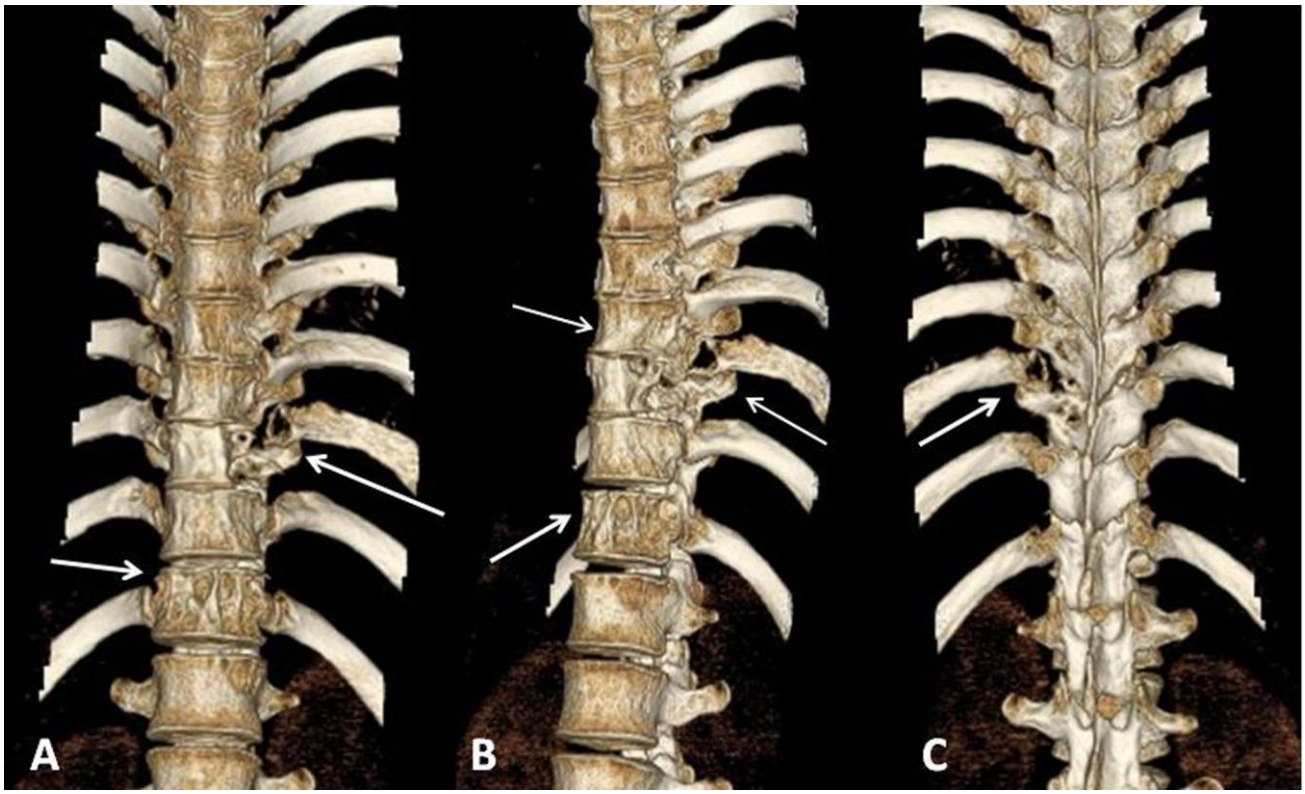

Figure 5: A 30 year old female with atypical spinal tuberculosis. Three dimentional (3D) reconstruction of the thoraco-lumbar spine showing the bone destruction in the lower thoracic vertebrae involving the vertebral bodies and the posterior elements (arrows). Technique: Multi detector CT scanner (64 slice), 3D reconstruction of 0.6mm slice thickness scan.

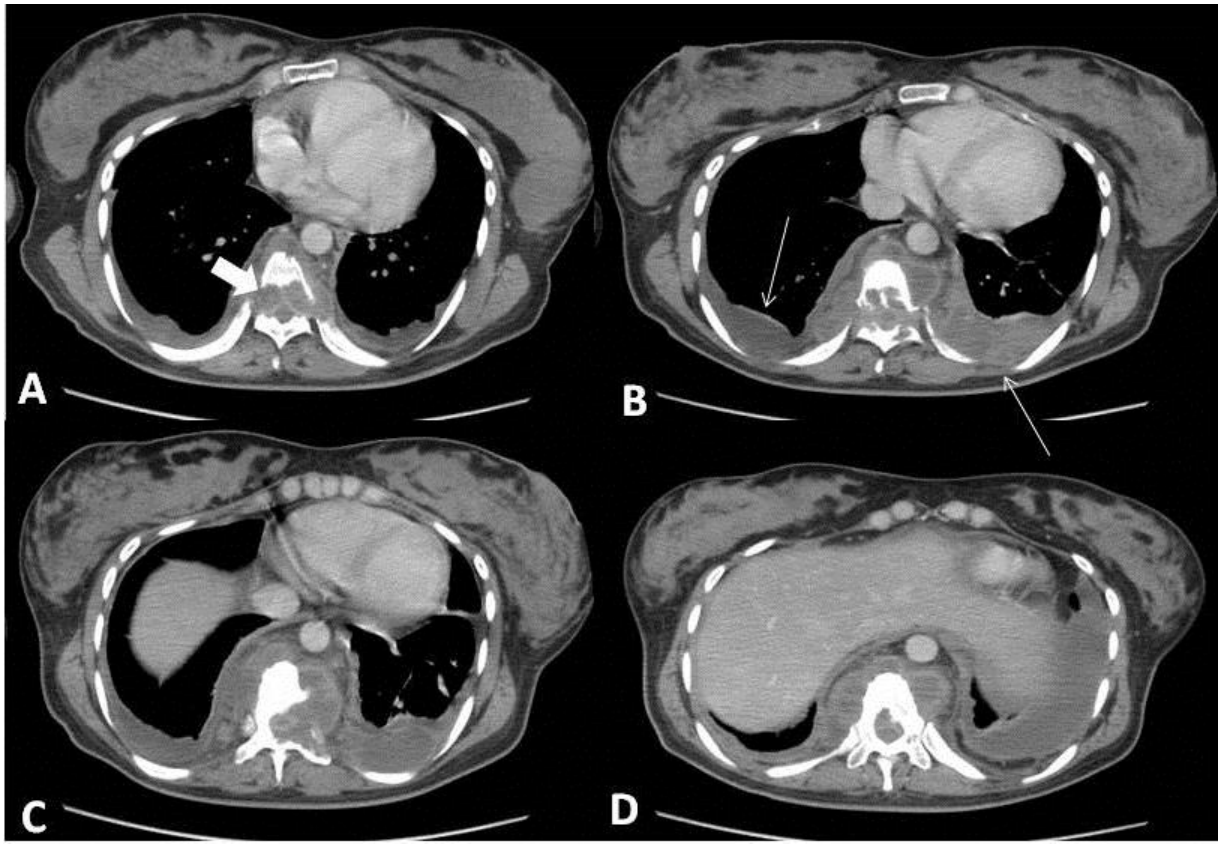

Figure 6: A 30 year old female with atypical spinal tuberculosis. Axial contrast enhanced CT sections of the chest. A- T 8 level, B- T 9 level, C- T 10 level, D- T 11 level, showing destructed vertebrae, para- and pre- vertebral abscesses with epidural abscess (wide arrow). There is also bilateral pleural empyema (arrows). Technique: Multi detector CT scanner (64 slice), mAs 91.7, kV 120, 3.7 mm slice thickness. Porto-venous phase of contrast study using 100 ml Omnipaque (300mg I/ ml).

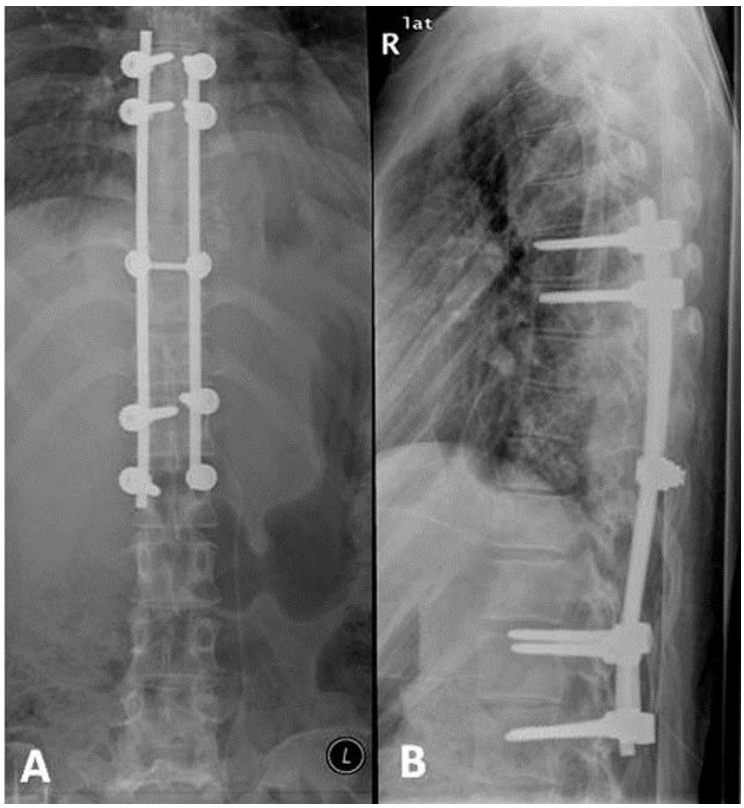

Figure 7: A 30 year old female with atypical spinal tuberculosis. Post-operative thoraco-lumbar spine x-ray. A-AP view (mAs 15.5, kV 77), B- Lateral view (mAs 35.7, kV 81) showing instrumented posterior spinal stabilization of the thoraco-lumbar spine.

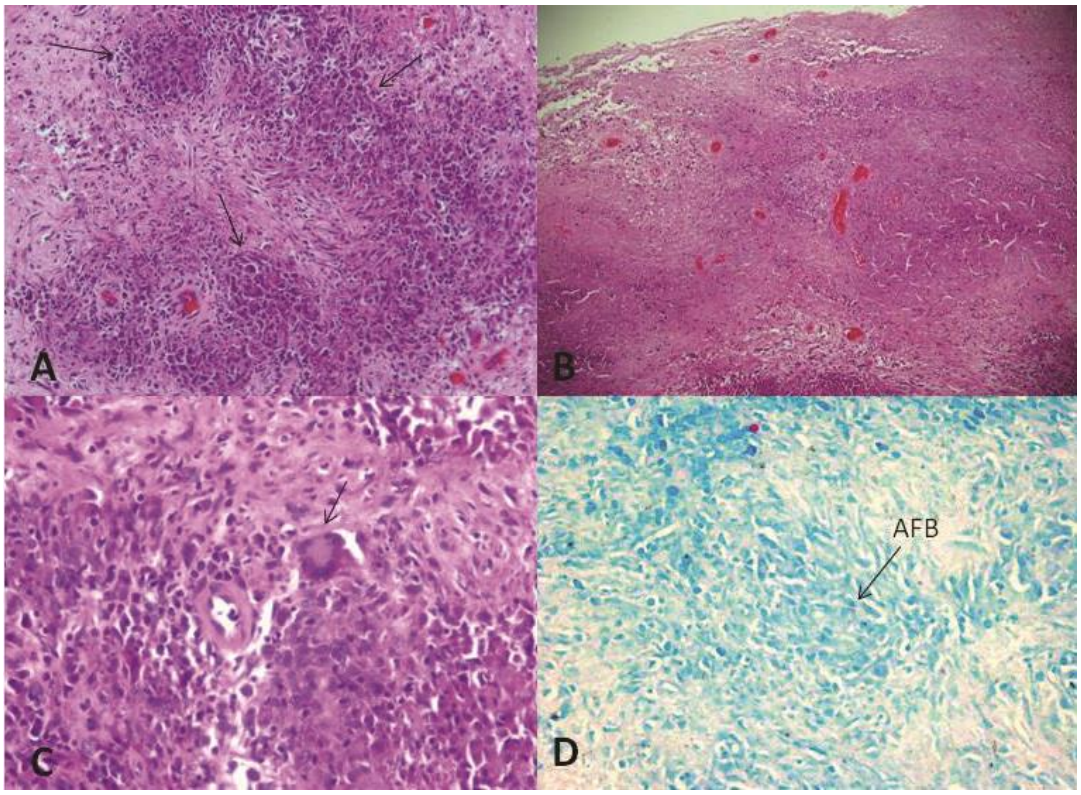

Figure 8: A 30 year old female with atypical spinal tuberculosis. Histopathology Photomicrographs. A- Low power view showing epitheloid granulomata (arrows) (H&E, original magnification X20). B- Low power view showing caseation necrosis (H&E, original magnification X10). C- High power view showing epitheloid granulomata with Langhans' giant cells (arrow) (H&E, original magnification X40). D- Ziehl Neelsen staining showing AFB (arrow) (original magnification X100).

Diagnosis	Commonest Location	X-ray	CT	MRI	Enhancement Pattern	Specific features
Atypical spinal TB	-Thoraco-lumbar spine -Can be multisegmental -Skip lesions	-Vertebral lytic lesion with preserved disc -Paraspinal shadow -No sclerosis	-Destructed vertebrae -Large paravertebral abscess	-Typically heterogeneous mixed T2 signal in involved bone. -High T2, low T1 signal in the intraosseous and paravertebral abscesses -Epidural extension with spinal cord compression -Acute stage shows restricted diffusion. -In chronic stages, no diffusion restriction with high ADC values.	-Heterogeneous in the vertebral lesions -Peripheral smooth rim enhancement in intraosseous and paraspinal abscesses	-Involvement of posterior vertebral elements. -Kyphotic deformity -Calcification in paravertebral abscess.
Pyogenic spondylitis	-Lumbar and cervical spine -Single segment	-Decreased disc height with vertebral lytic lesion -Sclerosis	As plain X-ray with small para-vertebral lesion and epidural extension	-Homogeneous high T2, low T1 signal in involved vertebrae and disc -Acute stage shows restricted diffusion. -In chronic stages, no diffusion restriction with high ADC values.	-Homogeneous enhancement of involved vertebral bodies -Intense ill-defined enhancement in paravertebral lesion/ abscess	-Rapid disc destruction -Sclerosis -Posterior elements are spared -No kyphosis
Brucellar spondylitis	-Lumbar spine most frequently involved -Multiple levels can be involved -Skip lesions can be seen	-Lytic lesion in disco-vertebral junction -Sclerosis -Anterior osteophytes	-Disco-vertebral lesion -Small paraspinal lesion	-Homogeneous high T2, low T1 in involved vertebrae and disc -Small paraspinal lesion with no or minimal epidural extension -Acute stage shows restricted diffusion. -In chronic stages, no diffusion restriction with high ADC values.	-Homogeneous enhancement of vertebral bodies and involved discs	-Anterior "parrot beak" osteophytes -Gas in discs -Facet joint enhancement -Kyphosis is rarely seen -Posterior elements are spared
Lymphoma	-Skip or contiguous multilevel involvement	-Most lesions are lytic -Rarely mixed lytic-sclerotic or sclerotic	-Vertebral lesions (body and posterior elements) -Paraspinal masses -Epidural lesions	-Focal or diffuse vertebral lesions -Variable/ high T2 signal in the vertebral and paraspinal lesions -High STIR signal -Low T1 signal -Restricted diffusion. -Low ADC values	-Heterogeneous diffuse enhancement	-Paraspinal masses with vertebral lesion but no extensive cortical bone destruction
Metastatic lesions	-Mostly thoracic spine	-Most lesions are lytic vertebral lesions -Some lesions are mixed lytic-sclerotic or sclerotic	-Bony destruction with epidural mass -Sclerotic lesions	-Vertebral lesions show low T1signal -High signal in T2 and STIR sequences -Sclerotic lesions are hypointense on all sequences -Restricted diffusion. -Low ADC values	-Diffusely enhancing	-Vertebral body and posterior element lesions -Preserved discs -Halo sign
Osteoporotic fractures	-Mostly mid thoracic and thoraco-lumbar vertebrae	-Decreased bone density -Decreased vertebral heights with wedging	-Vertebral body wedging -Associated posterior vertebral wall fracture with retropulsion	-Low T1, high STIR signal in fractured vertebra -No paraspinal or encasing epidural mass -No diffusion restriction -High ADC values	-Not used -Compressed vertebra may show mild enhancement	-Progressive kyphosis due to wedging of vertebral bodies -Spared or fractured pedicle. -MR-fluid sign

Table 1: Differential diagnosis table of atypical spinal tuberculosis (TB) - spondylitis without discitis

Etiology*	Haematogeneous spread of *Mycobacterium tuberculosis*
Incidence *	-Skeletal TB is seen in 1-5% of all TB patients -Spinal TB accounts for 50% of skeletal TB cases
Gender ratio*	Male: female = 4: 3
Age predilection	Average 40 years
Risk factors *	Poverty, overcrowding, illiteracy, malnutrition, alcoholism, drug abuse, diabetes mellitus, immunosuppressive treatment, chronic peritoneal dialysis, previous tuberculous infection and HIV infection.
Imaging Findings	-Computed tomography shows destruction of multiple vertebral bodies and posterior elements with preserved discs. Skip lesions can be seen. No sclerosis. Presence of a large paravertebral abscess that can show calcification. Kyphotic deformity can occur. -Magnetic resonance imaging shows typically heterogeneous mixed T2 signal, heterogeneous enhancement in the involved vertebrae with high T2 signal in the intraosseous and paraspinal abscesses with thin smooth rim enhancement. Epidural abscess causes spinal cord compression.
Treatment*	-Medical treatment -Surgical treatment is indicated in cases with neurological complications
Prognosis*	In 95% healing occurs with medical treatment in the predestructive stage

*Include all cases of TB spondylitis (typical and atypical)

Table 2: Summary table of atypical spinal tuberculosis (TB)

ABBREVIATIONS

ADC = Apparent diffusion coefficient
AFB = Acid fast bacilli
CT = Computed Tomography
DW-MRI = Diffusion weighted MRI
ETB = Ethambutol
H&E = Haematoxylin and Eosin
HIV = Human immune deficiency virus
INH = Isoniazid
MDR-TB = Multi-drug resistant TB
MRI = Magnetic Resonance Imaging
PZN = Pyrazinamide
RMP = Rifampicin
STIR = Short tau inversion recovery
TB = Tuberculosis

KEYWORDS

Tuberculosis; spondylodiscitis; spinal tuberculosis; Pott's disease; atypical tuberculous spondylitis; MRI

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