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Abstract: Tuberculosis (TB) represents a major public health problem worldwide. Any tissue may be infected. Involvement of the musculoskeletal (MSK) system account for 1-3% of all tuberculous infections. MSK TB may manifest as tuberculous spondylitis, arthritis, osteomyelitis, and soft tissue infections. Although TB spondylitis may present with distinctive imaging features compared to pyogenic infections of the spine, the imaging semiology of extraspinal TB infections is mostly nonspecific and may mimic other lesions. TB infections should therefore always be considered in the differential diagnosis, particularly in immunocompromised patients. The aim of this article is to review the imaging features of spinal and extra-spinal MSK TB. Magnetic Resonance Imaging is considered the modality of choice to make the diagnosis and to evaluate the extent of the disease.

Key words: Tuberculosis – Musculoskeletal – Conventional Radiography – Computed Tomography – Magnetic Resonance Imaging

KEY POINTS:

- MSK tuberculosis represents up to 3% of all TB infections.
- 50-70 % are located at the spine, while extraarticular TB accounts for 30% of cases.
- Late preservation of the disc space, calcifications, anterior vertebral corner destruction, multilevel involvement, skip areas and rarely posterior element involvement are useful signs to differentiate TB spondylitis from pyogenic spondylitis.
- Although Phemister triad on CR is a typical feature of TB arthritis, MRI is more sensitive for early detection.

Introduction

Tuberculosis- and poliomyelitis-related orthopedic and neurologic disorders such as scoliosis as well as paraplegia used to be the main etiology for hospitalization in dedicated orthopedic units in Central Europe during the early 20th century. This has dramatically changed due to increasing wealth, research and development of antibiotics and immunizations. However, tuberculosis-related infections remain a major challenge in some areas and territories with a lower socio-economic background [1-3].

Musculoskeletal (MSK) tuberculosis (TB) represents 1–3% of all TB [4 ,5]. It occurs in 25% of cases of extra-pulmonary TB [6,7]. The spine is the most frequent location, seen in 50-70% of MSK TB. Involvement of the peripheral joints is seen in 30%. Soft tissues infection, such as tenosynovitis, pyomyositis and bursitis are rare [6]. TB infections, that are commonly indolent with a variable clinical presentation, may mimic other conditions, such as pyogenic arthritis, rheumatoid arthritis or pigmented villonodular synovitis [8], resulting in a delayed diagnosis [9]. The aim of this article is to review the pathogenesis, clinical presentations, and most common imaging features of MSK TB.

Pathogenesis and predisposing factors

Causative microorganisms

TB is a chronic bacterial infection resulting in formation of caseous granulomas in infected tissues. The causative organism is a mycobacterium called the Mycobacterium tuberculosis complex, of which M. tuberculosis is responsible of 97–99% [4].

Atypical mycobacteria, such as Mycobacterium kansasii, Mycobacterium marinum, Mycobacterium scrofulaceum, and Mycobacterium avium complex account for the rest of the cases of TB [10-12].

After the phagocytosis of the TB bacillus, epithelioid histiocytes and lymphocytes gather in small clusters. (Micro-) granulomas form to restrict the disease [4]. Those microgranulomata conglomerate and join to form a mature noncaseating tuberculoma. Commonly, subsequent solid central caseous necrosis will develop, which may eventually liquefy. Pathologically, a central zone of solid caseation necrosis containing only scarce TB bacilli in the caseating granuloma, is depicted. A capsule of collagenous tissue, epithelioid cells, multinucleated giant cells, and mononuclear inflammatory cells surrounds the lesion. The diagnosis is based on the imaging presentation, that will be detailed later, whether the granuloma is noncaseating, caseating with a solid center, or caseating with a liquid center [13].

Infection may extend to the surrounding tissues and calcifications of granulomatous lesions can be seen in the late stage [9]. All organs may be infected but the lungs are the most involved (80–85%) [4, 14-16].

Microbiology

The final diagnosis is based on the microbiology test results. A special microbiology-request form is therefore needed with request to Acid-Fast Bacilli (AFB) smear, culture & identification or M. tuberculosis Complex Genotyping.

The microbiological diagnosis of active TB is based on AFB smear microscopy, Nucleic Acid Amplification Tests (NAAT), and culture. Culture is the gold standard for TB diagnosis, with possible diagnosis of TB at an early stage, allowing extrapulmonary TB diagnosis, species identification, and drug susceptibility testing. Weeks are however needed before results are known. NAAT have significantly reduced this delay [17].

The Xpert MTB/RIF tests (Cepheid) are the only U.S. Food and Drug Administration (FDA)-approved automated NAAT that can identify M. tuberculosis bacteria (MTB) and resistance to rifampicin (RIF) from respiratory specimens. It is an easy cartridge based real-time Polymerase Chain Reaction (PCR) method delivering results in 2 hours. It has a closed amplification system that reduces cross contamination risk. Moreover, no advanced biosafety equipment is needed [17].

Histology

Unlike pulmonary tuberculosis (TB), biopsy is required in the extrapulmonary forms of TB. Extrapulmonary TB is often paucibacillary, with common negative culture. In these cases, notably in endemic areas where molecular tests are unavailable, histological, and cytological examinations provide a rapid diagnosis allowing the initiation of the treatment. The hallmark of TB infection is necrotizing granulomatous inflammation [18].

Cytology

Fine needle aspiration (FNA) is a simple and safe outpatient procedure for the diagnosis of osseous TB, obviating the need of an open biopsy [19].

Immunohistochemistry

Immunohistochemistry (IHC) on biopsy specimens improves tuberculosis diagnosis. MPT64 is a unique antigen with high sensitivity and specificity, compared to other conventional techniques, in the diagnosis of TB and to differentiate it from nontuberculous mycobacteria. Pragati et al. showed in a study that immunohistochemical staining by anti-MPT64 is useful in establishing microbiological diagnosis of extra-pulmonal TB on biopsy specimens [20].

Molecular pathology

Molecular techniques on Formalin-Fixed Paraffin-Embedded Tissue (FFPET) can be an alternative tool for the diagnosis of TB when microbiological investigations have not been performed if the clinical features are in favor of a nontuberculous pathology. Confirmation of TB by molecular tests on FFPET avoid another biopsy, which would also be impossible if the lesion has been removed or if there is a high risk of anesthesia [18].

Staff-protection is recommended for imaging-guided biopsy and drainage of an abscess in suspected TB infection. In those cases where imaging guided biopsy or aspirate are performed, the radiologist should be informed of the possible diagnosis before the procedure. Protection is not needed for general radiology personnel unless in case of a procedure that may aerosolize a tuberculous collection or when the patient is multi drug resistant (MDR) positive. In those situations, or if there is a risk of aerosolizing tuberculous collections, a certified respirator is recommended. Protection is also not required for patients. Ideally, any imaging should be performed within the patient's isolation room. If the imaging is done in the department, it should be scheduled for off-peak times, away from immunocompromised patients. The patient should wear a surgical mask and rapidly imaged in his or her own well-ventilated area, and in the best-ventilated room available. If the patient is MDR positive, a certified respirator worn by the patient is recommended [21].

Spread of infection

A musculoskeletal TB infection is most commonly the result of a hematogenous dissemination or lymphatic spread from a primary lung infection or reactivated infected tuberculous focus in a bone or joint [10,22]. Synchronous active pulmonary TB is seen in less than 50% of the patients [30]. Musculoskeletal TB rarely results from a direct inoculation [11,24,25].

Predisposing factors

Immunosuppression resulting from a disease such as the acquired immune deficiency syndrome (AIDS) or drug therapy, represents the most frequent predisposing condition. Pre-existing joint disease is also a risk factor [6]. Other risk factors for reactivation include poor socioeconomic conditions [11].

Different manifestations of MSK TB

Tuberculosis of the osseous spine

Pathogenesis

TB spinal infection develop from arterial hematogenous spread of M. tuberculosis into vertebral body from a primary or reactivated focus, which is usually either a pulmonary or a genitourinary lesion [26-29].

Extension from a paraspinal infection is rare [28,29]. Lymphatic drainage from an adjacent infected tissue such as the pleura or a kidney may engender infection spread [30,29,28].

Venous spread was also suggested, although being less probable. It occurs through the Batson's plexus running in the subchondral bone of the vertebral body [29,31].

The anatomy of vascularization of the vertebrae explains the location of spinal infection (Fig. 1) [26,27,31].

There is an anterior and a posterior vertebral arterial supply in adults. Anterior paired segmental arteries perforate the vertebral cortex with non-anastomosing arterioles. Posterior spinal arteries form a craniocaudal anastomotic network with adjacent levels. End arterioles stop beneath the anterior aspect of the end plates, where Mycobacteria may be trapped [29,32].

Initially, the infection is limited to the vertebral body and the term "vertebral osteomyelitis or spondylitis" may be used. Spondylitis can be solitary or multifocal, contiguous, or not [26,27].

Further extension of the infection to the adjacent vertebral body by contiguity occurs beneath the anterior or posterior longitudinal ligament or through vascular anastomoses. Secondary spread to the disc may occur. At this stage, the designation "spondylodiscitis" is appropriate. Multiple vertebrae are commonly involved because of spread via the longitudinal vascular anastomosis and progressive destruction of the disc and vertebra may occur [33,34]. The infectious process may extend later to the paravertebral soft tissues or to the spinal canal, with paravertebral abscess formation due to tissue necrosis [26,27,30].

In the pediatric population, the disc can be primarily involved by the hematogenous route because the disc is still vascularized at this age group. In the latter scenario, "discitis" is the primary site of the infection [26,27,35,36].

Clinical presentation

The time interval from symptom onset to diagnosis is usually several months because of the insidious and slow development [28]. Clinical findings are non-specific and range from long-standing lumbar stiffness to local tenderness [29]. Night sweats are observed in 20% of cases, with fatigue and gradual weight loss. A moderate evening fever is observed in about 50% of cases [26,27]. Severe spinal deformity (gibbus) resulting from an acute kyphotic angulation may be depicted in advanced stages. Neurological symptoms and complications may be acute or delayed [28].

Imaging features

- *Tuberculous spondylodiscitis*

Tuberculous Spondylodiscitis has been historically designated as **Pott's disease** with involvement of the disc and vertebra and representing 47–94% of spinal TB [26,27,34,37]. 80% of spondylodiscitis are seen in the lower thoracic and upper lumbar spine [33,38,39], 4–15% of cases are seen in the cervical spine [37] and 2–3% in the lumbosacral spine [30,40]. In children, immigrants, and immunocompromised persons, multiple vertebrae lesions may be involved (5–23%) [26,27,38,41-44].

Conventional Radiography (CR): CR has a low sensitivity for early detection of TB spondylitis. Spondylitis can be detected if at least 50% of a vertebra is destroyed [45]. Detection of associated soft tissue extension is difficult, unless calcifications develop in the paravertebral area in advanced stage [28,29]. At the early stage of the infection, CR may show localized osteoporosis of the subchondral bone with loss of the "white stripe" of the vertebral end plate. The extension of the infection will disrupt the cortex and spread to the adjacent disc space with slight disc-space narrowing. Disc-space narrowing is usually less extensive and manifests later in TB compared to pyogenic spondylitis [29]. This can be attributed to the absence of proteolytic destructive enzymes in Mycobacterium tuberculosis [29]. Progressively, destruction of the lateral and anterior cortices of the vertebral body occurs with developing of angular collapse (Fig. 2), causing subsequent kyphosis. Craniocaudal extension of the infection to multiple adjacent vertebral bodies with typical anterior corner bone destruction happens subsequently to subligamentous spread underneath the anterior longitudinal ligament [29]. Large intravertebral cavities limited by thin sclerosis, containing sequestra continuous with the disc may develop later. Bone sclerosis is less common and less extensive than in brucellar or pyogenic spondylodiscitis [30,46,47]. The association of a normal disc height with intravertebral cavities is highly suggestive of TB spondylitis [26,27,48].

Computed Tomography: Early foci of bone infection and soft tissue extension (Fig. 3) are better depicted on CT than on CR [28]. CT is very helpful to assess early cancellous bone destruction, cortical resorption as well as deformity of the vertebral spine in advanced stages (gibbus deformity) [29]. It is also very sensitive to assess extension to the spinal canal and posterior elements, and to look for an associated facet joint arthritis [30]. Intradiscal hypodensity may indicate early disc involvement, although MRI is better suited for assessment of the disc [18,19,42]. Paravertebral tuberculous abscesses (Fig. 4) are usually large and bilateral [30,39,41], presenting as hypodense collections limited by thin and vascularized walls, containing central or peripheral calcifications, which is characteristic of TB [37]. Less commonly, epidural extension occurs, resulting in an epidural phlegmon or abscess underneath the posterior longitudinal ligament (Fig. 5), having a bilobed morphology on axial images [18,19,39,42]. CT may be used to guide percutaneous biopsy or for drainage of intradiscal or paravertebral abscesses [29,46].

CT is also used in case of suspicion of spinal instability or in the pre-operative assessment of secondary spine deformities [26,27,38,39,46].

Magnetic Resonance Imaging: Magnetic resonance imaging (MRI) is the method of choice for detection and follow-up of spinal TB [39,30,46,49-51]. MRI is the most sensitive imaging technique to detect early inflammatory bone marrow changes and end plate changes, even before they appear on CR and CT. MRI is helpful to delineate paravertebral, epidural, and intraosseous abscesses and to evaluate the extent of cord compression and the presence of intramedullary lesions. Assessment of the whole spine helps to look for additional asymptomatic spine locations, also called "skip infections," present in 16–70% of cases (Fig. 6) [26,27,35,37,40,44,52].

Characteristic findings consist of T1-hypointensity of both vertebral bodies and disc spaces, but with a marked T2-hypointensity in the vertebral body with a T2-hyperintensity in the intervertebral disc. In the late chronic stage, the signal is variable [26-28]. The T2-hypointensity in the vertebral body may be explained by fibrosis, macrophage infiltration and free radicals in caseous necrosis [9,53].

Location at the anterior surface of the vertebra (Fig. 7) is highly suggestive of spinal TB presentations [54,55]. They result from subperiosteal lesions at the anterior vertebral surface. Extension to multiple vertebral segments may occur along the anterior longitudinal ligament, sometimes with skip lesions. In addition, periosteal stripping may compromise the extra-osseous vasculature supplied by the periosteum, with resulting osteonecrosis and anterior vertebral scalloping [56]. Subligamentous

abscesses are depicted on MRI with typical preservation of disc [54] and peripheral rim enhancement and absence of central enhancement corresponding to central pus or caseated material [28, 29].

Intraosseous abscesses are centrally located in the vertebral body and manifest with a T1-hypointensity, very high T2-signal intensity with rim enhancement [30, 26,27, 28, 29]. Epidural phlegmon is an epidural enhancing mass with intermediate T1- and hyperintense T2-signal [49]. A peripheral rim enhancement is seen in epidural abscess. Like CT, epidural abscess and phlegmon have a typical "bilobed" shape (Fig. 5) [26,27,57].

- *Differential diagnosis of tuberculous spondylodiscitis (**Table 1**)*

-	Pyogenic/fungal	Brucellosis	Erosive osteochondrosis	Metastasis
Disc space	Early destruction	Gas within the disc	Narrowed disc space	No involvement
Extension	Disc/adjacent vertebra	Disc/adjacent vertebra Typical anterior osteophytes (Parrot's beak) on CR.	Absent	Disc/adjacent vertebra
Soft tissue component	Modest	Minimal paraspinal masses	Absent	Solid extraosseous soft tissue component.
Calcifications	Extremely rare	Extremely rare	Absent	Absent
Posterior involvement	Extremely rare	Extremely rare	Absent	Possible
Location	L>T>C	L>T>C	L=T=C	L=T=C

Table 1: Differential Diagnosis with characteristics & clues to the diagnosis of TB spondylodiscitis

- *Tuberculosis of the posterior elements*

Neural arch tuberculosis may occasionally be the initial site of tuberculosis and may be solitary or multifocal. It occurs commonly in the cervicothoracic spine [33, 37,49]. Spinal instability is a frequent complication and imaging helps in surgical planning [26,27,58].

CR: diagnosis on CR is only possible in less than 10% of cases [58]. Osteolysis of a spinous process, a lamina or a pedicle may be seen with possible extension to the posterior cortex of the vertebral body or the adjacent ribs. Well-defined or ill-defined bone sclerosis may also be observed [26,27,37].

Computed Tomography: Assessment of bone destruction and adjacent soft tissues is better evaluated than on CR [58,37,51]. Zygapophyseal, costovertebral, or costotransversal joints may be involved [26,27,37].

Magnetic Resonance Imaging: nonspecific, T1-hypointense and T2-hyperintense and enhancing, lesions are depicted (Fig. 7) [26,27].

Extra-spinal osteo-articular TB

TB of joints

Pathogenesis

In MSK TB, infection of the synovial joints is the second most frequent location after spinal TB [59,11], accounting for 30% [6]. TB arthritis is usually monoarticular in 90% [4,60], involving the large joints, such as the hip and the knee. The elbow, wrist, sacroiliac joint, glenohumeral joint, articulations of the hand and foot, and the sternoclavicular joint, may as well be affected [61]. Lower extremities are more involved than upper extremities [29]. Multiple joints may be involved in 10% of the cases [11].

TB arthritis is usually secondary to TB osteomyelitis, in which a primarily tuberculous metaphyseal focus crosses the epiphyseal plate. Transphyseal spread is highly suggestive of TB skeletal infection. TB arthritis, without pre-existing osteomyelitis, due to direct hematogenous spread of tubercle bacilli to the synovial membrane via sub-synovial vessels is less frequent [11,29].
Reactive inflammatory hyperemia leads to juxta-articular osteoporosis. Osseous erosions develop on both sides of the joint space (Fig. 8) [29].
When the infectious process reaches the subchondral region, the articular cartilage loses its nutrition and gets detached from the bone; this may result in loose bodies [12]. Cartilage loss and destruction (Fig. 9) develop relatively late because the exudate in TB arthritis lacks proteolytic enzymes.
Granulomatous inflammation leads to synovial thickening and joint effusion [29].
Rice bodies consisting of flakes or loose sheets of necrotic articular cartilage and accumulations of fibrinous material may develop in the synovial fluid [11].
Extension to adjacent para-articular soft tissue with formation of cold abscesses and sinus tracts develop if TB joint disease is left untreated [29]. Periosteal new bone formation may develop [11].
Damage to the physis in childhood may result in shortening or angulation of the limb [12].
TB arthritis should not be confused with Poncet's disease or reactive tuberculous rheumatism with poly-articular involvement in patients with active or inactive visceral TB. Its existence as a distinct entity is a matter of debate [8,12].

Clinical presentation
The onset is insidious. The symptoms are non-specific including chronic joint pain, restriction of joint motion, swelling, muscle wasting, and deformity. Cold abscess may reveal the infection [6,8].

Imaging features

CR: There are five radiographic stages of TB arthritis summarized in **table 2.**

Stage I manifests with a localized osteoporosis with soft tissue swelling, stage II is characterized by marginal erosions with mild joint space narrowing. Subchondral and subperiosteal cysts with loss of joint space is seen in stage III (Fig. 8). Gross joint destruction is seen in stage IV (Fig. 9). Joint ankylosis is seen in stage V.

Stages	Diagnostic clues on imaging
Stage I	Synovitis - Localized osteoporosis - Soft tissue swelling
Stage II	Early arthritis - Marginal erosions - Mild joint space narrowing

Stage III	Advanced arthritis - Subchondral and subperiosteal cysts - Loss of joint space
Stage IV	Late advanced arthritis - Gross joint destruction
Stage V	Joint ankylosis

Table 2: Radiographic stages of TB arthritis.

Phemister triad consist of peri-articular osteopenia, marginal erosions, and gradual narrowing of the joint space [8]. Lack of sclerosis or periostitis in the early stage, except for children, in whom a layered periosteal reaction is present, is a typical feature of TB arthritis [29].

Computed Tomography: The degree of bony destruction, bony sequestration, and peri-articular soft tissue extension is better evaluated on CT than on CR [29,46].

Magnetic Resonance Imaging: MRI can detect the early-stage disease before joint space loss occurs such as articular cartilage defects, subchondral bone erosions and bone marrow oedema. Inflammation of surrounding muscles and subcutaneous tissues or sinus tracts formation are best evaluated on MRI [8]. Fibrosis, macrophage infiltration and free radicals in caseous necrosis are of low signal on T2-weighted images (WI) [9,8,53]. Internal debris, septation, loose bodies, calcifications, and hemosiderin deposits due to bleeding may be T2-hypointense [3]. TB arthritis should be thus included in the differential diagnosis when an articular lesion with low or intermediate SI on T2-WI is seen [29,61]. Joint effusion is often heterogenous with T2-hyperintense area due to the presence of necrotic synovial debris, fibrinous tissue, or rice bodies (Fig. 10) [6]. Rice bodies, consisting of fibrin collagen and mononuclear cells, may be present in joint, bursa, or tendon sheath, and are not only seen in TB arthritis but also in chronic synovitis in various rheumatic diseases [9,62-64]. As findings in TB arthritis on MRI are non-specific, aspiration or synovial biopsy is commonly needed for final diagnosis [11].

Differential Diagnosis

The differential diagnosis of tuberculous arthritis includes:

- Pyogenic arthritis and inflammatory arthritis:

More prominent bone erosions, subchondral bone marrow edema, and inflammatory changes of the surrounding soft tissues.
Abscesses with typical irregular and thick walls (compared to smooth and thin in TB arthritis).

- Rheumatoid arthritis:

Symmetrical polyarticular disease
Heterogeneous T2-hypointense synovitis (like in TB arthritis).
Uneven and thick synovitis (compared to smooth and thin synovitis in TB arthritis).

- Diffuse Tenosynovial Giant Cell Tumor:

Soft tissue swelling and joint effusion in the initial phases.
Preserved joint space.
Extrinsic erosions of the bone in the form of subchondral lucent areas with thin rim of sclerosis.

Heterogeneous localized or diffuse synovial plaque like or nodular thickening on MRI with T1-intermediate signal and T2-hypointensity.
Hemosiderin deposition and resultant blooming on T2* imaging.

- Neuropathic joint disease in the foot:

Widespread destruction in the joints of the midfoot.

Tuberculous osteomyelitis

Pathogenesis

TB osteomyelitis is less frequent than TB arthritis. Hematogenous or lymphatic spread from a primary (often pulmonary) TB focus, may result in either solitary or disseminated TB osteomyelitis [11]. Multifocal tuberculous osteomyelitis, also called osteitis cystica tuberculosa multiplex, is more frequently seen in children [8,65]. Alternatively, TB osteomyelitis may develop as a reactivation following a decrease of the host immunity [9]. TB infection occurs commonly in the metaphysis, like other infections, but the diaphysis can also be affected [8]. The infection starts in the medulla of the metaphysis and a granulomatous lesion develops. Caseation, liquefaction necrosis and progressive resorption of bone trabeculae occurs. Later, macroscopic bone destruction, periosteal reaction, transphyseal spread of the infectious focus and finally joint involvement are seen [29]. Occasionally, extension of the infection through the cortex may result in a para-osseous mass or collection [11]. Epiphyseal extension is characteristic in children [4,21].

Pathologically, there are two types of lesions. Caseous exudative type manifest with a destruction of bony trabeculae with softening and caseation necrosis of the bone and abscess formation. Caries sicca type is characterized by formation of granulation tissue with minimal caseation [8,12,35].

Clinical presentation

The onset is insidious and gradual with nonspecific pain, swelling and low-grade fever [8]. This indolent course may result in a delayed diagnosis.

Imaging features

CR: radiographs may reveal considerable destruction at a late stage of the disease. Common signs consist of well-defined osteolytic lesions in the metaphysis of long bones with or without sclerosis, soft tissue swelling, cortical expansile remodeling, periostitis, and sequestrum. Sequestration is not frequent and less extensive than with pyogenic osteomyelitis [11,30,39]. Transphyseal spread, with an isolated epiphyseal lesion, is a characteristic feature of tuberculous osteomyelitis not seen in bacterial infection [4,9,11,29]. Complications, such as pathological fracture, dislocations, limb length discrepancy, and deformities may also be depicted on CR. Reactive hyperemia affecting the growing ossification center may cause enlarged epiphyses [29].

Computed Tomography: CT is particularly useful for assessment of subtle bone erosions and sequestrum [9].

Magnetic Resonance Imaging: Nonspecific bone marrow edema, trabecular resorption, spread to the epiphysis and joint space, intraosseous abscesses with cortical bone disruption and possible extension to the muscles may be seen (Fig 11) [11,66].

On MRI, it is possible to distinguish a noncaseating granuloma from a caseating granuloma with a solid center, or caseating granuloma with a liquid center.

The non-caseating granuloma is usually T1-hypointense relative to the surrounding bone and T2-hyperintense and enhances homogenously.

The solid, caseating granuloma shows a heterogenously enhancing central portion surrounded by a capsule with a ring-enhancing pattern of uniform thickness. The lesion is commonly T1-hypointense or isointense relative to the surrounding bone and T2-isointense to hypointense. The rim of a caseating TB granuloma is markedly hypointense on T2-WI and enhances after gadolinium contrast administration. The presence of paramagnetic free radicals in the enclosed macrophages may explain the shortening of the T2 signal.

Central liquefaction of the tuberculoma occurs later. A granuloma with central liquefaction of caseous material hypointense in its center on T1-WI and hyperintense on T2-WI with an associated intense rim enhancement. At this stage, distinguishing a true tuberculous or pyogenic abscess formation is difficult om MRI [13].

Differential Diagnosis

The differential diagnosis of tuberculous intraosseous abscesses includes pyogenic Brodie abscess, simple bone cyst, aneurysmal bone cyst, chondroblastoma, and intraosseous tophi.

The differential diagnosis of multifocal tuberculous osteomyelitis includes Langerhans's cell histiocytosis, bone metastases, and multiple myeloma.

TB dactylitis is a subtype of TB osteomyelitis involving the short tubular bone of hands and feet [9]. It typically involves one single digit, but multifocal tuberculous dactylitis affecting both hands have been reported as well [12,67]. This lesion affects children aged less than 6 years [63]. Patients present with soft tissue swelling and flessum [9]. On CR, TB dactylitis manifest with an expansile, fusiform cyst-like or bubbly osteolytic lesion with ballooning of the remaining thinned cortex with periosteal reaction, explaining the term *spina* (short bone) *ventosa* (infated with air) [8,9]. MRI features are similar to those of TB osteomyelitis elsewhere varying from non-caseating granuloma, solid caseating granuloma to caseating granuloma with central liquefaction (Fig. 12).

Differential Diagnosis

The differential diagnosis of TB dactylitis includes:

- Fungal infection: Absent or very minimal periosteal reaction.
- Giant Cell Tumor of the small bones: Marked cortical ballooning. Absence or minimal periosteal reaction, sclerosis, and matrix mineralization. Involvement of the subchondral bone. Low signal intensity on all sequences due to deposition of hemosiderin or collagen.
- Enchondroma: Well marginated lytic lesion arising from the medullary cavity. Cortical expansion. Pathological fracture. Chondroid matrix with popcorn-like, rings and arcs, or whorls of calcification. Endosteal scalloping.

Tuberculosis of the soft tissues

Tuberculous tenosynovitis and bursitis

Pathogenesis

TB bursitis and tenosynovitis account for only 2% of MSK TBC [12,68,69]. TB tenosynovitis usually results from hematogenous spread from other sites or due to direct extension from an infected joint. Involvement of flexor tendons of the hand is typical, but rare [6]. Tenosynovitis and bursal involvement are considered late sequelae of osteoarticular involvement [11].

The first manifestations of TB tenosynovitis and bursitis consist in serous effusion, followed by synovial thickening, and accumulation of tissue debris and caseous material (Fig. 13). Tendon tethering or rupture are possible complications [9].

Imaging features

CR: Findings are often normal but may depict soft tissue swelling along the course of the tendon sheath [6]. Associated soft tissue calcifications favor TB infection [9].

Ultrasound: US is the first line imaging modality to demonstrate the tendon or synovial sheath thickening or both. In the chronic stage, the amount of fluid decreases, in contradistinction to acute pyogenic infection where large fluid collections are typical [6,11]. Ultrasound is also useful to assess the degree and extent of tendon and tendon sheath involvement [11].

Magnetic Resonance Imaging: MRI helps to delineate the precise extent of soft tissue and osteo-articular involvement (Fig. 14, 15) [11].

Three progressive stages exist with different MRI features according to the stage and extent of disease (**Table 3)** [6]:

Stages	Diagnostic clues on imaging
Hygromatous stage	Serous fluid in tendon sheath No thickening of tendon sheath
Serofibrinous stage	Thickening of flexor tendons (most typical) and synovium Multiple tiny hypointense nodules (rice bodies) within the T2-hyperintense synovial fluid
Fungoid stage	Formation of a soft tissue mass involving the tendon and tendon sheath

Table 3: Progressive stages of TB tenosynovitis

Differential Diagnosis

- Pyogenic and fungal infections
- Posttraumatic lesions

Tuberculous myositis and abscess

Pathogenesis

TB infection of the muscles or deep fascia is rare and may be encountered in immunosuppressed patients [6,12]. Striated muscle is resistant to mycobacterial infection owing to the poor oxygen content, high lactic acid concentration, and a paucity of reticuloendothelial tissue [12,70]. Tuberculous myositis results from local extension from adjacent lymphadenopathy, arthritis, or osteomyelitis. Chest wall involvement is due to direct invasion from pleural disease or thoracic lymphadenopathy. Cold abscesses in the psoas muscles arise from lumbar spondylodiscitis (Fig. 4). Primary infection via hematogenous or lymphatic routes is extremely rare [6,11].

Clinical presentation
Tuberculous myositis can simulate a slow-growing tumor [9,70]. Abscesses of the chest wall are located in the parasternal region, costovertebral junction, and along the ribs [12,71]. In the anterior chest wall, TB abscesses of the anterior chest wall may be clinically confused with breast masses [72].

Imaging features
Computed Tomography: allows precise localization of collections and possible associated bony involvement. Typically, TB collections show necrotic, hypodense centers with rim enhancement (Fig. 16) [6].

Magnetic Resonance Imaging: Lesions are T2-hyperintense and T1-hypointense with irregular enhancement of the abscess wall (Fig. 17) [6]. The abscess wall is slightly hyperintense on T1-WI and hypointense on T2-WI due to the presence of oxygen free radicals and iron within macrophages in the wall of the abscess (Fig. 18) [12,70]. There is diffusion-restriction [6].

Differential Diagnosis

Abscesses with wall calcifications: other chronic bacterial infections.

Tuberculous abscesses of the anterior chest wall can mimic breast masses.

Treatment principles

Anti-tuberculous treatment should be started as early as possible. This medical treatment is the cornerstone of treatment. In some cases, surgery may be necessary, for instance, large abscess formation, severe kyphosis, an evolving neurological deficit, or lack of response to medical treatment. Generally, the prognosis is good with early diagnosis and early treatment [73].

Conclusion

The imaging pattern of MSK TB of the spine may be very suggestive, which may assist in early diagnosis. However, imaging features may be nonspecific, especially in extraspinal MSK TB and in the advanced stages, mimicking other disease processes.

Awareness of the variable imaging pattern of MSK TB in patients with a high index of clinical suspicion may result in an earlier diagnosis allowing appropriate treatment to avoid devastating complications.

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Captions to figures:

Fig. 1

Pathogenesis of tuberculosis of the spine.

A: Posterior arterial supply of the vertebral bodies, terminating in multiple complexes of endarterioles subjacent to the anterior aspect of the vertebral end plates.

B: Entrapment of Mycobacterium bacilli at these end arteriolar complexes (arrow).

C: Proliferation of tuberculous infection at the anterior end plates, resulting in focal destruction of the anterior cortices, and secondary spreading to the adjacent disc space.

D: Further extension of the infectious process occurs underneath the anterior longitudinal ligament, resulting in typical anterior corner destruction on multiple levels.

Fig. 2:

CR of the lumbar spine.

Late tuberculous spondylodiscitis of the lumbar spine. Height loss of the affected vertebral body with ventral collapse (arrow), causing subsequent “gibbus” formation. There is a relative sparing of the intervertebral disc spaces.

CT-Guided aspiration of cold abscess revealed a positive culture M. tuberculosis.

Fig. 3

CT scan of the lumbar spine.

A and B: sagittal CT scan images show cancellous bone destruction and cortical resorption of the endplates because of tuberculous spondylodiscitis at the level L1-L2 (arrowhead in B). C: axial CT image at the level L1-L2 shows anterolateral soft tissue extension (arrows in A and C).

CT guided aspiration was done, and culture was positive for M. tuberculosis.

Fig. 4

CT scan of the abdomen.

Tuberculous spondylodiscitis with destruction of the vertebral end plate with formation of large paraspinal soft tissue abscesses (arrows) and peripheral faint calcifications (arrowheads) (Pott’s disease).

CT-Guided aspiration of cold abscess yielded positive culture for M. tuberculosis.

Fig. 5

MRI of the thoracolumbar spine.

A: T1 hypointensity of the vertebral bodies T9 and T10. B-C: enhancement on the T1-WI after injection of gadoliniumcontrast (arrows). Note intra-osseous abscess formation in the vertebral body of T10 with rim enhancement (arrows). There is anterior epidural extension of the infection with a bilobed-shaped collection and peripheral enhancement (arrowheads). Note also a pre- and paravertebral abscess (asterisks).

TB was proven by demonstration of necrotizing granulomatous infection on histopathological examination after per operative aspiration and biopsy.

Fig. 6

MRI of the thoracolumbar spine.

A: Height loss of L1 with ventral collapse. The bone marrow of the vertebral body L1 is hypointense on T1-WI (arrow), B: The vertebral body L1 enhances (arrow) which is better seen on C: subtraction images before and after injection of gadolinium contrast on image C (arrow), in keeping with spondylitis.

Note also cancellous bone destruction and cortical resorption of the end plates at the level T9-T10 with similar signal changes of the bone marrow. B-C: The disc is enhancing, in keeping with spondylodiscitis.

Note the prevertebral abscess (arrowheads) and skip areas.

Known miliary pulmonary tuberculosis and positive response to tuberculostatics 9 month following onset of treatment provided indirect proof of TB.

Fig. 7

MRI of the thoracolumbar spine.

A-B: Multifocal spondylitis with T2 hyperintensity of the affected vertebral bodies (arrows). C: Contrast enhancement on T1-WI after injection of gadolinium contrast (arrows).

There is also involvement of the posterior elements with T2 hyperintensity of the processus spinosus of T5 on image A (arrowhead).

TB was proven by demonstration of necrotizing granulomatous infection on histopathological examination of lymph nodes at the left groin.

Fig. 8

CT scan of the right hip.

Advanced tuberculous arthritis (stage III) with loss of joint space, acetabular subchondral cyst formation and sequestration (arrows). Histology was positive for necrotizing granulomatous inflammation.

Fig. 9

CR of the wrist.

Late advanced tuberculous arthritis (stage IV) with gross joint destruction. Note the disruption of the Gilula lines, loss of joint spaces and subchondral cysts (arrows). There is also a destruction of the triquetrum and lunate bone and deformity of the distal radius and ulna and widening of the scapholunate space indicating ligamentous tear (asterisks). Histology was positive for necrotizing granulomatous inflammation.

Fig. 10

MRI of the knee.

Chronic tuberculous arthritis.

A-C: There is joint effusion (asterisks) and marked synovial nodular thickening with T2-hypointensity (arrows) and D: enhancement on fat suppressed T1-WI after IV injection of gadolinium contrast (arrow in D). Aspirated material from of the knee yielded positive IGRA-test (interferon-gamma release assay).

Fig. 11

MRI of the sacroiliac joints.

Tuberculous sacroiliac joint infection.

A: Coronal T1-WI on image A show multifocal hypointense lesions with hyperintense peripheral rim (arrows). B: Coronal fat-suppressed T1-WI shows a large lesion with heterogeneously increased signal within the left sacrum and relative hyperintense lesions in the right sacrum (arrows). C: Coronal, contrast-enhanced, fat-suppressed T1-WI shows contrast enhancement on both sides of the left sacroiliac joint, absence of contrast enhancement within the central part of the joint and multiple areas of contrast enhancement within the sacrum (arrows), due to associated disseminated tuberculous osteomyelitis. D: CT scan of the sacroiliac joints confirms marked destruction of the left sacroiliac joint (arrowheads). Ziehl-Neelsen stain was positive following peroperative aspiration of sacroiliac joint.

Fig. 12

Tuberculous dactylitis (spina ventosa). CR of the hands of a child shows expansile, bubbly osteolytic lesions within the diaphysis of the right second and third metacarpal bones (arrows). The patient was known with multifocal tuberculous osteitis and pulmonary TB.

Fig. 13

MRI of the wrist.

A: coronal FS PD-WI of the wrist showing distention of the synovium surrounding the flexor tendons and the ulnar bursa with intralesional rice bodies (arrow).

B: coronal T1-WI. The lesion is isointense to muscle (asterisks).

C: axial FS T2-WI confirms distention of the synovium surrounding the flexor tendons with intralesional rice bodies (arrow).

D: axial FS T1-WI after administration of gadolinium contrast shows peripheral enhancement of the flexor tendon sheaths and adjacent muscles (arrows).

Histopathology of image-guided synovial biopsy revealed tuberculous synovitis.

Fig. 14

MRI of the right ankle in a patient with TB tenosynovitis of the peroneal tendons.

A: T1 hypointense and B: T2 hyperintense effusion with synovial thickening and accumulation of tissue debris and caseous material in the peroneal tendons sheaths (arrows). Note the T2 hypointensity of the synovial thickening on image B. C: FS T1-WI after administration of gadolinium contrast shows marked enhancement of the peroneal tendons sheaths (arrow).

TB infection was proven by aspiration and positive culture.

Fig. 15

MRI of the right shoulder in a patient with TB bursitis of the subdeltoid bursa

A: T1 hypointense and B: T2 hyperintense effusion within the sub-acromial bursa (arrows). Note the peripheral high signal of the thickened wall of the sub-acromial bursa on T1-WI (image B) C: FS T1-WI after administration of gadolinium contrast shows peripheral enhancement of the bursal wall (arrows).

TB infection was proven by aspiration and positive culture.

Fig. 16

CT scan after intravenous contrast administration.

Tuberculous spondylodiscitis of the cervical spine with parapharyngeal soft tissue abscesses (arrows).

CT-Guided aspiration of cold abscess yielded positive culture M. tuberculosis.

Fig. 17

MRI of the neck.

A-C: Large, lobulated, cystic-necrotic lesion at the right side, with mural nodularities, high signal intensity on T2-WI on image A (arrows), low to intermediate signal on T1-WI on image B (arrows) and peripheral enhancement on the fat-saturated T1-WI after injection of gadolinium contrast on image C (arrows), in keeping with a necrotic tuberculous lymphadenopathy with abscess formation.

Histopathology of ultrasound-guided synovial biopsy revealed tuberculous lymphadenitis.

Fig. 18

MRI of the thoracolumbar spine.

A-C: Subcutaneous soft tissue mass in the lumbar region with central T2-hyperintense signal on image A and central T1-hypointense to intermediate signal on image B (arrows), a subtle peripheral T1-hyperintense and T2-hypointense wall and thick peripheral enhancement on the T1-WI after injection of gadolinium contrast (asterisks). There is peripheral T2-hypointense rim on image A suggesting a tuberculous subcutaneous abscess. D: Ultrasound of the lesion with an irregular thick wall and a central heterogeneous content (arrowheads).

CT-Guided aspiration of cold abscess yielded positive culture M. tuberculosis.