Section: Miscellaneous

Review Article

THE ROLE OF RADIOLOGICAL IMAGING IN SKELETAL TUBERCULOSIS

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ABSTRACT

Skeletal tuberculosis accounts for roughly 1 to 3 percent of all tuberculosis and 10 to 15 percent of extrapulmonary disease, yet it is one of the most frequently misdiagnosed manifestations of an infection that still causes more than a million deaths each year. Because its clinical picture is slow and non-specific, imaging usually provides the first objective evidence of disease and continues to guide management from diagnosis to the end of treatment. This review synthesises the role of each imaging modality. Radiography remains the initial test but is insensitive early, since roughly a third of local bone mineral must be lost before a lytic lesion is visible. Computed tomography resolves cortical destruction, sequestra and the calcification that characterises tuberculous cold abscesses and guides percutaneous biopsy. Magnetic resonance imaging is the reference standard: it detects marrow involvement before bone is destroyed, maps epidural and paraspinal spread, and identifies the features that separate tuberculosis from pyogenic infection, brucellosis and malignancy. Ultrasonography localises effusions and abscesses for aspiration, while 18F-FDG PET/CT detects clinically silent multifocal disease and provides a metabolic measure of treatment response. No finding is pathognomonic, so imaging raises or lowers diagnostic probability and directs biopsy—increasingly obtained under image guidance—rather than replacing it. Used together, these modalities shorten the interval to treatment and reduce the deformity and neurological injury that follow late diagnosis.

INTRODUCTION

Tuberculosis remains a leading cause of death from a single infectious agent, with an estimated 10.8 million cases and 1.25 million deaths in 2023.[1] Although most disease is pulmonary, Mycobacterium tuberculosis can involve the skeleton, which accounts for roughly 1 to 3 percent of all tuberculosis and 10 to 15 percent of extrapulmonary disease; the spine is affected in about half of skeletal cases, followed by the large weight-bearing joints [Figure 1].[2-6] The clinical problem is delay. Pain is often mild, systemic features may be absent, and in patients without pulmonary disease the chest radiograph is frequently normal, so a monoarticular arthritis or a collapsing vertebra is easily mistaken for degenerative change, pyogenic infection or metastasis.[3,7] During the resulting latency cartilage is lost, vertebrae collapse and, in the spine, abscess and granulation tissue reach the cord. Because the early clinical signs are unrevealing, imaging usually provides the first objective evidence of disease and then guides its management.[4,8]

No single modality serves every purpose. The tasks are detection before irreversible destruction, characterisation of pattern and extent, differentiation from mimics, guidance of tissue sampling and drainage, and assessment of response to prolonged therapy; contemporary practice is therefore multimodal. This review considers the pathological basis of the imaging appearances and then works through each modality, the major anatomical patterns, the central problem of differential diagnosis, image-guided intervention, response assessment and emerging techniques.

Pathological basis of the imaging appearances: The imaging findings follow from how tuberculosis behaves in bone. Skeletal disease is almost always haematogenous, seeded from a pulmonary or lymphatic focus that may be occult, and lodges in vascular red marrow—the vertebral bodies, long-bone metaphyses and the synovium of large joints.[3,4] In the spine, the blood supply to the anterior subchondral bone explains the characteristic paradiscal onset, with infection beginning on either side of a disc.[4,9,10] Two features distinguish tuberculosis from pyogenic osteomyelitis. First,

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mycobacteria lack the enzymes that digest cartilage, so the disc is relatively spared early even as the adjacent vertebrae are destroyed—the reverse of the early, disc-centred destruction of pyogenic disease, and one of the more useful discriminators.[4,11] Second, the response is granulomatous rather than suppurative: caseous necrosis forms a "cold" abscess that provokes little surrounding reaction, has a thin, well-defined wall, and tracks along fascial planes and beneath the longitudinal ligaments to reach levels remote from the focus, producing skip lesions and smoothly marginated paravertebral and psoas collections.[3,4,10]

Chronic cold abscesses frequently calcify, and amorphous calcification within a paraspinal or psoas collection is close to specific for tuberculosis, being rare in pyogenic infection.[3,10] Devascularised bone may separate as fragmented "kissing" sequestra across a destroyed disc. Where infection involves the posterior elements or spreads circumferentially, the pattern departs from the paradiscal norm and begins to imitate malignancy. In children disease is more often multifocal, the short tubular bones may show an expansile form (spina ventosa), and periosteal reaction is more prominent than in adults.[12,13]

Radiography: Radiography remains the first test and is useful for triage and for following gross structural change, but it is insensitive early: a lytic lesion appears only after roughly 30 to 50 percent of local bone mineral is lost, so a normal film does not exclude disease and, with suggestive symptoms, should prompt cross-sectional imaging.[6,8] In a peripheral joint the changes follow the Phemister sequence—juxta-articular osteoporosis, marginal erosions at the bare areas, and late, slow joint-space narrowing—with relative sparing of the joint space and little early reactive sclerosis, features that distinguish it from pyogenic arthritis; fibrous rather than bony ankylosis marks the burnt-out stage.[6,12]

In the spine, radiographs show the downstream consequences of paradiscal infection: endplate erosion, disc-space narrowing and anterior wedging or collapse producing the angular kyphosis, or gibbus, of advanced disease.[9,14,15] A fusiform paravertebral shadow may be visible, and calcification within it strongly favours tuberculosis.[10,16] In children, tuberculous dactylitis (spina ventosa) and a layered periosteal reaction are characteristic, and in the foot and ankle plain films remain a reasonable initial survey.[13,17] These are findings of established disease; serial films then give a cheap, reproducible record of progression or healing over the long course of treatment.

Ultrasonography: Ultrasound cannot assess bone beyond the cortex but is quick, radiation-free and excellent at detecting fluid. It demonstrates joint effusion and synovial thickening and identifies cold abscesses, including clinically silent collections that contain debris, septa and echogenic foci, and it can reveal a large abscess arising from a modest bony focus.[6,18] Its greatest value is interventional: real-time guidance for aspiration of joint fluid and abscess contents supplies material for microbiology and histology, and it can guide drainage of accessible collections, all without ionising radiation.[6] Its weakness is non-specificity—tuberculous fluid resembles pyogenic and other inflammatory fluid—so it localises and guides sampling rather than establishing the cause.

Computed tomography: Computed tomography excels at the features that matter in tuberculous osteomyelitis: the extent and pattern of cortical and trabecular destruction, sequestra, and calcification within collections.[10,19] It detects erosions and lytic foci earlier and more completely than plain films, and multiplanar reformatting resolves complex anatomy at sites where radiographs are hard to interpret—the craniovertebral junction, the posterior elements and the sternoclavicular, sacroiliac and pelvic joints.4 Fragmented or kissing sequestra flanking a destroyed disc are characteristic, and amorphous calcification in a paravertebral or psoas abscess is close to specific and far better seen on CT than on radiography or MRI.[3,10]

The other indispensable role of CT is guidance of percutaneous biopsy. Because the appearances are suggestive rather than diagnostic, tissue is almost always required, and CT allows accurate needle placement into a vertebral body, a paravertebral collection or an awkward peripheral focus, targeting viable tissue at a lesion's margin.19 Samples go for histopathology, culture and rapid nucleic acid amplification, which supply the diagnosis and drug-susceptibility data imaging cannot. Its costs are radiation and limited soft-tissue contrast, so for the spine it complements rather than replaces MRI.

Magnetic resonance imaging: MRI is the reference standard because it detects disease before bone is destroyed and maps, in one examination, the full extent of osseous, articular and soft-tissue involvement, including the epidural and paraspinal spread that determines neurological risk.[4,19,20] The earliest change is in the marrow, which loses its normal fat signal—low on T1, high on T2 and STIR—well before any cortical breach is visible elsewhere.[20,21] After gadolinium, marrow, synovium and abscess walls enhance. In the spine, MRI shows paradiscal marrow oedema with relative disc preservation, subligamentous spread beneath the longitudinal ligaments, and extension of granulation tissue and pus into the epidural space; it defines the level and severity of cord or cauda equina compression and, occasionally, associated myelitis and arachnoiditis.[10,19,21] The characteristic cold abscess—thin-walled, smoothly marginated, with a rim of enhancement around non-enhancing caseous contents—is shown to particular advantage.[20,22]

In peripheral joints MRI reveals synovial hypertrophy and pannus, effusion, erosions, subchondral oedema and soft-tissue collections long before radiographs change; low T2 signal within thickened synovium and intra-articular rice bodies are suggestive though not specific, and it also demonstrates tenosynovitis and bursitis.[5,22] Two

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caveats temper this. Its sensitivity makes findings frequently non-specific, overlapping with pyogenic infection and neoplasia, and marrow signal resolves slowly, lagging behind clinical improvement, so it is an imperfect early gauge of response if read in isolation.[23]

Nuclear medicine and FDG-PET/CT

Technetium-99m bone scintigraphy is sensitive to skeletal turnover but non-specific and unreliable, since some tuberculous lesions are photopenic; a negative scan does not exclude disease, and its role is now limited.[3,18] F-FDG PET/CT has been more useful. Active tuberculous inflammation is FDG-avid, and whole-body imaging in a single acquisition addresses two problems conventional imaging handles poorly.[24,25] First, it detects clinically unsuspected, spatially separate foci—skeletal disease is multifocal more often than is apparent—revealing additional bone, nodal or visceral sites that alter staging and guide biopsy.[25,26] Second, because uptake reflects metabolic activity rather than residual scarring, a fall in the maximum standardised uptake value between baseline and follow-up gives an early, quantitative measure of response, valuable in extrapulmonary and drug-resistant disease where microbiological monitoring is difficult.[24-26] FDG is not specific to infection—malignancy, other infections and surgery all take it up—so intense uptake still needs interpretation in context and tissue confirmation when in doubt; with cost and availability constraints, PET/CT is a problem-solving tool rather than a first-line test [Figure 2].[24]

Spinal tuberculosis: The spine is the commonest and most consequential site, and its imaging has a recognisable grammar; four patterns of vertebral involvement are described.[9,23,27] The paradiscal pattern is commonest: infection begins in anterior subchondral bone and spreads across the disc, producing paired paradiscal lesions, endplate erosion and disc-space loss.[9,10] The central pattern involves the body itself, sometimes producing vertebra plana that mimics malignant collapse or, in children, Langerhans cell histiocytosis.[21,27] The anterior subligamentous pattern tracks beneath the anterior longitudinal ligament, scalloping several contiguous bodies—a strong pointer to tuberculosis.[10,28] The posterior pattern, involving pedicles, laminae and facet joints, is uncommon, mimics neoplasm and may compress the cord from behind.[4,21]

Two consequences dominate management. The cold abscess collects in paravertebral tissues and tracks to distant sites—prevertebral and retropharyngeal collections in the cervical spine, fusiform paraspinal abscesses in the thorax, and psoas abscesses reaching the groin in lumbar disease—typically large, thin-walled and often calcified.[3,29] The neurological consequence is cord compression, usefully divided into a reversible early-onset form during active disease (abscess, granulation tissue, caseation) and a late-onset form from mechanical causes such as the internal gibbus of a healed kyphosis, which carries a worse prognosis; MRI alone distinguishes these and is indispensable for surgical planning.[9,30,31] Disease is frequently multilevel with non-contiguous skip lesions, so whole-spine MRI is appropriate when tuberculosis is suspected.[29,32]

Extraspinal osteoarticular tuberculosis: Outside the spine, tuberculosis usually causes a chronic monoarticular arthritis of a large weight-bearing joint. About 90 percent is monoarticular; the hip and knee predominate, followed by the ankle, sacroiliac joint, shoulder, elbow and wrist, with multifocal disease in roughly a tenth of cases [Figure 3].[5,6] The insidious course again favours delay, and MRI is usually needed to define extent. Early synovial infection produces effusion, synovial hypertrophy and marginal erosions at the bare areas, with relative joint-space preservation and little reactive sclerosis; as disease advances cartilage is lost, subchondral bone is destroyed on both sides, and the end stage is severe destruction with fibrous ankylosis.[6,12,22] On MRI, low T2 signal within thickened synovium, rim-enhancing collections and rice bodies are suggestive, and it maps associated abscesses, sinus tracts and tenosynovitis.[22]

Certain forms deserve mention. Tuberculous osteomyelitis without joint disease can mimic a Brodie abscess or a bone tumour in a long-bone metaphysis.[5] The foot and ankle, with many bones and joints affected in varied combinations, demand a high index of suspicion.[17] Soft-tissue and bursal disease, sometimes with minimal bony change, is well shown by ultrasound and MRI, and in children dactylitis and multifocal involvement are characteristic.[13,18]

Differentiating tuberculosis from its mimics: No finding is pathognomonic; the task is to weigh features that raise or lower probability against the main imitators—pyogenic spondylodiscitis, brucellar spondylitis, other granulomatous infections and neoplasm—recognising that imaging shapes pre-test probability and the urgency of biopsy rather than replacing tissue diagnosis. The most studied distinction is from pyogenic spondylodiscitis. Features favouring tuberculosis include a thoracic or thoracolumbar location, relative disc preservation despite extensive vertebral destruction, subligamentous spread over three or more levels and skip lesions, a thin- and smooth-walled paravertebral or intraosseous abscess, posterior-element involvement, heterogeneous enhancement, and calcification in a paraspinal collection [Figure 4].[11,22,33,34] Pyogenic infection more often affects the lumbar spine, destroys the disc early and centrally, produces ill-defined thick-walled phlegmon, and enhances more homogeneously.[22,33] No single feature is decisive, but combinations are, and several groups have distilled them into weighted MRI scores with good accuracy in derivation cohorts; external validation remains limited, so these support rather than replace clinical and microbiological assessment.[28]

Brucellar spondylitis is a particular difficulty where both are endemic, since it too can spare the disc;

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relative to tuberculosis it causes less destruction and deformity, smaller collections and a lower-lumbar predilection, but overlap is substantial and serology is usually required.[35] Diffusion-weighted imaging and the apparent diffusion coefficient are often oversold: infective lesions generally show higher values than malignant marrow, which can occasionally separate infection from tumour, but ranges overlap too much for reliable individual use and do not separate tuberculous from pyogenic infection; perfusion MRI is likewise supplementary rather than decisive.[36,37]

Separating tuberculosis from malignancy matters most for the central and posterior patterns and for solitary lesions. Endplate destruction with disc involvement, a paravertebral abscess and an intact posterior cortex favour infection; pedicle destruction with disc sparing, a focal soft-tissue mass and discrete marrow deposits favour metastasis—but each is a recognised mimic of the other, and where doubt persists biopsy is the arbiter.[5,21]

Image-guided intervention

Imaging is also therapeutic and confirmatory. Because the appearances are non-specific and drug resistance makes susceptibility testing essential, obtaining tissue is now the rule, and percutaneous image-guided biopsy has largely replaced open sampling; under CT, or ultrasound for accessible targets, a needle samples viable tissue at a lesion's periphery for histopathology, culture and molecular testing with detection of rifampicin resistance.[19] A negative culture does not exclude the diagnosis, and repeat or larger-bore sampling is sometimes needed. Image guidance also enables minimally invasive drainage of large paravertebral, psoas or soft-tissue cold abscesses, relieving symptoms, reducing mass effect and providing abundant material for microbiology, and it may defer or avoid open surgery in selected patients—procedures that depend entirely on prior cross-sectional mapping, making the diagnostic and interventional roles inseparable.[19]

Treatment response and follow-up: Skeletal tuberculosis is treated with prolonged multidrug chemotherapy, often a year or more in spinal disease, and response assessment is largely an imaging exercise—though structural and metabolic markers heal at different rates, and clinical and inflammatory-marker improvement remain primary, with imaging providing corroboration and detecting complications.[9,23] On MRI, healing appears gradually: resolution of marrow oedema with return of fatty signal, declining enhancement, shrinkage of collections, and bony bridging across the treated segment.[10,23] Timing is crucial: marrow signal and enhancement lag well behind clinical recovery, so persistent change on an early follow-up scan does not indicate failure and must not trigger premature changes in management; conversely, increasing destruction, enlarging collections, new deformity or progressive compression are genuine signals of failure warranting reassessment, including for drug resistance.[23]

Metabolic imaging offers an earlier readout, since FDG uptake tracks activity rather than scarring: a fall in standardised uptake values on serial PET/CT can indicate response before structural resolution, useful in multifocal and drug-resistant disease, though the usual constraints of cost, availability and non-specificity reserve it for selected cases.[24-26]

Emerging techniques and future directions

Several developments are extending imaging's contribution. Whole-body MRI, including diffusion-weighted sequences, allows radiation-free survey for multifocal disease in one examination, attractive in children and for skip lesions, and is increasingly feasible as acquisition times fall.[32] Advanced sequences—diffusion tensor imaging, chemical-shift imaging and dynamic contrast-enhanced perfusion—continue to be evaluated for separating tuberculosis from pyogenic infection, brucellosis and tumour, with promising but not decisive results.[35-37] Hybrid PET/MRI, combining metabolic information with high soft-tissue contrast at lower dose than PET/CT, is an appealing prospect as availability grows.[25] The most active frontier is computational: radiomics and machine-learning models applied to CT and MRI aim to classify infective spondylodiscitis by aetiology, with early studies reporting encouraging discrimination.[28,35] These remain investigational—mostly single-centre, retrospective and inadequately validated externally—and their value will depend on prospective, multi-institutional testing and integration with the microbiological and molecular diagnostics that remain the reference standard.

CONCLUSION

Skeletal tuberculosis is defined by delay: an indolent, non-specific illness in which imaging usually provides the first objective evidence of disease and then directs management to its conclusion. Each modality has a defined, largely non-overlapping role. Radiography triages and follows gross change but misses early disease; CT resolves bone destruction, sequestra and the calcified cold abscess and guides sampling; MRI is the reference standard, detecting marrow disease before bone is lost, mapping the epidural and paraspinal spread that governs neurological outcome, and identifying the features that shift probability toward or away from tuberculosis; ultrasound localises fluid and guides aspiration; and FDG-PET/CT finds occult multifocal disease and measures metabolic response. The unifying limitation is that no finding is pathognomonic, so imaging raises or lowers probability and directs biopsy rather than replacing it, with definitive diagnosis resting on histology and microbiology—now most often obtained under image guidance and supplemented by rapid molecular testing for resistance. Used together and interpreted against the clinical and epidemiological background, these tools shorten the interval to treatment and reduce the deformity and neurological

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injury that make an eminently treatable disease so damaging when it is recognised late. Emerging whole-body, hybrid and computational methods are likely to refine, but not overturn, this multimodal, tissue-anchored approach.

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