Tuberculosis

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Definition

Tuberculosis (TB) is a potentially fatal contagious disease that can affect almost any part of the body but is mainly an infection of the lungs. It is derived from the Neo-Latin word: "Tubercle" which means a round nodule/swelling and "Osis" which means condition. The causative organisms is *Mycobacterium tuberculosis*, which is of two types, namely: human and *Mycobacterium bovis* which is found in animals.

The other causative organisms are (1) *Mycobacterium africanum* and (2) *Mycobacterium microti*. The Non-Mycobacterium Genus are (1) *Mycobacterium leprae*, (2) *Mycobacterium avium*, and (3) *Mycobacterium asiaticum*, all together constitute *M. tuberculosis* complex along with *M. africanum*, *M. bovis*, *M. canetti,* and *M. microti*.

The characteristics of *Mycobacterium tuberculosis* is that it is a gram-positive organism, which is an obligatory aerobe, non-spore-forming and non-motile rod. It is a mesophile about 0.2–0.6 × 2.4 μm^3. It has a slow generation time of about 15–20 h when it may contribute to virulence. It has a lipid-rich cell wall which contains mycolic acid with 50% being its cell wall dry weight. It is acid fast which retains acid stains, and confers resistance to detergents and antibacterials.

Classification

1. Pulmonary TB which may be a primary disease or a secondary disease.
2. Extrapulmonary such as
   (a) Lymph node TB
   (b) Pleural TB
   (c) TB of upper airways

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(d) Skeletal TB
(e) Genitourinary TB
(f) Miliary TB
(g) Pericardial TB
(h) Gastrointestinal TB
(i) Tuberculous meningitis
(j) Less common forms

Epidemiology

In 2011, there were an estimated 8.7 million incidence cases of TB globally. It is equivalent to 125 cases in 1,00,000 population.

- Asian: 59%
- African: 26%
- Eastern Mediterranean Region: 7.7%
- The European Region: 4.3%
- Region of the America: 3%

India is the highest TB burden country accounting for more than one-fifth of the global incidence (global incidence is 9.4 million while India annual incidence is 1.96 million).

Incidence of tuberculosis: Its annual incidence in different age groups shows a maximum in the age groups above 55 years and the annual deaths from infections due to tuberculosis than others such as HIV, measles, STD, malaria, and tropical diseases.

Spread of Tuberculosis

Commonest form of spread is coughing without covering the mouth, crowded places with poor ventilation, and due to spitting everywhere.

Severe Symptoms

1. Persistent cough
2. Chest pain
3. Coughing with bloody sputum
4. Shortness of breath
5. Urine discoloration
6. Cloudy and reddish urine
7. Fever with chills
8. Fatigue

Types

Pulmonary TB

1. *Primary tuberculosis*: The infection of an individual who has not been previously infected or immunized is called primary tuberculosis or Ghon's complex or childhood tuberculosis. Lesions forming after infection is peripheral and accompanied by hilar which may not be detectable on chest radiography.
2. *Secondary tuberculosis*: The infection of an individual who has been previously infected or sensitized is called secondary or post primary or reinfection or chronic tuberculosis.

Extrapulmonary TB

These are found in 20% of patients of TB Patient.

Affected sites in body are:

1. *Lymph node TB (tuberculous lymphadenitis)*: Seen frequently in HIV-infected patients.

   Symptoms: Painless swelling of lymph nodes most commonly at cervical and supraclavicular (Scrofula) regions. Systemic systems are limited to HIV-infected patients.
2. *Pleural TB*: Involvement of pleura is common in primary TB and results from penetration of tubercle bacilli into pleural space.
3. *TB of upper airways*: Involvement of larynx, pharynx, and epiglottis. Symptoms: dysphagia, chronic productive cough.
4. *Genitourinary TB*: 15% of all Extrapulmonary cases. Any part of the genitourinary tract gets infected. Symptoms: urinary frequency, dysuria, hematuria.
5. *Skeletal TB*: Involvement of weight-bearing parts like spine, hip, knee. Symptoms: pain in hip joints and knees, swelling of knees, trauma.
6. *Gastrointestinal TB*: Involvement of any part of GI tract. Symptoms: abdominal pain, diarrhea, weight loss.
7. *TB meningitis and tuberculoma*: 5% of all extrapulmonary TB. Results from hematogenous spread of 10 and 20 TB.
8. *TB pericarditis*: 1–8% of all extrapulmonary TB cases. Spreads mainly in mediastinal or hilar nodes or from lungs.
9. *Miliary or disseminated TB*: Results from hematogenous spread of tubercle bacilli. Spread is due to entry of infection into pulmonary vein producing lesions in different extrapulmonary sites.
10. *Less common extrapulmonary TB*: It may manifest as uveitis, panophthalmitis, painful hypersensitivity, and related phlyctenular conjunctivitis.

Table 7.1 Interpretation of the tuberculin test

Diameter of induration	Interpretation	Action
Less than 6 mm	Negative	Previously vaccinated individuals may be given BCG, provided there are no contraindications.
6 mm or greater, but less than 15 mm	Hypersensitive to tuberculin protein. May be due to previous TB infection, BCG or exposure to atypical mycobacteria.	Should not be given BCG.
> – 15 mm	Strongly hypersensitive to tuberculin protein. Suggestive of TB infection or disease.	Should not be given BCG. Refer for further investigation and supervision which may include chemotherapy.

Diagnosis

1. Bacteriological test: (a) Ziehl-Neelsen stain, (b) Auramine stain (fluorescence microscopy).
2. Sputum culture test: (a) Lowenstein–Jensen (LJ) solid medium: 4–18 weeks, (b) liquid medium: 8–14 days, (c) agar medium: 7–14 days.
3. Radiography: Chest X-ray (CXR).
4. Nucleic acid amplification: Species identification; several hours; low sensitivity, high cost; most useful for the rapid confirmation of tuberculosis in persons with AFB-positive sputa; has utility value in AFB-negative pulmonary tuberculosis and extrapulmonary tuberculosis.
5. Tuberculin skin test (PPD): Injection of fluid into the skin of the lower arm. 48–72 h later checked for a reaction (Table 7.1). Diagnosis is based on the size of the wheal. 1 dose = 0.1 mL contains 0.04 μg tuberculin PPD.
6. Other biological examinations: Cell count (lymphocytes), protein (Pandy and Rivalta tests)—Ascites, pleural effusion, and meningitis.

Preventive Measures

1. Mask
2. BCG vaccine
3. Regular medical follow-up
4. Isolation of patient
5. Ventilation
6. Natural sunlight
7. UV germicidal irradiation

Table 7.2 Table showing the management with mechanism of action in tuberculosis

First-line drugs	Second-line drugs
Isoniazid	Cycloserine
Rifampin	Ethionamide
Rifapentine	Levofloxacin
Rifabutin	Moxifloxacin
Ethambutol	Gatifloxacin
Pyrazinamide	*p*-Aminosalicylic acid
	Streptomycin
	Amikacin/Kanamycin
	Capreomycin

Drugs	Mechanism of action
Isoniazid	Inhibits mycolic acid synthesis.
Rifampicin	Blocks RNA synthesis by blocking DNA-dependent RNA polymerase
Pyrazinamide	Bactericidal—slowly metabolizing organism within acidic environment of phagocyte orcaseous granuloma
Ethambutol	Bacteriostatic Inhibition of arabinosyl transferase
Streptomycin	Inhibition of protein synthesis by disruption of ribosomal function

BCG vaccine: Bacille Calmette Guerin (BCG), First used in 1921, is the only vaccine available today for protection against tuberculosis. It is most effective in protecting children from the disease, given 0.1 mL intradermally, duration of protection 15–20 years, efficacy 0–80%. It should be given to all healthy infants as soon as possible after birth unless the child presented with symptomatic HIV infection.

Management (Table 7.2).

Monitoring side effects of common antitubercular drugs (Table 7.3).

Dosage regimen:

1. Intensive phase + continuation phase.
2. HREZ (2 months) + HRE (4 months).

Recommended dosage for the initial treatment of tuberculosis in adults (Table 7.4):

Table 7.3 Table showing the side effects of drugs

Drug	Side effect	Management
Rifampin	Rash, liver dysfunction, flulike syndrome, red-orange urine, drug interactions, fever chills.	Observe patient, stop drug if significant. Monitor AST/limit alcohol consumption/monitor hepatitis symptoms. Administer at least twice weekly/limit dose to 10 kg/adults. Reassure patients. Consider monitoring levels of other drugs affected by Rifampin, especially with contraceptives, anticoagulants, and digoxin/avoid use of protease inhibitors. Stop drug.
Isoniazid	Hepatitis, peripheral neuritis, optic neuritis, seizures.	Monitor AST/limit alcohol consumption/monitor for hepatitis syndrome/educate patient/stop drug at first symptoms of hepatitis (nausea, vomiting, anorexia, flulike syndrome); administer B6/stop drug
Pyrazinamide	Hepatitis, hyperuricemia	Monitor AST/limit daily dosage to 15–30 mg/kg/ discontinue with signs and symptoms of hepatitis. Monitor uric acid levels only in cases of gout and renal failure.
Ethambutol	Optic neuritis	Use 25 mg/kg daily only for first 2 months (except in drug resistant tuberculosis), then lower daily dose to 15 mg/kg; when possible monitor/visible acuity (eye chart) and red and green color chart (Ishihara color book); stop drug at first change of vision.
Streptomycin, amikacin, capreomycin	Ototoxicity, renal toxicity	Limit dose and duration of drug as much as possible/avoid daily therapy in patients >50 years old/monitor BUN and serum creatine levels and possibly conduct audiometry before as treatment needed during therapy/question patients regarding tinnitus/stop drug if patients develop tinnitus.

Table 7.4 Table showing the dosage of antitubercular drugs used

Drug	Daily dose	Thrice weekly dose
Isoniazid	5 mg/kg, maximum 300 mg	15 mg/kg, maximum 900 mg
Rifampicin	10 mg/kg, maximum 600 mg	10 mg/kg, maximum 600 mg
Pyrazinamide	20–25 mg/kg, maximum 2 g	30–40 mg/kg, maximum 3 g
Ethambutol	15–20 mg/kg	25–30 mg/kg

Dots

Directly observed treatment, short-course.

DOT means that a trained health care worker or other designated individual provides the prescribed TB drugs and watches the patient swallow every dose (sure cure for TB).

Multidrug Resistance TB

TB is caused by strains of *Mycobacterium tuberculosis* that are resistant to at least isoniazid and rifampicin, the most effective anti-TB drug. Globally, 3.6% are estimated to have MDR-TB. Almost 50% of MDR-TB cases worldwide are estimated to occur in China and India.

Extensively Drug Resistance TB

Extensively drug-resistant TB (XDR-TB) is a form of TB caused by bacteria that are resistant to isoniazid and rifampicin (i.e., MDR-TB) as well as any fluoroquinolone and any of the second-line anti-TB injectable drugs (amikacin, kanamycin, or capreomycin).

Tuberculosis and HIV

Worldwide the number of people infected with both HIV and TB is rising. The HIV virus damages the body's immune system and accelerates the speed at which TB progresses from a harmless infection to a life-threatening condition. The estimated 10% activation of dormant TB infection over the life span of an infected person is increased to 10% activation in 1 year, if HIV infection is superimposed. It is the opportunistic infection that most frequently kills HIV-positive people.

Epidemiological Impact

Reactivation of latent infection: People who are infected with both HIV and TB are 25–30 times more likely to develop TB again than people only infected with TB. Primary infection that is new tubercular infection in people with HIV can progress to active disease very quickly. Recurring infection—in people who were cured of TB.

Diagnosis of TB in People with HIV

HIV-positive people with pulmonary TB may have a higher frequency of having sputum negative smears. The tuberculin test often fails to work, because the immune system has been damaged by HIV. It may not even show a response even though the person is infected with TB. Chest X-ray will show less cavitation. Cases of extrapulmonary TB are more common.

Tuberculosis of Bones and Joints

Tuberculosis of the Spine

Spine is the most frequent site of osseous involvement by TB in about 50% of cases. The disease was first described by Sir Percival Pott in 1779, hence the name Pott's disease. There has been a resurgence of the disease in the developed countries following the HIV pandemic. It is defined as an infection by *Mycobacterium tuberculosis* of one or more of the extradural components of the spine, namely the vertebra, intervertebral disks, paraspinal soft tissues, and epidural space.

Pathophysiology

It is usually by hematogenous route. Perivertebral arterial or venous plexus is still in debate, but arterial route is considered more important. The primary focus in the lung or other extra-osseous foci such as lymph nodes, GIT, or any other viscera. The lower thoracic and lumbar vertebrae are most often affected followed by middle thoracic and cervical vertebrae. The C2–C7 region is reportedly involved in 3–5% of cases and the atlanto-axial articulation in <1% of cases. Usually two continuous vertebrae are involved but several vertebrae may be affected, "skip lesions" and solitary vertebral involvement may occur. The so-called skip lesions or a second lesion not contiguous with the more obvious lesion is seen in 4–10% of cases. The infection begins in the cancellous area of the vertebral body commonly in the paradiscal location and less often in the centrum or anterior surface. The vertebral body becomes soft and gets easily compressed to produce either wedging or a total collapse. Anterior wedging is commonly seen in the dorsal spine. This produces kyphus with a gibbus deformity. Spread of infection can occur beneath the anterior longitudinal ligament, involving adjacent vertebral bodies. It spreads to the adjacent disc. The intervertebral disk resists infection by *Mycobacterium tuberculosis* probably due to a lack of proteolytic enzymes in the *Mycobacterium as compared with pyogenic infection and* disk destruction begins only when two vertebral bodies are so involved that the disk loses its nutritional support. Hence, disk space narrowing occurs later and is less marked in tubercular infection *as opposed to pyogenic infection.* The posterior elements may be affected initially or predominantly in some persons. A marked exudative lesion due to hypersensitivity reaction to *Mycobacterium* occurs results in formation of thick pus. This pus contains serum, leukocytes, caseous material, tubercle bacilli, and bone fragments which tracks through the pre and paravertebral soft tissues forming abscesses. The exudate penetrates ligaments and follows the path of least resistance along fascial planes, blood vessels, and nerves to distant sites from the original bony lesion as cold abscess. The abscesses may further extend into the spinal canal producing an epidural abscess and cord compression.

Clinical Features

It can affect any age group but majority under 30 years of age. Rare in the 1st year of life, but when it occurs, it *tends to be more severe* with greater bone destruction

and multiple vertebral involvement. Some patients may be afebrile and free of systemic symptoms until late stage of the disease, others may present with constitutional symptoms before symptoms related to the spine manifest. The usual presentation is with persistent spinal pain, local tenderness, and limitation of spinal mobility. The ESR is elevated in more than 80% of cases and tuberculin skin test is usually positive. Paraparesis in about 20–30% of all patients with a much higher incidence in cervical region (40% cases quadriparesis).

There is early onset paraplegia usually due to *cord compression* by epidural abscess or granulation tissue, pathological subluxation or dislocation, sequestered bone or disk fragments. *Nonmechanical causes* include inflammatory cord edema due to vascular stasis and toxins or cord granulation tissue due to passage of tuberculous inflammation to the meninges and eventually the cord. Rarely paraplegia may be due to cord infarction due to endarteritis. Late onset paraplegia is due to dural fibrosis, severe kypho-scoliotic deformity, spinal canal stenosis, gliosis of cord, or sequestra from vertebral body.

Late onset paraplegia has a *much less favorable prognosis* than early onset paraplegia.

Laboratory Investigations

1. Relative lymphocytosis, a low level of hemoglobin and a raised ESR are found in *active tubercular disease.*
2. The Mantoux test is nondiagnostic in an endemic region and may be negative in an immuno-deficient individuals.
3. The sensitivity of AFB staining may vary from 25% to 75%.
4. Culture of AFB requires a long incubation period of 4–6 weeks, although bactec radiometric culture takes <2 weeks.

Serological tests: Nondiagnostic in lesions with a low level of bacilli. IgG and IgM titers show significant differences between the initiation of treatment and at 3 months later (*can be used for follow up*). PCR is an efficient and *rapid* method of diagnosis which can differentiate between typical and atypical mycobacteria. However, a positive result is *not a substitute for culture and is NOT indicative of the activity* of the disease. It does not differentiate live from dead microorganisms.

Imaging Modalities

1. *Conventional radiographs*: Initial investigation which is often negative in early disease. More than 30–50% of mineral must be lost before a radiolucent lesion becomes conspicuous on the plain films and this takes about *2–5 months* with limited evaluation of cranio-vertebral junction, cervico-dorsal junction, posterior neural arches, and the sacrum.
2. *Computed tomography*:

   Advantages: It helps in the early detection of bone and soft tissue changes when plain films are normal and acts as a better anatomic localization and

characterization of lesions. It helps in the evaluation of areas difficult to evaluate on plain films such as cranio-vertebral junction, cervico-dorsal junction, and sacrum. It helps in providing guidance for biopsy and surgical approach with early signs as inflammatory marrow changes in the vertebral body are not well depicted. The effect of extradural disease on the thecal sac and its contained spinal cord and neural elements is difficult to evaluate properly.

3. *Magnetic resonance imaging*:
   It is the modality of choice with advantages being
   (a) Multiplanar capability
   (b) The direct demonstration of early bone marrow involvement or edema
   (c) Unsurpassable assessment of spinal canal and neural involvement
4. Soft tissue and intraosseous abscesses are also well demonstrated on MR imaging with a higher sensitivity for early infiltrative disease including endplate changes and marrow infiltration than bone scan and plain films.

MRI scores over CT in detection of early disease (marrow edema). Skip lesions are more easily and more often detected. Incidence of multilevel noncontiguous vertebral tuberculosis is generally reported to be between 1.1% and 16%. MRI scores over CT also in detection of epidural, meningeal, and cord involvement and in planning the surgical approach.

Diffusion weighted MR imaging has been applied in an attempt to distinguish between tubercular and neoplastic vertebral disease (mets, myeloma). In one study the authors concluded that *DW-MRI and ADC values may help in the differentiation* of spinal tuberculosis from other lesions of similar appearance.

Disadvantages:

(1) Calcification which is the hallmark of tubercular infection and small bone fragments is not readily detectable. (2) Gradient echo images (GRE) demonstrate calcification better. (3) Small bone fragments in an epidural abscess are important to detect when surgical management is being considered so that they can be accurately removed. (4) Interventional procedures are difficult to perform with MR imaging.

Nuclear Medicine Scintigraphy

Technetium di-phosphonate study is an economical but nonspecific tool for early detection. Sensitivity is 87.5–95%. Radiotracer uptake is usually increased in osseous tuberculous infection and may reveal multiple sites in disseminated disease which is *a nonspecific finding that may mimic metastases.*

False-negative bone scans may be obtained in disseminated tuberculosis, cervical spine lesions, isolated neural arch lesions and posterior neural arch lesions are more readily detectable when cross-sectional nuclear medicine imaging is performed.

The pitfalls of nuclear imaging

1. Limited anatomic resolution,
2. Non-specificity.
3. False-negative examinations.

Advantage: may help identify a focus of interest; further imaging of the area in question, along with additional tissue sampling, can then be performed to aid in diagnosis.

PET CT: PET-CT (FDG PET) have high sensitivity for detection of chronic osteomyelitis. The increased FDG uptake in regions of active granulomatous inflammation can delineate the sinus tracks without the need for contrast. With the incorporation of CECT in the PET/CT protocol, the complete extent of bone marrow and soft tissue involvement can be delineated. Metabolically active disease can be distinguished from residual fibrotic tissue.

As in oncological imaging, PET/ CT plays a useful role in determining multiple occult foci of involvement in a single scan. It can also serve as a valuable baseline for monitoring response to treatment and provide information on disease spread. It is also a useful tool to guide the site of biopsy or other interventional procedures.

Limitations of PET-CT

1. The uptake patterns that are indistinguishable from malignant processes.
2. Though high standardized uptake values (SUVs) greater than 2.5 have been attributed to malignant lesions, high values (up to 21) have been seen in tuberculosis as well.
3. 2 time points scan: It includes delayed imaging at *90–120* min. *At malignant sites, the FDG uptake continues to increase* for several hours.
4. *In inflammatory lesions, uptake peaks at approximately 60 min* after administration and the SUVs either stabilize or decline thereafter.
5. Another approach to increase the diagnostic accuracy of PET is the combined use of 18F-FDG and C-11 acetate as the latter accumulates in tumors and not in inflammatory lesion.
6. The exact role of FDG-PET and PET/CT in TB and other inflammatory diseases is evolving and is NOT as yet clearly defined.
7. With the development of newer and more specific radiotracers like positron emitter labeled antituberculous drug molecules such as INH and rifampicin in the future, PET/CT may play a significant role in establishing an early diagnosis and effective monitoring of therapeutic response.

Imaging Appearances

1. Vertebral, disk space, and the soft tissue changes.
2. Para-vertebral abscesses are an important early feature of Pott's spine.

3. The incidence varying from 55% to 96%—*rarely may precede any visible vertebral lesion*.
4. It is usually antero-laterally and is less often directly posteriorly in the peri-dural space.
5. The associated changes in the spinal cord and rare variants of disease such as extra-osseous extradural granuloma may be detected by modern imaging techniques.

Conventional Radiography

It depends on the initial focus of infection within the vertebra—paradiscal, central, anterior subperiosteal/subligamentous, and appendiceal/neural arch.

1. Paradiscal: It is the most common type, aka *marginal, intervertebral, subarticular, or metaphyseal lesion.* It most often begins in the anterior part of the vertebral body either superiorly or inferiorly adjacent to the endplate. Two adjacent vertebral bodies are involved in about 50% of cases. There is demineralization and loss of definition endplates with little or no periosteal reaction or reactive sclerosis affect the remainder of the vertebra. As infection spreads, the adjacent intervertebral disk becomes involved with narrowing of the disk space. Rarely the disk space may remain intact for a longtime. This makes the diagnosis difficult, since disk space narrowing constitutes an *important diagnostic feature of infection and serves to differentiate tuberculosis from fracture, malignant disease, solitary myeloma, and porotic collapse*. There is anterior wedging or collapse with varying degrees of kyphosis. The *scoliosis is asymmetric or unilateral destruction* of vertebral bodies and disks and is virtually confined to the lower thoracic and lumbar vertebrae.
2. Central: There is a lytic area with absence of normal trabeculae in the central portion. It gradually enlarges and the vertebral body may expand or balloon out like a tumor. In later stages concentric collapse occurs, almost resembling a vertebra plana. The paravertebral shadows may be absent or minimal. *The disk space is either not affected or only minimally affected.*
3. Anterior subperiosteal: It begins at the anterior vertebral margin underneath the periosteum and spreads beneath the ALL with subtle anterior erosions of multiple vertebrae. The clinical symptoms are severe in relation to the minor radiographic abnormalities. The disk destruction maybe late and anterior erosions are difficult to detect on plain radiographs.
4. Appendiceal or neural arch tuberculosis: Ranges from 2% to 30% of cases, usually in contiguity with vertebral body involvement. Isolated involvement of the neural arch is rare (<2% in non-endemic and < 5% in endemic areas). The plain films—limited evaluation of the neural arch lesions (CT and MR useful). NAT most commonly affects the cervical and upper dorsal spine (*unlike classical spinal tuberculosis which is most common at the lumbo-dorsal junction). They have a* tendency towards pedicular and laminar involvement in (*pyogenic*

spondylitis—predilection for the facet joints). The pedicle—most common site. Usually unilateral and the radiographic findings in NAT include—pedicular or laminar destruction with erosion of the *adjacent ribs* in the thoracic region. The erosion of *posterior cortex* of the vertebral body with relative sparing of the intervertebral disks along with a large para-spinal maas is indicative of NAT.

Importance of NAT: Recognition of coexisting posterior and anterior involvement is essential for presurgical planning because decompression can lead to instability, anterior and posterior stabilization must be performed. Cases of isolated NAT respond well to simple decompression and debridement followed by chemotherapy. Paraplegia associated with NAT reportedly has a better prognosis than that with typical spinal tuberculosis.

Abscess formation: Paravertebral soft tissue opacity and is usually out of proportion to the degree of osseous destruction. It is commonly bilateral and uniform and may be globular indicating pus under tension which may be minimal in the central variety of tubercular lesion. In the cervical region it is seen as widening of the prevertebral soft tissues and in the dorsal spine seen as the posteromedial pleural line is displaced laterally and the abscess produces as typical fusiform shape called the "birds nest" appearance.

The aneurysmal effect—may be found with an anterior paravertebral or subligamentous abscess between D4 and D10 levels. There are shallow erosions or gouge defects on the anterior surface of vertebral bodies due to transmitted aortic pulsations. The intervertebral disks being resistant to pressure atrophy are spared. An abscess at the dorsolumbar junction has an indistinct converging lower border and is referred to as a "petering abscess." In the lumbar region the abscess tends to track along the psoas producing bulging of the psoas outline. Calcification in the paraspinal abscesses is considered pathognomonic of tuberculosis as *nontubercular abscesses rarely calcify.* Tuberculous abscesses of the psoas muscle calcify in two distinct patterns: (1) faint amorphous deposits or (2) tear drop shaped calcification. *With healing the calcification tends to become more dense* and in rare instances may be seen to diminish or disappear on serial radiographs.

Computed Tomography

Four patterns of bone destruction have been described on CT:

1. Fragmentary—47%,
2. Osteolytic—33%,
3. Subperiosteal—10%,
4. Well-defined lytic with sclerotic margins—10%.

The fragmentary type: It is the most frequent and characteristic with numerous residual small bone fragments embedded in a soft tissue mass. Similar appearance has been described in involved areas of vertebral appendages. *Why bone*

fragments?—because tuberculous inflammatory exudates lack proteolytic enzymes required to lyse bone. These bone fragments may migrate into the surrounding structures including the spinal canal, paravertebral soft tissues, and psoas muscles. They are easily detected by CT. *This is in contrast to pyogenic spondylitis that shows multiple small erosions like a "pepper pot" and no calcification.* There is disk space narrowing, multilevel involvement with kyphosis (particularly well seen on multiplanar reconstructions). There is obliteration of the fat planes around the vertebral body very early in the evolution of abscess formation. These soft tissue abscesses are characterized by their CT attenuation values—high attenuation lesions being defined as *granulation tissue*, low density lesions defined as *abscesses or caseous material.* CT is ideally suited to demonstrate small amounts of calcification which are not visible on plain radiographs. Pre-contrast scan—thick nodular rim of increased tissue with attenuation of an abscess may be seen (*represents the hypervascular, hypercellular fibrotic wall of the inflammatory cavity*). IV contrast—usually strong rim enhancement around low attenuation multiloculated collection. This is also called the "rind sign." Granulation tissue shows a more homogenous enhancement with an epidural extension of these soft tissue masses with cord compression. There are some small bone fragments some distance away from the actual site of vertebral destruction.

A combination of multi-locular and calcified para-spinal abscess which are thick and well-enhancing irregular rim with the presence of vertebral body bony fragmentation is a strong indication of tuberculous (*rather than pyogenic infection or neoplasm*). A CT-guided biopsy procedures as well providing material for histo/cytopathology, AFB staining, and culture in equivocal cases.

Magnetic Resonance Imaging

T1—usually decreased marrow signal and loss of cortical definition.

T2—a relative increase in signal intensity within involved vertebral bodies and disks. Disk involvement has been reported in 46–72% of cases and occurs *relatively late compared to pyogenic spondylitis.* "Floating disk sign"—Occasionally the disk space is preserved despite extensive bone destruction. In children the hydrated *disks do not seem to form a good barrier* to infection and are involved in most patients.

Involvement of posterior elements—detected well by MR, *more common in tuberculosis than pyogenic infections.* The contrast study—may show inhomogeneous enhancement in the region of marrow infiltration.

Paraspinal soft tissue masses/abscesses—about 71% of cases on MRI, on T1W loss of the uniform psoas muscle signal intensity with enlargement of the affected muscle, on T2W—hyperintensities. Post contrast—thick rim enhancement around intra-osseous and paraspinal soft tissue abscesses; more uniform enhancement is seen with granulation tissue or phlegmon. Epidural extension—about 61% cases on MRI. It displaces the thecal sac. The spinal cord is distorted. Post-contrast fat sat T1W sequences—best to demonstrate meningeal and epidural inflammatory soft tissues, with improved definition of cord and nerve root compromise. Heavily T2W

FSE sequences can also be used to provide a *myelographic effect* showing thecal sac compression.

The MR imaging features, with high sensitivity and specificity for diagnosis of spinal tuberculosis are—(1) disruption of the endplate (100% and 81.4% respectively), (2) paravertebral soft tissue (96.8%, 85.3%), (3) high signal intensity of the intervertebral disk on the T2-W image (80.6%, 82.4%). The overall sensitivity and specificity for diagnosis are 100% and 88.2%, respectively.

Cord Changes

Conventional radiographs provide no information. Even CT cannot adequately assess the cord status. MR imaging provides invaluable information about the status of the cord. Cord involvement often results in neurological deficit or paraplegia. The spinal cord has physiologic reserve to with stand pressure, particularly when pressure develops slowly *40–50% reduction in cord diameter is often compatible with good cord function!!!*

Spinal Cord Changes

1. *Edema of the cord*—hyperintense signal on T2-weighted images but no signal alteration on T1
2. *Myelomalacia*—T1 hypointense signal (higher than that of CSF) may be associated with thinning of the cord
3. Atrophy of the cord
4. *Syringomyelia*—signal characteristics of CSF

PS

1. Edema is compatible with good neurological recovery following treatment.
2. Thinning of the cord with syrinx or myelomalacia leads to poor cord function.
3. Rarely, a small tuberculoma of the cord may be responsible for neurological deficits presenting as “spinal tumor syndrome.”

Importance of MRI in Management

1. MR shows a relatively preserved cord with evidence of myelitis or edema and a predominantly fluid collection in the extradural space, respond well to conservative treatment.
2. Early surgical decompression is indicated when MRI shows that the extradural compression is due to granulation tissue or caseous tissue, with little fluid component compressing the spinal cord.

Atypical Spinal Tuberculosis

Definition

Compressive myelopathy with no visible or palpable spinal deformity and without the radiological appearance of a typical vertebral lesion. It is relatively uncommon and difficult to diagnose and treat in the early stages with more chances of neurological complications.

Atypical lesions may present as an intra-spinal tubercular granuloma with involvement of the neural arch and compressive myelopathy in single vertebral disease with a sclerotic vertebra.

Tubercular granuloma should be considered in the differential diagnosis of spinal tumor syndrome in zones endemic for tuberculosis.

Extraosseous extradural granuloma: It is a rare variant. Hematogenous route is more common in men than women at the dorsal epidural space and in the thoracic segment. It is a clinically compressive radiculomyelopathy with the pathology being a granulomatous membrane which is found ensheathing and compressing the spinal cord or cauda equina. It may be easily diagnosed by MR imaging as isointense to cord on T1W images and have mixed signal intensity on T2W images. Enhancement after gadolinium will be uniform if the inflammatory process is phlegmonous in nature or peripherally enhancing if abscess formation or caseation has occurred.

Posttreatment Follow-up

Conventional radiographs show the following:

The healing is appreciated late on routine radiographs which lags behind by about 3 months. The bony changes may even progress till 14 months after starting treatment and should not necessarily be considered an adverse feature.

The soft tissue paravertebral masses may also progress while on treatment reaching a maximum size within 1.5 months, although they may take up to 15 months to resolve. Radiographic signs of healing seen are as follows:

In cases of a static lesion, regression of a lesion is seen as well-defined outlines of the lesion without any evidence of sclerosis. Fusion of adjacent vertebral bodies forming a large block of osseous mass and fusion of contiguous vertebrae have been regarded as the surest sign of healing of spinal tuberculosis. In the absence of reliable serological and immunological markers of healing, the “healed status” is achieved if there is clinical and radiological evidence of healing with no recurrence after 2 years.

Computed tomography: Features of resolution seen as an increase in vertebral bone density with reduction in the size of paraspinal soft tissue masses. The inflammatory reaction in the bone marrow, however, is not well depicted.

Magnetic Resonance Imaging:

Signs of healing: The earliest sign is reduction in the amount of inflammatory soft tissue. However, increasing soft tissue mass, bony destruction, or an alteration

in signal intensity do not indicate failed treatment. A high-signal intensity rim on the T1W sequences at the edge of the osseous lesion represents healing with reduction/loss of contrast enhancement. However, persistent or increasing enhancement is not necessarily indicative of either deterioration or treatment failure.

Reactivation of old tubercular spondylitis: The change of signal from low signal in healed tuberculosis to high signal on T2W indicates that reactivation may present with an isolated psoas abscess without evidence of bony lesions.

Differential Diagnosis

1. Pyogenic
2. Fungal
3. Degenerative disk disease
4. Brucellosis
5. Neoplasms

Degenerative spondylosis: Clinical findings (afebrile) with disk space usually not markedly narrowed. On MR, disk desiccation is manifested as low signal intensity on T2W images. After IV contrast *infected disks enhance strongly* whereas degenerated disks only occasionally enhance to a small degree.

Tuberculous v/s pyogenic spondylitis: Chronicity and slow progression with lack of sclerotic and reactive changes. On MR imaging there is a relative preservation of disk with involvement of multiple contiguous and more frequent involvement of posterior elements, a well-defined para-spinal lesion, disproportionally large para-spinal masses, especially with calcification or a thick rim of enhancement, subligamentous spread to three or more vertebral levels, and presence of skip lesions favor a tubercular etiology. However, differentiation from pyogenic infection can at times be difficult.

Brucellosis: Like tuberculosis the course is indolent and characteristic features of brucellar spondylitis include gas within the disk, only minimal associated paraspinal soft tissue mass, absence of gibbus deformity, or predilection for the lower lumbar spine. On MR images, vertebral body morphology and cortical margins are intact despite evidence of osteomyelitis. When a solitary vertebra is involved, it indicates a metastatic disease in adults or eosinophilic granuloma. In children the spinal lesions such as lymphoma and neoplasms such as multiple myeloma and chordoma that involve contiguous vertebrae and disks can add to the diagnostic difficulty.

Spine, joints—Tubercular arthritis, long and flat bones—tubercular osteomyelitis, short bones—tubercular dactylitis and tendon sheath and bursae.

Etiopathogensis

It begins in the synovium or in the metaphyseal spongiosa with contiguous spread or hematogenous spread. The metaphyseal lesion may involve the joint through subperiosteal space, through capsule, or through destruction of the epiphyseal plate.

Sequestration and peri-osteitis are NOT very common (c/w pyogenic infections). Ischemic necrosis and end arteritis may result in a very small sequestrum which is usually not visible radiologically (unless calcified). Granulation tissue spreads onto the free surface of cartilage eroding it in patches, later causing loosening and separation of the cartilaginous tissue as it proceeds causing necrosis of cartilage with erosion of exposed bone. Marginal erosions are common in TB of weight-bearing joints (hip, knee, ankle). Wedge-shaped necrotic foci may become evident on either side of joint leading to the appearance of "kissing sequestra." Necrosed cartilage and fibrinous material form "rice bodies" in synovial joints, tendon sheaths, and bursae. Abscesses that form may track along the fascia planes and form sinuses. Plaques of irregular bone, if present in walls of chronic abscess or sinus, suggest long-standing TB infection.

BCG osteitis: It can occur following BCG vaccination which follows a benign course. It resembles chronic osteomyelitis radiologically but responds to ATT.

Synovial sheath infections: They are common with non-typical mycobacteria (other than *M. tuberculosis* and *M. bovis*), with a history of trauma (puncture wounds), surgery, immunocompromised status, and exposure to contaminated marine life.

Infection of prosthetic joints: It is a late complication and usually 6–12 months after surgery. It is attributed to extensive surgery, use of implants, and favorable conditions for mycobacteria like DM, steroids, and immunocompromised status.

It can be divided into four radio-pathological stages:

1. Inflammatory edema and exudates (pre-destructive stage)
2. Necrosis and cavitation
3. Destruction and deformation
4. Healing and repair

Infection in bones is said to develop 2–3 years after primary focus (lung, lymph nodes), so diagnosis is usually delayed (c/w pyogenic infections which are seen 2–3 weeks after clinical presentation).

Clinical Features:

Insidious onset (c/w pyogenic infections) with low-grade fever, weight loss, and night sweat. There is movement restriction, muscle wasting, regional lymph node involvement, and neurologic symptoms. Weight-bearing joints like hip, knee, and ankle are commonly involved, though any part of the skeleton can get involved.

Imaging Modalities and Radiographic Features

Imaging Modalities

1. Radiography
2. USG
3. CT
4. MRI
5. Nuclear imaging

Radiography

AP and lateral views of the involved region and radiograph of the chest.

Radiological stages

1. Stage of synovitis
2. Stage of arthritis
3. Stage of advanced arthritis

1. *Stage of synovitis*: Soft tissue swelling and joint widening due to effusion and synovial hypertrophy. The first radiological sign may be juxta-articular osteoporosis. If there is secondary superadded infection, subperiosteal reaction may result. As a result of localized hyperemia growth plate may show overgrowth, especially in childhood.
2. *Stage of arthritis*: Articular margin and bony cortices become hazy (blurring and fuzzy) giving rise to "washed out appearance." Narrowing of joint space (involvement of articular cartilage) "Phemister Triad" consisting of (a) juxta-articular osteopenia, (b) peripherally located osseous lesions, and (c) gradual narrowing of joint space which are considered pathognomonic of tubercular osteoarthritis. Early loss of articular joint space is more typically seen in rheumatoid arthritis and thus helps in differentiating from tuberculosis.
3. *Stage of advanced arthritis*:
   (a) Collapse
   (b) Subluxation or dislocation
   (c) Migration of bone
   (d) Deformity of joint
4. Healing as follows:
   (a) Re-mineralization.
   (b) Cortical and articular margins become distinct.
   (c) Fibrous ankylosis may occur during healing phase (pyogenic arthritis—Bony ankylosis).
   (d) In contrast to pyogenic arthritis, the development of bone ankylosis is uncommon in tubercular arthritis.

USG

Helpful in the evaluation of large joints; demonstrates joint effusion, synovitis, and capsular thickening; synovial thickening—hypoechoic intra-articular soft tissue. Synovial sheath along tendons is thick and heterogeneous with minimal fluid suggestive of chronic tenosynovitis:

(a) Soft tissue abscess like psoas abscess
(b) Cortical disruption and irregularity of articular margins
(c) Smaller joints like wrist, hand, foot, and ankle and
(d) Guided joint fluid aspiration or synovial biopsy is possible.

CT

Lytic areas and marginal erosions seen much before plain radiographs.

Swelling in soft tissues, granulation, exudations, abscess, and early calcification can also be demonstrated much earlier; joint space better evaluated by CT—Limitation—The plain radiographs and CT scan are *not* likely to detect the stage of inflammatory edema and exudates. Computed tomography-guided aspirations and needle biopsy for difficult areas like sacroiliac joints.

MRI

Synovial hypertrophy is commonly seen

1. Hypointense areas on T2-weighted images suggesting hemosiderin deposition
2. Rim of synovial lesions on pregadolinium T1-weighted images and
3. Fluid loculations with enhancing synovial rims and erosions on postgadolinium images

PS: All the above features may be helpful in characterizing the lesion as tubercular when the radiographs are normal.

MRI highlights:

STIR shows fat saturation technique, results in markedly decreased signal intensity from fat and strikingly increased signal from fluid and edema. It is extremely sensitive tool for detecting tissue and marrow pathology. T2-weighted sequence shows hyperintense joint effusion. Synovial proliferation due to tubercular arthritis maybe hypointense on T2-weighted images and thickened synovium enhances vividly after gadolinium. Active pannus proliferating into the subarticular bone enhances on the postcontrast scans while chronic fibrosis does not enhance.

Caseating granulomas with solid centers give a characteristic hypointense signal to the synovium on T2-weighted images showing chondral lesions and subchondral bone erosions may be visible at a stage *when the joint space is still well preserved.*

Penumbra sign—A thin *intermediate signal intensity* rim along the periphery of a bone or soft tissue abscess on unenhanced T1-weighted images, due to layer of *granulation tissue* along its wall. It is *useful in identifying soft tissue abscesses.*

MRI helpful in detecting

1. Bone marrow inflammation.
2. Intra-osseous abscess.
3. Sequestrum.
4. Cortical destruction.
5. Cloaca and sinus tract formation.
6. Tenosynovitis may be seen.
7. Bursitis may be seen as distended bursa or multiple small abscesses.
8. Repeat imaging can be helpful in follow-up and if there is deterioration, then a representative biopsy is mandatory from the area.

Nuclear Imaging

The pre-destructive stage can be visualized by MRI and also probably by bone scans. Isotope bone scan or MRI may reveal subclinical active lesion in 40% of

patients in addition to the presenting lesion. Out of technetium-99 m, gallium-67, and indium-111 isotopes used in skeletal scintigraphy, technetium-99 m is the most sensitive, though not specific.

Positive scan helps in localizing and for follow-up. 18Fluorine fluorodeoxyglucose-positron emission tomography (18F-FDG-PET) has also been found useful in localizing tubercular disease and in differentiating soft tissue infection from osseous infection.

Tuberculosis of joints—arthritis: includes the joints (hip, knee, ankle and foot, shoulder, elbow, wrist and carpus and the sacro-iliac joints).

Tuberculosis of the Hip

Involvement in about 15% cases of osteo-articular TB. Lesions can arise in acetabulum, synovium, femoral epiphysis, or metaphysis or spread to the hip from foci in the greater trochanter or ischium. If upper end of femur is involved (being entirely intracapsular), the joint is involved early in disease. Erosion or lytic lesions may also occur in the greater trochanter or the overlying bursa, without involvement of the hip joint for a long period of time (Fig. 7.1).

Stage of synovitis: Radiography—plain radiograph usually normal shows: displacement of fat planes (effusion), soft tissue swelling, and deossification. Radiologically significant osteoporosis appears 12–18 weeks after onset of symptoms (c/w pyogenic arthritis).

Clinically seen as an irritable hip, positive obturator sign occur due to flexion deformity (reduced obturator foramina size).

Ultrasound, CT, and MRI: They are more sensitive in this stage to detect increased joint space and accumulation of fluid.

Investigations may be repeated, at 3–6 weeks interval, to help in establishing the diagnosis.

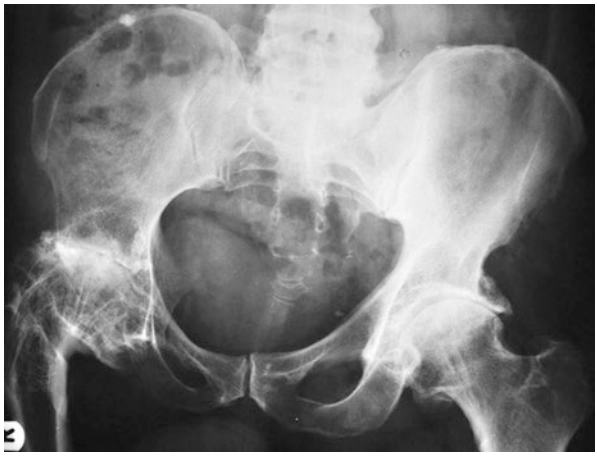

Fig. 7.1 TB of right hip along with OA of left hip (Courtesy: re-used with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

Differential diagnosis:

1. Traumatic or nonspecific transient synovitis
2. Perthes
3. Slipped capital femoral epiphysis
4. Low grade pyogenic infections

Stage of arthritis: Peri-articular erosions with reduction of joint space (destruction of articular cartilage). Lesions can usually be picked up on CT before they are apparent on plain radiographs.

Stage of advanced arthritis: Destruction of articular cartilage, acetabulum, femoral head, capsule, and ligaments. Capsule may get thickened and contracted. The upper end of femur may displace upwards and dorsally breaking the Shenton's line. Lower part of acetabulum empty (Wandering acetabulum). If femoral head, neck are grossly destroyed and collapsed in an enlarged acetabulum; this appearance is called "mortar and pestle" appearance.

Role of Surgery

It is indicated mainly (1) to establish diagnosis, (2) therapeutic and (3) failure of conservative treatment. Surgery is mainly in four forms, namely: (a) synovectomy, (b) debridement of joint, (c) arthrodesis, and (d) arthroplasty.

Indications for arthrodesis are as follows: Failure of conservative management or relapse, destructive lesion in the head or painful fibrous ankylosis/ankylosis with severe deformity. Arthrodesis is mainly of two types (Table 7.5): extra-articular or intra-articular or combined with intra-articular.

Excision arthroplasty: where the head, neck, proximal parts of femur, and acetabular rim are excised. This results in a painless, mobile hip, capable of squatting but with instability and shortening of the limb.

Replacement arthroplasty: Early cenmentless total hip arthroplasty in active TB, provided the patient is administered ATT and thorough debridement is done. Statistics according to recent authors is: cemented [1], uncemented [2].

Table 7.5 Table showing the differences between the types of arthrodesis of the hip joint

Intra-articular	Extra-articular
Active disease	Iliofemoral (Hibbs)
Painful fibrous ankylosis	Ischiofemoral (Brittains)
Lower rate of fusion	Abbot Lucas procedure: First stage is arthrodesis in wide abduction and second stage is subtrochanteric osteotomy to reposition the limb. (Best position is 30 degrees of flexion and 5/10 degrees of IR/ER.)

Tuberculous Arthritis of the Knee

Largest intra-articular space. Involved in about 10% of osteo-articular tuberculosis. Any age group; symptoms—pain, swelling, palpable synovial thickening, and restriction of mobility. Tenderness in the medial or lateral joint line and patello-femoral segment of the joint. The initial focus may be in synovium or subchondral bone of distal femora, proximal tibia, or patella.

Stage of synovitis: Osteoporosis, soft tissue swelling, joint/bursa effusion. Distension of supra-patellar bursa on lateral radiograph of knee. Infection in childhood can lead to accelerated growth and maturation resulting in big bulbous squared epiphysis.

Widening of the inter-condylar notch (synovitis).

Stage of arthritis: (1) Loss of definition of articular surfaces, (2) marginal erosion, (3) decreased joint space, and (4) osteoporosis.

Stage of advanced arthritis: (1) Osteolytic cavities with or without sequestra formation, (2) marked reduction of joint space, (3) destruction and deformity of joints, and (4) in advanced cases, there is triple deformity of the knee, that is, lateral, posterior, and superior displacement of tibia on femur.

Differential diagnosis:

1. Juvenile rheumatoid arthritis
2. Villonodular synovitis
3. Osteochondritis dissecans
4. Hemophilia

Biopsy of the synovial membrane and aspiration of the joint fluid followed by smear and culture can confirm the diagnosis.

Operative Treatment

1. In the synovial stage: Arthrotomy and synovectomy should be carried out.
2. In early arthritis, synovectomy, removal of loose/rice bodies, debris, pannus, loose articular cartilage, and careful curettage of osseous juxta-articular foci. Postoperatively triple drug therapy, combined with traction, intermittent active and assisted exercises, and suitable brace ambulation should be continued.
3. In adults with advanced arthritis or in cases which resulted in painful fibrous ankylosis during the process of healing, the knee joint may be treated by arthrodesis (Fig. 7.2).

   This option provides a painless stable knee, prevents recrudescence, corrects deformity and the patients can do long hours of standing and walking.
4. Arthroplasty: [3, 4] performed arthroplasty inadvertently in the preoperatively unsuspected cases of tuberculosis of the knee. Kim et al. [5] reported good results after total knee arthroplasty in selected cases of old healed tuberculosis of knee.

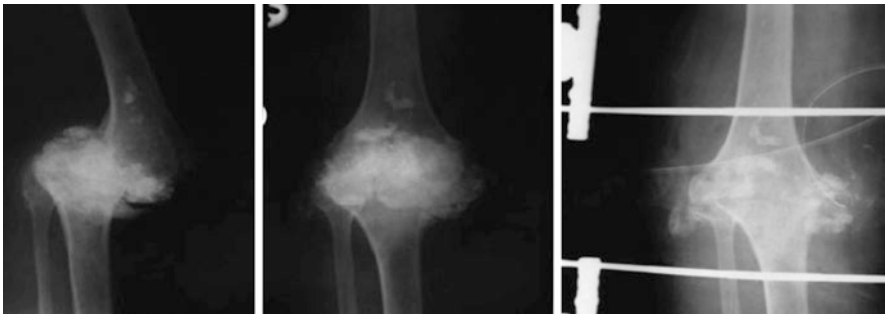

Fig. 7.2 Tuberculosis of the knee—treated by arthrodesis with Charnley's compression clamps (Courtesy: re-used with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

Gale and Hardinge [6] reported a short-term result of total knee arthroplasty in the presence of active disease. Indication for arthroplasty for a healed disease may be more justified for the knee than for any other joint. At present replacement arthroplasty of knee is being offered to selected patients. Most of the authors suggest this operation at least 5–10 years after the last evidence of activity of infection [2, 5, 6]. Mandatory coverage by modern antitubercular drugs for about 5 months after replacement surgery is advised.

Tuberculosis of the Ankle and Foot

Ankle: Swelling—in front of the joint, around the malleoli and tendoachilles insertion, marked osteoporosis with or without erosion with unsharpness of articular surfaces along with reduction of joint space. In long-standing cases, gross destruction of bones and sinus formation can result (Fig. 7.3). Pathological anterior dislocation can occur. Periosteal reaction is rare.

Foot: Common involvement of calcaneum, subtalar, and midtarsal joints (Fig. 7.4).

Anterior two-thirds of calcaneum being commonly affected. Radiograph can reveal the presence of osteolytic lesion with or without coke-like sequestrum. It rapidly spreads across the intercommunicating synovial channel*s*, so multiple bones are commonly involved. DD—Osteochondritis dissecans of talus can simulate a tuberculous lesion of the ankle. The foot bones can have isolated tubercular lesions as in the os calcis or as diaphyseal foci in metatarsal bones (tubercular dactylitis). A subchondral lesion in the calcaneum leading to talo-calcaneal arthritis. Talo-navicular and naviculo-cuneiform lesions and calcaneocuboid joint involvement can also occur, particularly in diabetes mellitus.

The tarso-metatarsal and metatarso-phalangeal joint can be involved. Lesions may look very similar to Madurella infection.

Differential diagnosis—neuropathic change in the foot, secondary to diabetes or leprosy.

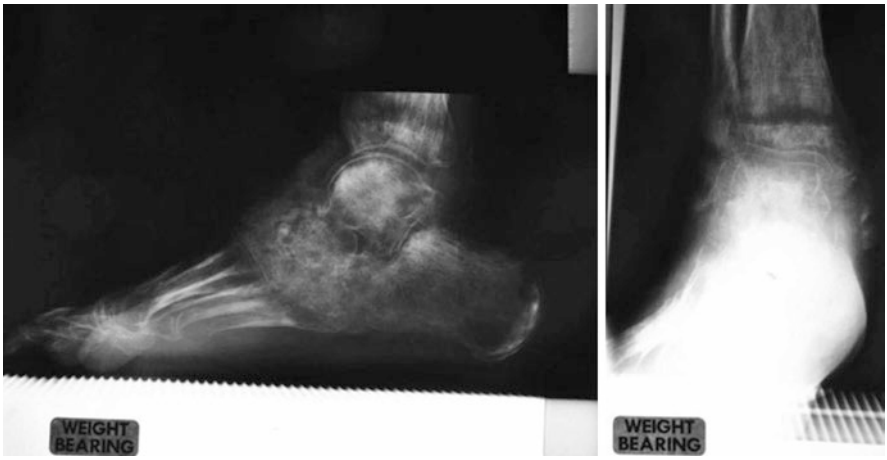

Fig. 7.3 TB of the ankle (Courtesy: re-used with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

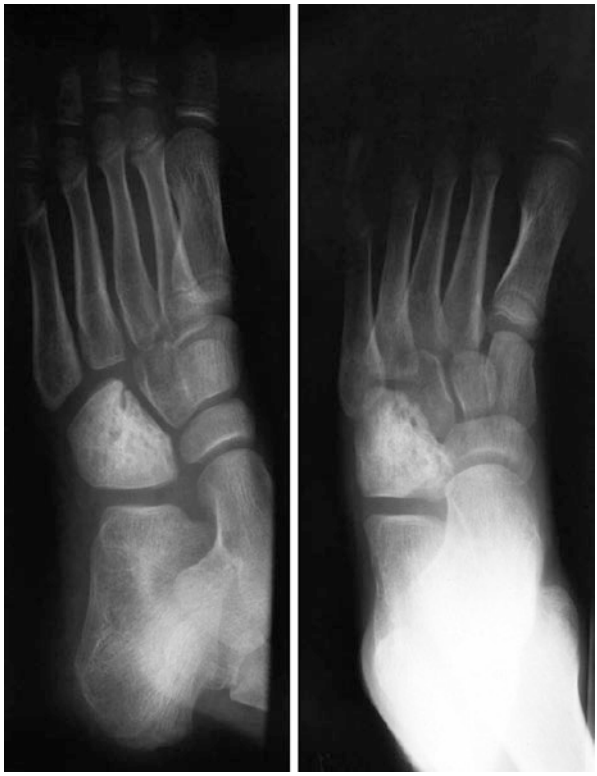

Fig. 7.4 TB of the cuboid (Courtesy: re-used with the kind permission of Magdi E. Greiss, Whitehaven, Cumbria, UK)

Tuberculosis of the Shoulder

It is very rare and more frequent in adults. The incidence of concomitant pulmonary tuberculosis is high.

The classical sites could be—head of humerus, glenoid, spine of the scapula, acromio-clavicular joint, or the coracoid process and rarely synovial lesion.

Iatrogenic due to steroid injection given for a stiff shoulder with the mistaken diagnosis of frozen shoulder, particularly in diabetics. Initial tubercular destruction is typically widespread (because of the small surface contact area of articular cartilage).

The symptoms—severe painful movement restriction particularly abduction and external rotation along with gross wasting of shoulder muscles.

Radiologically, there is osteoporosis with erosion of articular margins (fuzzy) and an osteolytic lesion involving head of humerus, glenoid, or both.

The lesion may mimic giant cell tumor. The joint space involvement and capsular contracture are seen early in the disease along with sinus formation, inferior subluxation of the humeral head, and fibrous ankylosis.

Caries sicca—It is an atrophic type of tuberculosis of the shoulder having a benign course without pus formation along with small pitted erosions on the humeral head.

The classical dry type is more common in adults, the fulminating variety with cold abscess or sinus formation is more common in children.

Differential diagnosis:

(1) Peri-arthritis of the shoulder, (2) rheumatoid arthritis, (3) post-traumatic shoulder stiffness.

Aspiration of the shoulder and FNAC might be necessary to establish the diagnosis. The patients usually respond well to antitubercular drugs.

Tuberculosis of the Elbow Joint

It is seen in 2–5% cases. The most frequent sites of involvement are the medial and lateral condyles of the humerus, articular surface of olecranon usually intra-articular but occasionally extra-articular in the head of radius. Rarely synovial in origin. Radiographic features: (1) *Articular type*—involvement of humerus and ulna, osteoporosis, blurring of articular cortex, and early diminution of joint space. (2) *Extra-articular type*—ulna is involved most commonly with destructive lesions seen in olecranon or coronoid process. Periostitis may also be seen. In infants and children, sequestra may be present. Peri-osteitis is a common feature and most commonly affects the ulna; pathological dislocation of elbow is very rare.

Differential diagnosis—(1) osteochondritis dissecans of the humeral condyle and (2) osteoid osteoma of the lateral condyle of the humerus which being intra-articular in location can be mistaken for tuberculosis of the elbow joint.

The diagnosis can be confirmed by aspiration or biopsy of synovium from the lateral side.

Tuberculosis of the Wrist and Carpus

It is a rare site and usually affecting adults. The anatomical sites of the lesions are: radius (distal end) and proximal row of carpal bones—scaphoid, lunate, capitate. There also may be concomitant involvement of the sheaths of volar or dorsal tendons might also occur.

Radiographic features—include intense osteoporosis, soft tissue swelling, erosions of articular margins and cartilage destructions, periosteal reaction and *early appearance of ossification centers*. All carpal bones tend to get involved in adults. More localized lesions in children (due to thicker articular cartilage in children).

Intense demineralization is present in carpus, distal radius, and ulna, *metacarpals being usually spared*. This serves as a *differentiating feature from rheumatoid arthritis.* Biopsy of the wrist can be easily done from the dorsal route, when in doubt.

Tuberculosis of the Sacroiliac Joints

It is more often in young adults than children. The involvement is usually unilateral and usually associated with tuberculosis of spine. Clinically there is tenderness over the sacroiliac joint and compression and distraction tests are painful.

Radiographic features: There is irregularity and fuzziness of articular surfaces starting at the inferior surface. Sub-articular erosions may be present causing widening of joint space.

Both the sclerosis and erosions predominate on the iliac side while punched out lesions may be seen in ilium or sacrum.

Tuberculosis at this uncommon site is frequently missed. The cold abscess can be either intra-pelvic or under the gluteus maximus muscle. Magnetic resonance imaging is the ideal means of evaluating SI joints—coronal imaging of the SI joints, parallel to the plane of the sacrum, allows direct comparison of one SI joint to the other; diagnosis is established by aspiration or FNAC. Antituberculosis therapy and protective bracing are the treatment of choice.

BCG Osteomyelitis and Arthritis

It is the vaccine of an attenuated bovine tubercular bacillus when generalized BCG infection and bone and joint infection can occur. Vaccination is not usually associated with immunologic disorder and has a favorable prognosis. BCG osteomyelitis affects children between 6 months and 6 years of age. It usually affects epiphysis and metaphysis of tubular bone, especially around the knee, ribs, the sternum, and the small bones of hand and feet. It is more common on same side of the body as the vaccine was injected. Solitary lesions predominate as well-defined lytic foci.

Diagnosis of osteitis after BCG vaccination is established according to criteria proposed by Foucard and Hjelmsted:

1. BCG vaccination in the neonatal period.
2. A period of less than 4 years between vaccination and symptom onset.
3. No contact between the child and any adults with TB.
4. A consistent clinical profile.
5. Histopathology suggestive of TB.

Long and Flat Bones—Tubercular Osteomyelitis

Long bones and flat bones: ribs, scapula, sternum, skull, pelvis, SC and AC joint. TB osteomyelitis about 3% of MSK tuberculosis. In 7% of them, skeletal site of lesions are multiple; the most frequent sites are the manubrium sterni, sternum and isolated spinous processes, spine of the scapula, and ischium and fibula.

The lesions tend to be simultaneous in onset and progression and generally affect persons with low immune resistance. There are symmetric well round, oval cystic lesions, with little or no periosteal reaction initially may be present. In untreated cases, laminated periosteal reaction may be seen. Sequestra formation is uncommon in adults, and large sequestra may be seen in children (intraosseous vasculature is more prone to thrombosis).

Joint involvement is rare as the lesions are diaphyseal or metaphyseal.

Closed cystic tuberculosis:

Disseminated lesions as bone cysts with NO sclerosis, abscess, or sinus formation.

Differential diagnosis:

1. Polyostotic fibrous dysplasia
2. Eosinophilic granuloma of the bone
3. Enchondromatosis

A firm diagnosis can only be established by biopsy of the lesion.

Rx—Antituberculosis regimens with curettage.

Pathology: Hematogenous spread from a primary focus. Granulomatous lesion develops within the bone at the site of deposition of the mycobacterium, usually metaphysis which is the site of infection. Earliest lesion appears as eccentric osteolytic lesion in the shaft near the epiphysis or metaphysic. Epiphyseal growth plate offers little resistance resulting in transphyseal spread of infections.

Two types of lesions have been described.

Pathologically:

1. Caseous exudative type—Destruction of bony trabeculae, softening and caseation necrosis followed by formation of tuberculous pus or cold abscess and.
2. Granular type caries sicca where predominantly granulation tissue is formed with minimal caseation.

Tuberculosis of Long Bones

Can be *Radiologically* classified as:

1. Metaphyseal type: oval or round focus in metaphysic; ultimately crosses into the epiphysis and joint; femur and tibia most commonly affected.
2. Cystic (diaphyseal type): affects children and young adults; diaphysis involved, well-defined round or oval lytic areas; expansile and large sequestra may be seen; tibia is the most common bone involved. Joint involvement is infrequent.

Solitary involvement is predominant. Disseminated skeletal TB is rare. Multifocal tuberculous osteomyelitis is also known as "osteitis cystica tuberculosa multiplex."

Multiple sites of involvement are seen in children, while in adults, involvement is more often confined to a single bone. Little or no surrounding reactive bone and local osteopenia are salient features.

The radiographic appearance:

In young patients: favor metaphyseal region, usually osteolytic and well defined and without sclerosis. It may show variable size. In adults: the lesions are smaller and located in the long axis of bone and some may show well-defined sclerotic margins.

MRI may show an early focus of altered marrow signal with irregular margins and cortical invasion with ill-defined soft tissue. Eccentric lesion with cortical breach. Small communicating abscesses are better appreciated on postcontrast T1W images as *enhancing rings* of juxta-cortical inflammatory tissue and are a strong predictor of tuberculosis.

Tuberculosis of Flat Bones

Ribs seen in about 2% cases of bone tuberculosis. Commonly adults being generally affected. One-third of patients will have pulmonary tuberculosis. Clinically—pain, tenderness, and fluctuant chest wall swelling. Radiology shows that the *posterior half of ribs* most commonly affected with bony rib expansion and punched out lesions and destruction which may be poorly marginated.

Scapula—Rare, acromion, spine, superior or inferior angle of scapula. Patient presents with pain and swelling. CT and MRI are helpful in early detection of lesions.

Sternum: Rare. An irregular destructive lesion with retrosternal and pre-sternal soft tissues with paucity of sclerosis or peri-osteitis.

Skull: Frontal bone most common site. Ill-defined lytic lesion may be the only radiological feature seen with overlying cold abscess (Potts' Puffy tumor). Button sequestrum sometimes seen. Facial bones and mandibular involvement is extremely rare.

Pelvis: Isolated tubercular lesion may occur in iliac bone, ischial tuberosity, and ischio-pubic ramus. Ischial tuberosity involvement was earlier recognized as "weaver's bottom" in which the overlying bursa was inflamed with secondary involvement of bone. Concomitant involvement of sacroiliac joints is common. Radiologically, lytic lesions without surrounding sclerosis or periosteal reaction may be seen.

Tuberculosis of Short Bones

Tubercular dactylitis: It is primarily a disease of childhood which affects short tubular bones distal to tarsus, and wrist bones of the hands are more frequently affected than bones of the feet. The proximal phalanx of the index and middle fingers and the metacarpals of the middle and ring fingers are being the most frequent locations. They frequently present as marked swelling on the dorsum of the hand and soft tissue abscess is normally a common feature. Monostotic involvement is common. It often follows a benign course without pyrexia and acute inflammatory signs, as opposed to acute osteomyelitis. Plain radiography is the modality of choice for evaluation and follow-up.

The radiographic features are: cystic expansion of the short tubular bones have led to the name of "spina ventosa" being given to tubercular dactylitis of the short bones of the hand. Spina—short bone and ventosa—expanded with air. Bone destruction and fusiform expansion of the bone: It is most marked in diaphysis of metacarpals and metatarsals in children. Periosteal reaction and sequestra are uncommon. Healing is gradual by sclerosis.

Differential diagnosis: (1) syphilitic dactylitis—bilateral and symmetric involvement, more periostitis, less soft tissue swelling and (2) chronic pyogenic osteomyelitis and mycotic lesions in the foot. Debridement and antitubercular regimen result in complete subsidence of the lesion.

Tuberculosis of the Tendon Sheaths and Bursae

The most common sites are flexor tendon sheaths of hand, subacromial bursa, olecranon bursa, and bursae under the medial head of gastrocnemius.

Clinically: In the volar aspect of the wrist, the classical presentation is a dumbbell-shaped swelling giving to cross fluctuation and crepitus. The spread to these sites is normally from the neighboring bone or joint but it could be due to hematogenous spread.

Primary investigation is ultrasonography. In chronic tenosynovitis, tendon and synovial thickening predominate, with relatively little synovial sheath effusion. In acute suppurative tenosynovitis, synovial sheath effusion is the predominant feature. Magnetic resonance imaging helps in delineating the precise extent of soft tissue involvement and any associated osseous or joint involvement.

Among bursal infections, the most commonly affected locations are

1. The trochanteric
2. Subacromial
3. Subgluteal
4. Radioulnar wrist bursa

Plain radiography shows local osteopenia (due to hyperemia) and calcifications within wall of the distended bursa. Antituberculosis regimes coupled with excision of the synovial sheath and bursae are the treatment of choice.

Atypical Mycobacterium Infection

It may be seen in immunocompromised patients, those with renal transplants or those receiving cortico-steroids. The infection can lead to osteomyelitis, septic arthritis, tenosynovitis, and bursitis.

Radiologically multiple lesions may be seen. The metaphysis and diaphysis of long bones are usually affected. Osteoporosis is NOT marked with abscesses and sinus tract seen.

Tubercular infection of prosthetic joint: It may develop due to reactivation of tubercular arthritis for which the operation had been performed. Arthrocentesis and specimens are required for establishing the diagnosis.

Interventions are mainly diagnostic: USG guided—fluid aspiration, pigtail placement (psoas abscess) or CT guided—aspiration/biopsies (SI joints).

Conclusion

Tuberculosis of bones and joints can have varied radiological appearances. Conventional radiographs are the usual initial imaging modality. Radiography along with biopsy/FNAC generally suffices for diagnosing infections of joints and bones (except spine).

USG has a limited role, mainly in detection of fluid/collections and in guided interventions. Spinal tuberculosis is the most severe among infective spondylitides.

MRI is the imaging modality of choice for spinal tuberculosis.

CT can be an alternative tool when MR is not available and for guided interventions.

Tuberculosis of Spine

Introduction: It was described first by Sir Percival Pott in 1779. The classic destruction of the disk space and the adjacent vertebral bodies, destruction of other spinal elements, and severe and progressive kyphosis subsequently became known as Pott's disease. Approximately 10% of patients with extrapulmonary tuberculosis have skeletal involvement. The spine is the most common skeletal site affected, followed by the hip and knee. Spinal tuberculosis accounts for almost 50% cases of skeletal tuberculosis, higher incidence in concomitant HIV infections.

Predisposing factors: Poverty, overcrowding, illiteracy, malnutrition, alcoholism, drug abuse, diabetes mellitus, immunosuppressive treatment, HIV infection, and FokI polymorphism in the vitamin D receptor gene [7].

Spinal involvement: Hematogenous spread of *M. tuberculosis* into the dense vasculature of cancellous bone of the vertebral bodies. Pulmonary lesion, genitourinary system infection as primary source. Spread: Subchondral arterial arcade derived from anterior and posterior spinal arteries, Batson's paravertebral venous plexus, anterior inferior portion of vertebral body. Spread to central part of disk. Common sites—paradiscal, anterior, and central. In younger patients, the disk is primarily involved because it is more vascularized. In old age, the disk is not primarily

Table 7.6 Table showing the factors leading to early onset paraplegia

Problem	Cause of involvement
Mechanical pressure	Mechanical pressure by tuberculous debris, sequestrum of bone or disk, abscess, subluxation and dislocations, concertina collapse, and internal gibbus.
Tuberculous granuloma	Tuberculoma in extradural, intradural, or intramedullary regions.
Tuberculous myelitis	Uncommon. May involve spinal cord parenchyma
Spinal artery thrombosis	Infective thrombosis of anterior spinal artery
Tuberculous arachnoiditis	Meningeal inflammation and fibrosis.

Table 7.7 Table showing the causes leading to late onset paraplegia

Problem	Cause of involvement
Transection of spinal cord by bony bridge	Transverse ridge of bone produced by severe kyphosis
Fibrosis of dura (pachymeningitis)	Formation of tough, fibrous membrane encircling the cord

involved because of its age-related avascularity. Segmental arteries bifurcate to supply two adjacent vertebrae. Multiple contiguous vertebrae involved if spread occurs below the anterior or posterior longitudinal vertebrae. Destruction of the intervertebral disk space and the adjacent vertebral bodies results in collapse of the spinal elements with anterior wedging.

Early onset paraplegia (Table 7.6).

Late onset paraplegia (Table 7.7).

Clinical Features

Local pain, local tenderness, stiffness and spasm of the muscles, cold abscess, gibbus. Slow and insidious progression, 4–11 months average duration. Constitutional symptoms. Pain may be aggravated by spinal motion, coughing, and weight-bearing due to advanced disk disruption and spinal instability, nerve root compression, or pathological fracture. Neurological deficits common with cervical and thoracic segment involvement. Can occur during any stage (Table 7.8).

Cold abscess: Dysphagia, respiratory distress, or hoarseness of voice due to retropharyngeal abscess. Extension into pleural cavity, mediastinum, and esophagus. Paravertebral abscess and psoas abscess.

"SADDER" spine: Sclerosis, abscess, disc space reduction, destruction, erosion of vertebrae and rarefaction.

Paravertebral soft tissue shadows calcifications presence due to lack of proteolytic enzymes. 50% and 75% of patients with osteoarticular tuberculosis and up to

Table 7.8 Table showing the causes seen in different types of lesions

Type of involvement	Mechanisms of involvement	Radiological appearances
Paradiskal	Spread of disease via the arteries	Adjacent margins of two consecutive vertebrae. The intervening disk space is reduced
Central	Spread of infection along Batson's plexus of veins	Involves central portion of a single vertebra; proximal and distal disk spaces intact
Anterior Marginal	Abscess extension beneath the anterior longitudinal ligament and the periosteum	Begins as destructive lesion in one of the anterior margins of the body of a vertebra, minimally involving the disk space but sparing the vertebrae on either side
Skipped lesions	Spread of infection along Batson's plexus of veins	Circumferentially involvement of two noncontiguous vertebral levels without destruction of the adjacent vertebral bodies and intervertebral disks
Posterior	Spread via the posterior external venous plexus of vertebral veins or direct spread	Involves posterior arch without involvement of vertebral body
Synovial	Hematogenous spread through subsynovial vessels	Involves synovial membrane of atlanto-axial and atlanto-occipital joints

67% of patients with spinal tuberculosis have an associated primary lung focus or have a reported history of pulmonary tuberculosis.

CT

CT demonstrates abnormalities earlier than plain radiography. Delineation of encroachment of the spinal canal by posterior extension of inflammatory tissue, bone, or disk material.

CT-guided biopsy.

MRI

Rapid determination of the mechanism for neurologic involvement. (1) Involvement of the vertebral bodies, (2) disk destruction, (3) cold abscess, (4) vertebral collapse, (5) spinal deformities.

Lab

Demonstration of acid-fast bacilli on pathological specimen. Histological evidence of a tubercle. Presence of epithelioid cells on the biopsy material.

Neuroimaging-guided needle biopsy from the affected site is the gold standard technique for early histopathological diagnosis. Open biopsy of the spine is usually

Table 7.9 Table showing the differential diagnosis of lesions seen

1. Pyogenic: Duration of illness in months—2–3 months; usual age of presentation—any age; anatomical location—lumbar; vertebral and other structures involved—vertebral bodies and intervening disc; common predisposing factors = systemic illness like diabetes mellitus; common clinical features = fever and marked pain, myelopathy; lab features = leukocytosis, raised ESR. C-reactive protein, neuroimaging (salient features) = present, raised ESR and C-reactive protein, destruction of vertebral bodies, marked enhancement of lesion, epidural abscess.
2. Tubercular: Duration of illness in months—3–6 months; usual age of presentation—children and young adults; anatomical location—lumbo-thoracic; vertebral and other structures involved—vertebral bodies and intervening disc, extensive soft tissue involvement (cold abscess); common predisposing factors = exposure to tuberculous infection; common clinical features = fever, malaise, weight loss, backache, myelopathy; lab features = leukocytosis, raised ESR. C-reactive protein, neuroimaging (salient features) = absent, raised, may be raised, destruction of vertebral bodies and disc spaces, rim enhancement of the soft tissue masses.
3. Brucellar: Duration of illness in months—2–6 months; usual age of presentation—middle aged; anatomical location—lumbar; vertebral and other structures involved—vertebral bodies and intervening disc, minimal soft tissue involvement, sacroiliitis; common predisposing factors = ingestion of unpasteurized milk; common clinical features = fever, weight loss, malaise, backache; lab features = leukocytosis, raised ESR. C-reactive protein, neuroimaging (salient features) = present, raised, may be raised, intact vertebral architecture despite diffuse vertebral osteomyelitis.
4. Metastatic: Duration of illness in months— < 2 months; usual age of presentation—middle aged and elderly; anatomical location—thoracic; vertebral and other structures involved—posterior wall of vertebral body (60%), pedicles (50%); common predisposing factors = presence of systemic malignancy; common clinical features = bone pain at night, back pain followed by radicular pain, myelopathy; lab features = leukocytosis, raised ESR. C-reactive protein, neuroimaging (salient features) = absent, not raised, not raised, low signal intensity on T1-weighted images. Hypersignal on T2-weighted images, and heterogenous enhancement.

performed when either closed techniques have proved insufficient or other procedures, such as decompression and possibly arthrodesis, are planned. Smear positivity for acid-fast bacilli may be seen in up to 52% of cases and culture positivity in about 83% of cases.

Histology: Epithelioid cell granulomas, granular necrotic background, lymphocytic infiltration, and scattered multinucleated and Langhans' giant cells (Table 7.9).

PCR: Detects as few as 10–50 bacilli; faster and more sensitive. Sensitivity ranging from 61% to 90%. Specificity of 80–90%. Multiplex real-time polymerase chain reaction: a practical approach for rapid diagnosis of tuberculous and brucellar vertebral osteomyelitis [8].

QuantiFERON-TB Gold assay: Cell-mediated inflammatory responses in vitro to tuberculosis infection. Measures interferon-gamma harvested in plasma from whole blood incubated with the *M. tuberculosis*-specific antigens. Sensitivity—84%. Specificity—95%.

Table 7.10 Table showing mechanism of action of different drugs

First line/anti-TB drugs	Mechanism of action	Administration	Dosage
Isoniazid	Bactericidal	GI absorption	300 mg/day
Rifampicin	Bacteriostatic or bactericidal	GI absorption	600 mg/day
Pyrazinamide	Bacteriocidal in an acid environment	GI absorption	15–30 mg/kg/day (<2 g/day)
Streptomycin	Bactericidal in alkaline environment	Parenterally	<1 g/day
Ethambutol	Bacteriostatic	GI absorption	40–55 kg: 800 mg/day 56–75 kg: 1.2 g/day >75 kg: 1.6 g/day

Treatment

1. WHO recommendation—9 months (2 HRZE + 7HR).
2. Middle path regime.
3. Supportive therapy—in the form of multivitamins, hematinics, and high protein diet.
4. X-rays and ESR at 3–6 months interval.
5. Gradual mobilization with the help of spinal braces.
6. Aspiration of abscesses.
7. Sinuses excision.
8. Decompression.
9. Surgical stabilization.

First line of AntiTB drugs (Table 7.10).

Indications

1. Without neurological complaints: Progressive destruction in spite of ATT with no response to conservative therapy or an increase in size of paravertebral abscess leading to mechanical or kyphosis deformity.
2. With neurological complaints: New or worsening neurological deficits with paraplegia of rapid onset or severe paraplegia, indicative of neural arch disease or spinal tumor disease.

Surgical approaches adopted at various levels by surgeons (Table 7.11).

The Hong-Kong Operation: First reported by Ito and later popularized by Hogsdon and Stock in 1956. Thorough excision (extirpation) of the tuberculous focus. Posteriorly-dura mater.

Cephalad and caudad till healthy, bleeding cancellous bone was exposed. Create surfaces suitable for docking of the strut graft (Rib/Tricortical iliac crest).

Table 7.11 Table showing the different approaches to treatment of tuberculosis of spine

1. Kirkaldy-Willis (1965): For the cervical spine—the anterior approach; From C7 to D1—through the bed of the 3rd rib; for the dorsal spine—anterolateral or transpleural; for the dorsolumbar spine—anterolateral approach; for the lumbar spine—retroperitoneal sympathetic or the ureter approach; for the L5S1 level—the transperitoneal paramedian approach in the Trendelenburg position [9].
2. Hodgson (1969): For C1C2—transoral or transthyroid; for cervical spine—through anterior posterior triangle; for C7D1—transpleural through the bed of the 3rd rib or split sternal for an extensive lesion; for the dorsal spine—transpleural decompression; for the dorsolumbar spine—through the bed of the 11th rib extrapleural and extraperitoneal or transpleural through the bed of the 9th rib; for lumbar spine—renal approach; for the L5S1—trans peritoneal in the Trendelenburg position, lower midline incision [10].
3. Kemp et al. (1973): For cervical spine—the anterior approach; for C7D1—anterior approach; for dorsal spine—trans-sternal for D3D4, anterior transpleural from D5 to D11; for dorsolumbar junction—through the bed of the 12th rib; for lumbar spine—retroperitoneal approach; for L5S1 junction—retroperitoneal through an oblique incision [11].
4. Smith and Robinson (1985): For C1C2—anterior approach; for cervical spine—anterior approach [12].
5. McAfee et al. (1987): For C1C2—retropharyngeal extramucosal approach [13].
6. Tuli et al. (1988): For C1C2—transoral for drainage; for cervical spine—anterior approach; for C7T1 junction—low anterior cervical approach; for dorsal—anterolateral or transpleural approach; for dorsolumbar junction—anterolateral approach; for lumbar spine—retroperitoneal approach; for L5S1 junction—retroperitoneal or retropsoas transverse vertebrotomy [14].

Role of posterior Stabilization in the management of tuberculosis of the dorsal and lumbar spine [15].

Aim: Study the progress of interbody union, the extent of correction of the kyphosis, and its maintenance with early mobilization. Incidence of graft and implant-related problems. The American Spinal Injury Association (ASIA) score was used to assess the neurological status.

Mean preoperative vertebral loss was highest (0.96) in the dorsal spine. The maximum correction of the kyphosis in the dorsolumbar spine was 17.8°. Loss of correction maximal in the lumbosacral spine at 13.7°. All patients had firm anterior fusion at a mean of 5 months. The incidence of infection was 3.9% and of graft-related problems 6.5%. Adjuvant posterior stabilization allows early mobilization and rehabilitation.

Graft-related problems were fewer and the progression and maintenance of correction of the kyphosis were better than with anterior surgery alone. There is no additional risk relating to the use of an implant either posteriorly or anteriorly even when large quantities of pus are present. There is an excellent article written on evaluation of the risk of instrumentation as a foreign body in spinal tuberculosis. Clinical and biologic study [16] show that few mycobacteria adhere to the stainless steel, whereas Staphylococcus heavily colonizes on stainless steel. Pyogenic organisms form a thick biofilm, whereas mycobacteria show a scanty biofilm. Hence, the use of implant in the presence of TB infection is theoretically safe.

Indications for stabilization: Pan vertebral disease, in which all three columns are diseased. Long-segment disease, in which after surgical decompression a bone graft >5 cm is inserted with instrumentation to prevent graft-related complications and consequent progression of kyphosis and neural complications. Surgical correction of a kyphosis is performed when both anterior decompression and posterior column shortening is required.

Kyphosis: Problems of progressive deformity and correction of residual severe deformity are current challenges. Patients with spinal tuberculosis treated conservatively have an average increase of 15° in deformity. 3–5% of the patients develop with a deformity greater than 60°.

Problems:

1. Cosmetic and psychological disturbance.
2. Costo-pelvic impingement.
3. Secondary cardiorespiratory problems.
4. Late onset paraplegia.
5. Correction difficult with high rate of complications.

It affects the anterior column in more than 90% of patients. Vertebral collapse can continue until the healthy vertebral bodies in the region of the kyphosis approximate anteriorly and consolidate.

Factors affecting the progression: Age—children have more severe deformity and more rapid progression to collapse and variable outcome after healing. Changes in healed phase.

Kyphosis in adults: Structural weakness of the vertebral column lead to pathological fracture in the diseased vertebrae and it can collapse into a kyphosis. Even after diagnosis and ambulant chemotherapy, kyphosis continues to grow despite being treated. Progression of kyphosis can be minimized by prescribing suitable braces. Patients treated nonoperatively have an average increase of 15° in deformity and 3–5% end up with a deformity greater than 60°. Kyphosis also continues to grow in a TB spine lesion which is surgically decompressed and bridged by bone graft with associated complications of slippage and breakage. Kyphosis, once healed, with osseous fusion does not grow alarmingly in adults in later life. When the lesion heals with fibrous or fibroosseous healing it may progresses further.

Kyphosis in children: The TB spine lesion in children causes more destruction as most of the vertebral bodies are cartilaginous. Even when a TB lesion heals by nonoperative treatment kyphosis continues to increase with growth. The anterior growth potential of the vertebral body is destroyed: The disease itself—Surgical excision of disease focus leads to an alteration in the growth resulting from the effect of biomechanical forces on the growth plate of both the fusion mass and the vertebral segments within the kyphosis. Patients treated with radical resection and anterior fusion showed the worst results with respect to progression of kyphosis particularly when the lesion was in thoracic spine and several segments were involved. Posterior elements contribute to growth and as far as possible anterior radical resection should be avoided in children [17].

Growth changes of solidly fused kyphotic bloc after surgery for tuberculosis—comparison of four procedures. The natural history of post-tubercular kyphosis in children [18].

Type I—increase in deformity until growth had ceased.

Ia—continuous while Ib—after a lag period of 3–5.

Type II—decrease in deformity with growth. IIa—occur immediately after the active phase, while IIb—after a lag period of 3–5 years.

Type III—progression showed minimal change during either the active or healed phases and was seen only in those with limited disease.

Influence of level of lesion:

Thoracic and thoracolumbar lesions have a greater deformity at presentation.

Natural thoracic kyphosis allows progression of deformity and lumbar lordosis protects against the deformity. In the thoracic spine, the more horizontally oriented articular facets allow earlier subluxation of the facet joints leading to a rapid collapse.

Buckling collapse: Complete destruction of more than two vertebral bodies. Dislocation of facet joints occurred at multiple levels leading to a kyphosis of more than 120°. Longitudinal overgrowth of vertebral segments is noted leading to stretching of the spinal cord at the apex of the kyphosis with possible secondary late onset paraplegia.

Risk factors for buckling collapse:

1. Age of less than 7 years at the time of the disease.
2. Thoracolumbar involvement.
3. Loss of more than two vertebral bodies.
4. Presence of radiographic spine-at-risk signs.

Risk factors for severe progression:

1. Age below 10 years.
2. Vertebral body loss of more than 1–1.5.
3. A pretreatment deformity angle of greater than 30°, especially in children.
4. Cervical thoracic and thoracolumbar junctional lesions.
5. The presence of “spine-at-risk” radiological signs.

Spine “At Risk” Signs

1. Separation of the facet joint: The facet joint separates at the apex of the curve with instability and loss of alignment.
2. Retropulsion: The posterior retropulsion of the diseased vertebral segment is identified by drawing two lines along the posterior surfaces of the first upper and lower normal vertebra.
3. Lateral translation: A line drawn through the middle of the pedicle of the lower vertebra does not touch the pedicle of the superior vertebra.

4. Toppling: In the initial stages of collapse, the line drawn along the anterior surface of the lower normal vertebra intersects the inferior surface of the upper normal vertebra. Toppling has occurred when the line intersects above the middle of the anterior surface of the upper vertebra.

Each sign is given a score of 1 with a maximum possible score of 4. A spinal instability score of more than 2 is associated with a significantly larger final deformity. A score of 3 or more accurately predicts an increase in the angles of deformity and kyphosis of more than 30 and a final deformity of more than 60.

Prediction of deformity progression: $Y = A + BX$; Y = final angle of gibbus deformity, X = measurement of initial loss of vertebral body. A and B are constants 5.5 and 30.5, respectively.

Patients reporting with classical disease: In adult patients, three or more vertebral body affection with initial vertebral body loss of 1–1.5 vertebral body height in a dorsal or dorsolumbar spine, need to be taken up for kyphosis correction. Children younger than 7 years of age with three or more vertebral body affection in dorsal or dorsolumbar spine. Two or more spine at risk segment will have a progressive kyphosis with growth.

Correction in active disease:

Correction of the kyphosis without opening the disease area and without anterior decompression will produce more prominent spinal cord indentation by retropulsed fragment (internal salient) and consequently neural deficit. Hence, internal salient should be removed by anterior debridement and corpectomy. Moderate to severe kyphosis which needs correction is a long standing problem. Abrupt correction of kyphosis with a column that is shortened anteriorly will produce lengthening of anterior column, which will stretch the spinal cord with consequent neural deficit. Thus, it is desirable to shorten the vertebral column posteriorly. Instrumented stabilization with anterior gap grafting and posterior fusion is needed as the spine becomes unstable following anterior corpectomy and posterior column fusion.

Methods:

1. Single stage transpedicular approach.
2. Single or two-staged anterior decompression with bone grafting followed by correction of kyphosis and posterior instrumentation.
3. Single stage kyphosis correction by extrapleural anterolateral approach.
4. Correction in healed lesion: Transpedicular decancellation osteotomy.

Late onset paraplegia/paraplegia with healed disease: Long-standing severe kyphosis with a history of being treated for spinal TB 10 or more years ago and now present with signs of upper motor neuron spinal cord injury or paraplegia. The cause may be a reactivation of old healed lesion at kyphus or intrinsic changes in spinal cord due to continued stretch on internal salient.

Anterior decompression and fusion: Internal salient is removed either by transthoracic transpleural anterior approach or by the extrapleural anterolateral approach.

Costotransversectomy/extrapleural anterolateral. Posterior vertebral column resection and intraoperative manual traction to correct severe post-tubercular rigid spinal deformities incurred during childhood: minimum 2-year follow-up.

Aim: evaluate the clinical and radiographic outcomes of posterior vertebral column resection (PVCR) and intraoperative manual traction to correct severe posttubercular spinal deformity incurred during childhood. Retrospective study of 11 patients (4M + 7F).

Clinical outcome assessment with Oswestry index and Visual Analog scale. Imaging measurements and fusion status were assessed using plain radiography and CT. Kyphosis improved from a preoperative average of 93.4° to a postoperative average of 18.7° for a correction of 80.1%. The Cobb angle in the coronal plane improved from an average of 48.1° to 10.3° postoperatively for a correction of 76.3%. At the last follow-up, two patients improved neurologically from ASIA grade C to grade D, and one patient improved from grade C to grade E. Perioperative complications occurred in 4 of the 11 cases. One patient had a dural tear. Three patients had temporary degradation of intraoperative neuromonitoring, and one experienced transient paralysis of the left lower extremity postoperatively.

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