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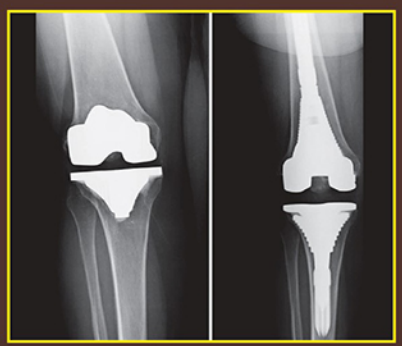
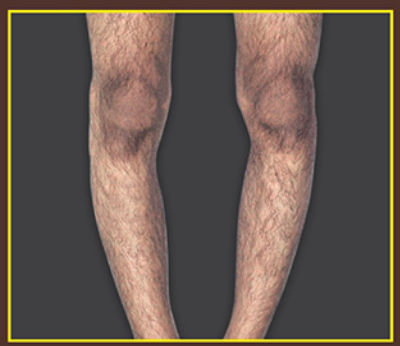
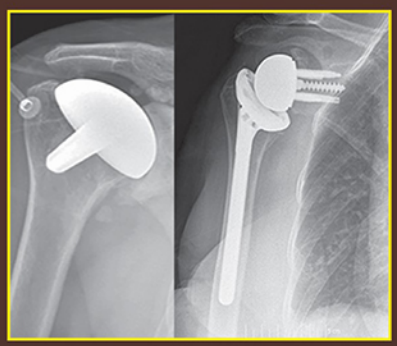

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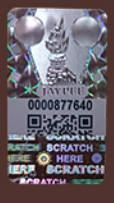

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Chapter

23 Tuberculosis of Bones and Joints

Topics

- General considerations
- TB of the spine
- Pott's paraplegia
- TB of the hip
- TB of the knee
- TB of other joints
- TB osteomyelitis

Competency

- **OR4.1:** Describe and discuss the clinical features, investigation and principles of management of tuberculosis affecting major joints (Hip, Knee) including cold abscess and caries spine.

GENERAL CONSIDERATIONS

Tuberculosis (TB) is still a common infection in developing countries. After lung and lymph nodes, bone and joint is the next common site of tuberculosis in the body. It constitutes about 1–4 per cent of the total number of cases of tuberculosis.

The spine is the *most common* site of bone and joint tuberculosis, constituting about 50 per cent of the total number of cases. Next in order of frequency are the hip, the knee and the elbow. Tubercular osteomyelitis more commonly affects the ends of the long bone, unlike pyogenic osteomyelitis which affects the metaphysis. This is also the reason for early involvement of the adjacent joint in tubercular osteomyelitis. Table 23.1 shows the common musculoskeletal structures affected by tuberculosis.

ETIOPATHOGENESIS

Common causative organism is *Mycobacterium tuberculosis.* Bone and joint tuberculosis is *always* secondary to some primary focus in the lungs, lymph nodes, etc. Mode of spread from the primary focus may be either haematogenous or by direct extension from a neighbouring focus.

Table 23.1: Musculo-skeletal tuberculosis.

Tissue	Disease	Remarks
Bone ▪ Long bone ▪ Short bone (phalanges) ▪ Spine	▪ TB osteomyelitis ▪ Tuberculous dactylitis ▪ TB spondylitis	▪ Tibia commonly affected ▪ Also called *spina ventosa* ▪ Also called Pott's disease
Joint ▪ Arthritis ▪ Synovium	▪ TB arthritis ▪ Synovial TB	▪ Hip joint commonly affected ▪ Knee-most common site
Others ▪ Tendon (synovium) ▪ Bursae	▪ TB tenosynovitis of flexor tendons at wrist ▪ TB bursitis of trochanteric bursa	▪ Compound palmar ganglion ▪ Trochanteric bursitis

Pathology: Tubercular infection of the bone and synovial tissue produces similar response as it produces in the lungs, i.e., chronic granulomatous inflammation with caseation necrosis. The response may be proliferative, exudative or both:

a. *Proliferative response:* This is the more common of the two responses. It is characterised by chronic granulomatous inflammation with a lot of fibrosis.
b. *Exudative response:* In some cases, particularly in immunodeficient individuals, elderly people and people suffering from leukaemia, etc., there is extensive caseation necrosis without much cellular reaction. This results in extensive pus formation. These are also termed *non-reactive* cases.

Natural history: Inflammation results in local trabecular necrosis and caseation. Demineralisation of the bone occurs because of intense local hyperaemia. In the absence of adequate body resistance or chemotherapy, the cortices of the bone get eroded, and the infected granulation tissue and pus find their way to the subperiosteal and soft tissue planes. Here they present as *cold abscesses,* and may burst out to form sinuses. The affected bone may undergo a pathological fracture.

A tubercular osteomyelitis in the vicinity of a joint may result in the involvement of the joint.

Joint involvement is usually in the form of a low-grade synovitis, with thickening of the synovial membrane. Unlike pyogenic arthritis where proteolytic enzymes cause severe early destruction of the articular cartilage, tubercular infection causes slow destruction. Once the synovium is inflamed, it starts destroying the cartilage from the periphery. This inflammatory synovium at the periphery of the cartilage is called *Pannus.* Eventually, the articular cartilage is completely destroyed. The joint gets distended with the pus. Joint capsule and ligaments become lax, and the joint may get subluxated. Pus and tubercular debris burst out of the joint capsule to form a cold abscess, and subsequently a chronic discharging sinus.

Healing: It occurs by *fibrosis,* which results in significant limitation or near complete loss of joint movement (fibrous ankylosis). If considerable destruction of the articular cartilage has occurred, the joint space is completely lost, and is traversed by bony trabeculae between the bones forming the joint (bony ankylosis) as shown in Figure 23.1. Fibrous ankylosis is a common outcome of healed tuberculosis of the joints, except in the spine where bony ankylosis follows more often.

Fig. 23.1: Types of ankylosis.

CLINICAL FEATURES

Clinical features depend upon the site affected. Patients of all ages and both sexes are affected frequently. The onset is gradual in most cases.

Usual presenting complaints are pain, swelling, deformity and inability to use that part. Sometimes, the presentation is atypical. The following general principles will help in making a diagnosis:

a. **High index of suspicion:** Tuberculosis should be included in the differential diagnosis of any slow onset disease of the musculoskeletal system, particularly in countries where tuberculosis is still prevalent. Because of its slow onset and progress, the symptoms and signs are often minimal and non-specific. A high index of suspicion and a close watch over such symptoms in susceptible individuals, is the key to early diagnosis.
b. **Fallacious history of trauma:** Very often the patient assigns all his symptoms to an episode of injury. One should not get carried away by such information, as the injury may be coincidental. A detailed inquiry in such cases will reveal a symptom-free period between the episode of trauma and the beginning of symptoms, thus establishing the non-traumatic nature of the disease.
c. **Lack of constitutional symptoms:** Symptoms like fever, loss of appetite, weight loss, etc., are present in only about 20 per cent cases. An active primary focus is detected only in about 15 per cent of cases

at the time of diagnosis; in the rest it has already healed by the time the patient presents.

Specific signs and symptoms in patients with tuberculosis at major sites will be discussed in respective sections.

INVESTIGATIONS

Radiological examination: X-ray examination of the affected part, anteroposterior and lateral views, is the single most important investigation. Findings in the early stages may be minimal and are likely to be missed. A comparison with an identical X-ray of the opposite limb or with an X-ray repeated after some period, may be helpful. Following are some of the general radiological features of tuberculosis of the bones and joints:

TB osteomyelitis: A tubercular osteomyelitis presents as a well-defined area of bone destruction, typically with minimal reactive new bone formation. This is unlike a pyogenic infection, where reactive periosteal new bone formation is an important feature.

TB arthritis: In tubercular arthritis there is reduction of the joint space, erosion of the articular surfaces and marked *peri-articular rarefaction.* This is unlike many other causes of joint space reduction such as osteoarthritis, septic arthritis, etc., where there is subchondral sclerosis instead.

X-ray features specific to different sites will be discussed in their respective sections. A chest X-ray should be done routinely to detect any tubercular lesion in the lungs.

Other investigations: Some of the following investigations may be helpful in diagnosis:

- *Blood examination:* Lymphocytic leukocytosis, high ESR.
- *Mantoux test* useful in children.
- *Serum ELISA* test for detecting anti-Mycobacterium antibodies.
- *Synovial fluid aspiration* (see Table 22.3, page 175).
- Aspiration of cold abscess and examination of pus for AFB.
- *Histopathological examination* of the granulation tissue obtained by biopsy or curettage of a lesion.

TREATMENT

Principles of treatment: Treatment of tuberculosis of bones and joints consists of control of the infection and care of the diseased part. In most cases, conservative treatment suffices; sometimes operative intervention is required.

Table 23.2: Common anti-tubercular drugs and their dosages.

Name/Daily dose (max.)	Side-effects
Bactericidal	
▪ Rifampicin (RF) 10 mg/kg (600 mg)	▪ Hepatotoxicity, pink coloured urine
▪ Isoniazide (INH) 5-10 mg/kg (300)	▪ Hepatotoxicity, peripheral neuritis
▪ Streptomycin (SM) 30 mg/kg (1 gm)	▪ Vestibular damage, nephrotoxicity circumoral paraesthesia
▪ Pyrazinamide (PZ) 25 mg/kg (1.5 gm)	▪ Hepatotoxicity
Bacteriostatic	
▪ Ethambutol (ETH) 25 mg/kg for 4 weeks (1000 mg), thereafter 15 mg/kg (800 mg)	▪ Optic neuritis, colour blindness
▪ Cycloserine 10 mg/kg (500 mg)	▪ CNS toxicity - headache, tremor, dysarthria
▪ Ethionamide 25 mg/kg (750 mg)	▪ Anorexia, nausea, vomiting
▪ Para-amino salicilate (PAS) 200-400 mg/kg (12 g)	▪ Anorexia, nausea, vomiting

Control of infection: It is brought about by potent anti-tubercular drugs, rest to the affected part and the building up of patient's resistance.

a. *Anti-tubercular drugs:* Table 23.2 shows common anti-tubercular drugs, their dosage, route of administration and common side-effects. It is usual practice to start the treatment with 4 drugs - Rifampicin, INH, Pyrazinamide, Ethambutol for 3 months. In selected cases with multifocal tuberculosis, 5 drugs - RF, INH, PZ, ETH and Streptomycin, may be required for the initial period. The patient is monitored* to detect any failure to respond or for any side-effects of the drugs.

b. *Rest:* The affected part should be rested during the period of pain. In the upper extremities this can be done with a plaster slab; in the lower extremities traction can be applied. In most cases of spinal tuberculosis bed rest for a short period is sufficient; in others, support with a brace may be necessary.

c. *Building up the patient's resistance:* The patient should be given a high protein diet and exposed to fresh air and sunlight to build up his general resistance.

* Multi-drug resistance is a serious upcoming problem. Anti-tubercular drugs, in proper combination, in proper dosages and under close supervision is the key to its prevention.

Care of the affected part: This consists of protection of the affected part from further damage, correction of any deformities and prevention of joint contractures. Once the disease is brought under control, exercises to regain functions of the joint are carried out. Care consists of the following:

a. *Proper positioning of the joint:* The joints should be kept in proper position so that contractures do not develop.
b. *Mobilisation:* As the disease comes under control and the pain reduces, joint mobilisation is begun. This prevents contractures and helps regain movement. In cases with extreme damage to the joint, it is best to expect ankylosis of the joint in the position of most useful function.
c. *Exercises:* As the joint regains movement, muscle strength building exercises are taught.
d. *Weight bearing:* It is started gradually as osteoporosis secondary to the disease is reversed.

Operative intervention may be required in some cases. Following are some procedures commonly used:

a. *Biopsy:* For cases where the diagnosis is in doubt, a fine needle aspiration cytology (FNAC) may be performed from an enlarged lymph node or from a soft tissue swelling. An open biopsy may be necessary from a bony lesion, or in case FNAC fails to confirm the diagnosis.
b. *Treatment of cold abscess:* A small stationary abscess may be left alone as it will regress with the healing of the disease. A bigger cold abscess may need aspiration or evacuation (discussed in detail on page 189).
c. *Curettage of the lesion:* If the lesion is in the vicinity of a joint, infection is likely to spread to the joint. An early curettage of the lesion may prevent this complication.
d. *Joint debridement:* In cases with moderate joint destruction, surgical removal of infected and necrotic material from the joint may be required. This helps in the early healing of the disease, and thus promotes recovery of the joint.
e. *Synovectomy:* In cases of synovial tuberculosis, a synovectomy may be required to promote early recovery.
f. *Salvage operations:* These are procedures performed for markedly destroyed joints in order to salvage whatever useful functions are possible, e.g., Girdlestone arthroplasty of the hip (page 196).
g. *Decompression:* In cases with paraplegia secondary to spinal TB, surgical decompression may be necessary.

TUBERCULOSIS OF THE SPINE (POTT'S DISEASE)

The spine is the *most common* site of bone and joint tuberculosis; the dorso-lumbar region being the one affected most frequently.

RELEVANT ANATOMY

Development of a vertebra (Fig. 23.2): A vertebra develops from the sclerotomes which lie on either side of the notochord. The lower-half of one vertebra and upper-half of the one below it, along with the intervening disc develop from each pair of sclerotomes and have a common blood supply. Therefore, infections via the arteries involve the 'embryological' section, as in the more common paradiscal tuberculosis of the spine.

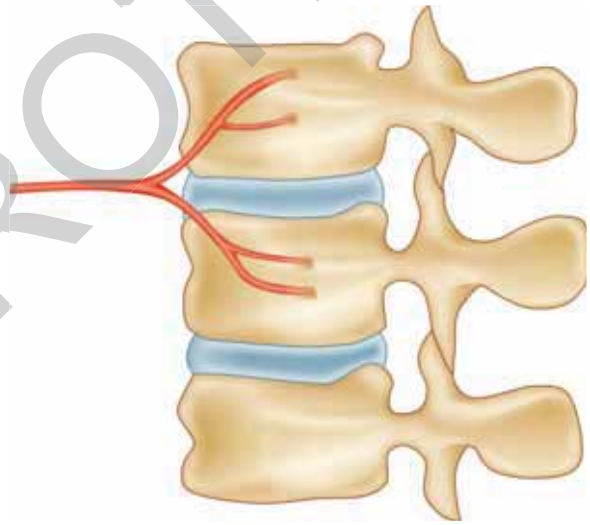


Fig. 23.2: Blood supply of vertebrae (diagrammatic, simplified).

Surface anatomy of the vertebral column: The only part of the vertebra which is accessible to palpation is its spinous process, hence this is used for localising the level of the vertebral segment. Table 23.3 shows the relationship between the vertebral spinous processes to that of some of the easily palpable anatomical landmarks. Once the affected vertebra

Table 23.3: Vertebral level – landmarks.

▪ Most prominent spinous process at the base of the neck	T_1
▪ At the level of the spine of the scapula	D_3
▪ At the level of lower angle of the scapula	D_7
▪ Floating rib	D_{12}
▪ At the level of the iliac crests	L_4
▪ At the level of the posterior superior iliac spine	S_2

Table 23.4: Relationship between spinal and cord segments.

Spinal segment	Cord segment
Cervical vertebrae	Add 1 to vertebral level
Upper dorsal vertebrae	Add 2 to vertebral level
Lower dorsal vertebrae	Add 3 to vertebral level
At D_{10}	All dorsal segments over
At D_{12}	All lumbar segments over
At L_1	All sacral segments over
Below L_1	Cauda equina

is known, the corresponding cord-segment can be found as discussed subsequently.

Cord-segment localisation: Because of the disproportionate growth of the vertebral column and spinal cord, the cord ends at the lower border of first lumbar vertebra. Beyond this, up to S2 there is only the dural sac containing a bunch of nerve roots (cauda equina). The segment of the cord which corresponds to a given vertebra is therefore above the level of that vertebra. Relationship between the spinal segment and cord segment in different regions of the spinal column is as shown in Table 23.4.

PATHOLOGY

Like tuberculosis of the bones and joints elsewhere in the body, TB of the spine is always secondary. The bacteria reach the spine via the haematogenous route, from the lungs or lymph nodes. It spreads via the paravertebral plexus of veins, i.e., Batson's plexus, which has free communication with the visceral plexus of the abdomen, a common site of tuberculosis.

Types of vertebral tuberculosis: Lesions in the vertebrae may be of the following types (Fig. 23.3):

a. *Paradiscal:* This is the *most common* type. In this, the contiguous areas of two adjacent vertebrae along with the intervening disc are affected.
b. *Central:* In this type, the body of a single vertebra is affected. This leads to early collapse of the weakened vertebra. The nearby disc may be normal. The collapse may be a 'wedging' or 'concertina' collapse (Fig. 23.4); wedging being more common.
c. *Anterior:* In this type, infection is localised to the anterior part of the vertebral body. The infection spreads up and down under the anterior longitudinal ligament.
d. *Posterior:* In this type, the posterior complex of the vertebra, i.e., the pedicle, lamina, spinous process and transverse process are affected.

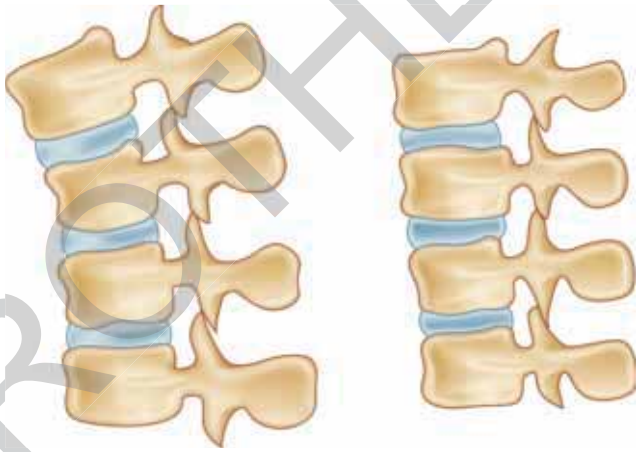


Fig. 23.4: Types of collapse of vertebrae.

Pathology: Basic pathology is the same as that in other bone and joint tuberculosis. In the more common paradiscal type, bacteria lodge in the contiguous areas of two adjacent vertebrae. Granulomatous inflammation results in erosion of the margins of these vertebrae. Nutrition of the intervening disc, which comes from the end-plates of the adjacent vertebrae is compromised. This results in disc degeneration, and as the process continues, complete *destruction.*

Weakening of the trabeculae of the vertebral body results in *collapse of the vertebra.* Type of collapse

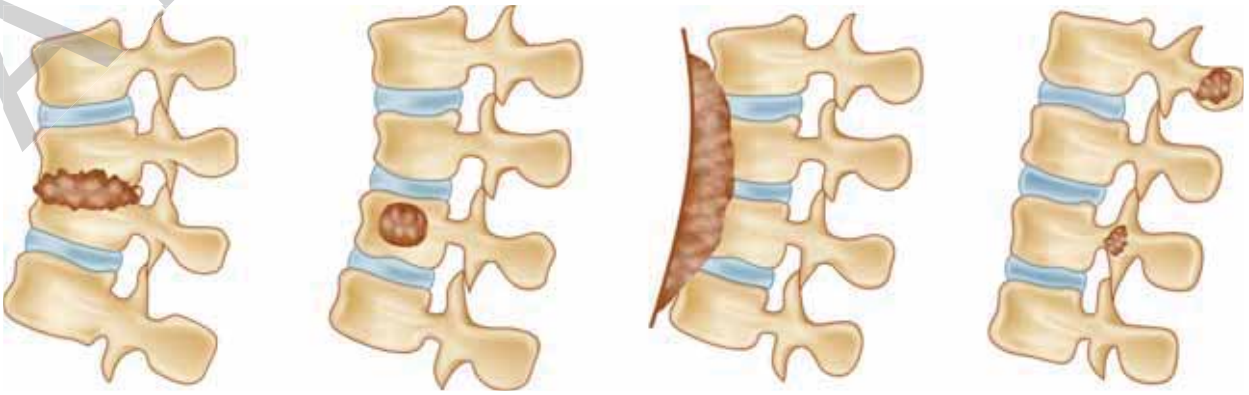


Fig. 23.3: Types of vertebral tuberculosis.

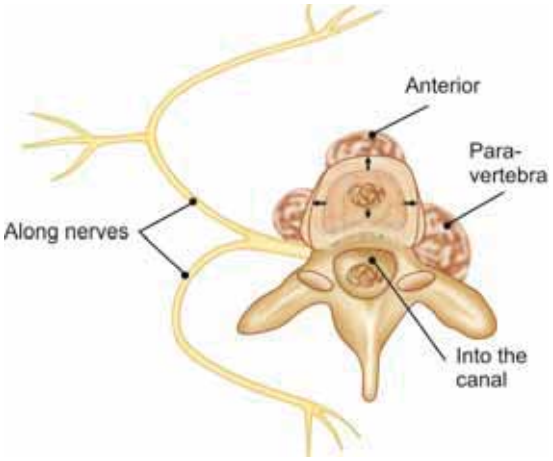


Fig. 23.5: Directions of tracking of tubercular pus from a vertebral focus.

is generally a wedging, occurs early, and is severe in lesions of the dorsal spine. This is because, in the dorsal spine the line of weight bearing passes anterior to the vertebra, so that the anterior part of the weakened vertebra is more compressed than the posterior, resulting in wedging. In the cervical and lumbar spines, because of their lordotic curvature (round forwards), wedging is less. Destruction occurs early, and is severe in children.

Cold abscess: This is a collection of pus and tubercular debris from a diseased vertebra. It is called a cold abscess because it is not associated with the usual signs of inflammation - heat, redness, etc., found with a pyogenic abscess. The tubercular pus can track in any direction from the affected vertebra (Fig. 23.5). If it travels backwards, it may press upon the important neural structures in the spinal canal. Pus may come out anteriorly (prevertebral abscess) or on the sides of the vertebral body (paravertebral abscess). Once outside the vertebra the pus may travel along the musculo-fascial planes or neurovascular bundles to appear superficially at places far away from the site of lesion.

Healing: As healing occurs, the lytic areas in the bone are replaced by new bone. The adjacent vertebrae undergo fusion by bony-bridges. Whatever changes have occurred in the shape of the vertebral body are, however, permanent.

CLINICAL FEATURES

Presenting complaints: Clinical presentations of a case of TB of the spine is very variable - from a seemingly non-specific pain in the back to complete paraplegia. Following are some of the common presenting complaints:

- **Pain:** Back pain is the most common presenting symptom. It may be diffuse; no more than a dull ache in the early stages, but later becomes localised to the affected diseased segment. It may be a 'radicular' pain i.e., a pain, radiating along a nerve root. Depending upon the nerve root affected, it may present as pain in the arm (cervical roots), girdle pain (dorsal roots), pain abdomen (dorso-lumbar roots), groin pain (lumbar roots) or 'sciatic' pain (lumbo-sacral roots).
- **Stiffness:** It is a very early symptom in TB of the spine. It is a protective mechanism of the body, wherein the para-vertebral muscles go into spasm to prevent movement at the affected vertebra.
- **Cold abscess:** The patient may present the first time with a swelling (cold abscess) or problems secondary to its compression effects on the nearby visceral structures, such as dysphagia in TB of the cervical spine. A detailed examination in such cases reveals underlying TB of the spine.
- **Paraplegia:** If neglected, which is often the case in developing countries, a case of TB of the spine presents with this serious complication. For details see Pott's paraplegia, page 189.
- **Deformity:** Attention to TB of the spine may be attracted, especially in children, by a gradually increasing prominence of the spine - a gibbus.
- **Constitutional symptoms:** Symptoms like fever, weight loss, etc., are rarely the only presenting symptoms.

EXAMINATION

The aim of examination is: (i) to pick up findings suggestive of tuberculosis of the spine; (ii) to localise the site of lesion; (iii) find skip lesions; and (iv) to detect any associated complications like cold abscesses or paraplegia. Following is the systematic way in which one should proceed to examine a case of suspected TB of the spine.

- **Gait:** A patient with TB of the spine walks with short steps in order to avoid jerking the spine. He may take time and may be very cautious while attempting to lie on the examination couch. In TB of the cervical spine, the patient often supports his head with both hands under the chin and twists his whole body in order to look sideways.
- **Attitude and deformity:** A patient with TB of the cervical spine has a stiff, straight neck. In dorsal spine TB, part of the spine becomes prominent (*gibbus or kyphus*).* Significant deformity is generally absent in lumbar spine tuberculosis; there may just be loss of lumbar lordosis.

* There are three types of kyphotic deformities:
i. Knuckle – prominence of one spinous process
ii. Gibbus – prominence of two or three spinous processes
iii. Kyphus – diffuse rounding of the vertebral column

Table 23.5: Presentation of cold abscesses from different regions of the spine.

Region of spine	Presentation			
	Anteriorly	On the sides	Along musculo-fascial plane	Along neurovascular plane
Cervical spine	Retropharyngeal abscess	Paravertebral abscess	At the posterior border of sternocleidomastoid muscle, in the *posterior triangle* of neck	To axilla, to arm along neurovascular bundle of the arm
Thoracic spine	Mediastinal abscess	Paravertebral abscess	Trickles downward and enters either of the two lumbocostal arches: ▪ Lateral lumbocostal arch - To present as *lumbar abscess* ▪ Medial lumbocostal arch - To present as *psoas abscess*	Along thoracic spinal nerves to present at ▪ Anterior chest wall ▪ Midaxillary line ▪ Posterior chest wall
Lumbar spine	Prevertebral abscess	Paravertebral abscess	Lumbar abscess or psoas abscess lower	Along neurovascular bundle of the leg to present in groin or lower down in the leg

- **Paravertebral swelling:** A superficial cold abscess may present as fullness or swelling on the back, along the chest wall or anteriorly. It is easy to diagnose because of its fluctuant nature. Sometimes, an abscess may be tense and it may not be possible to elicit fluctuation. A needle aspiration may be performed in such cases, to confirm the diagnosis. It is important to look for cold abscesses in not so obvious locations, depending upon the region of the spine affected (Table 23.5).
- **Tenderness:** It can be elicited by pressing upon the side of the spinous process in an attempt to rotate the vertebra.
- **Movement:** There is no necessity to examine for spinal movement in a patient with obviously painful spine. Spinal movement are limited in a case of TB of the spine, and can be tested, wherever considered suitable.
- **Neurological examination:** A thorough neurological examination of the limbs, upper or lower, depending on the site of tuberculosis should be performed. In addition to motor, sensory and reflexes examination, an assessment should be made of urinary or bowel functions. Aim of neurological examination is to find: (i) whether or not there is any neurological compression; (ii) level of neuro- logical compression; and (iii) severity of neurological compression.
- **General examination:** A general physical examination should be performed to detect any active or healed primary lesion. The patient may have some other systemic illness like diabetes, hypertension, jaundice, etc., which may have a bearing on further treatment.

RADIOLOGICAL INVESTIGATIONS

X-ray examination: One must *specify* the level of the suspected damage, when requisitioning an X-ray of the spine. Minimum of two views, AP and lateral, are necessary. A chest X-ray for primary focus or an X-ray of the abdomen – KUB, if a psoas abscess is suspected, may also be taken. Following are some of the important radiological features.

- *Reduction of disc space:* This is the *earliest sign* in the more common, paradiscal type of tuberculosis (Fig. 23.6A). In early stages, reduction in disc space may be minimal, and may be detectable only on comparing the height of the suspected disc with those above and below it. In advanced stages, disc space may be completely lost (Fig. 23.6B). A lateral X-ray is better for evaluation of disc space. Reduction of disc space is an important sign because in other diseases of the spine, e.g., secondaries in the spine, the disc space is well-preserved.
- *Destruction of the vertebral body:* In early stages, the contiguous margins of the affected vertebrae may be eroded. The diseased, weakened vertebra may undergo wedging. In late stages, a significant part or whole of the vertebral body may be destroyed (Fig. 23.6C), leading to angular kyphotic deformity. Severity of the deformity depends upon the extent of wedging and number of affected vertebrae (Table 23.6).
- *Evidence of cold abscess:* Radiological evidence of a cold abscess is a very useful finding in diagnosing a case of suspected spinal TB. Following abscesses may be seen on X-rays:
  - *Paravertebral abscess:* A paravertebral soft tissue shadow corresponding to the site of the affected vertebra in AP view indicates a paravertebral abscess. It may be of the following types: (i)

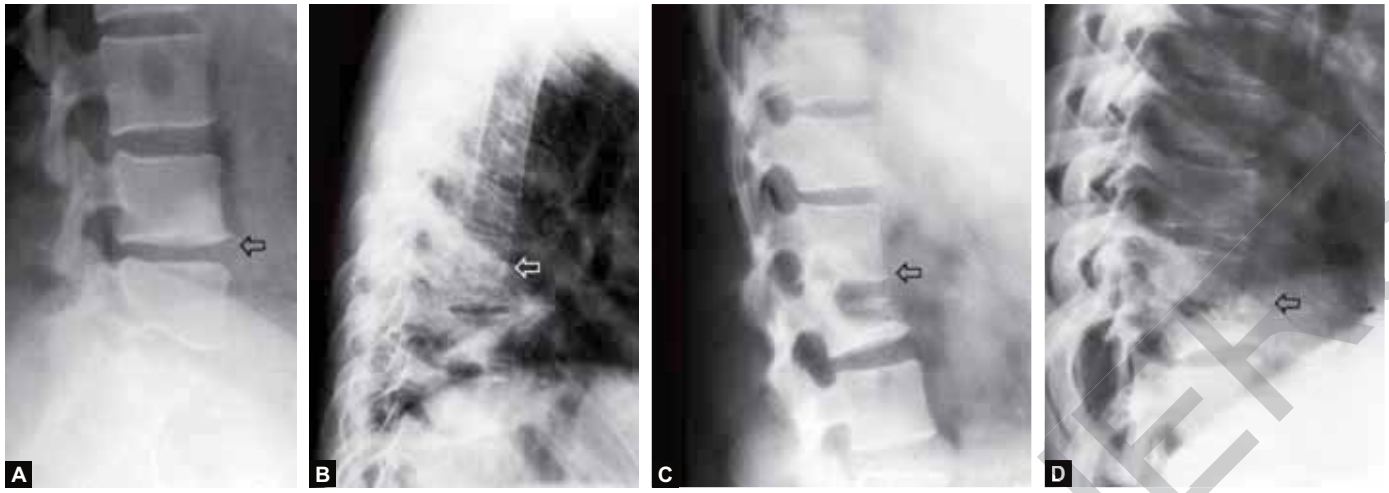


Fig. 23.6: X-ray findings in TB of the spine: (A) Early case, minimal loss of disc prolapse; (B) Complete loss of disc prolapse; (C) Destruction of vertebral bodies with loss of disc prolapse; (D) Advance destruction and wedging of vertebrae.

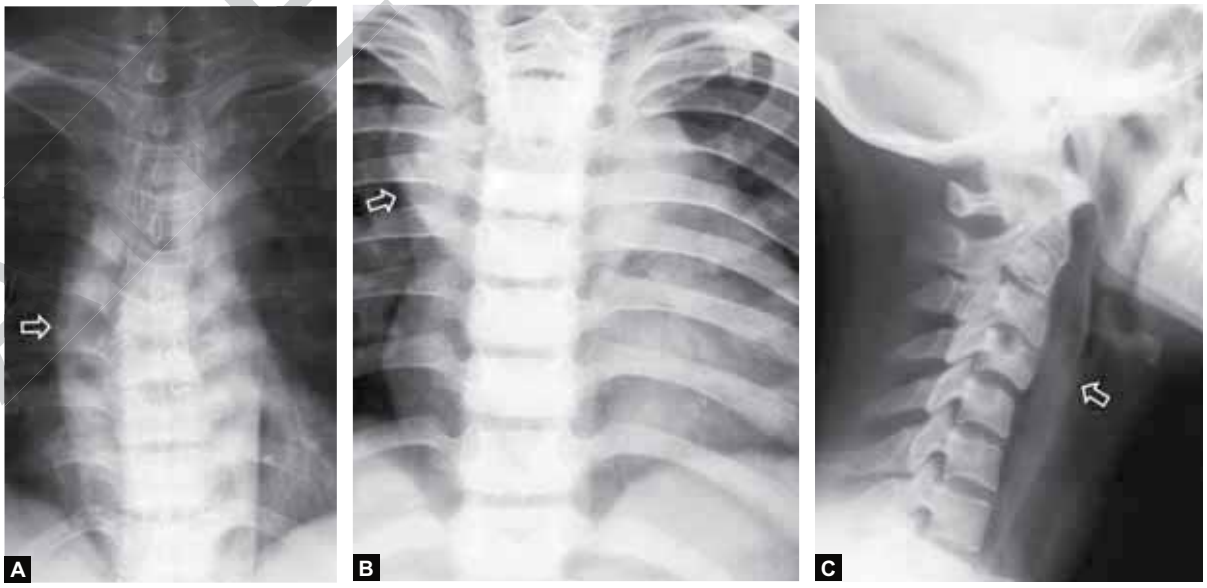
a fusiform paravertebral abscess (bird nest abscess - an abscess whose length is greater than its width (Fig. 23.7A); and (ii) globular or tense abscess - an abscess whose width is greater than the length (Fig. 23.7B). The latter indicates pus under pressure and is commonly associated with paraplegia.

Table 23.6: Number of affected vertebrae.

Type	No. of vertebrae involved
Knuckle	1
Gibbus	2–3
Angular kyphosis	3–4
Rounded kyphosis	>4

- *Widened mediastinum:* An abscess from the dorsal spine may present as widened mediastinum on AP X-ray.
- *Retropharyngeal abscess:* In cervical spine TB, a retropharyngeal abscess may be seen on a lateral X-ray. Normally, soft tissue shadow in front of the C3 vertebral body is 4 mm thick; an increase in its thickness indicates a retropharyngeal abscess (Fig. 23.7C).
- *Psoas abscess*: In dorso-lumbar and lumbar tuberculosis, psoas shadow on an X-ray of the abdomen may show a bulge.

- *Rarefaction:* There is diffuse rarefaction of the vertebrae above and below the lesion.
- *Unusual signs:* In tuberculosis involving the posterior complex, there may be erosion of the



Fusiform abscess Tense abscess Retropharyngeal abscess

Fig. 23.7: Types of paravertebral abscesses.

posterior elements of pedicle, lamina, etc. These are better visible on oblique X-rays of the spine. Anterior type of vertebral tuberculosis may show erosion of the anterior part of the body, much the same as that possibly seen sometimes in cases with aneurysm of aorta, thus termed *aneurysmal sign.* There may be lytic lesions in the ribs in the vicinity of the affected vertebra.

- *Signs of healing:* Once the disease starts healing, the density of the affected bones gradually improves. Areas surrounding the lytic lesion show sclerosis, and over a period of time these lesions are replaced by sclerotic bone. The adjacent vertebrae undergo bony fusion.

CT scan: It may detect a small paravertebral abscess, not otherwise seen on plain X-ray; may indicate precisely the extent of destruction of the vertebral body and posterior elements; and may show a sequestrum or a bony ridge pressing on the cord (Fig. 23.8). This is a very useful investigation in cases presenting as 'spinal tumour syndrome', where there may be no signs on plain X-rays.

MRI is the investigation of choice to evaluate the type and extent of compression of the cord (Fig. 23.9). It also shows condition of the underlying neural tissues, and thus helps in predicting the prognosis in a particular case.

Biopsy: CT guided needle biopsy, or an open biopsy may be required in a case with doubtful diagnosis.

Other general investigations: Investigations like ESR, Mantoux test, ELISA test for detecting anti-tubercular antibodies, chest X-ray, etc., to support the diagnosis of tuberculosis, may be carried out whenever required.

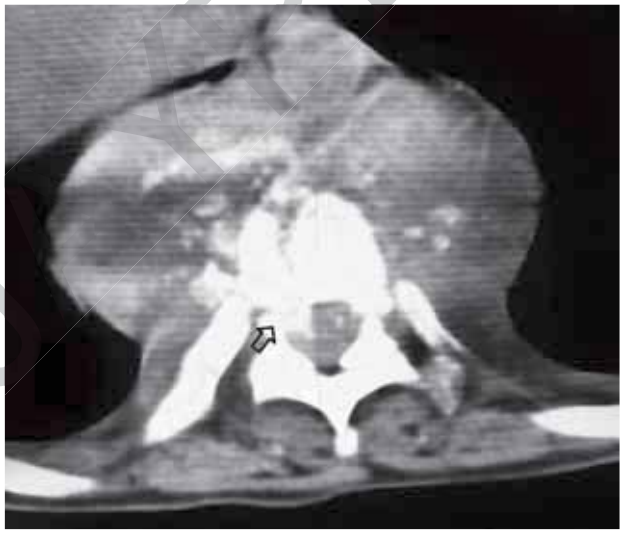


Fig. 23.8: CT scan of a case of TB spine. (Note bony fragments in the canal).

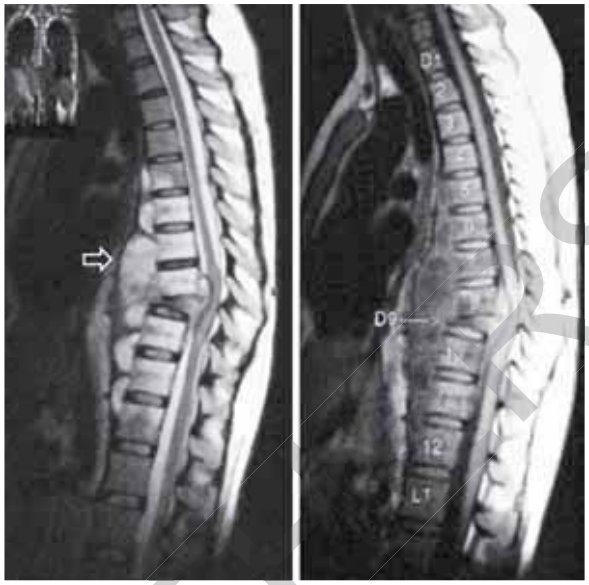


Fig. 23.9: MRI of the spine (T_1 and T_2 images), showing TB spine. (Note the compression on the cord and huge prevertebral abscess).

DIFFERENTIAL DIAGNOSIS

Cases with TB of the spine report fairly late in developing countries, so they present mostly with classic signs, symptoms and radiological features. In the early stages, and sometimes in some atypical presentations, diagnosis may be difficult. Some of the common differential diagnosis and their differentiating features are given in Table 23.7.

TREATMENT

Principles of treatment: Aim of treatment is: (i) to achieve healing of the disease and (ii) to prevent, detect early, and treat promptly any complication like paraplegia, etc. Treatment consists of anti-tubercular chemotherapy (page 182), general care (page 182), care of the spine, and treatment of the cold abscess. Only the latter two will be discussed here.

Care of the spine: This consists of providing rest to the spine during the acute phase, followed by guarded mobilisation.

- *Rest:* A short period of bed rest for pain relief may be sufficient during early stages of treatment. In cases with significant vertebral destruction, a longer period of bed rest is desirable to prevent further collapse and pathological dislocation of the diseased vertebrae. In children, a body cast is sometimes given, basically to force them to rest. Minerva jacket or a collar may be given for immobilising the cervical spine.
- *Mobilisation:* As the patient improves, he is allowed to sit and walk while the spine is supported in a collar for the cervical spine, or an ASH brace for the dorso-lumbar spine. The patient is weaned off

Table 23.7: TB of the spine: Differential diagnosis.

Symptoms	Clinical features	Investigations
Back pain		
Traumatic	History of trauma present No fever or abscess	▪ X-ray – disc height normal. ▪ Wedging of vertebrae present ▪ No paravertebral shadow seen
Secondaries/ myeloma	History of 'primary' elsewhere or myeloma	▪ Disc space normal ▪ Pedicles may be involved in secondaries ▪ Lesions in other bones present
Prolapsed disc	▪ Radiating pain ▪ SLRT – positive ▪ Localised nerve root deficit	Normal X-rays
Ankylosing spondylitis	▪ Chronic back pain, starts in lower back ▪ Diffuse morning stiffness ▪ Chest expansion reduced	▪ 'Bamboo spine' appearance on X-rays ▪ SI joints affected – hazy, fused
Neurological deficit		
Spinal tumour	▪ Present with gradually increasing neurological deficit ▪ No back pain, or other findings on spine examination	▪ X-ray – interpedicle space increased ▪ Pedicle erosion present ▪ CT myelogram confirms
Traumatic	▪ History of definite trauma present ▪ Weakness is sudden onset	X-ray suggestive of fracture-dislocation
Secondaries in the spine	▪ No history of trauma ▪ Back pain present ▪ History of 'primary' elsewhere	X-ray shows erosion of vertebrae

the brace once bony fusion occurs. He is advised to avoid sports for 2 years.

Treatment of cold abscess: A small cold abscess may subside with anti-tubercular treatment. Abscesses presenting superficially need treatment as discussed below:

- *Aspiration:* A thick needle is required because often there is thick caseous material. It should be an anti-gravity insertion with the needle entering through a zig-zag tract.
- *Evacuation:* In this procedure, the cold abscess is drained, its walls curetted, and the wound closed without a drain. This is unlike drainage of a pyogenic abscess, where a post-operative drain is always left. A psoas abscess can be drained extraperitoneally using a kidney incision.

Medical Research Council of Great Britain conducted controlled trials to study various aspects of TB spine and published findings in four reports (1973-74). Their conclusions were that: (i) Bed rest is not necessary; (ii) Streptomycin is not necessary; (iii) PoP jacket offers no benefit; and (iv) Debridement is not a good operation.

COMPLICATIONS

1. **Cold abscess:** This is the most common complication of TB of the spine. Treatment is as discussed above.
2. **Neurological compression:** At times the patient presents as a case of spinal tumour syndrome; the first clinical symptom being a neurological deficit (discussed subsequently).

POTT'S PARAPLEGIA (TB SPINE WITH NEUROLOGICAL INVOLVEMENT)

The incidence of neurological deficit has been reported to be 20 per cent. It occurs *most commonly* in tuberculosis of the dorsal spine because the spinal canal is narrowest in this part, and even a small compromise can lead to a neurological deficit.

PATHOLOGY

This consists of pressure on the neural tissues within the canal by products from the diseased vertebrae. It could occur in the following ways:

- **Inflammatory oedema:** The neural tissues become oedematous because of vascular stasis in the adjacent diseased area.
- **Extradural pus and granulation tissue:** This is the *most common* cause of compression on neural structures. The abscess formed around the diseased vertebrae may compress the neural structures from the front, much the same way as an extradural tumour.

- **Sequestra:** Devascularised bone and extruded disc material may be displaced into the canal.
- **Internal 'gibbus':** Angulation of the diseased spine may lead to formation of the bony ridge on the anterior wall of the spinal canal (Fig. 23.10). This is called the internal gibbus.

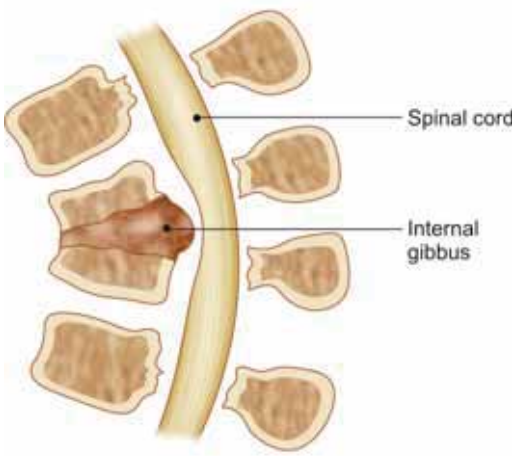


Fig. 23.10: Internal gibbus.

- **Infarction of the spinal cord:** This is an unusual but important cause of paralysis. It results from blockage of the anterior spinal artery, caused by the inflammatory reaction.
- **Extradural granuloma:** Very rarely, an extradural granuloma may form without any damage to the osseous structures. Such a patient presents with a clinical picture of a spinal tumour - the so-called *'Spinal tumour syndrome'*.

Types of Pott's paraplegia: It can be divided into two types:

a. **Early onset paraplegia,** i.e., paraplegia occurring during the active phase of the disease, usually within two years of onset of the disease.
b. **Late onset paraplegia,** i.e., paraplegia occurring several years after the disease has become quiescent, usually at least two years after the onset of disease.

Pathology of the two types is different, as also is the prognosis. Table 23.8 gives the causes of neurological deficit in early and late onset paraplegia.

CLINICAL FEATURES

Neurological complications can occur in a known case of tuberculosis of the spine; or the case may present for the first time with a neurological deficit. In the latter, tuberculosis as the cause of paraplegia is detected only on examination and further investigation. Onset of paraplegia is gradual in *most* cases, but in some it is sudden. Tubercular paraplegia is usually spastic to start with. Clonus (ankle or patellar) is the most prominent early sign. Paralysis may pass with varying rapidity, through the following stages:

- **Muscle weakness**, spasticity and in-coordination due to pressure on the corticospinal tracts which are placed anteriorly in the cord and are probably more sensitive to pressure.
- **Paraplegia in extension:** Tone of the muscles is increased due to absence of normal corticospinal inhibition, resulting in paraplegia in extension.
- **Paraplegia in flexion:** Absence of paraspinal tract functions in addition to the corticospinal functions leads to paraplegia in flexion.
- **Complete flaccid paraplegia:** Paraplegia becomes completely flaccid once all transmission across the cord stops.

Grades of Pott's paraplegia: Potts' paraplegia has been graded on the basis of degree of motor involvement, into four grades (Goel, 1967):

Grade I: Patient is unaware of the neural deficit; the physician detects Babinski positive and ankle or patellar clonus on clinical examination.

Grade II: Patient presents with complaints of clumsiness, in-coordination or spasticity while walking, but manages to walk with or without support.

Grade III: Patient is not able to walk because of severe weakness. On examination, he has paraplegia in extension. There may be partial loss of sensation.

Grade IV: Patient is unable to walk, and has paraplegia in flexion with severe muscle spasm. There is near complete loss of sensation with sphincter disturbances.

Table 23.8: Causes* of paraplegia in TB of the spine.

Early onset paraplegia
- *Inflammatory causes*
  - Abscess - most common
  - Granulation tissue
  - Circumscribed tuberculous focus
  - Posterior spinal disease
  - Infective thrombosis of the spinal blood supply
- *Mechanical causes*
  - Sequestrum in the canal
  - Infected degenerated disc in the canal
  - Pathological dislocation - a ridge of bone pressing on the cord

Late onset paraplegia
- Recurrence of the disease
- Prominent anterior wall of the spinal canal in case of severe kyphosis (internal gibbus)
- Fibrous septae following healing

* Although these several mechanisms have been described as acting separately to produce paraplegia, more than one cause may be responsible in a particular patient.

INVESTIGATIONS

It is usually possible to diagnose vertebral tuberculosis as a cause of paraplegia by typical radiological signs. In some cases, a MRI scan may be done to see: (i) type of vertebral destruction; (i) presence of paravertebral soft tissue abscess; and (iii) cause of paraplegia, i.e., whether it is pus, sequestra, etc. CT scan may be required in some cases to better evaluate the vertebral canal. MRI is the investigation of choice, wherever available.

TREATMENT

Principles of treatment: Aims of treatment are as follows:

a. To *promote recovery* of the affected neural tissues, by reversing the cause responsible for compression, either by drugs or by operation.
b. To *achieve healing* of the vertebral lesion, and to support the spine till the diseased segment becomes stable.
c. To undertake *rehabilitative* measures to prevent contractures, and to regain strength in the affected part.

Treatment of Pott's paraplegia has been the topic of considerable study and discussion. Following is the treatment considered most acceptable in the author's opinion. Treatment may be divided into conservative and operative. All cases of Pott's paraplegia must be treated under supervision, after admission to a hospital.

Conservative treatment: Anti-tubercular chemotherapy forms the mainstay of treatment. All patients are started on 4-drugs anti-tubercular chemotherapy as soon as the diagnosis is made. The spine is put to absolute rest by a sling traction for the cervical spine, and bed rest for the dorso-lumbar spine. The paralysed limbs are taken care of, as discussed in the Chapter 32. During treatment, repeated neurological examination of the limbs is carried out to detect any deterioration or improvement in the neurological status.

If paraplegia improves, conservative treatment is continued. Patient is allowed to sit in the bed with the help of a brace as soon as the spine has gained sufficient strength. Bracing is continued for a period of about 6 to 12 months.

Operative treatment: If paraplegia does not improve at a satisfactory rate, or if it actually deteriorates; surgical intervention is indicated. Following are the indications for surgery considered suitable in most centres.

Absolute indications

1. Paraplegia occurring during usual conservative treatment.
2. Paraplegia *getting worse* or remaining stationary despite adequate conservative treatment.
3. Severe paraplegia with *rapid onset* may indicate severe pressure from a mechanical accident or abscess.
4. Any *severe paraplegia* such as paraplegia in flexion, motor or sensory loss for more than six months, complete loss of motor power for one month despite adequate conservative treatment.
5. Paraplegia *accompanied by uncontrolled spasticity* of such severity that reasonable rest and immobilisation are not possible.

Relative indications

1. Recurrent paraplegia, even with paralysis that would cause no concern in the first attack.
2. Paraplegia with *onset in old age:* Indications for surgery are stronger because of the hazards of recumbency.
3. *Painful paraplegia,* pain resulting from spasm or root compression.
4. *Complications* such as urinary tract infection and stones.

Rare indications

1. Paraplegia due to posterior spinal disease.
2. Spinal tumour syndrome.
3. Severe paralysis secondary to the cervical disease.
4. Severe cauda equina paralysis.

Operative procedures for Pott's paraplegia: The operative method aims at removal of the agents causing compression on the neural structures. The following operations are commonly performed:

a. **Costotransversectomy (Fig. 23.11A):** As the name suggests, this operation consists of the removal of a section of rib (about 2 inches), and transverse process. As this is done, sometimes liquid pus comes out under pressure. This is considered by some as a tense abscess relieved, and thus enough to decompress the neural tissues. It is indicated

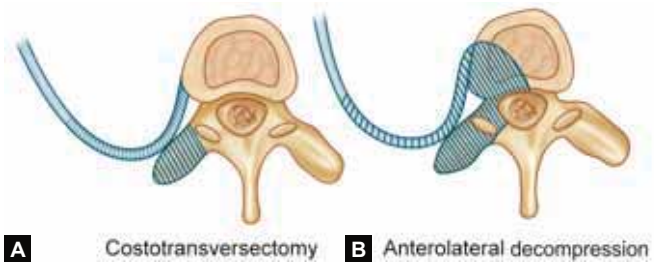


Fig. 23.11: Structures to be removed.

in a child with paraplegia, and when a tense abscess is visible on X-ray. In all other cases, it may not produce adequate decompression and an anterolateral decompression may be necessary.

b. **Anterolateral decompression (ALD):** This is the most commonly performed operation. In this operation, the spine is opened from its lateral side and access is made to the front and side of the cord, thus it is called anterolateral decompression. The cord is laid free of any granulation tissue, caseous material, bony spur or sequestrum pressing on it. Structures removed in order to achieve adequate exposure of the cord are; the rib, transverse process, pedicle and part of the body of the vertebra (Fig. 23.11B). Lamina or facet joints are *not* removed, otherwise stability of the spine will be seriously jeopardized.

c. **Radical debridement and arthrodesis (Hong Kong operation):** Wherever facilities are available, a radical debridement is performed by exposing the spine from front using transthoracic or transperitoneal approaches. All the dead and diseased vertebrae are excised and replaced by rib grafts. Advantage of this operation is early healing of the disease and no progress of the kyphosis.

d. **Laminectomy:** It is indicated in cases of spinal tumour syndrome, and those where paraplegia has resulted from posterior spinal disease.

Surgery for the cervical spine tuberculosis requires a separate technique; anterior decompression is preferable in this area.

PROGNOSIS

Prognosis of Pott's paraplegia depends upon the following factors:

1. **Age:** Children respond to treatment better than adults.
2. **Onset:** Acute onset paraplegia has a better prognosis.
3. **Duration:** Long-standing paraplegia has a worse prognosis.
4. **Severity:** Motor paralysis alone has a good prognosis. Sphincter involvement, i.e., urinary or bowel incontinence are bad prognostic indicators.
5. **Progress:** Sudden progress of the paraplegia has a bad prognosis.

TUBERCULOSIS OF THE HIP

After spine, the hip is affected, most commonly. It usually occurs in children and adolescents, but patients at any age can be affected.

PATHOLOGY

The basic pathology is the same as that discussed on page 181. The usual initial lesion is in the bone adjacent to the joint, i.e., either the acetabulum or the head of the femur (osseous tuberculosis). In some cases, the lesion may begin in the synovium (synovial tuberculosis), but quickly the articular cartilage and the bones are affected. A purely synovial tuberculosis, as seen in the knee joint, is uncommon in the hip. Common sites of initial bone focus in TB of the hip are as shown in Figure 23.12.

Natural history: The infected granulation tissue harbouring the bacilli, from the initial bony focus erodes the overlying cartilage or bone and reaches the joint. In early stage, this results in synovial hypertrophy and effusion. The pannus of hypertrophied synovium around the articular cartilage gradually extends over and under it. Cartilage is thus destroyed and the joint becomes full of pus and granulation tissue. Synovium gets thickened, oedematous, grey and ulcerated. Denuded of their protective cartilage, the bone ends become raw.

Multiple *cavitation* is typical of tuberculosis. Such cavities are formed in the femoral head and the acetabulum. Eventually, the head or the acetabulum gets partially absorbed. By the constant pull of the muscles acting on the hip, the remaining head of the femur may dislocate from the acetabulum onto the ilium, giving rise to the so-called *wandering acetabulum* (Fig. 23.13). In later stages, pus bursts through the capsule and spreads in the line of least resistance. It may present as cold abscess in the groin or in the region of the greater trochanter. Pus may perforate the acetabulum and appear as a pelvic abscess.

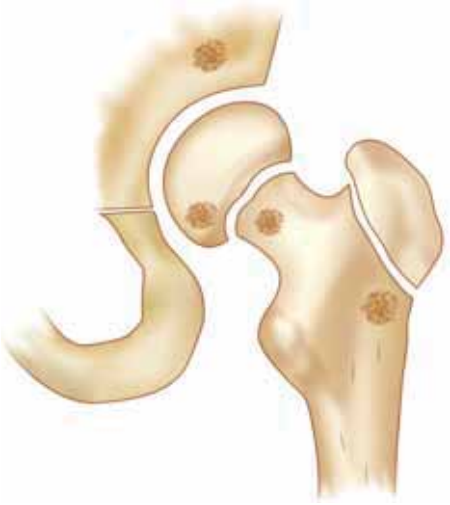


Fig. 23.12: TB hip: Common sites.

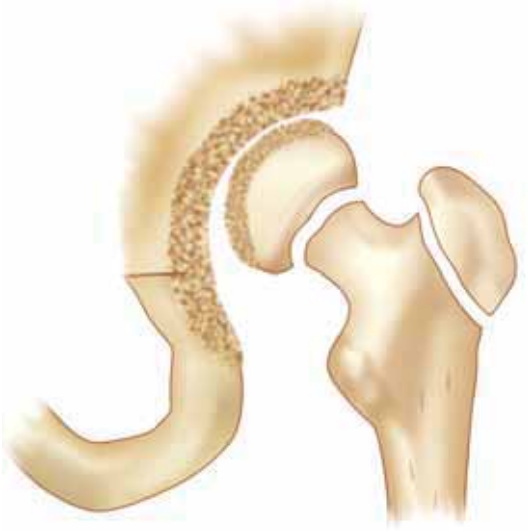


Fig. 23.13: 'Wandering' acetabulum.

Healing: If left untreated, healing may take place by fibrosis, leading to ankylosis of the hip usually in a deformed position (fibrous ankylosis).

CLINICAL FEATURES

Presenting complaints: The disease is insidious in onset and runs a chronic course. The child may be apathetic and pale with loss of appetite before definite symptoms pertaining to the hip appear. One of the first symptoms is stiffness of the hip, and it produces a limp. Initially, stiffness may occur only after rest, but later it persists all the time.

Pain may be absent in early stages, or if present, may be referred to the *knee*. The child may complain of 'night cries', the so called 'starting pain', caused by the rubbing of the two diseased surfaces, when movement occurs as a result of the muscle relaxation during sleep. Later, there may be cold abscesses around the hip or these may burst, resulting in discharging sinuses.

EXAMINATION

It should be carried out with the patient undressed. Following physical findings may be present:

- **Gait:** Lameness is one of the *first* signs. In the early stage, it is because of stiffness and deformity of the hip. Because of the flexion deformity at the hip, the child stands with compensatory exaggerated lumbar lordosis. While walking the hip is kept stiff. Forward-backward movement at the lumbar spine is used for propulsion of the lower limb. This is called the 'stiff-hip gait'. Later the limp is exaggerated by pain so the child hastens to take the weight off the affected side. This is called the 'painful or antalgic gait'.
- **Muscle wasting:** The thigh muscles and gluteal muscles are wasted.
- **Swelling:** There may be swelling around the hip because of a cold abscess.
- **Discharging sinuses:** There may be discharging sinuses in the groin or around the greater trochanter. There may be puckered scars from healed sinuses.
- **Deformity:** Gross deformities may be obvious on inspection. Minimal deformities are compensated for by pelvic tilt and can be made obvious by tests. Commonly it is flexion, adduction and internal rotation deformity of the hip. Method of measuring deformities is as discussed in Annexure I.
- **Shortening:** There is generally a true shortening in TB of the hip, except in Stage I, in which an apparent lengthening occurs (Annexure I). Limb length discrepancy can occur at this joint not only because of actual shortening of the bones (true length) but also because of the adduction-abduction deformity, which results in pelvic tilt and thus affects the length of the limb (apparent length). The method of measuring true and apparent lengths is as discussed in Annexure I. True and apparent length, and their relation to different deformities of the hip are given in Table 23.9.
- **Movements:** Both, active and passive movements are limited in all directions. An attempted movement is associated with muscle spasm.

There may be severe limitation of movements, both active and passive, in all directions in late cases of tuberculosis. This is called ankylosis of the hip. If there is no movement at all, it is bony ankylosis.

- **Abnormal position of the head:** In a dislocated hip, the head can be felt in the gluteal region.
- **Telescopy:** This test assesses the instability of the head if it is out of the acetabulum (details in Annexure I).

Table 23.9: Hip deformities, ASIS and limb length.

Deformity	ASIS level	Limb length
Adduction	Higher	Apparent < true (apparent shortening)
Abduction	Lower	Apparent > true (apparent lengthening)
No adduction-abduction	Same	Apparent = true

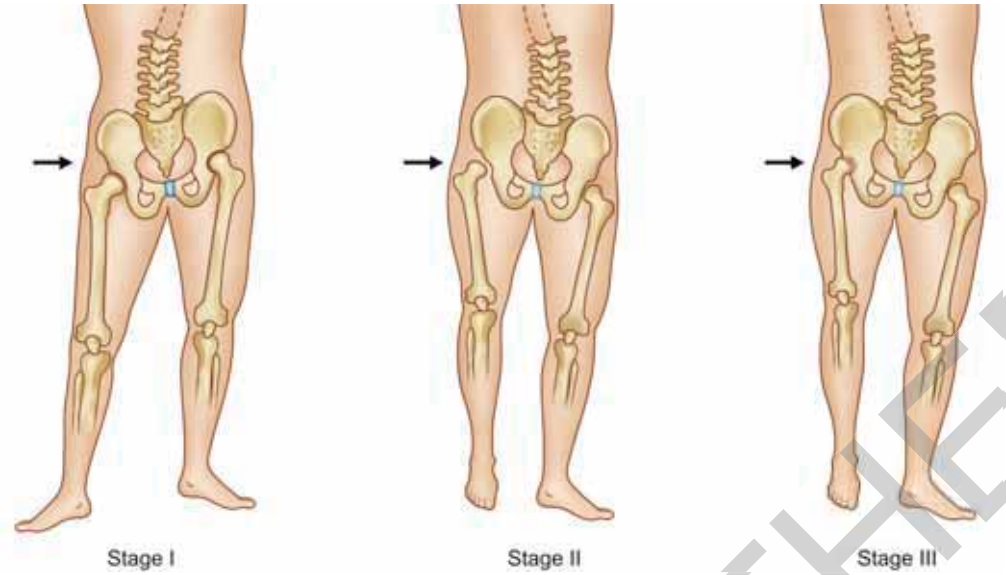


Fig. 23.14: Stages of TB hip.

STAGES OF TB OF THE HIP

TB of the hip has been arbitrarily divided into three stages in its clinical course (Fig. 23.14).

Stage I (stage of synovitis): There is effusion into the joint which demands the hip to be in a position of maximum capacity. This is a position of flexion, abduction and external rotation. Since flexion and abduction deformities are only slight and are compensated for by tilting of the pelvis, these do not become obvious. The limb remains in external rotation. As the pelvis tilts downwards to compensate for the abduction deformity, the affected limb appears longer (apparent lengthening), though on measuring true limb lengths, the two limbs are found to be equal. This stage is also called the *stage of apparent lengthening*. It lasts for a very short period. Very rarely does a patient present to the hospital in such an early stage of the disease.

Stage II (stage of the arthritis): In this stage, the articular cartilage is involved. This leads to spasm of the powerful muscles around the hip. Since the flexors and adductors are stronger muscle groups than the extensors and abductors, the hip takes the attitude of flexion, adduction and internal rotation. Flexion and adduction may be concealed by compensatory tilt of the pelvis but internal rotation of the leg is obvious. As the pelvis tilts upwards to compensate for the adduction, the affected limb appears shorter (apparent shortening), although on comparing the limb lengths in similar positions, the two limbs are equal. This is also called the *stage of apparent shortening*.

Stage III (stage of erosion): In this stage, the cartilage is destroyed and the head and/or the acetabulum is eroded. There may be a pathological dislocation or subluxation of the hip. Attitude of the limb is the same as that in Stage II, i.e., flexion, adduction and internal rotation except for the fact that the deformities are exaggerated. There is *true shortening* of the limb because of the actual destruction of the bone. In addition, apparent length of the limb is further reduced because of the adduction deformity.

INVESTIGATIONS

Radiological examination: An X-ray examination of the pelvis with both hips, AP and lateral views of the affected hip are essential. Inclusion of the normal hip in the same film on the AP view helps in comparing the joint spaces on the two sides. MRI scan and bone scan may be useful in early diagnosis. Some of the radiological signs in an established case of TB of the hip are as follows:

- *Haziness:* Haziness of the bones around the hip is the *earliest* sign. To appreciate it best, the affected hip is compared with the normal hip.
- *Lytic lesion:* There may be lytic lesions in the regions specified in Figure 23.12, on page 192).
- *Reduction of joint space:* This occurs because of destruction of the cartilage. It may be uniformly or irregularly diminished, better appreciated in the early stages on comparing it with the opposite side (Fig. 23.15A).
- *Irregular outline:* The outline of the articular ends of the bone becomes irregular because of destruction by the disease process. In severe cases, a significant part of the head or acetabulum may be destroyed (Figs. 23.15B and C).

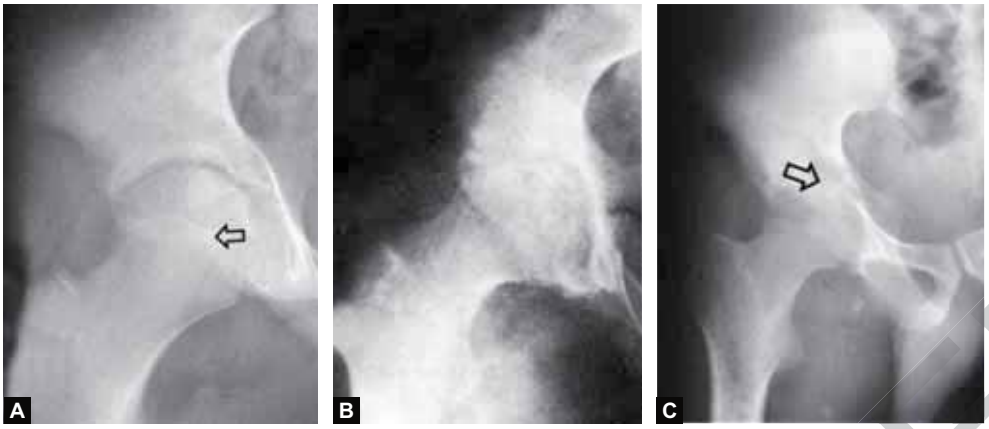


Fig. 23.15: Radiological features of TB of the hip.

- *Acetabular changes:* The head may be lying out of the acetabulum in a 'pseudo' acetabulum on the ilium - the *wandering acetabulum.* In some cases, the acetabulum simply gets enlarged and deepened with the deformed head shifted medially, giving the appearance of the *'pestle and mortar'.*
- *Signs of healing:* If the disease starts healing, there may be sclerosis around the hip.

Other investigations: The investigations that can be carried out to confirm the diagnosis are as discussed on page 171.

Biopsy: It may be needed in some doubtful cases. This is done by exposing the hip by the posterior approach and taking a piece of the synovium for histopathological examination. It is possible to do an arthroscopic biopsy.

DIFFERENTIAL DIAGNOSIS

TB of the hip is the *most common* cause of pain in the hip in children in countries where TB is still prevalent. Following differential diagnosis should be considered:

a. **Other causes of monoarthritis of the hip:** Subacute low grade monoarthritis due to low grade septic infection or rheumatoid arthritis also presents with pain and stiffness of the hip. Lack of supportive evidence for TB (like positive family history, past history) and destruction and sclerosis on X-ray, favour a diagnosis of septic arthritis. It may sometimes be difficult to differentiate the two. In rheumatoid arthritis, joint space is uniformly reduced.

b. **Inguinal lymphadenopathy or psoas abscess:** Patients with these extra-articular diseases often present with a flexion deformity of the hip because of spasm of the iliopsoas. An examination reveals that all movements of the hip *except extension* are pain free.

c. **Other diseases of the hip presenting at that age:** In a child presenting with a *limp without much pain* the following conditions should be considered:
   - *Congenital dislocation of the hip:* The limp is *painless.* It can generally be detected at birth, but is often noticed only when the child starts walking. An abnormal femoral head can be felt in the gluteal region. Telescopy test is positive. X-rays are decisive.
   - *Congenital coxa vara:* The limp is *painless.* The movements limited are abduction and internal rotation. In fact, adduction and external rotation may be increased. X-ray examination usually confirms the diagnosis.
   - *Perthes' disease:* This occurs in children in the age group of 5-10 years. The main complaint is a limp, which is generally painful. There is minimal limitation of movement, mainly that of abduction and internal rotation. Little or no shortening is present. Typically, X-ray changes are *out of proportion* to the physical findings. The joint space, unlike in TB of the hip, may even be *widened* (for details see page 309).

d. **Osteoarthritis:** This occurs in older individuals. Hip movements are limited in all directions but only terminally. There is associated pain and crepitus. Most cases are of osteoarthritis secondary to some other pathology (see Chapter 35).

TREATMENT

Principles of treatment: It is to control the disease activity, and to preserve joint movement. In early stages (Stages I and II), it is possible to achieve this by conservative treatment. In later stages (Stage II and after), significant limitation of joint functions occur despite best treatment. Treatment may be conservative or operative.

Essential
ORTHOPAEDICS

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He has had an illustrious career in the course of which, in addition to this book, he has published a number of other books on subjects related to orthopaedics. He has also published a number of papers in scientific journals. He was awarded the Commonwealth Fellowship, UK and the Johnson & Johnson Fellowship. He is an active member of orthopaedic associations at local and national levels. His expertise has been recognised worldwide, making him a sought after faculty at most national and international conferences on knee and shoulder surgery. He has a vast experience of teaching undergraduates, postgraduates and paramedical staff.

He is a stalwart in the field of knee and shoulder surgery, and is currently in private practice at New Delhi. He was earlier Head of the Department at Max Super Speciality Hospital, Saket, New Delhi. He is currently Director of Sports Injury Management Institute (SIMI) at Sitaram Bhartia Institute of Science and Research, New Delhi.

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Highlights

- All chapters have been thoroughly revised and updated. Contents have been tailored to the requirement of a medical student and those preparing for PG entrance examination
- The Annexure on 'Clinical Methods' makes it an 'all-in-one' book for medical students and residents
- The 'problem-solving approach' has been uniformly appreciated by students, teachers and practising doctors. This has been further improved
- The Annexure on 'Orthopaedic Terminology' helps to review-at-glance, the meaning of different terms used in orthopaedics
- A number of diagrams and X-rays have been replaced for better clarity and understanding.

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