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International Students Edition

3rd Edition

Practical Orthopedics

Biological Options and Simpler Techniques for Common Disorders

(With an Atlas of Rare Conditions)



SM Tuli

Foreword
Apurv Mehra

JAYPEE

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CHAPTER

2 Infections of Bones and Joints

INTRODUCTION

Infection of any bone caused by infective organisms is addressed as osteomyelitis. Commonly the infective organisms may be *Staphylococcus*, *Streptococcus*, typhoid and para-typhoid bacilli, pneumococci and *Haemophilus influenzae*. Granulomatous infections are a result of infection by *Mycobacterium tuberculosis* or syphilitic osteomyetitis. Mycetoma (fungal) infections may produce 'actinomycosis' or 'maduramycosis'. Rarely parasitic infestation may cause hydatid disease of bone or spine. Clinically the patient may present as acute osteomyelitis, subacute osteomyelitis, chronic osteomyelitis and an acute exacerbation of a pre-existing chronic osteomyelitis. Due to many factors there is a looming pandemic of "superbugs" of pyogenic, mycobacterial and viral organisms. We need affordable and widely available drugs for clinical use. The maximum threat for infection by superbugs is to immune compromised populations, critically ill patients, postoperative conditions, and geriatric population.

ACUTE HEMATOGENOUS OSTEOMYELITIS

This is a disease of childhood and adolescence generally occurring in under nourished children with poor defensive immune system. The commonest source of infection is due to hematogenous spread from a distant infected site such as ear, nose, mouth, respiratory tract, the bowel or genitourinary tract. The infecting organisms are carried to all parts of the skeleton, however, the bacteria get localized in the highly vascular and fast growing metaphyseal region of bones. A mild trauma near the bone ends may create a hematoma in the metaphyseal area which would act as a nidus for bacterial localization and infection. In the operating rooms and hospital environments, the most common source of infection is by nosocomial transmission. The commonest cause of osteomyelitis in the affluent societies is an open fracture or operative reduction with internal fixation of fractures.

Pathology

The pathological picture varies considerably depending upon the virulence of the organisms and host immunity. The initial focus of infection is usually the metaphyseal region of bone. The rich vascular pattern of this region favors the localization of infection. The initial focus of infection, if not treated, spreads outwards, penetrates the cortex and forms a subperiosteal abscess, which may burst to present as a subcutaneous abscess or pus discharging through sinuses. The pus can also spread subperiosteally or through medullary canal toward the diaphyseal area. In very young children, the infection may penetrate the physis, infect the epiphysis and spread to the joints. The joints get readily infected even if a part of metaphyseal area is intracapsular as in hip, elbow, and shoulder joints.

Exuberant periosteal reaction is seen between 2 to 7 years of age because the periosteum is very tough. Stripping of the

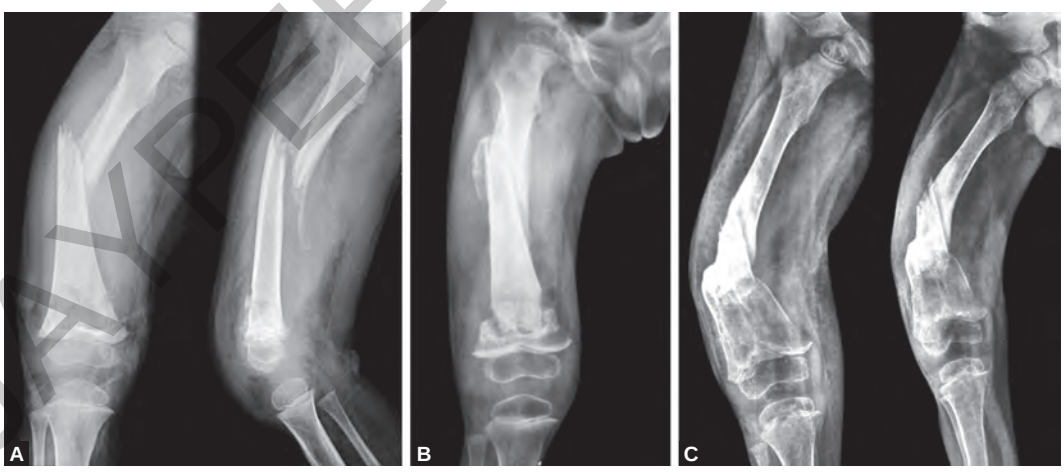
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periosteum, thrombosis of the vessels and increased intraosseous tension may result in ischemia of cortex of bone. Nonviable cortex becomes a "cortical sequestrum". When the infection is fulminant, varying extent of diaphysis may become necrosed and form a "diaphyseal sequestrum" and result in a pathological fracture.

The healing response of bone to infection (or any injury) is new bone formation under the raised periosteum. The new bone formed around an area of infected cavity would lead to the thickening of cortex; the new bone that forms around a sequestrum is called the involucrum. The involucrum generally has holes in it for discharge of pus and small sequestra, the holes or gaps are called 'cloacae'. Sequestrum is defined as a dead piece of bone surrounded by infected granulations and/or pus. The sequestrum gets separated from the parent bone by granulation tissue. Its surface is smooth, however, the margins and areas in contact with granulation tissue may have serrated edges and rough surfaces. In fulminating infections, sometimes, the periosteum may undergo necrosis, the involucrum is incomplete or deficient in such areas. Pus will continue to discharge through sinuses or ulcers, till spontaneous extrusion of sequestra or its surgical removal. In young children, once the infection is controlled, the 'sterile' sequestrum may undergo resorption and incorporation not unlike a bone graft **(Fig. 2.1A to C)**.

Clinical Presentation

The patient is usually a child or an adolescent presenting with history of fever, pain, toxemia, swelling and difficulty in the function of limb. Any bone can be involved, however, the commoner areas presenting with infection are upper end of tibia and lower end of femur. Examination would reveal a swollen, warm and tender area. The adjacent joints may also show swelling, which may be a 'sympathetic' effusion due to synovial irritation or actual involvement by infection. All the signs and symptoms may be subdued if the patient had already received antibiotics prior to your examination. Septic arthritis, rheumatic arthritis, bleeding disorder, and cellulitis are commoner conditions in differential diagnosis.



Figs. 2.1A to C: A neglected case of acute osteomyelitis of femur in a child. Distal half of femur has sequestrated, there is a pathological fracture in the distal metaphyseal region and in the middle of femur. The involucrum formation was incomplete, probably the periosteum was necrosed due to fulminating infection. Proximal part of uncovered sequestrum was exised, the distal part got incorporated (C). The follow-up revealed an excellent remodeling.

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Radiological Features

X-rays may not show any changes in the bone for 10–14 days except a soft tissue swelling. After about 2 weeks, X-rays may show patchy areas of destruction in the bone, elevation of periosteum with subperiosteal new bone formation **(Figs. 2.2A and B)**. In later stages, a cortical sequestrum or a diaphyseal sequestrum may be seen. In chronic stage, bone becomes more thick, there is more obvious patchy rarefaction (cavitations) with different shapes and sizes of sequestra. The living bone (metabolically active) will show osteoporosis in early stages because of reactive hyperemia which will ultimately show thickening due to formation and deposition of subperiosteal new bone. The dead bone (metabolically inactive) or sequestrum does not show any reaction except at the edges being separated from the living bone by granulation tissues. There is a natural biologic tendency to "wall off" foreign material or a sequestrum by encapsulating layers of inflammatory fibrous tissue.

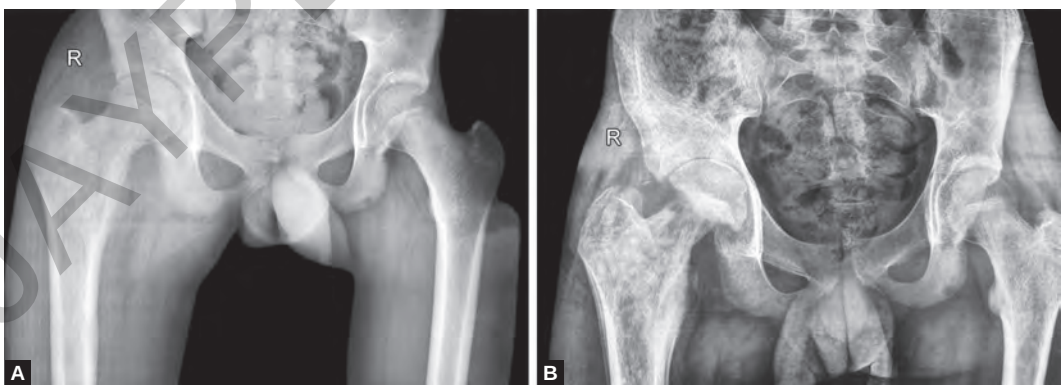
While examining X-rays in early stages of acute infection, soft tissue signs should not be forgotten. Edema of muscles is seen as soft tissue swelling, the fat planes in between the muscles get obliterated. Ultrasonography is a good investigation to show soft tissue changes. MRIs may help distinction between living and dead bone, and show the extent of soft tissue involvement.

Treatment

Without any delay, rest the limb with a suitable splint in the best functional position. The splint must permit periodic inspection of the infected area. Ideally before starting parenteral antibiotics, aspirate pus or tissue fluids from the depth of suspected area, and obtain a blood sample. Send the material for microbiological examination. Start with "best guess" antibiotics by intravenous route. Blood cultures are usually positive in 40–50%, and aspirate from the infected area positive in 50–60%.

Unfortunately, a large number of infective organisms are resistant to first line antibiotics like penicillin and many other commonly used antibiotics. As a rule, more than one antibiotic should be started, ideally by intravenous route as soon as the samples have been obtained for microbiological investigations and major bleeding disorders excluded. Parenteral antibiotics therapy for 2–4 weeks should be followed by oral antibiotics for another 4–6 weeks.

Patients with acute osteomyelitis or acute septic arthritis must be hospitalized and kept under close observation while on intravenous antibiotics. If rapid subsidence of local and



Figs. 2.2A and B: (A) Early radiological features of osteomyelitis of right femur. The comparison with contralateral femur shows irregular patchy areas of osteoporosis, faint periosteal reaction and soft tissue swelling. The limb at this stage must be splinted to prevent a pathological fracture; (B) About 1 month after the onset, more changes are visible with subperiosteal new bone formation.

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systemic signs of infection and inflammation does not occur, operative intervention is needed.

Operative Intervention for Acute Osteomyelitis

Make a standard incision at the site of maximum involvement of bone, up to the periosteum, evacuate all abscess and infected soft tissues. There is generally a debate regarding making multiple drill holes at the site of osteomyelitis. If no pus wells out, making drill holes is mandatory. It is wise to make holes in the longitudinal axis of bone. There are more chances of a pathological fracture if the holes are made right angle to the long-axis. Having exposed the infected area, obtain a few pieces of bone, infected soft tissues for histological examination. Infected granulation tissues and pus must be sent for microbiological investigations. Close the wound loosely over a drain. Ideally, two drains should be put in, one in the medullary canal and one in the soft tissues around the bones. Avoid negative suction on the intramedullary drain lest it may suck out too much of blood from the medullary vessels. If lymph nodes are available, send those also for histological studies. Splint the limb for about 6–8 weeks, you may permit nonweight-bearing ambulation (for lower limb infections) after 4–6 weeks of operation and initiation of antibiotic therapy. There is a real danger of a pathological fracture of the bone having acute osteomyelitis, because initial X-rays up to 2–4 weeks may practically "look normal".

FUNCTIONAL POSITION OF JOINTS

When a joint requires to be given rest for acute inflammation or a chronic condition, a plaster splint or an orthotic appliance should hold the joint in the best functional position as outlined below.

Hip: About 10–30 degrees of flexion almost related to age, neutral between adduction and abduction and 5 degrees of external rotation.

Knee: About 5–10 degrees of flexion to help foot to clear the ground while walking, and 5 degrees external rotation. Some flexion at knee joint helps sitting on chair.

Ankle: At right angle (plantigrade) and in 5 degrees external rotation.

Wrist: about 10 degrees of dorsiflexion to allow a firm grip.

Elbow: About 90 degrees of flexion and in mid-pronation.

Shoulder: "Saluting position"—about 60 degrees abduction, 40 degrees flexion and 40 degrees internal rotation.

SEPTIC ARTHRITIS (FIG. 2.3)

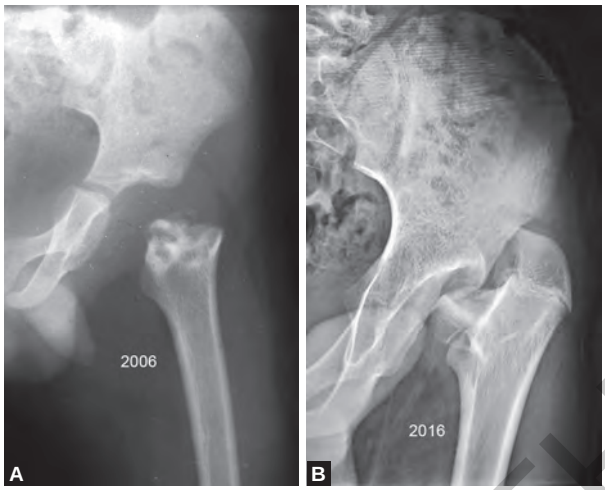
Septic arthritis is most common in children with 1–5 years of age. Typically the patient presents with pain, fever and inability to bear weight (lower extremity joints) or inability to use the limb (pseudoparalysis). In young children, the distinction between transient synovitis of hip and septic arthritis of hip can be difficult, however, four easily obtainable predictors like fever, inability to bear weight, erythrocyte sedimentation rate (ESR) 40 mm or greater, and serum white blood cell count (WBC) 12,000/mm^3 or greater can be used to establish the possibility of septic arthritis.

In neonates, it is difficult to clinically distinguish between septic arthritis of hip and septic arthritis associated with osteomyelitis of upper end of femur (upper end of femur is intracapsular). Similar difficulty would arise in other joints like shoulder and elbow. Antibiotic policy is same as for acute osteomyelitis.

Surgical Management

Arthrotomy of the infected hip and shoulder is mandatory for adequate decompression. Superficial joints like knee, ankle and elbow can be repeatedly aspirated with instillation of antibiotics, if desired. Aspiration should be done using 16G or 18G needles. The aspirate may be serosanguinous (earliest), cloudy or turbid, or frankly purulent (later).

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Figs. 2.3A to B: Functional treatment for articular infections can heal the disease with retention of functional range of motion. X-rays at presentation (A), regeniration on healing of disease (B) and follow-up.

If aspiration and instillation with antibiotics does not control the infection an arthrotomy is mandatory.

Arthrotomy should be done as early as possible, open the joint through a standard incision in the capsule, collect the infected material from the joint for microbiological and histological investigations. Irrigate joint with normal saline. Only close the skin after inserting a suction drain in the depth of wound. Either apply a traction for postoperation observation and management, or apply a plaster cast in the best functional position. The suction drain is removed after 2–4 days. If hip is dislocated, it is reduced and immobilized in hip spica, one may use 1 or 2 stout Kirschner's wire(s) to hold it in good position. Same principle is applicable for acute septic arthritis of any joint in children.

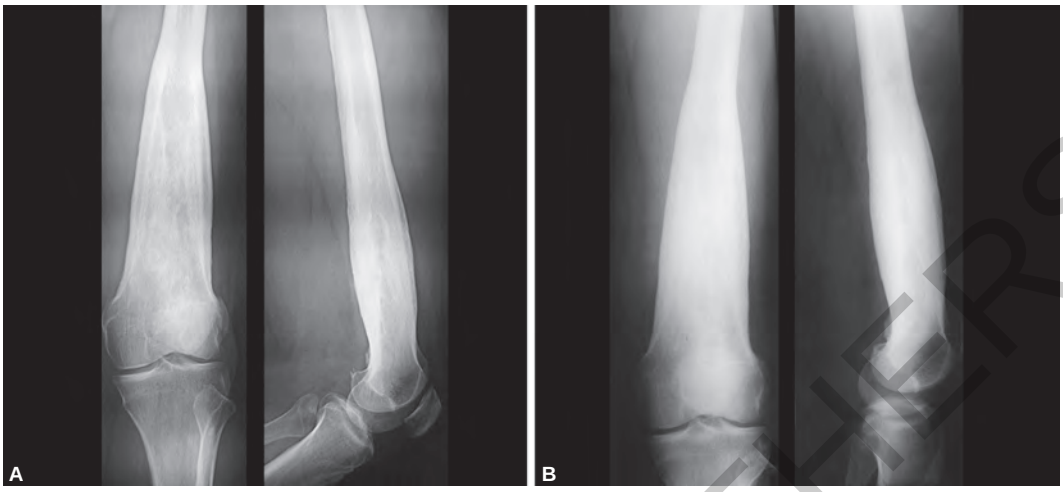
CHRONIC OSTEOMYELITIS

If treated well in time and adequately, the acute infection of bone would heal; swelling, pain, tenderness and toxemia would resolve or disappear and laboratory investigations would be normal. The infected bone may, however, show thickening in the X-rays for a long time. Depending upon the age of patient, the thickened area with growth would move away from the metaphyseal area and undergo gradual remodeling to near normal shape. To avoid reactivation of infection, one must maintain good nutritional status.

Some of the patients of acute osteomyelitis develop chronic osteomyelitis. The main factors are poor nutritional status, delayed or inadequate drainage of pus or infected cavities, ineffective or inadequate use of antibiotics, presence of sequestra, or foreign bodies (metallic or nonmetallic) in cases of infection following open injuries or operations. In addition to pyogenic infection, chronic osteomyelitis may also be caused by tuberculosis, fungal or syphilitic microbes.

The patient presents with one or more sinuses over the involved bone. There may be multiple scars due to old infection or operative incisions. The sinuses or scars may be adherent to the underlying bone, pouting/protruding granulation tissue may be seen through the sinuses. The involved bone is thick and tender, there would be a history of periodic acute "flare-ups" of pain, swelling and fever. The "flare-up" is generally associated with closure of sinuses, collection of pus in the tissues or a history of trauma. The flare-up symptoms would subside when the sinuses burst open, discharge pus and/

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Figs. 2.4A and B: Healed status of chronic osteomyelitis of distal femur. (A) Note thickening of bone, small patchy cavitations within the bone and periosteal reaction; (B) X-rays of the same patient after a gap of 2 years, clinically there has been no evidence of active infection, however, the bone thickening persists.

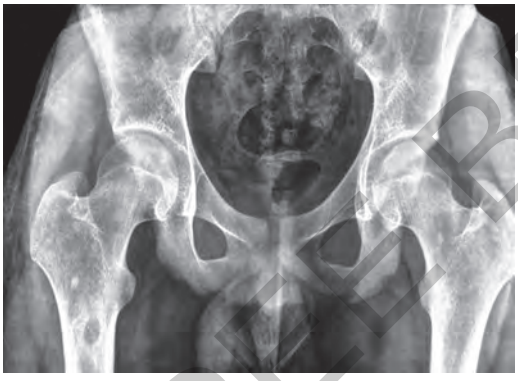


Fig. 2.5: A typical Brodie's abscess in right upper femur.

or small sequestra. X-rays would reveal the thickened bone with infected cavities, with or without sequestra, and involucrum **(Figs. 2.2 to 2.5)**. Involucrum formation suggests the presence of a viable periosteal sleeve around the sequestrum.

Sequestrum and Involucrum

Sequestrum is a dead piece of bone surrounded by infected granulations or frank pus. A bone may become necrosed or dead due to devascularization as a result of hematogenous osteomyelitis or due to extensive loss or stripping of soft tissues and periosteum because of trauma or rough iatrogenic handling.

A free bone graft, say harvested from iliac bone transplanted around nonunion of tibia, is practically a dead bone. Carefully performed (aseptic technique and gentle tissue handling) procedure would permit healthy granulations to surround the grafted bone ultimately leading to successful incorporation. If, however, there is infection and the free bone graft gets surrounded by infected granulations or frank pus, it is now a sequestrum.

Fate of Sequestrum

Sequestrum initiates a reaction in the surrounding bone and periosteum. Subperiosteal bone formation around the sequestrum forms an involucrum as a reparative process. In young children, sometimes, under the influence of effective antibiotics and supportive therapy the infected granulations surrounding a sequestrum become sterile, the healthy granulations would then invade the dead bone resulting in resorption of the sequestrum or reincorporation as a grafted

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bone. If sequestrum persists despite supportive therapy and antibiotics, the involucrum would grow around the sequestrum.

Behavior of Involucrum

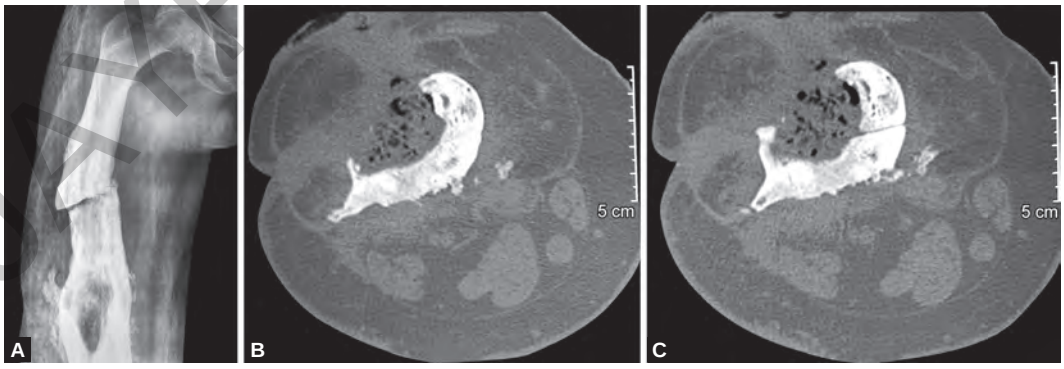
Formation of involucrum is the activity of the surrounding healthy periosteum. If periosteum is necrosed, the involucrum will not form. If the exposed bone is dead and not covered by periosteum, again involucrum will not form. The activity of the formation of involucrum can be judged by X-rays of the limb at 4–6 weeks intervals. If its outer surface is rough, the size of involucrum is likely to increase. If the outer surface of involucrum has become smooth, further increase of its size in unlikely.

Sequestrectomy

It is wise to wait till the formation of sufficient involucrum. Sequestrum should be carefully removed without jeopardizing the stability of the infected bone. The window in the involucrum should not be >1/3 of the diameter of the bone. Overzealous removal of involucrum or premature operation may lead to an iatrogenic fracture of bone. In case of loss of integrity of the parent bone, the infected cavity should be filled up with copious fresh autogenous bone grafts and the limb should be protected in a suitable plaster cast. Mechanical protection may be achieved by inserting an intramedullary device without reaming, or the use of an external fixation. Success is achieved by adequate debridement, however removal of dead bone through the cortical "window" must be compatible with mechanical stability of the parent bone **(Figs. 2.6A to C)**.

Papineau's Procedure, Open Bone Grafting

If the cavity left behind after sequestrectomy is large or you are dealing with infected nonunion or symptomatic infected large cavities in bone, Papineau's principles are a rational option. Remove grossly infected tissues, leave behind bleeding recipient surfaces. Harvest adequate amount of autologous cancellous bone grafts, fill up the cavity with copious bone grafts, or in case of infected nonunion place the slivers of bone around the site of nonunion. Cover the operated area by loosely stitching the soft tissues over it. If one cannot achieve a satisfactory soft tissue cover, which may be the situation due to scarring of long standing in chronic osteomyelitis or infected nonunions, cover the cancellous bone grafts with sterile vaseline gauze. The dressing would require a change 2 to 3 times a week. Mechanical stability in cases of infected fractures may be provided by the use of an external fixation or by intramedullary elastic rods or plaster cast.



Figs. 2.6A to C: A window to perform sequestrectomy must be made carefully. Overzealous window has created an iatrogenic pathological fracture of femur through the infected bone, an extremely difficult problem to treat.

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Papineau's technique can also be used for infected area through a window in plaster cast. While changing the vaseline gauze dressing, some superficial pieces of cancellous graft may become discolored (grayish) and fall off as loose pieces. Within 3–6 weeks, most of the graft gets covered with healthy granulations, which may epithelize on its own or may be covered by split skin graft. We have successfully used this technique for large infected cavities and infected nonunions with inadequacy of soft tissue covering.

Alternative methods: Alternative methods that are available entail plastic surgery procedures to replace the scarred tissue prior to bone grafting. This would increase the number of days in the hospital and the resultant financial burden.

Some workers have tried evacuation of the infected cavities followed by continuous antibiotic instillation and suction (ingress–egress) or use of antibiotic–impregnated cement beads with the aim of continuation of high concentration of local antibiotics. Simpler and cost-effective treatments, however, are preferred because many studies have shown adequate concentrations of antibiotics at the site of chronic osteomyelitis even when the patient was only on systemic parenteral or oral antibiotics.

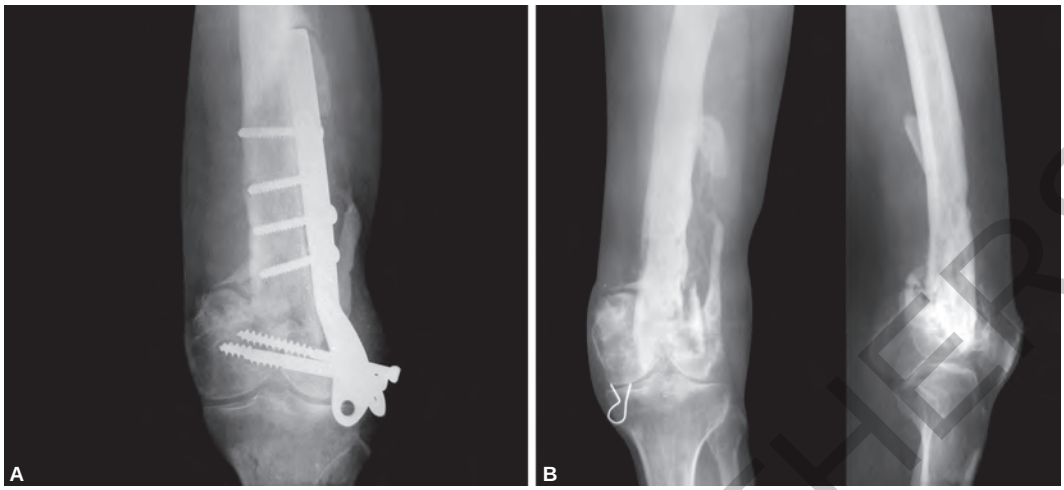
'Curing' chronic osteomyelitis is an illusion; at best one can achieve a lasting healed status. Some cases of chronic osteomyelitis may show no activity of infection despite the persistence of radiological evidence of infection. Such cases may be called **"dry chronic osteomyelitis"**, other cases keep on showing occasional activity of infection (local pain, swelling, collection of pus and spontaneous discharge) and may be called **"wet chronic osteomyelitis"**. Poor nutritional status, diabetes, prolonged use of steroids and immunosuppressive drugs and local trauma increase the chances of recrudescence of infections. Persistance of sequestra (necrotic bone), infected intraosseous cavities, cicatrix (scarring) formation in skin and soft tissues, poor body resistance (immunity), and diabetic state are generally responsible for persistance of chronic osteomyelitis. An unhealing ulcer associated with chronic osteomyelitis may rarely undergo malignant change after 15–20 years. The author encountered five such patients during 50 years of orthopedic outpatient services, one related to infection in humerus, three associated with infection of tibia, and one related to chronic infection after cementation of a cystic lesion in proximal tibia.

IATROGENIC INFECTIONS IN ORTHOPEDICS

One of the most dreaded complications of operative procedures in orthopedics is operation-induced (iatrogenic) infection **(Figs. 2.7 and 2.8)**. The commonest organisms related to such infections are aerobic or anaerobic pyogenic bacteria, however, in countries where tuberculosis is almost endemic, tuberculous infection after implant surgery or operative intervention must also be considered. The author has observed tuberculous infection after joint replacements around knee, hip and shoulder joints. Tuberculous infection was also seen around the implants used for open-reduction and internal fixation in general, and after arthroscopic exploration of the joints. The ubiquitous mycobacteria are capable of implantation or impregnation at the site of operative trauma, especially in patients with immune compromise.

Any practicing orthopedic surgeon should be cognizant to this type of infection. Strict aseptic precautions are mandatory for any physician and healthcare workers. Proper handwashing is one of the most important means of controlling nosocomial transmission during any operative intervention or an invasive procedure. The most effective method of sterilization of operative instruments, is still considered to be high pressure autoclaving. The endoscopic equipments, are not autoclaved and most

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Figs. 2.7A and B: Osteomyelitis of the femur as a sequel of fixation of femoral fracture. Distal half of the femoral shaft is sequestrated. Attempt at formation of involucrum is incomplete probably because the periosteum had undergone necrosis.



Fig. 2.8: An example of infected nonunion: Note a huge soft tissue swelling of whole of thigh, site of fracture fixed with a gap, an extra long plate probably resulted in extensive soft tissue stripping disturbing the osseous blood supply, a few sequestra are present around the site of nonunion.

of them are sterilized by Glutaraldehyde 2.45% formalin chamber, ethylene oxide vapors, or gas sterilization. It is wise to be reminded that postoperative discitis after open operation on the spine is 1%, whereas the incidence after endoscopic procedures is about 3%. Cannulated instruments have been implicated as potential source of contamination.

Diagnosis

A high suspicion index plays a crucial role in diagnosis at an early stage of infection. Clinical examination of the operated area must observe the degree of inflammation, the margins of operative incision; look for undermined edges of ulcers and sinuses, pouting granulations through sinuses and nature of discharge from the wounds. Every effort must be made to obtain material for microbiological investigations. Whenever debridement is done, the tissue must be sent for histological examination for pyogenic, tuberculous and fungal pathologies. Despite X-rays and modern imaging modalities, microbiological and histological investigations, the clinician may be confronted with final diagnosis as "chronic/subacute/acute inflammation/infection". Assessment by repeated clinical or interval assessment is supreme to help make therapeutic choices.

Patients at risk of iatrogenic infections are immunocompromised due to conditions like hemoglobinopathy, diabetes, liver disease,

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alcohol abuse, substance abuse, rheumatoid disorder, immune-suppressive and anti-mitotic drugs. Local tissue damage caused by operative intervention or injury provides a nidus for seeding for the microbacteria.

Therapeutic Options

Remove all stitches of the operative incision preferably up to the deepest layer. Do adequate debridement of infected area and ensure drainage or postural dependent drainage, possible active exercises of the limb would help pump out the collection from deeper parts.

Having sent the material for micro-biological and histological (when possible) studies, start a combination of at least 2 drugs of 'best guess' of broad spectrum antibiotics by parenteral route for about 2 weeks. The antibiotics may then be modified according to the available microbiological (and histological) reports; and appropriate oral antibiotic therapy must be continued for 6–8 weeks. Supportive therapy and improvement of nutritional status is equally important.

If the infection is coming under control, the implant may not be removed, however, if the infection is not coming under control or it is a case of fulminating infection, it is wise to remove the implant, and splint or rest the joint or limb in the best functional position. If one is impelled to try a second implant replacement, it may be wise to use an inert "spacer" during the waiting period till the infection is controlled and the tissues are ready for the next surgery.

TUBERCULOSIS OF BONES AND JOINTS

General Clinical Picture

World-wise nearly one million lives are lost each year because of tuberculous infection.

Skeletal tuberculosis mostly occurs during first three decades of life; no age, however, is immune. With increasing longevity, the disease is being reported now in the elderly as well. The characteristics are insidious onset, monoarticular or mono-osseous involvement and the constitutional symptoms like low-grade fever and lassitude (especially in the afternoon), anorexia, loss of weight, "night cries", painful limitation of movements, muscle wasting, and regional lymph nodes enlargement. During active stage, the protective muscle spasm is severe holding the diseased area immobilized. During sleep, the muscle spasm relaxes and permits movement between the inflamed surfaces resulting in pain causing the typical night cries (especially in children). Commonly involved joints in descending order are vertebral column, hip, knee, ankle-foot, wrist-hand, elbow and shoulder.

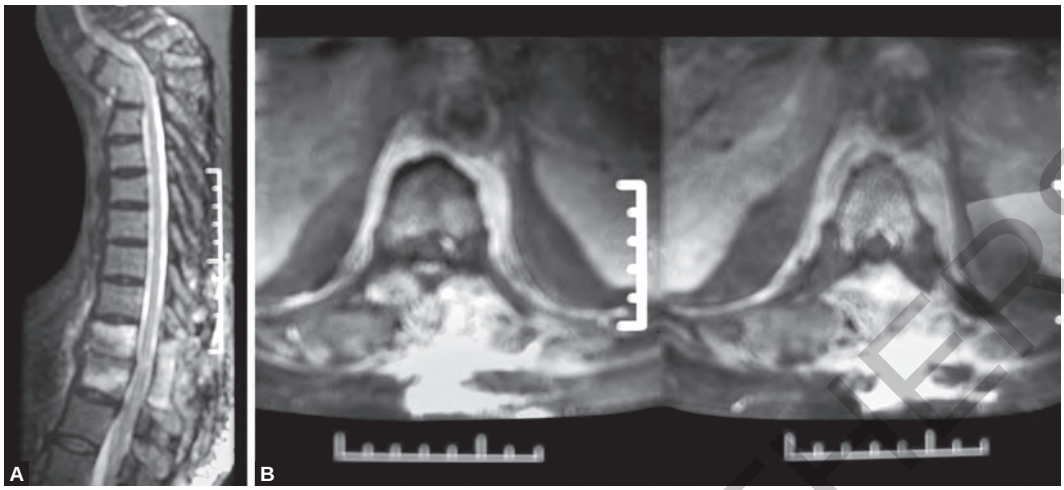
Diagnosis

In resources constrained countries, in general, diagnosis of tuberculosis of bones and joints can be reliably made on clinical and radiological examination. However, in early stages and whenever in doubt a positive proof of the disease must be obtained employing semi-invasive or invasive investigations. In the affluent societies, corticosteroids, alcoholism, prolonged illness, diabetic state, immunosuppressive drugs, and old age are the probable predisposing factors. Skeletal tuberculosis must be included in differential diagnosis of chronic/subacute mono-articular arthritis, chronic abscess, draining sinus, chronic swelling or osteomyelitis; tuberculous infection is a great mimic even today **(Figs. 2.9 to 2.11)**.

Roentgenograms and Images

Anteroposterior and lateral views of the part, and an X-ray of the chest are required. Localized osteoporosis is the first radiological sign of active disease. The articular margins and bony cortices become hazy, (giving a "washed out" appearance), there may be development of areas of trabecular or bony destruction and osteolysis. The synovial fluid, thickened synovium, capsule and pericapsular tissues may cause a soft tissue

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Figs. 2.9A and B: (A) In skeletal tuberculosis, one can at best achieve a lasting healed status. This case shows a healed tuberculosis at cervico-dorsal lesion in childhood. Patient now reported with a fresh active infection in lower dorsal spine after 2 years of the first lesion; (B) MRI-T2 shadows show bright signals (pus collection) in the vertebral bodies, perivertebral region and posterior elements of the lower dorsal spine.



Fig. 2.10: Tuberculous infection is a great mimic. Multiple lytic lesions in the humerus in this lady are due to tuberculosis. The soft tissue swelling around the lesion was a cold abscess, patient had concomitant active tuberculous lesions in the lungs and vertebral column.

swelling. With the involvement of articular cartilage, the joint space (articular cartilage space) shows diminution in the X-rays. As the destructive process advances, there may be collapse of bone, subluxation/dislocation, migration and deformity of the joints. In a typical paradiscal spinal tuberculosis earliest radiological signs are diminution of the disc space with erosion of paradiscal margins of vertebrae. MRIs would show bright signal in T2 and dark singal in T1 images in the paradiscal region, during the active phase of infection.

MRIs now available almost in all countries, it is the most useful investigation for disorders of the skeletal system, it does not expose the patient to radiations.

Mnemonic may help remember MRI images:

- T2 water white: T1 water black (dark); T1 and T2 fat white; T1 and T2, ligaments capsule, desiccated disc or scars are black (dark) "Water" = inflammatory fluid.

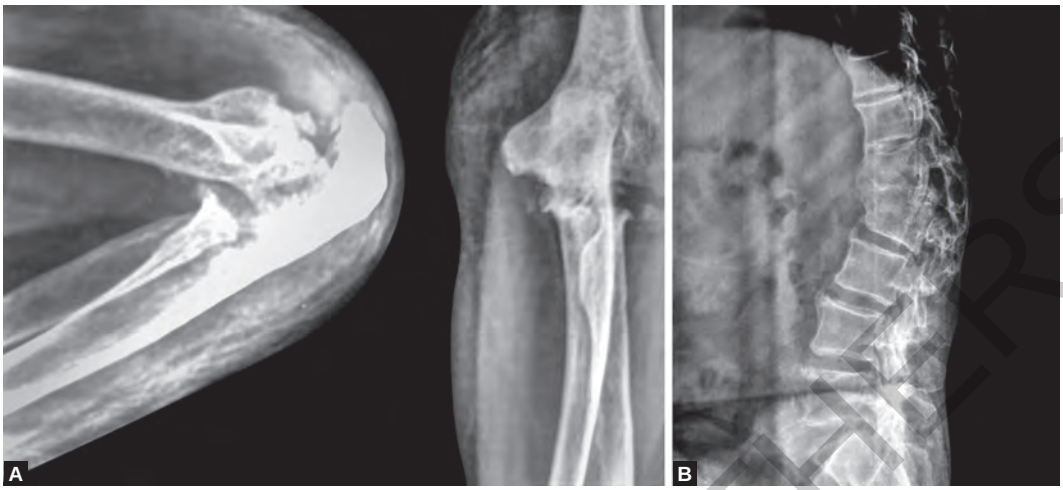
Blood

A relative lymphocytosis, low hemoglobin, and raised ESR are often found in the active stage of disease. ESR, however, is not necessarily a proof of activity of the infection.

Biopsy

Whenever there is doubt (particularly in early stages), it is mandatory to prove the

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Figs. 2.11A and B: (A) X-rays of advanced tuberculous arthritis of elbow in a 66 years male. Patient was treated by antitubercular drugs, and range of motion exercises (functional treatment). On healing, he could retain flexion-extension arc from 5 to 140 degrees; (B) X-ray of spine of the same patient: He was treated for spinal tuberculosis 15 years ago. The disease remained healed for many years. Patient reported for recurrence of infection in the elbow after developing diabetes.

diagnoses by obtaining the diseased tissue (granulation and/or synovium and/or bone and/or lymph nodes). At the time of open biopsy of a joint or an osseous lesion, a wise orthopedic surgeon would perform synovectomy or curettage as a part of therapeutic measures. The diseased tissue obtained should be submitted for histology, microbiology and serological tests.

Modern Imaging Techniques

MRI would confirm whatever one can see in the plain X-rays and CT scans. However, it also shows the predestructive lesions like edema or inflammation of the bone in active disease, which is more extensive than the areas of radiological destruction in the bones. Once the radiological changes have been established, no great advantage is gained by radioisotope scanning or MRI. Earliest radiological changes are discernable in the standard X-rays 2 to 4 months after the onset of disease whereas MRIs would reveal the changes between 3 and 6 weeks, radioisotope scanning can demonstrate a metabolically active lesion around 5–7 days. Generalized screening of any person presenting with active osteo-articular tuberculosis would reveal another subclinical active lesion in about 40% of patients.

Antitubercular Chemotherapy

The mainstay of treatment is multidrug antitubercular chemotherapy. No local operation can eradicate the disease as most of the patients have many other foci of tuberculous infection. Even when the disease has clinically healed, many tubercle bacilli survive for many years in the lymph nodes and the healed affected tissues. There are several recommended regimens of chemotherapy. At present, we use the following **(Fig. 2.12)**:

Intensive phase—consists of isoniazid 300–400, rifampicin 450–600 mg and fluoroquinolones 400–600 mg daily for 5–6 months. All replicating sensitive bacteria are likely to be killed by this bactericidal combination.

Continuation phase—is aimed to eliminate the slow-growing persisters, dormant or intracellular mycobacteria. This consists of isoniazid and pyrazinamide 1500 mg daily for 4–5 months followed by isoniazid and rifampicin for another 4–5 months.

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Practical Orthopedics

Biological Options and Simpler Techniques for Common Disorders

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SM Tuli graduated in 1955 and postgraduated in 1958 from Government Medical College, Amritsar, Punjab, India. He obtained PhD (1966, under Professor PK Duraiswami) from University of Delhi. He was awarded the fellowship of Academy of Medical Sciences (India) and International Fellowship of National Institute of Health, US, 1972. He founded the Department of Orthopedics at Banaras Hindu University (BHU) and at University College of Medical Sciences (UCMS), Delhi. Based upon the laboratory work of Marshall Urist (US), Dr Tuli established clinically applicable Bone Banks at BHU, UCMS and at Vidyasagar Institute of Mental Health and Neurosciences (VIMHANS). Presently he is working at Moolchand Hospital, New Delhi, India.

His major contributions have been the evolution of 'middle-path-regime' for the treatment of osteoarticular and spinal tuberculosis, use of 'allogenic bone grafts' for resconstruction of bone defects, development of an extra-articular operation—glenoplasty (for shoulder dislocation) and immune-potentiation technique for Multidrug-resistant Tuberculosis (MDR-TB). He worked as an Editor of the *Indian Journal of Orthopedics* with Varanasi team 1972–1987, and he also served as the President of Indian Orthopedic Association in 1984.

He has nearly 200 scientific publications, and contribution of chapters to *Essential Surgery* (WHO), *Children's Orthopedics* (Churchill Livingstone), *APLEY's Orthopedics* (Hodder-Arnold) and *Neglected Musculoskeletal Injuries* [Jaypee Brothers Medical Publishers (P) Ltd]. He is author of the book *Tuberculosis of the Skeletal System* (running VIth Edition), *Guidelines for Ageing with Health, Grace and Dignity (running II edition)*. He has been a surgeon to the common man, VIPs, medical doctors, and most prestigiously, to many orthopedic surgeons. After six decades of professional journey, he has not lost his passion for caring of orthopedic patients and for furthering orthopedic education.

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