

Bone & Joint Infections

Nasef Mohamed Nasef

Professor of Orthopaedic surgery

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- **Definition.**
 - **Prevalence.**
 - **Aetiology.**
 - **Risk factors.**
 - **Classification.**
 - **Pathology.**
 - **Clinical features.**
 - **Diagnosis & imaging.**
 - **Natural history.**
 - **Complications**
 - **Differential diagnosis.**
 - **Management.**

Definition

- Infection is a condition in which pathogenic organisms multiply and spread within the body tissues which gives rise to an inflammatory reaction (acute or chronic).
- Inflammation is the body's way of combating the invaders and destroying them ,or at least immobilizing them and confining them to a single area..



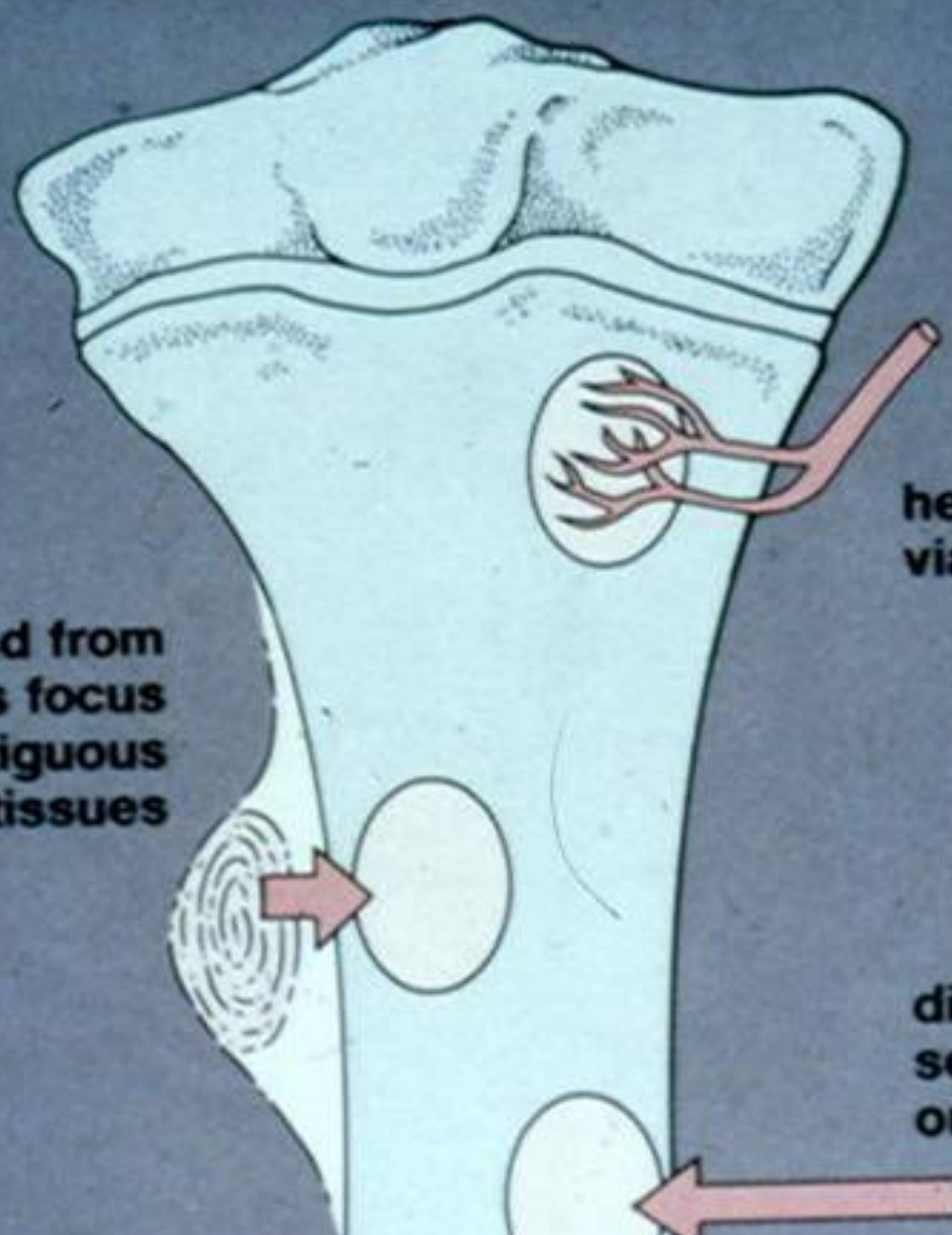
- **Route:**

Micro-organisms may reach the bones and joints either:

- *-directly* through a break in the skin

(a wound, an open fracture or an operation) or

- *-indirectly via the blood stream* from a distant site such as the nose or mouth, the respiratory tract or the genito-urinary tract.



The diagram shows a cross-section of a long bone with three distinct infection routes. At the top, a network of red nutrient arteries is shown entering the bone. In the middle, a red arrow points from a circular area of concentric lines (representing an infectious focus in soft tissue) into the bone. At the bottom, a red arrow points from the outside of the bone into a circular area within the shaft.

**spread from
infectious focus
in contiguous
soft tissues**


**hematogenous route
via nutrient arteries**

**direct implantation
secondary to trauma
or surgery**

- **Factors affecting the outcome** of Micro-organism reaching the bones and joints:
- -***Organism virulence*** and type
- -***The host response*** is crucial in determining the course of the disease. Resistance is likely to be depressed in the very young and the very old, in states of malnutrition or immune-suppression, and in certain diseases such as diabetes.
- -***Local factors*** also are important as damaged muscle and the presence of foreign material create a favorable environment for organisms

- **Outcome** of Micro-organism reaching the bones and joints:
- -A minor inflammatory reaction that *resolves* without significant pus or granuloma formation
- - *a pyogenic osteomyelitis or arthritis* (pus formation),
- -a chronic *granulomatous reaction* (as in tuberculosis), or an indolent response to an *unusual organism* (e.g. a fungal infection)..

- **Acute pyogenic infections** are characterized by the formation of **pus** - *a concentrate of dead and dying bacteria, leucocytes and tissue debris* - which is often localized in an abscess. Pressure builds up within the abscess and infection may then extend:
 - - directly along the tissue planes
 - -via lymphatics (causing lymphangitis and lymphadenopathy) or
 - -via the bloodstream (bacteraemia and septicaemia).

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- The accompanying systemic reaction varies from a vague feeling of lassitude with mild fever to severe illness, toxaemia and shock.
 - The generalized effects are due to the release of bacterial enzymes and endotoxins as well as cellular breakdown products from the host tissues.

- Bone, which consists of *a collection of rigid compartments*, is more susceptible than soft tissues to vascular damage and cell death from the build-up of pressure in acute inflammation;
- Unless it is rapidly controlled, bone infection will inevitably lead to *necrosis*.
- Bone structure - a honeycomb of inaccessible spaces - also makes it very difficult to eradicate infection once it is established.

- **Chronic infection** may follow on acute or may be chronic from the start.
- Some organisms provoke a non-pyogenic reaction involving the formation of cellular granulomas which consist largely of lymphocytes, modified macrophages and multinucleated giant cells as well as fibroblastic and vascular proliferation; this is seen most typically in *tuberculosis*.
- Systemic effects are less acute but may ultimately be very debilitating, with lymphadenopathy, splenomegaly and tissue wasting.



- **-Patient:**

- Acute osteomyelitis is mainly a disease of children.
- When adults are affected it may be because their resistance is lowered by debility, disease (as diabetes) or drugs (as immunosuppressive drugs).
- -Trauma may determine the site of infection, possibly by causing a small haematoma or fluid collection in a bone.

- -**The causative organism**
- is usually *Staphylococcus aureus*, less often one of the other Gram-positive cocci, such as *Streptococcus pyogenes* or *S. pneumoniae*.
- In children under 4 years of age the Gram-negative *Haemophilus influenzae* is a fairly common pathogen.
- Other Gram-negative organisms (e.g. *Escherichia coli*, *Pseudomonas aeruginosa*, and the anaerobic *Bacteroides fragilis*) occasionally cause acute bone infection.

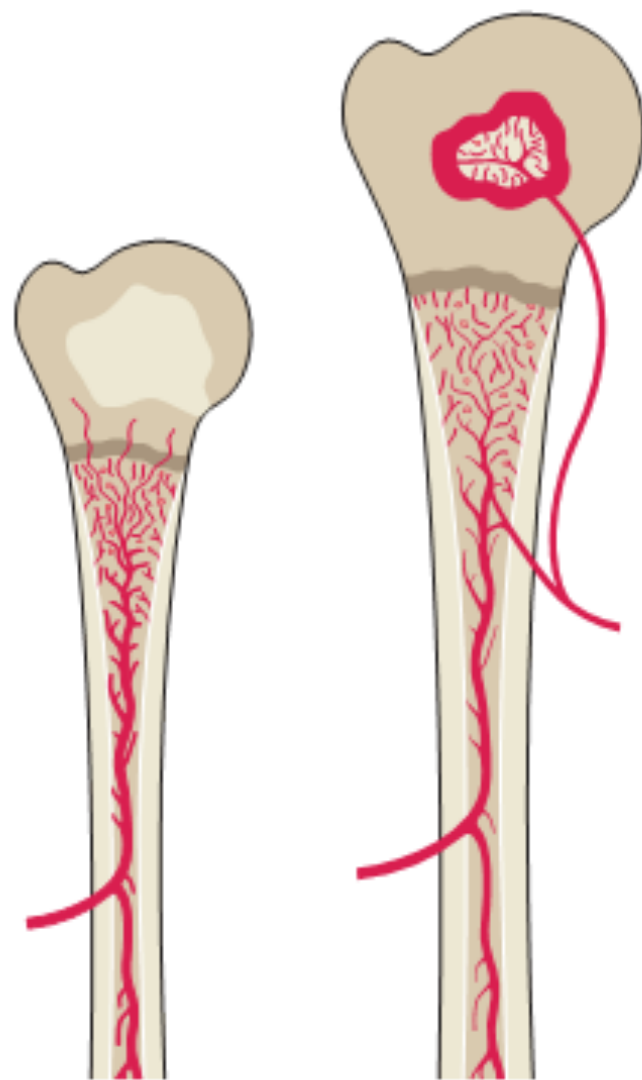


- **-Source:**

- The blood stream is invaded, perhaps from a minor skin abrasion, a boil, a septic tooth or
- - in the newborn - from an infected umbilical cord.
- In adults the source of infection may be a urethral catheter, an indwelling arterial line or a dirty needle and syringe.

- **-Site:**

- Organisms usually settle in the *metaphysis*, most often in the proximal tibia or in the distal and proximal ends of the femur.
- This may be due to the non-anastomosing terminal branches of the nutrient artery cause relative vascular stasis which favours bacterial colonization.
- **In young infants**, It is more common in the **epiphysis** in whom there is still a free anastomosis between metaphyseal and epiphyseal blood vessels.
- In adults, haematogenous infection is more common in the vertebrae than in the long bones



(a)

(b)

2.1 Epiphyseal and metaphyseal blood supply (a) In new-born infants some metaphyseal arterioles from the nutrient artery penetrate the physis and may carry infection directly from the metaphysis to the epiphysis. (b) In older children the physis acts as a barrier and the developing epiphysis receives a separate blood supply from the epiphyseal and peri-articular blood vessels.

- **Pathology:**
- The pathological picture shows a characteristic pattern marked by:
 - *inflammation,*
 - *suppuration,*
 - *necrosis,*
 - *reactive new bone formation* and, ultimately,
 - *resolution and healing.*

• INFLAMMATION:

- The earliest change is an *acute inflammatory reaction* with vascular congestion, exudation of fluid and infiltration by polymorphonuclear leucocytes PNLs.
- **The intraosseous pressure rises rapidly**, causing intense pain, obstruction to blood flow and intravascular thrombosis and impending ischaemia.



- **SUPPURATION:**

- By the second or third day, *pus forms* within the bone and forces its way along the Volkmann canals to the surface where it produces a subperiosteal abscess.
- From there the pus spreads into the surrounding soft tissues, bone and may be joints.

- *In infants, infection often extends through the physis into the epiphysis and thence into the joint.*
- *In older children the physis is a barrier to direct spread but where the metaphysis is partly intracapsular (e.g. at the hip, shoulder or elbow) pus may discharge through the periosteum into the joint.*
- *In adults the abscess is more likely to spread within the medullary cavity. Vertebral infection may spread through the end-plate and the intervertebral disc into the adjacent vertebral body.*

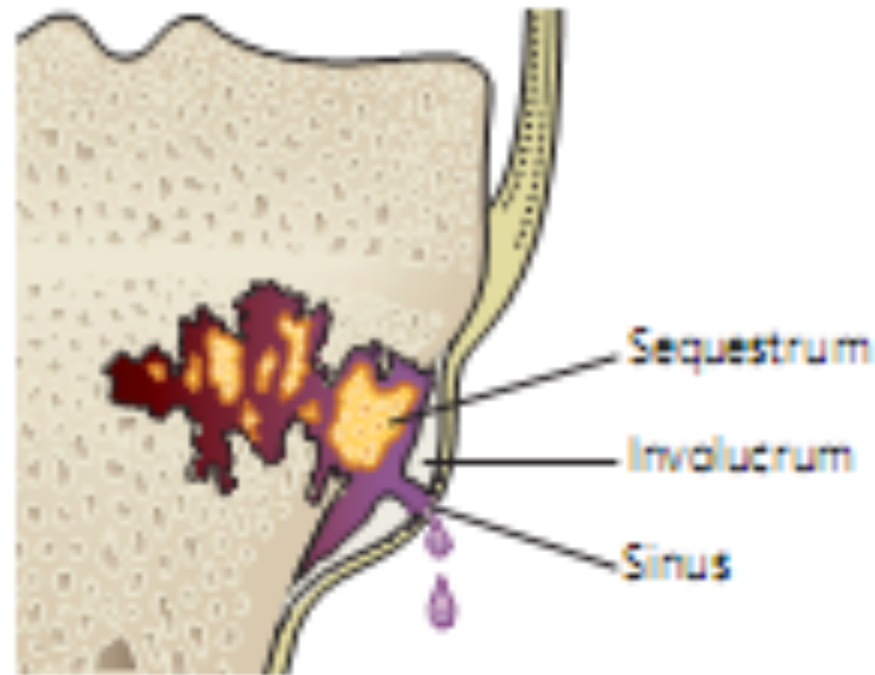
- Once suppuration occurs *outcome depends on the adequacy of surgical and medical treatment.*
There are two possibilities either:

- -Bone necrosis and new bone formation (=chronic osteomyelitis) OR
- -Resolution

- **NECROSIS:** by the end of a week there is usually microscopic evidence of *bone death* due to:
- -The rising intraosseous pressure, vascular stasis, infective thrombosis and periosteal stripping increasingly compromise the blood supply
- -Bacterial toxins and
- -Leucocytic enzymes also may play their part in the advancing tissue destruction.
- With the gradual ingrowth of granulation tissue the boundary between dead and living bone becomes defined.
- *Pieces of dead bone separate as sequestra* varying in size from mere spicules to large necrotic segments.


- Macrophages and lymphocytes arrive in increasing numbers and the debris is slowly removed by a combination of phagocytosis and osteoclastic resorption.
- However, the larger sequestra remain entombed in cavities of bone, inaccessible to either final destruction or repair.

- **NEW BONE FORMATION:** once sequestra are formed the condition is now established as a *chronic osteomyelitis*.
- New bone forms from the deep layers of the stripped periosteum. This is typical of pyogenic infection and is usually obvious by the end of the second week.
- With time the **new bone thickens** to form an **involucrum** enclosing the infected tissue and sequestra.
- If the infection persists, pus and tiny sequestrated spicules of bone may continue to discharge through **perforations** in the involucrum known as **cloacae** and track by sinuses to the skin surfaces.



(c)

2.2 Acute osteomyelitis (a) Infection in the metaphysis may spread towards the surface, to form a subperiosteal abscess (b). Some of the bone may die, and is encased in

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- Chronic osteomyelitis following an acute is nowadays rare.
 - If infection is controlled and intraosseous pressure released at an early stage, chronic osteomyelitis can be aborted.



- **RESOLUTION:**

- The bone around the zone of infection is at first osteoporotic (probably due to hyperaemia).
- With healing, there is fibrosis, appositional new bone formation; and periosteal reaction, which results in sclerosis and thickening of the bone.
- *In some cases, remodelling may restore the normal contours; in others, though healing is sound, the bone is left permanently deformed.*

● - -Clinical features:

- *Symptoms:*
- -usually a **child**,
- -There may be a **recent history of infection** - a septic toe, a boil, a sore throat or a discharge from the ear.
- -**Malaise** and a **fever**; in neglected cases, toxaemia may be marked.
- - **Severe pain and Loss of function**. The parents will have noticed that the child refuses to use one limb or to allow it to be handled or even touched
- The cardinal sign - and often the earliest - is **erosion and fuzziness of the sacroiliac joints**.
- Later there may be periarticular sclerosis, and finally bony ankylosis.

- **Signs:**
- -Typically the child **looks ill and feverish**; the **pulse rate is likely to be over 100** and the temperature is raised.
- - **Loss of function and loss of range of motion** of the limb; The limb is held still and even the gentlest manipulation is extremely painful
- -There is **severe tenderness** near one of the larger joints (e.g. above or below the knee, in the popliteal fossa or in the groin).
- -**Local redness, swelling, warmth and oedema** are later signs and signify that pus has escaped from the interior of the bone.
- -**Lymphadenopathy** is common but non-specific.
- It is important to remember that all these features may be attenuated if antibiotics have been administered.


- *In infants*, and especially in the newborn, the constitutional disturbance can be misleadingly mild; the baby simply fails to thrive and is drowsy but irritable.
- Suspicion should be aroused by a history of birth difficulties, umbilical artery catheterization or a site of infection (however mild) such as an inflamed intravenous infusion point.
- Metaphyseal tenderness and resistance to joint movement can signify either osteomyelitis or septic arthritis; indeed, both may be present, so the distinction hardly matters. Look for other sites - multiple infection is not uncommon.

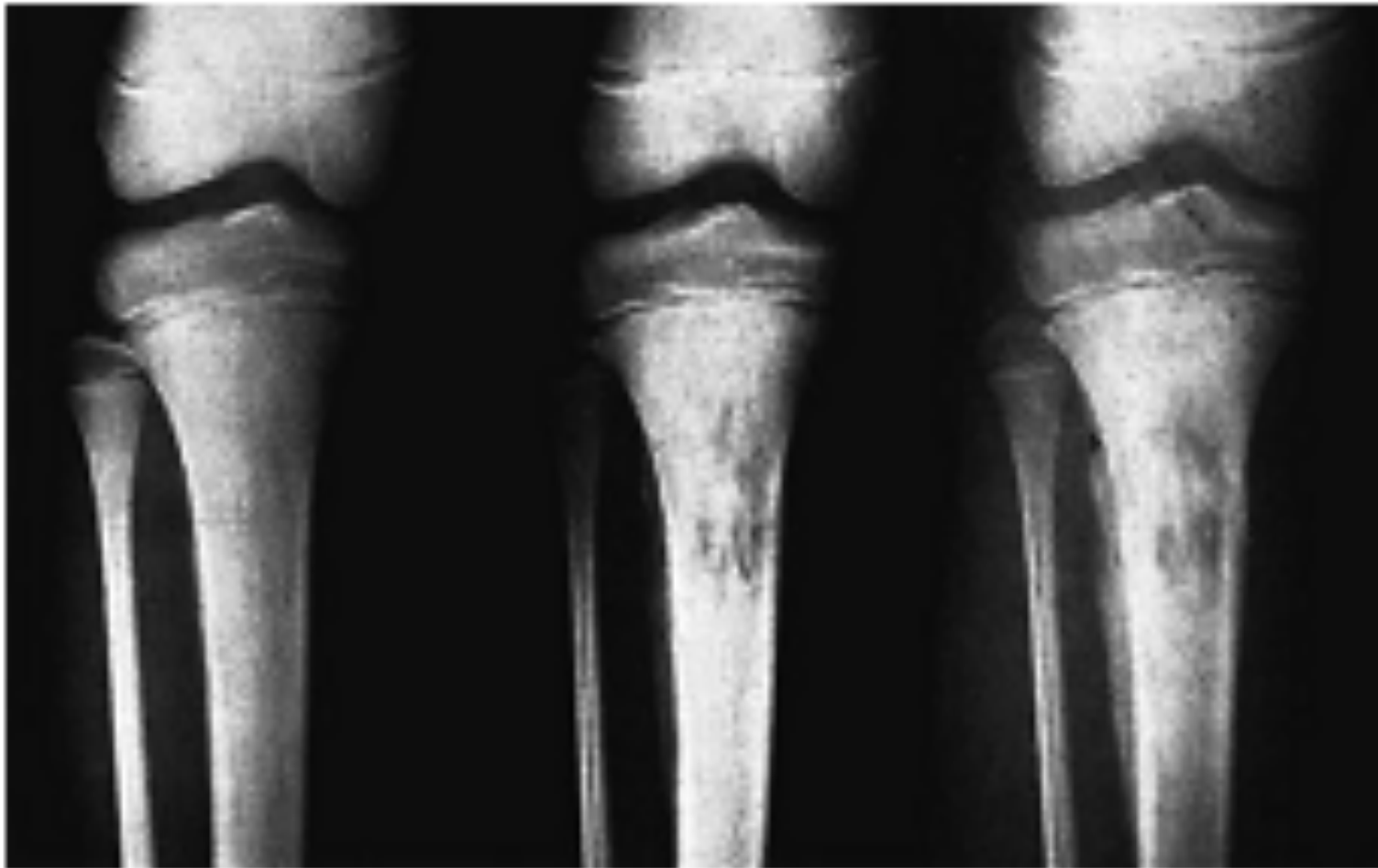
- **In adults** the commonest site of infection is the **thoracolumbar spine**.
- There may be a history of some urological procedure followed by a mild fever and backache.
- Local tenderness is not very marked and it may take weeks before x-ray signs appear; when they do appear the diagnosis may still need to be confirmed by fine-needle aspiration and bacteriological culture.
- Other bones are occasionally involved, especially if there is a background of diabetes, malnutrition, drug addiction, leukaemia, immunosuppressive therapy or debility.
- *In the very elderly*, and in those with immune deficiency, systemic features are mild and the diagnosis is easily missed.

● Diagnostic imaging:

● *Plain x-ray:*

- -During the first few days, no abnormality of the bone. Displacement of the fat planes signifies soft-tissue swelling, but this could as well be due to a haematoma or soft-tissue infection.
- -By the end of the second week, there may be a faint extra-cortical outline due to periosteal new bone formation. This is the classic x-ray sign of pyogenic osteomyelitis.
- -Later the periosteal thickening becomes more obvious and there is patchy rarefaction of the metaphysis.

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- -An important late sign is the combination of regional osteoporosis with a localized segment of apparently increased density.
 - Osteoporosis is a feature of metabolically active, and thus living, bone;
 - the segment that fails to become osteoporotic is metabolically inactive and possibly dead.



2.3 Acute osteomyelitis The first x-ray, 2 days after symptoms began, is normal – it always is; metaphyseal mottling and periosteal changes were not obvious until the second film, taken 14 days later; eventually much of the shaft was involved.

- **Ultrasound:** may detect a subperiosteal collection of fluid in the early stages of osteomyelitis, but it cannot distinguish between a haematoma and pus.
- **Radioscintigraphy:** with Technetium-HDP reveals increased activity in both the perfusion phase and the bone phase.
- This is a highly sensitive investigation, even in the very early stages, but it has relatively low specificity and other inflammatory lesions can show similar changes.
- In doubtful cases, scanning with Gallium citrate or Indium labelled leucocytes may be more revealing.



- **MRI:**

- is extremely sensitive, even in the early phase of bone infection, and can therefore help to differentiate between soft-tissue infection and osteomyelitis.
- The most typical feature is a reduced intensity signal in T₁-weighted images.

- **Lab Investigations:**
- -**Microbiological examination of pus aspirate** from the metaphyseal subperiosteal abscess or the adjacent joint: a simple Gram stain of a smear may help to identify the type of infection and assist with the initial choice of antibiotic as well as microbiological culture and tests for sensitivity to antibiotics.
- -Increased **white cell count** and **C-reactive protein** values and the **ESR** also rises but it may take several days to do so and it often remains elevated even after the infection subsides.
- -**Blood culture** is positive in only about *half* the cases of proven infection.

- **-Differential diagnosis:**
- **CELLULITIS**
- Skin infection where there is widespread superficial redness and lymphangitis.
- There is no bony involvement.
- The organism is usually staphylococcus or streptococcus.
- Mild cases will respond to an oral penicillinase-resistant penicillin; severe cases need intravenous antibiotics.



- **ACUTE SUPPURATIVE ARTHRITIS**

- Tenderness is diffuse, and all movement at the joint is abolished by muscle spasm.
- In infants the distinction between osteomyelitis and septic arthritis is somewhat theoretical, as both usually coexist.

- **ACUTE RHEUMATISM**

- The pain is less severe and it tends to fleet from one joint to another,
- and there may be carditis, rheumatic nodules or erythema marginatum.

● **STREPTOCOCCAL NECROTIZING MYOSITIS**

- A rare condition were group A Beta-haemolytic streptococci (the same organisms which are responsible for the common 'sore throat') occasionally invade muscles and cause an acute myositis which, in its early stages, may be mistaken for cellulitis or osteomyelitis.
- Intense pain and board-like swelling of the limb in a patient with fever and a general feeling of illness are warning signs of a medical emergency.
- This rapidly progresses out of control with extensive muscle necrosis, septicaemia and death.
- MRI will reveal muscle swelling and possibly signs of tissue breakdown.
- Immediate treatment with intravenous antibiotics is essential.
- Surgical debridement of necrotic tissue - and sometimes even amputation - may be life saving.

- **SICKLE-CELL CRISIS**

- The patient may present with features indistinguishable from those of acute osteomyelitis.
- In areas where *Salmonella* is endemic it would be wise to treat such patients with suitable antibiotics until infection is definitely excluded.

- **GAUCHER'S DISEASE**


- Pseudo-osteitis may occur with features closely resembling those of osteomyelitis.
- The diagnosis is made by finding other stigmata of the disease, especially enlargement of the spleen and liver.

● -Treatment:

- If osteomyelitis is suspected on clinical grounds, blood and fluid samples should be taken and then treatment started immediately without waiting for final confirmation of the diagnosis.
- There are four important aspects to the management of the patient:
 - (1) supportive treatment for pain and dehydration;
 - (2) splintage;
 - (3) antibiotic therapy; and
 - (4) surgical drainage.

- **GENERAL SUPPORTIVE TREATMENT** for pain, fever and dehydration
- **SPLINTAGE** of the affected part for comfort and also to prevent joint contractures. Simple skin traction may suffice and, if the hip is involved, this also helps to prevent dislocation. At other sites a plaster slab or half-cylinder may be used but it should not obscure the affected area.
- **ANTIBIOTICS** Blood and aspiration material are sent immediately for examination and culture, but the prompt administration of antibiotics is so vital that treatment should not await the result.
- Initially the choice of antibiotics is based on the findings from direct examination of the pus smear and a 'best guess' at the most likely pathogen; a more appropriate drug can be substituted, if necessary, once the organism is identified and its antibiotic sensitivity is known.

- The following recommendations are offered as a guide rather than a specific policy:
- *Older children and previously fit adults*, who probably have a staphylococcal infection, are started on intravenous **flucloxacillin and fusidic acid**.
- Fusidic acid is preferred to benzylpenicillin partly because of the high prevalence of penicillin resistant staph and as it is particularly concentrated in bone.
- However, for a known streptococcal infection benzylpenicillin is better.
- Patients who are allergic to penicillin should be treated with a **2nd or 3rd generation cephalosporin**.
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- *In children from 6 months to 6 years*
 - Empirical treatment in this age group should include cover against Haemophilus influenzae, unless it is known for certain that the child has had an antihaemophilus vaccination.
 - This is best provided by ***a combination of IV flucloxacillin and cefotaxime or cefuroxime.***


- *Neonates and infants up to 6 months of age:*
- Initial ttt should be effective against penicillin resistant staph aureus , Group B streptococcus and Gram negative organisms. Drugs of choice are *flucloxacillin* plus 3rd gen cephalosporin like *cefotaxime*.
- Alternatively, effective empirical ttt can be provided by *a combination of flucloxacillin* (for penicillin resistant staph) , *benzylpenicillin* (for Group B streptococci) and *gentamicin* (for Gram negative organisms)

- *Patients with sickle-cell disease* are prone to bone infection and in most cases this is due to salmonellae and/or other Gram negative organisms. The recommended treatment is with ***chloramphenicol*** . Nowadays the antibiotic of choice is a third generation ***cephalosporin or a fluoroquinolone like cripfloxacin***.
- *Heroin addicts and immunocompromised patients* often have unusual infections (e.g. pseudomonas, proteus or bacteroides). When the background is known, it is wise to start with one of third gen ***cephalosporins or a fluoroquinolone*** preparation , depending on the results of sensitivity tests.


Patients considered to be at risk of meticillin-resistant Staphylococcus aureus (MRSA) infection

- Patients admitted with acute haematogenous osteomyelitis and who have a previous history of MRSA infection, or any patient with a bone infection admitted to a hospital or a ward where MRSA is endemic, should be treated with intravenous **vancomycin** (or similar antibiotic) together with **a third-generation cephalosporin**.

- The usual programme is to administer the drugs intravenously (if necessary adjusting the choice of antibiotic once the results of antimicrobial sensitivity become available) until the patient's condition begins to improve and the CRP values return to normal levels – which usually takes 2–4 weeks depending on the virulence of the infection and the patient's general degree of fitness.
- By that time the most appropriate antibiotic would have been prescribed, on the basis of sensitivity tests; this can then be administered orally for another 3–6 weeks, though if bone destruction is marked the period of treatment may have to be longer.

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- While patients are on oral antibiotics it is important to track the serum antibiotic levels in order to ensure that the minimal inhibitory concentration (MIC) is maintained or exceeded.
 - CRP, ESR and WBC values are also checked at regular intervals and treatment can be discontinued when these are seen to remain normal.

- **DRAINAGE** If antibiotics are given early, drainage is often unnecessary.
- However, if the clinical features do not improve within 36 hours of starting treatment, or even before that if there are signs of deep pus (swelling, oedema, fluctuation), and most certainly if pus is aspirated, the abscess should be drained by open operation under general anaesthesia.
- If pus is found - and released - there is little to be gained by drilling into the medullary cavity.
- If there is no obvious abscess, it is reasonable to drill a few holes into the bone in various directions.
- The wound is closed without a drain and the splint (or traction) is reapplied.

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- **Postoperative management:**
 - Once the signs of infection subside, movements are encouraged and the child is allowed to walk with the aid of crutches.
 - Full weight bearing is usually possible after 3-4 weeks.

● Complications:

● METASTATIC INFECTION

- This is sometimes seen - generally in infants - and may involve other bones, joints, serous cavities, the brain or lung.
- It is easy to miss secondary sites of infection when attention is focused on one particular area;
- it is important to be alert to this complication and to examine the child all over and repeatedly.
- A lethal outcome from septicaemia is nowadays extremely rare; with antibiotics

● ***SUPPURATIVE ARTHRITIS***

- This may occur:
- (1) in very young children, in whom the growth disc is not an impenetrable barrier;
- (2) where the metaphysis is intracapsular, as in the upper femur; or
- (3) from metastatic infection.
- In infants it is so common as almost to be taken for granted, especially with osteomyelitis of the femoral neck

● **CHRONIC OSTEOMYELITIS**

- Despite improved methods of diagnosis and treatment, acute osteomyelitis sometimes fails to resolve and the patient is left with a chronic infection and a draining sinus.
- This may be due to neglect but is also seen in debilitated patients and in those with compromised defence mechanisms.




- **ALTERED BONE GROWTH**

- In infants, physical damage may lead to arrest of growth and shortening of the bone.
 - In older children, however, the bone occasionally grows too long because metaphyseal hyperaemia has stimulated the growth disc.
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● POSTOPERATIVE OSTEOMYELITIS

- **Definition:** Osteomyelitis can occur after any operation on bone, but especially after operating on open fractures and after procedures involving the use of foreign implants.
- **Incidence:** The reported incidence of infection after orthopaedic operations on a broad cross-section of patients varies from 0.2% to over 10%. Much depends on the criteria for diagnosing 'postoperative' infection.

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- **Risk factors:** the risk is considerably greater in the elderly, the obese, those with diabetes or other chronic diseases, patients with sickle-cell disease, Gaucher's disease or leukaemia, patients on corticosteroid or immunosuppressive therapy, and patients who have had multiple previous operations at the same site.

- **Local factors that favour bacterial invasion** are:
(1) *the foreign implant* is both a predisposing factor and an important element in its persistence. Bacteria as well as human tissue cells have an affinity for molecules on the surface of the implant. Both compete for occupancy of the same surface '***the race for the surface***'. If the tissue cells win, the implant is incorporated as an 'inert' biomaterial. If the bacteria win, the resulting infection usually persists until the implant is removed.
- (2) soft-tissue damage; (3) haematoma formation; and (4) bone death.

- **Organisms:** in postoperative osteomyelitis are usually a mixture of pathogenic bacteria (*Staph. aureus*, *Proteus*, *E. coli*, *Pseudomonas*) and others that are not normally pathogenic (e.g. *Staph. epidermidis*) but may become so in the presence of a foreign implant.
- **Route:** Organisms may be introduced:
 - -directly into the wound from the atmosphere, the instruments, the patient or the surgeon; or
 - -by haematogenous spread from a distant focus.



- **Classification of postoperative infection:**

- *A. Early infection*

- 1. Superficial

- 2. Deep

- 3. Superficial and deep


- *B. Late infection*

- 1. Following early infection


- 2. Covert infection appearing later

- 3. Following a long period of normality

- **Clinical features**
- *-Early postoperative infection (within 1 month)* is usually fairly obvious. With a purely superficial infection the symptoms are minimal, but if the infection is deep, the patient complains of persistent pain and may have a fever. The skin over the implant is inflamed, and there may be a purulent discharge from the wound. Often there is tenderness and pain on moving the limb. The ESR and white cell count are elevated, and blood culture may be positive. Bacteriological examination of the wound discharge will help to identify the organism and establish the antibiotic sensitivity.
- Rarely (e.g. in patients on immunosuppressive therapy) there may be a fulminant postoperative infection with septicaemia and toxæmia.

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- ***-Intermediate postoperative infection*** is seen between 1 month and 1 year after the operation. Often there is a history of 'wound problems' in the early postoperative period, followed by a long quiescent period during which both the surgeon and the patient may be falsely reassured while the organisms lurk in hidden corners, waiting to proliferate and spread when local conditions favour their emergence.

- **-Late postoperative infection** is much more difficult to diagnose. Several years may have elapsed since the operation, during which the patient was completely asymptomatic. Pain usually starts insidiously and may never become acute. Often there is no more than a low-grade inflammatory reaction, little different from that due to aseptic loosening of the implant; this is especially true of cemented joint prostheses. Local examination, x-ray signs of bone resorption and increased activity on radionuclide scanning may equally fail to distinguish between aseptic loosening and infection. However, if there is marked periosteal new bone formation and cortical destruction, with increased scinti-graphic activity in both the perfusion phase and the bone phase, the likelihood of infection is greatly increased. The MRI may show a localized area of high signal activity due to pus.

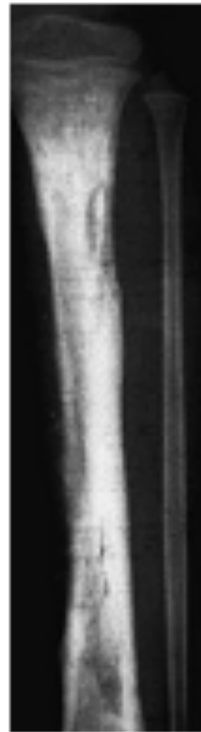
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- Blood investigations are sometimes helpful. The ESR is always elevated after joint replacement but it should return to normal by 6 months.
 - Confirmation of the diagnosis is obtained by aspirating purulent material from the area, or by culturing the organism in washings taken after attempted aspiration.

• **Prevention**

- The risk of implant-mediated infection can be reduced by:
- (1) avoiding operations on immune-depressed patients;
- (2) eliminating any focus of infection before operating;
- (3) insisting on optimal operative sterility; The introduction by Charnley of the ultra-clean air operating enclosure and special operating suits with body exhausts brought a substantial reduction in his incidence of wound infection (especially 'early' infection) after hip replacement. Wearing impermeable operative clothing also is of value. Using all methods combined, the sepsis rate for total hip replacement can be reduced to below 0.2%.

- (4) giving prophylactic antibiotics(e.g. one of the cephalosporins, given intravenously shortly before and then 8-hourly for two further doses after operation);
- (5) proper surgical technique and handling tissues gently;
- (6) using high-quality implant materials;
- (7) ensuring secure fixation of the implant; and
- (8) preventing or treating later intercurrent infection.

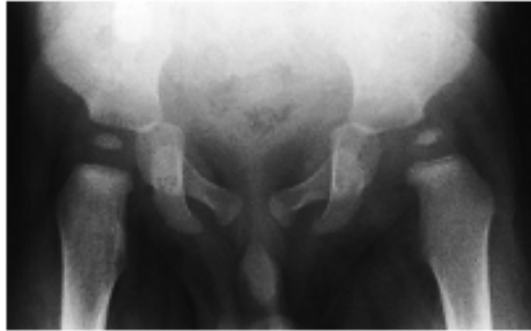
- **Treatment**
- Appropriate *antibiotics*, given intravenously and in large doses
- If there is an abscess, it should be drained, *debrided* and the wound left open followed by intermittent antibiotic irrigation and suction drainage
- If at all possible the *fixation device* should be retained until the fracture has united; even worse than a septic fracture is a septic unstable fracture. If the implant has to be removed in order to achieve adequate debridement, the fracture should be held securely with an external fixator



(a)

(b)

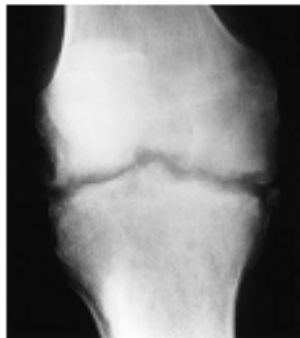
2.5 Chronic osteomyelitis
Chronic osteomyelitis may follow acute. The young boy (a) presented with draining sinuses at the site of a previous acute infection. The x-ray shows densely sclerotic bone. (b) In adults, chronic osteomyelitis is usually a sequel to open trauma or operation.



(a)



(b)



(c)

2.8 Suppurative arthritis – x-ray

(a) In this child the left hip is subluxated and the soft tissues are swollen. (b) If the infection persists untreated, the articular epiphysis may be entirely destroyed, leaving a permanent pseudarthrosis. (c) Septic arthritis in an adult knee joint.



(a)

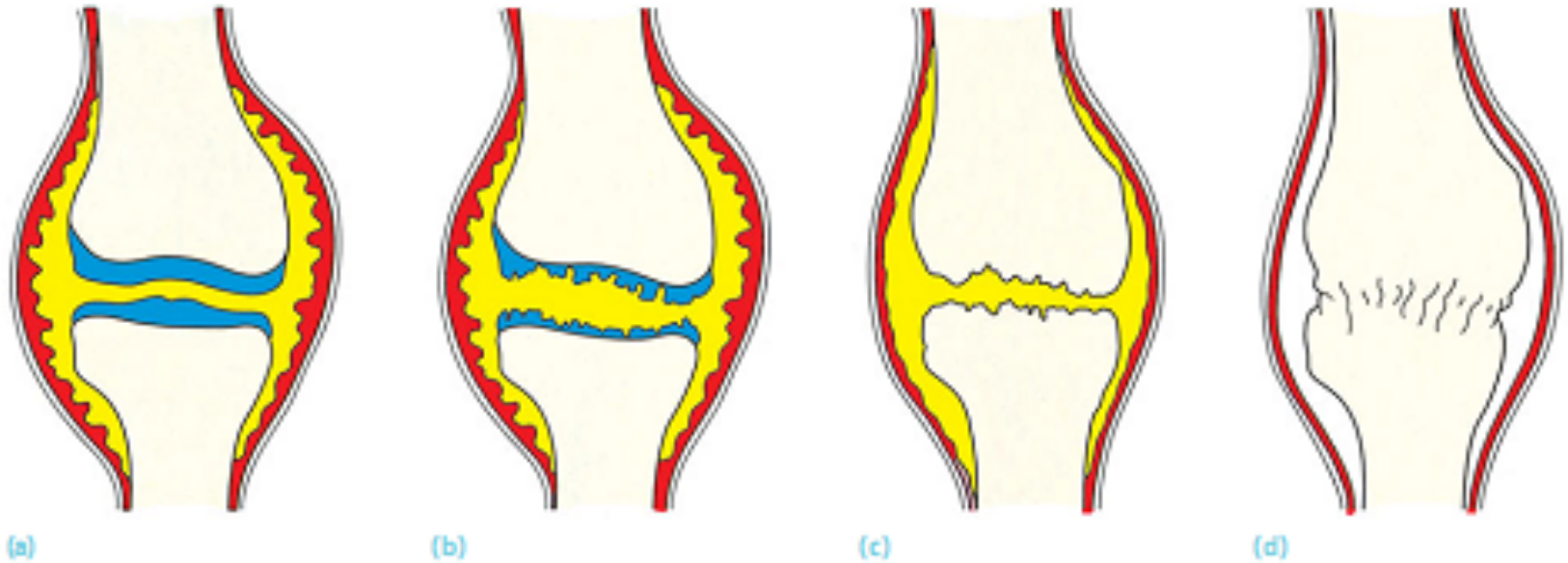


(b)




(c)

2.4 Subacute osteomyelitis (a,b)
The classic Brodie's abscess looks like a small walled-off cavity in the bone with little or no periosteal reaction;
(c) sometimes rarefaction is more diffuse and there may be cortical erosion and periosteal reaction.



2.7 Acute suppurative arthritis – pathology In the early stage (a), there is an acute synovitis with a purulent joint effusion. (b) Soon the articular cartilage is attacked by bacterial and cellular enzymes. If the infection is not arrested, the cartilage may be completely destroyed (c). Healing then leads to bony ankylosis (d).



THANK

YOU