OSTEOARTICULAR INFECTIONS

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Acute osteomyelitis and septic arthritis have a lot in common in terms of presentation and principles of management

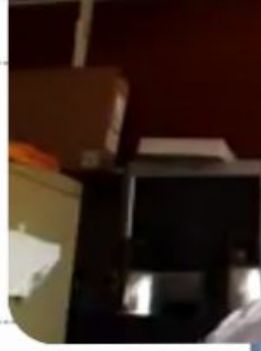
Acute haematogenous osteomyelitis: clinical presentation

1) Acute haematogenous osteomyelitis

2)Subacute haematogenous osteomyelitis.

Absence of systemic illness and presenting in less than 10 days

3)Chronic osteomyelitis where there maybe presence or absence of systemic illness, evidence of radiographic bone changes and a history of previous episode of infection



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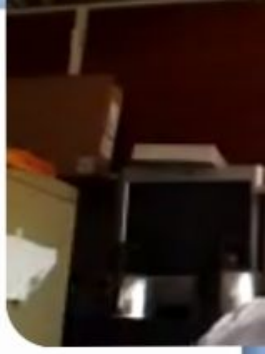
Cardinal features of acute osteomyelitis in children

1) Pain and swelling if a joint is involved. Pain near a joint may also suggest bone infection at its early stages.
2) Fever
3) Refusal to bear weight (DD trauma to bone or joint
4) Elevated ESR
5) Elevated CRP

Blood investigations in acute osteoarticular infections



- Complete haemogram with wbc differential
- ESR
- C-reactive protein
- For articular infections, aspiration of joints for culture and sensitivity. This may not be practical in childhood joint infections who ideally should have formal arthrotomy in which the material is sent for culture and sensitivity. This is usually done under GA.

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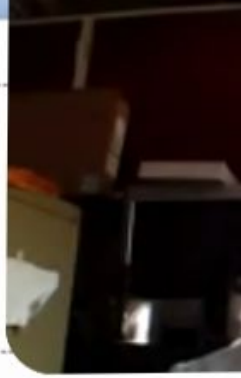
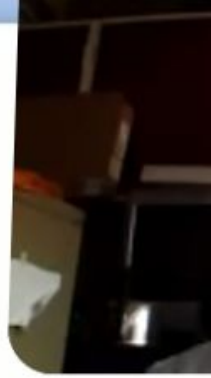


Points to consider in infection of bone :

Radiographic investigation

- Plane X-ray changes may start showing as periosteal elevation or soft tissue swelling if a joint is involved. Plane xray changes are usually seen between 11 and 14 days
- Radionuclide scanning – more sensitive than plane radiographs but has poor specificity T99
- MRI has replaced radionuclide scan and can be a guide as to location of abscess for aspiration, and also show marrow edema
- CT may be done but not as sensitive as MRI.



Septic Arthritis

Septic arthritis may be monoarticular or polyarticular.

Besides open arthrotomy which is what is commonly done, arthroscopic drainage and lavage can be also be done in any joint except probably in a paediatric hip for which treatment failure can be very costly that means the recommendation for the paediatric hip open arthrotomy should always be done.

Synovial fluid analysis

- Gram stain of aspirate
- Culture and sensitivity
- WBC and differential
- Glucose levels
- Crystals from the joint aspirate

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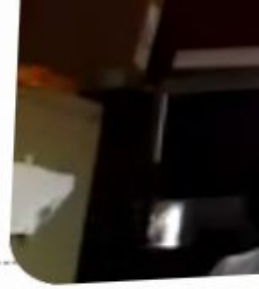

- Distinguishing septic arthiritis of the hip from transient synovitis is a common problem.
- The diagnosis of septic arthiritis is made in more than 90% of the cases when the Kochers criteria is met.

1. WBC count more than 12,000 cells/ml
2. ESR higher than 40mm/hr
3. Inability to bear weight
4. Temperature higher than 38.6 degrees centigrade

- Usually transient synovitis is a self limiting inflammation of the hip which usually settles without much intervention

Differential diagnosis in septic arthritis across all age groups

- Trauma
- Juvenile Rh.A
- Transient synovitis
- sickle cell crises
- Rheumatoid Arthiritis(tends to be in adults)
- Osteoarthiritis (in adults)
- Crystalline arthropathies
- Haemophilia- haemoarthrosis

Aspiration of joint material for culture and sensitivity or material obtained from formal arthrotomy.

Blood cultures especially where multiple joints are involved and septicaemia if suspected

There may be no bone changes except soft tissue swelling unless the bone has become involved as would happen in intracapsular joints.

Common sites of septic arthritis

- Knee 33%
- Hip 20%
- Shoulder 11%

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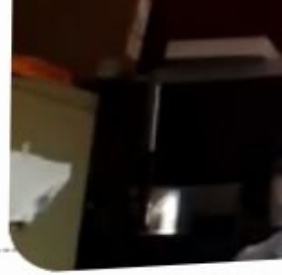
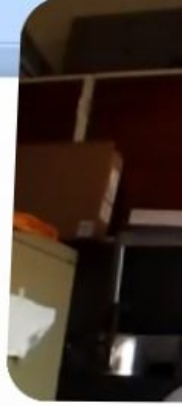


Differential diagnosis of haematogenous osteomyelitis

- Septic arthritis- pain near the joint and sympathetic effusion into the joint
- Soft tissue infections such as cellulitis pyomyositis which is infection of muscle planes
- Juvenile chronic arthritis
- Trauma
- Acute Rh. Fever
- Sickle cell crisis



A note on sickle-cell disease:

- There are three types of arthritis that should be considered and has been encountered in patients with sickle cell disease.

1. Aseptic arthritis
2. Aseptic arthritis associated with remote infection, often of the salmonella type
3. Septic arthritis where the organism is staphylococcus areus.

In general the commonest organism in SCD is staph areus.

Pathogenesis and natural history of acute haematogenou osteomyelitis as well as septic arthritis

In septic arthritis the organisms reach the joint through the synovium whose basement membrane is thin or absent. The inflammatory response leads to migration of polymorpho nuclear leukocytes which release proteolytic enzymes that may cause acute damage to articular cartilage. This damage may be irreversible. It must be noted the window of opportunity of clearing the infection in septic arthiritis which may save the cartilage is only 4 days(In acute haematogenous osteomyelitis is only 48 hours)

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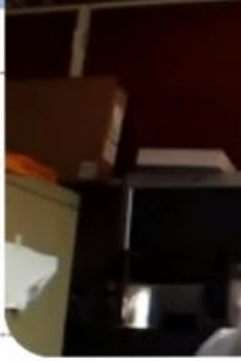
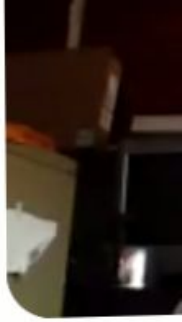


- Haematogenous osteomyelitis in infants below one year: blood supply between metaphysis and epiphysis - transepiphyseal vessels freely communicate. Spread into joint may happen and it may be difficult to tell the location of the initial infection (especially in the hip). After the age of 2 years there's a clear cut separation between the metaphysial and epiphysial blood supply and the epiphyseal plate acts a barrier in spreading from metaphseal to the epiphyseal region thus to the joint.

In acute haematogenous osteomyelitis the Capillaries branch from nutrient arteries and progress longitudina towards the physis but turn 180 degrees and reach and empty into much larger venous sinusoids. This is an area of slow and turbulence circulation. The capillaries do not anastomose and act as end vessels. Thrombosis of these vascular system can result in vascular stasis and with the presence of micro organisms lead to necrosis of bone.

Another postulated factor is decreased oxygen tension diminishing the phagocytic activity of macrophages.



The infection can spread via pathways of least resistance including harvesian system, Volkmans canal. In children older than 1 to 2 years infection can spread in two ways . It will generally spread and erode cortex forming subperiosteal abscess, thereby elevating the periosteum. The other direction of spread is in the diaphysis through the endosteal blood supply which is jeopardized by generated thrombotic pressure. The consequence of involvement of the harvesian systems and Volkmans canals is extensive formation of sequestrum. Ten percent of acute haematogenous osteomyelitis usually progress to chronic osteomyelitis. It should be understood this is usually related to late diagnosis. As the infection progresses the original cortical bone is devascularized forming a sequestrum while the new bone laid down will eventually develop as the involucrum.

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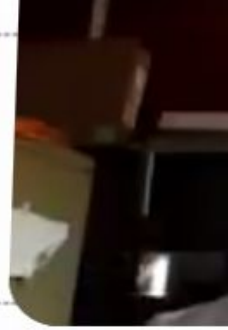

- What is a sequestrum? A sequestrum is a necrotic segment of bone without blood supply that acts as a nidus for infection in chronic osteomyelitis
- What is an involucrum? The involucrum is the new cortical bone laid down by the periosteum around the shell of the old cortex (sequestrum)

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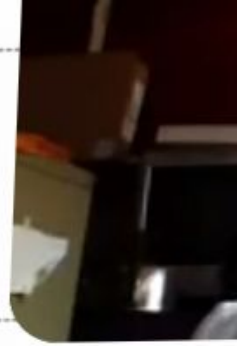

- Acute inflammatory reactions with vascular congestions. Exudation of fluid with infiltration by polymorphonuclear leucocytes. The increase in introsseous pressure rises rapidly causing intense pain and intravascular thrombosis. This early cascade of events threatens the bone with ischaemia, resorption due to a combination of phagocytic activity and the local accumulation of cytokines, growth factors, prostaglandins and bacterial enzymes. In the 2nd or 3rd day pus forms within the bone and forces its way along the volkmanns channel to the subperiosteal space, with the elevated periosteum also laying a layer of new bone along its length. Intraoseous pressure rise in joints that are partly intracapsular (eg hip, shoulder and elbow) and may discharge pus through the periosteum into the joint.

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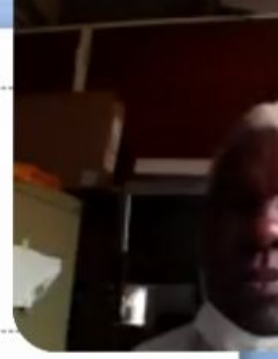

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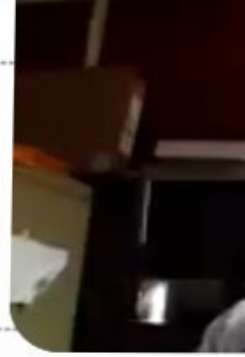

Bacterial colonization and resistance to antibiotics is enhanced by the ability of certain organisms (including staphylococcus) to adhere to avascular bone surface and foreign implants. This phenomenon tends to protect the bacteria from antibiotics and other host defences. The protein involved in this phenomena is a polysaccharide slime (glycocalyx or biofilm). Thus bacterial adherence to biomaterials should be considered when treating infections after ORIF.

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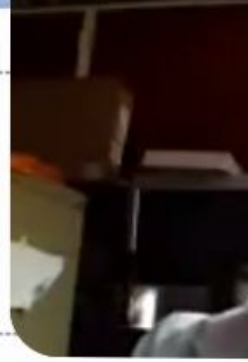

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Treatment

Prompt intravenous antibiotics is vital and should not wait for the laboratory results.

It is also important to have a supportive care plan which should include resting the joint or the limb in a functional position. This will prevent formation of unacceptable contractures. This especially true for the infected knee and hip.analgesics must be given. Backslab for the knee and short duration skin traction for the hip.

Empirical IV antibiotics must be started before the diagnosis has been comfirmed. In all the studies done show commonest organism is staph areus and therefore any antibiotic started empirically should cover this organism. In septic arthritis the window of opportunity of preventing permanent cartilage damage is only 4 – 7 days. In acute haematogeous osteomyelitis, the window of opportunity is as short as 48 hours.

The choice of antibiotics may be guided by the clinicians experiences of local condition or direct examination of pus smear.

Factors such as patients' age, general state of resistance, renal function , degree of toxaemia, and previous history of drug allergy must be taken into account

Supportive treatment:

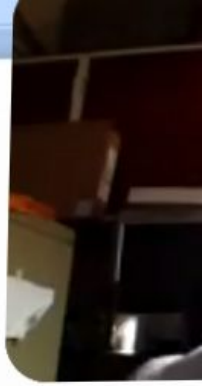
Temperature control by anti-pyretics which should also provide analgesia. The limb should always be rested in a functional position. Normally the hip is rested by simple skin traction until the pain has been controlled and this is especially true in children.

1.Neonates and infants up to 6 months

Drugs of choice to consider are flucloxacillin and a third generation cepharosporin . This will cover both penicillin resistant staph aureus and group B Streptococcus as well as gram negative organisms

2.Children 6 months to 6 years of age. If H. influenza vaccination has not been given then flucloxacillin and cefotaxime can be given.

3.In older children and previously fit adults the majority of them will have a staphylococcal infection and can be started on IV flucloxacillin and fusidic acid which is preferred to benzypenicillin because of possibility of resistant staph aureus and its better bone penetration.



Note: On adult haematogenous osteomyelitis common site may be thoracolumbar spine.

There maybe a history of a urological procedure, followed by mild fever and backache . Local tenderness may not be marked.

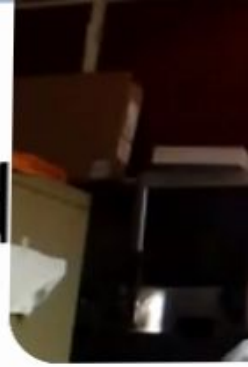
X-Ray changes may take weeks before appearing.

Diagnosis may additionally need fine needle aspiration and bacteriological culture

Other imaging which may be required are MRI, CT or Spect CT.

Surgical drainage of a deep abscess may be required . At the back of ones mind, tb spondylodiscitis should be considered. The history would definitely be much longer and various x ray features, may be different from an acute bacterial spondylodiscitis. Other bones and joints are occasionally affected especially if there is a background of DM, malnutrition , drug addition, leukemia or immunosuppressive therapy or HIV

Complications of acute haematogenous osteomyelitis and septic arthritis



- Progression to chronic osteomyelitis
- Chronic discharging sinuses
- In children, growth plate may be damaged leading to limb shortening and unequal growth
- Pathological fractures especially where the organism causing disease are of severe virulence and the patient's immunity is low. Usually, the formed new bone which is called involucrum is unable to support the bone.
- In septic arthritis there may be permanent joint damage which may lead to either fibrous or bony ankylosis

SUMMARY OF SEPTIC ARTHRITIS

1. Portal of entry haematogeneous , direct penetration into the joint su during operations, injections. Septicemia tends to affect more than one join

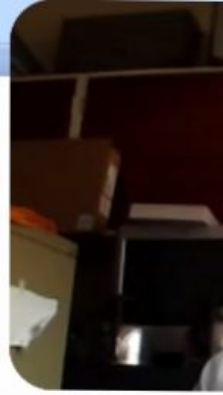
   In the hip spread into the joints of pus can also occur as a result of metaphyseal infection of the neck of femur which is intracapsular.

2. **Pathology**

   - Synovial membrane
   - thin or absent basement membrane of capillary vessels in synovial system.

3. **Cartilage damage**

   - Bacterial enzymes
   - Proteolytic enzymes released by synovial cells, inflammatory cells (Macrophages), Pus is chondrolytic and septic arthritis in children is an emergency irrespective of which joint is involved.



4. **Treatment**

Aspirations

May almost always be impossible in children, and this maybe especially true in the hip. While as arthroscopic clearance of pus can be done, this is not recommended in the hip where one has a single chance of clearing the pus completely. Open arthrotomy is recommended.

5. **Types of organisms causing septic arthritis can be a guide to appropriate anti-biotic treatment.**

Across all age groups, the commonest isolated organism is staphylococcus areus

Staph and Group B streptococci

Haemophilus influenza if not immunized

Salmonellas species occasionally isolated in people with sickle cell anemia

6. **The hip must be rested after arthrotomy.**

The most effective way to rest the hip is by skin traction. Follow up will determine when the skin traction is removed and mobilization started. In the literature it is said that an infected joint can cleaned through athroscopic washout. This is not recommended in the hip where formal opening of the joint should be done, so that no infection is missed in the synovial folds of the joint. You only have a short opportunity to get it right, the cartilage may have been damaged by the time you consider repeating the athrotomy.

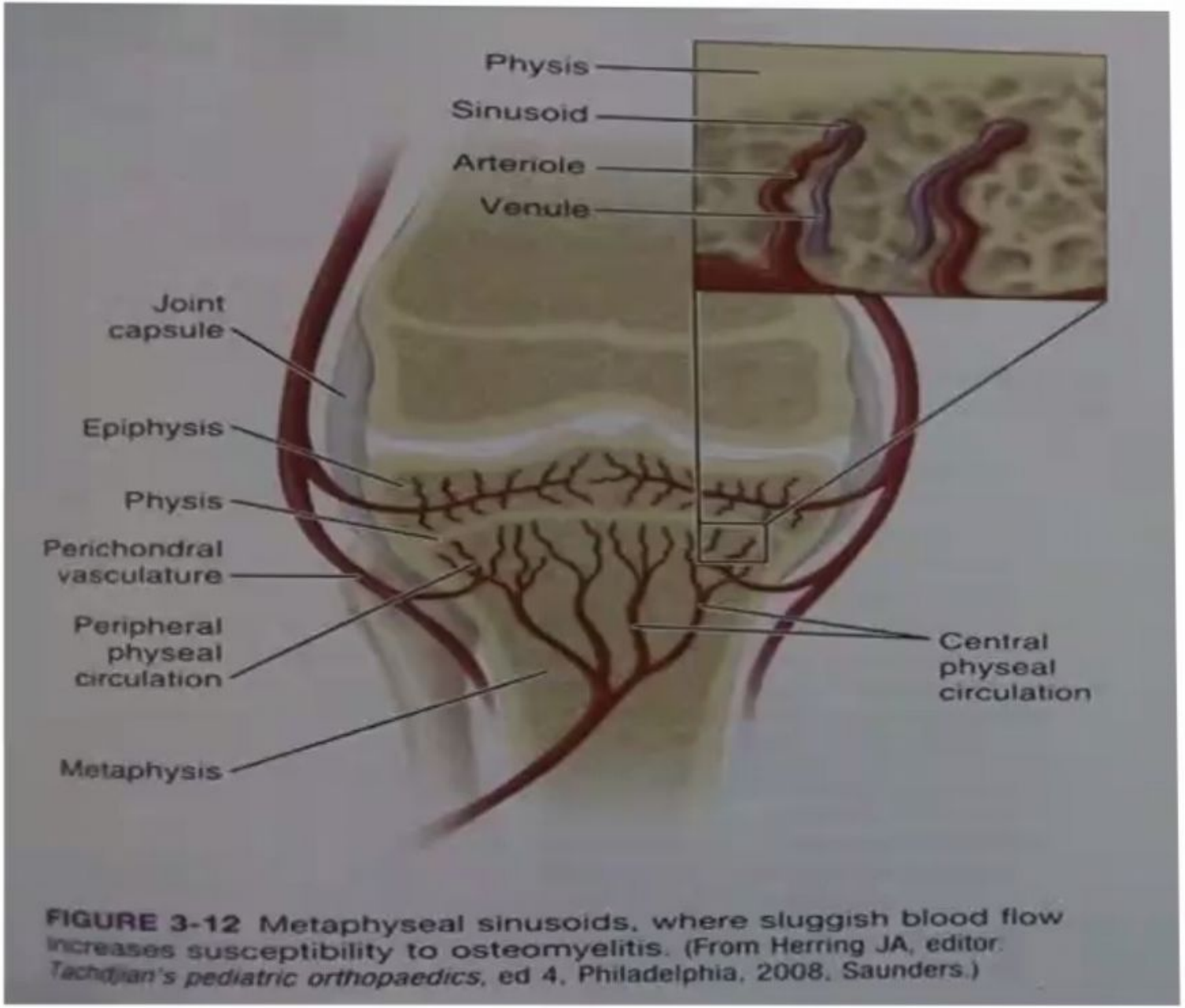


FIGURE 3-12 Metaphyseal sinusoids, where sluggish blood flow increases susceptibility to osteomyelitis. (From Herring JA, editor. *Tachdjian's pediatric orthopaedics*, ed 4, Philadelphia, 2008, Saunders.)

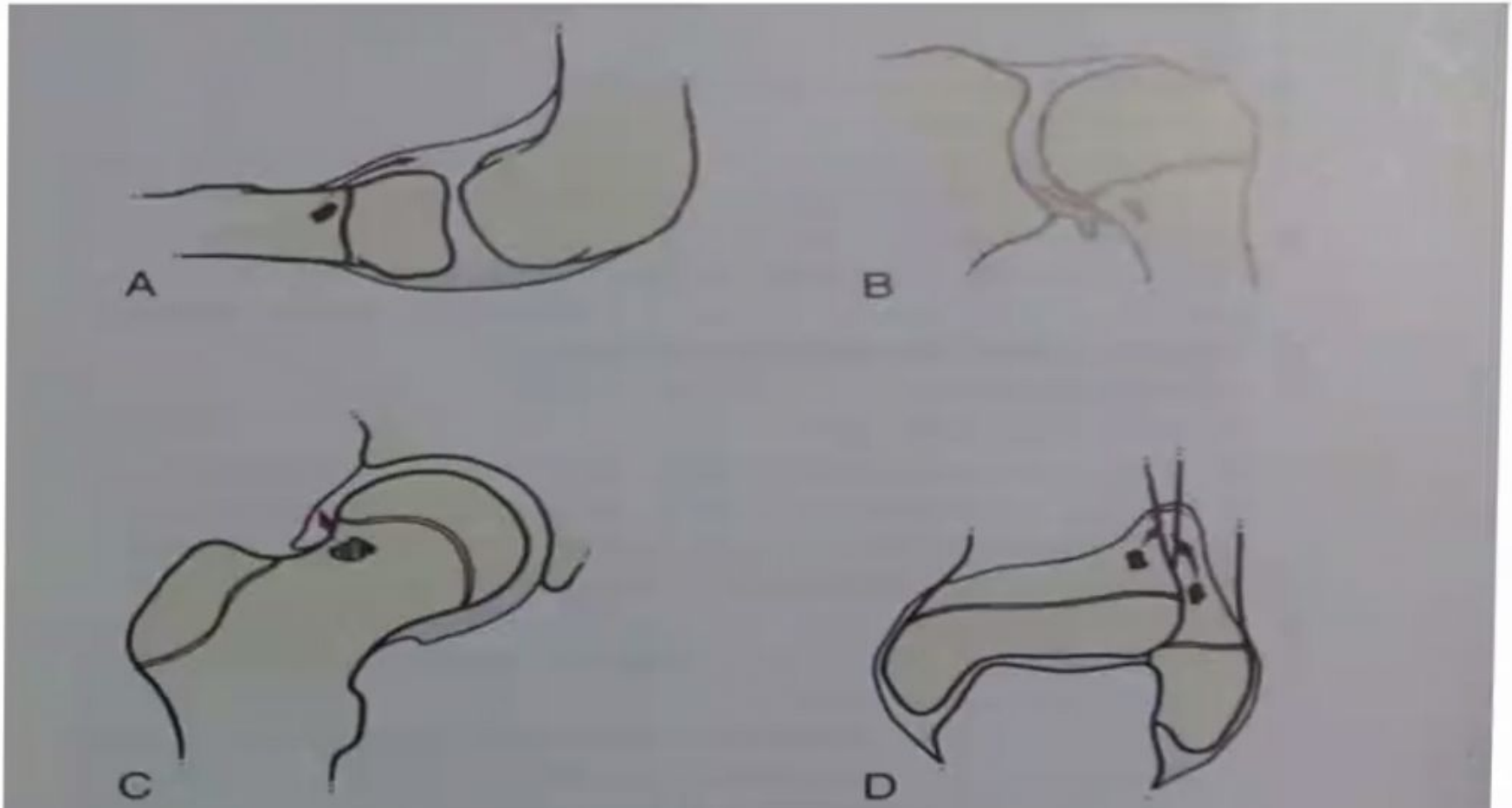
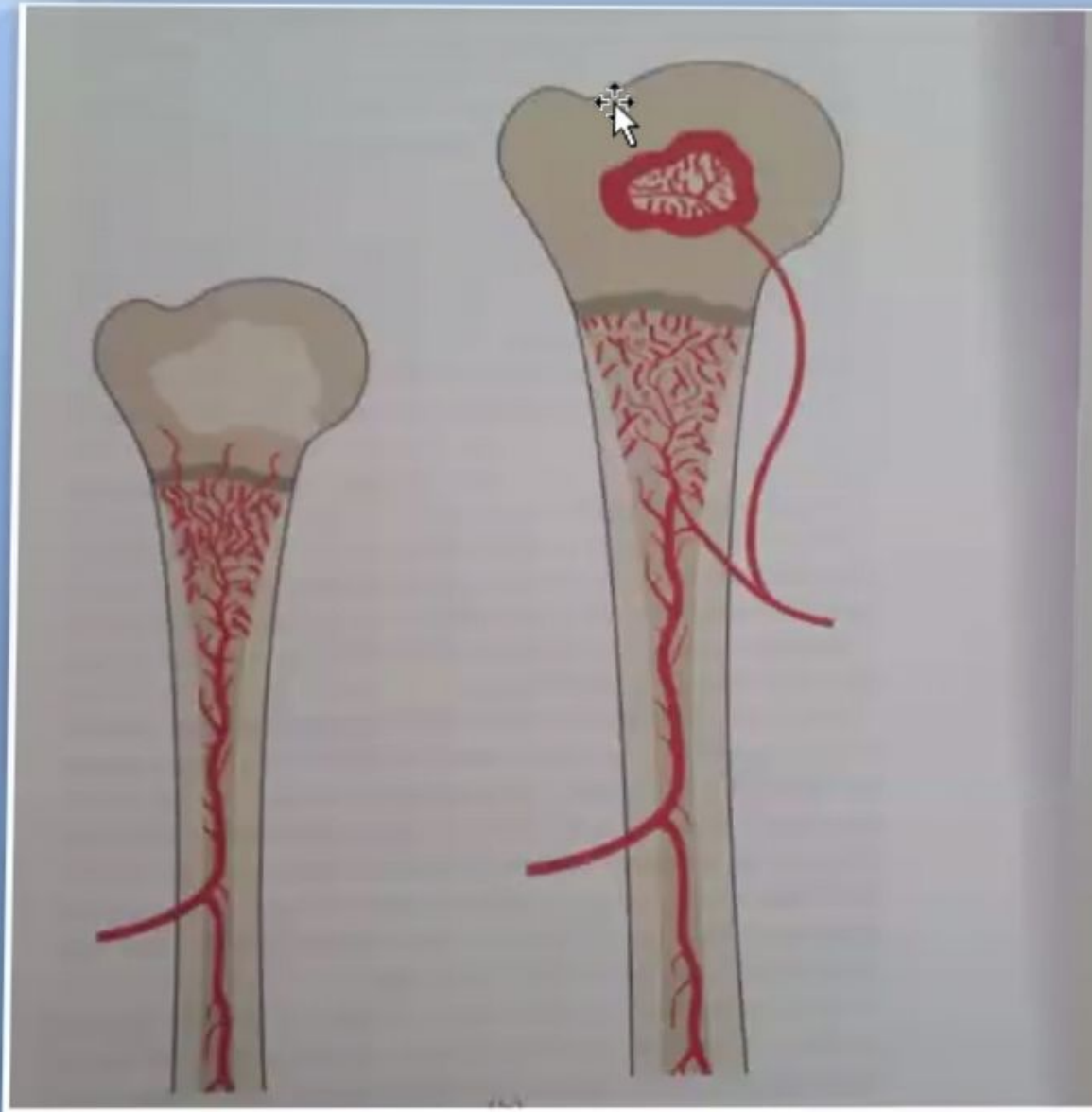
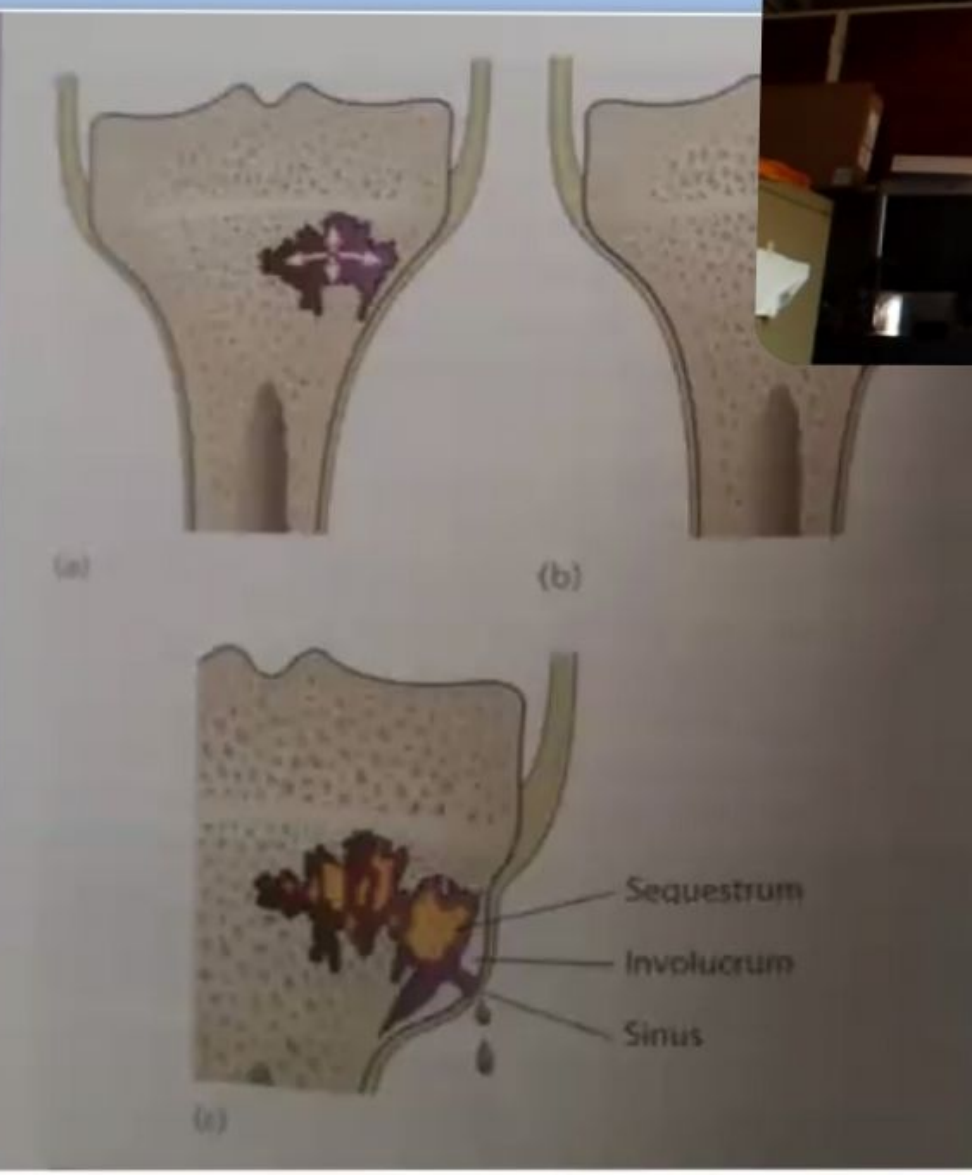


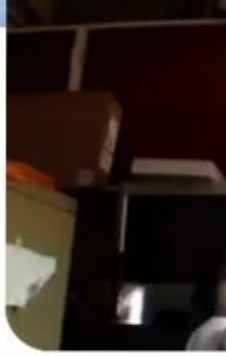
FIGURE 3-13 Metaphyses of the proximal radius **(A)**, proximal humerus **(B)**, proximal femur **(C)**, and distal tibia and fibula **(D)** are intraarticular. Osteomyelitis in these locations may decompress into the joint and produce concomitant septic arthritis. (From Herring JA, editor: *Tachdjian's pediatric orthopaedics*, ed 5, Philadelphia, 2014, Elsevier Saunders, Figure 27-23)

Table 3-6 Common Organisms in Septic Arthritis, by Age

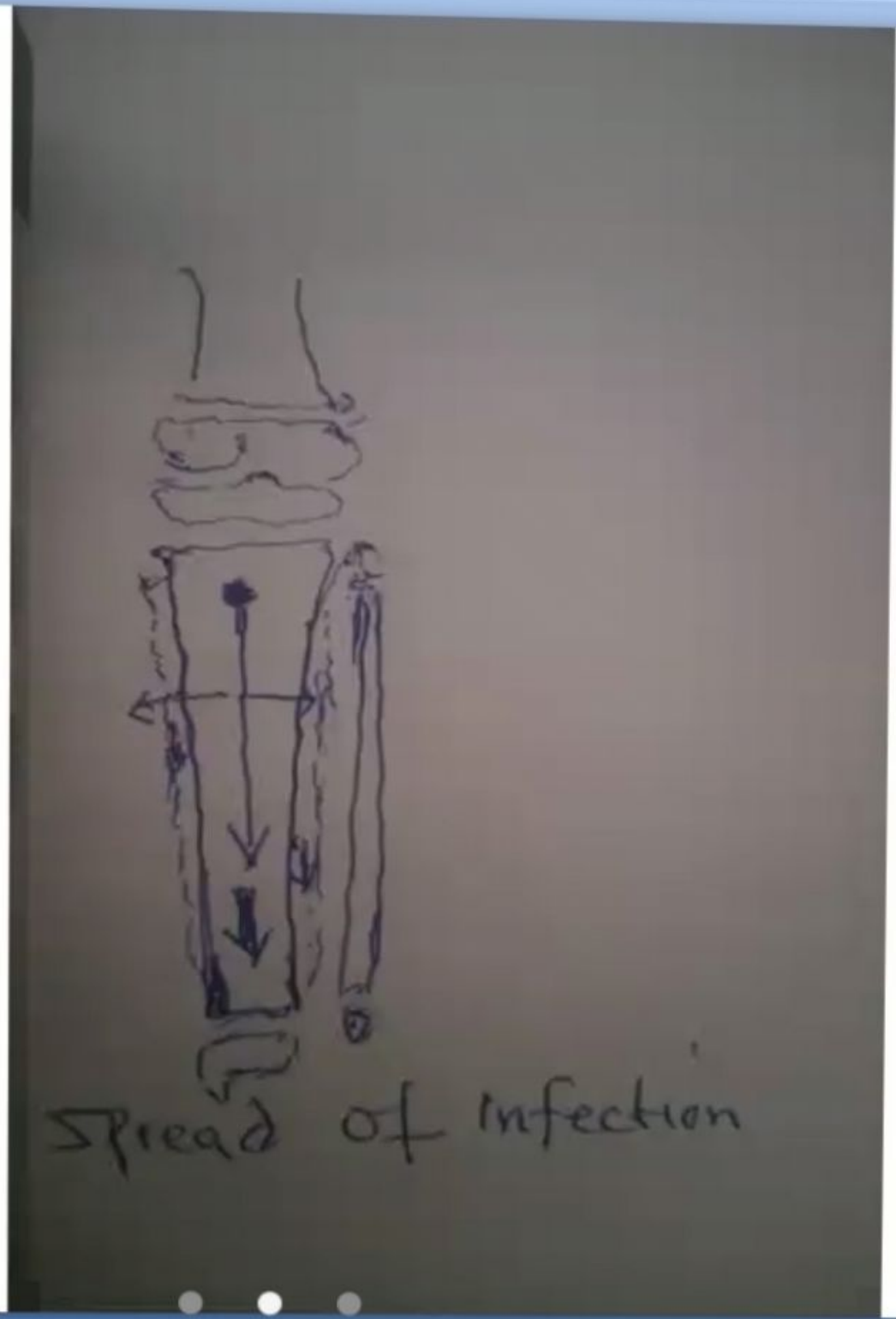
AGE	COMMON ORGANISMS	EMPIRICAL ANTIBIOTICS
<12 mo	*Staphylococcus* spp., group B streptococci	First-generation cephalosporin
6 mo-5 yr	*Staphylococcus* spp., *Haemophilus influenzae*	Second- or third-generation cephalosporin
5-12 yr	*Staphylococcus aureus*	First-generation cephalosporin
12-18 yr	*S. aureus, Neisseria gonorrhoeae*	Oxacillin/cephalosporin

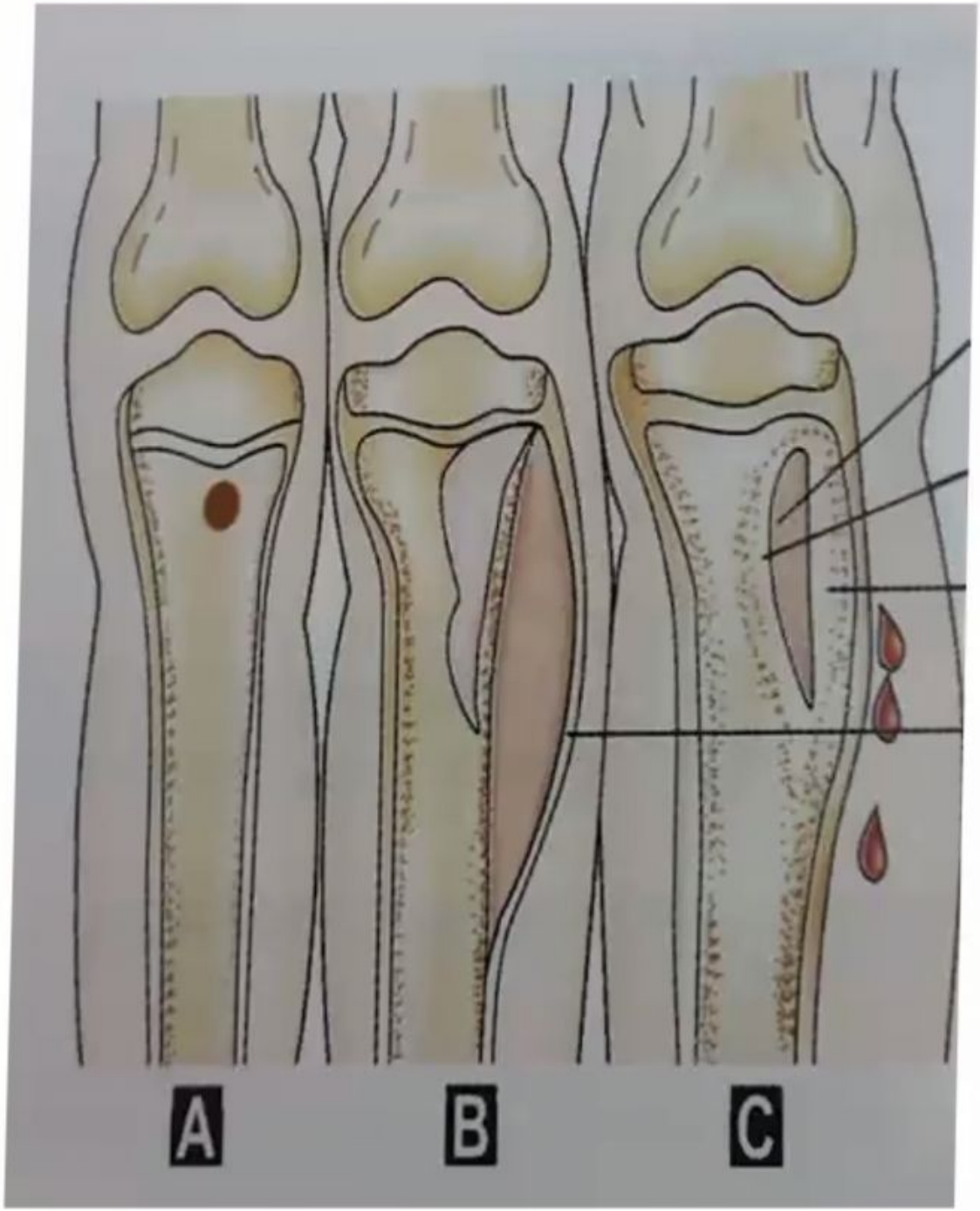



(a)
(b)
Sequestrum
Involucrum
Sinus
(c)

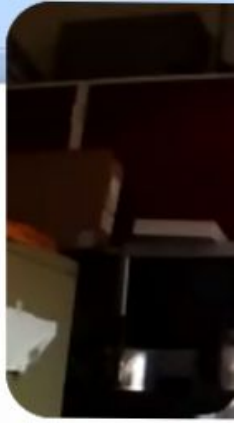


- After initial infection, the spread may take several routes including proximally where after the first year of life, the growth plate may act as a barrier. Besides forming pus, presenting as a sinus the spread may also destroy bone with its blood vessels as it spreads distally and if not treated early may render a large part of the cortical bone avascular and depending on host resistance versus virulence of the pathogen, may cause a pathological fracture of the weakened and dead bone whose periosteum has been destroyed.


Spread of Infection

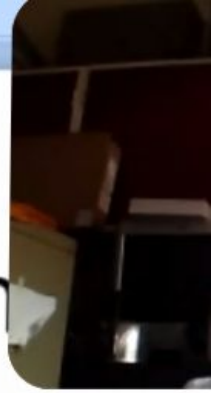

A
B
C

Chronic Osteomyeliti



- Bone destruction and formation are the characteristic findings in chronic infections. Serpiginous (creeping and spreading) areas of bone destruction which may be confused with bone tumour.
- Acute infections often produce cortical bone destruction and periosteal elevation which may again be confused which may be confused with a tumour like Ewings sarcoma which may also cause fever.

- Chronic infections with long standing skin damage presentin as a sinus may change to squamous cell carcinoma. Material that has been sent for culture should be subjected to histology (biopsy) and material sent for biopsy should likewise be subjected to culture.

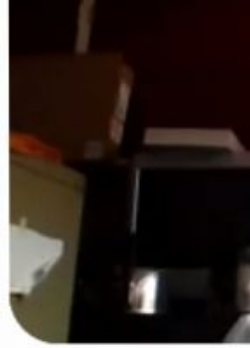


- Chronic infections with long standing skin damage presentin as a sinus may change to squamous cell carcinoma. Material that has been sent for culture should be subjected to histology (biopsy) and material sent for biopsy should likewise be subjected to culture.

Contd.

- Histology will reveal a mixed-cell population of inflammatory cells, plasma cells, polymorphonuclear leucocytes, eosinophils, lymphocytes and histiocytes.

Treatment of Chronic Osteomyelitis



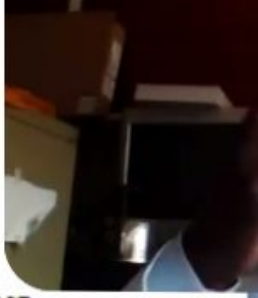
- "Staging" the condition helps in risk-benefit analysis but in general terms chronic osteomyelitis is seldom eradicated by antibiotics alone. Continuous collaboration with a microbiologist and as treatment progresses, input from a plastic surgeon is invaluable.

Treatment

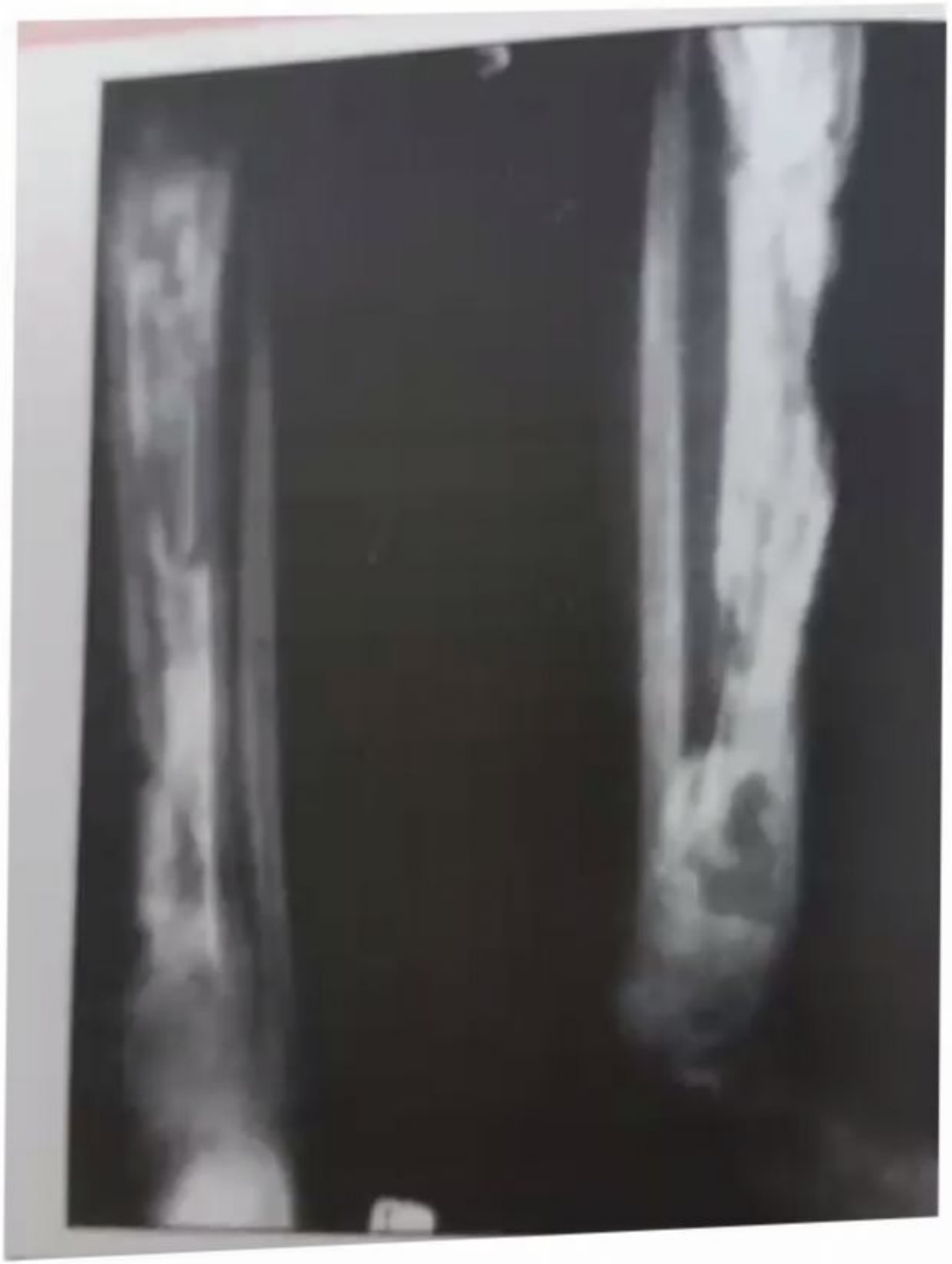
- Antibiotics
- Local treatment (sinuses)
- Surgery: A waiting policy? Primary cause of infection:

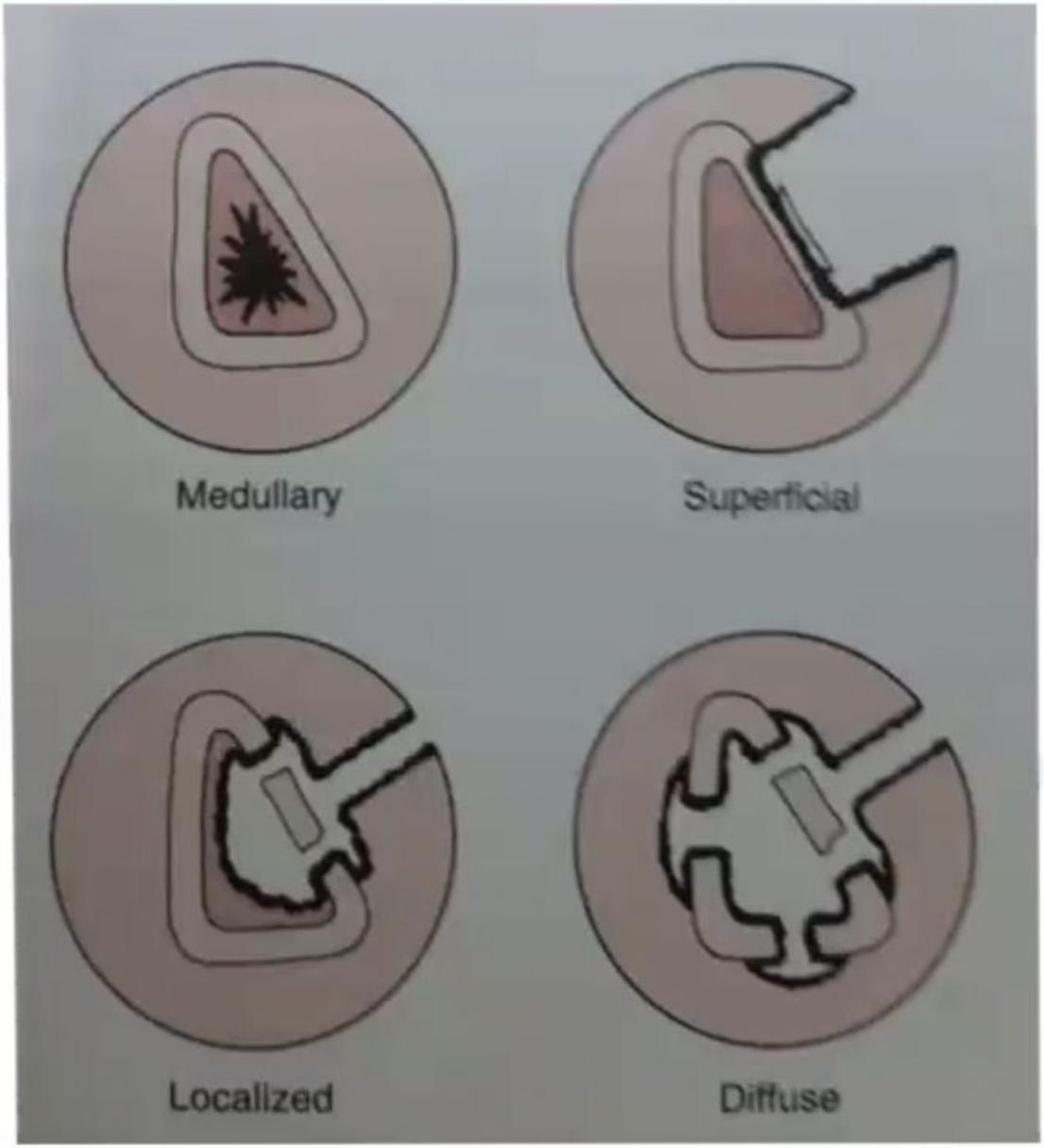
a) Chronic haematogenous infection
b) Post traumatic bone infection
c) Infection in the presence of an implant.

In developed countries where the medical services are good and patients are seen early by their general practitioner, the issue of haematogenous spread in children from sites like ear nose and throat has markedly reduced. The challenge in those countries is dealing with bone infection as a result of compound fractures and implants which have been complicated by infection.

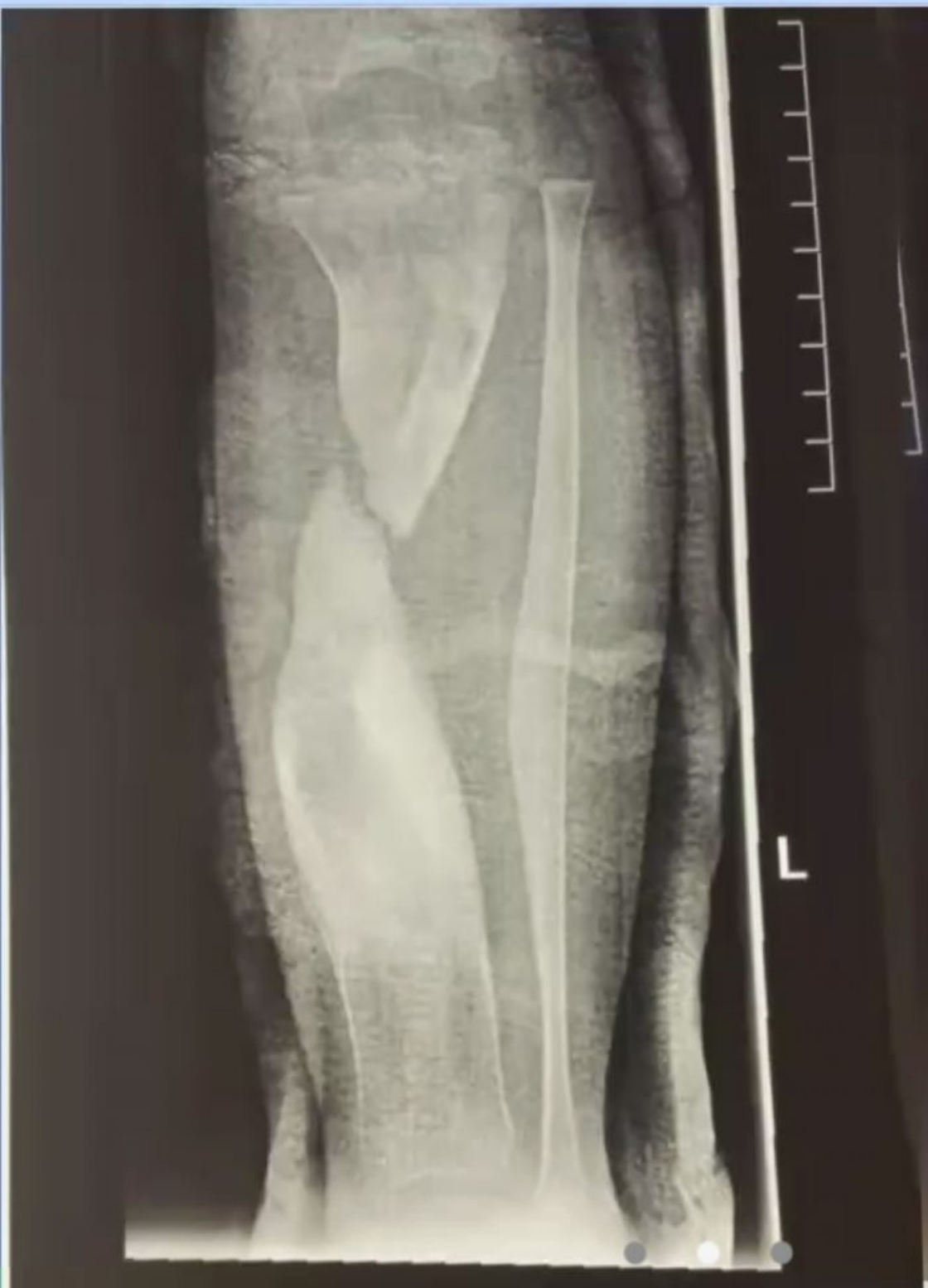


a) Post operative infection – presence of implant (foreign material)
b) Dealing with 'dead space'. Porous antibiotic impregnated beads
c) Bone grafts – Consider papineau technique – cavity packed with cancellous bone graft
d) Flap transfer
e) Lautenbach technique – Excision (radical) followed by closed irrigation e.g lifomycin but normal saline may be used
f) Refractory cases with bone loss may be treated by one or staged fibular graft or ilizarov method of bone transport.





Cierny and collegues in 2003 is based on both the local pathological anatomy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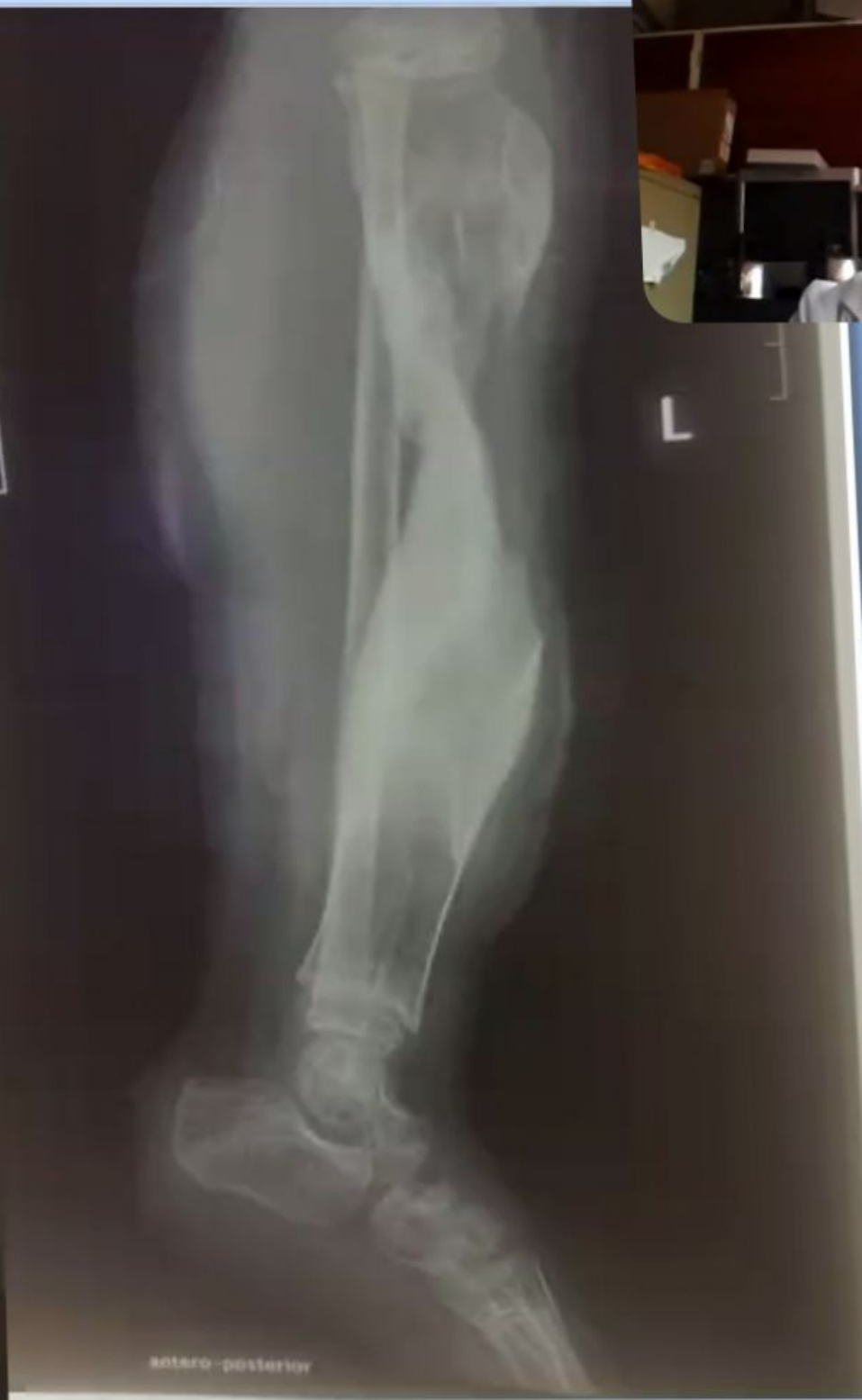
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antero-posterior