E-lecture 7

Myositis and Myonecrosis

Myositis and myonecrosis	
Pyomyositis	*S. aureus*
Streptococcal necrotizing myositis	*S. pyogenes*
Gas gangrene	*Clostridium* spp.
Nonclostridial (crepitant) myositis	Mixed aerobic and anaerobic bacteria
Synergistic nonclostridial anaerobic myonecrosis	Mixed aerobic and anaerobic bacteria

- Muscle involvement (inflammation or infection) can occur with:
- → *viral infection (such as influenza, dengue, or coxsackievirus B infection)*
- → Parasitic invasion (trichinellosis, cysticercosis, or toxoplasmosis).
- Myalgia (muscle pain) can occur in most of these infections, severe muscle pain is the hallmark of pleurodynia (coxsackievirus B), trichinellosis, and bacterial infection.

>Acute rhabdomyolysis (breakdown of damaged muscle) predictably occurs with clostridial and streptococcal myositis, as both these organism have enzymes that breakdown muscle.

> Rhabdomyolysis is less so associated with influenza *virus, echovirus, coxsackievirus, Epstein-Barr virus, and Legionella infections*.

Necrotizing myositis

- *S. pyogenes* (GAS) may induce primary myositis (referred to as streptococcal necrotizing myositis) in association with severe systemic toxicity
- → this is basically necrotizing fasciitis (Type2) that involves the muscle tissue.

- Myonecrosis occurs in about 50% of cases in typical necrotizing fasciitis without muscle involvement being the primary tissue infected!
- (meaning muscles are affected by necrosis when the surrounding fascia is involved, even without the infection being active in the muscle itself!)

pyomyositis

- Pyomyositis, or pus forming infection of the muscle tissue, is usually due to *S. aureus* (remember it is the typical pus former in skin)
- Pyomyositis is common in tropical areas, and generally *has no known portal of entry (in contrast to necrotizing fasciitis)*.
- Cases of pyomyositis caused by MRSA producing the PVL toxin have been described among children in the United States.
- Muscle infection begins at the exact site of blunt trauma or muscle strain

Cont.

- Pyomyositis infection usually remains localized, and shock does not develop unless organisms produce
- 1- *toxic shock syndrome toxin 1 (which acts as a super antigen, that causes exaggerated immune response that is many fold the normal response → shock.*
- 2-enterotoxins (exotoxins produced by *S. aureus).*

- If the patient lacks antibodies to the toxins above then they are prone to developing toxic shock when these toxins are produced.

Pyomyositis

- a purulent- PUS forming- infection of skeletal muscle.

- Abscess formation is the usual consequence when these pyogenic bacteria reach the muscle tissue.

- Pyomyositis usually arises from **haematogenous** spread (deeper infections, muscle and bone, are typically more related with haematogenous spread rather direct inoculation).

Epidemiology

- Occurs in M> F , and more in tropical climate in two main age groups:

→children (aged 2–5 years)

→adults (aged 20–45 years).

In temperate climes

→ pyomyositis typically affects adults or the elderly (not children).

→Patients usually have predisposing conditions such as HIV, infection, DM, malignancy, cirrhosis, renal insufficiency, organ transplantation (reduced cell immunity), immunosuppressive therapy.

Other risk factors include trauma, IDU, and concurrent infections (toxocariasis –roundworms-, VZV).

Microbiology

- *S. aureus* 90% of tropical cases 75% of temperate cases.
- GAS account for 1–5% of cases all around.
- *E. coli* ST131 is an emerging cause in patients with haematological malignancy.
- Uncommon causes B, C, and G streptococci and *S. pneumoniae, and S. anginosus*.
- Rare causes include *Enterobacteriaceae, Y. enterocolitica, N. gonorrhoeae, H. influenzae, A. hydrophila, anaerobes, B. mallei, B. pseudomallei, A. fumigatus, Candida spp., MTB, and MAC.*

Clinical features

- 20% and 50% of cases patient have had recent blunt trauma or vigorous exercise of the affected area –myolysis-
- The muscle area is damaged and becomes susceptible for infections).
- Seen more in the lower extremity (thigh, calf, gluteal muscles), but not limited to that area and can affect any muscle group.
- Multifocal infection occurs in up to 20% of cases!
- Since it is usually from a hematologic cause, the patient must be assessed for complications of bacteraemia (endocarditis)

clinical stages:

- • Stage 1 (early invasive stage)—

  crampy local muscle pain, swelling, and lowgrade fever. Induration (hardening) of the affected muscle + leucocytosis may be present

- • Stage 2 (suppurative stage)—at 10–21 days after onset of symptoms (most patients present at this stage).

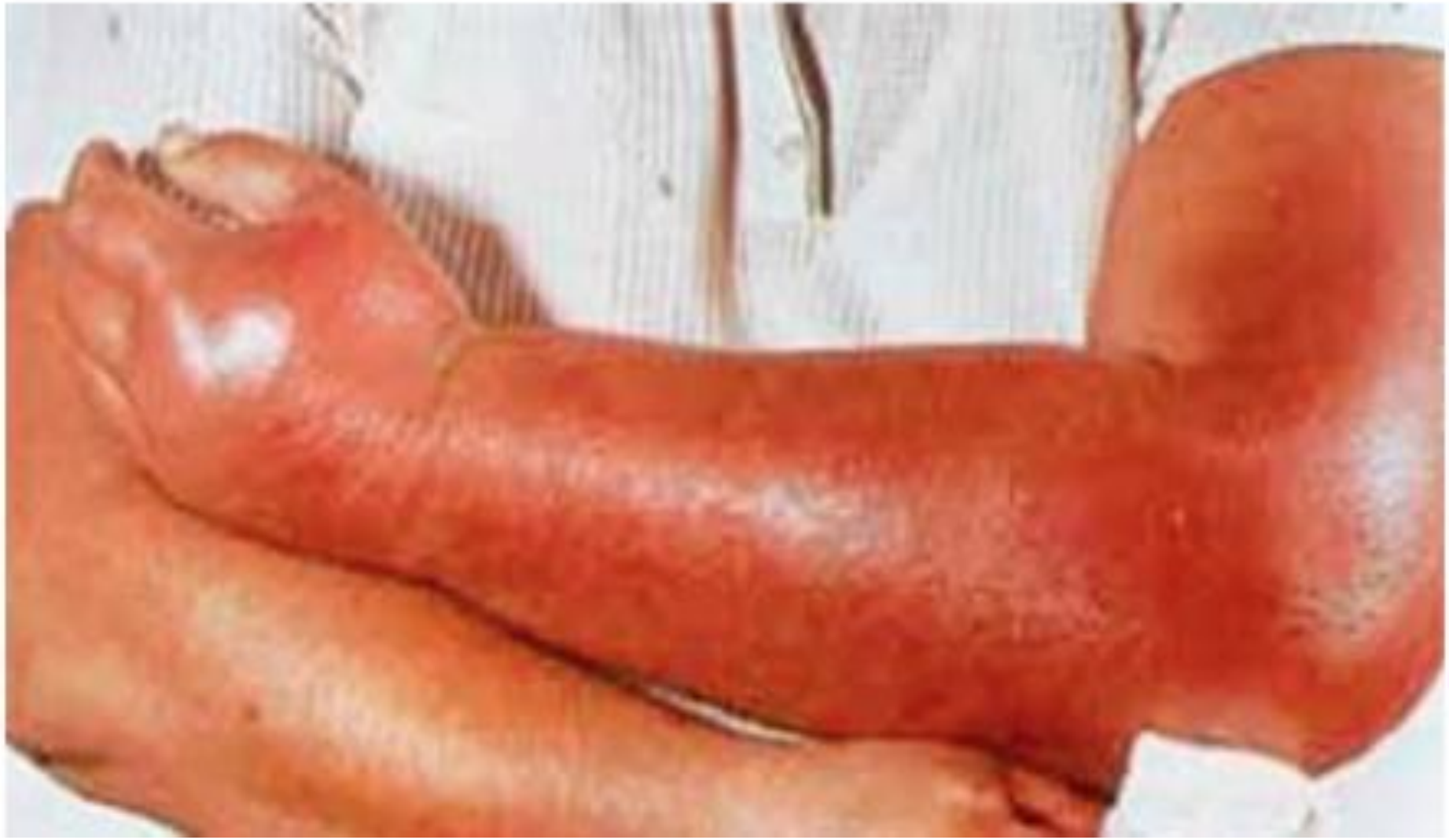
  fever, very sharp muscle tenderness and swellingAn abscess may be clinically apparent, aspiration of which yields pus. There is marked leucocytosis;

- • Stage 3 (systemic stage)—
- the affected muscle is fluctuant. Patients may present with complications of *S. aureus* bacteraemia, e.g. septic shock, endocarditis, septic emboli, pneumonia, pericarditis, septic arthritis, brain abscess, and ARF. Rhabdomyolysis may occur.

Diagnosis

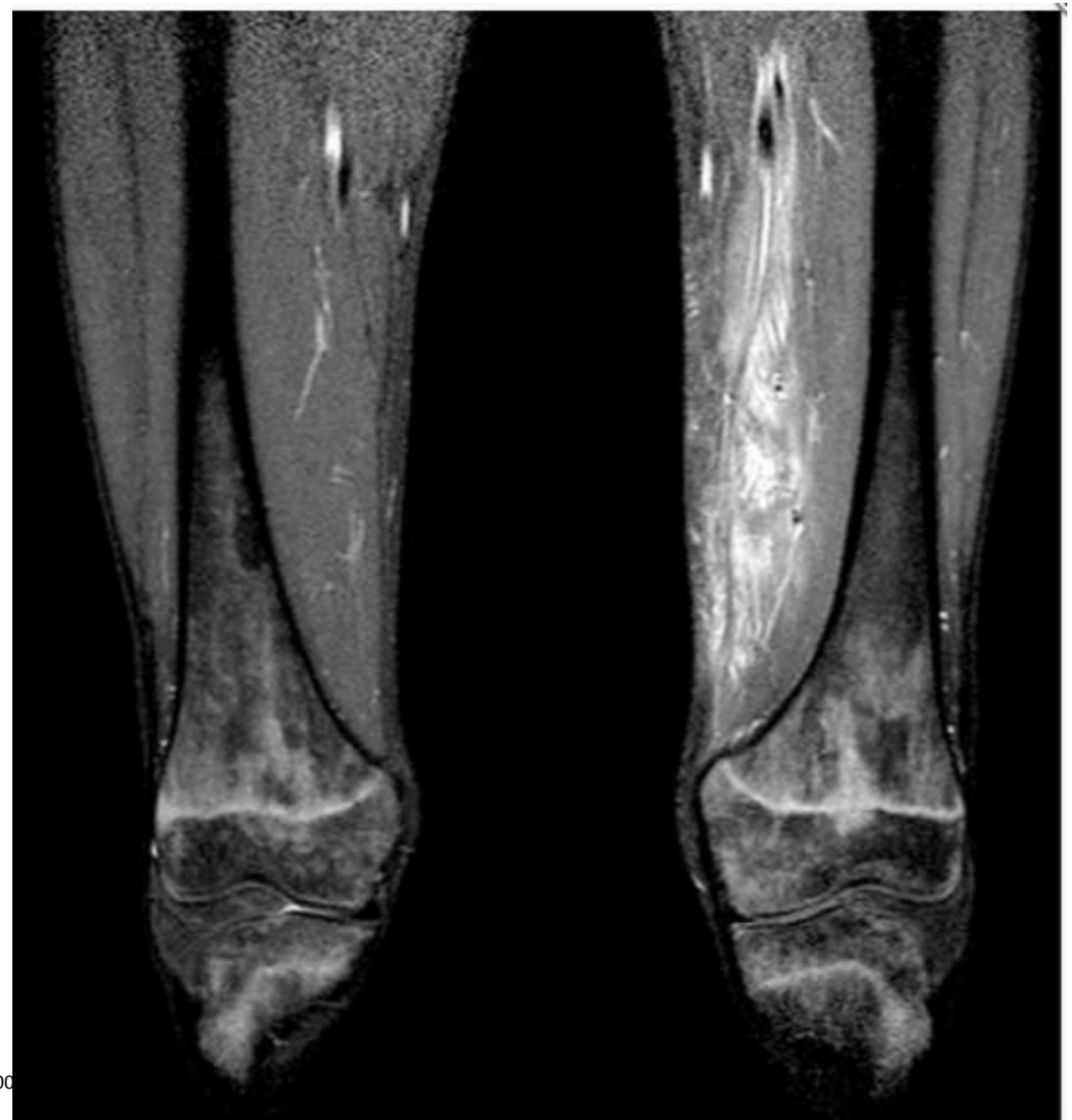
- • *Early pyomyositis is difficult to distinguish from other Dx (thrombophlebitis, muscle haematoma, muscle rupture, fever of unknown origin osteomyelitis).*
- Iliacus pyomyositis may mimic septic arthritis of the hip, and iliopsoas pyomyositis may mimic appendicitis.
  - Imaging—
- MRI is gold standard technique (may show muscle enhancement and intramuscular abscesses- see next).
- CT (may detect muscle swelling and well defined abscesses).
- Ultrasound can be helpful for Dx and Rx
  - Microbiology—
    - diagnostic aspirates before starting Abx to get a specific culture
    - BCs are only positive in 10% of tropical cases and 35% of temperate cases!

the muscle will not yield purulent material. Because of unspecific symptoms, pyomyositis would not be usually the initial diagnosis



https://www.researchgate.net/figure/Stage-1-of-pyomyositis-is-characterized-by-local-muscle-pain-swelling-and-erythaema_fig1_6178145

Pyomyositis inner thigh in young patients with severe aplastic anemia (E.coli)



- http://www.elsevier.es/ficheros/publicaciones/21735743/0000

Management

- Antibiotics— stage 1 antibiotics alone
- HOWEVER, most patients present with stage 2/3 disease and require antibiotics+ drainage.
- Empiric therapy for these stages:

Directed against *S. aureus and streptococci* (flucloxacillin or vancomycin – if MRSA is suspected or there is a risk of MRSA).

- *Immunocompromised patients →broader Abx such as piperacillin–tazobactam ± vancomycin.*
- Once culture is out → Tailored Abx for 3-4 weeks

- Drainage—percutaneous drainage Dx and Rx (drainage and send drain sample for Micro)

This may be CT-guided or ultrasound-guided.

Septic Arthritis

Defined as : An **inflammatory reaction** of the joint space caused by an infectious agent.

→ Usually caused by bacteria but may be caused by mycobacteria or fungi.

→ Very common and hard to treat due to use of prosthetic joints (2-10% of all prosthetic joints!)

→ Also common among immune compromised and eldery (45% of ppl with Septic arthritis are above 65 years and 56% are male)

Etiology:

- • *S. aureus* : commonest cause overall, especially in acute cases, the increase in incidence here matches that of increase use of Prosthetic joints.
- • Streptococci → groups A, B, C, and G streptococci, + *S. pneumoniae* and viridans groups (20%) of all cases.
- • CoNS.
- • *E. coli*.
- • *H. influenzae.*
- • *N. gonorrhoeae* (the commonest cause in sexually active young adults)
- • *N. meningitidis*.
- • *P. aeruginosa*.(sternoclavicular joints, sacroilliac joints)
- • *Salmonella* spp.
- + other causes, brucella, polymicrobial.

Prosthetic join infection

- According to presentation:

- Acute (*S. aureus*) within 3 months
- Subacute within 3-24 months
- Chronic >24 months

- Overall *S.aureus*, but CoNS and G-ve aerobes cause the delayed cases more

Epidemiology

- The reported incidence of septic arthritis varies from two to five cases per 100000 population or 8–27% of adults presenting with painful joints (20,000 cases /year in US)

- Risk factors for septic arthritis include
- Age >80 years,
- Diabetes mellitus
- Rheumatoid arthritis
- Prosthetic joint
- Recent joint surgery
- Skin infection/ulcers
- Intra-articular corticosteroid infection

injection drug use, and alcoholism.

Pathogenesis

- Septic arthritis usually occurs after haematogenous seeding of pathogenic bacteria- this is the most common route.
- But like osteomyelitis contiguous spread or direct inoculation can also be causes.
- Healthy synovial cells have phagocytic activity and normally able to clear any seeding from outside sources.
- Any weakness to immune system (SLE, Rhuematoic arthritis..etc) increases risk (hence old age!)

- Previously damaged joints are most susceptible to infection (arthritis)
- These joints show neovascularization and and adhesion factors, which promote bacteremia and consequent infection.
- *S. aureus* especially, binds to articular sialoprotein, collagen, elastic and prosthetic materials visa tissue adhesion factors that they possess.
- Infection typically damages the cartilage (chondrocyte proteases of *S. aureus* , the inflammation in turn causes further damage to the cartilage)
- Gonococcal arthritis exhibits much less influx of WBC into the joint, which explains why it is **not as destructive** to joints as other bacteria.

Clinical features

- • Children and adults with acute septic arthritis usually present with fever (60–80%) and monoarticular involvement (90%).
- • The knee is the most commonly affected joint, followed by the hip.
- Clinical features include pain, swelling, and reduced mobility in the joint.
- • Polyarticular infections occur in 10–20% of patients, especially those with rheumatoid arthritis and viral causes.
- • Infections with mycobacteria or fungi usually have an insidious onset.

Diagnosis

- Laboratory investigations frequently show a raised WCC and inflammatory markers.
- Joint aspiration shows purulent synovial fluid, with an elevated WCC (50 000–100 000 cells/mm3), mostly neutrophils.
- Gram stain is positive in 29–50%, and culture is positive in 80–90% of cases (synovial fluids in blood culture bottles may improve yield)
- Samples should also be sent for microscopy for crystals. BCs are positive in 75% of cases.

Imaging

- Radiographs of the affected joint may be normal at presentation.
- Typical changes are periarticular soft tissue swelling, fat pad edema, periarticular osteoporosis, loss of joint space, periosteal reactions, erosions, and loss of subchondral bone.
- Ultrasound can be used to confirm an effusion and guide aspiration.
- CT and MRI are highly sensitive for imaging early septic arthritis. CT is better for imaging bone lesions.
- MRI may not distinguish septic arthritis from inflammatory arthropathies.

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CLINICAL HISTORY

43-year-old female with a history of lupus treated with steroids, presents with developing left knee pain, swelling, and fevers.

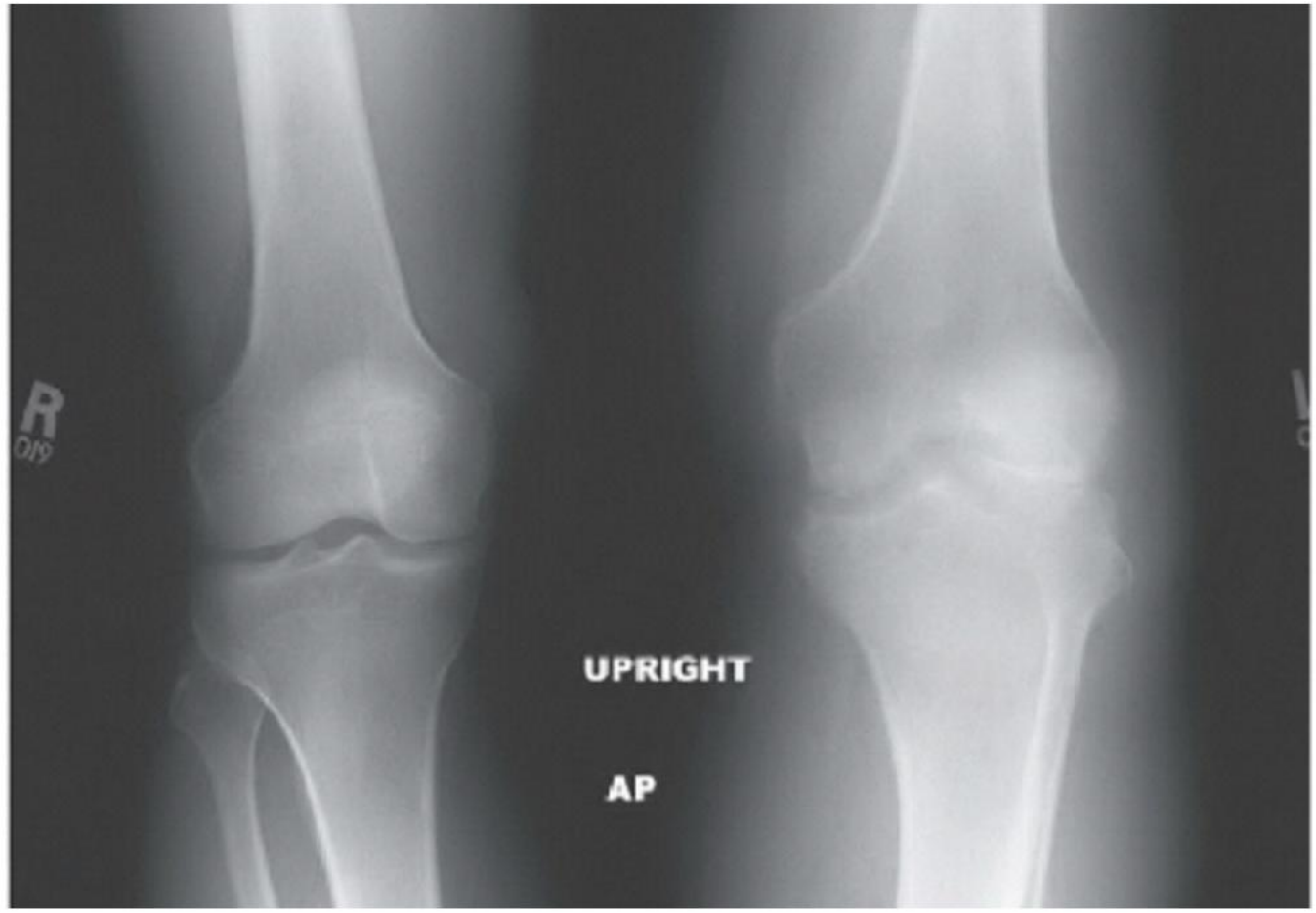

FIGURE 88A

Management

- • Drainage of the joint, either by closed aspiration or arthroscopic washout, should be performed **urgently.**

- Open drainage may be required either when repeated drainage has failed to control the infection or for drainage of hip joints.

- Prosthetic joint infections often require removal of the prosthesis.

Antimicrobial therapy

According to the initial Gram stain findings.

Empirically - IV piperacillin–tazobactam ± vancomycin.

- Definitive therapy is tailored to culture and sensitivity results
- • Adjunctive therapy with a short-course systemic corticosteroid treatment has been shown to be of benefit in children with haematogenous bacterial arthritis.

End