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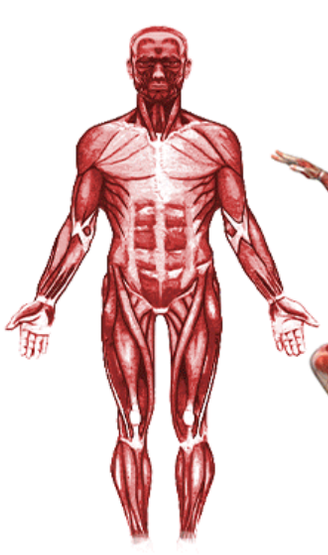

# MSS

Musculoskeletal System

# Microbiology

**Doctor 2018 | Medicine | JU**


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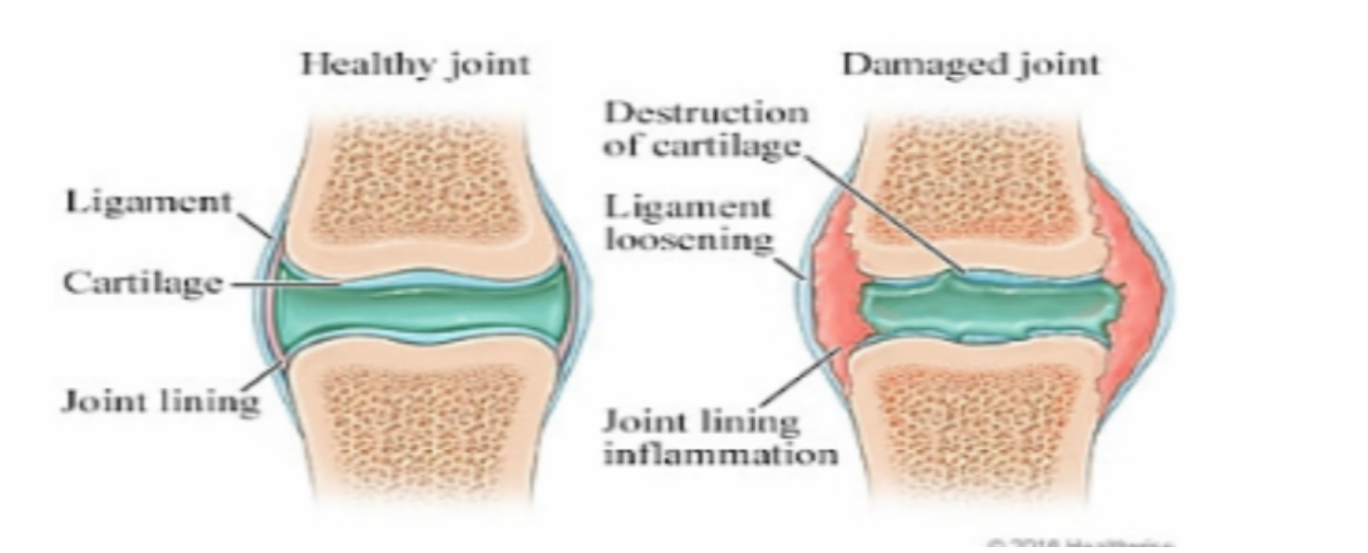
Septic arthritis: An inflammatory reaction of the joint space caused by an infectious agent.
It is usually caused by bacteria but may be caused by mycobacteria or fungi.

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

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




**Etiology:** **Septic arthritis** can be caused by bacterial, viral or fungal infections. Bacterial infection with Staphylococcus aureus (staph) is the most common **cause**. ... **Septic arthritis** can develop when an infection, such as a skin infection or urinary tract infection, spreads through your bloodstream to a joint.

• Strept• *S. aureus* (commonest cause).
ococci, e.g. groups A, B, C, and G streptococci, *S. pneumoniae*.
• CoNS.
• *E. coli*.
• *H. influenzae*.
• *N. gonorrhoeae*.
• *N. meningitidis*.
• *P. aeruginosa*.
• *Salmonella* spp.
• Others, e.g. *P. multocida, C. canimorsis, E. corrodens, S. moniliformis, Brucella* spp., *B. pseudomallei, Clostridium* spp.
• Polymicrobial infections.

**Epidemiology:** If patients have underlying **joint** disease, or prosthetic joints, the incidence increases

Risk factors for septic arthritis include **age** >80 years(the older you are the more likely to have it, because your ability to tolerate the seeding bacteria is less and less)
diabetes mellitus, rheumatoid arthritis, prosthetic joint, recent joint surgery, skin infection/ ulcers,
intra- articular corticosteroid infection(steroid inside the joint can introduce bacteria)
injection drug use, and alcoholism.

All of them you can reduce them because are overuse the joint or an inflammatory condition that is infecting the joint

**Pathogenesis:**

•Septic arthritis usually occurs after hematogenous seeding of pathogenic microorganisms(the most common) but may occur via direct inoculation, e.g. injection, surgery, or trauma.

contiguous spread can also be causes

Why hematogenous??
Joint after occurring the damage it will tried to repair itself ,the way it repairs itself is that neovascularization(new blood formation) this new blood route is the route of bacteria that use to get access into the joint.

•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.

#joint with arthritis is more susceptible to septic arthritis.

# Clinical features

•• Children and adults with acute septic arthritis usually present with **fever** (60–80%) and monoarticular involvement (only one joint is involved)(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 .

**CLINICAL HISTORY**

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

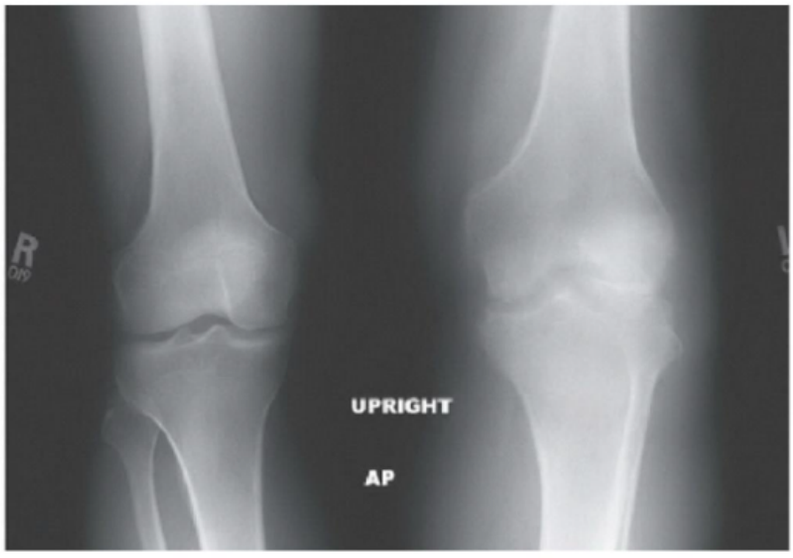



FIGURE 88A

Notes about the picture:

1)synovial fluid cloudy in the joint space .

2)the subtissue is very swelling .

## Management

•• Drainage of the joint, either by closed aspiration or arthroscopic washout, should be performed **urgently.** •open drainage( insert a tube and keep the tube in while keeping drainage out the fluid) 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

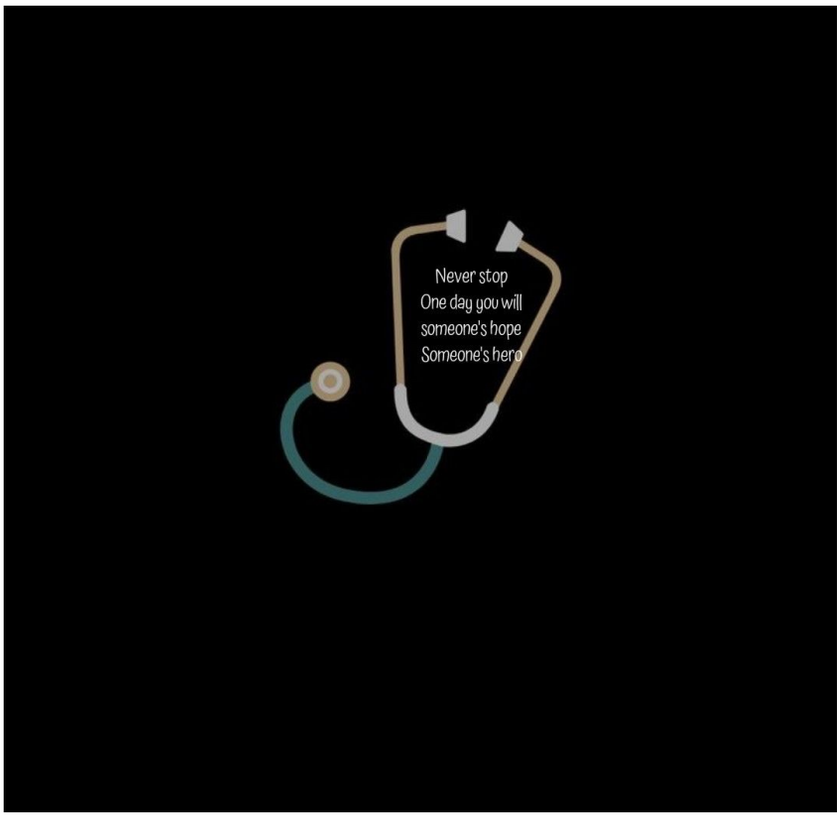
According to the initial Gram stain findings.
Empirically - IV piperacillin–tazobactam ± vancomycin.
•Definitive therapy is tailored to culture and sensitivity results

•• Adjunctive therapy with a short-course systemic corticosteroid treatment has been shown to be of benefit in children with haematogenous bacterial arthritis.

Useful video:
https://www.orthoatlanta.com/videos/septic-arthritis-of-the-knee#vm_A_9e1c8386

https://www.youtube.com/watch?v=498Px-BBdOo

Good luck



## 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 (systemic infections 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.
- Usually, it is not the virus itself invading the muscle tissue and causing myalgia. The cause of pain is the systematic inflammatory response to an intracellular pathogen.

Why systemic? Because muscles are well perfused tissues, so viremia is likely to cause inflammation at the muscle and therefore pain. Most individuals complain of muscle pain at their thighs or back.

- The same applies to systemic intracellular bacterial infections
- Myalgia differs from **myositis**, which is a localized infection to the muscle.
- **Acute rhabdomyolysis** (breakdown of damaged muscle) predictably occurs with clostridial and streptococcal myositis, as both these organisms 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! In other words, there may be fasciitis that may cause necrosis in the muscle without the bacteria actually infecting the muscle. This happens when the bacteria in the fascia is causing significant necrosis that spreads to surrounding tissues, including muscles.
- As can be seen, myonecrosis has two forms:
  - From necrotizing fasciitis that progresses into the muscle.
  - Necrotizing Myositis

## Pyomyositis

- Pyomyositis, a pus forming (purulent) infection of skeletal muscle tissue, is usually due to **S. aureus** (remember it is the typical pus former in skin), especially those that have PVL toxin.
- Abscess formation is the usual consequence when these pyogenic bacteria reach the muscle tissue.
- 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.

- Pyomyositis infection usually remains localized, and shock does not develop unless organisms produce
  i. Toxic shock syndrome toxin 1 - acts as a super antigen that causes exaggerated immune response that is many folds the normal response -> shock.
  ii. 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 usually arises from hematogenous spread (deeper infections, muscle and bone, are typically more related with hematogenous spread rather direct inoculation).

Epidemiology of Pyomyositis

- There are two main scenarios:
  1. In tropical climates:
     - Occurs more in males than females, and in two main age groups with no significant medical history:
       - children (aged 2–5 years)
       - adults (aged 20–45 years)
     - There is direct inoculation from the blood into the muscle.
     - Somehow this happens with high temperature and humidity, but we don't know why.
  2. In temperate climates:
     - 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), and immunosuppressive therapy.
     - Other risk factors include trauma, IDU (Injection drug use), and concurrent infections (toxocariasis caused by roundworms, VZV)
     - Typically, patients have past medical history, which allows bacteria to reach the blood. With trauma, some muscle necrosis may occur. Together, this will allow the bacteria to seed into the muscle. Then, three phases will occur (as discussed below in the "Clinical Stages" section).

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 hematological malignancy.
- Uncommon causes are B, C, and G streptococci, 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

- In between 20% and 50% of cases patients 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 it is not limited to that area and can affect any muscle group.
- Multifocal infection occurs in up to 20% of cases! (multifocal = more than one location)
- Since it is usually from a hematologic cause, the patient must be assessed for complications of bacteremia (endocarditis).

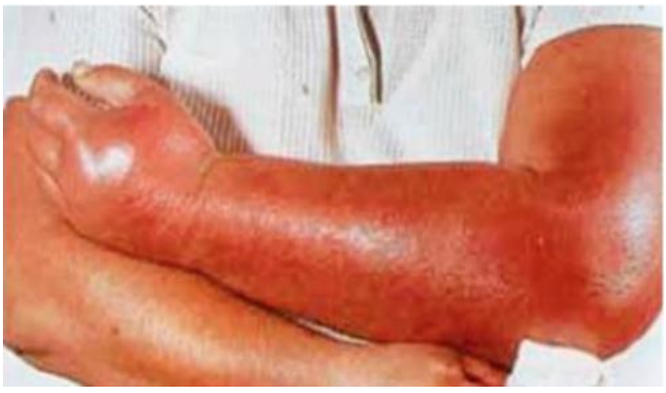
Clinical Stages

- Stage 1 (early invasive stage)
  - crampy local muscle pain, swelling, and low-grade fever. Induration (hardening) of the affected muscle + leukocytosis may be present.
  - The bacteria is everywhere in the area of trauma, and therefore causes non-specific symptoms.
- Stage 2 (suppurative stage)
  - at 10–21 days after onset of symptoms (most patients present at this stage). Fever, very sharp muscle tenderness and swelling. An abscess may be clinically apparent, aspiration of which yields pus. There is marked leukocytosis.
  - After 10 days, the abscess starts to form, and at the same spot, the patient complains of pain. There is also significant inflammation in the area. Since we are discussing skeletal muscle, this occurs in the upper or lower limb.
  - If phase 2 progresses into the system, it reaches phases 3.
- Stage 3 (systemic stage)
  - The affected muscle is fluctuant. Patients may present with complications of S. aureus bacteremia, e.g. septic shock, endocarditis, septic emboli, pneumonia,

pericarditis, septic arthritis, brain abscess, and ARF (acute renal failure). Rhabdomyolysis may occur

Diagnosis

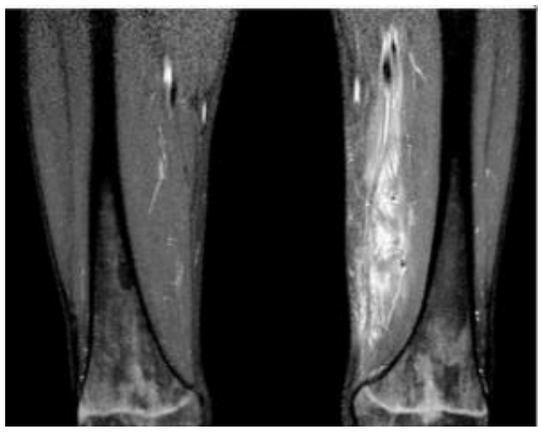
- Early pyomyositis is difficult to distinguish from other possible diagnoses (thrombophlebitis, muscle hematoma, muscle rupture, fever of unknown origin osteomyelitis).
- Iliacus pyomyositis may mimic septic arthritis of the hip, and iliopsoas pyomyositis may mimic appendicitis.
- Imaging:
  - MRI is the 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
  - Blood cultures are only positive in 10% of tropical cases and 35% of temperate cases!



Seeing this, you might assume the patient has cellulitis or some other infection, as it is still in the early stage. This is where an MRI is useful.

Pyomyositis in the inner thigh in a young patient with severe aplastic anemia (E. coli)

The white area is where all of the bacteria has been sequestered. The abscess spans along the side of this skeletal muscle.



Management

- Antibiotics— Stage 1 use antibiotics alone
- HOWEVER, most patients present with stage 2 or 3 of the disease and require antibiotics and 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). The drainage may be CT-guided or ultrasound-guided.