Micro 6+7 summary

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Osteomyelitis: an infection of bone that leads to tissue destruction and formation of sequestrum

- Bacteria are the predominant cause followed by fungi, mycobacteria and viruses.
- Management is tailored for each individual, depending on many factors like causative agent and host status,…
- Identified as a **spectrum,** ranging from localized to extensive (e.g. tibial, vertebral).

Three routes: Hematogenous, Contiguous, Traumatic/ surgical.

Collection of exudates in bone marrow → increased medullary pressure → extension to bone cortex → rupture through periosteum → blood supply interrupted → necrosis and sequestrum formation → new bone formation (involucrum).

Causative organism of osteomyelitis	Comments
S.Aureus	It's the most common (>50% of the cases, along with CoNS) , most aggressive and invasive. Metastatic foci with bacteremia
Coagulase negative staphylococci (CoNS)	Foreign bodies (Prosthetics), biofilm formation
Streptoccocci	Spread rapidly through soft tissues
Enterobacteriacaea (Ecoli,klebsiella)	Variation in Abx sensitivity + Resistance to antibiotics during therapy.
P.Aeruginosa	Resistance + successor to other bacteria when initial therapy fails
Anaerobics	Mixed with aerobic, synergistic, depend on devitalized tissue.
Brucella	Unpasteurized milk
Fungi	Candida is most likely
M.Tuberculosis	Vertebral osteomyelitis, in countries with poor resources.
Viruses	ASSOCIATED with varicella and variola

Classification systems:

1. lee and Waldvogel: acute or chronic, hematogenous or contiguous, with/without vascular compromise.
2. Cierny and mader system: for long bone osteomyelitis (affected portion of bone, status of host).

Anatomical types: stage1: medullary osteomyelitis

stage2: superficial

stage3: localized

stage4: diffuse

Physiological classes: A=normal host

B= host with local (BL) or systemic (BS) compromise

C= treatment worse than disease

Etiology: hematogenous → monomicrobial

contiguous → mono or polymicrobial

Bacteremia is a frequent cause (no wound)

Prosthetic joints → S.aureus bacteremia → metastatic osteomyelitis

Urinary tract → Ecoli, klebsiella bacteremia → vertebral osteomyelitis

Epidemiology: diabetic foot → 30-40% risk to develop osteomyelitis

More aging population → more diabetes and obesity→ high frequency of osteomyelitis

Rich countries → orthopedic related osteomyelitis

Poor countries → TB and brucella related

Pathogenesis: most common predisposing factor → devitalized tissue (less blood supply, less O_2, hindered venous and lymph outflow)

Diabetes, immunocompromise → risk factors

S.aureus: 1. has enzymes and toxins to destruct the bone.

2. can evade immune defenses (survive inside macrophages).

3. two populations: intra and extra cellular, intracellular replenishes extracellular bacteria.

4. can remain dormant (can't be killed by antibiotics).

CoNS: use biofilms on prosthetics to protect them from the immune system

In patients with sinuses or entry wounds, the superficial flora may not represent the true pathogen. So common skin flora are not typically targeted with antibiotics.

Clinical features: in pediatrics → hematogenous spread

in adults → subacute or chronic

Onset of pain.

Presentation of chronic → local signs inflammation, sinus tract, fractures

Non-healing ulcers can indicate osteomyelitis.

Dx: clinical and confirmed by radiology, inflammatory markers are elevated

X-ray → elevated periosteum

CT, MRI → investigations of choice

Needle aspiration → Dx and Rx

Biopsy during surgery with debridement → Dx and Rx

Stop antibiotics 2 days before biopsy

PCR

Rx: debridement + antibiotics IV (4-6 weeks)

s.Aureus → clindamycin, nafcillin, cephalosporins,…

MRSA → vancomycin, …

Streptococci → penicillins

Enterococci → penicillin + gentamicin

Enterobacteriacae → ciprofloxacin, ceftriaxone,..

p. Aeruginosa → ciprofloxacin

Complications: Sinus formation, fractures, sepsis, tumors (squamous cell carcinoma, sarcoma)

Prognosis:

Vertebral, immunocompromised, late Dx, → poorer prognosis

Local, early Rx → better prognosis

Prevention: Better pre-operative measures; Use mupirocin and chlorhexidine, Early Dx and Rx

Lec. 7: septic arthritis:

Inflammatory reaction of the joint space caused by an infectious agent.

Very common and hard to treat (prosthetic joints)

In immunocompromised, elderly (>50% males)

Most common → S.aureus

Sterptococci, CoNS, Ecoli, N.gonorrhoeae (commonest in sexually active young adults).

Types: 1. Acute (s.aureus) within 3 months

2. Subacute within 3-24 months

3. Chronic >24 months

8-27% of adults → painful joints

Risk factors:

>80 years, diabetes, rheumatoid arthritis, prosthetic joint, surgery, skin infections, injection drug use,…

Pathogenesis: hematogenous (commonest), contiguous, direct inoculation.

Healthy synovial cells have phagocytic activity (up to a limit).

Previously damaged joints → neovascularization + adhesion factors → bacteremia.

S.Aureus bind to articular sialoprotein and collage via adhesion factors they have.

Gonococcal arthritis → less WBC → less destructive

Clinical features:

Acute → fever and monoarticular involvement

Knee is most commonly affected, then hip

Pain, swelling, reduced mobility

Rheumatoid arthritis and viral causes → polyarticular

Mycobacteria, fungi → insidious onset

Dx:

elevated WBC (mostly neutrophils) and inflammatory markers

Aspiration → purulent fluid

Gram stain is positive in almost half of the cases, and cultures 90% positive

Radiography: periarticular soft tissue swelling, loss of joint space, inflammation,..

CT and MRI highly sensitive, may not distinguish from inflammatory arthropathies.

Management: **urgent** drainage + antimicrobials

Prosthetic joint infection may require removal of prosthesis.

Antibiotics: empiric: IV piperacillin tazobactam

definitive: according to culture and sensitivity results

Adjunctive therapy with steroids → children with hematogenous bacterial arthritis.