MSS INFECTION

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Q1. How it got there?

**Direct**: skin puncture, open fracture, operation.

**Systemic**: via hematogenic or lymphatic spread.

Q2. How it differ from soft tissue infection?

**First**: rigid compartment -> pressure build up more susceptible to vascular damage and cell death.

**Second**: honeycomb of inaccessible spaces make it hard to eradicate.
Q3. Risk factor??

Host risk factor:

local:

- trauma.
- poor circulation.
- foreign body; biofilm "glycocalyx".

Systemic

malnutrition, DM, rheumatoid, corticosteroid and immunosuppressant in addition to age "extreme of age"
Q4. Forms of infection?

Acute pyogenic:
- pus formation" leukocytes, tissue derbies, dead bacteria" .
- acute systemic manifestations " septicaemia and bactermia“ .

Chronic infection:
- Fibrosis or granulomas "lymphocyes, modified macrophages and multi-nucleated giant cell“.
- less systemic effect actually but ultimately disabling "lymphadenopathy, splenomegaly and tissue wasting“.

INTRODUCTION
Q5. Treatment?

Treatment outlines

- Initiate antibiotic or chemotherapy
- Evacuate the pus and remove dead tissue
- Stabilize the bone in case of fracture
- Maintain soft tissue and skin cover "regular dressing"
- Supportive measures and analgesia
**Acute osteomyelitis**

- **Osteomyelitis**: inflammation of the bone or bone marrow, typically result from infection.

- Almost invariably occurs in children over the age of 4 years because of rich blood supply to the growing bones, when adults are affected it may be because of lowered resistance or local trauma.

- In children, the vascular metaphysis of a long bone are usually affected, most often at the proximal or distal end of the femur or the proximal end of the tibia.

- In adults, the vertebrae and the pelvis are most commonly affected.
It will start in the metaphysis because of:
1. Defective phagocytosis in metaphysis (inherently depleted RES).
2. Rich blood supply.
4. Metaphyseal hemorrhage from repeated trauma (culture media).

Risk factors include:
- Diabetes mellitus
- Infected umbilical cord newborn
- Steroid use
- Iv drug abusers
- Urinary catheterization, m.c. source in adult
- Splenectomy
- Trauma to the area
- Etc.
EPIDEMIOLOGY

- The overall prevalence is 1 case per 5,000 children.

- The incidence of vertebral osteomyelitis is approximately 2.4 cases per 100,000 population.

- The overall incidence is higher in developing countries.

- No increased incidence of osteomyelitis is noted based on race.

- Males are at increased relative risk, which increases through childhood, peaking in adolescence and falling to a low ratio in adults.

- Osteomyelitis has a bimodal age distribution:
  Acute hematogenous osteomyelitis is primarily a disease in children. Vertebral osteomyelitis is more common in persons older than 45 years.
The causal organism is usually Staphylococcus aureus, less often Streptococcus pyogenes or S. pneumoniae.
Microorganisms reach bone by one of the following:

A. **Hematogeneous spread** in which blood stream is invaded, M.C.
B. **Contagious spread** eg, cellulitis.
C. **Penetrating trauma** including iatrogenic causes.

Once reaching the bone it will pass through 5 stages:

1. **Inflammation**
2. **Suppuration**
3. **Necrosis**
4. **New bone formation**
5. **Resolution or intractable chronicity**
The earliest change is an acute inflammatory reaction. The **intraosseous pressure rises**, causing intense **pain** and **obstruction of blood flow**.
SUPPURATION

By the second day pus appears in the medulla and forces its way along the Volkmann canals to the surface, where it forms a subperiosteal abscess.

It then spreads along the shaft, to re-enter the bone at another level, or bursts out into the soft tissues.
By the end of the 1st week there is evidence of necrosis forming a piece dead bone “sequestra”.

Necrosis result from vascular compromise that occurs due to rising intraosseous pressure, vascular stasis, infective thrombosis and periosteal stripping.
New bone forms from the deep layer of the periosteum.

With time the new bone thickens to form **involucrum** enclosing the infected tissue and sequestra.
The bone around the zone of infection becomes increasingly dense; this, together with the periosteal reaction, may result in overall thickening of the bone.
CLINICAL FEATURES

- **Severe pain, malaise and a fever**; in neglected cases toxemia may be marked.
- In **adults**, suspicious features are **backache** since it mostly affect vertebrae and a mild fever, possibly following a urological procedure.
- In **children**, **Irritability or lethargy**.
  Damage to the physis may eventually lead to **growth retardation**.

**Examination:**

- ‘**fingertip tenderness**’ near one of the larger joints.
- Painful and restricted joint movement (**pseudoparalysis**).
- **Deformity** at that site as it may spread to the epiphysis and then to the joints.
- Local redness, swelling, warmth and edema as later signs and signify the presence of pus.
LABORATORY INVESTIGATION:

- **White blood cell (WBC) count and erythrocyte sedimentation rate (ESR).**
- **Blood cultures** may be positive.
- **Aspiration**: pus from the subperiosteal abscess or the adjacent joint. Most certain way to confirm the clinical diagnosis.
- Blood C-reactive protein (CRP) is a sensitive marker for monitoring progress during the course of treatment.
**DIAGNOSIS**

**Imaging Study:**

**X-ray:**
- For the first 10 days, plain x-rays show no abnormality.
- By the end of the second week:
  - Refraction of the metaphysis and periosteal new bone formation.
  - Bone may appear increasingly ragged if tx is delayed or ineffective.
  - Sclerosis and thickening of the cortex that occurs with healing.
  - Sometimes sequestra are seen, separated from the surrounding bone.

**MRI:**
- Show pathological changes before x-rays.
- Can help to distinguish between bone and soft-tissue infection.

**Bone Scan**
X-ray:
Bone Scan

MRI:

A

B

Sequestrum

Involucrum

Cloaca
# Treatment

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Common pathogens</th>
<th>Treatment of choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates and infants up to 6 months of age</td>
<td>penicillin-resistant S. aureus, Group B streptococcus and Gram-negative organisms</td>
<td>flucloxacillin plus a third generation cephalosporin like cefotaxime.</td>
</tr>
<tr>
<td>Children 6 months to 6 years of age</td>
<td>H. influenza</td>
<td>combination of intravenous flucloxacillin and cefotaxime or cefuroxime</td>
</tr>
<tr>
<td>Older children and previously fit adults</td>
<td>staphylococcus species</td>
<td>intravenous flucloxacillin and fusidic acid</td>
</tr>
<tr>
<td>Elderly and previously unfit patients</td>
<td>Gram-negative infections</td>
<td>combination of flucloxacillin and a second- or third-generation cephalosporin</td>
</tr>
<tr>
<td>Patients with sickle-cell disease</td>
<td>Salmonella</td>
<td>Chloramphenicol, risk of aplastic anemia. Replaced by 3rd generation cephalosporine of fluoroquinolone</td>
</tr>
<tr>
<td>Heroin addicts and immuncompromised patient</td>
<td>Pseudomonas or Proteus</td>
<td>third generation cephalosporins or a fluoroquinolone preparation</td>
</tr>
<tr>
<td>Patients at risk of (MRSA)</td>
<td>-</td>
<td>intravenous vancomycin (or similar antibiotic) together with a third-generation cephalosporin.</td>
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</table>
Antibiotic

- The drugs should be administered intravenously.

- Patients 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 fitness.

- Thereafter, the antibiotic should still be administered orally for at least another 3–6 weeks.

- Treatment is discontinued only when minimum inhibitory concentration (MIC) is maintained. CRP, ESR and WBC values are seen to back to normal.
Supportive treatment:
Continuous bed rest is important. Osteomyelitis is extremely painful; the affected limb is splinted and adequate analgesics must be given.

Drainage
Open operation under general anaesthesia, If:
1. The clinical features do not improve within 36 hours of starting tx.
2. There are signs of deep pus (swelling, edema, fluctuation).
3. Most certainly if pus is aspirated.

About one-third of patients with confirmed osteomyelitis are likely to need an operation; adults with vertebral infection seldom do.
FOLLOW UP

- Once the signs of infection subside, movements are encouraged.
- Full weight bearing is usually possible after 3–4 weeks.
- Outpatient follow-up is important, to ensure that there is no recurrence of infection.
DIFFERENTIAL DIAGNOSIS

- **Cellulitis**: this is often mistaken for osteomyelitis. There is widespread redness and lymphangitis. A source of skin infection may not be obvious and should be searched for (e.g. between the toes). If doubt remains about the diagnosis, **MRI** will help to distinguish between bone infection and soft-tissue infection.

- **Sickle-cell crisis**: patients with sickle-cell disease may present with features like those of acute osteomyelitis. Where **Salmonella** is endemic it would be wise to treat these patients with suitable antibiotics until infection is definitely excluded.
**Complication**

- **Spread**: infection may spread to the joint (septic arthritis) or to other bones (metastatic osteomyelitis).

- **Pathological fracture**: occasionally the bone is so weakened that it fractures at the site of infection or operative perforation.

- **Growth disturbance**: if the physis is damaged there may later be shortening or deformity.

- **Persistent infection**: treatment must be prompt and effective. ‘Too little or too late’ may result in chronic osteomyelitis.

- **Vascular complication**: 30% of pediatric patients with long-bone osteomyelitis may develop deep venous thrombosis (DVT).
Sub acute haematogenous osteomyelitis

- Chronic low-grade infection of bone characterized by a lack of systemic manifestations.
  
  because the organism is less virulent or the patient more resistant.

- Characterized by mild to moderate pain often nocturnal, usually described as a persistent ache; intermittent symptoms; insidious onset; and, often, a long delay between the onset of pain (the most common presenting symptom) and the diagnosis. Usually, symptoms are present for 2 weeks or longer.

- The course is generally marked by few or no constitutional symptoms and no known previous acute disease. A systemic reaction is absent, and supportive laboratory data are inconsistent.
The distal femur and the proximal and distal tibia are the favorite sites.

The patient is usually a child or adolescent.

The most common organism cultured from a sub acute osteomyelitis is a *staphylococcus* species.
Clinical Features

History

- Mild-to-moderate localized pain frequently exacerbated following a period of unusual activity.
- Night pain that is relieved with aspirin is frequently reported.
- Minimal loss of function is another common symptom (e.g., limping in a patient with a lower-limb lesion).

Examination

- Localized tenderness associated with warmth, redness, and soft-tissue swelling with the involvement of subcutaneous bone.
- Pain may occur with movement of the adjacent joint, and some joint effusion may be present, but the pain and effusion are usually mild.
- The surrounding muscles may occasionally demonstrate some wasting.
LABORATORY STUDIES:

- The (WBC) count is usually within the reference range or occasionally slightly elevated, with a normal differential.

- The (ESR) and (CRP) measurements are usually mildly elevated, but they may be within the reference ranges in 30-50% of patients.

- Blood culture results are usually negative.
DIAGNOSIS

IMAGING STUDIES

- **X-ray**: small, oval cavity surrounded by sclerotic bone “the classic Brodie’s abscess”.

- **Radio-isotope scan**: will show increased activity.

- **CT**: detect lesions in difficult anatomic locations that could not be seen with plain radiography (e.g., the pelvis and sacrum).

- **MRI**: most sensitive investigation in the evaluation of bone marrow pathology. Signal intensity is decreased on T1-weighted images whereas it is increased on T2-weighted images with a rim of decreased intensity due to sclerotic bone.
X-ray
Bone scan

CT
Gledhill's classification include seven forms on the basis of morphology, location, and similarity to neoplasms.

**Type I (metaphyseal lesion)**

- **Type Ia** is a *central* metaphyseal lesion that is seen as a punched-out radiolucency.
  - Langerhans cell histiocytosis

- **Type Ib** is a metaphyseal lesion *eccentrically* located with cortical erosion.
  - Osteogenic sarcoma
Type II (diaphyseal lesion)

- Type IIa is a localized **cortical** and periosteal reaction.
  - osteoid osteoma

- type IIb lesion is a **medullary** abscess in the diaphysis without cortical destruction
  - Ewing sarcoma
**Type III (epiphyseal lesion)**

- Type IIIa is a primary *epiphyseal* osteomyelitis and appears as a concentric radiolucency.

- Type IIIb is a sub acute infection that crosses the epiphysis and involves both the epiphysis and metaphysis.
Type IV (metaphyseal-equivalent lesion, defined as the portion of a flat or irregular bone that borders cartilage).

- Type IVa involves the vertebral body with an erosive or destructive process.

- Type IVb involves the flat bones of the pelvis and is mostly sclerotic, with neither erosion nor destructive processes.

- Type IVc involves the small bones (e.g., tarsal bones, clavicle).
TREATMENT

Conservative

- If the diagnosis is not in doubt.
  - Immobilization
  - **Antibiotics** (flucloxacillin and fusidic acid) *intravenously* for 4 or 5 days and then *orally* for another 6 weeks.

- Often result in healing, though this may be slow.

Operative

- If the x-ray shows that there is no healing after conservative treatment.
  - **Open curettage** may be indicated.
  - This is always followed by a further course of antibiotics.
Post-traumatic osteomyelitis

- **Open fractures** are always contaminated and are therefore prone to infection.
- More commonly affects **adults** and typically occurs in the **tibia**.
- The most commonly isolated organism is **S aureus**, but other organisms such as Escherichia coli, Proteus mirabilis and Pseudomonas aeruginosa are sometimes involved.
- Local soft-tissue vascularity may be compromised, leading to interference with healing.

- Posttraumatic infection begins outside the bony cortex and works its way in toward the medullary canal.

- Soft tissue damage may lead to a greater risk of recurrence.
CLINICAL FEATURES

- Fever.

- Pain and swelling over the fracture site.

- The wound is inflamed and there may be a seropurulent discharge.
Blood tests reveal increased CRP levels, leukocytosis and an elevated ESR.

- These inflammatory markers are non-specific and may be affected by tissue trauma.

X-ray appearances may be difficult to interpret because of bone fragmentation.

MRI can be helpful in differentiating between bone and soft tissue infection, but is less reliable in distinguishing between long-standing infection and bone destruction due to trauma.

A wound swab is obtained for microbiological examination and (if necessary) to test for antibiotic sensitivity.
TREATMENT

- Management of open fractures

- In most cases a combination of flucloxacillin and benzylpenicillin (or sodium fusidate), given 6-hourly for 48 hours

- If the wound is contaminated, it is advisable also to give metronidazole for 4 or 5 days to control both aerobic and anaerobic organisms.
END OF LECTURE

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