Osteomyelitis / Bone and Joint Infections

Bone infections in children are usually from haematogenous bacterial seeding to a single joint, usually the lower limbs, but may be multifocal. Approximately 10% cases are due to spread from adjacent infection or penetrating injury. Most cases of septic arthritis are monoarticular (hip, knee, ankle or elbow), but in 10% of cases multiple joints may be involved. Occasionally osteomyelitis and septic arthritis can co-exist. This occurs when the metaphysis is intra-articular with a thin periosteum (such as the hip, shoulder, ankle and wrist). Vascular connections between the metaphysis & epiphysis make infants with osteomyelitis particularly prone to arthritis of the adjacent joint.

Clinical History and Examination:

Septic Arthritis:
<24hrs unwell, fever, pain in affected joint
unable to weight bear/limping
pseudoparalysis/asymmetric movement of limb (early signs in infants & neonates)
hot/swollen & tender joint
reduced joint movement

Acute Osteomyelitis:
similar to septic arthritis
swelling overlying the bone & tenderness

Chronic Osteomyelitis
less ill
+/- fever
local signs less obvious
draining sinus/bony deformity may be present

Differential diagnosis:
Cellulitis, subcutaneous abscess, fractures, & bone tumors (ALL, AML, neuroblastoma, Ewing's sarcoma and osteosarcoma). In newborns and infants in whom osteomyelitis may present as a pseudoparalysis, also consider NAI, CNS disease cerebral hemorrhage and trauma. Juvenile arthritis, Henoch-Schonlein purpura and reactive arthritis can be mistaken for septic arthritis.

Aetiology:

S. aureus is the most common pathogen (75%), followed by S. pneumoniae & pyogenes. Gram-negative bacteria and group B streptococci frequently are seen in newborns. Neisseria gonorrhoeae is also important in the neonatal period. P. aeruginosa is often associated with osteomyelitis and osteochondritis following penetrating wounds of the foot. Bony lesions due to Bartonella henselae (catscratch disease) are also reported. Salmonella is an important cause of osteomyelitis in children with sickle cell disease. Kingella kingae, a fastidious gram negative rod, is increasingly recognized as causing osteoarticular infections. Haemophilus influenza type b (Hib) is now uncommon thanks to the vaccination programme. HIV and some other immunodeficiencies predispose to atypical mycobacterial and fungal osteomyelitis (often multifocal).
Investigations

Elevated white cell count in 50% patients. CRP & ESR almost always are elevated and useful for monitoring response to treatment. Perform on admission, prior to changing to oral therapy and 2 weeks after switching to oral therapy.

Blood culture. Positive in 40% of septic arthritis and 60% of osteomyelitis.

Bone, and joint aspirate gram stain & cultures will improve diagnostic pickup. A synovial fluid WBC count >100,000/mL suggests septic arthritis, especially with a predominance of PMNLs.

Pneumococcal/group B streptococcal antigen detection in urine

Check sickle status if appropriate.

Imaging Studies

Xrays:

Local soft tissue swelling is the earliest sign. Periosteal reaction is present after a few days.

Useful in detecting bone tumours, fractures, & healing fractures. Osteopenia, lytic lesions, & periosteal changes are late radiographic signs. Absence does not exclude acute osteomyelitis.

Plain radiographs of newborns often have a lytic area at diagnosis.

Bone Scan:

Three-phase technetium radionuclide bone scanning demonstrates increased osteoblastic activity of the infected bone & distinguishes osteomyelitis from deep cellulitis. False-negative rate 20%, especially in the first few days of illness. Fractures, bone tumours, and surgery also cause enhanced technetium uptake.

Indium scan: Uses indium-labeled leukocytes but has limitations in newborns, infants, & neutropenic patients. Not a routine investigation.

MRI:

Increasingly used to define bone involvement in patients with a negative bone scan. Changes in bone marrow caused by inflammation result in an area of low signal intensity within bright fatty marrow. These abnormalities need to be clinically correlated before a diagnosis is made, as they are not specific for osteomyelitis.

Ultrasound:

Simple, sensitive, and inexpensive technique for detecting a hip effusion. Also useful to guide the aspiration needle if an effusion is detected.

Treatment

OSTEOMYELITIS and SEPTIC ARTHRITIS are medical emergencies. Patients will be managed jointly by the Paediatric and Orthopaedic teams. The paediatric registrar and attending consultant will coordinate investigations and management. Paediatric and Orthopaedic teams will liaise frequently – daily during the acute phase.

In osteomyelitis in children with sickle cell disease, liaise with Haematology regarding management.

Surgery

Early surgery indicated if pus is present in a joint or sub-periosteally or in the soft tissues

And/or:

i) If there is failure of clinical response within 24 hours of commencing therapy,

ii) In the severely ill child (after resuscitation and rehydration).

If having a general anaesthetic, consider what other procedures may be performed at the same time e.g. line placement.
Send pus/joint fluid (NOT swabs) in a plain sterile container (WITHOUT formalin). When sufficient pus is present, an aliquot should also be inoculated into blood culture bottles. If tissue obtained, send some for histology and some for microbiology.

Antibiotics:
- Please see the Trust antibiotic guidelines

Initial cover for staphylococci and streptococci:
Flucloxacillin 50mg/kg/dose QDS + Ceftriaxone 80 mg/kg OD
If penicillin allergic: Clindamycin 20mg/kg/dose, divided QDS). Change according to sensitivities. Consider changing antibiotics if clinical and laboratory progress suboptimal.
Initially IV: minimum 2-4 days for SEPTIC ARTHRITIS, then 2/52 of oral antibiotics (total 3/52) – see below*
Initially IV: minimum 2/52 for OSTEOMYELITIS, then 4/52 of oral antibiotics (total 6/52) – see below*
* Doses should be written per dose not per day.
Neonatal osteomyelitis, consider flucloxacillin & cefotaxime to cover Enterobacter, GBS & S aureus.
In children with penetrating trauma of the foot, consider an antipseudomonal penicillin or Ceftazidime + Gentamycin, & surgical debridement.

* Once symptoms and signs of inflammation have subsided and the ESR has started to fall, consider switching to oral antibiotics.
  - Only switch to oral from IV if:
    - Clinically improving
    - Afebrile
    - ESR/CRP/WCC improving
    - Can tolerate oral antibiotics
    - Family circumstances suitable.

Single oral agent: Clindamycin (culture –ve) (divided qds)
(amoxycillin/ clavulonic acid (as Augmentin-Duo) dose 0.3 ml/kg/dose, bd (max. 10ml / dose) is an alternative if culture negative or there are supply problems with clindamycin,)). Flucloxacillin (S.aureus) (divided qds) doses per dose.
Vancomycin is an alternative to clindamycin for empiric therapy in patients with methicillin-resistant S aureus.
Follow up in Paediatric clinic

OSTEOMYELITIS: 2 weeks after discharge. XR at end of treatment (6/52) then clinical review 2/52 later.
SEPTIC ARTHRITIS: 2 weeks after discharge (at end treatment). Then review 2 weeks later.
Follow up in Orthopaedic Clinic : 2 months after discharge.
Weekly ESR/CRP & FBCs to monitor response to treatment. Oral antibiotic dosages may need to be increased to keep peak serum-cidal levels of 1:8 or greater. If serum-cidal levels are not adequate with oral antibiotics, the patient may need parenteral treatment.
5-10% of patients may experience recurrence, which may lead to chronic osteomyelitis with discharging sinuses and other systemic sequelae.

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