Osteomyelitis

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As with septic arthritis, bone infection is usually caused by haematogenous spread. Infection starts in the metaphyses of long bones, where the slow flow through the looped vessels combined with microtrauma encourages seeding of infection during a bacteraemia (Figure 44.41a). Inflammation follows

Figure 44.39 Ultrasound scan of a hip joint. A large effusion is dis

tending the joint capsule. The dotted line represents the distance between the femoral neck and the joint capsule.

and, if purulent material forms, the pressure e ff ects secondary to the abscess formation lead to bony destruction. Pus passes through cortical bone and when it does so it elevates the strong periosteum, which may render the cortical bone avascular. As in cases of trauma or tumour, the periosteal elevation is a potent stimulus for new bone formation. In cases of untreated or chronic infection this new bone or involucrum may surround the dead bone, the sequestrum, leading to a ‘bone-within-a bone’ appearance (Figure 44.41b). The presentation and investigation of osteomyelitis can be similar to those for joint sepsis. The di ff erentiation between the two may be di ffi cult and a sympathetic joint e ff usion may occur with metaphyseal osteomyelitis. Thus, if there are no organ isms seen on microscopy of a joint aspirate, the possibility of a coexisting osteomyelitis must be considered. The metaphysis of a long bone may be intracapsular and infection may spread - easily into the joint once the periosteum is breached. In the neonate, proximal femoral osteomyelitis and septic arthritis are essentially the same condition (Figure 44.41c). - General principles for the management of infection should be followed. Pus needs to be drained but otherwise the treatment is medical. Debate continues over the duration of

(b) Figure 44.40 Septic arthritis of the right hip: (a) anteroposterior

(AP) pelvic radiograph with subtle signs of right hip subluxation; pelvic radiograph 6 months later showing destruction of the femoral head secondary to late treatment of a septic joint. sinusoidal colonies vessel Trauma Ve in Artery (with bacteraemia) (b) Dead and dying bone s Bone absces Periosteum Pus Cortex Medullary cavity (c) Joint cavity (b) AP Bone absces s Periosteum Figure 44.41 (a–c) Diagrams illustrating the pathology underlying the development of osteomyelitis. The longer the infection goes

untreated the greater the destruction, with the possibility of sequestrum forma

tion and secondary joint infection.

or oral: management varies from region to region and relates to the local bacteriological prevalences. Methicillin-resistant S. aureus (MRSA) is common in some areas and the presence of the Panton–Valentine leukocidin gene increases morbidity . The shortened intravenous and oral treatment regimes are for uncomplicated cases of osteomyelitis and septic arthri tis only , and only for patients who are improving clinically and haematologically . Summary box 44.22 Bone and joint infection /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Occurs by haematogenous spread, enhanced by microtrauma In untreated and/or chronic osteomyelitis, new involucrum envelops dead sequestrum In addition to antibiotics, treatment consists of: Rest/splintage of affected limb Analgesia A joint effusion may be sympathetic, a primary septic arthritis or caused by direct spread from the adjacent metaphyseal infection Treatment involves: Drainage of pus when present Appropriate and often prolonged antibiotic therapy: parenteral and then oral Treatment of the underlying condition, e.g. nutritional de /f_i ciency, sickle cell disease

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