Bone and joint infection

Philip Bejon

Esther Robinson

Abstract

Bone and joint infections are serious. They can be life-threatening or, more commonly, associated with long-term disability and reduced quality of life. The spectrum of disease has changed over time, and the proportion of iatrogenic disease caused by infected prosthetic joints is increasing annually. Osteomyelitis results when bacteria colonize bone, usually a sterile tissue, via direct inoculation from trauma or operation, haematogenous seeding from bloodstream infection or contiguous spread from another infected site. The pathophysiology of biofilm formation leads to a chronic infection that is not readily accessible to either host immunity or antibiotics. Presentation is with pain, swelling, lack of mobility and systemic symptoms. Diagnosis requires clinical symptoms, appropriate radiological imaging and microbiological sampling. Early or very limited osteomyelitis may be managed with antibiotics alone, but surgical debridement is key for many infections. Therapy should be undertaken by multidisciplinary teams including infection specialists, surgeons, radiologists and other clinical professionals.

Keywords Bone infection; diagnosis and management; MRCP; osteomyelitis; prosthetic joint infection; septic arthritis

Key points

- Antibiotics alone can be used for early and limited osteomyelitis; most other osteomyelitis requires surgical drainage or debridement
- Therapy should be undertaken by multidisciplinary teams including infection specialists, surgeons, radiologists and other clinical professionals
- Acute prosthetic joint infection in a non-loosened joint can be managed by 'DAIR' (debridement, antibiotics, implant retention), but revision is necessary if the implant is loose

Introduction

Infection of joint spaces and bones can be limb-, joint- or life-threatening. Spontaneous disease is uncommon, but the incidence of prosthetic joint infection (PJI) and diabetic foot osteomyelitis, is increasing because of rising arthroplasty rates and increasing prevalence of diabetes mellitus, respectively.

A high index of clinical suspicion and appropriate diagnostic testing are required to allow timely intervention. Invasive sampling and/or debridement are often required and necessitate collaboration between physicians, radiologists and surgeons.

Pathogenesis

Infection of bone or joints occurs as a result of traumatic inoculation, haematogenous seeding or contiguous spread. Causative organisms, with some characteristic clinical features, are given in Table 1.

Philip Bejon *MB BS DTM&H MRCP PhD is an Honorary Consultant Physician in Infectious Diseases at Oxford University Hospitals NHS Foundation Trust, working primarily on the Bone Infection Unit at the Nuffield Orthopaedic Centre, Oxford, UK. He also has interests in malaria epidemiology and vaccine trials. Competing interests: none declared.*

Esther Robinson *BM BCh DPhil FRCPath is an Associate Clinical Professor in Microbiology at Warwick Medical School and an Honorary Consultant Microbiologist at Heart of England Foundation Trust, Birmingham, UK. Competing interests: none declared.*

General principles in diagnosis of bone and joint infection

No biochemical or haematological test is diagnostic, but raised peripheral neutrophil count, serum C-reactive protein and newer biomarkers such as procalcitonin can support the diagnosis.

Joint aspiration allows white cell count, Gram staining and culture. Imaging can support diagnosis and guide sampling. Ultrasound is useful for assessing fluid in a joint but significant knee effusions can be aspirated at the bedside without imaging. Osteomyelitis and joint destruction might be visible on plain radiographs, but these are insensitive and changes occur late in disease. Magnetic resonance imaging is the most sensitive modality for osteomyelitis; computed tomography is an alternative.

Therapeutic principles

Antibiotics should usually be started after adequate microbiological sampling has occurred, but empirical treatment should not be withheld in a patient with life-threatening systemic sepsis. Empirical antibiotics should be broad spectrum and guided by local resistance patterns; antibiotic prescription should be tailored as soon as the pathogen is identified.

Antibiotics alone can be used for early and limited osteomyelitis; all other bone and joint infections discussed here require surgical drainage or debridement. Good surgical management requires removal of all necrotic or infected tissue, stabilization of the joint or cavity and complete skin closure. If the skin cannot be closed, specialist input from plastic surgeons is required.

Osteomyelitis

Epidemiology and pathogenesis

Osteomyelitis affects approximately 10–100 per 100,000 population per year.[1] Haematogenous osteomyelitis is most common in those aged <20 years, with >50% of cases in the under-5s.

Clinical features

Acute osteomyelitis presents with several days of pain, tenderness and swelling, possibly with signs of systemic sepsis. Concomitant septic arthritis can be the presenting feature. Vertebral and pelvic osteomyelitis often present with pain alone.

Chronic osteomyelitis, when bone has become necrotic, presents over a longer period, often in adulthood after an acute

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Summary of causative organisms and their risk factors in bone and joint infection

Disease		Causative organisms	Diagnostic clues
Osteomyelitis		*Staphylococcus aureus* (90% adults, >50% children)	Bacteraemia, soft tissue infection
		Streptococcus pyogenes (group A)	Skin infection, trauma
		Streptococcus groups C and G	Diabetes mellitus, immunocompromise
		Group B *Streptococcus*	Neonate, elderly patient
		Gram-negative bacilli (e.g. *Escherichia coli*, *Salmonella*)	Sickle cell disease
		Polymicrobial (5%)	Post-traumatic
		Mycobacterium tuberculosis	Geographical association; more common with immunocompromise; vertebral disease especially
		Brucella spp	Exposure to infected animals or milk; typically vertebral bodies and long bones; classically indolent presentation
		Fungi	Immunocompromised host; post-traumatic
		Haemophilus influenzae	Paediatric patients; very uncommon since introduction of Hib vaccine
		Kingella kingae	Children <3 years of age; may require specialized diagnostic methods
		Bartonella henselae	Exposure to cats; vertebral and limb girdle infection
Septic arthritis		*Staphylococcus aureus*	Bacteraemia; cellulitis around joint
		β-Haemolytic streptococci	Bacteraemia; cellulitis
		Streptococcus pneumoniae	May be multiple joints; associated with better functional outcome than other pathogens
		Neisseria gonorrhoeae	Often multiple joints; young, sexually active adults; also neonates
Prosthetic joint infection	Early (<3 months after surgery)	*Staphylococcus aureus*, β-haemolytic streptococci, enterococci	Inoculated at time of operation; strong association with surgical site infection
	Delayed (3–24 months after surgery	Coagulase-negative staphylococci, *Propionobacterium acnes*	Low pathogenicity, biofilm-forming organisms; indolent presentation.
	Late (>24 months after surgery	*Staphylococcus aureus*, Gram-negative bacilli	Haematogenous seeding from infection elsewhere

Table 1

episode in childhood, or following an acutely infected open fracture. A sinus tract over the infected area is pathognomonic.

Diagnosis

Acute osteomyelitis is likely if pus collections are seen on imaging and is confirmed by invasive sampling with microbiological culture. Chronic osteomyelitis is more likely to be culture-negative, but imaging supports the diagnosis. Malignancy is an important differential diagnosis, requiring histological assessment. Osteomyelitis resulting from diabetic foot infection can involve resistant organisms following prior antibiotic use and chronic soft tissue infection, and diagnostic imaging is complicated by neuropathic bone changes that mimic infection.

Treatment

Acute osteomyelitis might respond to antibiotics alone, especially in children, but collections require prompt drainage. Chronic osteomyelitis is characterized by necrotic bone (sequestrum) and periosteal changes. Curative treatment requires surgical excision. This risks fracture if the bone is weakened, or amputation if extensive resection is required. Optimal management is delivered by multidisciplinary teams in specialist units, including limb reconstruction and plastic surgeons. There are few randomized trial data to guide choice, route or duration of antibiotics. Typically, long courses are given, often including intravenous antibiotics.[2] However, recently reported randomized trials challenge the need for prolonged intravenous antibiotics.

Native joint septic arthritis

Epidemiology and pathogenesis

The incidence is approximately five per 100,000 population per annum. Risk factors include extremes of age, diabetes mellitus, immune suppression, joint surgery or injection, intravenous drug abuse and infection of overlying skin.

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