7: Soft tissue, bone and joint infections

Thomas Gottlieb, Bridget L Atkins and David R Shaw

Although many of these infections are trivial, some may threaten life or limb

Abstract

- Soft tissue infections are common and usually respond rapidly to oral antibiotics; if empirical therapy fails then exposure to unusual organisms should be considered.
- Septic arthritis requires early recognition, identification of the infecting pathogen and urgent joint washout to prevent irreversible cartilage and bone destruction.
- Prosthetic joint infection is uncommon but has high morbidity; the best outcomes are achieved with removal of the prosthesis and replacement after at least six weeks of antibiotic therapy.
- Osteomyelitis often complicates diabetic foot infection with ulceration and is rarely cured by antibiotics alone; early surgical intervention achieves the best outcome.

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SOFT TISSUE, BONE AND JOINT INFECTIONS cause considerable morbidity in hospitals and the community. Their severity varies from trivial to lethal. These infections commonly arise from skin breaches or bacteraemia, but occasionally they are a manifestation of a more generalised infection, such as endocarditis. Rarely, a localised soft tissue infection can produce a profound systemic illness with severe pain, tissue necrosis, hypotension and rapid progression to multisystem failure and death. Early recognition is essential, as, without treatment, mortality is high.

Soft tissue infections

Soft tissue infections can be subdivided according to the skin compartment involved (Box 1). Diagnosis is based on the appearance of lesions, degree of pain and systemic toxicity. Knowledge of the organisms involved does not always help define the tissue depth of disease, but aids choice of antimicrobial therapy. Investigation and management of common soft tissue infections are summarised in Box 1.

Impetigo

Impetigo is most common in children and usually involves the skin of the face, often around the mouth and nose. It has two forms:

- Non-bullous, which is most commonly caused by *Streptococcus pyogenes* (group A streptococci) and is typified by "honey-crust" lesions (Box 2); and
- Bullous, which is caused by *Staphylococcus aureus*;[4] rupture of the bullae leaves a thin "varnish-like" crust.

Folliculitis

Infection of the hair follicles may occur after exfoliation, use of a loofah sponge and shaving, or may be spontaneous. A specific form is "whirlpool" ("hot tub" or "spa") folliculitis, caused by *Pseudomonas aeruginosa*.[5]

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Skin abscesses and furuncles

S. aureus is the leading pathogen that causes furuncles and abscesses in the community. Certain phage types are associated with recurrent episodes of furunculosis and may spread among family members. Staphylococcal bacteraemia may result from a minor skin lesion or furuncle, with potentially severe complications, including osteomyelitis, septic arthritis and endocarditis.[6]

Recently, infections caused by community-acquired, methicillin-resistant *S. aureus* have been described in both adults and children in Australia.[7] These bacteria are resistant to β-lactam antibiotics, but retain susceptibility to agents such as clindamycin, in contrast to multiresistant *S. aureus*, which is usually hospital-acquired. This β-lactam resistance may delay effective therapy (case history, Box 3).

In patients with recurrent *S. aureus* soft tissue infection, attempts can be made to eradicate the causative strain from long-term carriage in the nasal passages and on skin with nasal mupirocin and skin antiseptic solutions.[8] Penicillins and cephalosporins are ineffective in eradicating nasal carriage.

Cellulitis

Cellulitis is an acute, spreading inflammation involving the epidermis, dermis and subcutaneous fat. The most common causes are *S. aureus* and ß-haemolytic streptococci (most commonly group A [*S. pyogenes*], but also groups C and G). Host factors may predispose to cellulitis and should be corrected if possible. These are infective (eg, varicella, tinea pedis and scabies) and non-infective (eg, eczema, trauma, local ischaemia, venous, arterial or vasculitic ulceration, lymphatic impairment, surgery and radiotherapy). Rarer

1: Investigation and management of soft tissue infections*

Syndrome	Common pathogens	Investigations	Management
Impetigo (epidermis)	*Streptococcus pyogenes* *Staphylococcus aureus*	■ None	■ *If uncomplicated and localised:* wash crusts off, apply topical mupirocin or sodium fusidate.[2] ■ *If multiple lesions present or not responding to topical treatment:* oral dicloxacillin. ■ *If severe or extensive, systemically unwell or not responding to oral treatment:* intravenous flucloxacillin.[3]
Folliculitis (hair follicle)	*S. aureus* *Pseudomonas aeruginosa* ("spa" or "whirlpool" folliculitis)	■ None	■ Usually self-limiting, no specific treatment required. ■ Avoid precipitating factor (eg, exfoliation). ■ In "spa" folliculitis, spa should be drained, cleaned and chlorinated on refilling.
Skin abscesses, furuncles (hair follicles)	*S. aureus*	■ Gram stain and culture of pus	■ Incision and drainage. ■ Antistaphylococcal antibiotics (initially intravenous if severe).
Cellulitis (epidermis, dermis, subcutaneous fat)	ß-Haemolytic streptococci *S. aureus* ***Rarely:*** *Haemophilus influenzae*, others	■ Blood cultures ■ Gram stain and culture of aspirate from blister or pustule, if present, or (in immunosuppressed patient) from leading edge of cellulitis ■ Consider unusual causes if relevant exposure (Box 4); may require tissue biopsy for histopathological examination, microscopy (Gram and acid-fast stain) and culture (including fungal and mycobacterial culture), or directed nucleic acid amplification tests.	■ Elevate limb. ■ Intravenous antibiotics directed at staphylococci and streptococci (eg, flucloxacillin, cephalothin or, for home-based therapy, cephazolin); or broader spectrum (eg, ceftriaxone and fluoroquinolones) if need to cover waterborne pathogens, such as *Aeromonas* or *Vibrio* spp., or patient is immunocompromised. ■ Consider hospital admission (Box 5).
Necrotising fasciitis (fascia)	*S. pyogenes* Mixed bowel flora	■ Gram stain and culture of surgical samples ■ Blood cultures	■ Urgent surgical debridement (multiple debridements may be needed). ■ Intravenous antibiotics: initially broad spectrum to cover β-haemolytic streptococci, enteric gram-negative rods, and anaerobes; and modified on culture and susceptibility results. Clindamycin should be added if *S. pyogenes* is identified.
Clostridial myonecrosis (gas gangrene) (muscle)	*Clostridium perfringens*	■ Gram stain and culture of surgical samples ■ Blood cultures	■ Urgent surgical debridement plus intravenous antibiotics (penicillin plus clindamycin). ■ Hyperbaric oxygen may have a role.

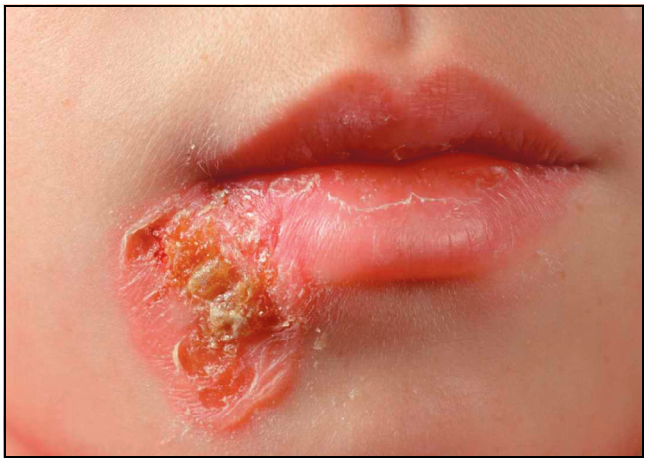
*Summarised from *Therapeutic guidelines: antibiotic.*[1]

causes of cellulitis are associated with specific exposures (Box 4). Infection with *S. pyogenes* may also be confined to the dermis, termed erysipelas.

Clinical features: Streptococcal infections often have an abrupt "wildfire" onset with localised pain in the affected limb. Rigors may precede visible signs of skin involvement or lymphangitis by 24 hours. Tender regional lymphadenitis often develops. Often fever abates, and leukocyte count and C-reactive protein level decrease before any improvement at the site of cellulitis. Indeed, the area involved, blistering and local necrosis may continue to increase for days after a systemic clinical response. Desquamation occurs on recovery.

Diagnosis and management: In practice, the specific causative organism is usually not isolated: blood cultures are usually negative,[10] and skin swabs are rarely diagnostic. However, culture of an aspirate from an intact blister may be helpful, and, in the immunosuppressed patient or after unusual

2: Non-bullous form of impetigo



"Honey-crust" lesion typical of Streptococcus pyogenes *impetigo.*

3: Case history — community-acquired methicillin-resistant *Staphylococcus aureus*

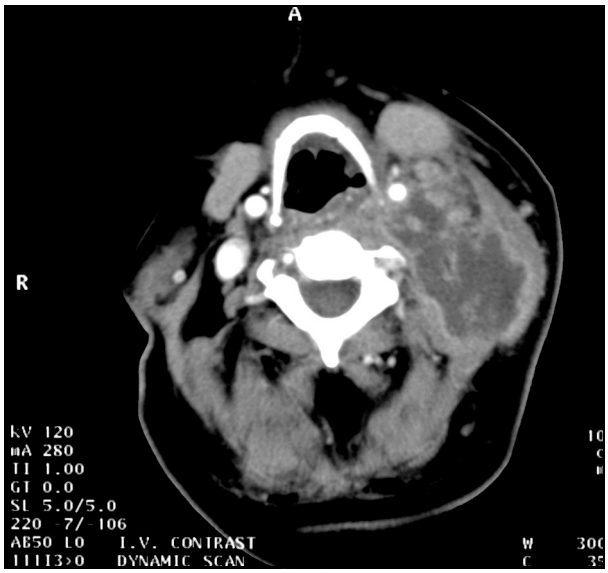
Presentation: A 14-year-old girl presented with a four-week history of progressive swelling in the left submandibular region. Despite treatment with oral flucloxacillin for 10 days she experienced increasing pain, fever and torticollis.

Management: The girl was admitted to hospital and given intravenous flucloxacillin and penicillin for three days without improvement.

Further history revealed that both the girl and her younger brother had experienced recurrent boils since a family visit to Samoa a year previously. Swabs of pus from a boil, as well as from her nose, axilla and groin, had failed to show any pathogens. The boils had responded slowly to flucloxacillin. Attempted eradication of staphylococci with intranasal mupirocin and triclosan washes had prevented further skin sepsis.

Investigations: Magnetic resonance imaging of the neck demonstrated necrotic submandibular lymphadenitis with a deep cervical abscess (right). The abscess was drained under anaesthesia. Culture of the pus revealed "community type" or "non-multiresistant" methicillin-resistant *Staphylococcus aureus*. The isolate was susceptible to erythromycin, tetracycline, gentamicin and ciprofloxacin, and resistant to β-lactams, flucloxacillin and cephalexin.

Management and course: Treatment was changed to clindamycin. Over the next 24 hours, the fever abated, and within seven days the swelling and pain decreased. Therapy was changed to oral clindamycin. After a further four weeks' therapy, the infection was completely resolved.



- *Methicillin-resistant* S. aureus (*MRSA) is no longer confined to patients with a history of recent hospitalisation.*
- *"Community" MRSA strains are increasingly reported in children and are often associated with pyogenic complications.*
- *Failure to consider or recognise MRSA may lead to inappropriate* β*-lactam therapy. We believe that boils and furuncles should be swabbed for Gram stain and culture.*

exposures (Box 4), an aspirate obtained through needling the leading edge of the cellulitis is valuable.[11]

Intravenous antibiotics are preferred initially except in mild infections (Box 1). Features that suggest a poor prognosis and need for hospital admission are shown in Box 5. Home-based intravenous therapy is an alternative for patients in a stable condition.[12]

If cellulitis does not respond to empirical β-lactam therapy, then environmental or occupational exposure to unusual pathogens should be considered (Box 4). Infections with these organisms may also present as single ulcers, nodules or nodular lymphangitis (case history, Box 6). Tissue biopsy may be required for diagnosis (Box 1).

Some patients have recurrent cellulitis and can be provided with a supply of antibiotics to take when symptoms recur. However, this is not always effective. Twice-daily penicillin V, cephalexin or erythromycin may be effective prophylaxis,[13] while some patients require monthly injections of benzathine penicillin.

Toxic shock syndromes

These syndromes of hypotension, fever, faint "sunburn-like" rash and multiorgan failure are caused by *S. aureus* or *S. pyogenes*. There may be obvious skin infection or, in the case of *S. aureus*, only vaginal or wound infection. Removing the source of bacterial toxin production (eg, wound debridement, removing vaginal tampon) is essential. Inclusion of clindamycin in the initial antibiotic regimen has been associated with a better outcome in streptococcal toxic shock, possibly as this antibiotic inhibits bacterial protein synthesis and thereby reduces toxin production.

Necrotising fasciitis

Necrotising fasciitis is a surgical emergency needing prompt recognition and radical debridement of devitalised tissue. It can take two forms:

- type 1, caused by multiple organisms, often of gut origin (synergistic gangrene); and
- type 2, caused by group A streptococci.

Both forms cause severe systemic toxicity that leads to hypotension, respiratory distress and multiorgan failure. There is usually severe pain in the affected area, with only modest overlying skin changes in the early stages, progressing rapidly to fascial and skin necrosis (Box 7) and deep tissue infarction, particularly of the muscle layers.

4: Unusual causes of cellulitis and nodular infections associated with specific exposures

Traumatic inoculation of soil, penetrating injury from thorns: Non-tuberculous mycobacteria (eg, *Mycobacterium fortuitum*, *M. chelonae*, *M. ulcerans*), *Nocardia* spp., fungi

Travel to tropical Australia[9] *or other tropical areas:* *Burkholderia pseudomallei* (melioidosis), chromoblastomycosis, *Chromobacterium violaceum*

Travel to tropical areas (beaches): Cutaneous larva migrans

Aquatic or marine trauma: *Mycobacterium marinum* (case history, Box 6), erysipelothrix (erysipeloid), *Vibrio* or *Aeromonas* infection

Animal bites: *Pasteurella multocida*, *Capnocytophaga canimorsus*

Human bites: *Eikenella corrodens*

Bathing in spas or tubs: *Pseudomonas aeruginosa*

Cat scratch: *Bartonella henselae*

Immunosuppression (eg, HIV, transplantation): *Nocardia* spp., *Cryptococcus neoformans*, *Mycobacterium tuberculosis* (reactivation)

Clostridial myonecrosis (gas gangrene)

Clostridial myonecrosis may follow trauma with penetration of contaminated material (eg, injection of contaminated recreational drugs) or occur spontaneously (often in association with underlying bowel malignancy). Gas production by clostridia may produce crepitus within the soft tissues. Mortality is high.

Bone and joint infections

Types of bone and joint infections and their investigation and management are summarised in Box 8.

Septic arthritis of a native joint

Septic arthritis requires prompt diagnosis, as delays in surgical drainage and antibiotic therapy may lead to progressive synovitis and irreversible cartilage and bone destruction.[14-16]

Clinical features: Acute septic arthritis usually presents with a warm, swollen, painful joint with an effusion. Restriction in movement differentiates it from bursitis. Occasionally, septic arthritis may be the presenting feature of endocarditis.

Diagnosis: This relies on a combination of clinical assessment, laboratory and radiological investigations. Attention should be paid to:

- distribution of involved joints;
- pre-existing joint disease, trauma or extra-articular infection;
- underlying diseases (eg, immunosuppression, malignancy or diabetes mellitus);
- sexual history (eg, recent genital tract discharge or urethritis); and
- activities and travel history (infections such as melioidosis and brucellosis have strong geographic associations).

Blood leukocytosis and raised C-reactive protein level and erythrocyte sedimentation rate are characteristic. Blood cultures are positive in about 50% of patients with acute non-gonococcal bacterial arthritis but in only 20% of those with gonococcal arthritis.[17] Serological tests are rarely helpful, except in chronic infections with *Brucella* spp., *Coxiella burnetii* (Q fever), *Treponema pallidum* (syphilis) and *Borrelia burgdorferi* (Lyme disease).

Culture and microscopy of synovial fluid are essential for specific diagnosis of a hot, swollen joint. Gram stain shows organisms in 50% of cases of septic arthritis. Ultrasonography is very sensitive for detecting joint effusions and guiding diagnostic aspiration, especially in the hip joint. Computed tomography and magnetic resonance imaging are useful in complicated or atypical cases.[18]

Management: Empirical antibiotic therapy directed against staphylococci should be given while awaiting culture results. Joint washout should be performed urgently and repeated often.

Prosthetic joint infection

Infection of prosthetic joints usually presents with insidious onset of increasing joint pain, modest swelling and possibly

5: Features suggesting a poor prognosis and need for hospital admission in cellulitis

- Pre-existing conditions (eg, renal impairment, diabetes, congestive cardiac failure, peripheral vascular disease, neoplasm, radiotherapy in proximity to cellulitis, immunosuppression, splenectomy, alcoholism and neutropenia)
- Extensive or rapidly progressive cellulitis
- Presence of bullae, necrosis or muscle involvement
- High fever, rigors
- Hypotension (with or without generalised rash)
- Development of renal impairment
- Suppurative wound or bite (especially on face or hand) requiring surgical drainage
- Animal or human bite; exposure to marine or riverine waters
- Lack of systemic or local response, or rising or unchanging C-reactive protein level despite adequate therapy
- Positive blood cultures

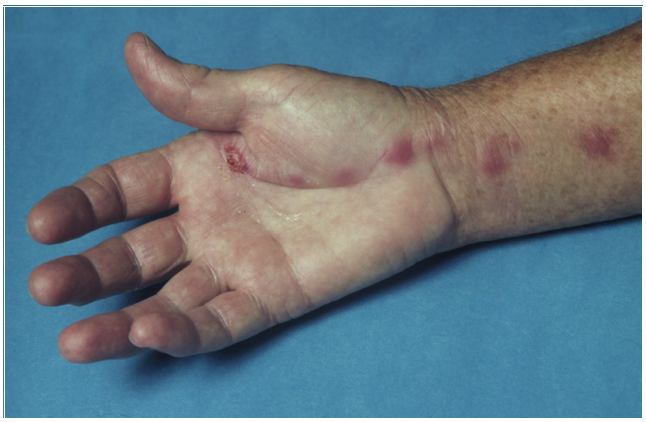
6: Case history — soft tissue infection caused by an unusual pathogen

Presentation: An elderly woman injured her hand with a fish hook while rock fishing on the New South Wales central coast. Over the next three weeks, she developed small, tender nodules along the lymphatic chain of the right forearm (pictured).

Investigation: A swab from a palmar ulcer grew *Staphylococcus aureus*. However, the illness had no systemic features (no fever, normal white cell count and no lymphadenopathy). She was assumed to have nodular lymphangitis. Biopsy of the lesion confirmed the presence of acid-fast bacilli. *Mycobacterium marinum* was isolated after three weeks' incubation.

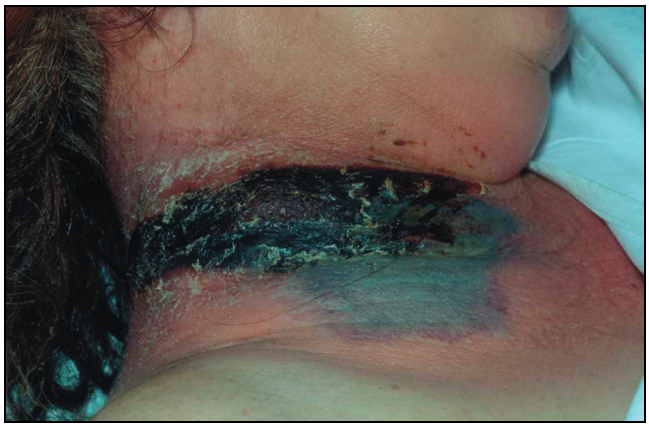
Management: She was treated with trimethoprim–sulfamethoxazole for three months with complete resolution.

- *This case illustrates the importance of a good history and review of the clinical presentation; the illness was not compatible with a staphylococcal infection despite a positive culture result.*
- *Post-traumatic infections caused by* Mycobacterium *or* Nocardia *spp. may persist for weeks or months; diagnosis may require biopsy.*



- *Biopsy specimens should be sent for histopathological and microbiological examination, and the possibility of an unusual pathogen (eg, a waterborne pathogen) should be specified, as the laboratory may not culture on the specific media required for isolation of these organisms unless prompted.*

7: Necrotising fasciitis



Necrosis involving skin and deep structures of the neck 48 hours after presentation with pain and fever.

sinus formation.[19] Presentation may be more acute if infection is haematogenous or occurs early after placement of a prosthesis.

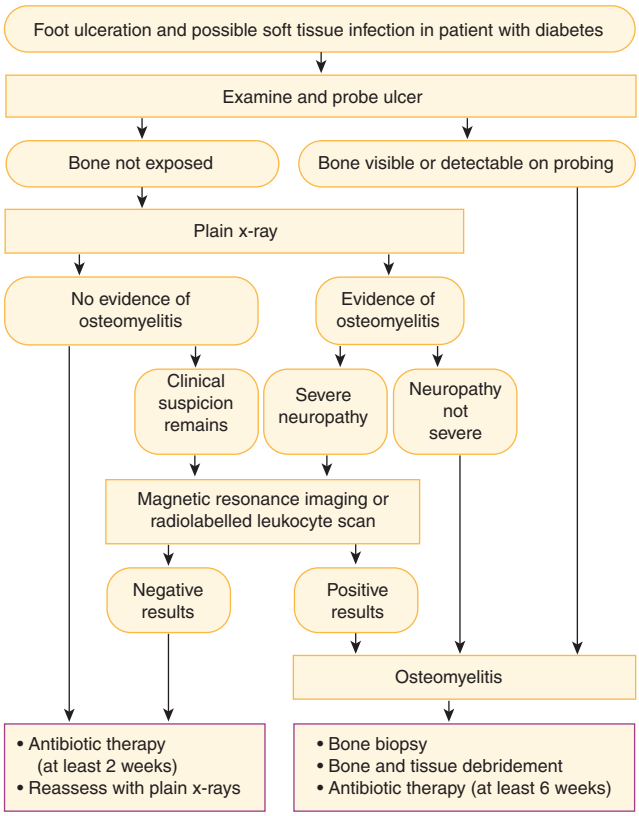
Diagnosis: Plain x-rays may show loosening of the prosthesis, but no clinical or radiological features can reliably differentiate infective from non-infective loosening. Radionuclide scans may show increased uptake at the site of infection but cannot provide a specific diagnosis. Blood cultures are rarely positive, except in acute infections, and microscopy and culture of swabs from sinuses are usually not helpful. However, specimens obtained from joint aspirates or deep debrided tissue may show the causative organism.[20,21]

Management: Prosthesis salvage can be attempted when the prosthesis is not loose and onset of symptoms is acute. With this approach, infections due to *S. aureus* have a greater than 50% chance of cure if surgical debridement is performed early.[22,23] Broader application of this approach has been shown to be cost-effective in the elderly.[24] If salvage is not realistic, the

8: Investigation and management of bone and joint infections

Syndrome	Common pathogens	Microbiological investigations	Management
Native joint septic arthritis	*Staphylococcus aureus* β-Haemolytic streptococci *Neisseria gonorrhoeae* ***Rarely:*** *Kingella kingae* (children), *Haemophilus influenzae*, *Mycobacterium tuberculosis*	■ Blood cultures ■ Synovial fluid microscopy (cell count, crystals and Gram stain) and culture (including mycobacterial and fungal, *if chronic*) ■ Genitourinary samples *(if gonococcal arthritis suspected)* ■ Consider synovial biopsy *(if chronic)*	■ Surgical washout of joint. ■ Intravenous antibiotics empirically (eg, flucloxacillin, cephalothin).
Prosthetic joint infection	Coagulase-negative staphylococci *S. aureus* Other gram-positive bacteria ***Rarely:*** gram-negative bacteria, *Candida* spp., *M. tuberculosis*	■ Blood cultures *(if acute)* ■ Gram stain and culture of joint aspirate and multiple deep surgical samples at debridement ■ Consider synovial biopsy *(if chronic)*	■ Early surgical debridement is essential. ■ ***If no loosening of prosthesis and short duration of symptoms:*** consider prosthesis salvage (joint washout and debridement together with intravenous antibiotics, followed by prolonged oral antibiotic therapy). ■ ***In other cases, if possible:*** replace prosthesis as one- or two-stage procedure with accompanying antibiotics.
Osteomyelitis	*S. aureus* Group A streptococci *M. tuberculosis* ***Infants:*** also group B streptococci, *Escherichia coli* ***Adults:*** also enteric Gram-negatives ***Intravenous drug use or penetrating nail injury to foot:*** *P. aeruginosa* ***Ulcer complication (diabetic, vascular insufficiency, or decubitus):*** as above, often polymicrobial, anaerobes ***Internal fixation device:*** *S. aureus*, coagulase-negative staphylococci ***Sickle cell disease:*** *Salmonella* spp, *S. aureus* ***Tropical exposure:*** *Pseudomonas pseudomallei* (melioidosis)	■ Gram stain and culture of surgically obtained pus, fine needle aspirate, bone biopsy or debridement samples (mycobacterial and fungal culture should be included, particularly in spinal infection) ■ Histological examination (to confirm osteomyelitis or detect neoplasm) ■ Blood cultures *(in acute osteomyelitis associated with internal fixation devices)*	■ Specific antibiotics according to culture results for at least six weeks (initially intravenously). ■ Monitor clinical response, erythrocyte sedimentation rate, C-reactive protein level and leukocyte count. ■ ***If unresponsive to antibiotics or bone tenderness is extreme:*** may require surgical decompression to release pus. ■ ***If chronic:*** requires surgery to debride all devitalised bone and soft tissue; soft tissue coverage is essential and often requires plastic surgery. ■ ***If associated with ulcer:*** also assess vascular supply, optimise diabetes control, podiatry and shoe fitting; surgical debridement (amputation is a last resort). ■ ***If associated with internal fixation device:*** may require debridement or removal of metalwork, but maintaining bone stabilisation is paramount; antibiotics may be needed until union if internal metalwork is retained.

9: Assessment and management of suspected diabetic foot infection and osteomyelitis



Practical tips for assessing a diabetic foot ulcer

- Previous episodes of osteomyelitis or neuropathic complications increase the risk of further episodes of osteomyelitis.
- Most diabetic patients with foot infection have no fever.
- Ulceration and soft tissue infection present for over two weeks, particularly over bony prominences, have a high risk for bony involvement.
- All ulcers with exposed bone have underlying osteomyelitis.
- Tapping bone while probing an ulcer with a sterile blunt probe is a sensitive (66%) and specific (85%) bedside test for osteomyelitis. However, as the negative predictive value is only 56%, this test cannot exclude osteomyelitis.[29]

infected prosthesis is removed or exchanged. A two-stage procedure is widely used, with an interval of about six weeks of antibiotic therapy before replacement of the prosthesis. If two operative procedures are not feasible, exchange of the prosthesis in one operation may achieve reasonable results.

Osteomyelitis (non-diabetic patients)

Bone becomes infected either through haematogenous spread of organisms, or, particularly in people with peripheral vascular disease, secondary to a contiguous focus of infection.[25] Osteomyelitis can be either acute or chronic. The latter usually reflects the presence of non-viable bone or other material and is characterised by bone loss, persistent drainage from sinus tracts and sequestra. The natural history often includes relapses and remissions.

10: Empirical antibiotic therapy for diabetic foot infection

Mild to moderate soft tissue infection
Metronidazole (400 mg orally, 12-hourly) *plus* cephalexin (500 mg orally, 6-hourly)
or amoxycillin–clavulanate (875 mg/125 mg orally, 12-hourly)

Severe soft tissue infection or osteomyelitis
Ticarcillin–clavulanate (3.1 g intravenously, 6–8 hourly)
or clindamycin (600 mg intravenously, 8-hourly) *plus* ciprofloxacin (750 mg orally, 12-hourly)

Diagnosis: Pain and swelling at the site of infection are cardinal features. Radiological changes may not appear for two or more weeks. Radionuclide scans lack specificity, as in joint infection. Computed tomography is a useful test for showing soft tissue changes (eg, paravertebral and psoas abscess) and for guiding biopsy. Magnetic resonance imaging delineates the extent of soft tissue involvement, bony changes and the presence of bone sequestra, and can be used for staging disease and surgical planning. It may be more sensitive than computed tomography early in the diagnosis of vertebral osteomyelitis. In the absence of positive blood cultures, a definitive microbiological diagnosis depends on isolation of the organism from the site of infection. This can be obtained from debrided tissue, pus obtained via fine needle aspiration or bone biopsy.

Management: Intravenous antibiotic therapy is required in all cases, at least initially (Box 8). Attempts should always be made to identify the causative organism, as selection of appropriate antibiotics is important for a good outcome.[26] Chronic osteomyelitis requires surgical debridement to remove devitalised bone and soft tissue for cure.

Diabetic foot infection

About 15% of people with diabetes mellitus develop foot ulceration,[27] which is complicated by osteomyelitis in two-thirds of cases.[28] Foot infection is a leading cause of hospital admission in people with diabetes and a major cause of lower-extremity amputation. Factors that increase the risk of osteomyelitis are:

- duration of diabetes mellitus over 10 years;
- peripheral neuropathy;
- abnormal foot structure with maldistribution of weight over the plantar surface of the foot;
- peripheral vascular disease;
- poor glycaemic control;
- disruption of skin integrity (eg, penetrating injury, fungal infection); and
- male sex.

Diagnosis: An approach to assessment and management of suspected diabetic foot infection and osteomyelitis, as well as practical tips, are summarised in Box 9. Infection complicating foot ulceration is suggested by spreading redness around the ulcer, local swelling and systemic features (eg, malaise, fever and night sweats). Assessing the depth of

infection beneath the ulcer and differentiating infection from neuropathic change is particularly difficult, as both cause bone destruction.

Imaging for osteomyelitis of the foot has poor specificity in diabetic patients, as in non-diabetic patients. In addition, neuropathic osteoarthropathy and healing fractures in diabetic patients may further reduce specificity.[30] Culture of superficial swabs is not useful, as diabetic foot ulcers are invariably colonised by multiple organisms. Bone biopsy and culture is the definitive investigation, with an estimated sensitivity of 95% and specificity of 99%,[31] but its value is much reduced by concurrent antibiotic therapy.

Prevention and management: Patients with diabetes should be educated about avoiding skin trauma; wearing protective, well-fitting footwear; skin moisturising; and inspecting daily for skin pressure. Regular review by a podiatrist is important.

Established osteomyelitis is difficult to cure in the presence of diabetic arteriopathy. Relapse is common, and suppression is often the best that can be achieved. Surgical revascularisation is feasible in some patients and leads to better short- and long-term outcomes. Antimicrobial therapy should be begun pending results of deep tissue culture and is required for at least two weeks for soft tissue infection, and for at least six weeks (often considerably longer) for osteomyelitis[32] (Box 10). Surgical resection of infected bone is the most effective means to eradicate infection, with maintenance of functional integrity of the foot as the goal.

With aggressive, targeted antibiotic therapy and early debridement, the short-term outcome is generally good. However, the three-year recurrence rate is high (amputation in more than 22% of patients), as is three-year mortality (27% in patients with primary healing of the initial ulcer, and 41% in those requiring amputation as initial treatment).[33]

Evidence-based recommendations

- Septic arthritis can be diagnosed on the basis of the clinical picture, supported by results of microscopy and culture of joint fluid[17] (E3).
- In prosthetic joint infection, the most consistent results rest with prosthesis removal and antibiotic therapy before replacement; long term success depends on many factors,[23,34] and it may not be practical for some patients. In selected patients, retention of an infected prosthetic hip arthroplasty plus prolonged antibiotic therapy may be successful (E2).
- Osteomyelitis is suggested in diabetic foot infection if the overlying ulceration is deep, or bone can be reached by probing[27,29] (E2).
- Resection of devitalised bone and tissue plus antibiotic therapy can be an effective foot-sparing approach in diabetic foot osteomyelitis[35] (E2).

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