DIABETIC FOOT INFECTION

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The global prevalence of diabetes has increased exponentially in recent years. Foot infections are a leading cause of hospital admissions in this group, with an annual incidence of foot complications of 1–2% per year owing to the combined influ - ence of macro- and microvascular insu ffi ciency , mechanical disruption, peripheral and autonomic neuropathy , immune defects and impaired tissue healing. Ulceration of the calca - neum and bones of the foref oot is common (Figure 43.11) and will result in amputation in up to one-fifth of cases. Infection begins as invasion of bacteria into the com - promised tissues and will rapidly spread to deep structures. Diagnosis is made on the clinical signs and symptoms of local inflammation and systemic upset. The presence of a wound/ ulcer, spreading cellulitis, fevers or critical ischaemia indicate a e infection and the need for urgent treatment. more sever Blood tests are frequently unhelpful, as inflammatory mark - ers may be normal or only mildly raised. Plain radiographs may show evidence of osteomyelitis but can be normal (par - ticularly ear ly in infection). MRI is the most sensitive imaging modality for diagnosis of bone involvement. Superficial swabs - - - terials -

The Figure 43.11 A severe diabetic foot infection, with marked infection, necrosis and tissue loss. The patient was neuropathic and had ankle and hindfoot deformity. The foot was salvaged with a corrective triple fusion of the hindfoot, excision of the infected ulcer, antibiotic therapy and primary closure of the lateral soft tissues.

mining the organisms responsible for underlying deep-seated infection. A combination of the ‘probe-to-bone’ test with ele vated inflammatory markers and abnormal plain radiographs confirms the diagnosis. The aetiological agents of diabetic f oot infection are the same as for bone infection in non-diabetic individuals, namely S. aureus , β -haemolytic streptococci and aerobic Gram-negative bacilli. Pseudomonas is over-represented, and empirical therapy for severe infections should include cover for this organism. Anaerobes may also be present and addition of metronidazole (particularly for abscesses and/or devitalised tissue) should be considered. Surgical debridement is required for collections, necrotic areas or more extensive osteomyelitis. Thought should be given to distinguishing superficial osteitis, resulting from loss of soft-tissue cover (often in association with v ascular compro mise), from more extensive bone involvement. In the former, biopsy and antibiotic therapy may be of limited importance and optimising glycaemic control, improving vascular supply and relieving pressur e, with appropriate footwear, much more important. This approach may avoid more extensive tissue loss or later amputation. Many patients with diabetes with foot infection have sig nificant vascular compromise and neuropathy , which makes healing after surgery unreliable. A full vascular assessment is mandatory in those with poor peripheral pulses. Proximal angioplasty or bypass surgery may impro ve distal vascularity to a level where infection surgery in the foot may be more successful. Amputation is not an easy option in diabetic foot disease and wound healing can be problematic. In general, excision should be adequate to remove all infected material and excess bone may need to be resected to allow

tension-fr ee skin clo sure. If there is extensive peripheral neuropathy , a below-knee amputation in an area with better sensation may be more appropriate. Summary box 43.10 Diabetic foot infection /uni25CF /uni25CF /uni25CF /uni25CF

The most important risk factor for osteomyelitis is the presence of a foot ulcer Ulcer swabs are not reliable in determining the pathogens responsible for osteomyelitis Bone biopsy for culture should be considered in extensive/ complex infection but may not be necessary in mild disease In severe disease, surgical debridement of collections and/ or necrotic tissue is required, followed by antibiotics tailored according to culture results

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