Diabetic Foot

The diabetic foot continues to be a major cause of morbidity, posing a global threat. Substantial progress has been accomplished in the treatment of foot lesions, but further improvement is required. It is now understood that the diabetic foot represents one of the major chronic complications of diabetes, posing a tremendous impact on morbidity and mortality of the diabetic population. In 2005, it was estimated that a lower limb was lost every 30 seconds due to diabetes in some part of the world ***(Bakker et al., 2005).***

Main aspects of pathophysiology

Three major pathologies, mutually interacting, result in the diabetic foot: ischaemia, neuropathy and infection ***(Connor. 2008***).

Ischaemia was recognized in the 19th century as a manifestation of peripheral arterial disease (PAD),which is more common in diabetes, and affects multiple vessels, with a predilection for the infra-popliteal arteries (anterior tibial, posterior tibial and peroneal artery) (***Bakker et al., 2005).***

The role of neuropathy was only appreciated in the second half of the 20th century. Neuropathy is responsible for stocking distribution sensory loss: the feet lose sensation of noxious stimuli, such as trauma induced by stepping on a sharp object or skin injury due to ill-fitting shoes ***(Boulton et al., 2004).***

Initially, foot injury may be trivial, but remain unperceived, eventually leading to progressing deep tissue destruction. Moreover, intrinsic foot muscles are deprived of normal innervation. Loss of innervation may result in muscle atrophy and foot deformities, mostly prominent metatarsal heads and claw or hammer toes. Thus, pressures are gradually abnormally distributed on the plantar aspect of the foot, in a way that some plantar sites have very high pressures and become prone to ulceration (***Frykberg et al., 2006).***

Impaired pressure distribution is aggravated by limited joint mobility (LJM). LJM is a generalized phenomenon in diabetes, mediated by increased non-enzymatic collagen glycosylation in the periarticular tissues. In the foot, it mainly affects the first metatarsophalangeal joint and contributes to elevated plantar pressure at the first metatarsal head ***(Papanas & Maltezos 2007).*** Of note, a rather frequently underestimated manifestation of neuropathy is reduced sweating, alternatively called sudomotor impairment. This is responsible for dry skin and callus formation. Skin fissures may become gates of entry for bacteria and increase the likelihood of infection ***(Tentolouris et al., 2009).***

Ischaemia and neuropathy predispose to infection. The vast majority of chronic foot ulcers become infected. In acute infections, gram-positive cocci predominate. In chronic cases, though, infection is multimicrobial by gram-positive cocci, gram-negative bacteria and anaerobes ***(Papanas &Maltezos 2007).*** Methicillin-resistant Staphylococcus aureus (MRSA) is becoming a nightmare for diabetic foot clinics ***(Dang et al., 2003***)

Indeed, tissue immune response to infection is hampered by ischaemia and neuropathy. Infection may even extend to ligaments, tendons and bones, compromising survival of both limb and patient. For this reason, it is vital to diagnose infection early and assess its severity, as well as vascular status and co-morbid conditions **.**In general practice, it is, probably, more convenient to distinguish between limb-threatening and not limb-threatening infections. In the latter, presenting features include: cellulitis > 2cm; oedema, pain or lymphangitis; drainage or foul odour; infection extending to the bone or joint; systemic signs and symptoms; severe ischemia ***(Papanas & Maltezos 2007).***

Main aspects of treatment

Treatment options may be classified into established measures (revascularization, casting and debridement) and new modalities ***(Papanas & Maltezos 2007).***Established treatment addresses the three major aetiologic factors discussed above. It attempts to restore blood flow to the limb, off-load high pressure areas and tackle infection ***(Frykberg et al., 2006).***

Restoration of blood flow is called revascularization or arterial reconstruction. This is achieved either by the open surgical approach (by-pass graft surgery) or by endovascular techniques (percutaneous transluminal angioplasty, PTA). Both modalities have proved effective in restoring adequate arterial perfusion among diabetic patients ***(Sigala et al., 2006).***

In neuropathic foot ulcers with adequate arterial perfusion, it is important to reduce mechanical stress at the site of the ulcer**.** Pressure relief is achieved by off-loading techniques involving the application of some form of cast. The "golden standard" in off-loading techniques is the Total Contact Cast (TCC), an irremovable cast made of stockinet; low-density foam, elastic plaster and fiberglass, with arockerbottom sole ***(Papanas & Maltezos 2007).***

A useful variant is "instant Total Contact Cast", a simple cast walker made irremovable by a layer of cohesive bandage. This "instant Total Contact Cast" is much simpler to make and does not call for experienced personnel (***Armstrong et al., 2005).***

Removal of non-viable material, foreign bodies, and poorly healing tissue from a wound is called debridement and is, typically, performed surgically by means of a scalpel. Debridement aims to diminish plantar pressure by taking away excessive callus, to remove necrotic tissue, bacteria and any foreign bodies, to promote drainage of exudates and to facilitate healthy granulation. An additional advantage is that it reveals true ulcer depth