Diabetic neuropathic foot care: the team approach

Diabetes remains a major healthcare challenge. Ulceration, deep infection and Charcot arthropathy of the foot and ankle are all consequent upon diabetic neuropathy. Regular screening and patient education are essential for successful prevention of complications. **Mr Matthew Solan** and **Professor David Russell-Jones** outline how a clear referral pathway to secondary care is essential for feet that are newly ischaemic, ulcerated or swollen.

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Diabetes affects 200 million people worldwide, and by 2030 this figure is expected to double[1]. The life-threatening cardiac and renal complications are prevented by primary and secondary care physicians through optimal blood glucose control. Limb threatening complications, consequent upon vascular insufficiency and/or neuropathy, are also best treated by prevention[2]. Ulcers, infection and Charcot arthropathy are potentially devastating problems that on occasion require surgical treatment. Careful surveillance and patient education programmes keep morbidity to a minimum. Diabetic foot care is best managed through a team approach (*Tables 1 and 2, overleaf*)[3].

Diabetic neuropathy

Foot problems are the reason for 20 per cent of hospital admissions in patients with diabetes. The majority of these patients have infections from ulceration attributable to their neuropathy. The lifetime risk of ulceration has been estimated at 15–25 per cent[4]. Of the 40,000 diabetic amputations each year, 85 per cent follow from non-healing ulcers. Approximately half of all these are considered preventable[5].

Pathophysiology — cellular

The cellular mechanisms underlying neuronal injury are complex and subject to much ongoing research endeavour. Reactive oxygen species (nitric oxide, H_2O_2 and superoxide ions) are affected by hyperglycaemia, resulting in chronic ischaemia from vasoconstriction[6]; poor myelination results from diminished phospholipid production[7]; and reduced neurotrophic factor production (insulin-derived growth factor and nerve growth factor) impairs neuron homeostasis[8].

Table 1. Primary care pathway

Primary care:
> General practitioner
> Community podiatrist

Annual check:
> 10g monofilament
> Pulses
> Deformity
> Footwear

When to refer:
> New ulcer
> New swelling
> New ischaemia

Table 2. Multidisciplinary team

Secondary care:
> Secondary care podiatrist
> Diabetologist
> Microbiologist
> Vascular surgeon
> Orthopaedic surgeon
> Amputation rehabilitation
> Plaster technicians
> Surgical appliances

Painful neuropathy and mononeuropathy

These mechanisms, along with chronic hypoxia due to microvascular changes, can damage large and small fibre nerves in the lower extremity and elsewhere. Symmetrical polyneuropathy affects sensorimotor and autonomic nerves. This may produce a painful, rather than the classical painless, neuropathy. It is important to distinguish this from other causes of lower limb pain, such as plantar fascitis or fracture.

Pain from vascular insufficiency must also be considered. More proximally, compressive mononeuropathy of the common peroneal nerve causes foot-drop in the same way that carpal tunnel and cubital tunnel syndromes may present in diabetic patients from compression of median and ulnar nerves, respectively. Treatment of neuropathic pain is best organised through the pain clinic. Newer agents such as pregabalin are useful for some, but by no means all, cases of painful neuropathy.

Charcot arthropathy

In the diabetic foot, sensory neuropathy and autonomic disturbance combine to cause development of Charcot arthropathy. Poor sensation and proprioception allow repetitive minor

injuries to 'go unnoticed'; autonomically controlled blood flow to the bones is also altered reducing the strength of the subchondral plate. The effect is dislocation and fracture, commonly of the midfoot[9]. Deformity and subsequent ulceration are inevitable sequelae in untreated cases.

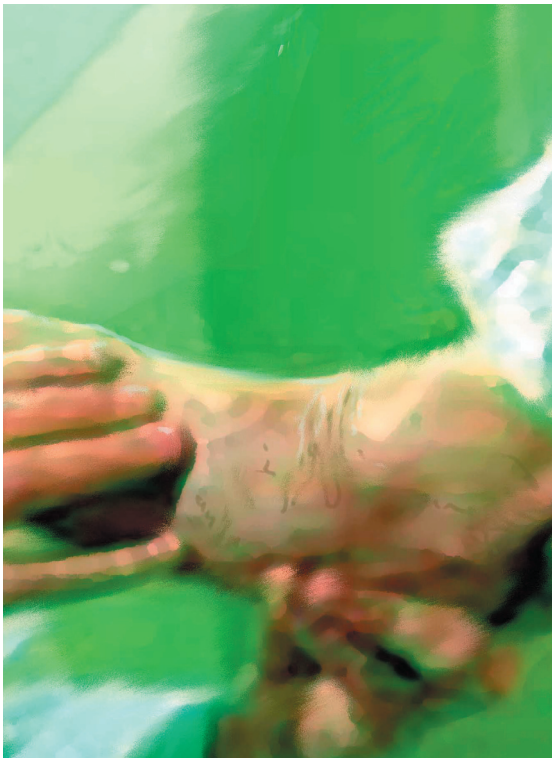
Eichenholz classified the stages of Charcot arthropathy. In Stage I, bone fragmentation predominates. The clinical picture is of swelling, warmth and erythema. It is important to distinguish this presentation from cellulitis and deep vein thrombosis. In contrast to cutaneous infection, the erythema of a Charcot foot will reduce with elevation of the extremity[10].

Stage II is the stage of coalescence, with reduced swelling and erythema. In Stage III, attempts at repair may produce a stable but often deformed foot — in effect a malunion after a fracture. Continuing instability leads to progressive skeletal destruction and disability. Bony prominences then predispose to ulceration.

Recognition is the key to successful management of the Charcot foot and ankle. Stage I limbs can be treated by total contact casting and strict non-weight bearing. The aim is to prevent collapse of the foot. Compliance is a major problem and patient education is essential for success. Weekly cast changes become less frequent as Stage I progresses to Stage II, but until the foot is cool and there is radiological evidence of coalescence non-weight bearing must be continued.

During the period of casting regular radiological surveillance allows consideration to be given to surgical stabilisation. In selected cases timely intervention to prevent loss of normal bony architecture is indicated[11].

Once the foot is cool and stable, attention turns to prevention of ulceration. Bony prominences are common even after successful total contact casting. Provision of a total contact insole, to increase the area over which weight is borne by the foot, is essential[12]. There should be no interval between contact cast and contact insole. This requires close co-operation between the providers of each. Accommodative footwear is usually required as well, since ordinary shoes will seldom hold both the foot and the orthotic. Prescription shoes allow extra room for insoles, and are available in wide fittings with deep toe-boxes to accommodate even severe toe deformities. Patients should be reassured that



the modern hospital shoe can be aesthetically very satisfactory.

Neuropathic ulcers

Ulcers are present in 84 per cent of diabetic patients undergoing amputation. The prevalence of neuropathic ulcers is as high as 15 per cent in diabetic patients over 65 years of age[10]. Counter-intuitively, the majority of neuropathic foot ulcers occur in type 2 diabetics. Sensory neuropathy is the most important risk factor. Ulcers develop in response to pressure and often begin where tissue necrosis occurs because of shear forces acting on skin and subcutaneous tissues.

These areas of necrosis may be hidden beneath calluses. Neuropathic ulcers classically form under the first or fifth metatarsal heads or the heel, as these are the weight-bearing parts of the foot. A contracture of the Achilles tendon, or an isolated gastrocnemius contracture, increases plantar forefoot pressures and is a surgically correctable cause of ulceration[13]. Ulcers may, however, develop rapidly on the dorsum, for example hammer-toes, in response to ill-fitting shoes.

Ulcer treatment is guided by classification

Traditionally ulcers have been classified according

Table 3. PEDIS ulcer classification

- Perfusion
- Extent
- Depth
- Infection
- Sensation

to their depth and the extent to which the limb is ischaemic (depth–ischaemia classification). The Wagner classification is best known, but has been subsequently modified[10]. The PEDIS system is currently used to help guide management of ulcers and associated infections (*Table 3*).

Location of the lesion in the forefoot or hind foot is also relevant. Heel ulcers respond slowly because of the relative hypovascularity of the specialised tissues of the heel pad. Forefoot ulcers that are slow to heal may benefit from surgical debridement of the underlying bony prominence; eg, shaving medial sesamoid or condylectomy fifth metatarsal head.

The established treatment of a neuropathic ulcer is to off-load the pressure area. Total contact casting is the gold standard method for this, although it is not entirely free of risk of iatrogenic complications[14]. An Achilles lengthening operation (usually performed percutaneously and in established neuropathy without any anaesthetic requirement) can reduce plantar forefoot pressure. The contracture may, however, recur[13].

Infection

All ulcers are contaminated and are portals of entry for deep infection. Not all ulcers require antibiotic treatment, however. Only if there is underlying bone exposed or significant (>2cm) surrounding cellulitis, should treatment be started[15]. Established infections are commonly polymicrobial. Swabs cannot be relied upon. Probing the depth of an ulcer gives an indication of the likelihood of underlying osteomyelitis, but not the causative organism(s).

In the presence of Charcot arthropathy neither plain x-rays, nor bone scans nor magnetic resonance imaging are sufficiently specific to reliably diagnose osteomyelitis. Biopsy and culture of deep tissues is the investigation of choice[16]. Expert microbiology advice then determines the best antibiotic regimen.

Key points

- Diabetic foot care requires a team approach.
- Annual screening must include assessment of pulses and sensation.
- Refer ulcers, ischaemia or new swelling.
- All ulcers are contaminated and are portals of entry for deep infection.
- Swelling equals Charcot arthropathy until proven otherwise.
- Routine swabs are not helpful in the management of ulcers.

Soft tissue infection, usually in the inter-metatarsal spaces, requires drainage. The ever-increasing array of sophisticated dressings are useful means of promoting healing, but only after proper debridement has removed all necrotic tissue. Vacuum-assisted wound healing is an excellent means of reducing the time taken for healthy granulation tissue to develop[17, 18].

Preventing recurrence

Once an infection or ulcer is healed the question of preventing recurrence must be addressed, and the following surgical questions asked:

- is there a contracture of the Achilles/ gastrocnemius?
- do weight bearing x-rays demonstrate bony spurs on the plantar surface beneath the ulcer?
- are foot or toe deformities so pronounced that surgical correction should be considered?

These 'local' questions should be addressed in tandem with review of blood glucose control and vascular status.

Multidisciplinary team

Identifying neuropathy and managing the sequelae is beyond the remit of any one group within the medical community. Primary care screening pathways form the cornerstone of prevention[19], but require accessible support from the hospital. Expertise is required from several quarters in the hospital and good lines of communication are the key to successful management.

Vascular and orthopaedic foot and ankle surgical advice is best sought early, to allow limb sparing surgery to be considered when non-operative treatments have failed[11]. With a co-ordinated team approach the morbidity associated with chronic ulcers and deep infections can be reduced.

Conflict of interest: none declared.

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