# Preventing chronic diabetic foot pathology from progressing to amputation: a podiatric case study

**Peters J**

## Abstract

Diabetes is one of the most potentially destructive and deforming disease processes in the human foot. The complications associated with diabetes are the primary cause of morbidity seen in hospital-based podiatry practice. Damage occurs at random in large and small blood vessels, sensory, motor and autonomic nerve fibres, often progressing to ulceration, Charcot joint disease, gangrene and limb loss. This paper takes a snapshot of one man's disease process, examining his foot complications over a 3 year period and describing the way they were managed in a hospital-based podiatry practice setting. The emphasis of the article centres on management of the mechanical causes of ulceration and the utilisation of a simple and safe procedure (a percutaneous tendo Achilles lengthening – TAL) [1, 2] to address those complications.

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## Introduction

Foot ulcers are the most common and destructive foot pathology associated with diabetes mellitus (diabetes) and they are significant risk factors for diabetes-related lower limb amputation [3]. Diabetic foot ulcers increase the risk of mortality and induce a greater utilisation of health care services [4]. The incidence of foot ulceration in people with diabetes lies between 5.3-7.8%, and the lifetime risk of foot ulceration in any diabetic patient is up to 15% [5]. The overall risk of lower limb amputation in diabetes is 15 times that of the non-diabetic population; in total, >50% of all lower limb amputations are associated with diabetes [3].

The pathogenesis of diabetic ulceration is variable, complex and often-healed ulcers will recur. The underlying aetiologies of plantar foot ulcers are multifactorial, encompassing both physiological and biomechanical factors [6]. These include what Boulton *et al.* [7] described as the "the widely accepted primary risk factor of injuries to diabetic feet" neuropathy or absence of protective pain response, whereby a combination of sensory, motor and autonomic neuropathic changes in the feet leave the foot unprotected, deforming and susceptible to trauma of skin, soft tissue and bone.

These tissue changes lead to elevated plantar pressures. Altered foot mechanics, including joint deformities, intrinsic muscle and fat pad wastage and limited joint motion, increase weight bearing pressure and are the precursors to injury due to elevated plantar pressures [8]. This repetitive pressure-induced injury can lead to the development of neuropathic fracture-dislocations in the foot, Charcot foot or Charcot joint disease. Charcot joint disease is defined as a progressive non-infective arthropathy. Joint destruction may occur in one or more joints and the surrounding soft tissue structures. Simultaneous bone and joint destruction results in fractures, dislocations and subluxations, followed by remodelling of bone and joints [9].

Peripheral vascular disease encompasses macroangiopathy (large blood vessel disease) and microangiopathy (small blood vessel disease) and causes ischaemia. Ischaemia results

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in hypoxic skin changes with impaired potential for healing and decreased resistance to infection [10]. Other underlying factors contributing to the development of ulceration include previous ulceration or amputation, loss of vision and poor glycaemic control. The major risk predictors for lower limb amputation are severe ischaemia, infection and wound depth [11]. Managing diabetic foot ulceration should be directed into three areas; controlling foot infections [12], reducing peripheral ischaemia [5] and controlling elevated plantar pressures.

The medical dictionary definition of ulceration is a "persistent breach in an epithelial lining". It is the clinician's primary task to heal that breach and prevent access to potentially limb threatening pathogens [11, 13]. Oral antimicrobial agents as first line management for infection, followed by hospitalisation and intravenous antimicrobial agents in the case of cellulitis is accepted practice [10]. In the author's opinion, topical management of the wound is equally important; debridement, irrigation, wound dressings and topical antimicrobials can all reduce wound bioburden and the likelihood of an acutely infected wound becoming cellulitic.

Peripheral vascular disease in diabetes encompasses macroangiopathy or atherosclerosis in large blood vessels [5]. Microangiopathy is a decrease in small blood vessel lumen diameter, due to thickened basement membranes, intimal scarring or thrombus formation. Vasomotor neuropathy is impairment of microvascular circulation to neurovascular tissue. These changes leave the ischaemic foot with thin, dry, atrophic skin, hypertrophic nails, absent hair growth and susceptible to trauma. Reducing peripheral ischaemia entails appropriate footwear and sock/stocking, establishing normoglycaemia and, in severe cases, revascularisation by a vascular surgeon.

It is generally accepted that the diabetic foot with neuropathy demonstrates higher plantar pressures [6]. Peripheral neuropathy, muscle atrophy, fat pad wastage, joint deformity, weight bearing pressure or shearing stress, with or without ulceration, result in elevated plantar pressures [11, 14, 15]. Controlling elevated plantar pressures by offloading compromised areas entails a range of options; these include callous reduction, padding and strapping, insoles and orthoses, customised and custom made footwear, walking cast boots, total contact casts [5] and surgery [1, 2].

## Case study

### Medical history

Mr A is a 54 year old man who was diagnosed with type 2 diabetes mellitus 12 years ago. He weighs 126kgs, and takes the following medication – Diaformin (100mg bd), Norvasc (10mg), Coversyl Plus (4mg), Amaryl (3mg), and 0.5 aspirin. He has no known allergies and at the time of referral he was taking Dicloxacillin 500mg qid.

### Presentation to the podiatry department

Mr A presented on 25 July 2000 at the podiatry department of Sir Charles Gairdner Hospital. He was referred by his consultant endocrinologist for management of acute injury related ulceration under the plantar aspect of his left 1st metatarsophalangeal joint (Figure 1). The lesion was 10mmx12mm in diameter, demonstrated a low-grade peripheral cellulitis and was surrounded by macerated callous at its margins.

The ulcer base demonstrated healthy granulation tissue and a mildly malodorous serous discharge was swabbed for microorganisms. Swab results identified an abundant growth of *Staphylococcus aureus*, which was sensitive to Flucloxacillin.

An initial neurovascular assessment of the feet found normal macrovascular perfusion with sound pulses and normal ankle brachial indices, and adequate microvascular perfusion in both feet. Moderate sensory neuropathy in the form of distal digital peripheral neuropathy, with an absence of perception to a 5.07 Semmes Weinstein monofilament (10g) and vibratory stimulus (Biothesiometer) was found. Motor neuropathy with a complete absence of ankle jerk reflex in both feet was observed. Biomechanically, both feet were mildly pronated, with bilateral equinus deformities at the ankle joints and mild anterior cavus at the midtarsal joints. His gait was appropulsive, with an early heel lift and significant abductory twist just prior to toe off.

The ulcer was aggressively debrided to healthy tissue, liberally irrigated with saline solution, dressed with a Povidone Iodine impregnated gauze and covered with foam dressing. Pressure was offloaded from the area with a 10mm self-adhesive orthopaedic felt pad, which ran proximal to the ulcer back under the first metatarsophalangeal joint.

Over the following weeks the ulcer was debrided weekly and dressings were applied second daily with the assistance of Silver Chain, a domiciliary service. The infection did not improve; the neuropathic nature of the foot meant that Mr A had no protective pain response and he continued going to work and weight bearing on the foot.

Three weeks post presentation the foot became oedematous, blisters appeared on the apex of the first, second and third digits and under the first metatarsophalangeal joint.

Mr A was hospitalised for 7 days to manage this episode of acute cellulitis (Figure 2). He was kept non-weight bearing, treated with intravenous antibiotics and all wounds were treated topically. Blisters were drained and covered with alginates and transparent films. On discharge, Mr A was managed orally with Ciprofloxacin and Clindamycin antibiotics. He was seen in the podiatry department weekly to manage ulcerations on the apicies of first, second and third digits and under the left first metatarsophalangeal joint and interphalangeal joint. We manufactured a removable cast boot (a total contact cast was not appropriate due to the large amounts of oedema in the foot and ankle and the ankle equinus), which fitted into a Darco$^{TM}$ post-op boot (Figures 3 & 4).

Nine months after initial presentation, with four changes of cast boot, fortnightly wound management and dressings, all ulcers were healed. At this point, in May 2001, we took the opportunity to take plaster casts of both feet to manufacture functional orthoses and custom-made footwear. These were manufactured and issued within 14 days of assessment. Mr A was then reviewed monthly and the ulceration sites were reassessed; they remained healed and both the orthoses and shoes provided good functional support.

In October 2001, Mr A presented again with a hot, swollen, oedematous left foot. There were no skin lesions and a diagnosis of Charcot joint was postulated. Radiographs identified the first metatarsocunieform joint as the site of breakdown and Indium scan confirmed the diagnosis, without the presence of osteomyelitis. At this stage the left foot was recast in a walking cast boot to prevent the deformation associated with the disease process. Mr A was reviewed fortnightly with no change, until in November when he stubbed his toes and developed lesions over the left first and second digital apicies.

## Development of a Charcot joint

The next 12 months saw the foot dressed fortnightly, with changes to the cast boot (repairs and replacement), resolution and then reulceration due to neuropathic weight bearing. Despite our regular attention, the deforming complications associated with soft tissue glycosolation progressed, with maximal pronation of the left foot; this resulted in neuroarthritic osteopenic collapse of the first metatarsocunieform joint (Charcot joint), enabling the arch to contact the ground (Figures 5 & 6). This ground contact increased plantar pressure under the Charcot joint. Eventually and inevitably, we came to the point where, after a short period of unshod weight bearing (to the ablutions), blistering occurred under the joint (Figure 7).

This pronatory twisting motion of the foot caused the first, second and third digits to become retracted and clawed, due to shortening of the tendons. The apicies of each of these digits contacted the ground on weight bearing and similarly started blistering. At this point of time, in December 2002, Mr A was attending the clinic on a weekly basis; we were spending 30 minutes debriding lesions and dressing and padding the foot. We had him in a pair of $1500 custom-made surgical boots, with functional orthoses.

In February 2003, options for realigning the foot and to resolve his now chronic ulcerations were discussed with Mr A. The Charcot joint deformity had significantly deformed the foot and was now quiescent. Fortunately, the foot itself

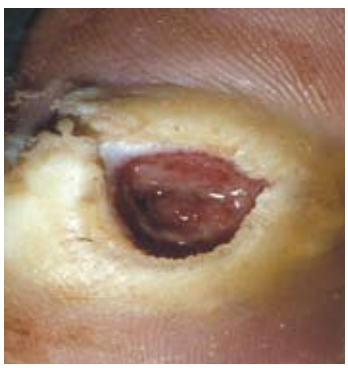

*Figure 1. Neuropathic ulceration under the left first metatarsophalangeal joint.*

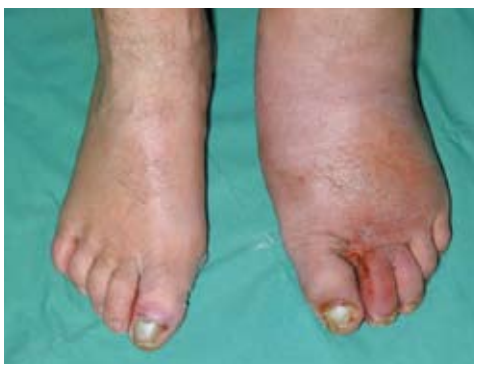

*Figure 2. Cellulitic infection of the left foot, post ulceration of first metatarsophalangeal joint.*

*Figure 3. Custom-made removable cast boot.*

was still remarkably mobile. We thoroughly examined the deformity and concluded that the primary deforming force was the ankle equinus. On maximum active dorsiflexion the foot would not dorsiflex past 100 of plantarflexion (Figure 8).

Two recent research papers looked into the effects of tendo Achilles lengthening (TAL) and discussed the minimally invasive option of percutaneous lengthening of the tendon [1, 2]. The findings of these papers were applicable to our needs; the first found a significant reduction in forefoot pressure after TAL [1]. The second concluded, after TAL, "ulcer recurrence was 75% less at 7 months and 52% less at 2 years..." and that TAL "should be considered an effective strategy to reduce recurrence of neuropathic ulceration of the plantar aspect of the forefoot in patients with diabetes mellitus and limited ankle dorsiflexion" [2].

The digital retraction deformities included the hallux being forced into a hammertoe position and the lesser digits all being clawed. We discussed the option of correcting all of these digital deformities once the TAL was healed and working functionally.

## Surgery performed on the foot

On 13 March 2003, a subcutaneous triple hemisection of the tendo Achilles was performed (Hoke procedure) – Figure 9 and post-operative Figure 10 [16]. This procedure was performed under local anaesthetic, with a load dose of 2gms of Cephalexin 2 hours pre-operatively and two thorough Hexachlorophene foot and leg scrubs in the 12 hours preceding the procedure. Mr A was sent home wearing an Aircast walker, with limited weight bearing over the following 48 hour period (Figure 11.)

Sutures were removed from the TAL 11 days post-operatively; all plantar digital and forefoot ulcerations were resolved within 4 weeks of surgery. Mr A was kept in the Aircast walker for 6 weeks post-operatively, at which time he was prescribed an off-the-shelf pair of depth boots (Figure 12).

*Figure 4. Darco post-operative shoe, encompassing cast boot.*

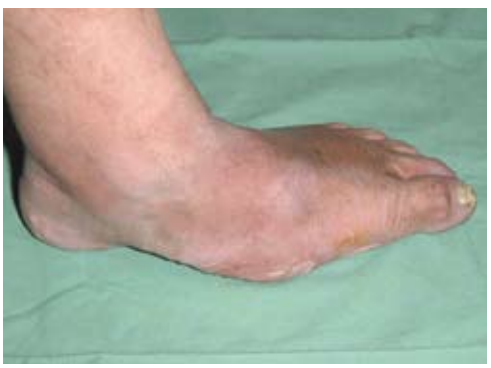

*Figure 5. Charcot joint changes, lateral view of foot.*

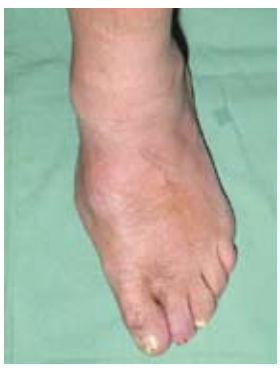

*Figure 6. Dorsal view of foot.*

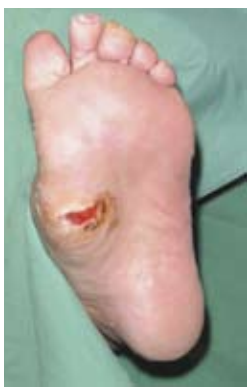

*Figure 7. Charcot joint plantar view, overlying blister.*

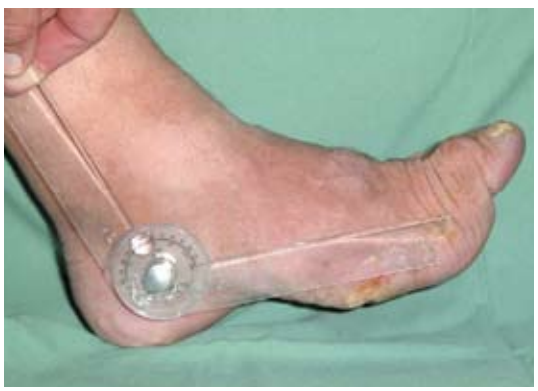

*Figure 8. Ankle equinus, the foot would not dorsiflex past 100 plantarflexed.*

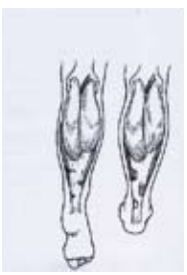

*Figure 9. Diagram of percutaneous TAL procedure.*

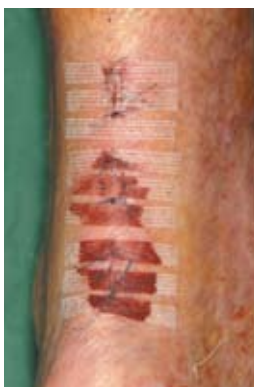

*Figure 10. Post-operative view of three longitudinal incisions used to perform TAL.*

On 8 December 2003, Mr A underwent surgery to his hallux, second and third digits (Figure 13). The hallux was re-aligned with an extensor tenotomy and an interphalangeal joint fusion. The second and third digits were realigned with simple flexor tenotomies. These surgeries were again performed under local anaesthetic with oral Cephalexin and topical Hexachlorophane anti-microbial management. They healed in 14 days without complications (Figure 14). The remaining fourth and fifth digits were realigned on 8 April 2004. Simple flexor tenotomies were performed under local anaesthetic, utilising the above anti-microbial protocol (Figures 15, 16 & 17).

## Discussion

This study highlights the multifactorial nature of diabetic foot complications and the progressive nature of the disease process, including pedal complications such as peripheral neuropathy, peripheral vascular disease, impaired healing, ulceration, intrinsic muscle atrophy, fat pad wastage, joint deformity, increased weight bearing pressure and Charcot joint disease. These complications, which may occur in any combination, are further compounded by systemic disease changes, including retinopathy, nephropathy and cognitive dysfunction.

*Figure 11. Aircast walker.*

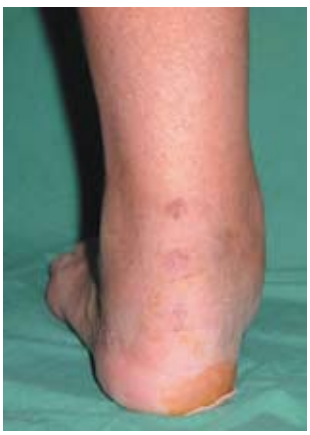

*Figure 12. Incision site, 4 weeks post-operatively.*

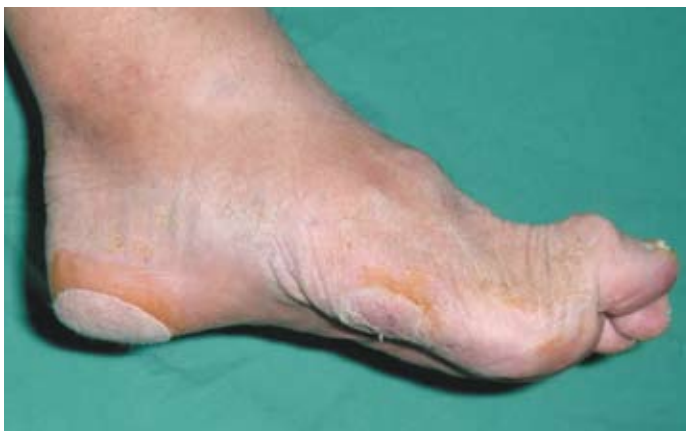

*Figure 13. Digital deformities, hallux hammertoe and retracted lesser digits.*

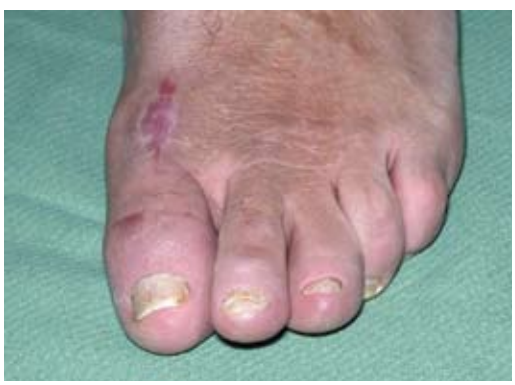

*Figure 14. Dorsal view of foot 14 days post-operatively.*

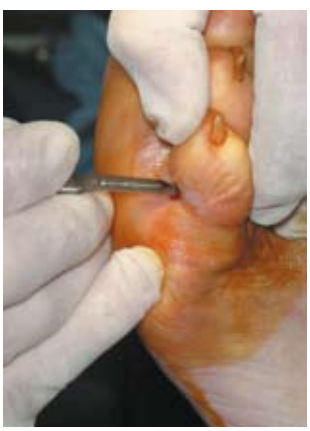

*Figure 15. Flexor tenotomy (under local anaesthetic, a stab incision is made under the proximal interphalangeal joint).*

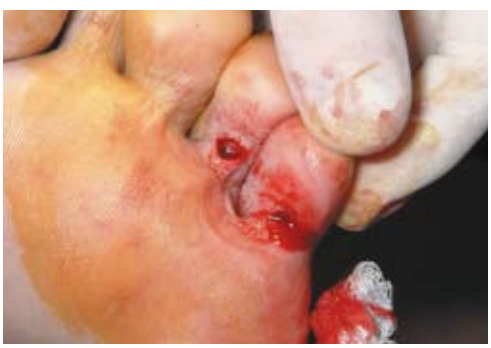

*Figure 16. Flexor tenotomy (the flexor tendon is transected and the toes are then straightened).*

We need to understand that each diabetic patient is different in the mix and severity of complications and therefore requires individual treatment management regimes. This diversity and the extent to which the diabetic epidemic has affected the community demonstrates the need to work together in multidisciplinary teams to manage the workload. We need a full armoury of screening tools, treatment and management options and education strategies to save limbs.

There is a perception that surgery of the diabetic foot is fraught with danger. This is verified by the reluctance of many health care workers to trim the nails of the diabetic patient. This article demonstrates that sound assessment of the underlying etiologic factors causing foot pathology, coupled with an absence of vascular complications and controlling for infection, surgery is a viable option for managing diabetic foot complications.

## Conclusion

This study is only a snapshot in the ongoing management of this patient. Have no illusions; these simple surgical procedures will never be a panacea to all Mr A's future foot complications. The need to provide ongoing regular maintenance, refurbishment of footwear and orthoses, annual screening and emergency care are ever necessary. The fact that this intervention was able to increase the patient's independence, reducing the need for weekly ulcer dressing changes, enabling him to purchase footwear off the shelf and extending the period between regular visits to eight weekly, are all very positive facts. To achieve positive change in a chronically diseased foot, improving it from its state in 2000 to where it is now in 2004, is immensely rewarding (Figures 18 & 19).

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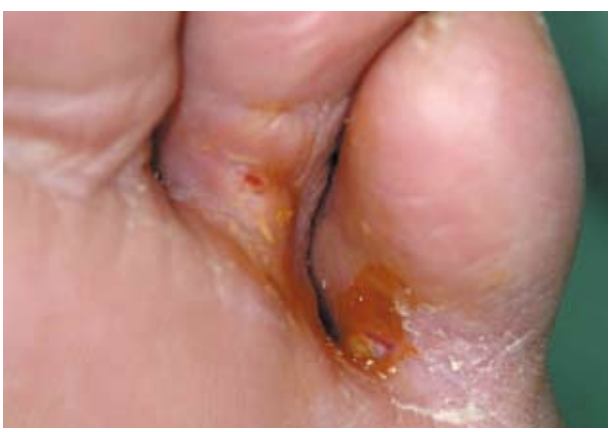

*Figure 17. 14 days post-operatively, incision sites are healed.*

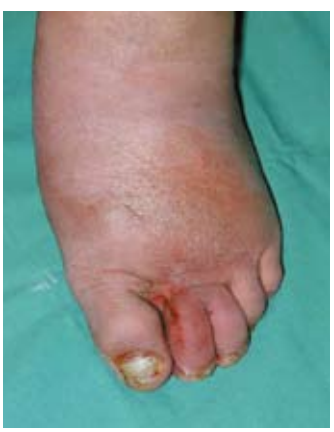

*Figure 18. August 2000.*

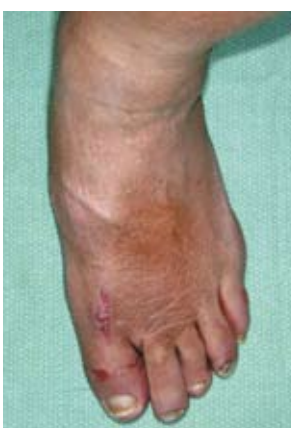

*Figure 19. June 2004.*