From 'minor ulcer' to complex wound:
management of a patient with a neuro-ischaemic foot ulcer complicated by verrucous hyperplasia

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Abstract

Diabetic foot ulceration can present a major challenge to healthcare professionals. Even very small lesions have the potential for a disastrous outcome; particularly those overlying joints. This case study discusses the management of a 60-year-old male Maori with type 2 diabetes and a neuro-ischaemic foot ulcer, who underwent a 1st ray and 2nd toe amputation followed by a split skin graft. Friction to the graft site led to the development of verrucous hyperplasia; a hyperkeratotic, papillomatous verrucous lesion. Despite excision and regrafting, the lesion recurred. Structural changes to the foot caused a new foot ulcer to develop. In this paper the impact of the complications of diabetes on the patient's feet and his mobility as well as the psychosocial factors related to his foot ulceration and foot deformity will be discussed.

Case study

History

George, a 60-year-old Maori male mental health caregiver, with type 2 diabetes of 8 years' duration and poor glycaemic control, was referred to an outpatient diabetic foot ulcer podiatry clinic by his GP in August 2004 with a small foot ulcer overlying his R1st metatarsophalangeal joint (MTP) which had been present for 6 weeks. He had no history of ulceration. His comorbidities included hypertension, hyperlipidemia, chronic renal impairment and moderate retinopathy. He was blind in his left eye due to an accident. Previously a heavy smoker, he was trying to give up. His medication included Protophane 10u bd, Cartia od, Lipex 40mg od, Losec 20mg bd and Betaloc od.

Examination

Examination revealed sparse hair growth, anhidrotic skin, onychomycosis and tinea pedis with interdigital fissuring. Dorsalis pedis and posterior tibial pulses were not palpable and capillary refill was sluggish. Intermittent claudication was reported after walking 200 metres. There was no venous insufficiency or oedema. An Ankle Brachial Pressure Index (ABPI) was not carried out at the initial assessment. ABPI results do need to be interpreted cautiously in patients with diabetes due to vascular calcification and toe pressures are generally more reliable [1].

Bilateral hallux abducto-valgus, clawed toes and anterior displacement of the plantar fat pad were observed. Limited joint mobility including rigid, plantarflexed 1st rays and ankle equinus were also noted. A 10-gm monofilament test revealed loss of protective sensation.

An area of macerated, haemorrhagic callus was present overlying the plantar aspect of his R1st MTP. There was hemoserous strike-through on the dressing but no clinical signs of infection. Debridement revealed a small, granulating area of ulceration; Texas classification [2] (grade 1A, category 2), measuring 4mmx3mm, which could be probed to 3mm

Treatment

As diabetic ulcers are usually heavily colonised [3], it was decided to use an antimicrobial dressing. Acticoat™ (Smith & Nephew) and Telfa™ (Kendall) were applied. A temporary, redistributive insole was fitted and he was referred to the orthotic centre for orthoses and extra depth footwear. He was strongly advised to give up smoking because of its known detrimental effects [4]. Lamisil spray was prescribed to treat the interdigital tinea. Follow-up dressings were arranged with the district nurses to be changed every 3 days. A referral was made to the vascular surgeon.

Weekly follow-up appointments were planned for debridement, to monitor the wound and contra-lateral foot

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and to check the offloading. He was managed by a diabetes nurse specialist, to improve his glycaemic control. At his follow-up appointment the ulcer had healed.

He returned to the clinic 3 weeks later, wearing a pair of scuffs with no dressing in place and said his shoes and insole had been stolen while on an overseas holiday. The foot had re-ulcerated, although the ulcer was superficial. He had returned to smoking four cigarettes a day. An HbA1C of 8.3% indicated ongoing suboptimal control.

At his next 3-week follow-up appointment, the ulcer had deteriorated and was highly exudating and malodorous. The wound bed could be probed to 7mm. The margins were undermined, granulation tissue was friable and there was erythema overlying the 1st MTP, with cellulitis extending 4cm proximally. He had finally given up smoking.

Deep tissue swabs were taken, Augmentin was prescribed and dressings were changed to Aquacel AG™ (ConvaTec) and Alleyvn™ (Smith & Nephew) to absorb exudate and prevent further maceration and undermining. Swabs cultured a heavy growth of coagulase negative *Staphylococcus aureus*. He was fitted into an Orthowedge healing shoe (Darco®) (Figure 1) and a referral was made to the diabetes consultant to review his management. At his vascular appointment, an arterial duplex showed occlusion of the distal femoral, popliteal, posterior tibial and anterior tibial arteries. He was waitlisted for revascularisation.

Figure 1. Orthowedge Healing shoe (Darco®).

He continued to be managed at the foot ulcer clinic on a 3-weekly basis for a period of 18 months while awaiting revascularisation, with district nursing visits in between. His priority for revascularisation remained low due to the small size of the ulcer and the considered risk factors associated with vascular surgery. There were two further episodes of cellulitis, treated with oral antibiotics. He developed tape allergy and was prescribed Locoid cream. The ulcer continued to increase in depth and developed a sloughy base. It was not possible to probe to bone and x-rays showed no evidence of osteomyelitis. A number of dressings were used during this period including Acticoat™ (Smith & Nephew), Curafil™ (Kendall)/Adaptic™ (Johnson & Johnson) and Iodosorb™ (Smith & Nephew).

George returned to smoking seven cigarettes per day. He was experiencing significant back pain due to the functional limb length difference caused by the Orthowedge healing shoe (Darco®), so he was fitted into a Wound Healing Shoe System (Darco®) (Figure 2) with a cavity at the 1st MTP.

Figure 2. Wound Care Shoe System (Darco®).

In February 2006 he was booked into hospital for inguinal hernia surgery and developed a deep tissue infection of his foot ulcer leading to extensive necrosis (Figures 3-4). He underwent revascularisation followed by an amputation and split skin graft (Figure 5) harvested from his right thigh.

George was issued with a modified postoperative sandal to prevent shearing forces and graft loss; however, he arrived at his follow-up appointment, in June 2006, wearing a pair of lace-up shoes. An area of macerated tissue was present,

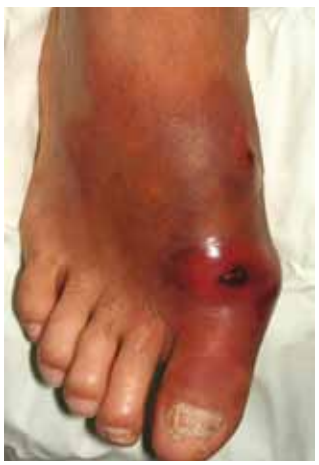

Figure 3. Deep tissue infection.

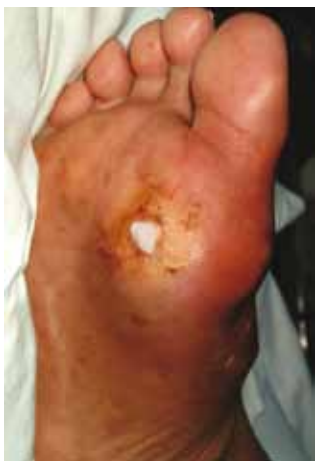

Figure 4. Initial ulceration with deep tissue infection.

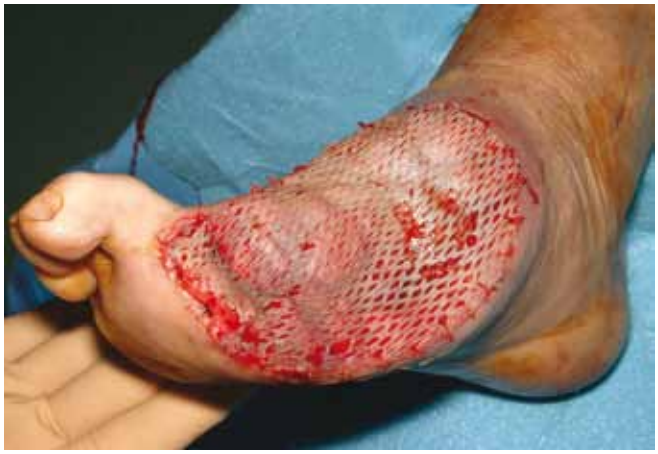

Figure 5. Split skin graft.

with hypertrophied papillae measuring 4cmx3cm and an underlying superficial ulcer at the distal aspect of the graft site measuring 2cmx4mm (Figures 6 and 7). A rolled up piece of plastic was found inside his right shoe.

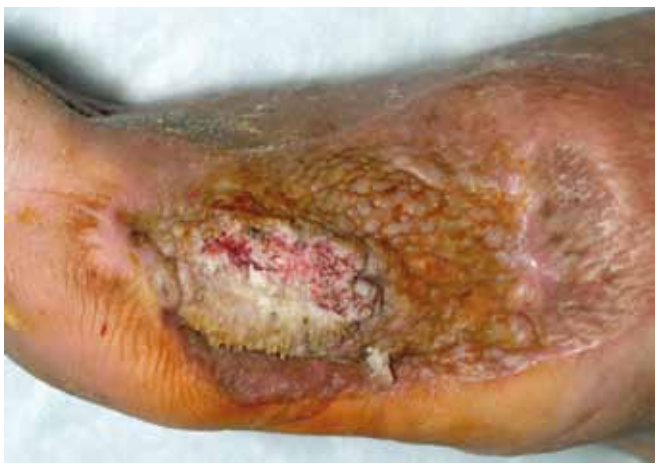

Figure 6. Macerated tissue with hypertrophied papillae.

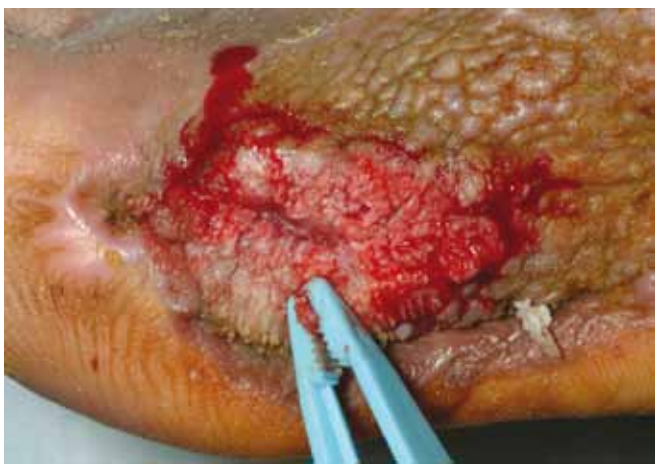

Figure 7. Superficial ulceration exposed after debridement.

Multiple biopsies did not show any particular pathology or organism. The lesion continued to grow and was later diagnosed histologically as verrucous hyperplasia (Figures 8 and 9).

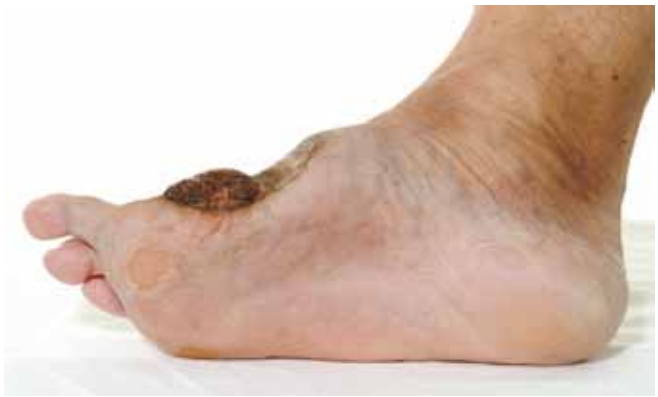

Figure 8. Verrucous hyperplasia.

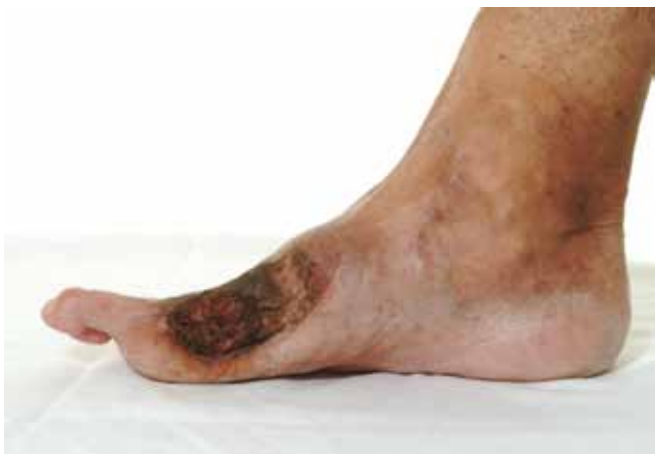

Figure 9. Verrucous hyperplasia.

Despite various treatments including offloading, debridement, excision and regrafting, the condition recurred. Structural changes led to a new foot ulcer under his R3rd MTP and he was referred for total contact casting. The ulcer healed and later re-ulcerated; however, the verrucous hyperplasia completely resolved (Figures 10-11). In September 2009, he underwent orthopaedic surgery including a 3rd metatarsal shortening osteotomy, extensor digitorum longus tendon lengthening and gastrocnemius slide. The outcome of this procedure is not yet known.

Impact of diabetes on the foot

There is evidence to suggest a strong link between persistent hyperglycaemia, peripheral neuropathy and the development of a number of other pathological changes which could have increased George's risk of developing a foot ulcer [5]. Smoking has been linked to peripheral neuropathy [6] and to insulin-

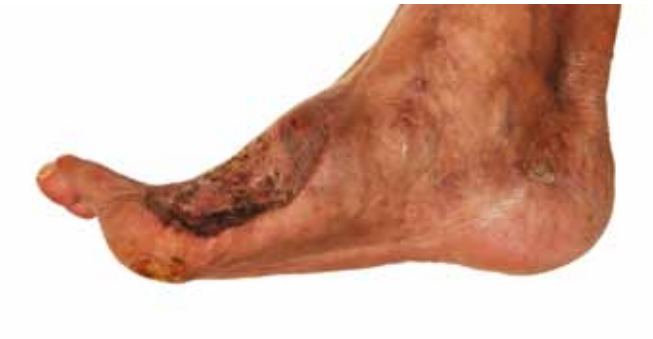

Figure 10. Re-ulceration 3rd MTP (NB Verrucous hyperplasia resolved after TCC).

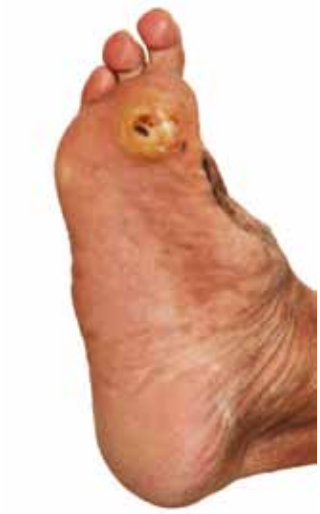

Figure 11. Re-ulceration 3rd MTP plantar view.

resistance [7]. Hyperlipidaemia and hypertension have also been linked to insulin resistance [8].

George presented with ankle equinus, which could have been associated with his neuropathy. This is supported by a study by Williams *et al.* [24], which demonstrated less ankle joint mobility in patients with diabetes and peripheral neuropathy [16], during the late stance phase of gait, compared with diabetic patients without peripheral neuropathy.

Tissue changes

Motor neuropathy can cause intrinsic muscle weakness, resulting in claw toe deformity and anterior displacement of the fat pad [9]. One of the signs of autonomic neuropathy is decreased sweating, resulting in anhidrosis and reduction of the skin's visco-elastic and anti-frictional qualities [10]. Sensory neuropathy is a precursor to loss of protective sensation [11].

Loss of protective sensation and limited joint mobility caused elevated peak plantar pressure under the 1st MTP, leading to the formation of callus, which was further exacerbated by his retracted toes and thin sub-metatarsal fat pad [12]. Loss of protective sensation caused unperceived chronic pressure on his nails from his footwear [13].

Peripheral neuropathy is also associated with loss of foot muscle volume [9] and an increased thickness of the plantar fascia, causing reduced dorsiflexion of the hallux [14].

Verrucous hyperplasia

Verrucous hyperplasia presented as an area of macerated, friable, 'warty' tissue over the graft site. Although this condition frequently occurs in patients with diabetes with neuropathy over amputation sites [15], very little is reported on it in the literature. Verrucous hyperplasia has also been termed lymphostatic or stasis papillomatosis and aetiologies include friction, severe stasis dermatitis, poor prosthetic fit and alignment and bacterial infection [16].

As verrucous hyperplasia recurs after removal, compression therapy and the reduction of shearing forces, rather than excision, are indicated [16]. It is also necessary to carry out a biopsy to differentiate this condition from verrucous carcinoma [16]. In George's case, total contact casting proved to be an effective method of removing shearing forces and the lesion totally resolved.

Ischaemia

George presented with numerous established risk factors for peripheral vascular disease, which resulted in reduced blood flow to the foot, including diabetes, smoking, hypertension, hyperlipidaemia, advanced age and the presence of peripheral neuropathy [17-18].

He had a long history of hyperglycaemia, which can cause endothelial cell and extra cellular matrix dysfunction, increased permeability of blood vessels and an inelastic wall, affecting blood flow [14]. He presented with a number of signs of ischaemia including cool, anhidrotic skin, sparse hair growth, prolonged capillary refill time and absent foot pulses [19].

Weight-bearing forces, in conjunction with callus can cause an impaired hyperaemic response leading to foot ulceration [6]. Hyperglycaemia decreases oxygen exchange, damaging the autonomic nervous system and leading to shunting of oxygenated blood [19].

Impaired wound healing

Diabetes is associated with impaired wound healing [20]. Hyperglycaemia has been shown to cause reduced leukocyte function, impaired collagen synthesis, decreased strength of collagen and impaired angiogenesis [14]. George's initial foot ulcer healed but re-ulcerated partly due to ischaemia. While his subsequent foot ulcer healed with total contact casting, the tissue remained vulnerable and re-ulcerated.

Infection

George presented with onychomycosis, which affects approximately 34% of patients with diabetes [21]. His skin was at risk of trauma and secondary bacterial infection from his thickened nails. Tinea pedis is also more prevalent in patients with diabetes and can create portals of entry for bacterial invasion [13]. Anhidrosis results in a loss of skin integrity, providing an ideal environment for infection [14].

The location of George's foot ulcer, overlying a joint, increased the risk of deep tissue infection and osteomyelitis [3]. When his foot ulcer deteriorated while in hospital, urgent revascularisation was necessary, followed by a 1st ray and partial 2nd ray amputation to remove all infected tissue and prevent more proximal limb loss [3]. The infection may have been initially overlooked, as the cardinal signs of infection are sometimes absent in patients with diabetes [11].

Impact on mobility

Peripheral neuropathy can affect gait, causing reduced walking velocity, reduced cadence and step length, shorter stride length, less ankle motion and less rhythmic acceleration [22]. Motor neuropathy affects the muscles required for normal foot movement and alters the distribution of forces during walking, contributing to the formation of callus [14]. Peripheral neuropathy leads to impaired peripheral sensation, reaction time and balance, increasing the risk of tripping [23].

Gait changes can be seen with peripheral vascular disease, including leg fatigue, reduced walking speed and duration of walking; however, claudication is less common in patients with diabetes due to peripheral neuropathy [19].

A study by Maluf *et al.* [25], found that measurement of peak plantar pressure during level walking was an efficient method to screen for maximum levels of stress on the foot when patients with diabetes and peripheral neuropathy perform activities of daily living.

George developed callus over his 1st MTP due to limited joint mobility, elevated peak plantar pressure and prolonged, repetitive, shearing and frictional forces during walking [10]. The callus initially protected the tissue from pressure damage, but eventually caused additional peak plantar pressure [6]. George did not demonstrate an antalgic gait and developed a foot ulcer beneath the callus, due to loss of protective sensation and his impaired circulation.

Exercise assists with hyperlipidaemia, hypertension, metabolic control and depression [13]. Patients with diabetes are recommended to participate in daily physical activity [26]. However, in order for George's foot ulcer to heal, it was essential to remove all pressure and stop his weight-bearing activities [3].

George's partial foot amputation caused a decrease in the surface area of the foot and altered foot function. Due to limited joint mobility, further alterations of peak plantar pressure occurred during walking and an ulcer developed over the plantar aspect of the R3rd MTP, placing him at risk of further amputation [14].

Some patients with diabetes and peripheral neuropathy, particularly those with prior foot surgery and severe deformity, have such high peak plantar pressure they can ulcerate after just a few steps barefoot [27].

Psychosocial impact

The economic impact of foot ulceration on patients with diabetes has been well-documented [8]. Depression is more common, especially in those with peripheral vascular disease and peripheral neuropathy [28]. Hyperglycaemia and hypoglycaemia have been linked to a decline in cognitive function [13].

Socio-economic factors influence the occurrence of foot problems, particularly in the Maori population [8]. Health-related quality of life is influenced by effective metabolic control and positive mental health [29] and decreases in relation to the number of complications [30].

A qualitative study by Kimmond *et al.* [29] highlighted that psychosocial factors impact greatly on health-related quality of life for patients with diabetes living with a foot ulcer. This can lead to "poor healthcare beliefs, poor self-care behaviour and attitudes and a lack of self-awareness" [18]. Reactions of patients may be influenced by the healthcare they receive and lead to "feelings of helplessness and a lack of control" [31].

Non-weight-bearing therapy can have an extremely negative effect on health-related quality of life [29]. In an effort to regain some sense of efficacy, patients with diabetes can rebel against the restrictions placed on their lifestyle [30].

George was part of a supportive family. To a certain extent, he had good self-care behaviour. He attended all podiatry appointments, attempted smoking cessation and demonstrated meticulous foot hygiene. However, he was forced to take leave, became tired of clinic appointments and was embarrassed by the appearance and smell of his foot. The offloading shoe caused him significant back pain. His restricted life, associated with his foot ulcer and reduced mobility, led to a "loss of sense of self" [29].

George showed signs of intense anger and frustration and returned to smoking. While he arrived at his appointments wearing his offloading shoe, it became evident he was wearing shoes of his choice outside the clinic. He eventually appeared defeated and bluntly refused to return to any offloading modality, saying "what will be, will be".

Conclusion

George had several of the known risk factors for foot ulceration, including loss of protective sensation and limited joint mobility, leading to altered foot mechanics and elevated peak plantar pressure. The development of his foot ulcer, combined with peripheral vascular disease and loss of protective sensation, meant he was at a greater risk of amputation. George's foot ulcer, the subsequent amputation and the growth of verrucous hyperplasia impacted on his health-related quality of life. A more holistic approach to his management might have resulted in a better outcome. As little has been published on verrucous hyperplasia, this condition could form the basis of a further study.

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