Severe thermal injury of the neuropathic diabetic foot: A case report

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Article points

1. Thermal injuries are a common cause of ulceration in the diabetic foot and are often the result of unusual aetiologies.
2. This case describes a thermal injury sustained by a 47-year-old man with diabetic neuropathy.
3. Thermal injuries of the neuropathic foot should be considered preventable.
4. People with diabetic neuropathy should be offered education on how to protect their feet from injury, including avoiding high temperatures.

Key words
- Burns wound
- Diabetic neuropathy
- Thermal injury

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Thermal injuries are a common precursor to ulceration and infection in the neuropathic diabetic foot. Here, a case of thermal injury sustained in a steam shower in a 47-year-old man with type 2 diabetes and neuropathy is reported. Thermal injuries among those with diabetic neuropathy should be considered preventable and the avoidance of heat should be highlighted in patient education.

Thermal injuries have been described as a cause of injury and ulceration in the neuropathic foot, and are often the result of uncommon aetiologies (Balakrishnan et al 1995; Thng et al, 1999; Al-Qattan, 2000; Jose et al, 2005; Putz et al, 2008). The authors present a case of thermal injury of the neuropathic diabetic foot following a steam shower. The implications of such injuries are discussed.

Case study

Patient history

A 47-year-old white European man, Mr F, with type 2 diabetes (duration 8 years) was first referred to the authors' diabetes clinic 2 years following diagnosis, at which time his HbA_{1c} level was 11% (97 mmol/mol) and treatment with oral antidiabetes agents was intensified (repaglinide added to metformin).

Mr F's compliance with lifestyle advice was poor and his alcohol consumption high. Rosiglitazone was added to his regimen in 2007 but his glycaemic control worsened and insulin therapy was initiated during a hospital stay for foot ulceration in 2008.

Between 2007 and presentation for the present case, Mr F sustained numerous minor injuries to his toes – typically the result of ill-fitting shoes or trauma from objects encountered while walking – resulting in minor, non-chronic episodes of ulceration. During this period, Mr F also experienced three episodes of major ulceration to the left 3rd and 4th toes, the right hallux and latterly the right 2nd, 3rd, 4th and 5th toes. The first episode healed uneventfully following a 4-week course of antibiotics. The second and third episodes occurred in quick succession and healed, the latter requiring hospitalisation. Throughout this period Mr F remained under regular review by the podiatry team.

Clinical assessment of Mr F's lower limbs revealed patchy neuropathy to the mid-shin and absent vibration perception to the medial malleoli bilaterally. There was no evidence of peripheral arterial disease, with strong foot pulses and ankle–brachial pressure indices >1.0 in both feet.

Presentation

Mr F presented with a thermal injury to his left forefoot in November, 2009. Ulceration

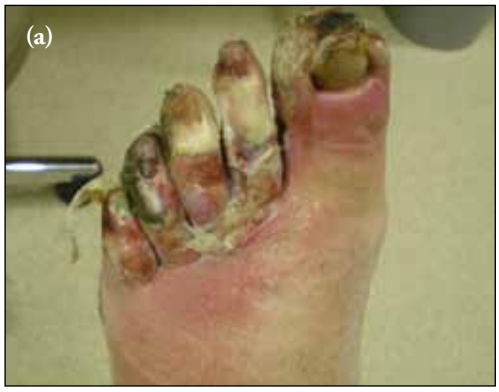


Figure 1. Mr F's left foot at presentation, the (a) dorsal and (b) plantar aspects are shown. Note the surrounding cellulitis, blackened toes.

extended to both the dorsal and plantar aspects of the foot with surrounding cellulitis, blackened toes and purulent exudates (*Figure 1*). Mr F reported noticing blistering on his foot after attending a sauna for a steam shower 1 week earlier.

Mr F was referred immediately for inpatient care under the joint management of the diabetes and plastic surgery teams. He was commenced on intravenous benzylpenicillin and flucloxacillin, his feet were elevated and the ulcerated foot was dressed with a dry, non-adherent dressing (Inadine; Systagenix, West Sussex).

Surgical review excluded vascular insufficiency and confirmed the appropriateness of conservative management. Wound swabs revealed Group G *Streptococcus*, *Pseudomonas* species and coliforms sensitive to the prescribed antibiotic regimen. Radiography showed no evidence of osteomyelitis.

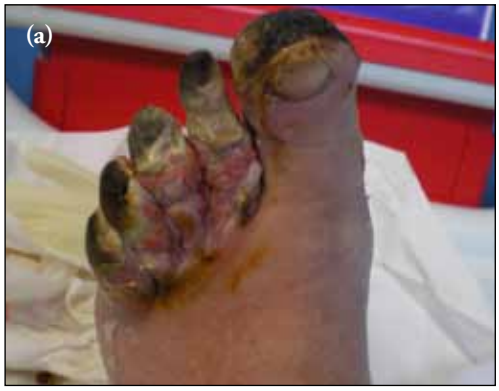
Four days after presentation and admission, erythema had reduced and medicated dressings were discontinued in favour of Melolite (Smith & Nephew, Hull) and Allevyn (Smith & Nephew) to encourage epithelialisation.

Intravenous antibiotics were administered continuously during Mr F's 2-week inpatient stay. Dressing changes were daily. *Figure 2* shows Mr F's left foot at the time of discharge, at which time he was switched to oral antibiotics for a further 2 weeks.

Mr F was followed-up in the podiatry clinic and *Figures 3* and *4* illustrate the continued healing of the foot. At the time of writing, the 5th toe was completely healed and the rest of the wound was progressing to healing.

Discussion

Aetiologies of diabetic foot ulcers differ, but untreated they may result in severe morbidity and mortality, with 5-year mortality rates following new-onset ulceration resulting in a lower-limb amputation as high as 74% (Robbins et al, 2008). Typical 5-year mortality rates in this cohort – usually the result of cardiovascular disease – ranges between 43% and 55%, and are higher than for cancers of the prostate, breast, colon and Hodgkin's lymphoma (Robbins et al, 2008).



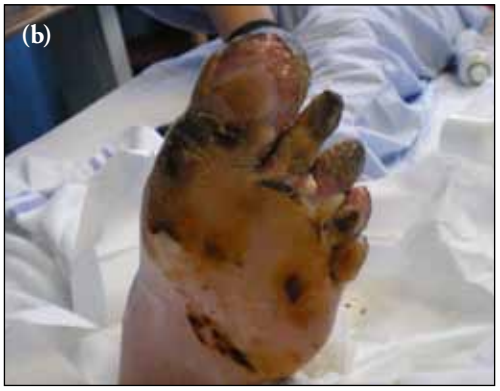


Figure 2. Mr F's left foot at discharge from hospital (2 weeks after presentation), the (a) dorsal and (b) plantar aspects are shown.

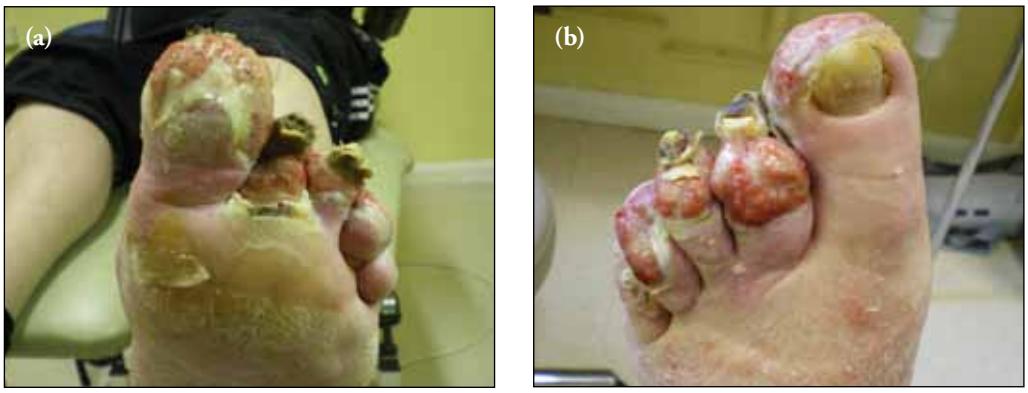


Figure 3. Mr F's left foot at (a) 6 and (b) 9 weeks after presentation.

Peripheral neuropathy, vascular insufficiency and structural deformity have long been known to increase the risk of ulceration in the diabetic foot (Prompers et al, 2008). Thermal injury is a leading cause of ulceration among people with established peripheral diabetic neuropathy (Putz et al, 2008). Thermal injuries are often unperceived by the person with neuropathy and there is sometimes a considerable delay in presentation – the median delay in one series being 27 days (range, 1–56 days; Dijkstra et al, 1997). In the present case, Mr F waited a week before attending the podiatry clinic.

Small-fibre neuropathy with impaired thermal sensation is the most common sensory deficit associated with diabetes (Chao et al, 2007). One cohort of people with type 2 diabetes (n=498), when subjected to quantitative sensory testing and nerve conduction tests showed higher thermal thresholds than a control group matched for age and sex (Chao et al, 2007). Chao et al also demonstrated a direct linear correlation between thermal thresholds of the upper and lower limbs and HbA_{1c} on multiple linear regression analysis. The most frequent abnormality demonstrated in this group was elevated warmth threshold in the big toe (60.2%), which may partly explain the frequency with which ulcers manifest at this location.

In a smaller cohort (n=34) the severity of peripheral neuropathy and warmth sensation thresholds were also shown to be directly related to HbA_{1c} – with HbA_{1c} being the only independent predictor of warmth sensation thresholds (Olmos et al, 1997). Participants in that study with a mean HbA_{1c} level of <9.5% (<80 mmol/mol) had a warmth perception threshold of 35.6±3.7°C, while for those with an HbA_{1c} level of ≥9.5% (≥80 mmol/mol) it was 39.0±3.8°C.

People with diabetes account for a large number of burns cases annually (Memmel et al, 2004). In a series of 37 people admitted for the treatment of lower-limb burns, 27% (10/37) had burns related to diabetes-related neuropathy, most occurring due to self-care measures (Katcher and Shapiro, 1987).

Burns of the neuropathic foot may be caused by unusual aetiologies. Standing or walking

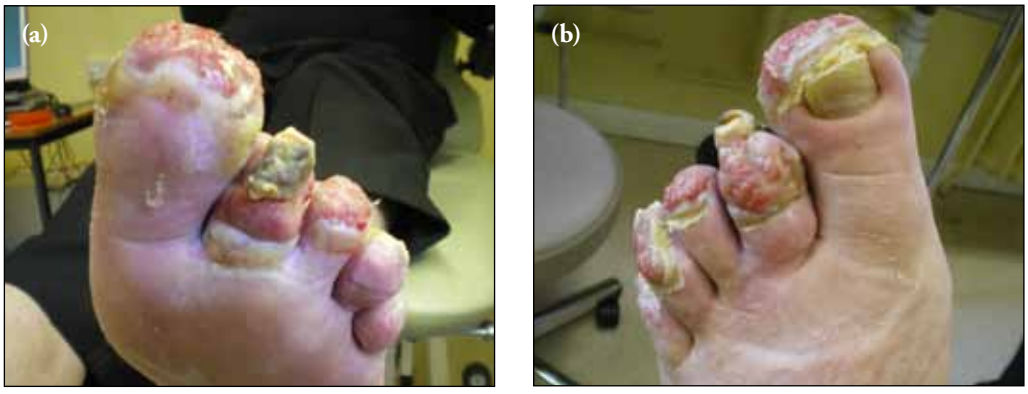


Figure 4. Mr F's left foot 12 weeks after presentation, the (a) plantar and (b) dorsal aspects are shown.

barefoot on the street at high temperatures (50–60°C) has been reported as the cause of burns to both diabetic and non-diabetic feet by Al-Qattan (2000). In that study the author reported second-degree burns to the anterior part of the sole in non-diabetic feet and deep burns involving the entire sole in diabetic feet under the same conditions.

Foot spas, therapeutic foot baths (Balakrishnan et al, 1995) and hot water bottles (Jose et al, 2005) have also been identified as causes of preventable thermal injuries to neuropathic diabetic feet. These include self-care modalities used to treat the symptoms of painful neuropathy on the advice of lay people. Burns to neuropathic diabetic feet have also been reported as the result of alternative medical practices (Ewins et al, 1993).

People with diabetes and burn wounds have an increased incidence of sepsis and community-acquired wound cellulitis (Memmel et al, 2004). Common isolates from such infections are *Streptococcus, Proteus, Pseudomonas, Candida* and meticillin-resistant *Staphylococcus aureus* (Memmel et al, 2004). Due to multisystemic complications associated with serious burns, it has been suggested that targeted infection prevention measures and aggressive clinical intervention among people with diabetes and burns may reduce the mortality and morbidity associated with thermal injuries in this group (McCampbell et al, 2002).

In the present case, Mr F reported being unaware that thermal injuries to his feet could be so severe. While he had been instructed in footwear choices and to examine his feet regularly, Mr F said that definitive advice on avoiding high temperatures may have prevented the injury reported here.

Conclusion

The case presented here highlights the need to warn people with diabetic neuropathy of the dangers of thermal injury to their feet. The application of heat to ease pain associated with diabetic neuropathy should be carefully monitored and clinicians should ask direct questions about such practices during review consultations. Printed information about thermal injury should be made available for discussion and distribution at medical and podiatry clinics.

Thermal injuries among people with diabetic neuropathy should be considered preventable. As chronic hyperglycaemia is the main cause of peripheral neural damage, intensive glycaemic control has an important part to play in the prevention of this complication. However, once neuropathy is established, clinicians should be conscious of highlighting to the person with diabetes the need to protect their feet from injury, including exposure to high temperatures. Morbidity and mortality associated with diabetic foot ulceration, regardless of aetiology, make prevention a clinical priority. ■

"Once neuropathy is established, clinicians should be conscious of highlighting to the person with diabetes the need to protect their feet from injury."

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