Persistent plantar ulceration associated with midfoot Charcot deformity

KEY WORDS
- Charcot foot
- Diabetic foot ulcer
- Multidisciplinary team

This case report describes a patient with Charcot neuroarthropathy, which has developed as a complication of his type 2 diabetes. The case is further complicated by a persistent neuropathic plantar foot ulcer. Charcot deformity significantly increases the chance of ulcers developing. In cases of ulceration, prompt treatment from a specialist multidisciplinary team is vital.

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The risk of foot ulceration for a person with diabetes may be up to 25% (Setacci et al, 2009). The most important risk factors are previous foot ulceration, peripheral neuropathy, foot deformity, peripheral arterial disease, age and living alone (Boulton et al, 2008; Cheer et al, 2009). Early recognition and management of these risk factors is important, especially in people with Charcot arthropathy (Jeffcoate, 2008).

This case and literature review highlights some fundamental considerations in the management of a patient with persistent foot ulceration as a consequence of a rocker-bottom foot deformity caused by Charcot neuroarthropathy.

CASE REPORT

Mr P is a 65-year-old man with well-controlled type 2 diabetes managed with metformin. He smokes ten cigarettes a day, consumes four units of alcohol a day, and has a history of hypertension and osteoarthritis of the spine. He has been attending the multidisciplinary diabetic foot clinic on a regular basis.

At his first referral in 2003, peripheral neuropathy was diagnosed, but there was no evidence of peripheral vascular disease. Mr P presented with an uninfected neuropathic ulcer over the apex of the distal phalanx of his right fourth toe. The ulcer was dressed with povidone iodine knitted viscose dressing and non-occlusive bandages. The orthotist measured his feet for surgical offloading shoes. Mr P was also advised to rest and given recommendations about footwear and walking.

Over the next 16 months he was regularly followed up in the foot clinic. Mr P did not wear his surgical shoes and seemed unable to take appropriate rest to offload his feet. In this period he also suffered from a complicating osteomyelitis – the ulcer on his right toe was probing to bone and he was treated with clindamycin and ciprofloxacin for 6 weeks. By April 2005 the ulcer on the right fourth toe had completely healed.

In June 2007 he visited the foot clinic with a first presentation of an ulcer on the plantar site of his right foot. His right foot had the classic rocker-bottom shape associated with Charcot mid-foot deformity (*Figure 1*). The condition was confirmed by X-ray and magnetic resonance imaging (MRI). There was no history of minor injuries to his foot, and it was unclear over what period the deformity had developed.

The ulcer had no signs of infection or discharge. It measured 1 cm × 1 cm, and was not probing to bone. There were no palpable arterial pulses. A povidone iodine knitted viscose dressing, and non-occlusive bandages were applied. The orthotist prescribed newly measured footwear.

The deformity was difficult to offload with footwear and thus remained prone to ulceration. As a consequence of the Charcot arthropathy and protrusion of bone, Mr P was referred and underwent corrective orthopaedic surgery at the end of 2007.

Fourteen months later, in April 2009, he was reviewed at the multidisciplinary foot clinic. The ulcer on the right foot had healed, but the orthopaedic surgeon was not keen to undertake

any further corrective surgery to reshape the foot. The decision was made to follow a conservative treatment plan in the diabetic foot clinic.

A new neuropathic ulcer had developed on the dorsum of the left first toe, for which conservative treatment was followed. The ulcer healed slowly and preventive footwear was continued.

In August 2009, Mr P's GP referred him to the diabetic foot clinic due to a trophic ulceration on the right foot in the same location as his June 2007 ulcer.

An MRI of the right foot (*Figure 2*) showed excessive osteomyelitis and significant disorganisation of the mid foot with extensive marrow oedema, tenosynovitis and generalised swelling of the soft tissue, particularly on the dorsum of the foot.

The orthopaedic surgeon stated that a below-knee amputation would be the only solution. Mr P was given time to adjust. In the meantime he was treated at home with oral antibiotics (clindamycin / ciprofloxacin for 6 weeks). The ulcer was dressed with a povidone iodine knitted viscose dressing and non-occlusive bandages. The district nurse performed the follow-up, which included dressing changes and monitoring of the ulcer. He Mr P reviewed weekly in the fracture clinic (*Figure 3*). By October 2009 the right foot had improved and there were no overt signs of infection.

Mr P did not have an amputation. He is still managed conservatively and is seen periodically in the clinic.

DISCUSSION

Charcot arthropathy

Charcot deformity is not widely recognised and often misdiagnosed. Two theories are believed to be important in the pathophysiology of Charcot arthropathy (Jeffcoate, 2008). The neurotraumatic theory states that progressive destruction of bones and joints is a consequence of continued exercise and weight-bearing in a foot which has lost its protective sensation. The neurotrophic theory focuses on weakness of the bones as a consequence of hyperaemia caused by damage to the vasomotor nerves; the latter because of changes in circulation or nutrition of trophic nerve fibres. In essence, the condition itself and the course of Charcot arthropathy might mostly be based on a combination of these two theories, because neither of the theories on its own can explain why Charcot arthropathy

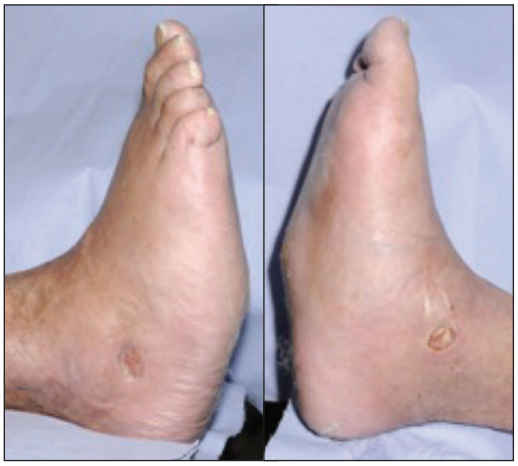

Figure 1. Two views of Mr P's Charcot deformity in October 2009. Note the classic rocker-bottom shape and two superficial ulcers at lateral / medial malleoli.

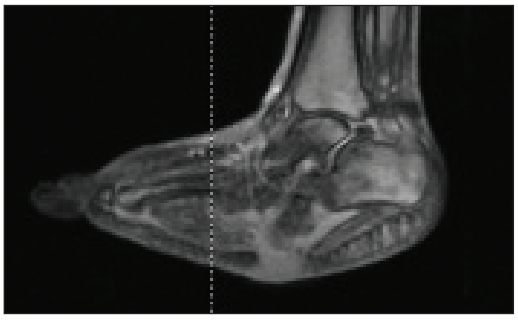

Figure 2. MRI of Mr P's right foot in August 2009.

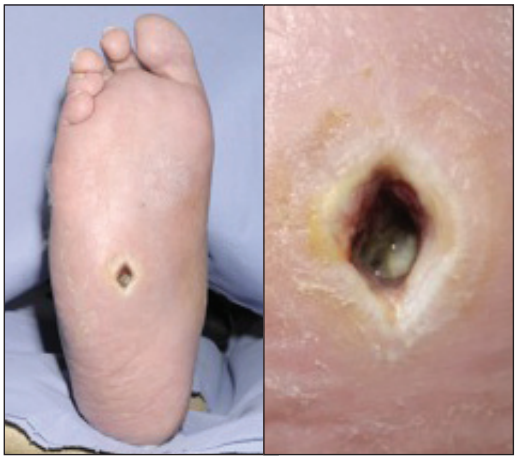

Figure 3. Mr P's plantar ulcer (1.5 cm × 1.0 cm; probing to bone; no overt signs of infection).

"Charcot deformity is not widely recognised and often misdiagnosed."

"Charcot is commonly misdiagnosed as a sprain or cellulitis."

mostly occurs unilaterally, why it is self-limiting and why it is rare (Jeffcoate, 2008).

The inflammatory theory has been more recently proposed (Baumhauer et al, 2006; Rogers et al, 2011). This states that acute trauma of the foot leads to the release of pro-inflammatory cytokines, including tumour necrosis factor and interleukin-1, which induce the increased expression of receptor activator of nuclear factor-B ligand (RANK-L), leading to osteoclast maturation, osteolysis and osteopenia. People with diabetes and peripheral neuropathy lack pain perception and consequently do not make sure their foot is immobilised, thereby causing ongoing trauma and inflammation (Molines et al, 2010).

A further complication is Charcot arthropathy, which is often characterised by the collapse of the arch of the mid-foot, resulting in abnormal bony prominences (Jeffcoate et al, 2000). The bony plantar prominence in the classic rocker-bottom foot becomes a site of abnormally high pressure that exposes the patient to the risk of chronic ulceration (Molines et al, 2010).

A lack of autonomic tone in the capillary circulation is present in the diabetic foot, which causes shunting of blood from arteries directly into veins, bypassing the tissues that need nutrition. This results in a foot that feels warm and has distended veins and bounding pulses. Despite these apparent signs of adequate perfusion, the foot is vulnerable to local microvascular gangrene and wound development (Jeffcoate et al, 2000). These wounds heal very poorly and slowly, and are often further complicated by secondary infection.

Charcot neuroarthropathy is commonly misdiagnosed as a sprain or cellulitis (Jeffcoate, 2008). A review of 36 Charcot neuroarthropathy cases found an average diagnostic delay of 29 weeks (Pakarinen et al, 2002). A previous traumatic event is not always clear and patients may primarily present with non-trauma-related erythema, warmth and oedema in the lower extremity. Often, patients are prescribed antibiotics and continue putting weight on the affected foot. Mechanical trauma cumulates in the acute phase, which results in significant bone and joint destruction. Diagnosis is based on history and clinical findings and confirmed by X-ray and/or MRI (Rogers et al, 2011). Early diagnosis and intervention is essential and leads to a significant lower incidence of deformity (Chantelau et al, 2005).

Management of Charcot neuroarthropathy

Treatment of patients presenting with an acute Charcot neuroarthropathy focuses on stopping the inflammatory process, breaking the vicious circle, relieving pain, and maintaining the architecture of the foot and ankle to prevent deformity (Frykberg and Mendeszoon, 2000). Offloading, protection, and stabilisation are key components of therapy (Armstrong and Lavery, 1998; Rogers et al, 2011; (Game et al, 2012).

A minimum of eight weeks of non-weight-bearing has been recommended for disease of the mid-foot, progressing through partial weight-bearing in a cast brace to full weight-bearing in about four to five months. Regular podiatry for management of callus formation and well-fitting shoes, to offload the medial long arch, are essential at this stage (Giurini et al, 1991).

An alternative approach for patients with early stage neuroarthropathy of the joints of the feet involves long term immobilisation with a total contact cast or an instant total contact cast at the time of diagnosis (Pinzur et al, 2006; Game et al, 2012). Appropriate duration of contact casting may take up to 18.5 weeks, with an average duration of 10.5 weeks (Armstrong et al, 1997). Data from a large audit in the UK (288 cases of acute Charcot foot) suggest that the use of non-removable offloading at presentation may shorten the time to resolution of disease (Game et al, 2012).

Oedema and temperature of the foot as well as radiographic signs of bony union should be taken into account when deciding on continuous immobilisation (Baravarian and Van Gils, 2004).

Possible benefits of targeted medical management alongside immobilisation have been demonstrated by researchers, following the recognition of the involvement that RANK-L might have in the osteolysis found in Charcot (Jeffcoate, 2004). Use of intranasal calcitonin has been shown to reduced bone turnover by acting directly on the RANK-L signalling pathway in patients with acute Charcot neuroarthropathy (Bem et al, 2006).

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Common deformities seen are the rocker-bottom foot caused by collapse of the medial arch, medial convexity deformity caused by medial displacement of the talonavicular joint and tarsometatarsal dislocation. Such deformities transfer weight bearing to areas that tolerate it poorly, and that lack sensation as a result of peripheral neuropathy; the latter is one of the main reasons patients continue to walk on subluxed joints. Late presentation of Charcot arthropathy may mean that offloading, routine foot assessment and medical management alone are inadequate and surgical intervention is required. In a structurally misaligned foot with an ulceration, the most important goal is to restore a stable, plantigrade foot with ulcer healing and elimination of infection (Garapati and Weinfield, 2004). Foot and ankle reconstruction with external fixation has been shown to be an effective method of correcting the deformity and providing a stable, plantigrade foot (Farber et al, 2002; Cooper, 2003).

Surgical correction can be avoided in most patients. However, in carefully selected cases, surgery can give acceptable alignment, thereby preserving soft tissue integrity and viability and avoiding amputation (Bono et al, 1993).

In a study reviewing conservative versus surgical treatment for diabetic Charcot foot (Pinzur, 2004), with a minimum 1-year follow-up, 87 out of 147 feet with mid-foot deformity (59.2%) achieved the pursued endpoint (long-term management with commercially available, therapeutic footwear and custom-made foot orthoses) without surgical intervention; 60 (40.8%) required surgery. According to this study, more than half of patients with Charcot arthropathy at the mid-foot can be successfully managed without surgery.

Impact on health-related quality of life

Diabetes itself and also its more severe complications may affect daily life considerably. Patients with foot ulceration or Charcot arthropathy may have a poorer health-related quality of life (HRQoL) than those who have experienced a diabetes-related amputation (Price and Harding, 2000; Willrich et al, 2005).

During a 1-year follow-up study (Ribu et al, 2008) significant differences were observed between patients with healed ulcers and patients with persistent ulcers on social functioning and mental health. Poor HRQoL was associated with poor ulcer prognosis. Delay in diagnosis of a Charcot foot deformation of more than 3 months was found to adversely affect quality of life and functional outcome (Pakarinen et al, 2009).

These findings suggest that the management of these patients should include support in coping with impaired mobility to enable them to continue regular daily activities as long as possible. The ultimate aim of wound care is to maintain a healed status and mobility as well as quality of life.

CONCLUSION

In summary, this case review illustrates a recurrent plantar foot ulceration which is associated with the presence of Charcot mid-foot deformity in a patient with diabetes. Within the clinic and in the community a multidisciplinary specialist team supported Mr P.

Conservative treatment in general is preferred over surgical correction, but despite this, ulcers readily become chronic wounds and often recur at the same site. Optimal management and support in the home situation will enable the patient in this case to keep leading his own life as long as possible, and ultimately this may be the best treatment goal. WUK

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