

# Impact of the podiatrist on foot lesion diagnosis

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A podiatrist's specialist knowledge within a medical team can contribute to the diagnosis of less common foot pathologies, which can often be the first manifestation of a systemic condition.

We report two case histories where patients presented in the dermatology

department, Brighton General Hospital. In each case, the podiatrist recognised previously undiagnosed diabetes from the presenting skin condition.

This paper highlights the effectiveness of multidisciplinary teams in managing diabetic patients, emphasising that referral pathways and liaison are essential to this process.

## Case report I

A 74-year-old man was referred to podiatrists at the dermatology department by a colleague from the community. He presented with a large, painless bulla on the plantar aspect of the left first and second toes, which resembled a burn-induced blister. This had developed overnight, with no history of trauma or ill-fitting footwear. The previous year an episode of sudden bullous eruption on the right foot had resolved without scarring, following a course of antibiotics.

On examination the bulla involved the entire plantar surface of the hallux and extended, via the interdigital cleft, along the medial side of the second toe. Peripheral erythema defined the demarcation line and the fluid content was clear.

The patient's general health was good, alcohol intake was moderate (21 units per week), and he smoked one cigar per day. All peripheral pulses were present and bounding. Other than the affected area, skin condition was good. On assessment there was evidence of sensory deficit suggestive of peripheral sensory neuropathy. The dermatologist suspected that the lesion was trauma induced.

Differential diagnoses included

drug reaction, burns, bulla secondary to trauma, bullous pemphigoid, bullous impetigo, acute pompholyx and bullosis diabeticorum (Marks, 1993). The last diagnosis in this list was suspected and diabetes was confirmed by a blood glucose reading of 22.3 mmol/litre.

The lesion was drained by aseptic incision but not deroofed (Figure 1). A dressing protocol of sterile saline irrigation and non-adherent sterile dressings was used. Direct pressure was restricted as much as possible with deflective padding and open-toed footwear.

A week later the bulla, now dehydrated, had turned a cyanotic bluish-purple colour, similar to pre-gangrenous necrosis (Figure 2). The necrotic skin shed several days later to reveal a neuropathic ulcer surrounded by healthy tissue (Figure 3). The ulcer swab cultures yielded no growth.

The patient was subsequently referred to the diabetologist and attended an education programme. Once diabetic control was established, the bulla healed in 6 weeks, and the neuropathic ulcer in 14 weeks (Figure 4). The patient was seen 6 months later with no new lesions or sequelae.

## ARTICLE POINTS

**1** Community podiatrists are able to diagnose uncommon foot lesions in 'at-risk' patients.

**2** Rapid referral is possible where community podiatrists are affiliated with specialist hospital-based teams and community clinics.

**3** A rota system for podiatrists within community and hospital teams facilitates continuous updating and optimal use of their podiatry skills.

**4** Podiatrists can aid rationalisation of caseloads, in readiness for the projected increase in the numbers of at-risk patients in the 21st century.

## KEY WORDS

- Foot pathology
- Podiatrist
- Diagnosis
- Undiagnosed diabetes

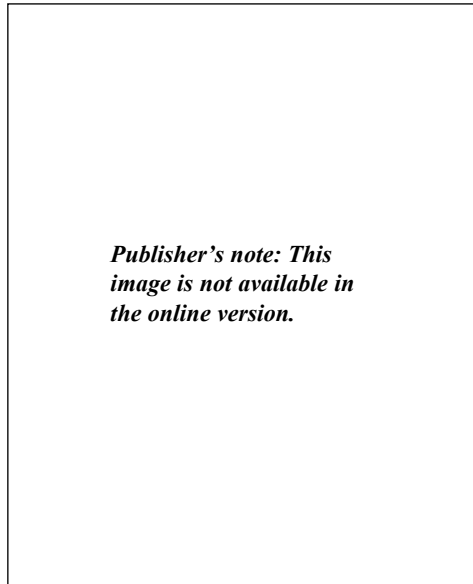
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**PAGE POINTS**

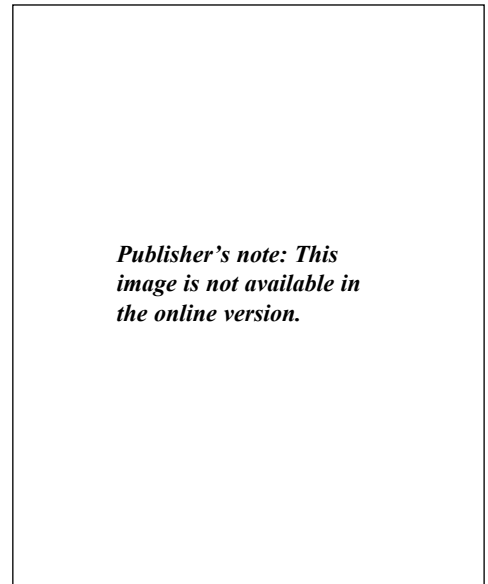
**1** Bullosis diabeticorum is a rare complication of longstanding diabetes mellitus.

**2** The aetiology is unknown.

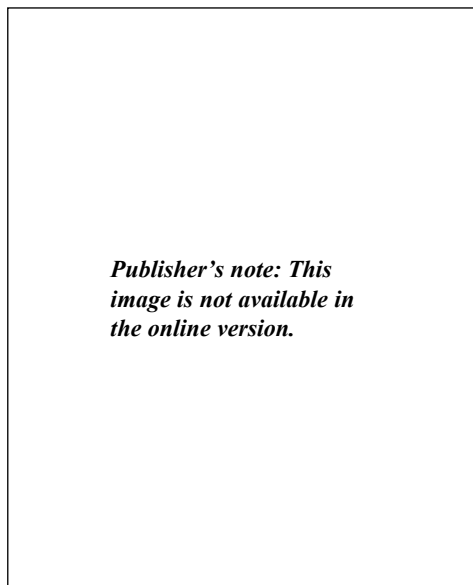
**3** In the few reported cases, the only common element was the presence of diabetes.



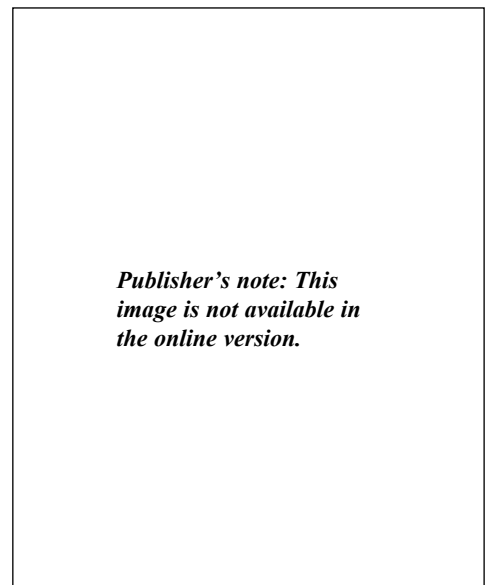
**Figure 1. Flaccid bullae following aspiration of serous fluid.**



**Figure 2. Ulcer at base of second toe and epidermal necrosis of great toe.**



**Figure 3. Superficial ulceration of first and second toe.**



**Figure 4. Complete healing showing hypopigmentation at base of second toe.**

**Comment on case report I**

Bullosis diabeticorum is a rare complication of longstanding diabetes mellitus (Bodman et al, 1991). In this instance the patient's diabetic state was undetected until presentation in the clinic; it is therefore questionable whether the diabetes was long-standing. Recurrence of idiopathic bullae is common (Bodman et al, 1991). Therefore it is reasonable to presume, because a previous eruption had occurred, that the patient's diabetes had been present for some time.

This cutaneous manifestation of diabetes mellitus was named bullosis diabeticorum by Cantwell and Martz (1967) and has been referred to as 'phlyctaenae', from the Greek phlytaina, meaning a blister made by a burn (Pirota et al, 1995)

The aetiology of bullosis diabeticorum is unknown. Most of the suggested causal and contributory factors are linked to other known complications of diabetes mellitus; however, no common element, other than the existence of diabetes, has been noted in the few reported cases (Pirota et al, 1995).

**Case report 2**

A 52-year-old man, who had recently moved to the district, was referred by the community podiatrist to a colleague working in the diabetes unit. The patient had a large, open lesion under his right mid-foot, which had been present for 6 months (Figure 5).

The community podiatrist had received the patient from the dermatologist, who had initially suspected a squamous cell carcinoma or a melanotic melanoma. Histology showed no histopathological abnormalities, and a referral to the neurologist was made for suspected neuropathy.

The patient reported a feeling of total deadness in both legs for six years, the right being more severely affected. He did not complain of any paraesthesiae, pins and needles or burning sensation. He had not been aware of any change in walking or any apparent weakness.

Neurological examination revealed sensory neuropathy in an extended stocking distribution, impaired proprioception at the great toe, impaired light touch to high shin bilaterally, and absent vibration sensation. The knee jerks were depressed, the ankle jerks were absent, and both plantar responses were flexor. The peripheral pulses, i.e. dorsalis pedis and posterior tibial, were present. The lesion was diagnosed as a neuropathic ulcer.

Systematic enquiry revealed no previous operations and no other abnormalities. He had a history of gout, for which he was taking allopurinol, and low vitamin B<sub>12</sub> levels, for which he had received appropriate therapy. His past medical history included an accident in 1962 when he had been knocked off his bike and rendered unconscious; there had been no other injuries. In 1994 he had tripped on a concrete step and injured his right mid-foot.

His alcohol intake was moderate

at 28–30 units a week, and he had recently started smoking. He was a little overweight at 93.7kg, his height was 182cm, and his blood pressure was raised at 170/100mmHg. He had no retinopathy and no albuminuria. His glycated haemoglobin (HbA<sub>1c</sub>) was 5.2%, creatinine was 74µmol/litre and cholesterol was 4.6 mmol/litre.

On examination, the right mid-foot had a rocker-bottom deformity and medial convexity, suggesting a Charcot's neuroarthropathy. The podiatrist requested an X-ray, to rule out osteomyelitis. Diagnosis of Charcot's neuroarthropathy was confirmed. The radiologist reported that the appearance was of a Charcot joint in the tarsal region, with general disruption of the anatomy and degenerative change. There was distal osteoporosis, but no evidence of osteomyelitis. The podiatrist arranged for a Scotchcast boot to be made.

The dermatologist had taken blood samples for vitamin B<sub>12</sub>, folate, VDRL (Venereal Diseases Research Institution test for syphilis), glucose and gamma glutamyl transpeptidase levels, liver function tests and a full blood count. Although the plasma glucose level was 7.4 mmol/litre, the presence of a neuropathic ulcer aroused a high index of suspicion of impending diabetes in the patient.

Two months after his first hospital visit, an oral glucose tolerance test showed that he had diabetes. His 2-hourly glucose was 12.1 mmol/litre.

The patient is now retired from work, and complies well with advice on avoidance of weightbearing. He has attended the diabetes education programme, and continues his visits to the podiatrist for debridement of the ulcer and regular foot check-ups. He now has some orthotics, made in the podiatry laboratory, and his ulcer is healing well (Figure 6).

**PAGE POINTS**

**1** The aetiology of Charcot's neuroarthropathy is debated.

**2** The condition is characterised by peripheral neuropathy, injury (often occult), continued repetitive stress, and adequate vascularity.

**3** It has been suggested that an intraneural lesion induces a hyperaemic response, resulting in fracture and bone loss, and progresses to neuroarthropathy.

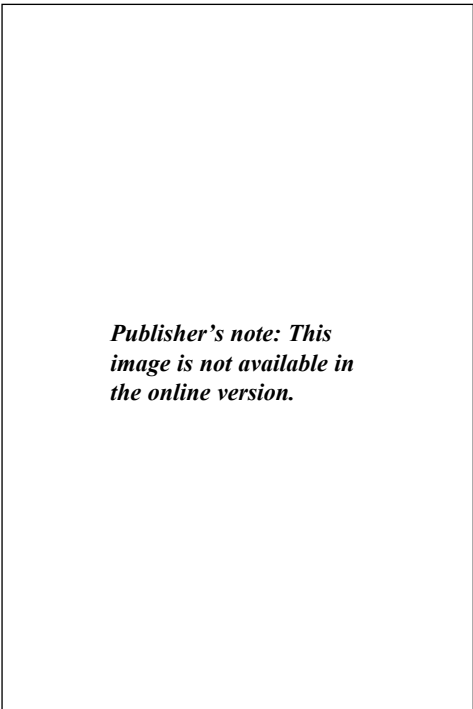
**PAGE POINTS**

**1** Chantelau and Schnabel (1997) suggest that Charcot foot is most likely to be caused by stress fracture.

**2** Bullosis diabeticorum and Charcot's neuroarthropathy have both been shown to cause foot ulceration.

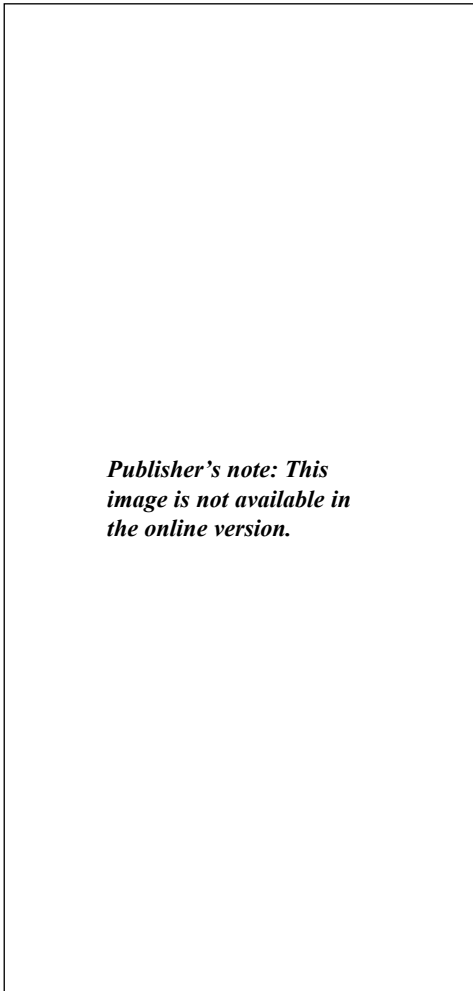
**3** All of those involved in the care of diabetic patients therefore need to be familiar with the presentation of these two disorders.

**4** Good liaison between hospital and community podiatrists enables a unified concept of the at-risk foot to be achieved.



*Publisher's note: This image is not available in the online version.*

**Figure 5. Ulcer pre-debridement, on first presentation at the dermatology clinic.**



*Publisher's note: This image is not available in the online version.*

**Figure 6. Ulcer healing well, following casting and debridement.**

**Comment on case report 2**

The aetiology of Charcot's neuroarthropathy has been debated. The symptoms are peripheral neuropathy, injury, often occult, continued repetitive stress, and adequate vascularity (Anderson, 1997). The neurovascular theory maintains that an intraneural lesion produces a vasodilatory response, resulting in fracture and bone loss, and progressing to neuroarthropathy. Bone resorption and softening result from the hyperaemic response (Beeson, 1995). Chantelau and Schnabel (1997) suggest that Charcot foot is most likely to be caused by stress fracture.

The degree of neuropathy in our patient was considerable, but no association with his accident in 1962 was established by the neurologist's report. Perhaps tripping on the concrete step contributed to the patient's Charcot foot.

Although the patient's intake of alcohol was moderate, a higher intake in the past may be relevant. It seems that a combination of mild glucose intolerance and increased alcohol intake can be a fairly potent cause of neuropathy (Vaughan, 1997). As anticipated in our patient, a diagnosis of diabetes eventually emerged.

**Discussion**

Bullosis diabeticorum and Charcot's neuroarthropathy are two fairly uncommon complications of longstanding diabetes.

Both of these conditions have been demonstrated to cause foot ulceration.

For this reason, familiarity with their presentation is important for those involved in the examination of the diabetic foot.

In each of these cases the suspicion of diabetes as the underlying factor was raised by the podiatrist working in the community, and was implemented by a podiatrist already established as a member of a multidisciplinary medical team.

This was facilitated by the Department of Podiatry's existing structure, whereby podiatrists are affiliated with specialist hospital-based teams as well as community clinics. This arrangement not only promotes ease of referral, but also increases the speed of referral.

Good liaison between hospital and community is established using a rota system

whereby podiatrists work for a spell in specialist hospital teams. This enables podiatrists to achieve continuous updating within the department; in addition, they are able to share information and expand their experience. Most importantly, a unified concept of the 'at-risk' foot may be achieved.

Fletton et al (1995) suggest that whereas the concept of the at-risk foot has developed in diabetic and other hospital-based practice, it is by no means clear that primary care workers or podiatrists subscribe to that concept or indeed have a clear idea of what constitutes at risk.

Involvement of community podiatrists with hospital teams ensures that they experience for themselves the definitions of the at-risk foot and can accurately identify the need for appropriate care. It also helps the clinician to distinguish between the not-at-risk patients who are receiving treatment, so that they may be deprioritized without any cost to the high-risk groups.

Many NHS trusts are scrutinising the type of caseloads treated in the community, and some are implementing a discharge policy that ensures they are targeting these vulnerable groups (Department of Chiropody/Podiatry, South Downs NHS Trust, 1997). Discharge may be indicated for not-at-risk patients who are non-compliant with footwear advice, for example, or where the patient (or their relatives) could carry out the appropriate foot care.

Positive education towards discharge needs to be gradual; it requires time and resources. It is, however, necessary in order to give the at-risk patient priority. This need for positive education was demonstrated by Fletton et al (1995) who found that half of the at-risk patients in a district were not receiving podiatric care, and a relatively high proportion of not-at-risk patients were receiving it.

In both of the cases reported here, the underlying systemic condition had not been detected before presentation. This supports the views of Currie et al (1995) who claim that the proportion of NHS resources used for the treatment of patients with diabetes has been significantly underestimated, as there is still likely to be a proportion of undiagnosed people with diabetes in the population.

The number of at-risk patients is projected to rise in the early part of the next century. Amos et al (1997) suggest that an increase in complications will undoubtedly follow. If this is the case, and if 30% of diabetic patients are affected by numerous cutaneous complications of diabetes (Pirootta et al, 1995), it is likely that undiagnosed diabetes will be increasingly observed in dermatology departments and all other outpatient settings in the future. ■

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PAGE POINTS

**1** Involvement of community podiatrists with hospital teams ensures that they can accurately identify the need for appropriate care.

**2** In one study, half of the at-risk patients in a district were not receiving podiatric care, whereas a high proportion of those not at risk were receiving it.

**3** It is likely that the proportion of NHS resources used for diabetic patients is significantly underestimated, given that there may be many people with undiagnosed diabetes in the population.

**4** With the numbers of at-risk patients projected to rise in the next few decades, undiagnosed diabetes will increasingly be observed in dermatology and other outpatient clinics.