A user's guide to foot screening. Part 3: Skin and joints

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Introduction

This article is the third in a series of three that aims to provide a clear, simple and practical approach to diabetic foot assessment. Each of the three articles has focused upon a particular aspect of foot examination and, although they have appeared individually, they should be combined to give an overall approach to foot examinations. This article focuses upon examining the skin and joints of the feet of the person with diabetic foot complications.

Diabetic foot complications are common and vary considerably in both their pathophysiology and clinical presentations, ranging from subtle skin and joint changes through to full thickness ulceration and overt gangrene. A good healthcare professional must be able to interpret observations and have excellent methods of inquiry to ascertain a comprehensive medical history. It is, therefore, important that all those involved in screening, assessment, health education and treatment of people with diabetic foot problems are appropriately educated and trained, and are working within clearly defined guidelines and well supported infrastructures.

Encouraging patients to be responsible for their own diabetes, including basic foot care, is essential, but many patients are unable to monitor or inspect their own feet because of poor eyesight and/or reduced mobility (Thomson and Masson, 1992; Masson et al, 1989). Therefore, for the healthcare professional involved in foot care, maintaining regular contact with patients is important (Edmonds et al, 1996). Additionally, regular monitoring and screening by healthcare professionals provides an ideal opportunity to reinforce messages of self-care and its importance (Boulton and Malik, 1998).

Annual screening for neurological and vascular problems is the cornerstone of risk identification (Abbott et al, 1998; National Institute for Cllinical Excellence [NICE], 2004); however, there are several other essential issues to consider when screening for the at-risk foot. These include: the presence of trauma-related callus, infections, limited and excessive joint mobility, skin and nail conditions, footwear, lack of self-awareness, poor healthcare beliefs/attitudes and limited access to appropriate foot care (Fletcher, 1990; Murray et al, 1996; Reiber et al, 1999; Uccioli et al, 1995). It is the aim of this article to cover those aspects related to skin and joints.

General appearance

A good habit to get into at the start of every appointment is to take time, stand back and make some general observations about the feet to be examined (*Table 1*). All of this information can be assimilated very quickly and should help confirm or refute the findings of assessment test results.

Skin and nails

The skin on the plantar surface of the feet is subject to high levels of repetitive shear, compressive and frictional forces during walking and standing. The visco-elastic nature and structure of skin is designed to maintain its integrity (Ryder et al, 1988). Tissue damage can occur due to altered soft tissue mechanics together with an increased magnitude and/or duration of shear, compressive or frictional forces. A simple rule of thumb

Table 1. Questions to ask yourself before commencing a clinical examination of a patient.

- What is your first clinical impression?
- Do the feet look impoverished?
- Can you see any distended veins or evidence of hair growth?
- What is the overall colour and quality of the skin?
- Are there any distinct peculiarities in the shape of the rear-, mid- or forefoot?

to identify such tissue damage is to see if the skin can be depressed easily and it remains depressed. An appreciation of, and the ability to identify, the components that may contribute to such conditions is an important part of the screening process and intervention strategies.

Xeroderma

Xeroderma (dryness of the skin) caused by a decrease in moisture and lipid content within the stratum corneum is a common finding in people with diabetic neuropathy and is considered to be concomitant with the normal skin ageing processes (Pigatto et al, 1996; Nicander, 1988). Loss of moisture from the skin can result in functional deficits within both the dermis and epidermis and a reduction in its visco-elastic and anti-frictional qualities. Such functional losses can lead to callus formation (which can lead to ulceration) and skin fissures which can expose the dermis to potential pathogenic invasion, tissue loss and ulceration (Boulton et

al, 1999). The frequent and regular use of bland emollients can help address this problem and additionally reinforces the importance of self-care and foot examination.

Fissures

Skin fissures are a common clinical finding occurring most frequently on the heel borders. They can deteriorate rapidly to ulcers and frequently become necrotic in patients with neuroischaemia (NICE, 2004). Deteriorated fissures must be dealt with swiftly and appropriately by referring the individual to a podiatrist for careful callus debridement at the fissure margins, which will allow the edges to knit together. Patients must be encouraged to apply bland emollients at least once daily to allow rehydration of the affected skin, preferably using a urea- or glycerine-based cream (Miettinen et al, 1999; Loden, 1996). The regular use of such emollients has been shown to reduce and replenish moisture loss, thus increasing suppleness (Loden, 1996).

Hard skin/callus

Plantar callus (*Figure 1*) is a common characteristic of the diabetic neuropathic foot. Such calluses are typically located over the toe apices and metatarsal heads and are generally thick and relatively soft (Bevans and Bowker, 1999). In contrast, calluses in the neuroischaemic or ischaemic foot are typically thin, dry, glassy and very hard. These may also be found over main weight-bearing areas but are particularly evident on the borders of the feet (Bevans and Bowker, 1999).

The presence of a bloodstained callus is highly predictive of ulceration being present below the callus. Upon removal

Figure 1. Example of a plantar callus.

Figure 2. Fungal infections of the nail (left) and skin (right).
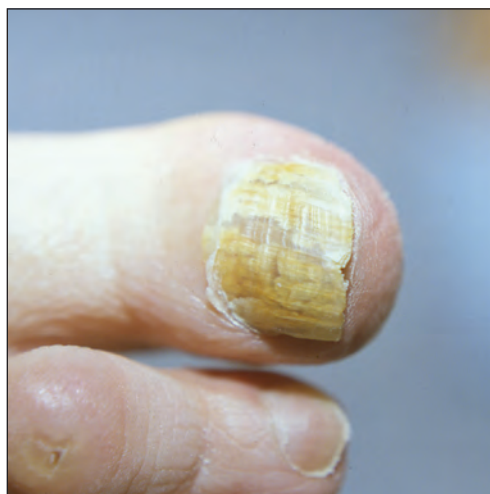
of the callus an ulcer can be found in up to 80% of cases (Rosen et al, 1985; Harkless and Dennis, 1987). Bloodstained calluses look like raspberry or blackcurrant jam under the skin. Individuals presenting with such calluses should be referred to a podiatrist for urgent debridement and pressure relief.

Fungal infections

Fungal skin and nail infections (*Figure 2*) in the foot can cause considerable discomfort and distress and have a reported prevalence of between 3% and 8% in the UK (Roberts, 1992; English et al, 1967; Howell et al, 1988); however, the precise prevalence in the population with diabetes is not known (*see pages 194–200 for a study on fungal foot infections in diabetes*). The common skin manifestations include interdigital fissuring, flaking, vesicular eruptions, inflammation and intense puritus while in onychomycosis (fungal infection of nails) the nails may be thickened, discoloured, friable and deformed.

Fungal infections of the skin are not a primary cause of foot ulceration; however, because they erode the epidermis there is an increased risk for subsequent bacterial infections. The most common site for such epidermal erosion to occur is between the toes, where bacterial infection develops quickly and may lead to deep ulceration and cellulitus (Yosipovitch et al, 1998; Semel and Goldin, 1996). It is therefore important to examine between patients' toes during foot screening. Fungal infections of the

skin can be treated quite easily by the topical application of anti-fungal creams (Bolognia and Braverman, 1998). Nail infections, however, are more difficult to treat and generally require systemic anti-fungal therapies sometimes in conjunction with topical anti-fungal nail lacquers.

Diabetic dermopathy

Diabetic dermopathy is the most common cutaneous finding in people with diabetes – appearing in 30% of people with diabetes (Huntley, 1982). Diabetic dermopathy often results in pigmental patches/lesions which are often bilateral, multiple and hyper-pigmented macules on the pre-tibial areas (Sueki and Fujisawa, 1986). These patches are also known as shin spots. Lesions may also appear on the forearm, the side of the foot and the anterior surface of the lower thigh. The lesions are round or oval, reddish-brown, scaly papules and plaques, ranging in size from 0.5–1 cm. The incidence of diabetic dermopathy has been shown to correlate with an increase in glycosylated haemoglobin and duration of diabetes (Shemer et al, 1998).

Due to the frequent location of the lesions over bony prominences, diabetic dermopathy may simply be a magnified response to trauma such as heat, cold, or blunt objects in people with diabetes. There may be a link between diabetic dermopathy and other complications of diabetes such as microangiopathy and neuropathy (Jelinek, 1994). Thus, patients with diabetic dermopathy should be further evaluated for detection of other

PAGE POINTS

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Figure 3. Bullosis diabeticorum – a blister containing a clear sterile fluid.

concomitant complications. To date, there is no effective treatment for diabetic dermopathy.

Bullosis diabeticorum

Bullosis diabeticorum, although rare, is a distinct complication of diabetes (Bolognia and Braverman, 1998; see *Figure 3*). The bullae (blisters) appear spontaneously from normal skin, usually on the feet and toes, but occasionally on the hands and fingers. They are usually 0.5 to several centimetres in size, and contain a clear, sterile, viscous fluid.

The bullae could be intra-epidermal or sub-epidermal. Intra-epidermal bullae are clear, sterile, non-haemorrhagic blisters that generally heal on their own within 2–5 weeks without scarring or atrophy. This type of bullae is more common in men with long-standing diabetes, peripheral neuropathy and good circulation to the involved extremity (Bolognia and Braverman, 1998).

Sub-epidermal bullae are the least common of the two types of bullosis diabeticorum (Huntley, 1982). These blisters are similar to the intra-epidermal blisters except they are occasionally haemorrhagic and may heal with scarring and atrophy. Bullosis diabeticorum is usually self-limiting; however, bacterial infections can result in deep infections (Huntley, 1982).

Local care to avoid blister eruption and appropriate antibiotic treatment for secondary infection is recommended.

Infection

Foot infections are common in patients with diabetes and sometimes are the presenting feature of undiagnosed type 2 diabetes. It is important to note that the cardinal signs of infection – redness, heat, swelling and pus – may be reduced and therefore are not as obvious as in those without diabetes. Unexplained raised blood glucose levels, sudden localised pain or discomfort in neuropathic ulcers can signify the onset of infection well before the presence of any clinical signs. Foot infections are serious and can progress very quickly, which, if not managed correctly and aggressively, can lead to lower-limb amputations. Any infection that does not show signs of responding to antibiotic therapy after 3 days should be treated very seriously and referred to a specialist foot clinic. Additionally, any patient with spreading cellulitis requires an immediate hospital referral as it is a limb-threatening condition (NICE, 2004).

Joints
Limited joint mobility

Collagen abnormalities – principally, thickening and increased cross-linking – are common in people with diabetes and are due to advanced glycation end-products (Goodfield and Millard, 1988). This is clinically evident by the skin having a tight and waxy appearance.

Testing for reduced joint mobility in the foot is important, but not very easy and requires a good understanding of foot anatomy and skill. Additionally, it can also be very time consuming in the hands of a novice. Reduced joint mobility in those with diabetic neuropathy has been shown to significantly increase foot pressures and the risk of ulceration (Fernando et al, 1991). As previously mentioned this is further increased when plantar callus is present.

Since limited joint mobility in the hands/wrists is sometimes indicative of joint problems in the feet and ankles (Ismail et al, 1996; Brik et al, 1991), perhaps the quickest way of screening for limited joint mobility is to ask the patient to perform

PAGE POINTS

1 Ankle joint equinus is deemed to be present when there is less than 10° dorsiflexion from its neutral position.

2 Although limited joint mobility is known to be associated with neuropathic ulceration, excessive joint mobility, especially at the mid-tarsal joint, can significantly alter forefoot time–pressure integrals and shear forces, thereby increasing the potential for callus formation and ulceration.

3 Although Charcot neuroarthropathy is not common it is important that it is identified early to help prevent devastating foot deformity and potential ulceration or amputation.

the 'prayer sign'. This is undertaken by asking patients to put the palms of their hands together with the fingers extended as if praying. If the palms do not meet the sign is said to be positive indicating limited joint mobility (Rosenbloom et al, 1981). Obviously this test is of little use if there are hand or finger deformities present such as Dupuytren's contracture.

Perhaps two of the most important joints in the foot to examine are the ankle and big toe joint (first metatarso-phalangeal joint). This is because reduced ankle and first metatarso-phalangeal joint dorsiflexion increases the force and duration of loading at the forefoot and the plantar surface of the big toe respectively, which may increase ulceration risk in these areas.

Testing for reduced ankle joint dorsiflexion
Ankle joint equinus is deemed to be present when there is less than 10° dorsiflexion from its neutral position.

With the lower leg fully extended, place your hand under the plantar surface of the foot so that the hand forms a rigid platform upon which the foot can rest. Gently push the foot upwards until it is at 90° to the lower leg, not allowing it to roll in or out as you do so. From this position gently push a little harder to obtain the maximum amount of dorsiflexion as possible. There should be at least 10° further dorsiflexion from this starting point; if this is not evident ankle equinus is likely to be present. This is a factor of high risk for ulcers in the presence of neuropathy (Fernando et al, 1991). Stretching exercises may be of some benefit.

First toe dorsiflexion
Gently grasp the base of the first toe between your thumb and forefinger with one hand and with the other do likewise just before the first metatarsal head. With the toe and first metatarsal head level, gently but firmly move the toe upwards. You should expect the toe to move up between 60°–90° from the starting point. If this cannot be achieved, the joint has a reduced range of movement. Once again

this increases the risk of ulceration under the first toe especially in the presence of peripheral neuropathy (Fernando et al, 1991).

Excessive joint mobility
Although limited joint mobility is known to be associated with neuropathic ulceration, excessive joint mobility, especially at the mid-tarsal joint, can significantly alter forefoot time–pressure integrals and shear forces, thereby increasing the potential for callus formation and ulceration. The simplest way to examine for this is to observe what happens to the feet when the patient stands. If feet roll-in excessively or the medial arches flatten markedly it is most likely that the joints are very mobile.

In the author's opinion this is a useful test to perform in order to determine the presence of motor neuropathy when high arches and clawed toes are observed at rest. If the toes remain clawed and the arches high upon standing, motor neuropathy can be strongly suspected; however, if the arches flatten motor neuropathy is unlikely even if the toes remain clawed.

Charcot neuroarthropathy
Although Charcot neuroarthropathy is not common it is important that it is identified early to help prevent devastating foot deformity and potential ulceration or amputation (Saunders and Frykberg, 1991). Typically, an active Charcot neuroarthropathy would present as a foot and leg that is intensely pink, warm and swollen with deformity that has occurred fairly suddenly (Jeffcoate et al, 2000; Jude and Boulton, 2001). Always compare the suspected foot and leg with the contra-lateral limb. Additionally, patients may describe pain which, if present, is generally a dull to intense aching and may be experienced in the foot or calf. Pulses will be readily palpable and abnormally high ankle systolic pressures are common due to calcification. Dilated dorsal foot veins are also often present. Charcot neuroarthropathy only occurs in patients with marked peripheral neuropathy and not in those with ischaemia.

Foot deformity

Foot deformities (*Figure 4*) commonly occur as a result of altered foot mechanics, poorly fitting footwear, neuropathy and surgery. The most common deformities are clawed or hammered toes and hallux valgus (bunions). These are frequent causative factors for abrasions, callus, blisters and ulcers, due to shoe rubs both in patients with neuropathy and those with neuro-ischaemia. Thus footwear examination and advice to the patient is essential. It is important to remember that the shoes worn to clinics may not be worn most of the time – people commonly 'dress to impress'.

Conclusion

Any advice that is given needs to be relevant and structured to each individual along with being realistically achievable. In conclusion, this article has tried to give an overview of some aspects of foot screening that involve observational skills rather than the use of diagnostic tools. Risk assessment and categorisation must involve combining a thorough clinical examination with diagnostic tests, balanced by good history taking. Recognising subtle skin or joint changes, which on their own may appear trivial but are major when combined with neuropathy or ischaemia, is very important. Combining such skills and expertise in diabetic foot screening is the secret to lesion prevention and good quality foot care. When examining feet, the opportunity should always be taken to educate the patient in order to reinforce the importance of self-care and identifying unresolved motivational barriers. Finally, it is hoped that this article, combined with the others in this series (Baker et al, 2005a; 2005b), should provide a 'user's guide to diabetes foot screening'. ∎

Figure 4. An example of deformity in a neuroischaemic foot.

Abbott CA, Vileikyte L, Williamson S, Carrington AL, Boulton AJ (1998) Multicenter study of the incidence of and predictive risk factors for diabetic neuropathic foot ulceration. *Diabetes Care* **21**(7): 1071–5

Baker N, Murali-Krishnan S, Rayman G (2005a) A user's guide to foot screening: peripheral neuropathy. *The Diabetic Foot* **8**(1): 28–37

Baker N, Murali-Krishnan S, Fowler D (2005b) A user's guide to foot screening: peripheral arterial disease. *The Diabetic Foot* **8**(2): 58–70

Bevans JS, Bowker P (1999) Foot structure and function: aetiological risk factors for callus formation in diabetic and non-diabetic subjects. *The Foot* **9**(3): 120–7

Bolognia JL, Braverman IM (1998) Skin and subcutaneous tissues. In: Lebovitz HE ed. *Therapy for diabetes mellitus and related disorders*. 3rd edition. American Diabetes Association, Alexandria, USA, 290–302

Boulton AJM, Malik RA (1998) Diabetic neuropathy. *The Medical Clinics of North America* **82**(4): 909–29

Boulton AJ, Meneses P, Ennis WJ (1999) Diabetic foot ulcers: A framework for prevention and care. *Wound Repair and Regeneration* **7**(1): 7–16

Brik R, Berant M, Vardi P (1991) The scleroderma-like syndrome of insulin-dependent diabetes mellitus. *Diabetes/Metabolism Reviews* **7**(2): 120–8

Edmonds M, Boulton A, Buckenham T, Every N, Foster A, Freeman D et al (1996) Report of the Diabetic Foot and Amputation Group. *Diabetic Medicine* **13**(9 Suppl 4): S27–42

English MP, Wethred RR, Duncan EH (1967) Studies in the epidemiology of tinea pedis. 8. Fungal infection in a long-stay hospital. *British Medical Journal* **3**(558): 136–9

Fernando DJ, Masson EA, Veves A, Boulton AJ (1991) Relationship of limited joint mobility to abnormal foot pressures and diabetic foot ulceration. *Diabetes Care* **14**(1): 8–11

Fletcher EM (1990) Foot care in the community for people with diabetes. *Practical Diabetes International* **7**: 171–2

Goodfield MJ, Millard LG (1988) The skin in diabetes mellitus. *Diabetologia* **31**(8): 567–75

'Recognising subtle skin or joint changes, which on their own may appear trivial but are major when combined with neuropathy or ischaemia, is very important.'

Harkless LB, Dennis KJ (1987) You see what you look for and recognize what you know. *Clinics in Podiatric Medicine and Surgery* **4**(2): 331–9

Howell SA, Clayton YN, Phan QC (1998) Tinea Pedis. The relationship between symptoms, organisms and host characteristics. *Microbial Ecology in Health and Disease* **1**: 131–5

Huntley AC (1982) The cutaneous manifestations of diabetes mellitus. *Journal of the American Academy of Dermatology* **7**(4): 427–55

Ismail AA, Dasgupta B, Tanqueray AB, Hamblin JJ (1996) Ultrasonographic features of diabetic cheiroarthropathy. *British Journal of Rheumatology* **35**(7): 676–9

Jeffcoate W, Lima J, Nobrega L (2000) The Charcot Foot. *Diabetic Medicine* **17**(4): 253–8

Jelinek JE (1994) Cutaneous manifestations of diabetes mellitus. *International Journal of Dermatology* **33**(9): 605–17

Jude EB, Boulton AJ (2001) Update on Charcot neuroarthropathy. *Current Diabetes Reports* **1**(3): 228–32

Leyden JL (1994) Tinea Pedis pathophysiology and treatment. *Journal of the American Academy of Dermatology* **31**(3 pt2): S31–3

Loden M (1996) Urea-containing moisturizers influence barrier properties of normal skin. *Archives of Dermatological Research* **288**(2): 103–7

Masson EA, Angle S, Roseman P, Soper C, Wilson I, Cotton M, Boulton AJM (1989) Diabetic foot ulcers: do patients know how to protect themselves? *Practical Diabetes International* **6**(1): 22–3

Miettinen H, Johansson G, Gobom S, Swanbeck G (1999) Studies on constituents of moisturizers: water-binding properties of urea and NaCl in aqueous solutions. *Skin Pharmcology and Applied Skin Physiology* **12**(6): 344–51

Murray HJ, Young MJ, Hollis S, Boulton AJ (1996) The association between callus formation, high pressures and neuropathy in diabetic foot ulceration. *Diabetic Medicine* **13**(11): 979–82

National Institute for Clinical Excellence (NICE; 2004) *Type 2 diabetes – prevention and management of foot problems.* NICE, London. Available at http://www.nice.org.uk/CG010NICEguideline (accessed 25.11.2005)

Nicander I (1988) *Electrical impedance related to experimentally induced changes in human skin and oral mucosa.* Thesis (PhD). Karolinska Institute, Stockholm

Pigatto P, Bigardi A, Canistraci C, Picardo M (1996) 10% Urea cream (Lanceran) for atrophic dermatitis: A clinical and laboratory evaluation. *Journal of Dermatology Treatments* **7**: 171–5

Reiber GE, Vileikyte L, Boyko EJ, del Aguila M, Smith DG, Lavery LA, Boulton AJ (1999) Causal pathways for incident lower-extremity ulcers in patients with diabetes from two settings. *Diabetes Care* **22**(1): 157–62

Roberts DT (1992) Prevalence of dermatophyte onychomycosis in the United Kingdom: results of an omnibus survey. *The British Journal of Dermatology* **126**(suppl 39): 23–7

Rosen RC, Davids MS, Bohanske LM, Lemont H (1985) Hemorrhage into plantar callus and diabetes mellitus. *Cutis* **35**(4): 339–41

Rosenbloom AL, Silverstein JH, Lezotte DC, Richardson K, McCallum M (1981) Limited joint mobility in childhood diabetes indicates increased risk for microvascular disease. *New England Journal of Medicine* **305**(4): 191–4

Ryder RE, Marshall R, Johnson K, Ryder AP, Owens DR, Hayes TM (1988) Acetylcholine sweatspot test for autonomic denervation. *Lancet* **1**(8598): 1303–5

Saunders LJ, Frykberg RG (1991) Diabetic neuropathic osteoarthropathy: The Charcot foot. In: Frykberg RG, ed. *The high Risk Foot in Diabetes Mellitus.* Churchill Livingstone, New York, 297–338

Semel JD, Goldin H (1996) Association of athlete's foot with cellulitis of the lower extremities: diagnostic value of bacterial cultures of ipsilateral interdigital space samples. *Clinical Infectious Diseases* **23**(5): 1162–4

Shemer A, Bergman R, Linn S, Kantor Y, Friedman-Birnbaum R (1998) Diabetic dermopathy and internal complications in diabetes mellitus. *International Journal of Dermatology* **37**(2): 113–5

Sueki H, Fujisawa R (1986) [Pigmented pretibial patches, with special references to the clinical classification and the correlation to HbA1 which serves as an index of diabetic control]. *Nippon Hifuka Gakkai zasshi – The Japanese Journal of Dermatology* **96**(3): 156–63

Thomson FJ, Masson EA (1992) Can elderly diabetic patients co-operate in routine foot-care? *Age and Ageing* **21**(5): 333–7

Uccioli L, Faglia E, Monticone G, Favales F, Durola L, Aldeghi A et al (1995) Manufactured shoes in the prevention of diabetic foot ulcers. *Diabetes Care* **18**(10): 1376–8

Yosipovitch G, Hodak E, Vardi P, Shraga I, Karp M, Sprecher E, David M (1998) The prevalence of cutaneous manifestations in IDDM patients and their association with diabetes risk factors and microvascular complications. *Diabetes Care* **21**(4): 506–9