

Diabetic foot ulcers

Pathophysiology, assessment, and therapy

C. Keith Bowering, MD, FRCP, FACP

abstract

OBJECTIVE To review underlying causes of diabetic foot ulceration, provide a practical assessment of patients at risk, and outline an evidence-based approach to therapy for diabetic patients with foot ulcers.

QUALITY OF EVIDENCE A MEDLINE search was conducted for the period from 1979 to 1999 for articles relating to diabetic foot ulcers. Most studies found were case series or small controlled trials.

MAIN MESSAGE Foot ulcers in diabetic patients are common and frequently lead to lower limb amputation unless a prompt, rational, multidisciplinary approach to therapy is taken. Factors that affect development and healing of diabetic patients' foot ulcers include the degree of metabolic control, the presence of ischemia or infection, and continuing trauma to feet from excessive plantar pressure or poorly fitting shoes. Appropriate wound care for diabetic patients addresses these issues and provides optimal local ulcer therapy with débridement of necrotic tissue and provision of a moist wound-healing environment. Therapies that have no known therapeutic value, such as foot soaking and topical antiseptics, can actually be harmful and should be avoided.

CONCLUSION Family physicians are often primary medical contacts for patients with diabetes. Patients should be screened regularly for diabetic foot complications, and preventive measures should be initiated for those at risk of ulceration.

résumé

OBJECTIF Examiner les causes sous-jacentes de l'ulcération diabétique des pieds, présenter une évaluation pratique des patients à risque et exposer une approche fondée sur des données probantes pour la thérapie des patients diabétiques souffrant d'ulcères aux pieds.

QUALITÉ DES DONNÉES Une recension a été effectuée dans MEDLINE de 1979 à 1999 pour trouver des articles portant sur les ulcères diabétiques aux pieds. La plupart des études trouvées comportaient des séries de cas ou des essais contrôlés de petite envergure.

PRINCIPAL MESSAGE Les ulcères aux pieds chez les patients diabétiques sont fréquents et aboutissent souvent à l'amputation du membre inférieur, à moins qu'une thérapie prompte, rationnelle et multidisciplinaire soit entreprise. Au nombre des facteurs qui influencent le développement et la guérison des ulcères aux pieds chez les patients diabétiques figurent le degré de contrôle métabolique, la présence d'ischémie ou d'une infection et le traumatisme constant aux pieds causés par une pression plantaire excessive ou des chaussures mal ajustées. Un traitement approprié de la blessure chez les patients diabétiques règle ces problèmes et prévoit une thérapie optimale locale de l'ulcère, notamment débrider les tissus nécrosés et assurer un environnement humide pour la guérison de la blessure. Des traitements n'ayant aucune valeur thérapeutique connue, comme les bains de pied et les antiseptiques locaux, peuvent être en fait dommageables et devraient être évités.

CONCLUSION Les médecins de famille sont souvent le premier point de contact médical pour les patients souffrant de diabète. Un dépistage régulier d'éventuelles complications du diabète apparaissant aux pieds devrait être fait chez ces patients et des mesures de prévention devraient être prises chez les personnes à risque d'ulcération.

This article has been peer reviewed.

Cet article a fait l'objet d'une évaluation externe.

Can Fam Physician 2001;47:1007-1016.

Diagnosed diabetes currently affects approximately 5% of the general Canadian population. That figure is expected to double over the next decade.^{1,2} Unfortunately, many diabetic patients over time will develop the chronic complications of diabetes: retinopathy, nephropathy, peripheral neuropathy, and atherosclerotic vascular disease. From a patient's perspective, one of the most feared of these complications is loss of a leg as a consequence of peripheral neuropathy or ischemia.

Diabetes is the most common disease process associated with lower limb amputation, accounting for approximately half of nontraumatic amputations in North America and Europe.^{3,4} Up to 85% of lower limb amputations in diabetic patients are preceded by foot ulcers that fail to heal.⁵ About 2% to 3% of all diabetic patients will develop a foot ulcer every year, and many of these will require prolonged hospitalization for treatment of complications of ensuing infection or gangrene.^{6,7} These foot problems affect the health care system considerably. A recent report estimated that direct annual costs of diabetic foot care in the United States were approximately \$5 billion (US).⁸

Despite these chilling statistics, progress has been made in treating diabetic foot ulcers and complications. Some centres specializing in diabetic foot management have documented reductions in amputation rates in the range of 50% after a coordinated, multidisciplinary effort incorporating evidence-based wound care is instituted.^{9,10} Before patients reach specialized foot clinics, however, primary care of foot ulcers is frequently provided by family physicians. This article reviews a general approach to diabetic patients with foot ulcers and examines recent developments in wound therapy.

Quality of evidence

The information presented in this review is based on a MEDLINE search of relevant articles on assessment and management of diabetic feet published during a 20-year period from January 1979 to September 1999. Important research that predated 1979 and that was referenced in these articles was also evaluated. Key search terms included diabetic foot ulcer, peripheral vascular disease, peripheral neuropathy, infection, osteomyelitis, wound and ulcer care, and amputation.

.....
Dr Bowering is Medical Director of the multidisciplinary Diabetic Foot Clinic at the Royal Alexandra Hospital in Edmonton, Alta. He is a Clinical Professor of Medicine and a member of the Division of Endocrinology at the University of Alberta.

Unfortunately, there are no randomized controlled, prospective trials on many aspects of diabetic foot therapy. Most reports are descriptive case series, although more recent multicentre studies on newer technologic approaches to diabetic ulcers are more scientific. Recommendations for assessment and management are based on an overall consensus derived from available literature.

Etiology of diabetic foot problems

The main underlying risk factors for foot ulcers in diabetic patients are peripheral neuropathy and ischemia.

Neuropathy. Epidemiologic studies have found a point prevalence of distal lower limb neuropathy ranging from 30% to 50% of the diabetic population studied.^{11,12} Both type 1 and type 2 diabetic patients are similarly affected. With such frequent occurrence of neuropathy, it is no surprise that more than 60% of diabetic patients' foot ulcers are primarily due to underlying neuropathy.¹³

The distal neuropathy of diabetes affects all components of the nervous system: sensory, motor, and autonomic, each of which contributes to foot ulcer development. Loss of nerve function correlates with chronic hyperglycemia, as reflected in the mean level of glycosylated hemoglobin over time.¹⁴ Ischemia of the endoneurial microvascular circulation induced by metabolic abnormalities from hyperglycemia is believed to be the underlying mechanism for nerve deterioration.^{15,16}

Motor nerve involvement: Loss of neural supply to the intrinsic muscles of the foot produces an imbalance of the long flexor and extensor tendons. Contraction of the more powerful flexors of the lower limb induces the classic high-arched foot and claw-toe deformity seen in as many as 50% of patients with diabetes.⁷ Hyperextension of the toes with resultant overriding of the metatarsal-phalangeal joints forces the metatarsal heads downward, thereby increasing their prominence. Hyperextension of the toes displaces the metatarsal fat pads distally, further reducing the natural cushioning of the metatarsal heads. These mechanical changes increase plantar pressures inducing callus formation and underlying skin breakdown.

Spaying of the foot from loss of the intrinsic muscles, in combination with disruption of the normal bony relationships of the distal foot, culminates in a foot that is wider and thicker than normal. Shoes that once fit that patient therefore no longer fit. Ill-fitting shoes cause areas of local trauma (**Figure 1**).

Figure 1. Lateral foot ulcer in a diabetic patient with peripheral artery disease: Lesion was precipitated by poorly fitting shoes.



Autonomic neuropathy: Autonomic dysfunction of the foot from diabetic neuropathy results in loss of sweat and oil gland function. Anhidrosis leads to dry, fissured skin susceptible to bacterial invasion. Furthermore, loss of peripheral sympathetic vascular tone in the lower limb increases distal arterial flow and pressure, which, by damaging the capillary basement membrane, might contribute to peripheral edema.¹⁷ Edema increases the risk of foot ulceration by adding another element of minor trauma caused by wearing shoes that fit even more poorly as the edema increases.

Sensory neuropathy: The motor and autonomic neural abnormalities would have far less effect were it not for the concurrent loss of protective sensation in the foot. Normally, if the foot developed a fissure or blister, or if bony structure changed, patients would feel the discomfort and take appropriate corrective measures. Unfortunately, with onset of the peripheral neuropathy of diabetes, this protective response diminishes and can eventually disappear with progressive reduction in nerve function. This sequence of events allows patients to walk with apparent comfort on ever-deepening ulcers. The lack of pain lulls patients, and often physicians, into a false sense of security, a misguided “but it doesn’t hurt; therefore it cannot be a serious problem” mentality.

Ischemia. The other major underlying cause of diabetic foot ulcers is peripheral vascular disease. Primarily ischemic ulcers without substantial accompanying neuropathy account for approximately 15% to 20% of foot ulcers, and another 15% to 20% have a mixed neuropathic-vascular etiology.¹³ Overall, atherosclerosis of the lower limbs in people with diabetes

occurs at least two or three times more often than in people without diabetes and has a predilection for affecting the tibial and peroneal arteries of the calf with relative sparing of the arteries of the foot.^{18,19} This pattern differs from the general population, where more proximal atherosclerotic changes predominate.

Involvement of the peripheral autonomic nervous system has been proposed to explain the more distal distribution of lower limb atherosclerosis among diabetic patients. Autonomic dysfunction reduces the normal vasoconstriction that occurs in the lower leg arteries with standing and results in an increase in the intraluminal flow and pressure that is aggravated by gravitational forces.²⁰ Reduced vasoconstrictive ability further reduces vessels’ capacity to expand in response to systolic pressure. The combination of high flow and reduced wall motion encourages formation of plaque in calf arteries.²¹

Interestingly, among diabetic patients, smoking does not seem to be associated with recurrent foot ulcers or risk of amputation from ischemia.^{5,22,23} These data conflict with information about patients without diabetes, which clearly indicates that smoking is a risk factor for claudication and amputation.²⁴ Regardless of whether smoking affects lower limb complications, diabetic patients who smoke have all-cause mortality twice that of non-smoking diabetic patients and should be strongly counseled to end cigarette consumption.²⁵

Assessment of diabetic feet

Despite the frequency of complications involving diabetic patients’ lower limbs, primary care practitioners frequently neglect to examine their feet. Surveys of physicians and patient chart evaluations have determined that fewer than 50% of diabetic patients receive appropriate foot evaluation as part of their annual medical checkups.²⁶⁻²⁸

Patients themselves are often unaware of serious foot problems because neuropathy removes the pain that would normally alert them. A recent community study discovered that 10% of patients diagnosed with diabetic foot ulcers did not know themselves that they had ulcers until they were advised by physicians.²⁹ Consensus panels have recommended annual screening foot examinations be performed for all diabetic patients older than 15 years and even more frequent assessments if patients are at risk from peripheral ischemia or neuropathy.^{18,30}

Neurologic assessment. Assessment of peripheral neuropathic involvement of the foot in diabetic patients

CME

Diabetic foot ulcers

should include determination of ankle reflexes, of vibration threshold (by means of a 128-Hz tuning fork), and presence or absence of protective sensation with a 10-g Semmes-Weinstein monofilament. Loss of any of the above parameters has been shown to correlate with development of neuropathic ulcers (**Figure 2**). Of these assessments, the 10-g filament is the most practical approach. A prospective study published in *Diabetes Care* in 1992 showed the loss of sensation to the 10-g filament on the sole of the foot was associated with a 10-fold risk of foot ulceration and a 17-fold risk of amputation over a 32-month follow-up period.³¹ The most important areas to assess are uncalled regions of the plantar surface of the metatarsal heads, although some authors advocate assessing as many as 10 spots over the sole of the foot from the toes to the heel.^{32,33}

Figure 2. Neuropathic ulcer on metatarsal head



Pinprick sensation and temperature discrimination are also affected by diabetic neuropathy. Their usefulness in identifying patients at risk of ulcer or amputation is limited because various clinical observers could not reproduce one another's observations.

Circulatory assessment. Assessment of peripheral circulation in diabetic patients includes the standard evaluation for pedal pulses; however, examiners should be aware of the possible pitfalls of using presence of pulses alone to exclude clinically significant peripheral ischemia. Rivers et al³⁴ describe a series of diabetic patients who had sufficiently severe peripheral ischemia to warrant distal surgical bypass procedures despite the presence of readily palpable pedal pulses.³⁴ Consequently, in addition to clinical parameters, noninvasive measures of circulation are frequently used to complement physical examination in assessing the degree of arterial obstruction.

Although the ankle-brachial index (ABI) as determined by Doppler ultrasonography is used to indicate

adequacy of peripheral blood flow in patients without diabetes, the ABI is less reliable in diabetic patients because calcification of the media of the distal arteries is common. This calcification makes the vessels relatively non-compressible, resulting in an artificially high systolic pressure in the ankle.³⁵

More reliable methods of assessing potential for healing foot ulcers in diabetic patients suspected of having peripheral ischemia involve systolic toe pressure measurements by photoplethysmography or measurement of distal transcutaneous oxygen tension.^{36,37} Both these latter assessments are performed in specialty diabetic foot clinics or vascular laboratories and offer an indication of potential for healing before angiography is considered. A contrast angiogram remains the criterion standard of assessment in patients with peripheral vascular problems but has to be undertaken with caution among patients with diabetes who often already have nephropathy. Using contrast dye in patients with renal disease can result in complete renal shutdown.

Radiologic assessment. All patients with foot ulcers should have a baseline x-ray examination as part of their initial evaluation to find any unknown radiopaque foreign body and to evaluate foot anatomy for changes indicating neuroarthropathy (Charcot joint) or osteomyelitis. A plain x-ray film alone, however, is an insensitive method of determining whether an ulcer is complicated by osteomyelitis. In the early stages of osteomyelitis, a plain x-ray film can show normal structures, and it can take 3 weeks after onset of bony infection before radiologic changes are evident. Even with chronic osteomyelitis, the plain x-ray film was positive in only 25% of cases in one small series.³⁸ The three-phase technetium bone scan has a greater sensitivity for osteomyelitis than plain x-ray films but in one study had a sensitivity of only 69% and a specificity even lower (39%).³⁹

Gallium scanning in combination with technetium improves the test's specificity. White blood cell scanning with indium-111 results in an even better sensitivity of 89% and a specificity of 69% for detecting osteomyelitis in diabetic patients.³⁹ Combining technetium scanning with indium white blood cell scanning improves the sensitivity and specificity to 100% and 80%, respectively, but has the drawback of being expensive and time-consuming.⁴⁰ Magnetic resonance imaging could also be an effective tool for assessing osteomyelitis, although at this point studies examining its role for diabetic patients have involved only a few patients.^{41,42}

A low-cost method of clinically evaluating diabetic patients with foot ulcers for osteomyelitis is highly specific but of low sensitivity. The method involves palpation of bone through a foot ulcer using a blunt-tipped steel probe. If bone can be felt beneath a foot ulcer, there is almost always underlying bone infection.⁴³

Therapy for diabetic foot ulcers

An understanding of the processes that precipitate and propagate foot ulcers in diabetic patients should dictate a rational approach to therapy.

Blood sugar control. Most foot ulcers have their origins in inadequate control of blood sugar, which results in development of lower limb neuropathy. There is now excellent evidence that improved control of diabetes can markedly reduce the incidence of neuropathy.⁴⁴ On the other hand, no convincing evidence yet shows that improved glucose levels definitively reduce development of peripheral atherosclerosis. Nevertheless, trials incorporating intensive blood glucose control in diabetic patients to reduce long-term complications suggest a trend to fewer macrovascular events in groups receiving intensive therapy. The trend is not statistically significant but deserves further study.

To date, no randomized controlled studies have been performed to determine whether improved glucose control has benefit once a foot ulcer has developed in humans. Animal studies do indicate that hyperglycemia impairs normal wound healing and that healing improves as blood sugar is reduced to more reasonable levels.⁴⁵ Host defenses against infection, particularly white blood cell function, are also adversely affected in the presence of elevated blood sugar levels but improve as blood sugar is reduced below 14 mmol/L.⁴⁶ The abnormal white blood cell response, in combination with a reduced neuro-inflammatory reaction, could explain why up to 50% of diabetic patients with deep foot infections do not have clinical fever or elevated white blood cell count at time of presentation.^{47,48}

Pressure relief and avoiding further trauma.

Unrecognized injury from poorly fitting footwear is a frequent precipitating event for foot ulcers in patients with peripheral neuropathy and loss of protective sensation.⁴⁹ Teaching patients to purchase appropriate shoes with sufficient width, depth, and arch support can prevent many foot ulcers from developing.

Once an ulcer develops, ongoing trauma from continuing to walk on the affected area prevents healing.

Offloading the affected limb is therefore most important. Although in clinical practice offloading often involves using a wheelchair or crutches in combination with therapeutic shoes, these methods are limited in their usefulness by how much patients use them. The most studied and effective offloading technique has been the total contact cast.

Use of total contact casts for neuropathic, non-ischemic ulcers has resulted in healing rates of 90% within 1 or 2 months of initial application.⁵⁰ Unfortunately, not every diabetic patient with a foot ulcer is a candidate for a total contact cast. These casts are unsuitable for infected or ischemic ulcers and require experienced technicians who have been specifically trained in application. The casts completely enclose neuropathic feet and lower limbs in plaster with little padding except in a few critical areas. A noncompliant patient or a cast applied to an inappropriate limb by an inexperienced technician can have disastrous consequences.

An alternative to a total contact cast is removable plastic walkers (**Figure 3**), which also have proven efficacy in helping to offload plantar ulcers.⁵¹ Although removable walkers are gaining popularity because of ease of use, it remains to be determined whether they are as efficacious as total contact casts in healing neuropathic plantar ulcers.

For many patients, maintaining offloading is the most difficult aspect of healing diabetic foot ulcers. Work commitments, or simply performing the activities of daily living, result in repetitive injury

Figure 3. Plastic walker



CME

.....

Diabetic foot ulcers

to the area. A social worker is often an essential member of the health care team to assist in addressing patient needs. The importance of absolute respite from weight bearing on the affected foot needs to be reinforced continually.

Peripheral vascular disease. Improved peripheral blood flow after distal revascularization surgery has frequently salvaged limbs in diabetic patients with limb-threatening obstruction in the tibial or peroneal arteries who otherwise would have proceeded to amputation.⁵² Popliteal to plantar-arch vein-graft bypasses have demonstrated long-term patency rates in the range of 80% and justify pursuing vascular reconstruction aggressively in suitable surgical candidates.

Unfortunately, comorbidity often dictates a non-surgical approach to the problem of peripheral vascular insufficiency. Few medical options are available. Arterial vasodilators have largely been abandoned because they are ineffective. Pentoxifylline, officially defined as a hemorheologic agent that improves deformability of stiff red blood cells, has been used for peripheral vascular disease to improve distal flow, but no randomized controlled large-scale prospective trials showed benefit in diabetic patients. Anecdotal reports suggest pentoxifylline is occasionally helpful when used in higher dosages over longer periods in diabetic patients, but essentially the agent is used as a last resort.

Antiplatelet agents can modify the natural course of peripheral arterial insufficiency, delaying progression of occlusive disease and decreasing the need for arterial reconstruction when used for primary prevention.⁵³ Their role is essentially to maintain the flow that already exists and to reduce the potential for further thrombotic occlusion.

Infection. Infection arising from diabetic foot ulcers frequently leads to amputation.⁵ Deep infections require early, aggressive surgical débridement in addition to antibiotics. The choice of antibiotics ultimately, of course, depends on the results of bacterial culture. Initially, while cultures are being obtained, the spectrum of antibiotics should reflect the broad variety of bacteria isolated from these wounds in diabetic patients. Mixed bacterial flora involving Gram-positive and Gram-negative aerobes and anaerobes are common. Deep infections have a reported mean of 4.8 to 5.8 different types of bacteria, typically requiring a combination of antibiotics such as ciprofloxacin and clindamycin orally, or imipenem-cilastatin parenterally.^{54,55}

Culture technique is critical in obtaining reliable results from diabetic foot infections. Surface swabs from a foot ulcer site are notoriously inadequate for identifying the type of bacteria causing limb-threatening deep infection. The most accurate technique involves removing surface exudate from the ulcer, obtaining a small tissue biopsy from the base of the ulcer, and sending the sample of tissue to the laboratory in appropriate aerobic and anaerobic culture material.⁵⁵

Topical ulcer therapy. Primary principles of good wound care incorporate regular débridement to remove necrotic material from the ulcer site and application of appropriate dressings. Encrusted, callused areas should be deroofed. Débridement is most efficient when forceps and scalpel are used. Foot soaking and whirlpool therapy are unhelpful in débridement and can actually promote further tissue damage from maceration or excessive heat.⁵⁶ Topical antiseptics, such as hydrogen peroxide, are toxic to healing dermal cells and are to be avoided.⁵⁷ Two new agents, cadexomer iodine (Iodosorb) and an antimicrobial silver dressing (Acticoat) have shown promise as topical antimicrobials that do not harm underlying tissue.^{58,59} No studies have shown traditional topical antibiotic creams and ointments to be beneficial in therapy of diabetic foot ulcers.

For all wounds, preservation of a moist wound environment aids healing. Ideal topical therapy for ulcers should keep the ulcer bed moist, but not excessively wet so as to promote maceration. Moistened gauze dressings are frequently used but have a tendency to dry out unless changed several times daily. This changes the moist dressing to a wet-to-dry dressing, which loses the proven benefits of the moist environment. Removal can also damage healing tissue. A variety of commercially available hydrogels, such as Intrasite Gel or Duoderm Gel, can maintain the moist environment necessary to the wound and can be applied topically every 1 to 3 days depending on moisture requirements. A non-adherent top dressing, such as ETE or Allevyn, can then be applied over the hydrogel. (A referenced guide to specific wound care is available through the Capital Health Authority, Regional Wound Care Guidelines, #300, 10216-124 St, Edmonton, AB T5N 4A3.)

Other therapies

Hyperbaric oxygen: Hyperbaric oxygen has been used as adjunct therapy for diabetic foot ulcers. A series of small studies demonstrated more rapid

healing and reduced amputation rates in patients treated with hyperbaric oxygen than in control subjects.^{60,61} Most trials using hyperbaric oxygen, however, have not been randomized or have been otherwise criticized for their methods. The current consensus recommendation is that hyperbaric oxygen could be a useful adjunct therapy for standard approaches to wound care in diabetic patients but that this field requires further investigation.⁶²

Tissue engineering and growth factors: Despite adequate blood flow and good wound care, some neuropathic diabetic foot ulcers fail to heal. Impairment of the normal cellular functions involving growth factors and fibroblasts necessary for wound healing has been postulated to account for the failure to close these wounds.^{63,64}

Recent advances in technology have produced growth factors (such as platelet-derived growth factor) and tissue engineering of living skin replacements to replenish components of the wound care process. Dermagraft and becaplermin are two examples of these new technologies that have a proven benefit in some diabetic patients with nonhealing ulcers. Dermagraft is a cryopreserved, cultured human dermis grown from fibroblasts. It has been shown to induce more rapid and frequent healing of chronic neuropathic diabetic foot ulcers than standard care in a study where the tissue was implanted weekly in the ulcer for up to 8 weeks.⁶⁵ Becaplermin is a recombinant platelet-derived growth factor in a gel form that, when applied daily to diabetic patients with chronic foot ulcers, also demonstrated greater healing than regular wound care over a 20-week assessment period.⁶⁶ Because these newer technologies are expensive, however, they are generally reserved for patients with neuropathic foot ulcers who continue to exhibit poor healing despite optimal wound care with débridement, offloading of pressure surfaces, provision of a moist wound environment, and control of infection and ischemia.

Patients at high risk for recurrence

Once foot ulcers have healed, the challenge for both physicians and patients is to maintain skin integrity. Patient education is essential. For example, daily self-inspection of feet for areas of erythema or callus buildup will alert patients to excessive pressure from tight shoes or inadequate plantar pressure relief before underlying skin breaks down.

A patient instruction sheet outlining such protective measures is shown in **Table 1**. The reward for providing patients with such information has been demonstrated quite well in a study where diabetic

Editor's key points

- Diabetes is the most common medical condition leading to lower limb amputation, and 85% of amputations are preceded by foot ulcers that fail to heal.
- The main risks from diabetes are peripheral ischemia and neuropathy (both sensory and motor).
- Assessment includes testing ankle reflexes and vibration threshold, determining the degree of protective sensation, evaluating circulation (pulses are often present despite serious ischemia), and considering x-ray examinations and bone scans for osteomyelitis.
- Management includes good blood sugar control, avoiding pressure or trauma with good shoes or a special total contact cast, treating infection, improving circulation, and using topical therapy.

Points de repère du rédacteur

- Le diabète représente l'état pathologique le plus communément rencontré aboutissant à l'amputation d'un membre inférieur et 85% des amputations sont précédées par des ulcères non guéris aux pieds.
- Les principaux risques du diabète se situent dans l'ischémie et la neuropathie périphériques (sur le plan sensoriel et de la motricité).
- L'évaluation se fait notamment par une vérification des réflexes et du seuil de vibration au niveau des chevilles, la détermination du degré de sensation protectrice, l'évaluation de la circulation (le pouls est souvent perceptible malgré une ischémie sérieuse) et on peut envisager une radiographie ou une scanographie pour détecter une ostéomyélite.
- La prise en charge comporte le contrôle de la glycémie, le port de bonnes chaussures ou d'un plâtre spécial de contact total pour éviter la pression ou le traumatisme, le traitement de l'infection, l'amélioration de la circulation sanguine et le recours à une thérapie topique.

patients given specific foot-care instructions by their physicians were compared with a control group given no specific teaching.⁶⁷ The intervention group had less than half the number of serious foot lesions over the ensuing 12 months than the control population. Regular visits to a podiatrist or chiropodist to remove calluses and properly trim toenails encourage patients not to risk performing their own "bathroom surgery."

Footwear that eliminates pressure points and custom orthotics that more evenly distribute plantar pressures also unequivocally reduce recurrence of ulcers in patients who have lost protective sensation. In one

Table 1. Foot care advice for patients with diabetes

INSPECTION

- Take time every day to look at your feet from all sides, including the space between your toes. If you cannot see the bottom of your feet well, use a mirror or ask a family member to look for you.
- Look for dry patches and cracks in the skin. These areas need special care with moisturizing lotion.
- Look for white, moist, wrinkly skin (especially between the toes). Changes in the skin between the toes might require prescription medication from your physician.
- Check for cuts, blisters, corns, calluses, swelling, ingrown toenails, or places that are red or pale. Feel for increased heat in the skin. Areas of concern should be discussed with your doctor.

BATHING

- Wash your feet daily in warm (not hot) water. Test the water temperature with your wrist or elbow to avoid burning your feet.
- Do not soak your feet. Soaking actually dries out your skin by removing the natural oils.
- Gently dry your feet with a soft towel, making sure to dry between each toe. Never use a hair dryer to dry or warm your feet.
- Apply a good moisturizing lotion to your feet after every wash. Do not apply lotion between the toes, which can make this area too wet. Spaces between the toes should be kept very clean and dry.

CORNS AND CALLUSES

- When feet are dry, gently file away mildly callused areas with a pumice stone (available at your pharmacy) to control callus buildup. Afterward, apply lotion to all callused areas to keep the callus flexible and to stop it from cracking.
- Corns and calluses are a response to pressure, often resulting from poorly fitting shoes that are too tight. Appropriate footwear and arch supports could be necessary to stop these problems from recurring.
- Avoid do-it-yourself chemical corn or callus removers. These can cause burns and damage healthy skin surrounding the corn or callus. Do not use corn pads.
- Never perform "bathroom surgery" on your feet with a razor blade or similar instrument.

TOENAILS

- Cut your toenails after bathing, when they are soft and easy to trim.
- Use a toenail clipper or file to shorten the nail but never cut the nail too short; always leave 1/16 to 1/8 inch (1 to 2 mm) of free nail. Nails may be cut straight across or shaped to follow the contour of the toe. Sharp edges need to be filed with an emery board to avoid cutting the toes next to them.
- Do not use sharp objects to poke or dig under the toenail or around the cuticle.
- Ingrown nails, or nails that are thick or tend to split when cut, should be cared for by a professional trained in foot care.

SOCKS

- Wear clean socks every day. Socks with at least 80% cotton or wool are best to absorb perspiration and allow skin to breathe. Avoid nylon socks as much as possible.
- Socks should fit well, without tight elastic at the top or bulky seams at the toe.

SHOES

- Wear shoes and socks in the house and outside at all times to protect and support your feet.
- Suggestions for proper footwear: shoes should have thick flexible rubber soles, closed toes, and closed heels; shoes should have sufficiently wide and deep toe boxes (always avoid shoes with pointed toes); the top part of shoes should be soft and flexible; linings should not have ridges, wrinkles, or seams; laces, buckles, or velcro closures are required (avoid slip-on shoes); and running or walking shoes are often the best type of shoes to buy.
- Each time you put on your shoes, you should shake them out and then carefully feel inside for stones or other articles that could have fallen in them unnoticed.
- Shop for shoes when your feet are largest, generally later in the day. Buy shoes that feel good and have room for all the toes to wriggle and be in their natural place. Avoid shoes that pinch or are too tight. Try on both shoes and, if one of your feet is larger, buy for the larger foot.
- If your feet are numb from nerve damage caused by diabetes, have someone trace an outline of your feet while you are standing on stiff paper, cut out the outline of each foot, and bring it with you when you shop for shoes. Insert the outline into the new shoes to help determine the proper fit.
- Wear new shoes for only short periods each day at first. Frequently inspect your feet, looking for areas of redness that indicate potential problems.

CIRCULATION

- If you smoke, stop.
- Exercise regularly, 30 minutes at least three times weekly (check with your physician before embarking on any exercise program).
- Avoid using heating pads or hot water bottles. These can burn the skin without warning if you have nerve damage in your feet from diabetes. If your feet are cool, use wool socks to keep your feet warm.

TREATMENT OF INJURIES

- If you stumble or bump into a hard object, look at your feet to be sure there is no damage.
- If your foot is hurt, do not keep walking on it (even if there is no pain), as continued walking can cause more damage.
- Treat blisters, cuts, and scratches right away. Never use strong chemicals, magnesium sulfate (Epsom salts), or antiseptics on your feet. Opening blisters yourself can lead to infections. Simply clean wounds with mild soap and water.
- Cover all injuries with clean dressings until they can be assessed by your physician.
- Call your doctor if any open sore shows any of the following: heat, redness, swelling, pus, or pain.

Reprinted with permission from the Diabetic Foot Clinic and Rehabilitation Services of the Royal Alexandra Hospital in Edmonton, Alta.

study, healed foot ulcers recurred within 1 year in 58% of patients who returned to wearing their regular footwear. The 1-year recurrence rate among those who wore therapeutic wider and deeper shoes fitted with custom insoles was less than 28%.⁶⁸ Unfortunately, the success of specialized footwear in preventing redevelopment of foot ulcers is dependent on patients' wearing the shoes consistently. Patients who wear their therapeutic shoes and orthotics only part of the day have twice as many ulcers as patients who wear their prescribed footwear regularly.⁶⁹

Conclusion

Foot ulceration is a common complication of diabetes that has potentially disastrous consequences for patients. Fortunately, better control of blood sugar levels, early recognition of complications of peripheral neuropathy and ischemia, and using a multidisciplinary approach to therapy when an ulcer develops can dramatically reduce this problem. Primary care physicians are key players in this approach when they identify diabetic patients at risk for ulceration and then initiate appropriate early management plans. ❀

Competing interests

None declared

Correspondence to: Dr C. Keith Bowering, Medical Director, Diabetic Foot Clinic, Royal Alexandra Hospital, 10240 Kingsway Ave, Edmonton, AB T5H 3V9; telephone (780) 944-0589; e-mail kbowerin@ualberta.ca

References

1. Tan MH, MacLean DR. The epidemiology of diabetes mellitus in Canada. *Clin Invest Med* 1995;18(4):240-6.
2. Tan MH, Daneman D, Lau DC, MacLean DR, Ross SA, Yale JF. *Diabetes in Canada: strategies towards 2000*. Toronto, Ont: Canadian Diabetes Advisory Board; 1997. p. 3.
3. Pohjoainen T, Alaranta H. Epidemiology of lower limb amputees in Southern Finland in 1995 and trends since 1994. *Prosthet Orthot Int* 1999;23(2):88-92.
4. National Diabetes Data Group. *Diabetes in America*. Bethesda, Md: Department of Health and Human Services; 1995.
5. Pecoraro RE, Reiber GE, Burgess EM. Pathways to diabetic limb amputation: basis for prevention. *Diabetes Care* 1990;13:513-21.
6. Moss SE, Klein R, Klein BE. The prevalence and incidence of lower extremity amputation in a diabetic population. *Arch Intern Med* 1992;152(2):610-6.
7. Borssen B, Bergenheim T, Lithner F. The epidemiology of foot lesions in diabetic patients aged 15-50 years. *Diabet Med* 1990;7:438-44.
8. Amato DA, Persson U, Lantini M, Basso K, Martens L. The cost of illness of patients with diabetic foot ulcers [abstract]. *Diabetes* 1999;48(Suppl 1):A191.
9. Edmonds ME, Blundell MP, Morris ME, Cotton LT, Watkins PJ. Improved survival of the diabetic foot: the role of a specialized foot clinic. *Q J Med* 1986;60:763-71.
10. Larsson J, Apelqvist J, Arardh CD, Stenstrom A. Decreasing incidence of major amputation in diabetic patients: a consequence of a multidisciplinary foot care team approach. *Diabet Med* 1995;12:770-6.
11. Adler AI, Boyko EJ, Ahroni JH, Stensel V, Forsberg RC, Smith DG. Risk factors for diabetic peripheral sensory neuropathy. *Diabetes Care* 1997;20:1162-7.
12. Harris M, Eastman R, Cowie C. Symptoms of sensory neuropathy in adults with NIDDM in the U.S. population. *Diabetes Care* 1993;16:1446-52.
13. Grunfeld C. Diabetic foot ulcers: etiology, treatment and prevention. *Adv Intern Med* 1991;37:103-32.
14. Dyck PJ, Davies JL, Wilson DM, Service FJ, Melton LJ III, O'Brien PC. Risk factors for severity of diabetic polyneuropathy. *Diabetes Care* 1999;22:1479-86.
15. Younger DS, Rosoklija G, Hays AP. Diabetic peripheral neuropathy. *Semin Neurol* 1998;18(1):95-104.
16. Cameron NE, Cotter MA. Metabolic and vascular factors in the pathogenesis of diabetic neuropathy. *Diabetes* 1997;46(Suppl 2):S31-7.
17. Katz MA, McCuskey P, Beggs JL, Johnson PC, Gaines JA. Relationships between microvascular function and capillary structure in diabetic and nondiabetic human skin. *Diabetes* 1989;38(10):1245-50.
18. Mayfield JA, Reiber GE, Sanders LJ, Janisse D, Pogach LM. Preventive foot care in people with diabetes. *Diabetes Care* 1998;21:2161-77.
19. LoGerfo FW, Coffman JD. Vascular and microvascular disease of the foot in diabetes; implications for foot care. *N Engl J Med* 1984;311:1615-8.
20. Rayman G, Hassan A, Tooke JE. Blood flow in the skin of the foot related to posture in diabetes mellitus. *BMJ (Clin Res Ed)* 1986;292(6513):87-90.
21. McMillan DE. Blood flow and the localization of atherosclerotic plaque. *Stroke* 1985;16:582-7.
22. Apelqvist J, Agardh CD. The association between clinical risk factors and outcome of diabetic foot ulcers. *Diabetes Res Clin Pract* 1992;18(1):43-53.
23. Mantey I, Foster AV, Spencer S, Edmonds ME. Why do foot ulcers recur in diabetic patients? *Diabet Med* 1999;16:245-9.
24. Krupski WC. The peripheral vascular consequences of smoking. *Ann Vasc Surg* 1991;5(3):291-304.
25. Muhlhauser I. Cigarette smoking and diabetes: an update. *Diabet Med* 1994;11:336-43.
26. Bailey TS, Yu HM, Rayfield EJ. Patterns of foot examination in a diabetes clinic. *Am J Med* 1985;78:371-4.
27. Payne TH, Gabella BA, Michael SL, Young WF, Pickard J, Hofeldt FD, et al. Preventive care in diabetes mellitus: current practice in urban health-care system. *Diabetes Care* 1989;12:745-7.
28. Mayfield JA, Rith-Najarian SJ, Acton KJ, Schraer CD, Stahn RM, Johnson MH, et al. Assessment of diabetes care by medical record review. *Diabetes Care* 1994;17:918-23.
29. Walters DP, Gatling W, Mullee MA, Hill RD. The distribution and severity of diabetic foot disease; a community study with comparison to a non-diabetic group. *Diabet Med* 1992;9:354-8.
30. Meltzer S, Leiter L, Daneman D, Gerstein HC, Lau D, Ludwig S, et al. 1998 clinical practice guidelines for the management of diabetes in Canada. *Can Med Assoc J* 1998;159(Suppl 8):S1-S29.
31. Rith-Najarian SJ, Stolusky T, Gohdes DM. Identifying diabetic patients at high risk for lower-extremity amputation in a primary health care setting. *Diabetes Care* 1992;15:1386-9.
32. Mueller MJ. Identifying patients with diabetes mellitus who are at risk for lower-extremity complications: use of the Semmes-Weinstein monofilament. *Phys Ther* 1996;76:68-71.
33. McGill M, Molyneaux L, Spencer R, Heng LF, Yue DK. Possible sources of discrepancies in the use of the Semmes-Weinstein filament. *Diabetes Care* 1999;22:598-602.
34. Rivers SP, Scher L, Veith FJ. Indications for distal arterial reconstruction in the presence of palpable pedal pulses. *J Vasc Surg* 1990;12(5):552-7.
35. Goss DE, Stevens M, Watkins PJ, Baskerville PA. Falsely raised ankle/brachial pressure index: a method to determine tibial artery compressibility. *Eur J Vasc Surg* 1991;5(1):23-6.

36. Apelqvist J, Castenfors J, Larsson J, Stenstrom A, Agardh CD. Prognostic value of systolic ankle and toe blood pressure in outcome of diabetic foot ulcer. *Diabetes Care* 1989;12:373-8.
37. Ballard JL, Eke CC, Bunt TJ, Killeen JD. A prospective evaluation of transcutaneous oxygen measurements in the management of diabetic foot problems. *J Vasc Surg* 1995;22(4):485-90.
38. Newman LG, Waller J, Palestro CJ, Schwarz M, Klein MJ, Hermann G, et al. Unsuspected osteomyelitis in diabetic foot ulcers. *JAMA* 1991;266(9):1246-51.
39. Newman LG. Imaging techniques in the diabetic foot. *Clin Podiatr Med Surg* 1995;12(1):75-86.
40. Johnson JE, Kennedy EJ, Shereff MJ, Patel NC, Collier BD. Prospective study of bone, indium-111-labelled white blood cell, and gallium-67 scanning for the evaluation of osteomyelitis in the diabetic foot. *Foot Ankle Int* 1996;17(1):10-6.
41. Cook TA, Rahim N, Simpson HC, Galland RB. Magnetic resonance imaging in the management of diabetic foot infection. *Br J Surg* 1996;83(2):245-8.
42. Morrison WB, Schweitzer ME, Wapner KL, Hecht PJ, Gannon FH, Behm WR. Osteomyelitis in feet of diabetes: clinical accuracy, surgical utility, and cost-effectiveness of MR imaging. *Radiology* 1995;196(2):557-64.
43. Grayson ML, Gibbons GW, Balogh K, Levin E, Karchmer AW. Probing to bone in infected pedal ulcers. A clinical sign of underlying osteomyelitis in diabetic patients. *JAMA* 1995;273(9):721-3.
44. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment on the development and progression of long-term complications in insulin-dependent diabetes. *N Engl J Med* 1993;329:977-86.
45. Gottrup F, Andreassen TT. Healing of incisional wounds in stomach and duodenum; the influence of experimental diabetes. *J Surg Res* 1981;31:61-8.
46. Bagdade JD, Stewart M, Walters E. Impaired granulocyte adherence: a reversible defect in host defense in patients with poorly controlled diabetes. *Diabetes* 1978;27:677-81.
47. Eneroth M, Apelqvist J, Stenstrom A. Clinical characteristics and outcome in 223 diabetic patients with deep foot infections. *Foot Ankle Int* 1997;18(11):716-22.
48. Walmsley D, Wiles PG. Early loss of neurogenic inflammation in the human diabetic foot. *Clin Sci* 1991;80:605-10.
49. Reiber GE, Vileikyte L, Boyko EJ, del Aguila M, Smith DG, Lavery LA, et al. Causal pathways for incident lower-extremity ulcers in patients with diabetes from two settings. *Diabetes Care* 1999;22:157-62.
50. Myerson M, Papa J, Eaton K, Wilson K. The total contact cast for management of neuropathic, plantar ulceration of the foot. *J Bone Joint Surg Am* 1992;74(2):261-9.
51. Lavery LA, Vela SA, Lavery DC, Quebedeaux TL. Reducing dynamic foot pressures in high-risk diabetic subjects with foot ulcerations: a comparison of treatments. *Diabetes Care* 1996;19:818-21.
52. Verhelst R, Bruneau M, Nicolas AL, Frangi R, El Khoury G, Noirhomme P, et al. Popliteal-to-distal bypass grafts for limb salvage. *Ann Vasc Surg* 1997;11(5):505-9.
53. Jackson MR, Clagett GP. Antithrombotic therapy in peripheral arterial occlusive disease. *Chest* 1998;114(Suppl 5):666s-82s.
54. Louie TJ, Bartlett JG, Tally FP, Gorbach SL. Aerobic and anaerobic bacteria in diabetic foot ulcers. *Ann Intern Med* 1976;85(4):461-3.
55. Sapico FL, Witte JL, Canawati HN, Montgomerie JZ, Bessman AN. The infected foot of the diabetic patient: quantitative microbiology and analysis of clinical features. *Rev Infect Dis* 1984;6(Suppl 1):S171-6.
56. Flores-Rivera AR. Risk factors for amputation in diabetic patients; a case-control study. *Arch Med Res* 1998;29(2):179-84.
57. Lineaweaver W, Howard R, Soucy D, McMorris S, Freeman J, Crain C, et al. Topical antimicrobial toxicity. *Arch Surg* 1985;120:267-70.
58. Wright JB, Lam K, Burrell RE. Wound management in an era of increasing bacterial antibiotic resistance: a role for topical silver treatment. *Am J Infect Control* 1998;269(6):572-7.
59. Sundberg J, Meller R. A retrospective review of the use of cadexomer iodine in the treatment of chronic wounds. *Wounds* 1997;9(3):68-86.
60. Zamboni WA, Wong HP, Stephenson LL, Pfeiffer MA. Evaluation of hyperbaric oxygen for diabetic wounds: a prospective study. *Undersea Hyperb Med* 1997;24(3):175-9.
61. Faglia E, Favales F, Aldeghi A, Calia P, Quaranteillo A, Oriani G, et al. Adjunctive systemic hyperbaric oxygen therapy in treatment of severe prevalently ischemic diabetic foot ulcer. *Diabetes Care* 1996;19:1338-43.
62. American Diabetes Association. Consensus development conference on diabetic wound care. *Diabetes Care* 1999;22:1354-60.
63. Doxey DL, Ng MC, Dill RE, Iacopino AM. Platelet-derived growth factor in wounds of diabetic rats. *Life Sci* 1995;57:1111-23.
64. Loots MA, Lamme EN, Mekkes JR, Bos JD, Middlekoop E. Cultured fibroblasts from chronic diabetic wounds in the lower extremity (non-insulin-dependent diabetes mellitus) show disturbed proliferation. *Arch Dermatol Res* 1999;291(2-3):93-9.
65. Pollak RA, Edington H, Jensen JL, Kroeker RO, Gentzkow GD. A human dermal replacement for the treatment of diabetic foot ulcers. *Wounds* 1997;9(6):175-83.
66. Wieman TJ, Smiell JM, Su Y. Efficacy and safety of a topical gel formulation of recombinant human platelet-derived growth factor-BB (becaplermin) in patients with chronic neuropathic diabetic ulcers. *Diabetes Care* 1998;21:822-7.
67. Litzelman DK, Slemenda CW, Langfeld CD, Hays LM, Welch MA, Bild DE, et al. Reduction of lower extremity clinical abnormalities in patients with non-insulin-dependent diabetes mellitus. A randomized, controlled trial. *Ann Intern Med* 1993;119(1):36-41.
68. Uccioli L, Faglia E, Monticone G, Favales F, Durola L, Aldeghi A, et al. Manufactured shoes in the prevention of diabetic foot ulcers. *Diabetes Care* 1995;18:1376-8.
69. Chantelau E, Haage P. An audit of cushioned foot wear: a relation to patient compliance. *Diabet Med* 1994;11:114-6.