Clinical perspective article

Diabetic foot ulcers: A framework for prevention and care

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Complications secondary to diabetes, such as diabetic foot ulcers, continue to be a major worldwide health problem. At the same time, health care systems are changing rapidly, causing concern about the quality of patient care. While the ultimate effect of current changes on health care professionals and patient outcomes remain uncertain, measures commonly used to reduce costs, e.g., disease and multi discliplinary management strategies, have been shown to help prevent the occurrence of diabetic ulcers. In addition, ultilizing a multi discliplinary approach, the principles of off-loading and optimal wound care, the vast majority of diabetic foot ulcers can be expected to heal within 12 weeks of treatment. Education of primary care providers and patients is paramount. **(WOUND REP REG 1999;7:7–16)**

It is well known that diabetic foot ulceration is a significant end stage complication of diabetes with considerable economic and public health implications. In the United States 5–6% of the population has diabetes, and it is considered one of the most costly diseases.[1] In all populations, the prevalence and incidence of noninsulin-dependent diabetes mellitus (NIDDM) is higher than insulin-dependent diabetes mellitus (IDDM), and NIDDM is particularly common in developing countries.[2]

In 1987, diabetes or its complications accounted for 9 million days in the hospital, was found to be the sixth leading cause of death, and accounted for 50% of all nontraumatic lower-limb amputations in the United States.[3] Programs to reduce the number of major amputations are considered worthwhile because amputations cause significant morbidity and mortality. In addition, they are costly. In a 1988 US study, hospital costs alone averaged $25,000 per amputation.[4] Similarly, using 1990 cost data, a Swedish study showed that 82%, or SEK 282,080 (~ $34,000) of amputation costs were in-patient care charges.[5] By contrast, in the same study, the average total cost for primary healing of diabetic foot ulcers, e.g., in-patient care, antibiotics, out-patient visits, topical treatment and orthopedic appliances, was SEK 51,000 (~ $ 6000). Using 1992 data, others have reported that the average cost of diagnosing and treating a foot ulcer with growth factors in special wound care centers in the United States was somewhat higher ($16,602).[6] However, the cost of amputation in terms of dollars and morbidity remains still higher.

IDDM	Insulin-dependent diabetes mellitus
NIDDM	Non-IDDM

Screening and prevention

All health care providers need to become familiar with the natural history of diabetes related complications since many can progress to end stages relatively asymptomatically.[7] Relatively simple, and noninvasive preventative strategies, such as foot-care education and wearing appropriate shoes, do reduce the likelihood of developing serious foot lesions.[8] An increased awareness of the problem and screening efforts may not only help individual patients, it will also improve our understanding of the incidence and prevalence of foot ulcers. For example, it has been noted that precise data on the incidence and prevalence of foot ulcers in

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Table 1. Examining the at-risk patient

Examination to determine presence of risk factors	Assessments: basic and comprehensive	Risk factors
Patient & family history	History of generalized atherosclerosis, coronary artery disease, ulcers, surgery, leg or foot pain. Current and past medication (s) alcohol/ tobacco use, mobility, edema	Patients with diabetes and a history of problems with glucose control, nephropathy or retinopathy
General physical assessment	Height, weight, blood pressure, mobility, gait, foot shape, foot X-ray, foot pressure studies	High body mass index, high blood pressure, less-than-optimal gait, limited mobility, foot/ toe (Charcot) deformities, unable to reach feet, callus formation
Assessment of vascular status	Foot pulses, color and temperature of skin, presence of edema, ankle/brachial index*, noninvasive Doppler studies, transcutaneous oxygen measurement	Absence of foot pulses, pallor, cold feet, edema, diminished hair growth on extremities
Neurological assessment		
Motor function	Ankle reflexes, muscle strength, foot shape, electrophysiological tests	Reduced/absent ankle reflexes, weakness, muscle wasting, flattened arch and prominence of metatarsal heads
Autonomic function	Skin condition and temperature, aspect of foot veins, quantitative sweat test, thermography for skin temperature	Dry skin (reduced sweating), callus formation, warm foot, distended veins
Sensory function	Touch (Semmes-Weinstein filaments), temperature, vibration (128-HZ fork), thermal threshold testing, biothesiometry	Unable to perceive 10 *g* force (5.07 filament), vibration perception threshold ≥ 25 V, reduced thermal perception

*Ankle/brachial index (ABI): Obtain ankle pressure and divide by brachial pressure. In patients with non-compliant arterial walls, the ABI may be falsely elevated.

diabetic patients is remarkably scarce compared to our knowledge of retinopathy or nephropathy in this patient population.[9]

Unfortunately, most experts agree that the feet are an often neglected part of the physical assessment. Indeed, current guidelines for monitoring the quality of care of patients with diabetes recommends asking the patient if they have removed their shoes and socks at least once during a physician visit in the past year.[1] While 15–20% of patients with diabetes will develop a foot ulcer in their lifetime, fewer than 20% of diabetic patients are given regular foot examinations by their primary care physician at the time of an office visit. Specifically, the rate of foot examinations during a 1-year period in a physicians office has been found to range from 30 to 50%.[10–13] In addition, Mills et al. reported that 29% of patients with infections or gangrene were delayed in their referral for definitive care.[14] An underestimation of severity and lack of recognition of ischemia were cited as reasons for the delay. Depending on the healthcare environment, economic considerations may also play a role in referral patterns. For example, in the United States today, there is concern about the referral patterns of providers who are financially rewarded for not sending patients to specialists or specialty clinics.

Screening efforts should include all diabetic patients (see Table 1). In practice, this means that health care professionals in all patient care environments need to know what to look for and how to conduct a basic risk assessment. For example, health care providers who visit patients in the home are in an ideal position to assess mobility, foot care practices, glucose control, dietary habits, patient knowledge about foot care and ulcers, and to make timely referrals.[15] Reducing pressure, one of the direct causes of ulcer formation (Figure 1), remains the key to preventing and healing foot ulcers.

Callus formation

It has long been recognized that elevated plantar dynamic pressures, together with neuropathy, can lead to ulcer formation (Figure 1).[16] Recently, the focus has expanded to include the formation of calluses as a possible marker for ulceration, and careful examination of callus is an essential component of patient screening and assessment programs. Callus exacerbates already high plantar pressures in patients with neuropathy and functions as a predictor of future ulceration.[17] However, it is important to remember that the presence, or absence, of neuropathy is not related to callus formation, suggesting that other factors such as age and footwear may be responsible.[18]

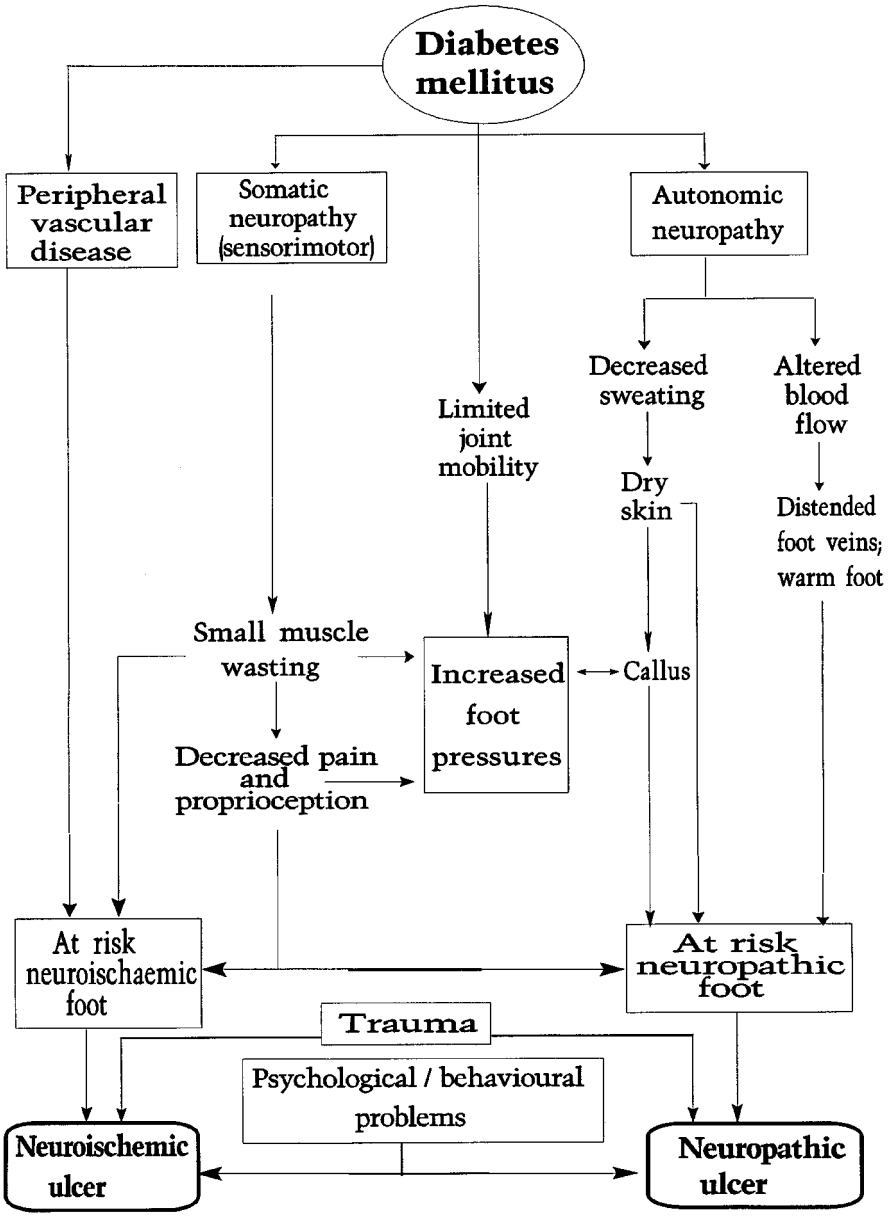

Figure 1. Pathways to neuropathic and neuroischemic foot ulceration in diabetic patients. Reprinted with permission from Ref. 23.

The normal process of keratinization is stimulated by the impact of excessive pressures on the plantar surface of the foot. Callus is considered the bodies "natural response" to the stress of the elevated pressure. The callus transmits further pressures into the subcutaneous tissue.

In addition to increasing pressure, this process leads to bruising and, eventually, extravasation of blood from the capillaries in the area. This may be secondary to a microangiopathy and occurs significantly more in diabetic than nondiabetic plantar calluses.[19] Removal of the callus tissue results in lowered plantar pressures and is an essential part of the treatment program of all diabetic foot patients.[20]

Neuropathy

The presence of somatic as well as autonomic neuropathy is a significant risk factor for the development of foot ulcers (Figure 1).[21] With respect to the development of foot ulcers, the effects of autonomic neuropathy are: decreased sweating (causing dry skin and a predisposition to callus formation) and alterations in blood flow. The latter results in distended dorsal foot veins and a warm, though insensitive, foot.[22] In addition to resulting in insensitivity, the presence of somatic neuropathy increases the risk of developing abnormal foot posture, weakness and wasting of the small muscles of the foot and subsequent excessive pressure at the metatarsal heads and the heel.[23] Early

detection of sensorimotor deficits and implementation of preventive strategies will help reduce the risk of developing foot ulcers. In patients with leprosy, early detection of neuropathy will help target educational, preventive and treatment efforts to identify nerve damage and prevent its devastating consequences.[24]

While some patients will seek medical care because their neuropathy is painful, many patients will develop chronic, painless, sensorimotor neuropathy and may not seek care until significant problems (e.g., an ulcer) have developed. For example, in a study of patients who underwent amputations at a US Veterans Administration Medical Center, 41% were found to be unaware of their sensory deficit.[25]

Reported prevalence rates of neuropathy in patients with Type I and II diabetes mellitus vary considerably (ranging from 15 to 47%), but all studies indicate that the prevalence of neuropathy increases with age and diabetes duration.[23] Similarly, in nondiabetic patients, increased age is associated with reduced peripheral nerve function and foot abnormalities.[26,27] While it is well known that the normal aging process is accompanied by a reduction in peripheral vibration sense and ankle reflexes, little is known about the prevalence of neuropathy or the risk of developing foot ulcers in the nondiabetic population. If the results of a recent study, in which 18% of nondiabetic elderly patients with foot problems had neuropathy,[27] are confirmed, the consequences for clinicians caring for an aging population are clear. A careful neurological examination of the feet of all at-risk and elderly patients should be performed on a regular basis. While diminishing reflexes, peripheral vibration sense and proprioception may reflect normal aging, a patient's risk of developing foot ulcers increases with every risk factor detected (see Table 1).

The appearance of the neuropathic foot, in its classic presentation, should help a clinician arrive at a diagnosis. The foot appears well nourished with a normal distribution of hair, a normal arch, healthy nails, and normal pulses. The skin tends to be dry. The intrinsic muscles of the feet may be atrophied which can result in clawing of the foot. The metatarsal heads can be prominent, especially when the fat pads are displaced. The sensory loss seen in diabetic neuropathy appears to be of greater importance as a predictor of subsequent ulceration than the presence of vascular disease.[28–30]

Vascularity

Problems with perfusion contribute to the development of ulcers and result in delayed healing. Regardless of the presence of vascular disease, exposure of tissues to prolonged pressure, shearing forces or friction will result in tissue anoxia and cell death. While the majority of ulcers on the bottom of the feet are seen in patients with diabetes mellitus, the deleterious effects of impaired perfusion in all patients, particularly over bony prominences such as the ankle or the heel, should not be overlooked. Limited mobility, a compromised overall health condition or nutritional status, and disease states that affect sensory perception (e.g., neuropathy) all increase the risk of developing pressure ulcers.[31]

Reduced tissue perfusion, secondary to macrovascular or microcirculatory disease, also contributes to the formation of diabetic foot ulcers and impairs healing of existing ulcers. The high prevalence of macrovascular disease in patients with Type I and Type II diabetes mellitus is related to the presence of virtually all major risk factors including: abnormalities in lipoprotein components, abnormal hemostatic properties of the blood and disorders within the arterial structures.[32] For example, in the Framingham study, the average adjusted incidence of claudication was 12.6/1000 for diabetic compared with 3.3/1000 for nondiabetic men, and 8.4/1000 for diabetic vs. 1.3/1000 for nondiabetic women.[33] In diabetic patients, large and small vessel diseases do not always progress at the same rate and it is not uncommon, for example, for small vessels in the toes to have evidence of ischemia while the dorsalis pedis or posterior tibial pulses are present and of adequate quality.[4] Hence, the "classical" ischemic ulcer is often seen on the toes whereas a combination of neuropathy and peripheral vascular disease will result in the more frequently observed foot ulcer in high pressure areas at the bottom of the feet.

The physical examination provides many clues to the diagnosis of ischemia in the diabetic patient. The skin is shiny and atrophic, the pulses are weak or absent, and fissures are common on the heels. Nails are thickened and overgrown with dry scales. Small microabcesses, which may occur behind or alongside the nails and in fissures, can be very painful to walk on. Small, punctate dermal ulcerations, with a flat, dry, necrotic center, may be present on the toes. The immediate periwound tissue can appear darkly erythematous due to maximal capillary vasodilation, giving the appearance of a crimson corona. Small infections can lead to lymphangitic streaking or spreading cellulitis which should be considered an emergency in the diabetic patient. Occasionally the infection leads to a local digital vessel thrombosis and, subsequently,

a black toe. A more sudden proximal vascular occlusion can lead to wet gangrene. In this situation there is inadequate time for formation of collateral circulation and the tissue changes from pallor to rubor with a blister forming over the classic blue-black moist tissue. Frequently, this macerated, necrotic tissue is infected with *Pseudomonas aeruginosa*, which gives the tissue a classic "fruity" odor. When performing a Doppler exam (Table 1), it is important to remember the potential for false elevation of the ankle pressures secondary to medial calcification of the vessels. For this reason, it is probably safer to measure transmetatarsal or toe pressures in the diabetic patient. It is also useful to assess qualitatively the Doppler signal by obtaining a printout or sonogram. The absence of a bi- or triphasic signal (i.e., presence of a monophasic signal) identifies a proximal stenosis in the macrovascular tree, and warrants further assessment.

Recently, there has been a renewed interest in transcutaneous pO_2 monitoring, a useful technique in the management of the diabetic foot.[34] The probe should be placed on the dorsum of the foot and measurements should be compared to those obtained on the chest wall. Values over 30 mm Hg indicate adequate tissue perfusion for healing in diabetes and are a reflection of the patency of the microcirculation.[35]

Assessment

At the initial visit, a complete medical, surgical, social, medication and family history has to be obtained. In addition, a detailed diabetic history (onset, glucose control, medications used, weight changes) is of the utmost importance. Review factors for cardiovascular disease and look for evidence of an underlying "triopathy" (nephropathy, retinopathy, neuropathy).[36] A complete physical examination, including an assessment of the patient"s vascular status, sensory, motor and autonomic nerve function, is undertaken (Table 1). Shoes are evaluated for signs of abnormal, excessive, or irregular patterns of wear, and the feet are examined to look for signs of impaired perfusion, infection, neuropathy and the presence of callus as described earlier.

To conduct a basic examination, most clinicians only have to add a set of Semmes-Weinstein monofilaments (Gillis W. Long, Hansen's Disease Center, Carville, LA) to their standard array of tools. While sensory function can be assessed using standard equipment, most experts recommend including use of these filaments because they have been tested and provide quantitative data.[37,38] Specifically, it has been found that most patients with neuropathy can not feel the 5.07 probe (10 *g*). Because prolonged (15–30 s) application of the filament to the area increases cutaneous pressure threshold, it is recommended that the filament be applied in a consistent manner for approximately 5 seconds.[39] For rapid screening purposes, loss of temperature sensation can be assessed using a cold tuning fork, while pin-prick and two-point discrimination can also be examined using standard (blunt) instruments.[38]

Wound assessment

At this time, the Wagner system is still the most widely used and evaluated method for classifying foot ulcers (Table 2). While the treatment of Grade 0 conditions consists of preventative efforts (see above), including these lesions in an ulcer grading classification system is helpful because it serves as a reminder that these patients are at high risk for developing wounds. As with all wound staging and classification systems, their major benefit lies in standardizing the terminology used to describe them, and their major disadvantage is that they are primarily based on wound depth and appearance.[40,41] The diabetic wound classification system developed at the University of Texas Health Science Center consists of three grades and 4 categories (based on depth, the presence of infection and/or ischemia).[41,42] In addition to assessing wound depth, the wound bed needs to be assessed for the presence of devitalized tissue, granulation tissue, exudate and odor. Furthermore, it is important to document the condition of the wound edges (undermining, callus, maceration) and to measure the size of the wound. While there is no optimal method for measuring wound size serial measurements help

Table 2. Wagner classification for grading foot ulcers*

Grade	Characteristics
0	Intact skin (preulcerative lesion Healed ulcers Presence of bony deformity
1	Superficial ulcer without subcutaneous tissue involvement
2	Penetration through the subcutaneous tissue. May expose bone, tendon, ligament or joint capsule
3	Osteitis, abscess, or osteomyelitis
4	Gangrene of digit
5	Gangrene of the foot requiring disarticulation

*Wagner, FW. The dysvascular foot: A system for diagnosis and treatment. Foot Ankle 1981;2:62-122.

clinicians assess the effect of care by quantifying changes in the wound area over time.

While the "classical signs" of infection (swelling, redness, pain, odor) are useful for diagnosing infection in acute wounds, they may, or may not be helpful when attempting to diagnose infection in chronic wounds.[40] For example, repeated injury will cause inflammation (redness), pain may, or may not, be present, most chronic wounds are contaminated with a variety of bacteria and many emit an odor. In the diabetic foot ulcer, hematological and bacterial indices of infection may also be misleading.[22] When attempting to diagnose infection in chronic wounds, experience is the best teacher. An unexplained delay in the healing process, the presence of a purulent discharge, crepitation from gas forming organisms or deep sinuses are often indicative of an infection.[22] Specifically, it has been shown that if the ulcer extends down to bone, osteomyelitis and/or joint infection may be present.[43] Following debridement, all grade 1, 2, and 3 wounds should be examined with a sterile probe to determine the presence of an abscess, sinus tracts or exposed bone. X-rays and deep cultures are useful, but, if osteomyelitis is suspected, a bone scan is needed. The topic of infection will be discussed in the next section on treatment.

Treatment: The multidisciplinary approach

Treatment of the diabetic foot still requires the multidisciplinary approach discussed by Edmonds et al. in 1986, even though advances in wound care and technology have resulted in some modifications of the protocols.[44] Multidisciplinary approaches to wound and foot ulcer care have been successfully implemented in different countries with varying health care delivery systems.[5,6,45–47] The services of an orthotic or prosthetic specialist are particularly helpful for providing off-loading devices or specialty shoes that are comfortable and easy to use, thereby increasing patient compliance. With respect to the latter, education is the key to preventing and healing these wounds. Brochures and hand-outs are very useful for reinforcing patient teaching, providing they are easy to read. Poor reading skills are very common in industrialized and nonindustrialized countries.[48] Hence, brochures may increase the value, but can not replace, verbal communication and instructions.

Treatment: Wound care

Regardless of ulcer depth, measures to reduce pressure are needed. Indeed, it has been argued that deep foot ulcers are superficial ulcers that have continued to be walked on.[49] While other measures, e.g., tight glucose control, may be helpful, re-injury of the wound as a result of unrelieved pressure will certainly impair healing and may increase the risk of complications. Some experts advocate the use of extra depth or custom made shoes, insoles, or specially designed padded socks for this purpose. An impression of the foot can be made to determine where pressure relief needs to be obtained. The total contact casting technique, with a rocker on the bottom of the foot, has also been successfully used for many years.[50,51]

Grade 4 and 5 ulcers (Table 2) often require amputation. However, in the case of grade 5 ulcers the extent of amputation may be reduced by preamputation arterial surgery whereas angioplasty and proximal reconstructive surgery may eliminate the need to amputate part of the foot when a grade 4 ulcer is present.[49] Prompt treatment of the infection is always needed. Fortunately, grade 4 and 5 ulcers are not as common as grade 1, 2 and 3 ulcers. Prompt and appropriate care of the latter may prevent the dreaded complications of the former.

The basic principles of wound care should be applied to all wounds. Because devitalized tissue in a wound may delay healing, predispose it to infection and hinder assessment, it needs to be removed. Removal of devitalized tissue and callus is often accomplished by sharp or surgical debridement. It is the most rapid method and most commonly used debridement technique for diabetic foot ulcers. Sharp debridement is particularly useful for removing black, dried eschar and callus. When limited to removing dried, black eschar only, sharp debridement can usually be performed at the bed-side or in the out-patient setting. Debridement has been shown to improve outcomes independent of topical treatment, in a growth factor study treating diabetic foot ulcers.[52] However, there are risks associated with surgical debridement and it may not always be the treatment of choice for removing yellow, fibrinous slough. When potential complications (e.g., bleeding) are a concern, when anesthesia is needed and not available, or when there are no adequate facilities to perform aggressive surgical debridement, less invasive (and slower) debridement methods such as using wet-to-dry dressings, enzymes or moisture retentive dressings should be considered. Alternatively, a combination of debridement methods such as sharp debridement of dry necrotic tissue followed by using moisture retentive dressings to facilitate autolytic debridement of sloughy necrotic tissue, may be very helpful and effective.[53] The potential role of wound cleansing tech-

niques to help dislodge devitalized tissue should also not be underestimated. Wound cleansers or saline, applied with adequate pressure will help the debridement process.

Bacteria are present in all chronic wounds and diabetic foot ulcers are no exception.[54] There is a natural balance between the quantity of bacteria present (bioburden) and the host's immune status. If an inoculum of bacteria is $> 10^5$ organisms/gram of tissue or the host suffers from a decrease in immune function, clinical infection occurs.[55] Although the gold standard for wound cultures remains the quantitative biopsy,[56] many facilities do not provide the service. Studies have shown that antibiotics are not universally effective in chronic or acute wounds and specifically in uncomplicated diabetic neuropathic forefoot ulcers.[57–59] When an infection is present, debridement of all devitalized tissue should be performed immediately and antibiotic treatment initiated. While many different antibiotics are commonly used, it is important to remember that Gram-negative as well as Gram-positive organisms are often present in these wounds. Serious infections require hospitalization and use of parenteral antibiotics so as to achieve higher concentrations of antibiotics in the peripheral tissues.[4] Oral antibiotics and out-patient management may be successful, but patients have to be assessed frequently and instructed to call immediately when signs and symptoms of worsening of the infection develop, or when their blood sugar levels begin to rise. The indiscriminate, nonculture directed use of antibiotics has led to the tremendous problem of antibiotic resistance.[60–63] The use of sound clinical judgment coupled with appropriate culture information is critical when caring for the patient with a diabetic foot ulcer.

Dry cells are dead cells. Hence, following debridement, tissues should be kept moist so as to prevent the formation of devitalized tissue and subsequent deepening of the wound. Some modern dressings not only protect the wound against dehydration, but they also prevent contamination and provide an environment which facilitates healing by retaining cells needed to phagocytose bacteria and facilitate repair. For example, studies have shown that viable polymorphonuclear leukocytes, macrophages, lymphocytes and monocytes as well as platelet-derived growth factor, fibroblast growth factor and epidermal growth factor can be found in the fluid of wounds covered with a moisture-retentive hydrocolloid dressing.[64] In addition, some of these dressings have been shown to provide a barrier against environmental contamination as well as bacteria and some viruses.[65]

These findings may, in part, explain why wounds dressed with a moisture-retentive dressings heal more expediently and are less likely to become infected than wounds dressed with traditional gauze-type dressings. For example, the reported infection rate of 3047 wounds of varying etiologies dressed with traditional gauze type dressings was 7.1% compared to 2.6% for wounds dressed with moisture-retentive dressings.[66] While few studies on the treatment of diabetic foot ulcers report infection rates, indeed, most are limited to reporting "worsening" of the wound, the trend does seem to hold for these wounds. Laing reports that, in his clinical practice, 2% of diabetic foot ulcers become infected when using a hydrocolloid dressing and total contact casting.[50] Similarly, in the Manchester diabetic clinic, a retrospective study of clinical outcomes showed that 2.5% of ulcers treated with the hydrocolloid dressing became infected compared to 6% of ulcers managed with traditional gauze-type dressings.[67] This difference was statistically significant.

In addition to the above mentioned treatments there are several therapies that have arrived on the scene as treatment options. The use of growth factors in wound care, and specifically for the diabetic foot ulcer, where over 900 patients have already been studied, has opened up a new treatment option for the recalcitrant diabetic foot ulcer patient.[68,69] Two hundred 81 patients at 20 centers were studied to evaluate the effectiveness of a human dermal replacement dermis.[70] In this study, at 12 weeks, 50.8% of treated patients healed compared to 31.7% of controls for a p-value of 0.006. Hyperbaric oxygen therapy has been shown useful for diabetic foot ulcers and refractory osteomyelitis.[71] A novel recent treatment option includes the use of a foam dressing with the application of subatmospheric pressure.[72] Orthopedic bone resections and flap coverage offers still another option when treating the diabetic foot ulcer.[73,74]

Wound care outcomes

Using the principles of wound care, the majority of Grade 1, 2 and 3 diabetic foot ulcers will heal within a reasonable period of time. When evaluating the time to healing of diabetic foot ulcers treated in a hospital based wound clinic, we found that the vast majority of wounds (88.1%; $n = 84$) healed (Figure 2). There was no significant difference between the healing rate for NIDDM (84.6%; $n = 26$) and IDDM (89.7%; $n = 58$) patients. This data includes 45 patients with 84

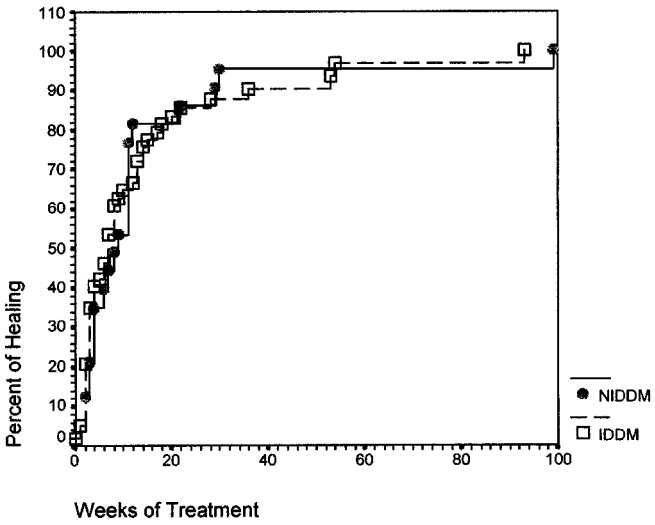

Figure 2. Kaplan-Meier time until healing curves of foot ulcers in insulin dependent (IDDM) and noninsulin (NIDDM) dependent patients. IDDM: Mean 14 weeks, median 7 weeks; NIDDM: Mean 14 weeks, median 9 weeks.

wounds treated prospectively over a 2 year course. Our findings are very similar to the clinic follow-up data reported by others using the multidiscliplinary team and standard wound care approach in different countries.[44,50,75] Also, in one of the studies, a direct comparison between clinics in two different countries (United States and Italy), it was found that, despite the differences in the two systems, similar success rates were achieved.[75] These results are particularly encouraging in light of the fact that they were not obtained under a protocol with exclusion criteria and enrollment limitations. As a result, they reflect clinical practice and may help clinicians understand what to expect. We hope that by incorporating all of the new technologies limb salvage rates and wound healing rates will continue to improve.

Wound care: The future

As we approach the 21st century, one thing is certain: no health care system in the world is stable. In many countries, concerns about health care cost and access have made it to the top of the political agenda.[76] While the ultimate effect of these changes on health care professionals remain uncertain, it seems likely that current trends toward individual and community-based health care and attempts to cut costs is not going to reverse itself.[77] With respect to wound care, attempts may be made to lower costs by purchasing inexpensive wound care products, by purchasing products "designed" to reduce caregiver time or by lowering the requirements of caregiver level of training and related salary.[78]

Fortunately, measures commonly employed to reduce costs, such as disease and multidisciplinary management strategies, consumer education and standards of care, are not new to providers in wound and diabetic clinics. On the contrary, they are the reason for their success and consistent results. Many studies have shown that prevention and multidisciplinary care programs reduce both the incidence and major complications of chronic wounds. The individual and collective price of providing less than optimal care in many of these patients, amputation, is simply too high.

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