Foot ulcers in the diabetic patient, prevention and treatment

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Abstract: Lower extremity complications in persons with diabetes have become an increasingly significant public health concern in both the developed and developing world. These complications, beginning with neuropathy and subsequent diabetic foot wounds frequently lead to infection and lower extremity amputation even in the absence of critical limb ischemia. In order to diminish the detrimental consequences associated with diabetic foot ulcers, a common-sense-based treatment approach must be implemented. Many of the etiological factors contributing to the formation of diabetic foot ulceration may be identified using simple, inexpensive equipment in a clinical setting. Prevention of diabetic foot ulcers can be accomplished in a primary care setting with a brief history and screening for loss of protective sensation via the Semmes-Weinstein monofilament. Specialist clinics may quantify neuropathy, plantar foot pressure, and assess vascular status with Doppler ultrasound and ankle-brachial blood pressure indices. These measurements, in conjunction with other findings from the history and physical examination, may enable clinicians to stratify patients based on risk and help determine the type of intervention. Other effective clinical interventions may include patient education, optimizing glycemic control, smoking cessation, and diligent foot care. Recent technological advanced combined with better understanding of the wound healing process have resulted in a myriad of advanced wound healing modalities in the treatment of diabetic foot ulcers. However, it is imperative to remember the fundamental basics in the healing of diabetic foot ulcers: adequate perfusion, debridement, infection control, and pressure mitigation. Early recognition of the etiological factors along with prompt management of diabetic foot ulcers is essential for successful outcome.

Keywords: diabetes, ulcer, prevention, infection, amputation

Introduction

The rapid rise in the incidence of diabetes, a serious life-long condition, is of alarming concern to health care professionals. Recent data from the Center of Disease Control and Prevention approximate that 20.8 million people, roughly 7% of the United States population, have diabetes (2005). In 2005 alone, 1.5 million new cases of diabetes were diagnosed in people aged 20 years or older (2005). Diabetes mellitus is a disease known for its multifaceted complications, and foot ulceration, which often results in lower extremity amputations, is one of the most common complications associated with the disease (Boulton and Vileikyte 2000; Reiber 2001; Dang and Boulton 2003; Pinzur et al 2005). The prevalence of foot ulcers ranges from 4% to 10% among persons diagnosed with diabetes mellitus (Singh et al 2005). This translates to an annual population-based incidence of 1.0% to 4.1%, and the lifetime incidence may be as high as 25% (Singh et al 2005). Diabetic foot ulcers frequently become infected and are a major cause of hospital admissions (Dang and Boulton 2003; Pinzur et al 2005). They also account

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for more than half of non-traumatic lower limb amputations in this patient population (Dang and Boulton 2003). Diabetic foot ulcers impose tremendous medical and financial burden on our healthcare system with costs conservatively estimated as high as $45000 per patient (Stockl et al 2004). These estimations, however, do not include the deleterious psychosocial effects on the patient's quality of life because of impaired mobility and substantial loss of productivity (Ragnarson Tennvall and Apelqvist 2004). Ulcerations are pivotal events in limb loss for two important reasons. They allow an avenue for infection (Armstrong and Lipsky 2004), and can cause progressive tissue necrosis and poor wound healing in the presence of critical ischemia. Approximately 56% of diabetic foot ulcerations become infected (Block 1981; Gibbons and Eliopoulos 1984; Smith et al 1987) and 20% of these patients with infected foot wounds end up with some type of lower extremity amputation. Therefore the timely prevention and healing of diabetic ulcerations are fundamental for amputation prevention (Schwegler et al 2002; Wu et al 2005). This manuscript will focus on the prevention and treatment of diabetic foot ulcerations.

Prevention

Prevention and prophylactic foot care have been advocated to decrease patient morbidity, the utilization of expensive resources, as well as the risk for amputations (Pinzur et al 2005). These interventions, which include the identification of risk factors, patient education, and intensive podiatric care (Moreland et al 2004; Singh et al 2005), have been shown to be cost effective or even cost saving (Ragnarson Tennvall and Apelqvist 2004). Increased awareness of the potential influence of reimbursement systems on prevention, management, and outcomes of diabetic foot lesions has improved in recent years (Ragnarson Tennvall and Apelqvist 2004).

Diabetes is a multifactoral disease and the multidisciplinary approach has been advocated for the comprehensive treatment of diabetes and prevention of its complications (Ronnemaa et al 1997; Plank et al 2003). Patients with diabetes who present for care should be under the concomitant management of a primary care physician with appropriate referrals to an endocrinologist, ophthalmologist, nephrologists, vascular surgeon, podiatrist, physical therapist, nutritionist, and a diabetic educator to help ensure adequate care (Dang and Boulton 2003; Schaper et al 2003; Singh et al 2005; Van Damme and Limet 2005). These different perspectives and approaches were the basis for the American Diabetes Association Position Statements and the International Consensus on the Diabetic Foot, resulting in a worldwide network of professionals involved in the management of diabetic patients with foot problems (Schaper et al 2003). Applying evidence-based multidisciplinary treatment has been shown to result in a 50% reduction of major lower-limb amputation in this high risk group (Van Damme and Limet 2005).

Lavery et al implemented a lower extremity disease management program consisting of screening and treatment protocols for the diabetic foot in a managed care organization and noted its effectiveness to reduce hospitalizations and amputations (Lavery et al 2005). Based on the presence or absence of diabetic neuropathy, peripheral vascular disease, foot deformities and pressures, as well as the history of lower extremity pathology, the authors stratified patients into low and high-risk groups, and implemented preventive or acute care protocols (Lavery et al 2005). They noted a 47.4% decrease in the incidence of amputations from 12.89 per 1000 diabetics per year to 6.18 ($p < 0.05$), and a 37.8% decrease in foot related hospital admissions, from 22.86 per 1000 members per year to 14.23 (37.8%), after implementation of the disease management program (Lavery et al 2005). They further noted a 21.7% reduction in the average patient length of stay from 4.75 to 3.72 days ($p < 0.05$), a 69.8% reduction in the number of skilled nursing facility admissions per 1000 members per year, and a 38.2% reduction in the average length of stay in a skilled nursing facility 8.72 to 6.52 days ($p < 0.05$) (Lavery et al 2005).

Singh et al conducted a literature review of the efficacy of various diabetic foot ulcer prevention methods in the primary care setting from articles published between January 1980 and April 2004 available through EBSCO, MEDLINE, the National Guideline Clearinghouse databases, Cochrane Library, and relevant Web sites (Singh et al 2005). The authors noted substantial evidence to support screening of all patients with diabetes to identify those at risk for foot ulceration (Singh et al 2005). The authors further noted that patients may benefit from certain prophylactic interventions, including patient education, prescription footwear, intensive podiatric care, and evaluation for surgical interventions (Singh et al 2005). However, the patient and their health care professional must be fully informed of their problems, understand the management process, and be willing to make the necessary lifestyle changes to minimize complications (Helfand 2003).

A thorough history and physical is fundamental to identify risk factors for the development of diabetic foot ulcers. This includes an assessment of loss of protective sensation, foot structure, limited joint mobility, vascular status, and a history of previous foot ulceration, amputation or Charcot neuroarthropathy (Lavery et al 1998; Mayfield et al 2003).

History

Known factors for foot ulcerations include a current ulcer, past history of previous ulceration, prior lower extremity amputation, or the presence of neuropathic fractures (Lavery et al 1998; Boyko et al 1999; Abbott et al 2002), which increase the risk for further ulceration, infection and subsequent amputation (Goldner 1960; Pecoraro zet al 1990; Lavery et al 1998). Within one year of wound healing following ulceration, up to 60% of patients with a positive ulceration history will develop another because the skin plantar to that site may be less resilient and less well fortified to accept repetitive stress and therefore more prone to subsequent breakdown (Helm et al 1991; Uccioli et al 1995). This population segment has the highest risk of developing subsequent foot ulceration (Lavery et al 1998; Peters and Lavery 2001) and is the easiest risk group to identify. This patient population is also the group most in need of frequent foot assessment, intensive education, therapeutic shoes, padded stockings and rigorous blood glucose control.

Foot exam

Annual foot examinations are recommended for all individuals with diabetes to identify high-risk foot conditions including peripheral vascular insufficiency, structural foot deformities, and loss of protective sensation for which specific interventions have been shown to be effective in reducing amputation risk (Mayfield et al 2000).

Vascular exam

Peripheral arterial disease (PAD) is twice as common in persons with diabetes as in persons without (Gregg et al 2004) and is also a major risk factor for lower extremity amputation (2003). However, PAD is not the most common cause of foot ulceration, and is a component factor in only about a quarter of all cases (Edmonds 1987; Thompson et al 1991). In a recent two-center study PAD partially contributed to 30% of all foot ulcers (Reiber et al 1999). Vascular assessment should include palpation of all lower extremity pulses, including femoral, popliteal, posterior tibial, and dorsalis pedis pulses. The palpation of pulses is a learned skill with a high degree of inter-observer variability and high false positive and false negative rates (2003). The dorsalis pedis pulse has been reported to be absent in 8.1% of healthy individuals and the posterior tibial pulse is absent in 2.0% (2003). Although the absence of both pedal pulses when assessed by a person experienced in this technique, strongly suggests the presence of vascular disease, the presence of palpable pulses cannot exclude peripheral vascular disease. Ankle brachial pressure index (ABPI) (Figure 1), in contrast to the variability of pulse assessment and the often nonspecific nature of information obtained via history, is an easily reproducible and reasonably

Figure 1 Obtaining ankle brachial pressure index.

accurate method of diagnosing vascular insufficiency in the lower limbs (2003). However, a normal ABPI may be deceiving, as medial arterial calcification of the foot vessels results in hardening of the arteries thereby falsely elevating the ankle brachial pressure index (Brooks et al 2001; Bonham 2006). Parameswaran et al (2005) assessed doppler waveform of lower extremity arteries, ABI and pulse oximetry in 57 consecutive patients with type 2 diabetes and no symptoms of PAD and found that 31% of the patients had PAD in the lower extremity (Parameswaran et al 2005). The authors noted that ABPI only had a sensitivity of 63% (95% CI, 46%–77%) and a specificity of 97% (95% CI, 91%–99%) (Parameswaran et al 2005).

In addition, clinical evidence of dependent rubor, pallor on elevation, absence of hair growth, dystrophic toenails, and cool, dry, fissured skin should also be noted as they may be concomitant signs of vascular insufficiency (2003).

Due to the high estimated prevalence of PAD in patients with diabetes, the ADA consensus statement issued the following recommendations (2003).

A screening ABPI be performed in all diabetic patients >50 years of age; if the results are normal, the test should be repeated every 5 years.

A screening ABPI should be considered in patients with diabetes <50 years of age who have other peripheral arterial disease risk factors. These risk factors include smoking, hypertension, hyperlipidemia, or duration of diabetes >10 years.

A diagnostic ABPI should be performed in any patient with symptoms of PAD.

Protective sensation

Protective sensation, a level of sensory loss that allows patients to hurt themselves without recognizing injury, is a major component of nearly all diabetic ulcerations (Reiber et al 1999). The consequent vulnerability to physical and thermal trauma increases the risk of foot ulceration sevenfold (Singh et al 2005). All patients with diabetes should be screened for loss of protective sensation to identify those at risk for foot ulceration (Olaleye et al 2001; Singh et al 2005). The absence of protective sensation may be determined using simple, non-invasive instruments such as a 128 Hz tuning fork, a Semmes-Weinstein 5.07/10 gram monofilament nylon wire, a calibrated vibration perception threshold (VPT) meter, or by a comprehensive physical examination (Abbott et al 2002).

The conventional 128 Hz tuning fork test is an easy and inexpensive tool to assess vibratory sensation. The tuning fork is held over bony prominences such as the first metatarsal head and the lateral malleolus (Figure 2), and the test is considered positive when the patient is unable to perceive any vibration that the examiner can perceive (Singh et al 2005). The 5.07/10 g Semmes Weinstein monofilament consists of a plastic handle supporting a nylon filament (Figure 3) and is one of the most frequently utilized screening tools to identify loss of protective sensation in the United States (Armstrong 2000; Singh et al 2005). Testing via the Semmes Weinstein monofilament is administrated with the patient sitting supine in the examination chair with both feet level. The monofilament is applied perpendicular to the skin until it bends or buckles from the pressure, left in place for approximately one second and then released (Singh et al 2005). The patient with his or her eyes closed responds "yes" each time he or she perceives the application of the monofilament.

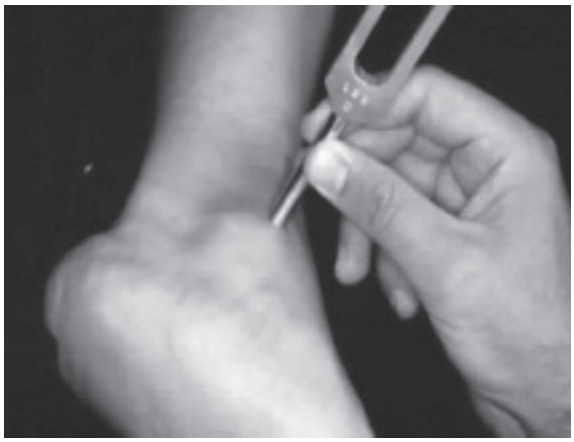

Figure 2 Use of 128 Hz tuning fork.

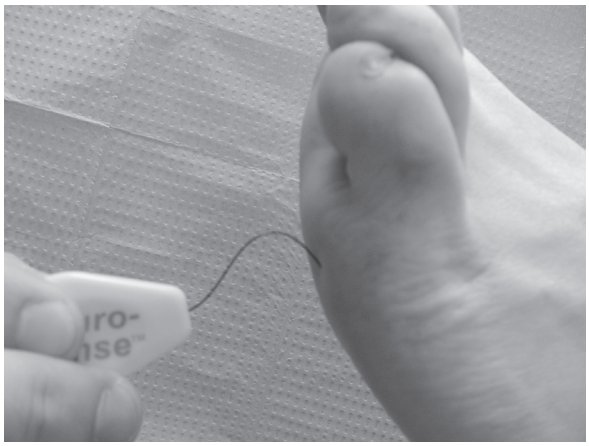

Figure 3 Semmes Weinstein monofilament. The monofilament is applied perpendicular to the skin until it bends or buckles from the pressure, left in place for approximately one second and then released.

The patient's inability to perceive 10 g of force applied by the 5.07 monofilament is clinically significant for large-fiber neuropathy. Although vibratory testing has demonstrated greater sensitivity (Sorman and Edwall 2002), the Semmes Weinstein monofilament test is sensitive enough to identify patients with the highest risk of foot complications (Gin et al 2002; Sorman and Edwall 2002).

The VPT meter, also known as Biothesiometer or Neurothesiometer, is a hand-held device with a rubber tactor that vibrates at 100 Hz. The hand-held device is connected to a base unit displaying a linear scale of applied voltage, ranging from 0 to 100 v (Armstrong 1999; Pham et al 2000). The device is generally held with the rubber tactor balanced vertically on the pulp of the big toe. The voltage is increased until the patient perceives a vibration. A mean of three readings measured in Volts is generally used to determine the vibration perception threshold for each foot. In a prospective four year study, a vibration perception threshold greater than 25 v had a sensitivity of 83%, a specificity of 63%, a positive likelihood ratio of 2.2, and a negative likelihood ratio of 0.27 for predicting foot ulceration (Young et al 1994; Mason et al 1999). A comparison of the screening methods is illustrated in Table 1.

Foot deformities and biomechanics

Foot deformities and limited joint mobility impose excessive pressure on the plantar aspect of the foot. This limitation in joint mobility is secondary to non-enzymatic glycosylation of periarticular soft tissues and reduces the foot's ability to accommodate for ambulatory ground reactive force to increase plantar pressure (Fernando et al 1991; Birke et al 1995; Lavery et al 1995; Frykberg et al 1998; Armstrong et al 1999; Van Damme and Limet 2005). This excessive pressure combined with the repetitive or constant stress from daily ambulation along with neuropathy will ultimately lead to failure of the protective integument and ulceration (Figure 4). Although the precise pathophysiological mechanisms underlying the development of diabetic foot ulcerations are complex (Van Damme and Limet 2005), it is generally associated with the presence of peripheral neuropathy and repetitive trauma due to normal walking activities which exposes the foot to moderate or high pressure and shear forces (Brand 1991; Cavanagh et al 2005; Wu et al 2005). Brand (1983) theorized that local inflammatory response, focal tissue ischemia, tissue destruction, and ulceration may occur when these types of forces are applied to a specific area over an extended period of time. Ulceration sites correlate with the highest plantar pressure points (Duckworth et al 1982; Boulton 1987; Birke et al 1991; Cavanagh et al 1996; Armstrong et al 1998). Foot deformities, limited joint mobility, partial foot amputations and other structural deformities often predispose diabetic persons with peripheral neuropathy to abnormal weight bearing areas of concentrated pressure that significantly increase their risk of ulceration (Lavery et al 1995; Boulton 1996; Lavery et al 1996). In one study of patients with peripheral neuropathy, 28% with high plantar pressure developed a foot ulcer during a 2.5 year follow-up compared with none with normal pressure (Veves et al 1992).

Lavery et al reported that patients with neuropathy but no deformity or history of ulcer or amputation are at 1.7 times greater risk for ulceration compared with patients without neuropathy (Lavery et al 1998). Neuropathy with concomitant

Table 1 Comparison of screening methods to help identify persons with diabetes at increased risk for foot ulceration

	Tuning fork	Monofilament	Biothesiometer
No. and type of studies	1 case control study (Coppini et al 1998); 1 prospective cohort study (Boyko et al 1999)	3 prospective cohort studies (Rith-Najarian et al 1992; Boyko et al 1999; Pham et al 2000)	2 prospective cohort studies (Young et al 1994; Pham et al 1998)
Criteria for positive screening test result	Patient loses vibration while examiner still perceives it	≥1 insensate site	Vibration perception threshold >25 v
Sensitivity, %	55–61	66–91	83–86
Specificity, %	59–72	34–86	57–63
Predictive Value %			
Positive	16*	18–39	20–32
Negative	93*87	94–95	95–97
Likelihood ratio			
Positive	1.5–2.0	1.4–4.7	2.0–2.2
Negative	0.63-0.66	0.3–0.5	0.3

Note: *Data not available in case-control study to calculate a positive and a negative predictive value.

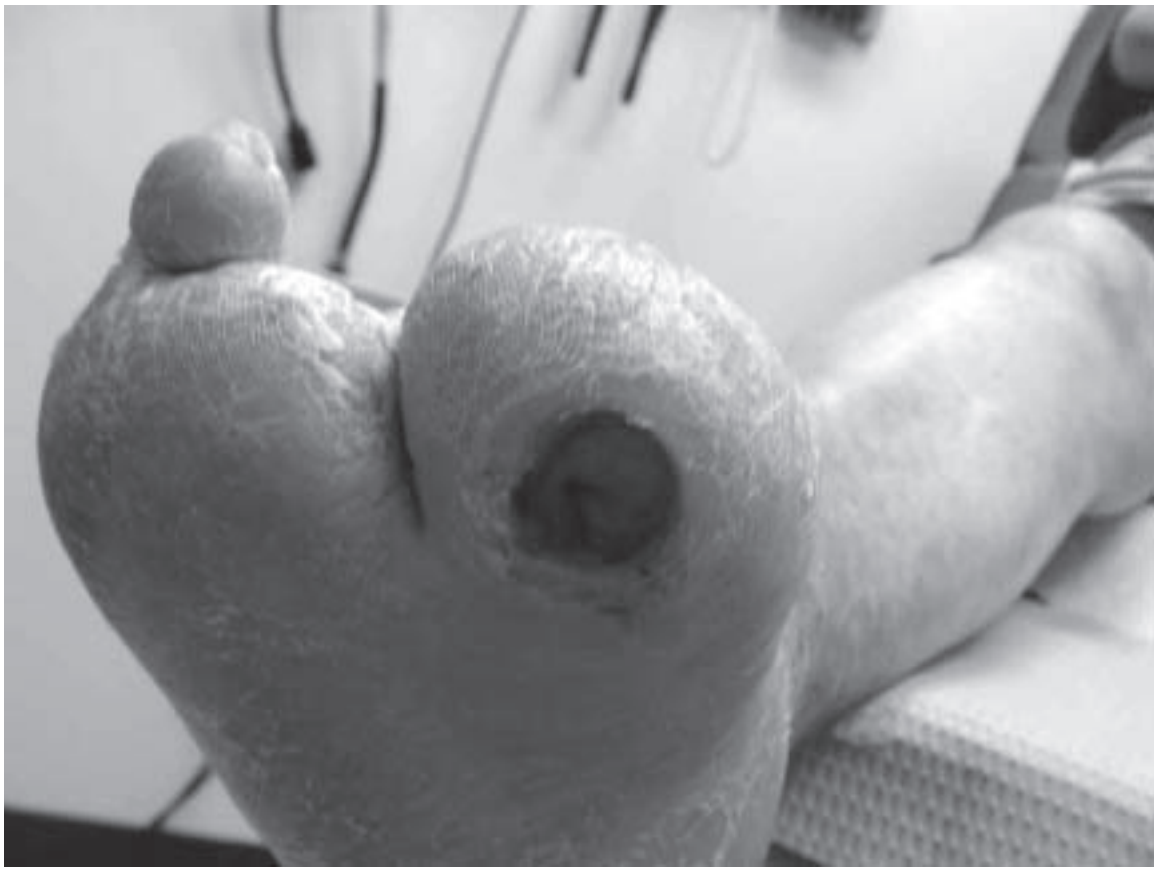

Figure 4 Neuropathic foot ulceration secondary to excessive pressure (from foot deformity) in combination with the repetitive stress from daily ambulation.

deformity or limited joint mobility yields a 12.1 times greater risk, and patients with a history of previous ulceration or amputation have a 36.4 times greater risk for presenting with another ulcer. Additional studies by Peters and Lavery, and Mayfield and co-workers (2001) by and large support these findings (Mayfield et al 1996; Peters and Lavery 2001).

Other contributing factors

Clinicians should also examine the patient for other contributing risk factors. Cutaneous manifestations associated with diabetes such as dry or fissured skin, calluses, tinea, or onychomycosis should all be noted.

Patient education

Educating patients at risk for diabetic foot ulceration have been shown to be beneficial (Malone et al 1989; Litzelman et al 1993; Singh et al 2005). Malone et al (1989) assessed the effectiveness of diabetic foot education by randomizing 103 patients (203 limbs) to receiving an hour foot care education and 100 patients (193 limbs) to receiving an hour of general diabetes mellitus education for 24 months total. The authors noted a lower incidence of foot ulcers in the group that received an hour of foot care education (4.5% vs 14.7%; RR, 0.31 [95% CI, 0.14 to 0.66]; ARR, –0.10 [95% CI, –0.16 to –0.04]; p = .002) (Malone et al 1989). Litzelman and co-workers (1993) conducted sessions on foot care, telephone reminders, and postcard reminders in 191 patients and gave standard care to 205 patients for a period of 12 months and noted fewer serious foot lesions in the group that received sessions on foot care and telephone/postcard reminders (OR, 0.41 [95% CI, 0.16–1.00]; p =.05) (Litzelman et al 1993).

Patients along with their family members or care takers should understand the implications of the loss of protective sensation, and the importance of daily foot examinations and the proper foot care (Mayfield et al 2003).

Treatment

There have been advances in managing diabetic foot ulceration with the development of new dressings, growth factors, bioengineered skin and tissue substitutes, hyperbaric oxygen, negative pressure wound therapy, and other novel approaches to stimulate wound healing (Steed 1995; Steed et al 1996;

Gough et al 1997; Donaghue et al 1998; 2000; Hopf et al 2001). Allogeneic bi-layered cultured skin equivalent (Apligraf, Organogenesis Inc., Canton, MA) is a living, biological dressing developed from neonatal foreskin and consists of living cells and structural proteins. It is FDA approved for the treatment of diabetic foot ulcers (2000) and was shown to heal more noninfected, nonischemic chronic plantar diabetic foot ulcers faster and in more patients than conventional therapy in a large-scale multi-center randomized prospective clinical trial (Veves et al 2001). Becaplermin (Regranex, Johnson & Johnson, New Brunswick, NJ) is a hydrogel that contains 0.01% platelet derived growth factor-BB (rhPDGF-BB) and is currently the only commercially available topical growth factor for use in cutaneous wound healing. Although the efficacy becaplermin has been demonstrated in several studies (Rees et al 1999; Embil et al 2000) this has not been translated equivocally onto daily practice. It has been suggested that bercaplermin's lack of clinical success may be secondary to the imbalance between levels of matrix metalloproteases and their inhibitors in the fluids of ulcers causes elevated levels of proteases (Yager et al 1996). The proteases in turn destroy essential growth factors, extracellular matrix proteins, and receptors, including the ones specific for PDGF-BB to ultimately prevent wounds from healing (Ladwig et al 2002). Many suggest combining bercaplermin gel with collagen and oxidized regenerated cellulose to help bind the matrix metalloproteases and potentiate becaplermin's effects on wound healing.

Collagen and oxidized regenerated cellulose (Promogran, Johnson & Johnson, New Brunswick, NJ) is a sterile, freeze dried matrix sheet. ORC absorbs wound exudate and forms a soft, biodegradable gel that binds and inactivates matrix metalloproteases (MMP) which have been shown to have a detrimental effect on wound healing when present in excessive quantities (Wysocki et al 1993; Ladwig et al 2002). In addition, ORC binds growth factors within the wound, protects them from degradation and releases them back into the wound in an active form as the matrix is slowly broken down. The effectiveness of ORC has been demonstrated in several studies (Vin et al 2002; Omugha and Jones 2003).

Negative pressure wound therapy (Vacuum Assisted Closure, Kinetic Concepts Inc., San Antonio, TX) is a non-invasive wound closure system that uses controlled, localized sub-atmospheric pressure to help promote healing in chronic and acute wounds. This sub-atmospheric or negative pressure can be conveyed either continuously or intermittently though a sterile, latex free polyurethane or polyvinyl alcohol foam dressing. Negative Pressure Wound Therapy has been advocated my numerous authors as a safe and effective adjunctive modality in the treatment of diabetic foot wounds (Armstrong et al 2002; Eginton et al 2003; Armstrong and Lavery 2005; Mendonca et al 2005).

Despite these advances, it is imperative to remember the fundamental basics in the healing of diabetic foot ulcers: adequate perfusion, debridement, infection control, and pressure mitigation.

Vascularity

Adequate vascular perfusion is of utmost importance in wound healing (Dang and Boulton 2003). If pulses are not palpable, doppler ultrasound, ankle-brachial blood pressure indices as well as other non-invasive vascular studies such as segmental pressures, pulse volume recordings, and transcutaneous oxygen tension are warranted. A prompt vascular surgery consult and possible intervention such as angioplasty, stenting, or femorodistal bypass to improve perfusion and thereby effect healing is also indicated (Cavanagh et al 2005).

Infection control

Since infection is not well defined, the genesis, diagnosis and resolution of infection remains a clinical endeavor (Cavanagh et al 2005). Cultures, laboratory results, and subjective symptoms are helpful adjuncts. Wound cultures reveal the causative pathogens, however, tissue specimens are strongly preferred over wound swabs for wound cultures (Cavanagh et al 2005). While the diagnostic criteria for infection are imprecise, there is little doubt that infection is a major cause of lower extremity morbidity that frequently eventuates into wet gangrene and subsequent amputation. Antimicrobial therapy should be guided by culture results, focused at curing the infection rather than healing the wound (Cavanagh et al 2005).

Debridement

Debridement, the removal of hyperkeratotic and devitalized tissue, foreign materials, and particulate matter from a wound, is often the key first step of effective wound care (Lewis et al 2001; Armstrong et al. 2004; Steed 2004). Debridement helps reduce the rate of infection and provides an ideal healing environment by converting chronic wounds into acute (Falanga 2004). Wound debridement helps reduce chronic inflammatory byproducts (Nwomeh et al 1999; Jude et al 2001; Armstrong and Jude 2002) and may be accomplished surgically, chemically, mechanically, biologically, or by autolysis (Lewis et al 2001). Sharp or

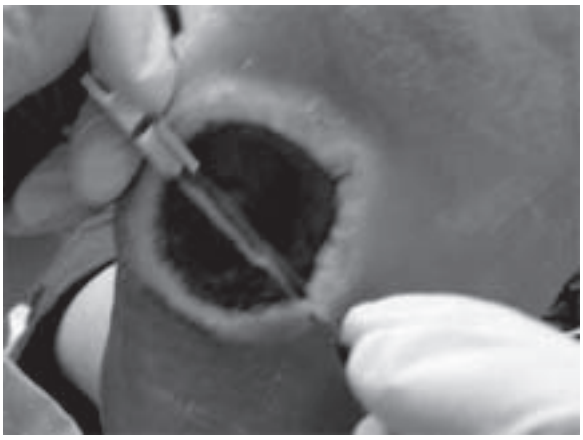

Figure 5 Debridement of wound margins to mitigate the "edge effect".

surgical debridement, the most direct and efficient method to clean the woundbed (Blanke and Hallern 2003), is generally considered the gold standard.

In addition to the wound base, it is also important to debride wound margins to mitigate the "edge effect" (Armstrong and Athanasiou 1998; Armstrong et al. 2004) (Figure 5). The edge effect is secondary to the skin interruption which increases both vertical and shear stresses on the edges of that interruption (Armstrong and Athanasiou 1998). The vertical force progressively deepens the wound, while the shear force from the underlying epithelium widens the wound via undermining (Armstrong and Athanasiou 1998).

Pressure mitigation

With sufficient vascular supply, appropriate debridement, moist wound environment, and infection control, the primary mode of healing a diabetic neuropathic foot ulcer is pressure dispersion (Armstrong et al 2004; Wu et al 2005). Of the plethora of offloading modalities including bed rest, wheel chairs, crutches, total contact casts, felted foam, half shoes, therapeutic shoes, custom splints, and removable cast walkers, total contact casting (TCC) (Figure 6) is considered by many to be the "gold standard" in achieving pressure redistribution (Coleman et al 1984; Walker et al 1985; Boulton et al 1986; Sinacore et al 1987; Walker et al 1987; Kominsky 1991; Lavery et al 1997). TCC has been shown to reduce pressure at the site of ulceration by 84%–92% (Lavery et al 1996) and is quite effective with healing rates ranging from 72%

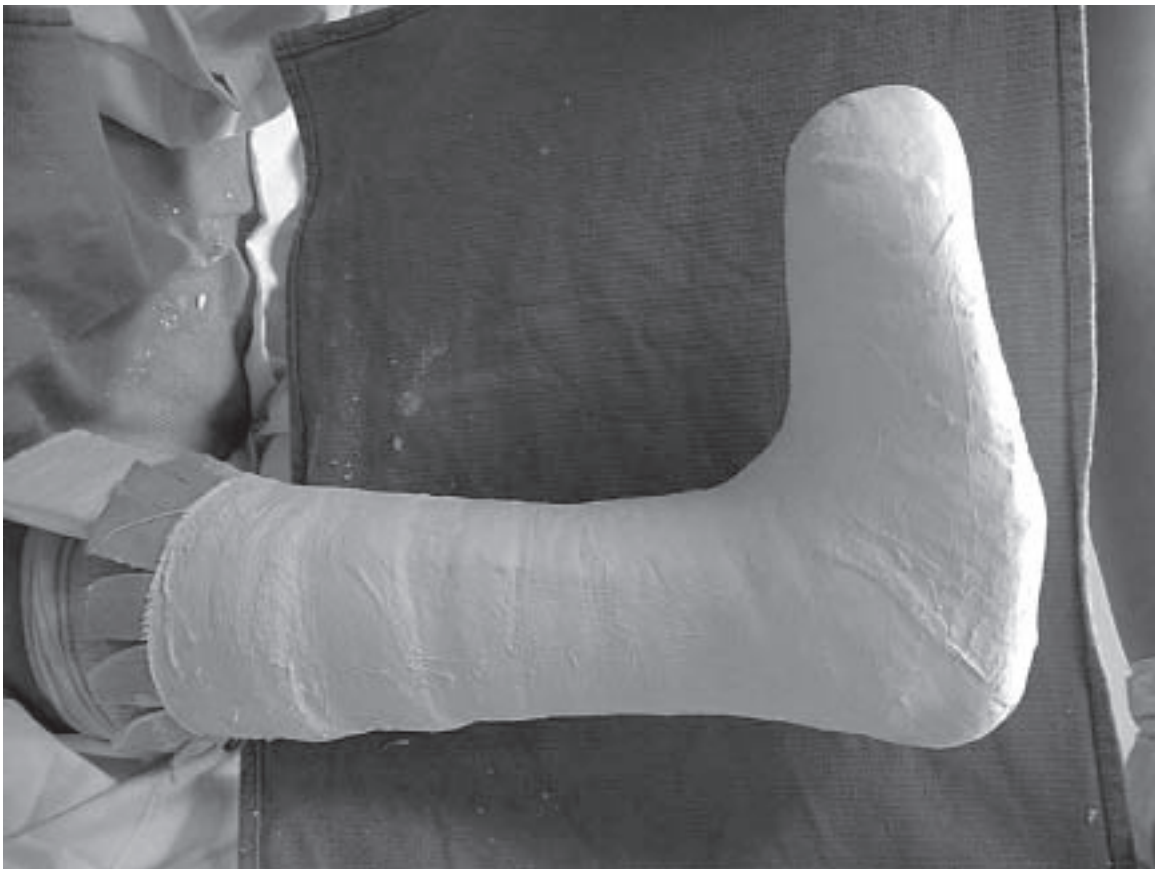

Figure 6 Total contact cast.

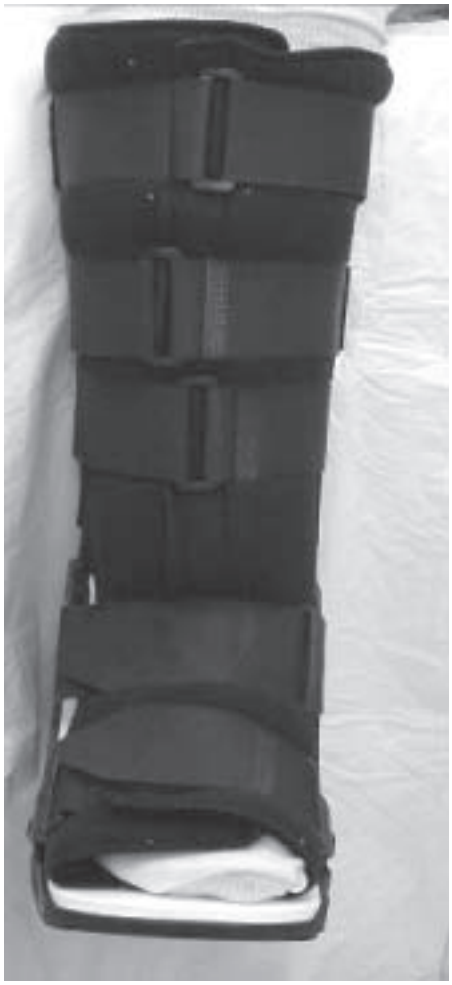

Figure 7a Removable cast walker.

to 100% over a course of 5–7 weeks (Helm et al 1984; Walker et al 1985; Sinacore et al 1987; Walker et al 1987; Myerson et al. 1992). However, the application of TCC is time consuming and often associated with a learning curve. Most centers do not have a physician or cast technician available with adequate training or experience to safely apply a TCC; improper cast application can cause skin irritation and in some cases even frank ulceration. In addition TCCs do not allow daily assessment of the foot or wound and are therefore often contraindicated in cases of soft tissue or bone infections. Recent studies have shown that an instant total contact cast (iTCC), made by simply wrapping a removable cast walker with a single layer of cohesive bandage, elastoplast, or casting tape (Figure 7a, b, c), may be just as effective as a TCC in pressure mitigation and the healing of diabetic foot ulcers (Wu et al 2005). A randomized controlled study compared the standard TCC with an iTCC (Katz et al 2005) and found no differences in healing rates and mean healing time between patients who received the TCC versus the iTCC . The proportions of patients that healed within 12 weeks in the iTCC and TCC groups were 80% and 74%, respectively (Katz et al 2005). There were no differences in complications between the two groups, however, the cost in materials and personnel was much lower for the iTCC compared with the TCC. The study concluded that the iTCC is not only equally efficacious in healing diabetic foot ulcers, when compared with the TCC but is quicker, easier, and more cost effective than the TCC (Katz et al 2005).

Conclusion

There is a high occurrence of foot ulcers within the population of people with diabetes. Foot ulcerations may lead to infections, lower extremity amputations and are major causes of disability to patients, often resulting in significant morbidity, extensive periods of hospitalization, and mortality. In order to diminish the detrimental consequences associated with diabetic foot ulcers, a high standard of care must be provided. Many of the etiological

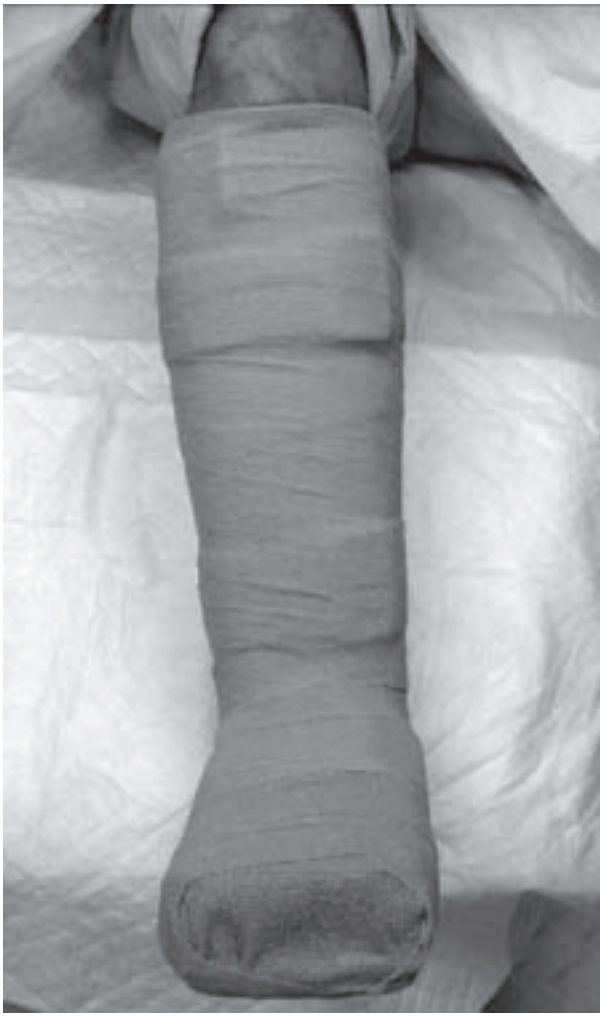

Figure 7b Instant total contact cast: made by wrapping the removable cast walker with a layer of cohesive bandage.

Figure 7c Instant total contact cast: made by wrapping the removable cast walker with a layer of plaster of paris.

factors contributing to the formation of diabetic foot ulceration may be identified using simple, inexpensive equipment in a clinical setting, and early recognition of these factors along with prompt management of the ulcers are essential for successful outcome (Van Damme and Limet 2005). Aggressive treatment of infections, correction of vascular occlusive disease, adequate wound care, and appropriate pressure mitigation are essential steps in the treatment protocol (Van Damme and Limet 2005). With the implementation of good prevention and treatment programs, a significant reduction of lower extremity complications well within reach.

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