Diabetic Foot Ulcers: Principles of Assessment and Treatment

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Foot ulcers are a major cause of morbidity among people with diabetes. At any given time, 2–3% of diabetics have an active foot ulcer, and 15% of all patients with diabetes will develop an ulcer during their lifetime [1–3]. Since pedal ulceration is the precursor of 70–90% of all diabetic amputations, the appearance of an ulcer should be regarded as a serious complication [2–5]. Although the number of amputations is on the rise, most pedal ulcers and subsequent amputations in diabetic patients are preventable when proper standards of care are followed [6–8]. Therefore, all practitioners who treat diabetic patients should have knowledge of the basic principles involved in the assessment and treatment of diabetic foot ulcers.

History and Physical Examination

Initial assessment

On the first visit, a general medical history is taken with particular attention given to conditions that may impede wound healing [Table 1]. The patient should be asked about onset, duration, course, and previous treatment of his/her wound. A general evaluation of the foot is conducted with special attention given to the detection of the major causes of ulceration: neuropathy, peripheral vascular disease, and musculoskeletal deformity [reviewed in ref. 9]. Next, the wound is examined for the presence of odor, granulation tissue, necrotic or fibrotic tissue, and pus or exudate. The wound is then debrided and measured. A photograph or sketch records the size and location of the wound for follow-up evaluation.

Diagnosis of infection

The diagnosis of infection in the diabetic foot is often subtle and difficult to reach. Since most patients with diabetic wounds have various underlying degrees of neuropathy, peripheral vascular disease, and abnormalities of the immune system, the classic findings of serious infection (inflammation, pain, fever, and elevated white blood cell count) are usually greatly diminished or absent [6,10]. Infection should certainly be suspected when there is purulence, exposed tendon or bone, radiographic changes in underlying bone, a complaint of pain in an insensate foot, a sinus tract, a non-healing wound, fever, or a sudden loss of glycemic control [1,6,10]. Culture and sensitivity alone cannot serve as a guide since all diabetic foot ulcers are contaminated though not necessarily infected [1,10]. A blunt, sterile steel probe should be used to check the base of the wound for hidden sinus tracts – sometimes the only sign of occult infection [1,6,11,12].

Table 1. Impediments to wound healing

Poor glycemic control	Impaired immune system
Hyperlipidemia	Infection
Electrolyte depletion	Necrotic tissue
Nutritional deficiencies	Repetitive trauma
Ischemia	Malignancy
Venous insufficiency	Chemotherapeutic agents
Hepatic disease	Steroids
Chronic renal failure	Cigarette smoking
Anemia	

When the wound is longstanding (6 or more weeks) or there is a sinus tract to bone, diagnostic X-rays should be ordered to check for osteomyelitis [1,8,13]. Since a 30–50% resorption of bone matrix must occur before lytic changes are seen on X-ray, the absence of positive findings on standard radiographs cannot rule out osteomyelitis [14]. The diagnostic usefulness of three-phase bone scans is limited in the diabetic foot because they are unable to detect low grade osteomyelitis and to differentiate between osteomyelitis and neuropathic bone disease [14,15]. Leukocyte scanning with indium oxyquinolone is the leading imaging technique for diagnosis of osteomyelitis in terms of both sensitivity and specificity [14,15]. Technical difficulties in performing this test, however, have hindered its use. Magnetic resonance imaging is less sensitive than leukocyte scanning in detecting low grade osteomyelitis and is unnecessarily expensive for diagnosing osteomyelitis in the severely infected foot [14]. MRI can be useful in planning the surgical debridement of deep plantar space abscesses. In contrast to diagnostic imaging, bone biopsy provides a definitive diagnosis as well as material for culture [15].

Detection of ischemia

As with the diagnosis of infection, evaluation of peripheral vascular disease in the diabetic can often be subtle, since the manifestations of diabetes often conspire to obscure and confound the diagnosis of ischemia [16,17]. Sensory neuropathy, for example, may mask the symptoms of intermittent claudication and rest pain. Autonomic neuropathy may cause

the foot to be warm even though nutrient capillary flow is poor [17]. Tibialis posterior and dorsalis pedis pulses may be palpable, yet, with the predilection of diabetics for distal arterial disease, occlusions may occur distal to the palpated pulse [18]. Even the results of Doppler ultrasound may be skewed by calcification of the intima in the arterial walls [6,16–19]. To obtain as true an assessment as possible, the vascular lab should perform a battery of tests that are usually unnecessary in the non-diabetic, such as segmental leg pressures, pressure of the hallux, and wave form analysis [17]. If deemed necessary, a stress test and transcutaneous oxygen measurements are performed [17].

Classification of the diabetic ulcer

Once the history and physical examination of the wound have been completed, the wound should be graded according to a classification system that is descriptive, reproducible, and understood by all healthcare professionals treating the patient. Ideally, the grade should help guide treatment, offer a rough prognosis for complete healing or risk for amputation, and provide a gauge to progress or deterioration.

Although a number of different grading systems has been used, the new University of Texas Diabetic Wound Classification System is rapidly gaining wide acceptance [Table 2] [20]. With this method, wounds are classified not only by depth, but also by the presence or absence of infection and ischemia. Severity and risk for an amputation increase as we move downward and to the right on the chart.

Principles of Treatment

Six modes of treatment have been established for diabetic foot wounds [1]. Three of these treatments are applicable to every wound; the other three are applicable in the presence of specific criteria.

Debridement

Frequent debridement of neuropathic wounds has been shown to enhance and shorten the healing process [1,21]. When necrotic tissue, crusted exudate, and fibrinogen are removed, growth factors are released and fibroblasts and keratinocytes migrate more easily into the wound. The benefits are obtained only by sharp, surgical debridement [1]. The practices of enzymatic and mechanical debridement such as whirlpool or soaks in soap and water are not supported by clinical evidence and may lead to maceration and infection [1,6].

Off-loading

The second standard of care, referred to as "off-loading," is the avoidance of all mechanical stress on the wound [1,22]. The devices to off-load the diabetic foot wound are too numerous to recount here. Examples of the better-known modalities are total contact casts, surgical shoes, walkers, healing sandals, felted foam dressings, and bed rest. The method of choice is determined by the location of the wound and by the patient's level of activity.

Dressings

The third treatment applicable to all diabetic wounds is the dressing [1]. Dressings not only protect the wound from trauma and infection but also affect the wound environment [1,23]. A moist but not heavily exudative environment has been shown to enhance healing, therefore the choice of dressing will promote or preserve this environment. The dressing for a dry wound should moisturize; the dressing for a macerated wound, desiccate.

Control of infection

The fourth principle of treatment is aggressive management of infection. In the infected wound, debridement of purulent debris controls the infection and shortens the course of antibiotics [1]. Attempting to cure deep plantar space abscesses and osteomyelitis through antibiotic therapy alone may result in prolonged treatment or even unnecessary amputation. A culture should be performed only after the wound has been debrided, since superficial swabbing of an undebrided ulcer is believed to yield results that have poor correlation to the actual pathogens [1,15]. However, the assumption that superficial swabs are unreliable may be unfounded. Our recent (unpublished) data indicate that

Table 2. The University of Texas Diabetic Wound Classification System

(Increasing complication by infection and/or ischemia)	Grade (increasing depth)			
	Stage	**0**	**I**	**II**	**III**
	A	Pre- or postulcerative lesion completely epithelialized	Superficial wound, not involving tendon, capsule or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
	B	Pre- or postulcerative lesion, completely epithelialized with infection	Superficial wound, not involving tendon, capsule or bone with infection	Wound penetrating to tendon or capsule with infection	Wound penetrating to bone or joint with infection
	C	Pre- or postulcerative lesion, completely epithelialized with ischemia	Superficial wound, not involving tendon, capsule or bone with ischemia	Wound penetrating to tendon or capsule with ischemia	Wound penetrating to bone or joint with ischemia
	D	Pre- or postulcerative lesion, completely epithelialized with infection and ischemia	Supercifial wound, not involving tendon, capsule or bone with infection and	ischemia	Wound penetrating to tendon or capsule with infection and ischemia

superficial swabs may be identical to deep tissue specimens up to 90% of the time.

Antibiotic therapy should be instituted immediately, with selection being based on clinical judgment [Table 3]. A mild infection from a superficial wound can often be treated with an oral agent that is active against the usual microbes in skin infections, namely the gram-positive cocci [6,15]. However, infections penetrating to the level of tendon or bone should be treated with broad-spectrum antibiotics that provide coverage for gram-positive cocci, gram-negative bacilli and anaerobes [6,15]. Serious limb- or life-threatening diabetic infections are usually polymicrobial and may often contain a pathogen that is resistant even when a regimen of broad-spectrum antibiotics is followed. Therefore, as soon as the results of the culture and sensitivity are available, the antibiotics should be adjusted accordingly.

Revascularization

Although ischemia is rarely the most important cause of ulceration, it is often an impediment to healing [17]. Since the metabolic requirements rise significantly when a wound or infection develops, a foot with sufficient perfusion to sustain intact skin may not have enough perfusion to sustain healing [17]. There are no rigid indications for revascularization [17,18]. Poorly perfused wounds will sometimes heal, while relatively well-perfused wounds may not close until revascularization has been performed. When a wound has failed to heal despite optimal care, and the ankle-brachial and toe-brachial indices are less than 0.8 and 0.6 respectively, vascular reconstruction should be considered [16,18].

Table 3. Initial antimicrobial regimens for diabetic foot infections

Non-limb threatening infection*
Oral treatment
Cephalexin
Cefuroxime
Clindamycin
Amoxicillin-clavulanate
Limb-threatening infection**
Oral treatment
Ciprofloxacin and clindamycin
Parenteral treatment
Ampicillin–sulbactam
Ticarcillin–clavulanate
Life-threatening infection***
Parenteral treatment
Imipenem–cilastatin
Vancomycin, metronidazole, and aztreonam
Cefotaxime and clindamycin

* Local cellulitis with no systemic involvement.
** Osteomyelitis or deep plantar space abscess without systemic involvement.
*** Osteomyelitis or deep plantar space abscess with systemic involvement.

Amputation

Amputation is not only the most feared sequela of ulceration but also a possible treatment. Accepted criteria for amputation are gangrene, uncontrollable infection, and intractable ischemic pain [24]. However, all too often the impetus behind an amputation is the doctor's frustration and impatience with treating a recalcitrant wound. Furthermore, too many amputations are performed without any thought of biomechanical function [24,25]. These amputations simply serve as a prelude to more proximal amputations.

Adjunctive treatments

Besides the established treatments mentioned above, other adjunctive modalities have been advocated. These include growth factors, hyperbaric oxygen therapy, vacuum suction, maggots, living skin equivalents, electrical stimulation, and cold laser. Of these adjunctive therapies, only growth factors (becaplermin and platelet-derived wound growth factor) have proven efficacious when used as supplements to good wound care [1]. Hyperbaric oxygen therapy for neuropathic, non-ischemic wounds has not been supported by randomized controlled trials [1]. This treatment should be considered in the treatment of serious wounds that have not responded to established treatments, especially when complicated by ischemia [1]. All other modalities are based on anecdotal evidence or are still under investigation [1].

Conclusion

Management of the diabetic patient with a pedal ulceration presents a difficult, but not insurmountable, challenge. Most ulcerations can and should heal when diagnosed and treated appropriately. Assessment of the wound should be thorough, identifying both the etiology and the current impediments to healing. Only after a precise assessment and classification have been done can an effective treatment regimen be established. Since optimal wound treatment often needs to be directed at both local and systemic factors, a multidisciplinary diabetic team comprising an internist, a podiatrist, a vascular surgeon, and other specialists as needed, is essential for achieving quality care [26,27].

References

1. American Diabetes Association. Consensus development conference on diabetic foot wound care. *Diabetes Care* 1999;22:1354–60.
2. Reiber GE, Vileikyte L, Boyko EJ, Del Aguila M, Smith DG, Lavery LA, Boulton AJM. Causal pathways for incident lower-extremity ulcers in patients with diabetes from two settings. *Diabetes Care* 1999;22:157–62.
3. Sedory Holzer SE, Camerota A, Martens L, Cuerdon T, Crystal-Peters J, Zagari M. Costs and duration of care for lower-extremity ulcers in patients with diabetes. *Clin Ther* 1998;20:169–81.
4. Ollendorf DA, Kotsanos JG, Wishner WJ, Friedman M, Cooper T, Bittoni M, Oster G. Potential economic benefits of lower-extremity amputation prevention strategies in diabetes. *Diabetes Care* 1998;21:1240–5.
5. Apelquist J, Bakker K, Van Houtum WH, Nabuurs-Franssen MH, Schaper NC. International Consensus on the Diabetic Foot. Amsterdam: International Working Group on the Diabetic Foot, 1999.

6. Caputo GM, Cavanagh PR, Ulbrecht JS, Gibbons GW, Karchmer AW. Assessment and management of foot disease in patients with diabetes. *N Engl J Med* 1994;331:854–60.
7. Apelquist J, Ragnarson-Tennvall G, Persson U, Larsson J. Diabetic foot ulcers in a multidisciplinary setting: an economic analysis of primary healing and healing with amputation. *J Intern Med* 1994;235:463–71.
8. Levin ME. Foot lesions in patients with diabetes mellitus. *Endocrinol Metab Clin North Am* 1996;25:447–61.
9. Slater R, Mizrahi A, Rapoport M. Podiatric screening and assessment of the diabetic foot. *Harefuah* 1999;136:502–5 (Hebrew).
10. Armstrong DG, Perales TA, Murff RT, Edelson GW, Welchon JG. Values of white blood cell count with differential in the acute diabetic foot infection. *J Am Podiatr Med Assoc* 1996;86:224–7.
11. Frykberg RG. Diabetic foot ulcerations. In: Frykberg RG, ed. The High Risk Foot in Diabetes Mellitus. New York: Churchill Livingstone, 1991:151–95.
12. LeFrock JL, Joseph WS. Bone and soft-tissue infections of the lower extremity in diabetics. *Clin Podiatr Med Surg* 1995;12:87–103.
13. Kominsky SJ. Outpatient management. In: Kominsky SJ, ed. Medical and Surgical Management of the Diabetic Foot. St. Louis: Mosby, 1994:223–47.
14. Newman LG. Imaging techniques in the diabetic foot. *Clin Podiatr Med Surg* 1995;12:75–86.
15. Joseph WS. Bone and joint infections. In: Joseph WS, ed. Handbook of Lower Extremity Infections. New York: Churchill Livingstone, 1990:45–63.
16. Campbell DR. Diabetic vascular disease. In: Frykberg RG, ed. The High Risk Foot in Diabetes Mellitus. New York: Churchill Livingstone, 1991:33–47.
17. Sykes MT, Godsey JB. Vascular evaluation of the problem diabetic foot. *Clin Podiatr Med Surg* 1998;15:49–83.
18. Gibbons GW. Vascular evaluation and long-term results of distal bypass surgery in patients with diabetes. *Clin Podiatr Med Surg* 1995;12:129–40.
19. Logerfo FW, Gibbons GW. Vascular disease of the lower extremities in diabetes mellitus. *Endocrinol Metab Clin North Am* 1996;25:439–45.
20. Armstrong DG, Lavery LA, Harkless LB. Who is at risk for diabetic foot ulceration? *Clin Podiatr Med Surg* 1998;15:11–19.
21. Steed DL, Donohoe D, Webster MW, Lindsley L. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. *J Am Coll Surg* 1996;183:61–4.
22. Armstrong DG, Lavery LA. Evidence-based options for off-loading diabetic wounds. *Clin Podiatr Med Surg* 1998;15:95–104.
23. Higgins KR, Ashry HR. Wound dressings and topical agents. *Clin Podiatr Med Surg* 1995;12:31–40.
24. Stuck RM, Sage R, Pinzur M, Osterman H. Amputations in the diabetic foot. *Clin Podiatr Med Surg* 1995;12:141–55.
25. Pulla RJ, Kaminsky KM. Toe amputations and ray resections. *Clin Podiatr Med Surg* 1997;14:691–739.
26. Ragnarson-Tenvall G, Apelquist J. Cost-effective management of diabetic foot ulcers. *Pharmacoeconomics* 1997;12:42–53.
27. Apelquist J. Wound healing in diabetes: outcome and costs. *Clin Podiatr Med Surg* 1998;15:21–39.

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