Exploring the complex care of the diabetic foot ulcer

Wound care, infection treatment, and pressure relief are the cornerstones of managing diabetic foot ulcers. A multidisciplinary approach to prevention and patient education are essential components of care.

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Diabetic foot ulcers are a complication affecting approximately 15% of people with diabetes. Treating these ulcers is costly, and the resulting disfigurement can be devastating. Twenty-five percent of all hospital admissions are for diabetic foot ulcers. The average hospital stay among patients with foot ulcers is 60% longer than the stay for other causes and costs $20,000 to $60,000 per patient, or up to $6 billion per year. The risk of amputation is 15 to 40 times greater in a person with diabetes than in one who does not have the disease.[1-6]

An ulcer is defined as a break in the cutaneous layer of the skin extending to the dermis.[5] Diabetic foot ulcers account for more than 50% of nontraumatic amputations and are associated with high rates of mortality, reamputation, and contralateral limb amputation.[2,7] Vascular insufficiency and peripheral neuropathy are the most common risk factors for developing foot ulcers[6,8] (see Table 1, page 33). The risk factors for amputation are identical to those for foot ulcers because 85% of amputations result from foot ulcers.[9]

CME

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Learning objectives

- Describe the history and physical assessment of a patient with a diabetic foot ulcer
- Outline how to classify diabetic foot ulcers to determine the treatment plan
- Discuss the evaluation and treatment of diabetic foot infections

Prevention as a first step

Health maintenance is an ongoing process, and optimal ulcer prevention is achieved through a multidisciplinary approach[2,6] (see Table 2, page 33). Clinicians should always remind patients with diabetes to eat a proper diet, exercise regularly, and practice good hygiene.[10] The American Diabetes Association recommends examining these patients' feet at each visit, in addition to performing an annual comprehensive foot examination that includes using a monofilament to test for possible loss of sensation.[5] All patients with diabetes should be evaluated by a podiatrist regularly.[11] Patients should be counseled to avoid smoking cigarettes, walking barefoot, using heating pads, and stepping into a bath without first checking the temperature.[5] Those with severely deformed feet need to wear custom-molded shoes or have corrective prophylactic surgery.[2] Research has demonstrated that the majority of foot ulcers are caused by poorly fitting shoes. Other sources of foot trauma are foreign bodies in the shoes, burns from hot water (see Figure 1, page 34), and ingrown toenails. Therapeutic footwear and socks are essential in foot ulcer prevention.[6,10]

Evaluation of foot ulcers

Patients often have had glucose abnormalities for years before receiving a diagnosis of diabetes. Complications begin to occur 10 to 15 years after diagnosis, earlier if

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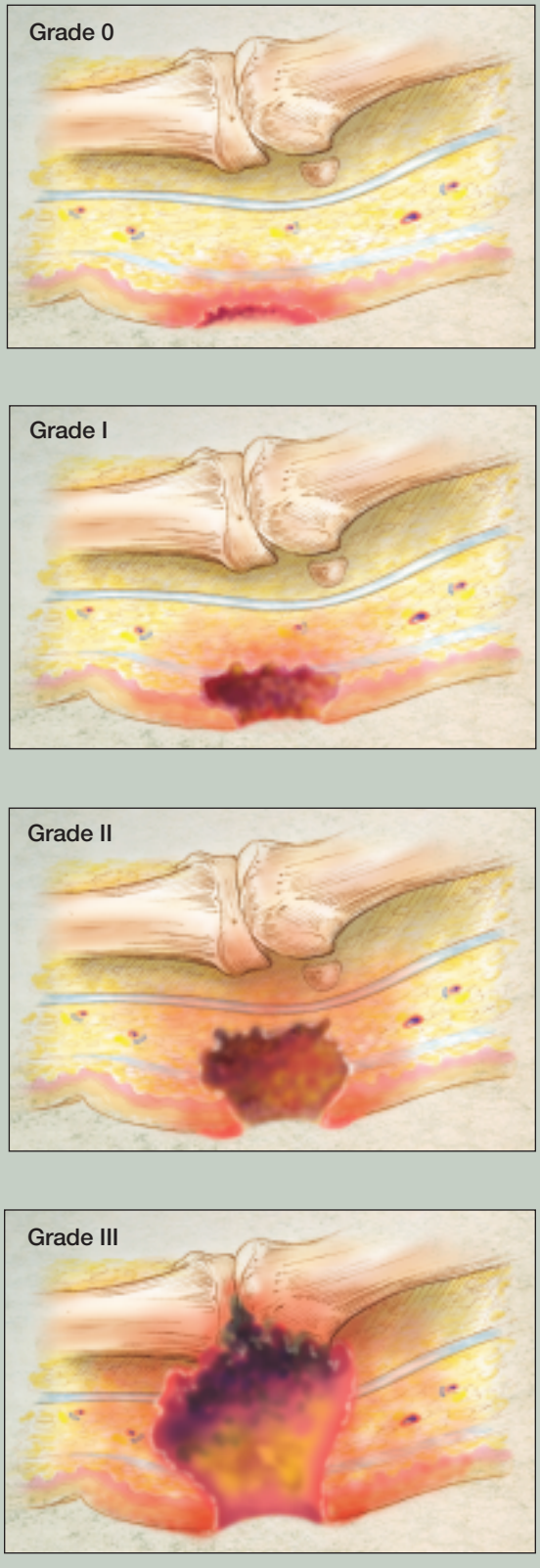
Diabetic foot ulcer progression



Here and on the cover: Michele Graham

glycemic control is poor. During the initial evaluation of a patient with a foot ulcer, the clinician should obtain a comprehensive history (see Table 3, page 34) and perform a complete physical examination.[11-13] At each visit, a thorough written description of the ulcer's characteristics, including appearance, size, depth, and location, should be recorded and a photograph taken. This provides a map of progress during treatment.[11] Patients should be questioned about leg discomfort including sensations, location, and timing, as well as aggravating and alleviating factors.[5,13] Neuropathic ulcers occur on the pressure points of the foot, while vascular ulcers occur on the side and heel of the foot and are painful.[8,10]

Neuropathy is defined as decreased ability to feel pain as a result of the loss of protective sensation.[6,10] The nerve axon causing vasodilation in response to painful stimuli is impaired.[1] Inability to perceive the pressure sensation of a 10-g monofilament indicates sensory neuropathy. The neurologic examination also includes measurements of proprioceptive, vibratory, pain, and temperature sensations and deep tendon reflexes.[2,4,5,9]

Next, the foot should be examined for Charcot's joint, contraction of the toes, dry skin, hammer toes, depressed metatarsal heads, and hallux valgus.[14,15] Charcot's joint is defined as pathological fractures that cause joint dislocation and destroy the normal shape of the foot.[2] Patients with diabetes who have a Charcot's joint typically complain of swelling and discomfort, and ulcers develop on the bony prominences.

Vascular disease

Peripheral vascular disease (PVD) is two to four times more common in patients with diabetes than in those without diabetes.[6,12] Large-vessel disease manifests 10 years earlier and accounts for 70% of all deaths among these patients.[12] Microangiopathy occurs at a younger age, affects both sexes equally, and typically involves the tibial and peroneal arteries.[8] Cigarette smoking aggravates vascular insufficiency by increasing peripheral vasoconstriction.[6]

Vascular compromise develops from the inability of the capillaries to vasodilate in response to trauma.[8] Patients complaining of claudication and presenting with decreased pulses, absence of hair on the legs, and cool skin temperature require prompt vascular evaluation. Other signs of vascular insufficiency are pain at rest, ischemic rubor when the feet are not elevated, and tissue necrosis.[11,13,15] Pain due to vascular insufficiency is aggravated by walking and relieved by keeping the feet in a dependent position.[13]

The presence of both the pedal and popliteal pulses is the most reliable indicator of arterial perfusion in the foot.[9] The vascular examination also includes assessment of capillary refill, evaluation for the presence of

hair, measurement of skin temperature, and evaluation for stasis edema.[2,11,14] Absence of a pedal pulse with presence of a popliteal pulse is a classic sign of diabetic arterial disease. Arterial Doppler studies should be performed, but arteriography should be reserved for patients in whom revascularization is being considered.[9,15] A noninvasive method for assessing PVD is the ankle-brachial index (ABI). An ABI less than 0.9 indicates PVD. If the ulcer does not appear to be healing or if there are signs of PVD, the patient should be referred to a vascular specialist.[2,11-13]

Classification of the diabetic foot ulcer leads the clinician to the best treatment course and its anticipated outcome.[2,9] There are two commonly used classification systems. The University of Texas system was developed specifically for staging diabetic foot ulcers[2,6] (see Table 4, page 35). The second commonly used system is the Wagner system, which was initially developed for PVD and later adapted for diabetic foot ulcers[2,6] (see Table 5, page 36). The principles of treating diabetic foot ulcers are simple: good wound care, treatment of infections, and pressure relief.[16]

Wound care

Wound-bed preparation reduces the wound margins to well-vascularized granulation tissue without signs of local infection and promotes the healing process.[1,2] The Wound Healing Society defines a chronic wound as one that fails to proceed through an orderly and timely repair process.[2,6] There are four phases to the wound healing process: coagulation, inflammation, proliferation, and tissue remodeling.

Debridement of necrotic tissue is a crucial first step in treating diabetic foot ulcers. Patients should be referred for surgical debridement early.[8] The procedure should be comprehensive, removing all nonviable tissue and extending the wound margins 2 to 3 mm into healthy, nonhyperkeratotic skin.[2,9,17] There are four types of debridement: surgical, enzymatic, autolytic, and mechanical. Surgical and mechanical debridements use some type of trauma to remove tissue, while autolytic and enzymatic debridements use a more natural method. Debridement removes local bacteria, stimulates healing, documents absence of hyperkeratosis, and decreases local infection.

The dressing used on a debrided ulcer should provide a moist environment to minimize trauma and reduce risk of infection.[1,2,6] Wet-to-dry dressing with normal saline is considered the standard of care. The tissue must be kept moist to avoid devitalization and deepening of the ulcer.[9] One alternative to wet-to-dry dressings is the use of platelet-derived growth factor (PDGF), an endogenous protein that exerts a tremendous influence on tissue granulation and thereby

IN THIS ARTICLE

Key Points

- Twenty-five percent of all hospital admissions are for diabetic foot ulcers. The average hospital stay among patients with foot ulcers is 60% longer than that for other causes.
- A thorough description and a photograph of the ulcer taken at each visit provide a map of progress during treatment.
- The principles of treating diabetic foot ulcers are simple: good wound care, treatment of infections, and pressure relief.

Competencies

Medical knowledge	◆◆◆◆◆
Interpersonal & communication skills	◆
Patient care	◆◆◆
Professionalism	◆
Practice-based learning and improvement	◆
Systems-based practice	◆

For an explanation of competencies ratings, see the table of contents.

TABLE 1
Risk factors for developing foot ulcers

Age >65 y	Peripheral neuropathy
Foot deformity	Poor glycemic control
Length of time with diabetes	Previous ulcer or amputation
Male sex	Vascular insufficiency

Data from Frykberg RG et al,[2] and McCulloch DK.[5]

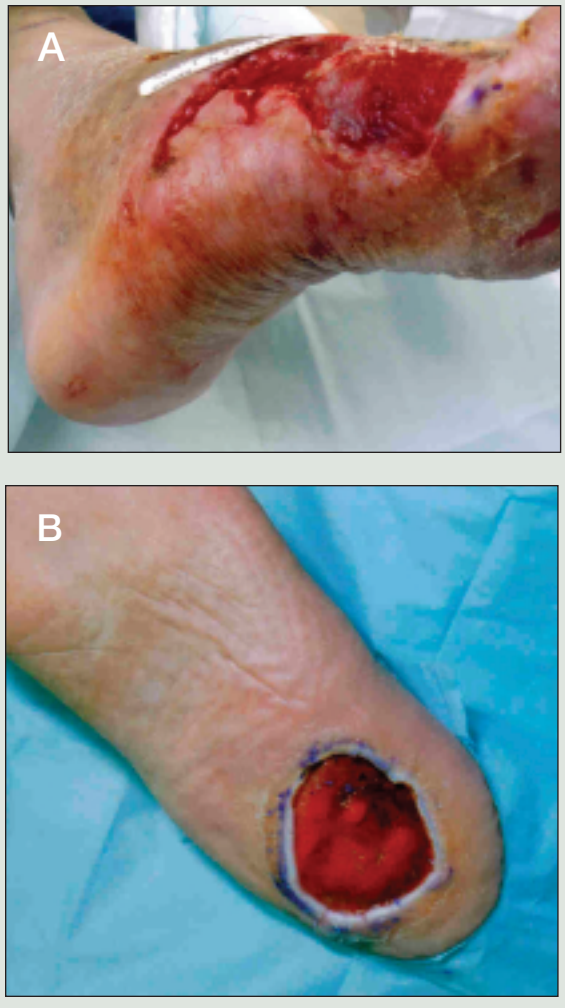
TABLE 2
Multidisciplinary team for foot ulcer prevention

Cardiologist	Nutritionist
Endocrinologist	Orthopedic surgeon
Infectious disease physician	Pedorthist
General internist	Podiatrist
Nephrologist	Teaching nurse

Data from Frykberg RG et al,[2] and Snyder RJ.[12]

FIGURE 1

Common types of diabetic foot ulcers



These photographs show a superficial ulcer caused by a burn (A) and an ulcer induced by the patient's stepping on an object while walking barefoot (B).

TABLE 3

Elements of a comprehensive history

Alcohol and tobacco use	Glycemic control
Claudicating symptoms	Inciting trauma
Duration of diabetes	Neuropathy symptoms
Duration of ulcer	Previous foot surgery
Eye complications	Previous ulcers
Footwear	Renal complications

Data from Frykberg RG et al,[2] McCulloch DK,[5] Daugherty KK et al,[6] Inlow S et al,[11] Snyder RJ,[12] and McCulloch DK and Hordon LD.[13]

improves wound healing.[2,4,18] PDGF has been shown to stimulate chemotaxis and mitogenesis of the neutrophils, fibroblasts, and monocytes by which wound healing can occur.[2,4,9,13,18] A third option is to use dermal/skin substitutes. They function as biological dressings, delivery systems for growth factors, and extracellular matrix components.[1,2,9,13]

Intermittent hyperbaric oxygen (HBO) therapy may help salvage marginally perfused tissue. With HBO therapy, the patient breathes 100% oxygen while inside a pressurized treatment chamber.[19,20] It is believed that this increases the oxygen tension in the wound, thereby increasing granulation tissue formation and accelerating wound contraction. Some reports show that HBO therapy reduced the number of major amputations in patients with diabetes.[21] HBO therapy relies on the presence of an adequate blood supply to the wound, and conflicting data on its effectiveness make this treatment controversial. Therefore, it should be used in conjunction with standard therapy and only after other methods have failed. Patients who have dysbarism and those who have claustrophobia should not receive this treatment.[4,6,19-21]

Infection treatment

Because of the high incidence of infection associated with diabetic foot ulcers, a systematic approach for a complete assessment is required. Foot ulcers are entry portals for systemic infection, and when left untreated, they are a threat to life and limb. Determining the severity of infection is important according to the Infectious Diseases Society of America.[22] In a mild infection, there is less than 2 cm of erythema; in a moderate one, there is more than 2 cm of erythema; and in a severe one, both infection and systemic toxicity are present.[22] Infection is presumed if erythema, warmth, tenderness, swelling, or pus oozing from the wound is noted.[5] Initial indications are an increase in temperature compared to the other foot, a malodorous discharge, erythema, swelling, and tenderness.[4,11]

Patients with cellulitis often do not feel pain because of the sensory neuropathy. The diagnosis is usually made by clinical appearance.[22] Typically, osteomyelitis occurs gradually over a few days to a week and produces fever and rigors.[23] Constitutional symptoms such as nausea, vomiting, malaise, and fatigue are other important clinical clues to osteomyelitis. Frequently, however, the only indication of infection may be unexplained hyperglycemia. Infections often begin around the toenail bed, at cracks in the skin, or in ischemic ulcers.[2,22]

If an ulcer is infected, the patient should be evaluated for extent of infection, glycemic control, and loss of sensation.[22] The ulcer should be probed to determine bone involvement, sinus tract formation, and extension into the tendon sheaths.[2] Deep cultures should be taken because superficial cultures grow only skin

flora.[1,6,8,9] Diabetic foot infections are commonly polymicrobial, containing both aerobic and anaerobic bacteria.[1] Necrosis or a fetid odor indicates an anaerobic infection.[17]

Leukocytosis is usually found in acute osteomyelitis but not in chronic osteomyelitis.[23] If the bone is exposed, the patient is assumed to have osteomyelitis.[2] Patients with soft tissue infections lasting longer than 2 weeks, especially over a bony prominence, are at high risk for this condition. The definitive method of diagnosing osteomyelitis is by bone biopsy, but this is not always practical or possible.[22] The ESR is a specific but insensitive diagnostic tool; it is frequently elevated above 100 mm/h in osteomyelitis but can be normal. Furthermore, the ESR often is inaccurate in patients with end-stage renal disease or nephrotic syndrome. However, an ESR higher than 70 mm/h with a 3-mm ulcer is specific for osteomyelitis. A high ESR can be particularly helpful in detecting a relapse.[23]

Imaging studies can be helpful in assessing diabetic foot infections. However, radiologic changes occur late in the course of osteomyelitis, so a negative radiograph does not rule out this diagnosis.[13] Osteomyelitis is not visible on plain radiographs until 10 to 14 days after the onset of bone involvement.[2] Radionuclide bone scans may show abnormalities earlier than radiographs, but they are less specific when bone turnover is high.[1,2] MRI is the procedure of choice for diagnosing osteomyelitis in patients with diabetes. A drawback is that it cannot distinguish whether abnormal bone marrow edema is due to neuropathic changes or osteomyelitis. The changes seen on MRI are not pathognomonic of osteomyelitis but can be used as a basis for initiating treatment or proceeding to bone biopsy. Indium-labeled leukocyte scanning uses radioactive indium as the tracer; it accumulates in the bone marrow at the site of infection or inflammation. It is more reliable than plain imaging but has a lower specificity and sensitivity than MRI.[13,22,23]

Diabetic foot infections are divided into non-limb-threatening and limb-threatening. In non-limb-threatening infections, cellulitis is less than 2 cm deep and does not extend to the bone or joint, and there is no evidence of systemic toxicity. In limb-threatening infections, cellulitis is more than 2 cm deep and extends to the bone or joint, and systemic infection is present.[2,6] Hospitalization for limb-threatening infections is mandatory.[2,22] Mild soft tissue infections are treated with oral antibiotics for 1 to 2 weeks, but deep infections require broad-spectrum IV therapy and deep surgical debridement.[1,6,9,15,22]

Empiric therapy should be started initially and cultures obtained. Once the pathogen causing the infection is identified, therapy can be switched to an agent that is specific for that organism. IV therapy should usually continue for 6 weeks, but shorter courses can be used if the affected bone has been resected. Moderate and severe infections, which often are polymicrobial, require 2 to 4 weeks of IV therapy, switching to oral therapy after a good response.[2,6,22]

Pressure relief

Off-loading, also called pressure relief, is the third principle of treatment[24] (see Table 6, page 36). Bed rest, an ideal pressure relief measure, often is impractical.[16] Off-loading is limited by the patient's physical characteristics and ability to comply with treatment and by the location and severity of the ulcer.[24,25] The heel is the most difficult plantar area to off-load; a wheelchair or crutches may be required.[25] Despite these drawbacks, off-loading remains the gold standard for ulcer treatment and for preventing recurrence.[25]

Total contact casting (TCC) is the most effective method of off-loading. A total contact cast is a special

TABLE 4
University of Texas classification system

Stage	Grade 0	Grade I	Grade II	Grade III
A	Pre- or post-ulcerative lesions completely epithelialized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infected	Infected	Infected	Infected
C	Ischemic	Ischemic	Ischemic	Ischemic
D	Infected and ischemic	Infected and ischemic	Infected and ischemic	Infected and ischemic

Adapted from Frykberg RG, Armstrong DG, Giurini J, et al. Diabetic foot disorders: a clinical practice guideline. American College of Foot and Ankle Surgeons. *J Foot Ankle Surg.* 2000;39(5 suppl):S1-S60.

TABLE 5
Wagner classification system

Grade	
0	Preulcerative state
1	Superficial ulcer
2	Full-thickness ulcer
3	Deep ulcer with or without osteomyelitis, abscess, or joint sepsis
4	Gangrene of a geographical portion of the foot
5	Extensive gangrene rendering the foot beyond salvage

Data from Frykberg RG et al,[2] and Epstein DA and Corson JD.[8]

TABLE 6
Off-loading techniques

Accommodative dressings	Patellar tendon-bearing braces
Assistive devices	Removable walking braces
Callus removal	Scotchcast boot
Foot casts	Shoe cutouts
Half, wedge, or surgical shoes	Surgical correction of deformity
Orthoses	Therapeutic shoes
Padded hosiery	Total contact casting

Data from Frykberg RG et al,[2] and Spencer S.[16]

cast designed to redistribute the patient's weight off the ulcer site, allowing ambulation while the ulcer is healing. The major drawbacks are that TCC limits access to the ulcer for dressing changes and wound inspection and that it requires a technician specifically trained in its application.[1,2,4,6,8,9] The patellar tendon-bearing brace is expensive and challenging to make but allows for considerable mobility while off-loading the entire foot.[25] Surgical shoes can be used in place of TCC, but they are not as effective and must be worn even when the patient takes one or two steps.[17] In patients with foot deformities, an orthopedic evaluation should be considered.[13] Surgical treatment corrects the foot deformity and improves foot mechanics.[25]

Conclusion

Many amputations resulting from diabetic foot ulcers are preventable with a thorough evaluation and aggressive treatment. The most important primary preventive measures are properly fitting socks and shoes and good control of blood glucose and lipid levels and of BP. A comprehensive, multidisciplinary evaluation, including determination of etiology, is essential in treating the diabetic foot ulcer.

The primary objective is to heal the ulcer and prevent amputation. Patients with an infected foot ulcer benefit from early, aggressive treatment that encompasses debridement, good wound care, and pressure relief. An infected diabetic foot ulcer requires close supervision whether it is limb threatening or not. A radiograph of the foot is acceptable initially, but a technetium scan or MRI may be warranted to confirm the clinical assessment. Antibiotic coverage should be broad spectrum and tailored to the patient. □

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