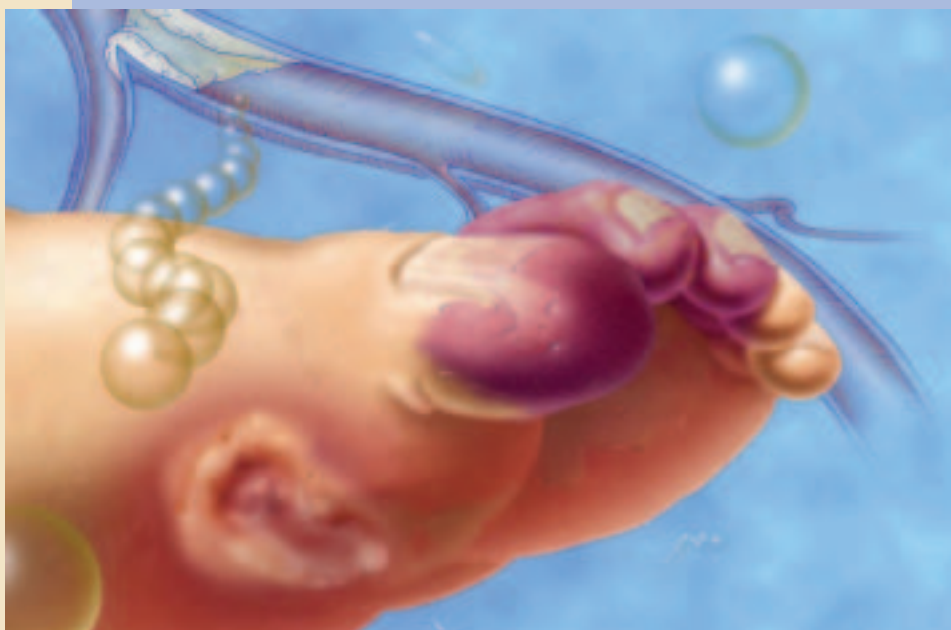


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July 2004

The Diabetic Foot Ulcer: *Management and Prevention Strategies in Primary Care*



CME-CE Certified Activity

Sponsored by the University of Medicine & Dentistry of New Jersey (UMDNJ)–Center for Continuing and Outreach Education



Release Date: July 2004
Expiration Date: June 30, 2005

Nursing credit for this activity will be provided through June 30, 2005.

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The Diabetic Foot Ulcer: Management and Prevention Strategies in Primary Care

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The Diabetic Foot Ulcer: *Management and Prevention Strategies in Primary Care*

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Center for Continuing and Outreach Education



Release Date: July 2004 • Expiration Date: June 30, 2005



This activity is supported by an unrestricted educational grant from Novo Nordisk.

Introduction:

The purpose of this activity is to educate health care providers on the management and prevention of diabetic foot ulcer.

Target Audience:

This activity is designed for primary care practitioners, nurses, and pharmacists.

Learning Objectives:

Upon completion of this activity, participants should be able to:

- Discuss the causes and risk factors of foot ulcer in diabetic patients
- Determine the severity of foot ulcer in a particular patient
- List application of appropriate treatment
- Describe measures to prevent occurrence of foot ulcer

Method of Instruction:

Participants should read the learning objectives and review the activity in its entirety. After reviewing the material, complete the post-test/self-assessment test consisting of a series of multiple-choice questions.

Upon completing this activity as designed and achieving a passing score of 70% or more on the post-test, participants will receive a CME-CE credit letter awarding AMA/PRA category 1 credit, nursing continuing education credit, pharmacy continuing education credit, and the test answer key four (4) to six (6) weeks after receipt of the post-test, registration, and evaluation materials.

Estimated time to complete this activity as designed is 1.0 hour.

Physician Accreditation:

UMDNJ–Center for Continuing and Outreach Education is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

UMDNJ–Center for Continuing and Outreach Education designates this educational activity for a maximum of 1.0 category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.


The activity was prepared in accordance with the ACCME Essentials.

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UMDNJ–Center for Continuing and Outreach Education is an approved provider of continuing education by the New Jersey State Nurses Association (NJSNA), Provider Number P173-9/2003-2006. Provider Approval is valid through June 30, 2005. NJSNA is accredited by the ANCC Commission on Accreditation. This activity is approved for 1.0 contact hours.

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The Diabetic Foot Ulcer

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Drs Angelo, Nam, and Vamos, and Ms Caputo have no significant financial relationships to disclose.

Field Tester Disclosure Declarations:

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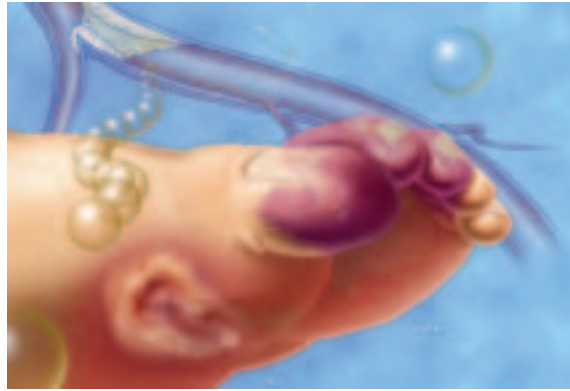
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The Diabetic Foot Ulcer: Management and Prevention Strategies in Primary Care

Foot ulcers are a major cause of morbidity and mortality in patients with diabetes. Health care providers who appreciate the scope of the problem and have a thorough understanding of the pathophysiology of diabetic neuropathy and known risk factors can prevent many of these foot ulcers and their complications. Instructing patients to observe for first signs of foot ulcers can lead to early intervention and prevention of complications. Treatment of established lesions depends on wound characteristics, causative organisms, and comorbidities and may require topical or systemic antimicrobial therapy and/or surgical intervention.

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Most recent estimates by the Centers for Disease Control and Prevention in Atlanta point to a prevalence of 18.2 million people in the United States with diabetes,¹ approximately 15% of whom will have a foot ulcer—commonly referred to as “diabetic foot”—in their lifetime.² Frequently limb threatening, as many as 14% to 24% of diabetic patients with a foot ulcer will require amputation.³ The vast majority of diabetic foot ulcers are caused by vascular and mechanical factors in conjunction with diabetic neuropathy. Diabetic neuropathy is apparent as damage to the sensory, motor, and autonomic nervous systems and is present in approximately 30% of the diabetic population.² The economic impact of the compli-

cations of diabetic neuropathy is considerable. In 2001, approximately \$10.9 billion was spent on diabetic neuropathy and associated complications, up to 27% of total medical costs of diabetes.³ This article discusses risk factors for the development of foot ulcers, the pathogenesis of diabetic neuropathy, foot ulcer classification, and management and prevention strategies.

Who Is at Risk for Diabetic Foot Ulcer?

Factors associated with an increased risk for foot ulcers include having diabetes for more than 10 years, male gender, poor blood glucose control, and coexistent cardiovascular, retinal, or renal complications.³ Specific foot-related conditions also increase the risk for foot ulcerations, including peripheral neuropathy with loss of protective sensation, altered foot biomechanics in the presence of neuropathy, bony deformity, significant peripheral vascular disease, history of ulcers or amputation, and severe nail pathology.

Pathogenesis of Diabetic Neuropathy

The pathogenesis of diabetic neuropathy involves a complex interre-

relationship between metabolic and ischemic factors and nerve repair mechanisms (Figure 1). Major emphasis has been placed on the polyol pathway, as depicted in Figure 2. Sorbitol, which appears to function as a tissue toxin, has been implicated in the development of neuropathy, retinopathy, nephropathy, and aortic disease.^{4,5} Under physiologic concentrations of substrate, aldose reductase has a low affinity for glucose, and little sorbitol is produced. However, in the presence of profound and chronic hyperglycemia, much greater amounts of sorbitol are produced. In an experimental model of diabetic neuropathy, sorbitol accumulation was associated with a decrease in myoinositol content, abnormal phosphoinositide metabolism, and a decrease in $\text{Na}^+\text{-K}^+\text{-ATPase}$ activity.⁴ The primacy of the polyol pathway in the initiation of neuropathy is supported by evidence showing that inhibition of aldose reductase corrects the level of myoinositol in nerves and restores full $\text{Na}^+\text{-K}^+\text{-ATPase}$ activity.⁶

Another factor of pathogenic importance is the glycation of serum and tissue proteins—such as plasma albumin, lens protein, fibrin, collagen, and lipoproteins—from chronic hyperglycemia.⁷ In this process, excess glucose combines with free amino acids on serum or tissue proteins, initially forming reversible, early glycated products and later, irreversible, advanced glycation end products (AGE).^{7,8} Receptors for AGE are present on macrophages and endothelial cells, and the binding of AGE to its receptors may induce the synthesis and release of cytokines, vascular adhesion molecules, endothelin-1, and tissue factor. AGE may also decrease endothe-

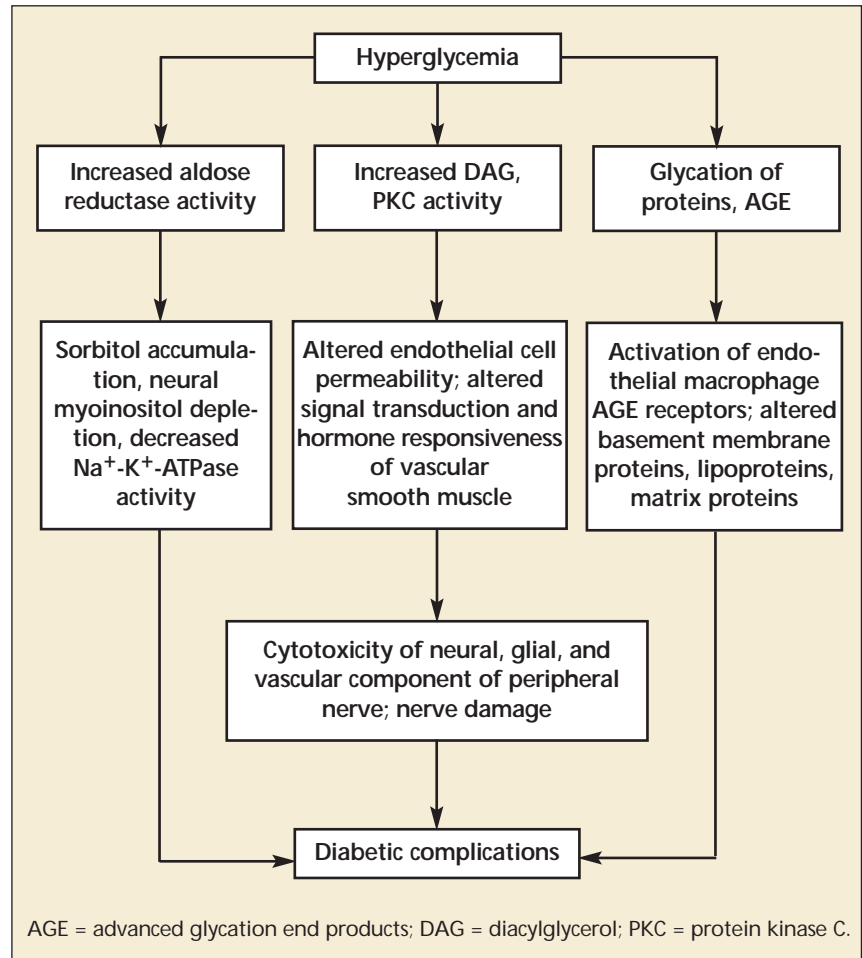


Figure 1—Pathogenesis of diabetic neuropathy. Adapted from Rose BD, McCulloch DK. Glycemic control and vascular complications in type 1 diabetes mellitus. *UpToDate*. www.uptodate.com.

lial-derived nitric oxide as well as alter basement membrane proteins, lipoproteins, and cellular matrix.

Activation of vascular protein kinase C (PKC) isoforms also appears to be important to the development of diabetic neuropathy. PKC activities are increased in the glomeruli, retina, aorta, and heart of diabetic animals.⁹ Heightened activity is thought to be caused by enhanced de novo synthesis of diacylglycerol (DAG), a major endogenous activator of PKC.¹⁰ Evidence shows that activated PKC increases levels of messenger ribonucleic acid encoding matrix components in

glomeruli and produces many of the vascular abnormalities induced by high glucose levels.⁸

Causes of Foot Ulcer

Pathophysiologic factors involved in the development of diabetic foot ulcers are neuropathy, arterial insufficiency, musculoskeletal abnormalities, and poor wound healing (Figure 3). Microbial pathogens, which will be discussed later, also play a key role. Additionally, poor nutrition compromises the healing process. Therefore, especially in elderly patients or those with other comorbidities, a nutri-

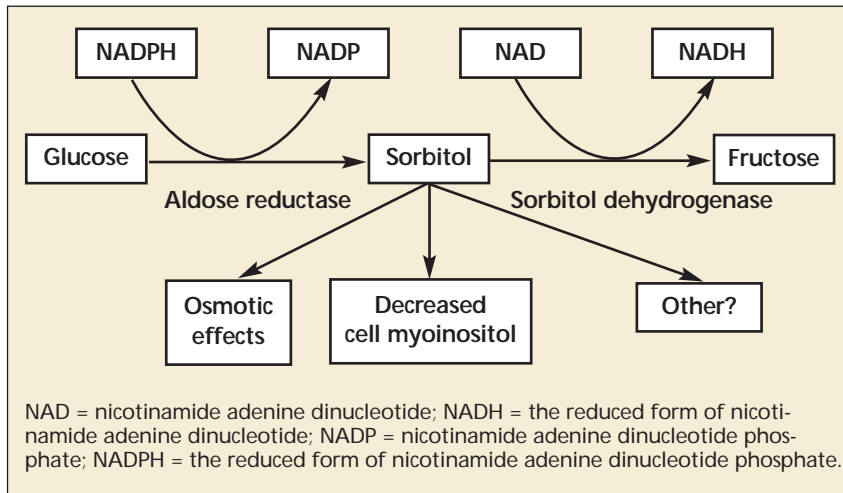


Figure 2—Role of sorbitol in diabetic microvascular disease. Adapted from Frank RN. On the pathogenesis of diabetic retinopathy. A 1990 update. *Ophthalmology*. 1991;98: 586-593.

tionist should be consulted to provide guidance and assist with food choices and supplements as needed.

Neuropathy

The cytotoxic, ischemic/hypoxic effects of sorbitol, AGE, and DAG/PKC are thought to disrupt signal transduction in the peripheral nerves. Malfunction of the sensory nervous system leads to a segmental demyelination process in type-A sensory fibers, which are involved with proprioception and the sensations of

activation of signal transduction along these pathways, leading to the pain and paresthasias of diabetic neuropathy, which, coupled with lack of sensation, leaves the patient with an ataxic gait. The clinical consequence of these disrupted fibers is the loss of crucial protective sensation, which heightens susceptibility to mechanical, chemical, or thermal injury.¹¹ The trauma can remain undetected, resulting in inflammation, further mechanical injury, and ulceration.¹²

typical foot posture in peripheral neuropathy, shifts the foot position so that the toes bear less weight and the metatarsal heads become more prominent. The metatarsal-phalangeal joints can become unstable secondary to wasting of the lumbrical and interosseous muscles, and overpowering extrinsic muscles can depress the metatarsal heads, contract the digits, and cock up the toes.¹¹ These changes increase weightbearing on the metatarsal heads and are manifested by a dysfunctional gait. As the pathologic process continues, the soft tissue covering the bones is exposed to abnormal compressive and shearing stress as it rubs against footwear during walking. With continuing exposure to shearing stress, the skin forms callus. Although this is initially a protective response, as time progresses, all that remains between the callus and the affected bone is a thin layer of tissue that is subjected to additional shearing stress that may destroy tissue and cause inflammation, bleeding, and eventual ulcer formation.¹²⁻¹⁴

Finally, the autonomic nervous system directly influences peripheral circulation in the extremities by supplying the sympathetic adrenergic fibers that regulate arteriole vasomotor tone and blood flow through arteriovenous shunts.¹⁵ Failure of sympathetic control results in arteriolar vasodilatation, which reduces peripheral resistance, increases arteriovenous shunt flow, and increases cutaneous blood flow at the expense of perfusion of the deeper structures. The greater cutaneous flow is responsible for the frequently encountered elevated foot temperatures and distended dorsal veins observed in patients with diabetic neuropathy. Blood that is bypassing the capil-

light touch, pressure, and vibration. The type-C sensory fibers, which are associated with free nerve endings that sense noxious, painful, and thermal stimuli, are similarly affected.¹¹ Damage to these fibers may initially result in pathologic

In persons with diabetic neuropathy, the small intrinsic muscles of the foot atrophy as a result of demyelination in distal motor nerves. This leads to an imbalance of the flexor and extensor muscles and clawing or curling of the toes. Clawing, a

In persons with diabetic neuropathy, the small intrinsic muscles of the foot atrophy as a result of demyelination in distal motor nerves.

The Diabetic Foot Ulcer

lary bed via the arterio-venous shunting may increase capillary pressure and neuropathic edema in the foot.^{16,17} This edema can further exacerbate foot pressures and contribute to ulceration.

Adding to the problems caused by autonomic dysfunction is diabetic anhidrosis. This represents a sudomotor impairment characterized by decreased sweating that results in dry, scaly, cracked skin, which facilitates the introduction of infectious agents.¹⁸

Arterial insufficiency

In diabetes, ischemia secondary to vascular disease interferes with healing by limiting the supply of oxygen, nutrients, and the cellular and soluble mediators involved in the repair process.¹⁹ Blood flow to the foot is decreased, primarily because of atherosclerotic obstruction of the major conduit vessels, characteristically involving the tibial and peroneal vessels, while sparing those in the pedal arch. Microvascular dysfunction adds to the vascular problems.²⁰ Defective hyperemic responses and endothelial dysfunction may also be important in the pathogenesis of foot ulcers in patients with diabetic neuropathy. A gradient of oxygen tissue pressure is required for fibroblast growth and the initiation of angiogenesis, while chronic hypoxia impairs wound healing.²

Musculoskeletal abnormalities

Diabetic patients are susceptible to musculoskeletal abnormalities of the foot, such as neuropathic arthropathy, previously known as Charcot's foot. Neuropathic arthropathy is characterized by chronic, progressive, degenerative disease of 1 or more joints and is

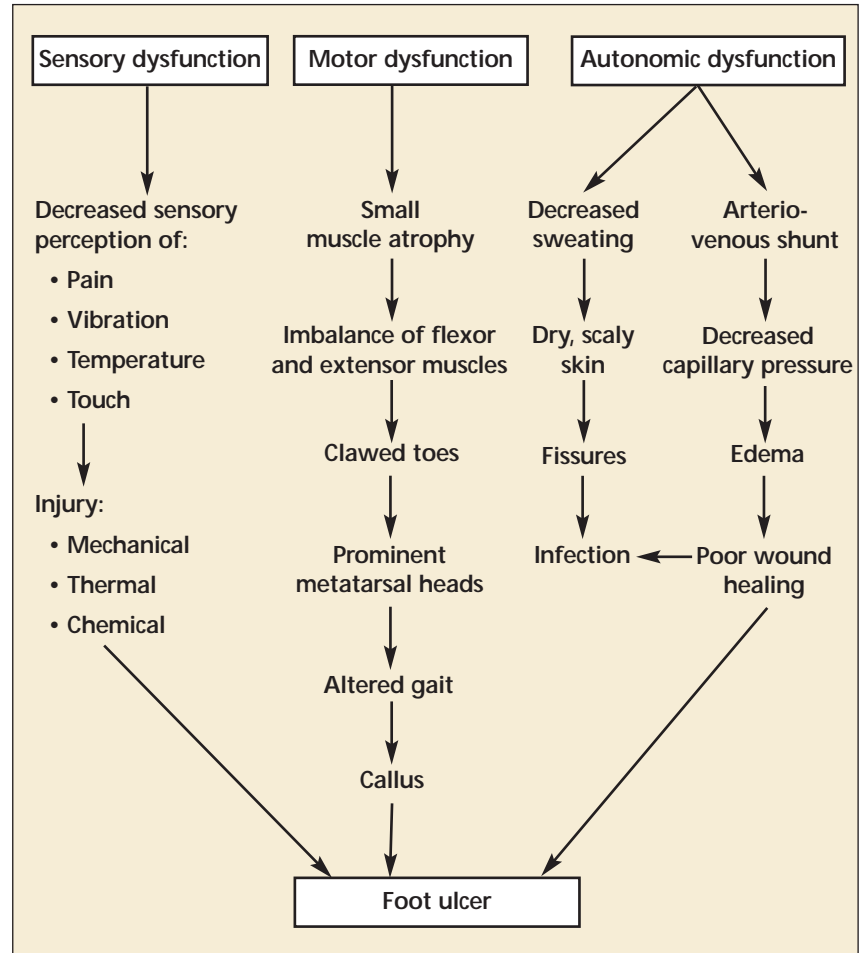


Figure 3—Pathophysiology of foot ulcers from diabetic neuropathy. Adapted from Zangara GA, Hull MM. Diabetic neuropathy: pathophysiology and prevention of foot ulcers. *Clin Nurse Spec*. 1999;13:57-65.

identified by swelling, bleeding, increased temperature, bone changes, and joint instability. This disease process likely stems from recurring trauma secondary to loss of pain and proprioception or from the previously described autonomic neuropathy that shunts blood to the skin away from the deep tissues and results in osteopenia.²¹ The end result of these musculoskeletal abnormalities is improper loading of the joints, uneven weight distribution, and repeated trauma.²¹

Poor wound healing

The biology of wound healing can be thought of as a succession of

unique cellular and physiologic events.²² Normally, at the time of injury, blood vessels rupture, exposing matrix proteins and leading to platelet aggregation, clot formation, and hemostasis.² Platelets release cytokines and growth factors that stimulate further proliferation of the clot and the recruitment and mitogenesis of cellular elements.² Neutrophils are recruited within minutes to hours of the injury, and more mediators and chemotactic substances are released. Subsequently, monocytes are activated to form tissue macrophages, which play a critical role in suppressing bacterial growth and clearing exis-

tent bacteria and necrotic tissue.² Macrophages continue releasing cytokines and growth factors, which bring fibroblasts and endothelial cells to the wound.² Reperfusion depends on angiogenesis, which, in turn, depends on the migration of endothelial cells to the site of injury.² The clonal expansion of cells, particularly fibroblasts, appears to define wound healing. Wound closure, occurring from the rim of the wound inward, is characterized by complete epithelialization. The end of healing is heralded by the migration of keratinocytes to the wound site. During this remodeling phase, tensile strength and cellular organization improve, skin integrity returns, and the wound contracts.²

Chronic hyperglycemia in diabetic patients disrupts the normal cellular and inflammatory pathways involved in wound healing and increases susceptibility to infection.⁴ Individuals with diabetes have been found to have abnormal cellular function, particularly of fibroblasts and neutrophils.² In addition, AGE alters endothelial and macrophage activation, which interferes with cytokine production and cellular migration. AGE may also be detrimental to basement membrane proteins, extracellular matrix production, and endothelial-derived nitric oxide production. There is also evidence to suggest that increased DAG and PKC activity changes endothelial cell permeability, signal transduction, and the hormone responsiveness of vascular smooth muscle.^{9,10}

Clinical Presentation

Common sites of foot ulcers are the metatarsal heads and distal phalanx, medial and lateral mid-foot, and the heel. Various methods

of grading the severity of diabetic foot infection have been used. Joshi and colleagues recently described a useful classification system that divides ulcers into those that do and do not threaten loss of limb.²³

Mild ulcers

Mild, non-limb-threatening ulcers are often shallow lesions with a clean base and less than 2 cm of surrounding cellulitis, with no evidence of fasciitis, abscess, or osteomyelitis. No ischemic process is involved, and the patient has good blood glucose control. Initial treatment includes oral antibiotics and wound care. The causative organisms for these ulcers are primarily aerobic gram-positive cocci (eg, *Staphylococcus aureus* and *streptococci*).²³



Mild ulcer.

Severe ulcers

Limb-threatening ulcers are those characterized by deep infection and more than 2 cm of cellulitis. Such ulcers involve ischemia, and their metabolic control is poor. At presentation these foot ulcers often have evidence of fasciitis or frank abscess formation. X-ray films of the foot may demonstrate periosteal lifting associated with osteomyelitis. Palpation of bone with a metal probe would also signify osteomyelitis. If the situation is unclear as to the presence of an

infectious process involving the bone, an MRI should be obtained.

Diabetic patients presenting with this type of ulcer should be hospitalized immediately for intravenous antibiotic therapy and surgical consultation. The causative organisms of such ulcerations are characteristically polymicrobial— aerobic gram-positive cocci, strict anaerobes (eg, *Bacteroides fragilis*), or gram-negative bacilli (eg, *Escherichia coli*).



Severe ulcer.

Treatment

Localized care

Localized care of diabetic foot ulcers consists of topical antimicrobial therapy, appropriate dressing application, foot elevation, debriding agents, and limitation of weight-bearing activities.²⁴ The success of localized care depends on a high degree of compliance by the patient and caregiver. Topical antimicrobial agents, such as silver sulfadiazine (eg, Silvadene cream, Thermazene) and mupirocin (Bactroban), can stimulate wound healing by eliminating bacteria on the wound surface. They should be used, however, only as ad-

junctional therapy. The use of povidone-iodine solutions (Betadine) and ointments (Betadine ointment) on healing tissue remains controversial. Foot soaks and lubricating lotions have been recommended in the past, but their efficacy has not been proven in controlled trials.

Chemical or enzymatic debriding agents are reserved for patients with mild ulcers (as defined above) or those who refuse surgical debridement. Options for topical debriding agents include collagenase (Santyl) or papain-urea (Accuzyme, Panafil).

Becaplermin gel (Regranex) has been found to significantly increase the incidence of complete wound closure and significantly reduce the time to complete closure of diabetic foot ulcers.²⁵

A moist environment is most conducive to formation of a nascent tissue matrix. Several existing products promote maintenance of a moist wound. A recent *Cochrane Review* examining 3 randomized controlled trials of hydrogel dressing products found a significant improvement over saline dressings alone.²⁶

Larval therapy with sterile maggots has been described in patients failing other types of therapy for debridement of necrotic tissue.²⁷

Surgical intervention

Surgery, which can consist of drainage, debridement, and often amputation, is important for preserving tissue, maximizing revascularization, and promoting healing.²⁸ The goal of early, aggressive debridement and drainage is to remove all necrotic soft tissue and bone.² Even for patients with poor circulatory status, it is important to establish dependent drainage to prevent pooling of pus. There is no

evidence to support soaking an ulcerated foot in a whirlpool or other hydrotherapies.² In fact, such measures could result in maceration, infection, or burns. The decision to perform surgery must be made with caution because, while the intervention can remove the source of infection, too much manipulation can impair healing by further damaging the tissues.²⁸

Intravenous antimicrobial therapy is initiated when systemic or extensive infection is suspected, most commonly in patients who have developed septicemia or osteomyelitis.²⁹ The recommended agents include a beta-lactam plus a beta-lactamase inhibitor (eg, ampicillin sodium/sulbactam sodium [Unasyn]) or clindamycin plus a gram-negative drug (eg, a third-

Diabetic patients must be instructed about proper and consistent foot care. Feet should be kept clean and dry at all times. Patients with neuropathy should not walk barefoot, even in the home. Properly fitted shoes are essential.

Systemic antimicrobial therapy

The choice of antimicrobial agent is based on the suspected bacterial flora, appearance of the infected site, history of the lesion, and general condition of the patient. It is important to use drugs that are bactericidal, since diabetic patients have poor immune defenses, and if the pathogen is not eliminated, the infection will recur. Oral antimicrobial therapy can be used to treat mild, superficial lesions when the infection is in the early stages, drainage is minimal, gangrene is absent, and the patient does not have systemic symptoms. More severe ulcerations will require intravenous antimicrobial therapy.²⁹

Recommended oral agents include cephalexin (Biocef, Keflex), clindamycin (Cleocin), and amoxicillin/potassium clavulanate (Augmentin).²⁹

generation cephalosporin, a fluoroquinolone, or aztreonam [Azactam]). Caution should be used with fluoroquinolones in diabetic patients because these medications may worsen glycemic control. Vancomycin (Vancocin, Vancoled) plus imipenem-cilastatin (Primaxin) is used for life-threatening infections.²⁹ Linezolid has been recently evaluated and has shown promise as an alternative therapy. However, there appears to be no benefit of linezolid over ampicillin/sulbactam. The cost of linezolid often will limit its availability, especially in an outpatient setting.³⁰

The recommended length of therapy for soft tissue infection is 2 to 3 weeks, for osteomyelitis it is 4 to 6 weeks.^{24,31} Ultimately, the length of treatment depends on the severity of the infection, the causative organism, and the clini-

cal response to the chosen medication. It is important to continue antibiotic therapy until an infection has been eliminated.

Prevention Is Key

Glycemic control is paramount in the prevention of diabetic neuropathy and the development of diabetic foot ulcers. A chronically elevated blood glucose concentration is the initial factor that sets in motion the pathogenic process of neuropathy.

Diabetic patients must be instructed about proper and consistent foot care to prevent ulcers. Feet should be kept clean and dry at all times. Patients with neuropathy should not walk barefoot, even in the home. Properly fitted shoes are essential. This is a particular problem with women, since an adequate shoe is not often stylish. Patients should be told to inspect their feet carefully daily for callus, infection, abrasions, foreign bodies, or blisters and to consult the physician about any potentially troublesome lesion.

The National Diabetes Education Program recommends that all patients with diabetes have a thorough foot examination at least annually to assess the condition of the skin and nails and to evaluate for the presence of any sensory or skeletal abnormalities.³² Proprioception and standardized sensory evaluation should be performed to help identify patients at risk. Standardized sensory evaluation is best accomplished with the Semmes-Weinstein monofilament at various locations of the foot, including the toes, the metatarsal head area, and the heel. "High-risk" patients are defined as those who lack palpable pedal pulses, have abnor-

malities of protective sensation, foot deformities, and a history of foot ulcers or amputations; they should have a visual foot inspection at every visit. High-risk patients should also be referred to the appropriate foot care specialist.

Patients with diabetes and high-risk foot conditions should be educated regarding their risk factors and appropriate management. A nonjudgmental assessment of a person's current knowledge and care practices should be obtained first. Patients at risk should understand the implications of the loss of protective sensation, the importance of foot monitoring on a daily basis, the proper care of the foot, including nail and skin care, and the selection of appropriate footwear. The patient's understanding of these issues and their physical ability to conduct proper foot surveillance and care should be assessed. Patients with neuropathy should be advised to break in new shoes gradually to minimize the formation of blisters and ulcers. Patients with visual difficulties, physical constraints preventing movement, or cognitive problems that impair their ability to assess the condition of the foot and to institute appropriate responses will need other people, such as family members, to assist in their care. Patients at low risk may benefit from education on foot care and footwear.³³

Conclusion

Peripheral diabetic neuropathy affects nearly one third of all patients with diabetes. Foot ulceration is a preventable complication in those with peripheral neuropathy. Better understanding of the pathophysiologic basis of foot ulcers can

assist health care providers in the evaluation, diagnosis, and treatment of diabetic foot ulcers. It is also critical to provide thorough, regular foot assessments with standardized sensory evaluations, skin assessment, and bony alignment evaluation as well as patient and family education. If begun early, these interventions for health promotion and disease prevention can help patients identify the early signs and symptoms of diabetic foot ulcers before extensive complications occur, thereby improving their quality of life, preventing pain and amputation, and reducing the health care costs of this chronic disease. ■

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CME-CE Questions for The Diabetic Foot Ulcer

- During a routine examination, it is determined that Mr Smith, a patient with long-standing diabetes, has developed a foot ulcer. It is a 1-cm shallow ulcer on the plantar aspect of the foot under the first metatarsal head. There appears to be a clean base with 1.5 cm of surrounding cellulitis. Which of the following is NOT an appropriate initial choice of management for this patient?
 - cephalexin
 - clindamycin
 - linezolid
 - mupirocin topical
 - amoxicillin/clavulanate
- Ms Johnson is a 52-year-old female diagnosed with type 2 diabetes 7 years ago. She notes that she has had variable control over her diabetes in the past, although her hemoglobin A_{1c} has been excellent for the past 4 years. Which of the following would classify this patient as “high risk” for development of a diabetic foot ulcer?
 - absence of palpable pedal pulses
 - abnormalities of sensation via the Semmes-Weinstein monofilament test
 - presence of musculoskeletal foot deformities
 - previous history of foot ulcers
 - all of the above
- The patient in question 2 is determined to have “low-risk” feet by your history and physical examination. In the absence of other complaints, the most appropriate management of this patient is:
 - arterial evaluation via the ankle-brachial index
 - proper education of the patient regarding appropriate footwear, daily foot inspection, appropriate nail care, and foot hygiene
 - thorough annual foot examination
 - all of the above
 - b and c only
- Of the 18.2 million patients in the United States with diabetes mellitus, what percentage exhibit signs or symptoms of peripheral neuropathy?
 - 5%
 - 15%
 - 30%
 - 45%
 - greater than 50%
- Severe or limb-threatening diabetic foot ulcers are characterized by all of the following EXCEPT:
 - singular microbial organism predominance
 - more than 2 cm of surrounding cellulitis
 - deep wounds with evident fasciitis
 - arterial insufficiency
 - abscess formation

CME-CE Questions CONTINUED

6. When choosing an antimicrobial agent for the treatment of a diabetic foot ulcer, it is important to consider:
- history of the ulcer
 - bacterial flora
 - appearance of the wound site
 - overall health of the patient
 - all of the above
7. Failure of appropriate oral antimicrobials and topical treatments often results in hospitalization of patients with diabetic foot ulcers. A multidisciplinary approach to these recalcitrant infections is best. Which of the following is NOT part of the initial evaluation and management of the severe diabetic foot ulcer?
- x-rays of the affected foot
 - surgical debridement to uncover the extent of the infection
 - arterial evaluation of the lower extremity
 - single-drug therapy with cefazolin
 - All of the above are appropriate for the initial evaluation and management of patients with a severe diabetic foot ulcer.
8. All of the following are true regarding localized care for diabetic foot ulcers EXCEPT:
- Mild ulcers may respond to chemical or enzymatic debriding agents such as papain-urea.
 - Topical antimicrobials (eg, silver sulfadiazine) stimulate wound healing by eliminating bacteria.
 - Foot soaks have proved to be effective for the treatment of foot ulcers because they promote wound healing.
 - Localized care is most successful when patients and caregivers are extremely compliant in their use.
 - Patients should be restricted from weightbearing activities and appropriate footwear should be assessed.
9. Patients with diabetes complicated by neuropathy should be taught:
- to visually inspect their feet daily
 - never to walk barefoot, even in the house
 - to soak their feet to aid in healing foot ulcers
 - options a and b
 - all of the above
10. Patients with diabetes at risk for foot ulcers should understand:
- the implications of the loss of protective sensation
 - the importance of foot monitoring on a daily basis
 - the proper care of the foot, including nail and skin care
 - the selection of appropriate footwear
 - all of the above

The Diabetic Foot Ulcer



University of Medicine & Dentistry of New Jersey Center for Continuing and Outreach Education

The Diabetic Foot Ulcer: *Management and Prevention Strategies in Primary Care*

REGISTRATION FORM

There is no charge for this CME-CE activity.

In order to obtain credit, participants are required to:

- (1) Read the learning objectives, review the activity, and complete the self-assessment quiz.
- (2) Complete this registration form and the activity evaluation form on the following page, and record your test answers in the box below.
- (3) Send the registration and evaluation forms to:
UMDNJ-Center for Continuing and Outreach Education
via mail: PO Box 1709, Newark, NJ 07101-1709
via fax: 973-972-7128
- (4) Retain a copy of your test answers. Your answer sheet will be graded and if a passing score of 70% or more is achieved, a CME-CE credit letter awarding AMA/PRA category 1 credit, nursing continuing education credit, pharmacy continuing education credit, and the test answer key will be mailed to you within four (4) to six (6) weeks. Individuals who fail to attain a passing score will be notified and offered the opportunity to complete the activity again.

SELF-ASSESSMENT TEST

Circle the best answer for each question on pages 10-11.

- | | | | | | | | | | | | |
|----|---|---|---|---|---|-----|---|---|---|---|---|
| 1. | A | B | C | D | E | 6. | A | B | C | D | E |
| 2. | A | B | C | D | E | 7. | A | B | C | D | E |
| 3. | A | B | C | D | E | 8. | A | B | C | D | E |
| 4. | A | B | C | D | E | 9. | A | B | C | D | E |
| 5. | A | B | C | D | E | 10. | A | B | C | D | E |

REGISTRATION

First Name	M.I.	Last Name	Degree
Daytime Phone		Evening Phone	
Fax	E-mail		
Preferred Mailing Address <input type="checkbox"/> Home <input type="checkbox"/> Business			
City	State	Zip Code	
Affiliation, Specialty			

I attest that I have completed the activity "The Diabetic Foot Ulcer: Management and Prevention Strategies in Primary Care" as designed and I am claiming 1.0 AMA/PRA category 1 credit.

Signature

Date

Credit for this activity is available until June 30, 2005.

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University of Medicine & Dentistry of New Jersey
Center for Continuing and Outreach Education

The Diabetic Foot Ulcer: *Management and Prevention Strategies in Primary Care*

ACTIVITY EVALUATION FORM

The planning and execution of useful and educationally sound continuing education activities are guided in large part by input from participants. To assist us in evaluating the effectiveness of this activity and to make recommendations for future educational offerings, please take a few moments to complete this evaluation form. Your response will help ensure that future programs are informative and meet the educational needs of all participants. **Please note: CE credit letters and long-term credit retention information will only be issued upon receipt of this completed evaluation form.** Thank you for your cooperation!

PROGRAM OBJECTIVES: Having completed this activity, are you better able to:

	Strongly Agree				Strongly Disagree
• Discuss the causes and risk factors of foot ulcer in diabetic patients	5	4	3	2	1
• Determine the severity of foot ulcer in a particular patient	5	4	3	2	1
• List application of appropriate treatment	5	4	3	2	1
• Describe measures to prevent occurrence of foot ulcer	5	4	3	2	1

OVERALL EVALUATION:

	Strongly Agree				Strongly Disagree
The information presented increased my awareness/understanding of the subject.	5	4	3	2	1
The information presented will influence how I practice.	5	4	3	2	1
The information presented will help me improve patient care.	5	4	3	2	1
The faculty demonstrated current knowledge of the subject.	5	4	3	2	1
The program was educationally sound and scientifically balanced.	5	4	3	2	1
The program avoided commercial bias or influence.	5	4	3	2	1
Overall, the program met my expectations.	5	4	3	2	1
I would recommend this program to my colleagues.	5	4	3	2	1

If you anticipate changing one or more aspects of your practice as a result of your participation in this activity, please provide us with a brief description of how you plan to do so.

Please provide any additional comments pertaining to this activity (positives and negatives) and suggestions for improvement:

Please list any topics that you would like to be addressed in future educational activities:

FOR PHARMACY PROFESSIONALS ONLY:

Please explain how this activity did or did not meet the needs of the pharmacy community for this disease.

Please comment on the aspects of this activity that contributed toward improving your professional effectiveness and ability to communicate with and counsel patients and/or monitor patient disease status.

Please list any specific areas of the activity that could be improved to assist us in providing valuable education to the pharmacy community.
