Foot Care

Diabetes Canada Clinical Practice Guidelines Expert Committee

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KEY MESSAGES

• Lower extremity complications are a major cause of morbidity and mortality in people with diabetes.
• The treatment of foot ulcers in people who have diabetes requires an interprofessional approach that addresses glycemic control, infection, off-loading of high-pressure areas, lower-extremity vascular status and local wound care.
• Antibiotic therapy is not required for uninfected neuropathic foot ulcers.
• Proprietary adjunctive wound dressings and technologies, including antimicrobial dressings, lack sufficient evidence to support routine use in the treatment of neuropathic ulcers.

KEY MESSAGES FOR PEOPLE WITH DIABETES

• Diabetes can cause nerve damage (also known as “diabetic peripheral neuropathy”) and poor blood flow to the legs and feet (also known as “peripheral arterial disease”).
• As a result, people with diabetes are less likely to feel a foot injury, such as a blister or cut. Diabetes can make these injuries more difficult to heal. Unnoticed and untreated, even small foot injuries can quickly become infected, potentially leading to serious complications.
• A good daily foot care routine may help keep your feet healthy:
  ◦ Examine your feet and legs daily
  ◦ Care for your nails regularly
  ◦ Apply moisturizing lotion if your feet are dry (but not between the toes)
  ◦ Wear properly fitting footwear
  ◦ Test your bath water with your hand before you step in, to make sure the water is not too hot
• If you have any corns (thick or hard skin on toes), calluses (thick skin on bottom of feet), ingrown toenails, warts, splinters or other wounds, have them treated by your doctor or other foot care specialist (such as a foot care nurse, podiatrist or chiropodist). Do not try to treat them yourself.
• If you have any swelling, warmth, redness or pain in your legs or feet, see your health-care provider or foot specialist right away.

Introduction

Foot complications are a major cause of morbidity and mortality in people who have diabetes, and contribute to increased health care use and costs (1–7). People with diabetes who have peripheral neuropathy and peripheral arterial disease are at risk of developing foot ulcers and infection that may lead to lower-extremity amputation (8–11). The frequency of amputation is much higher in people with diabetes than people without diabetes (12,13). This is especially true in developed nations, such as Canada, where adults with diabetes have 20-fold greater likelihood of being hospitalized for nontraumatic lower limb amputation than adults without diabetes (14). In the United States, the frequency of lower-extremity amputation decreased by 28.8% from 2000 to 2010, but the use of other orthopedic treatments for diabetic foot ulcers increased by 143% during this period (15). Preventive measures, foot care education, and early and aggressive treatment of diabetic foot problems are important components of diabetes care.

Risk Assessment

Risk factors for developing foot ulcers in people with diabetes include peripheral neuropathy, previous ulcer or amputation, structural deformity, limited joint mobility, peripheral arterial disease, microvascular complications, increased levels of glycated hemoglobin (A1C) and onychomycosis (16,17). Loss of sensation to the 10 g Semmes-Weinstein monofilament at the plantar surface of the foot is a significant and independent predictor of future foot ulcer and lower-extremity amputation (18–20).

Several wound classifications have been developed to provide objective assessment of foot ulcer severity. The simple Wagner classification is used commonly: Wagner Grade 0, skin intact; Grade 1, superficial ulcer; Grade 2, ulcer extending to tendon, capsule or bone; Grade 3, deep ulcer with osteomyelitis or abscess; Grade 4, gangrene of toes or foot; Grade 5, gangrene of midfoot or hindfoot. The University of Texas Diabetic Wound Classification System has been validated as a predictor of serious outcomes in people with diabetes who have foot ulcers (21,22) (Table 1).

In people who have ischemia, the distribution of peripheral arterial disease is greater in the arterial tree below the knee in people with diabetes compared with people without diabetes (23). Non-invasive assessments for peripheral arterial disease in people with diabetes include the blood pressure (BP) ankle-brachial index (ratio of ankle to brachial systolic BP), systolic toe pressure by photoplethysmography, transcutaneous oximetry and Doppler arterial flow studies (24,25). Although the ankle-brachial index in some clinical settings is a readily available and easy-to-perform technique, it may underestimate the degree of peripheral arterial disease.
obstruction because of medial arterial wall calcification in lower-extremity arteries (26,27). Photoplethysmography assesses the intensity of light reflected from the skin surface and red blood cells, which is indicative of arteriolar pulse flow; measurement of systolic toe pressure by photoplethysmography may be more accurate than ankle-brachial index in determining the presence of arterial disease in people with diabetes (28).

It is important to recognize the potential limitations inherent with noninvasive diagnostic tests for peripheral arterial disease (29,30). Other studies that are available for the evaluation of lower-limb ischemia that do not require arterial access include intraarterial digital subtraction contrast arteriography, magnetic resonance angiography and computed tomographic angiography, but these studies may be complicated by contrast-induced renal failure or gadolinium-associated nephrogenic systemic fibrosis (31–35). Consultation with a specialist in vascular medicine or surgery should be undertaken as soon as possible for people who have suspected lower extremity ischemia (30,36).

The foot examination is important and should include footwear assessment (19,37,38) (Table 2). Assessment of skin temperature is important because increased warmth may indicate the presence of inflammation or acute Charcot neuroarthropathy in a foot that has lost protective sensation (39–41). In addition, erythema and swelling may be indicators of cellulitis or Charcot neuroarthropathy (42,43). The clinical and radiographic differentiation between acute Charcot foot and infection may be difficult (44). Plain radiographs have low sensitivity and specificity in differentiating osteomyelitis from Charcot changes. Magnetic resonance imaging (MRI) of the foot may help clarify this differential diagnosis, but no diagnostic imaging studies are definitive, and the results of all imaging studies must be interpreted carefully and correlated with the clinical presentation (45,46).

### Table 1

<table>
<thead>
<tr>
<th>Stage</th>
<th>Grade</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>A</td>
<td>I</td>
<td>Pre- or post-ulcerative lesion completely epithelialized</td>
</tr>
<tr>
<td>B</td>
<td>II</td>
<td>Superficial wound not involving tendon, capsule, or bone</td>
</tr>
<tr>
<td>C</td>
<td>III</td>
<td>Wound penetrating to tendon or capsule</td>
</tr>
<tr>
<td>D</td>
<td></td>
<td>Wound penetrating to bone or joint</td>
</tr>
</tbody>
</table>

* Adapted from reference 21.

### Table 2

<table>
<thead>
<tr>
<th>Element</th>
<th>Parameter</th>
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</thead>
<tbody>
<tr>
<td>Inspection</td>
<td>- Paint morphology (Charcot arthropathy, bony prominences)</td>
</tr>
<tr>
<td></td>
<td>- Foot morphology (clawtoe, hammertoe, number of toes)</td>
</tr>
<tr>
<td></td>
<td>- Skin: blisters, abrasions, calluses, subkeratotic hematomas or hemorrhage, ulcers, absence of hair, toe nail problems, edema, abnormal color</td>
</tr>
<tr>
<td></td>
<td>- Status of nails</td>
</tr>
<tr>
<td></td>
<td>- Foot hygiene (cleanliness, tinea pedis)</td>
</tr>
<tr>
<td>Palpation</td>
<td>- Pedal pulses</td>
</tr>
<tr>
<td></td>
<td>- Temperature (increased or decreased warmth)</td>
</tr>
<tr>
<td>Protective sensation</td>
<td>- Sensation to 10 g monofilament*</td>
</tr>
<tr>
<td>Footwear</td>
<td>- Exterior: signs of wear, penetrating objects</td>
</tr>
<tr>
<td></td>
<td>- Interior: signs of wear, orthotics, foreign bodies</td>
</tr>
</tbody>
</table>

* Adapted from references 19 and 38 to 43.

** See Appendix 12. Monofilament Testing in the Diabetic Foot.

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### Preventive Care and Treatment

Preventive measures against the risk of amputation include regular foot examination, evaluation of amputation risk, regular callus debridement, patient education, professionally fitted therapeutic footwear to reduce plantar pressure and accommodate foot deformities, and early detection and treatment of diabetic foot ulcers (47,48). Many studies that have assessed interventions to prevent and treat diabetic foot ulcers have had limited quality of supportive evidence because of problems in study design and methods (49,50). However, the treatment of foot ulcers typically is most effective with an interprofessional approach and includes measures to improve glycemic control, decrease mechanical pressure with off-loading, treat infection, ensure adequate lower-extremity arterial inflow and provide local wound care (51–55).

Specific recommendations about wound dressing types cannot be made for typical diabetic foot ulcers because there is insufficient evidence to support any type of dressing over another (56–60). The essentials of good wound care include maintaining an optimal wound environment, off-loading pressure from the ulcer and regular debridement of nonviable tissue (58,61,62); wound dressings that maintain a physiologically moist wound environment should be selected. There are insufficient data to support the use of specific dressing types or antimicrobial dressings in the routine treatment of diabetic foot wounds (48,51–59). There is also insufficient evidence to make any recommendation about the role of suction wound dressings (referred to as “negative pressure wound therapy”) in the routine treatment of neuropathic wounds, but there is some evidence in favour of suction wound dressings for more advanced diabetic foot ulcers or after extensive debridement (58,61,63–66). Other adjunctive measures for wound healing, such as topical growth factors and dermal substitutes, have been evaluated for the treatment of diabetic foot ulcers, but the studies have been limited in sample size, duration and follow up, and the results are not sufficiently conclusive to support the use of these therapies (57,58,67–70).

Pressure off-loading may be achieved with temporary footwear until the ulcer heals and the tissues of the foot stabilize. Removable and nonremovable walker boots and total contact casts are effective in decreasing pressure at plantar surface ulcers (71–76). Although total contact casts are effective in supporting the healing of noninfected, nonischemic plantar surface neuropathic ulcers, total contact casting requires careful patient selection and personnel who have specialized training to minimize the risk of developing iatrogenic complications (74,75,77–79). When bony foot deformity prevents the fitting of appropriate footwear or off-loading of pressure-related ulcers, consultation with a surgeon skilled in foot surgery may be considered to evaluate and treat the deformity (80–82).

Treatment of the acute Charcot foot requires immobilization of the foot, typically for several months, in a total contact cast, removable walker boot or custom orthosis until consolidation occurs (63). Surgical stabilization may be indicated for Charcot arthropathy associated with marked instability, deformity or nonhealing ulcers.
Table 3
Empiric antimicrobial therapy for infection in the diabetic foot*

<table>
<thead>
<tr>
<th>Infection Severity</th>
<th>Antimicrobial Agent†‡§</th>
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</table>
| **Localized infections:**  
Neither limb nor life threatening  
Usually associated with cellulitis surrounding an ulcer  
Purulent debris may be present at the base of the ulcer  
Usual organisms: aerobic gram-positive cocci (S. aureus and β-hemolytic streptococci) | • Cloxacillin  
• Amoxicillin-clavulanic acid  
• Cephalaxin  
• SMX-TMP  
• Clindamycin  
• Doxycycline |
| **More extensive infections:**  
• Includes more severe infections, including more extensive cellulitis, plantar abscess and deep space infections  
• The choice of oral or parenteral should be guided by the extent of the infection and the patient’s overall clinical status  
• Initial antimicrobial therapy against staphylococci, streptococci, anaerobes and common Enterobacteriaceae species  
• Empiric treatment targeting P. aeruginosa is generally unnecessary unless risk factors present (e.g. history of foot soaking, severe or chronic infection)  
• Patients who are not toxic may be treated with debridement and oral antimicrobial therapy  
• Patients who are ill or toxic despite moderate local signs are treated as having a severe infection:  
  ◦ Limb or life threatening  
  ◦ Frequently polymicrobial  
  ◦ Immediate hospitalization, early surgical debridement and parenteral antimicrobial therapy  
• If MRSA is present or suspected, consider adding vancomycin, linezolid or daptomycin | • SMX-TMP plus metronidazole or clindamycin  
• Ciprofloxacin or levofloxacin plus clindamycin or metronidazole  
• Amoxicillin-clavulanic acid  
• Mosfloxacin  
• Linezolid  
• Carbapenem |
| **Osteomyelitis:**  
• Treat with intravenous therapy or long-term oral antimicrobial therapy using agents that are well absorbed from the gastrointestinal tract and have good distribution to bone and tissue  
• Surgical debridement indicated to remove necrotic debris, abscess or sequestrum  
• Therapy should be based on culture results whenever possible  
• If MRSA is present or suspected, consider adding vancomycin, linezolid or daptomycin | • Cefoxitin  
• 1st, 2nd or 3rd generation cephalosporin plus metronidazole  
• Piperacillin-tazobactam  
• Clindamycin plus 3rd generation cephalosporin  
• Carbapenem |

* Modified and used with permission from reference 90.
MRSA, methicillin-resistant Staphylococcus aureus; SMX-TMP, sulfamethoxazole-trimethoprim.
† Many of the agents identified in this table do not have Health Canada approval specifically for treatment of diabetic foot infections, including osteomyelitis, but may have an indication for the treatment of skin and soft tissue infections or antimicrobial activity against typical pathogens encountered in osteomyelitis of the diabetic foot.
‡ Duration of therapy is based on clinical response. However, typical treatment courses for skin and soft tissue infections range from 7 (mild) to 21 (severe) days, and the treatment of osteomyelitis may require 4 to 6 weeks of parenteral or several months of oral antimicrobial therapy. Whenever possible, it is desirable to switch to oral antimicrobial therapy to avoid complications from parenteral administration.
§ The agents suggested in this section are for empiric therapy prior to the availability of final culture and susceptibility results. Knowledge of local epidemiology and antimicrobial resistance profiles must also guide therapeutic choices.

Although bisphosphonates have been considered for the treatment of Charcot arthropathy, further studies are necessary to fully evaluate these agents and other medical therapies in the routine treatment of Charcot arthropathy (83–89).

Infection may complicate foot ulcers and may progress rapidly to become limb and/or life threatening (90). When infections begin, the most frequent pathogens typically include Staphylococcus aureus, Streptococcus pyogenes (group A streptococcus) and Streptococcus agalactiae (group B streptococcus). With persistent infection and the presence of devitalized tissue, gram-negative and anaerobic pathogens may cause polymicrobial infection (36,91). Specimens for culture from the surface of wounds are unreliable, and specimens from deeper tissues obtained by debridement are more likely to determine the correct bacterial pathogens for antimicrobial therapy (92–96). Initial therapy typically includes empiric, broad-spectrum antibiotics, and subsequent antibiotic selection is tailored to the sensitivity results of cultured specimens. With the exception of a few antimicrobial agents that have a specific indication for the treatment of diabetic foot infections, most agents available for use are selected for their antibacterial spectrum (36,95–97). Guidelines are available for antimicrobial choices in the empiric treatment of diabetic foot infections (Table 3) (98).

Achieving target glycemic control may be associated with decreased amputation frequency (99). Poor glycemic control may be associated with immunopathy and blunted cellular response to infection. Many people (50%) who have diabetes and a major limb infection may not have fever or leukocytosis at presentation (100). Deep infections require prompt surgical debridement and appropriate antibiotic therapy (36,101).

In medically suitable individuals who have peripheral arterial disease and a history of ulceration or amputation, distal limb revascularization may improve long-term limb salvage. Endovascular techniques with angioplasty and stenting for infrarenal arterial lesions may be effective to achieve limb salvage, but the long-term success is less in people with diabetes than people without diabetes (83,102). A specific evidence-based recommendation about the type of revascularization technique cannot be made, and the preferred method is based on the judgment of the vascular surgeon, in consideration of medical and surgical risks (29,30).

There is limited evidence to confirm an added benefit of hyperbaric oxygen therapy in reducing the indication for amputation or improving wound healing in individuals with diabetes. Therefore, hyperbaric oxygen therapy is not recommended for the routine treatment of infected or noninfected neuropathic or ischemic foot ulcers.
3. There is insufficient evidence to recommend any specific dressing type for typical diabetic foot ulcers [Grade C, Level 3 (103)]. Debridement of nonviable tissue [Grade A, Level 1A (104)] and general principles of wound care include the provision of a physiologically moist wound environment, and off-loading the ulcer [Grade D, Consensus].

5. There is insufficient evidence to recommend the routine use of adjunctive wound-healing therapies (e.g., topical growth factors, granulocyte colony-stimulating factors or dermal substitutes) for typical diabetic foot ulcers. Provided that all other modifiable factors (e.g., pressure off-loading, infection, foot deformity) have been addressed, adjunctive wound-healing therapies may be considered for nonhealing, nonischemic wounds [Grade A, Level 1 (69,70)].

Abbreviations: A1C, glycated hemoglobin; BG, blood glucose; BP, blood pressure; MRI, magnetic resonance imaging.

Other Relevant Guidelines

Targets for Glycemic Control, p. S42 Neuphropy, p. S217

Relevant Appendices


Author Disclosures

No authors have anything to disclose.

References


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**Literature Review Flow Diagram for Chapter 32: Foot Care**

- **Identification**
  - Citations identified through database searches N=5,199
  - Additional citations identified through other sources N=4

- **Screening**
  - Citations after duplicates removed N=3,376
  - Title & abstract screening N=2,214
  - Full-text screening for eligibility N=1,003
  - Full-text reviewed by chapter authors N=283
  - Studies requiring new or revised recommendations N=2

- **Eligibility**
  - Citations excluded* N=1,211
  - Citations excluded* N=720
  - Citations excluded* N=281

- **Included**

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