

Diabetic foot care plan

Gordon Dow MD FRCPC and The Diabetic Foot Care Plan Working Group*

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Diabetes mellitus is the number one cause of limb loss in North America, and is associated with growing, unacceptable rates of morbidity, mortality and economic loss. Approximately 80% of these amputations are preceded by the development of foot ulceration. Various disciplines have studied the prevention and management of foot ulceration in those with diabetes. The present care plan was constructed to incorporate the important contributions from these disciplines into practical therapeutic guidelines. The care plan has been divided into three basic sections: assessment, general management and antibiotic therapy. Each of these sections is described in detail and borrows heavily from previous Canadian position papers. Application of the care plan is illustrated by multiple diabetic foot clinical scenarios, which have been categorized according to the Wagner classification.

Key Words: *Diabetes; Foot infection; Skin ulcer; Osteomyelitis*

Plan de soins pour le pied diabétique

Le diabète sucré est la première cause d'amputation en Amérique du Nord et elle est associée à des taux inacceptables et croissants de morbidité, de mortalité et de perte de revenus. Environ 80 % des amputations sont précédées d'un ulcère du pied. Diverses disciplines se sont penchées sur la prévention et le traitement de l'ulcère pédiéux chez les patients atteints de diabète. Le présent plan de soins a été élaboré de façon à incorporer les importantes contributions de ces diverses disciplines aux directives thérapeutiques pratiques. Le plan de soins se subdivise en trois principales sections : évaluation, prise en charge générale et antibiothérapie. Chacune de ces sections est décrite en détails et emprunte largement aux énoncés de position canadiens antérieurs. L'application du plan de soins est illustrée par de multiples scénarios cliniques de pieds diabétiques qui ont été catégorisés selon les classifications de Wagner.

People with diabetes have a 15% lifetime risk of developing a foot ulcer and have an amputation rate of 6/1000 people with diabetes/year (1). Recent data has suggested that 80% of foot ulcers and/or amputations can be prevented by daily foot examinations by the patient, foot care and appropriate footwear (2). Unfortunately, foot infection continues to be a pervasive problem for people with diabetes living in Canada, where the risk of amputation is 12 to 15 times that of the general population, and amputation is the most common cause of

nontraumatic limb loss (3). This morbidity is compounded by the fact that of people with diabetes, 30% require amputation of the remaining limb at three years and 50% at four years after the first amputation (4). The mortality rate of diabetic amputees is also increased, with a three-year survival rate of 50% (5).

Extremity complications in people with diabetes are complex in their etiology and management, and hence do not fall under the purview of one specific medical discipline. The objective of the present paper, therefore, was to design a concise

Section of Infectious Diseases, Department of Internal Medicine, The Moncton Hospital, Moncton, New Brunswick

**Ms Sylvia Fournier, CHUM, Campus Hotel-Dieu, Montreal, Québec; Ms Lisa Huatkooper, St Boniface General Hospital, Winnipeg, Manitoba; Dr Tung Chuo Yang, Surrey Memorial Hospital, Surrey, British Columbia; Dr Omer Choudri, Memorial Hospital Bowmanville, Bowmanville, Ontario; Dr Shaun MacCormick, Department of Health, Truro, Nova Scotia; Mr Bud Jenkins, St John's Health Care Corporation, St John's, Newfoundland; Ms Marie-Claude Michel, Centre Hospital de l'Université Laval, Ste Foy, Québec; Ms Chantel Bearden, Dufferin-Caledon Health Care Corporation, Orangeville, Ontario*

Correspondence and reprints: Dr Gordon Dow, Section of Infectious Diseases, Department of Internal Medicine, The Moncton Hospital, 135 MacBeath Avenue, Moncton, New Brunswick E1C 6Z8. Telephone 541-857-5670, fax 541-857-5671

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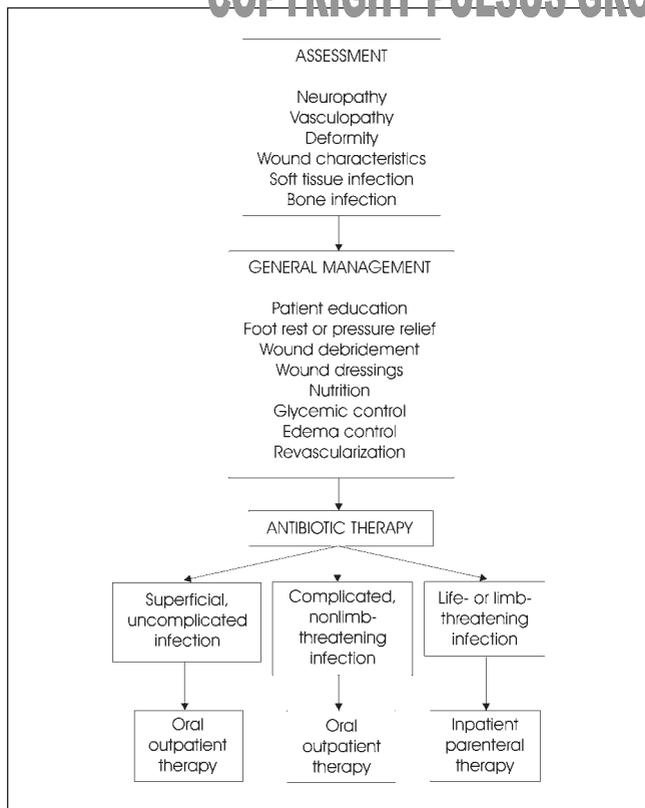


Figure 1) Diabetic foot infection care plan

and straightforward care plan for the management of the diabetic foot using a multidisciplinary approach. The objective of this undertaking was not to provide a detailed review of the literature, as has been undertaken by several authors (6,7), but rather to incorporate the current state of knowledge into a workable care plan. The care plan was organized as per the format in Figure 1, with sections discussing assessment of the diabetic foot (Table 1), general management based on the findings of that assessment (Table 2) and finally a description of empirical antibiotic therapy based on the severity of infection (Table 3). Where possible, photographic examples have been provided to illustrate various teaching points.

Empirical antibiotic therapy for diabetic foot infections has recently been reviewed (8,9), and these guidelines have been incorporated into this care plan with only minor modifications (Table 3). The empirical antibiotic choice should be altered based on appropriate culture results, once obtained.

The present care plan has been further supplemented by management guidelines based on the Wagner Classification (Table 4). Previous research has indicated that the use of protocols based on the Wagner Classification may be associated with higher rates of ulcer healing (10).

This care plan was developed with the input of a multidisciplinary panel and is intended to function as a general guideline from which deviation can and should be made, based on the particular clinical problem at hand and the opinion of the health care team.

TABLE 1
Care plan for diabetic foot infections – Assessment

Pathology	Assessment	Photographic example
Neuropathy	<ul style="list-style-type: none"> Consider significant if ulcer is painless, there is claw-toe deformity or patient is unable to perceive 10 g Semmes-Weinstein monofilament 	 <p>Testing for neuropathy with monofilament</p>
Foot deformity	<ul style="list-style-type: none"> Presence of claw-toe deformity, prominent metatarsal heads and high arch (suggesting sensory motor neuropathy) Consider Charcot neuroarthropathy if there is evidence of polyarticular joint destruction; severe neuropathy with bounding pulses and venodilatation; one or more fractures unrelated to infection; midfoot plantar ulcer; or pes planus (loss of arch) Presence of midfoot eversion or inversion Assess adequacy of footwear 	 <p>Charcot foot deformity with loss of plantar arch (pes planus) leading to 'rocker bottom' foot. Note typical superficial venous dilation of autonomic neuropathy</p>

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TABLE 1 (continued)

Care plan for diabetic foot infections – Assessment

Pathology	Assessment	Photographic example
Vasculopathy	<ul style="list-style-type: none"> Consider significant if pulses are absent and feet are cold, or if systolic pressure is 50 mmHg or less at ankle or less than 30 mmHg at toe using Doppler tests (note one-third of these patients will not heal unless perfusion is improved) Consider angiography if vasculopathy is present and there is severe claudication that is unacceptable to the patient, limb-threatening ischemia (progressive tissue necrosis) or ischemic leg or foot pain at rest or at night; patient is a surgical candidate; or angiography is not contraindicated (severe renal failure, anaphylaxis to contrast) 	 <p><i>Ischemic foot with typical digital tip ulceration</i></p>
Wound	<ul style="list-style-type: none"> Measure length, width and depth Classify wound bed as necrotic (yellow and/or black) or granulating (red) Classify ulcer according to the Wagner classification (see Table 4) 	 <p><i>Necrotic 'black/yellow wound' requiring surgical debridement. Extensive cellulitis also present</i></p>  <p><i>Conversion of wound P₄ to a 'red wound' (granulation tissue) after debridement and use of hydrogel dressings</i></p>
Infection	<ul style="list-style-type: none"> Ulcers without inflammatory changes should not be cultured (unless patient is on immunosuppressive treatment or severe ischemia is present and infection is suspected) Technique: cleanse ulcer base vigorously with saline and obtain deep swab for aerobic (and anaerobic) culture; if abscess is present, aspirate and culture the fluid. Anaerobes are seldom present if ulcer is superficial and present for less than four to eight weeks Microbiology from a swab approximates a tissue biopsy and is adequate to guide antibiotic treatment (11,12) Osteomyelitis is diagnosed if there is visible bone or if bone is detected with a sterile probe; it is unlikely if ulcer is less than four weeks in duration (13) Serial x-rays should be performed to follow bone remodelling during treatment and to diagnose occult osteomyelitis Perform bone scan with or without gallium or white blood cell scan, only if serial x-rays are negative and osteomyelitis is strongly suspected (14) Assess infection severity (as per Table 3) 	 <p><i>Infected Charcot ulcer with spreading cellulitis</i></p>

TABLE 2
Care plan for diabetic foot infections – Management

Pathology	Management
Neuropathy	<ul style="list-style-type: none"> • General measures: optimize glycemic control, control heart failure, discontinue alcohol and improve nutrition • Foot care preventive education and foot care (15); appropriate footwear and insoles (16) • Amitriptyline if painful peripheral neuropathy is present
Foot deformity	<ul style="list-style-type: none"> • Cushioned, extra deep footwear • Customized orthotics (insoles) • Total contact casting (if there is Charcot neuroarthropathy or resistance to healing despite adequate perfusion in a nonedematous limb) (17)
Vasculopathy	<ul style="list-style-type: none"> • Discontinue vasoconstricting medications, smoking, tight stockings, tensor bandages • Treat peripheral edema • Enteric-coated acetylsalicylic acid 325 mg once daily • Revascularization (angioplasty or bypass surgery) • Consider pentoxifylline • Manage hyperlipidemia
Wound	<ul style="list-style-type: none"> • Black and/or yellow wounds (necrotic tissue): Surgically debride all necrotic tissue, fibrin and callus (as perfusion permits) followed by autolytic debridement using hydrogel dressings; debridement dressings (saline wet to dry, one-quarter to one-half strength Dakin's solution) are generally avoided and are used only if the wound is necrotic or infected and sharp debridement cannot be performed; 10-min foot soak in dilute acetate 0.25% (vinegar) may aid debridement and disinfection at this stage • Deep ulcers or cavities: treat with saline-soaked gauze, salt-impregnated gauze, hydrogel fibre or nuga gauze covered with Flamazine (Smith & Nephew, Canada) • Red wounds (granulation tissue): treatment with hydrogel, bacitracin zinc ointment (or Flamazine if the infection is superficial); for large defects with slow response to standard care, consider platelet-derived growth factor (becaplermin), skin substitute (Dermagraft [Advanced Tissue Sciences, USA]) or skin grafting • See Management according to the Wagner classification (Table 4)

TABLE 3
Care plan for diabetic foot infections – Antibiotic therapy

Infection	Patient characteristics	Organism coverage	Antibiotic, dose and duration
Superficial, uncomplicated infections	<ul style="list-style-type: none"> • 80% of initial presentations • One-month's duration or less • 2 cm of inflammation or less • Negative probe to bone • Negative x-ray for osteomyelitis 	<ul style="list-style-type: none"> • <i>Staphylococcus aureus</i> • <i>Streptococcus</i> species 	<ul style="list-style-type: none"> • Cephalexin 500 to 1000 mg four times daily for 14 days, or • Cotrimoxazole 160/800 mg bid for 14 days, or • Amoxicillin/clavulanate 500/125 mg tid for 14 days, or • If patient is penicillin allergic, clindamycin 300 mg tid for 14 days
Complicated, nonlimb-threatening infections	<ul style="list-style-type: none"> • 20% of infections • Acute aggressive infections • Chronic progressive infections • Greater than 2 cm of cellulitis • Involve deep soft tissue • Involve bones or joints • Require outpatient management • Occasionally require home IV therapy or inpatient management 	<ul style="list-style-type: none"> • <i>S aureus</i> • <i>Streptococcus</i> species • Anaerobes • Coliforms (enteric Gram-negative bacilli) 	<ul style="list-style-type: none"> • Treatment duration: 14 days for soft tissue infections; average of 12 weeks for osteomyelitis (18,19) • Cephalexin 500 to 1000 mg four times daily plus Metronidazole 500 mg bid, or • Cotrimoxazole 160/800 to 320/1600 mg bid plus Metronidazole 500 mg bid, or • Amoxicillin/clavulanate 500/125 mg tid, or • Cotrimoxazole 160/800 to 320/1600 mg bid plus Clindamycin 300 mg four times daily, or • Clindamycin 300 mg four times daily plus ciprofloxacin 500 mg bid
Limb- and life-threatening infections	<ul style="list-style-type: none"> • Acutely evolving infections • Involve sepsis • Involve bacteremia • Require inpatient management including surgical intervention if necessary • Occasionally require home IV therapy postdischarge 	<ul style="list-style-type: none"> • <i>S aureus</i> • <i>Streptococcus</i> species • Anaerobes • Coliforms (enteric Gram-negative bacilli) • Add aminoglycoside for 48 to 72 h pending cultures • If infection is hospital-acquired, Gram-negative bacilli are suspected 	<ul style="list-style-type: none"> • Treatment durations as per complicated nonlimb-threatening infection • Intravenous ceftriaxone 1 g every 24 h plus either IV metronidazole 500 mg every 12 h or IV clindamycin 600 mg every 8 h, or • IV cefotaxime 1g every 8 h, plus either IV metronidazole 500 mg every 12 h or IV clindamycin 600 mg every 8h
		<ul style="list-style-type: none"> • Broad spectrum coverage including antipseudomonal coverage • Add aminoglycoside for 48 to 72 h pending cultures 	<ul style="list-style-type: none"> • Intravenous piperacillin/tazobactam 4.5 g every 8 h or 3.375 g every 6 h, or • Intravenous ceftazidime 1 to 2 g every 8 h plus IV clindamycin 600 mg every 8 h, or • Intravenous imipenem/cilastatin 500/500 mg every 6 h

IV Intravenous. Table adapted from reference 8

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TABLE 4
Management according to the Wagner classification

Grade	Description	Management	Photographic illustration
0	High risk foot based on neuropathy, vasculopathy or deformity; no ulcer present	<ul style="list-style-type: none"> • Assess for neuropathy and vasculopathy (see Table 1) • Provide foot care education • Provide emollients for dry skin • Pare callouses, trim nails, consider referral to podiatry • Provide appropriate foot wear and orthotics based on degree of foot deformity (see Table 1) • If there is severe claudication or ischemic rest pain, refer for vascular surgery • For less severe vascular disease or nonsurgical candidates, consider medical vascular management (see Table 2) 	 <p><i>Sensory motor neuropathy producing contractions of intrinsic muscles of foot leading to claw-toe deformity</i></p>
1	Superficial ulcer that does not penetrate into subcutaneous tissue	<ul style="list-style-type: none"> • Management as per grade 0 • Surgically debride all necrotic (black and/or yellow) tissue and callous as perfusion permits (consider chemical or enzymatic debridement if sharp debridement is not possible) • Follow sharp debridement by the use of topical hydrogel to permit autolytic debridement and granulation • Cleanse with saline between dressing changes • Manage infection as per Tables 1 and 2 • Provide pressure relief (eg, crutches, walkers, wheelchairs, healing sandals, air casts, total contact casts and extra deep footwear or orthotics) 	 <p><i>Superficial neuropathic plantar ulcer of forefoot</i></p>
2	Deep ulceration with penetration into subcutaneous tissue but not other deep structures	<ul style="list-style-type: none"> • Management as per grade 1 	 <p><i>Neuropathic plantar forefoot ulcer extending into subcutaneous tissue</i></p>
3	Involvement of deep subcutaneous tissues with either abscess formation, septic tenosynovitis, septic arthritis or osteomyelitis	<ul style="list-style-type: none"> • Management as per grade 2 • Deep surgical debridement is often indicated; surgical reperfusion may be required to permit debridement • Assess and manage infection as per Tables 1 and 2: Intravenous therapy may be required for approximately one to two weeks before oral step-down • Osteomyelitis is considered healed once the wound has closed, the erythrocyte sedimentation rate returns to baseline and bone remodelling is demonstrated on x-ray; prolonged (average 12 weeks) oral antibiotic therapy is usually required for osteomyelitis; if all necrotic bone can be debrided, a six-week course of antibiotic is adequate (20) 	 <p><i>Deep neuropathic-ischemic ulcer extending into the fifth metatarsal bone</i></p>

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TABLE 4 (continued)

Management according to the Wagner classification

Grade	Description	Management	Photographic illustration
3			
4	Localized gangrene of toe, forefoot or heel	<ul style="list-style-type: none"> • Management as per grade 3 • Lesions in this class are often ischemic, and vascular assessment is critical • Gangrenous toes may be allowed to petrify and autoamputate; may be the safest option where surgical wounding of ischemic tissue is not well tolerated (unless reperfusion is performed). In the setting of ischemia, aggressive surgical debridement may lead to further tissue loss; 0.25% vinegar soaks for 10 min daily and chemical or enzymatic debridement (especially of heel necrosis) may be preferable • 0.25% vinegar foot soaks appear to prevent progression from dry to wet gangrene if focal dry gangrene is present (mix 1 tablespoon of vinegar per cup of warm tap water) 	
5	Extensive gangrene of whole or most of foot	<ul style="list-style-type: none"> • Refer for amputation 	
			

*Radiographic bone destruction of fifth metatarsal**Gangrene of single digit (ischemic)**Extensive dry gangrene of forefoot**Extensive necrotizing fasciitis leading to gangrene of whole foot*

Table adapted from reference 13

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REFERENCES

1. Most RS, Sinnock P. The epidemiology of lower extremity amputations in diabetic individuals. *Diabetes Care* 1983;6:87-91.
2. Edmonds ME, Blundell MP, Morris HE, Thomas EM, Cotton LT, Watkins PJ. The diabetic foot: Impact of a foot clinic. *Q J Med* 1986;232:763-71.
3. Lawee D, Csima A. Diabetes-related lower extremity amputations in Ontario: 1987-88 experience. *Can J Pub Health* 1992;83:298-302.
4. Ebskov B, Josephsen P. Incidence of amputation and death after gangrene of the lower extremity. *Prosthet Orthot Int* 1980;4:77-80.
5. Palumbo PJ, Melton LJ. Peripheral vascular disease and diabetes. In: *Diabetes in America: Diabetes Data Compiled 1984*. Washington, DC: US Government Printing Office, 1985:1-21.
6. Caputo G, Cavanagh PI, Ulbrecht J, Gibbons G, Karchaner A. Assessment and management of foot disease in patients with diabetes. *N Engl J Med* 1994;331:854-60.
7. Lipsky BA, Pecoraro RE, Wheat LJ. The diabetic foot: soft tissue and bone infection. *Infect Dis Clin North Am* 1990;4:409-32.
8. Harding G, Field S, MacMahon R, Louie T, and the Prairie Consensus Conference Group. The antibiotic puzzle: Guidelines for the family physician. *Can J Infect Dis* 1997;8(Suppl C):2C-16C.
9. Fong IW and the Committee on Antimicrobial Agents. Management of diabetic foot infection: A position paper. *Can J Infect Dis* 1996;7:361-5.
10. Calhoun J, Cantrell J, Cobos J, et al. Treatment of diabetic foot infections: Wagner Classification, therapy and outcome. *Foot Ankle* 1988;9:101-6.
11. Sapico FL, Witte JL, Canawati HN, Montgomerie JZ, Bessman AN. The infected foot of the diabetic patient: quantitative microbiology and analysis of clinical features. *Rev Infect Dis* 1984;6(Suppl 1):S171-6.
12. Wheat LJ, Allen SD, Henry M, et al. Diabetic foot infections: bacteriologic analysis. *Arch Intern Med* 1986;146:1935-40.
13. Grayson ML, Balogh K, Levin E, Karchmer AW. "Probing to bone" – a useful clinical sign of osteomyelitis in diabetic fetid feet. 30th Interscience Conference on Antimicrobial Agents and Chemotherapy, Atlanta, October 21-21, 1990. (Abst)
14. Newman LG, Waller J, Palestro CJ, et al. Unsuspected osteomyelitis in diabetic foot ulcers: diagnosis and monitoring by leukocyte scanning with indium in 111 oxyquinoline. *JAMA* 1991;266:1246-51.
15. Young MJ, Cavanagh PR, Thomas G, Johnson MM, Murray H, Boulton AJM. The effect of callus removal on dynamic plantar foot pressures in diabetic patients. *Diabet Med* 1992;9:55-7.
16. Soulier SM. The use of running shoes in the prevention of plantar diabetic ulcers. *J Am Podiatr Med Assoc* 1986;76:395-400.
17. Mueller MJ, Diamond JE, Sinacore DR, et al. Total contact casting in treatment of diabetic plantar ulcers: controlled clinical trial. *Diabetes Care* 1989;12:384-8.
18. Peterson LR, Lissack LM, Canter K, et al. Therapy of lower extremity infections with ciprofloxacin in patients with diabetes mellitus, peripheral vascular disease, or both. *Am J Med* 1989;86:801-8.
19. Bamberger DM, Daus GP, Gerding DN. Osteomyelitis in the feet of diabetic patients: long-term results, prognostic factors, and the role of antimicrobial and surgical therapy. *Am J Med* 1987;83:653-60.
20. Dow G, Thompson W, Brunham R, et al. Duration of antimicrobial therapy for diabetic foot osteomyelitis. *Clin Invest Med* 1994;17(Suppl):Abst 440.



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