ORIGINAL INVESTIGATION

Practical Criteria for Screening Patients at High Risk for Diabetic Foot Ulceration

Lawrence A. Lavery, DPM, MPH; David G. Armstrong, DPM; Steven A. Vela; Terri L. Quebedeaux, DPM; John G. Fleischli, DPM

Background: A comprehensive understanding of clinical risk factors for developing foot ulcerations would help clinicians to categorize patients by their risk status and schedule intervention resources accordingly to prevent amputation.

Objective: To evaluate risk factors for foot ulcerations among persons with diabetes mellitus.

Method: We enrolled 225 age-matched patients, 46.7% male, with a ratio of approximately 1:2 cases: controls (76 case-patients and 149 control subjects). Case-patients were defined as subjects who met the enrollment criteria and who had an existing foot ulceration or a recent history of a foot ulceration. Control subjects were defined as subjects with no history of foot ulceration. A stepwise logistic regression model was used for analysis.

Results: An elevated plantar pressure (>65 N/cm^2), history of amputation, lengthy duration of diabetes (>10 years), foot deformities (hallux rigidus or hammer toes), male sex, poor diabetes control (glycosylated hemoglobin >9%), 1 or more subjective symptoms of neuropathy, and an elevated vibration perception threshold (>25 V) were significantly associated with foot ulceration. In addition, 59 patients (78%) with ulceration had a rigid deformity directly associated with the site of ulceration. No significant associations were noted between vascular disease, level of formal education, nephropathy, retinopathy, impaired vision, or obesity and foot ulceration on multivariate analysis.

Conclusions: Neuropathy, foot deformity, high plantar pressures, and a history of amputation are significantly associated with the presence of foot ulceration. Although vascular and renal disease may result in delayed wound healing and subsequent amputation, they are not significant risk factors for the development of diabetic foot ulceration.

Arch Intern Med. 1998;158:157-162

From the Department of Orthopaedics, The University of Texas Health Science Center at San Antonio (Drs Lavery, Armstrong, Quebedeaux, and Fleischli and Mr Vela); the Diabetic Foot Research Group (Drs Lavery, Armstrong, and Fleischli and Mr Vela); and the Mexican American Medical Treatment Effectiveness Research Center (Dr Lavery), San Antonio, Tex.

FOOT ULCERATION is one of the most common precursors to lower extremity amputations among persons with diabetes mellitus.[1,2] Ulcerations are pivotal events in limb loss for 2 important reasons. They allow an avenue for infection, and they can cause progressive tissue necrosis and poor wound healing in the presence of critical ischemia. Infections involving the foot rarely develop in the absence of a wound in adults with diabetes, and ulcers are the most common type of wound in these patients.[3] Foot ulcers, therefore, play a central role in lower extremity amputation. Clearly, the identification of an increased risk for ulceration is of central importance in any plan for amputation prevention.

Some descriptive studies suggest that 40% to 85% of amputations among persons with diabetes can be prevented with a team approach.[4-9] To help accomplish this goal, a comprehensive understanding of clinical risk factors for the development of foot ulcerations would help primary care providers to categorize patients by their risk status and schedule intervention resources accordingly. Previous studies of risk factors for ulcerations have identified peripheral neuropathy, vascular disease, limited joint mobility, foot deformities, abnormal foot pressures, a history of ulceration or amputation, and impaired visual acuity as important factors.[10-13] We have been unable to identify any existing work that considers all of these factors and their possible interactions in the same report. Therefore, the purpose of this study was to evaluate risk factors for foot ulcerations among persons with diabetes mellitus.

RESULTS

On univariate analysis, we noted numerous significant associations between

©1998 American Medical Association. All rights reserved.

Downloaded From: http://archinte.jamanetwork.com/ on 01/23/2017

PATIENTS AND METHODS

This project was conducted as a case-control study at the Texas Diabetes Institute and The University of Texas Health Science Center Clinics at San Antonio. The Texas Diabetes Institute is a model program for multidisciplinary diabetes care and includes the core participation of practitioners in general internal medicine, endocrinology, ophthalmology, podiatry, diabetes nurse education, and social services. Following informed consent, all patients were enrolled sequentially from these clinics. Before recruitment, institutional review board approval was secured. We enrolled 76 case-patients and 149 control subjects who met the following criteria: (1) the presence of diabetes mellitus based on World Health Organization criteria[14]; (2) evaluation by medicine and ophthalmology services within the past 3 months at the time of enrollment; (3) glycosylated hemoglobin, urinalysis, creatinine, and blood urea nitrogen laboratory studies performed in the past 3 months; and (4) age 18 to 80 years.

Case-patients were defined as subjects who met the above criteria with an existing or a recently healed (<4 weeks) foot ulceration. Control subjects were defined as those who had never had a foot ulceration. Patients with ulcers on the ankle or leg were eliminated from the study. Descriptive statistics for the study patients are summarized in **Table 1**.

Study participants were given a physical examination and interviewed for exposure variables, including demographic data; general medical, surgical, and social history; diabetes and diabetes-related complication history; visual acuity; and lower extremity vascular, neurologic, musculoskeletal, and dynamic foot pressure assessment. Diabetes mellitus was stratified into type 1 or 2 based on the criteria described by the National Institutes of Health's National Diabetes Data Group.[15] Renal function was stratified using the following criteria: no albuminuria (<20 µg/min) vs microalbuminuria (20-200 µg/min), macroalbuminuria (>200 µg/min), or chronic renal insufficiency (creatinine level, >350 µmol/L [>4.0 mg/dL]; current dialysis; history of renal transplantation).[16,17] The presence and severity of diabetic retinopathy was assessed from centrally graded retinal photographs taken with a wide-angle camera. These were evaluated by 1 grader who had no knowledge of the patients' case or control status. Retinopathy was classified as none vs background or proliferative. Proliferative retinopathy was differentiated from background retinopathy by the presence of any neovascularization, fibrous proliferations, preretinal hemorrhage, vitreous hemorrhage, or photocoagulation scars.[18] In addition, best-corrected visual acuity was evaluated using a Rosenbaum eye chart at the standard distance of 36 cm.[19] Subjects were allowed to wear glasses or contact lenses to assess their best-corrected vision. Corrected vision was scored as normal (<20/20), impaired (20/25 to 20/200), or legally blind (>20/200). We also asked subjects to attempt to place their foot in a position that would allow them to see the bottom of their foot regardless of their visual acuity. For this examination, self-evaluation skill was considered impaired if subjects were unable to position their foot and successfully read 0.5-cm type.[10]

We assessed the presence of peripheral sensory neuropathy using vibration perception threshold testing at the distal great toe with a Biothesiometer (Biomedical Instrument Co, Newbury, Ohio).[20] Peripheral vascular disease of the lower extremities was evaluated using several dichotomous variables. These included the Rose Intermittent Claudication Scale (history of claudication = score >10 points),[21] the absence of palpable dorsalis pedis and posterior tibial pulses in the foot, transcutaneous oxygen tension on the dorsal aspect of the first intermetatarsal space (<30 mm Hg),[11] and the ankle-brachial systolic blood pressure index (<0.80).[22] The CAGE questionnaire for evaluating ethanol abuse was used to dichotomize patients by the presence of alcoholism.[23] A value of 3 or higher was considered evidence of ethanol abuse using these criteria. Tobacco use was defined as any history of past or current smoking.

We averaged 3 measurements of the first metatarsophalangeal joint, the subtalar joint, and ankle joint range of motion to assess limited joint mobility of the forefoot, rear foot, and ankle. From these measurements, we determined the presence of ankle joint equinus (<0° dorsiflexion), limited subtalar joint motion (<20° total joint motion), and hallux rigidus (<50° hallux dorsiflexion).[24-26] To categorize forefoot deformities in addition to hallux rigidus, we evaluated the foot for the presence of hallux valgus, toe contractures (hammer-toe, claw-toe, or mallet-toe deformities), subluxation or dislocation of the metatarsophalangeal joints, and prominent metatarsal heads on the sole of the foot. In addition, we used the EMED pressure platform system (Novel, Dusseldorf, Germany) to evaluate dynamic barefoot pressures on the sole of the foot. An average of pressures from 3 midgait steps was used for the purposes of analysis.

In the initial analysis of the data, we stratified continuous variables into normal and abnormal categories and then performed χ^2 tests. We calculated odds ratios and 95% confidence intervals for exposure variables with an α of .05. To control for possible confounding variables, we then used a stepwise logistic regression analysis to model the effects of exposures and interactions. Only variables in the univariate model that achieved a *P* value of less than .25 were included in the regression analysis model.[27,28] Following multivariate analysis, to compare the additive risk, we restratified cases into 4 categories based on theorized increasing risk for ulceration. These categories included the following: intact protective sensation (category 0); loss of protective sensation, no deformity, and no history of ulceration or amputation (category 1); loss of protective sensation, deformity, but no history of ulceration or amputation (category 2); and loss of protective sensation, deformity, plus a history of ulceration or amputation (category 3).[29,30] Odds ratios were then calculated for each of these categories.

groups. Men with diabetes mellitus of durations longer than 10 years with 1 or more subjective symptoms of neuropathy and a history of an amputation or lower extremity bypass were more likely to present with an ulcer. Twenty-nine patients had a history of amputation, 27 (93%) of whom were in the group with ulcers. Of the patients with amputation, 20 had an amputation at the level of the digit, 4 at the midfoot level, and 5 were below-knee. Of these amputations, 7 were performed on the contralateral extremity (5 below-knee and 2 digital amputations). The 2 amputations performed in the control group were trauma-related. In addition, subjects with any

©1998 American Medical Association. All rights reserved.

Downloaded From: http://archinte.jamanetwork.com/ on 01/23/2017

Table 1. Descriptive Characteristics of Patients With Diabetes Mellitus*

Characteristic	Ulcer	No Ulcer
Patients, No.	76	149
Age, y	52.7±10.4 (28-75)	51.8±10.4 (20-76)
Male sex, %	74†	33
Type 2 diabetes mellitus, %	95	93
Duration of diabetes mellitus, y	14.5±9.1 (0-35)	9.2±8.8 (1-49)
Education, y	9.3±3.7 (1-18)	9.0±3.9 (0-16)
Glycosylated hemoglobin, %	9.9±2.4 (5.0-17.4)	8.6±1.9 (4.6-15.3)
Body mass index‡	30.8±5.7 (22.4-48.9)	32.3±6.2 (17.8-51.6)
Peak plantar pressure, N/cm^2	81.9±24.5 (10.0-125.0)	62.7±21.4 (7.3-113.0)
Ankle-brachial index	0.96±0.17 (0.38-1.19)	0.99±0.16 (0.25-1.19)
Transcutaneous oxygen tension, mm Hg	45.8±18.5 (10.0-88.0)	41.0±13.9 (6.0-88.0)
Vibration perception threshold, V	38.9±12.6 (8.0-50.0)	15.1±10.8 (4.0-50.0)

**See text for criteria for enrollment. Values are expressed as mean±SD (range) except where noted otherwise.*
†P<.05.
‡Measured in kilograms per meter squared.

concomitant nephropathy, retinopathy, or poor glucose control were at a significantly higher risk for ulceration. On physical examination, a loss of protective sensation, increased plantar pressure, and the presence of a foot deformity or a limited joint were signs significantly associated with the presence of ulceration. These data are summarized in **Table 2**.

Using a stepwise logistic regression model, we evaluated the possible interactions of the univariate associations. These data are summarized in **Table 3**. Not surprisingly, numerous variables that were statistically significant in the univariate analysis did not meet the criteria for significance in the logistic regression model after the correction for confounding. In this model, increased plantar pressure (>65.0 N/cm^2), a history of amputation, prolonged diabetes mellitus (>10 years), foot deformities (hallux rigidus, hallux valgus, or toe deformities), male sex, poor diabetes control (glycosylated hemoglobin >9.0%), 1 or more subjective symptoms of neuropathy, and an elevated vibration perception threshold (>25 V) were significantly associated with foot ulceration. In addition, 78% of patients with an ulcer had a rigid deformity directly associated with the site of ulceration.

We then evaluated the cumulative risk associated with common foot-specific factors (neuropathy, foot deformity, and foot amputation history) that can be easily incorporated into a normal clinical examination of a diabetic foot. As expected, the cumulative risk for ulceration increased with the addition of each variable. Patients with only peripheral neuropathy and no other risk factors were at 1.7 times greater risk for ulceration. Patients with both neuropathy and foot deformity were 12.1 times more likely to have an ulcer, and patients with neuropathy, deformity, and a history of amputation were 36.4 times more likely to have a wound develop. These data are summarized in **Table 4**.

Table 2. Independent Risk Factors for Ulceration: Univariate Model*

Risk Factor	Ulcer vs Nonulcer Group, % vs %	Odds Ratio	*P*
Historical data			
Male sex, %	74 vs 33	5.7	<.001
Diabetes duration >10 y	54 vs 21	4.5	<.001
≥1 Subjective symptoms of neuropathy	92 vs 66	6.1	<.001
Previous amputation	36 vs 1	40.5	<.001
Lower extremity bypass	4 vs 0	3.0	<.04
Current or past tobacco use	64 vs 56	0.8	.74
Alcohol abuse	16 vs 9	1.8	.19
Intermittent claudication	13 vs 6	2.3	.08
Diabetes comorbidities			
Any nephropathy	71 vs 44	3.1	<.001
Microalbuminuria	14 vs 20	0.7	.90
Macroalbuminuria	43 vs 22	2.7	<.001
End-stage renal disease	13 vs 3	5.5	<.003
Any retinopathy	66 vs 35	3.6	<.001
Background retinopathy	43 vs 26	2.2	<.009
Proliferative retinopathy	22 vs 9	3.0	<.005
Glycosylated hemoglobin >9.0%	68 vs 49	3.0	<.001
Impaired vision	48 vs 47	1.0	.97
Legally blind	14 vs 7	2.4	.09
Physical examination			
Loss of protective sensation (VPT >25 V)	84 vs 14	32.5	<.001
Plantar pressure >65 N/cm^2	72 vs 42	3.6	<.001
Hallux rigidus, hallux valgus, or rigid toe deformity	86 vs 56	4.6	<.001
Ankle equinus	41 vs 23	2.3	<.005
Limited subtalar joint range of motion	47 vs 30	2.1	<.009
Unable to see bottom of foot	54 vs 49	1.2	.49
≥1 Palpable foot pulse	87 vs 94	0.4	.09
Ankle-brachial index <0.80	10 vs 6	1.2	.1
Transcutaneous oxygen pressure <30 mm Hg	16 vs 17	1.1	.85

**VPT indicates vibration perception threshold.*

Numerous associations were conspicuous in their absence. No significant multivariate associations were noted between vascular disease (ankle-brachial systolic pressure index <0.80, palpable pedal pulses, or transcutaneous oxygen tension <30 mm Hg), level of formal education, nephropathy, retinopathy, impaired vision, ethanol abuse, or obesity and foot ulceration. Retinopathy and nephropathy, although significant on univariate analysis, did not meet the criteria for significance in the multivariate model. Furthermore, there was no significant association between past or current tobacco use and ulceration. This was true whether past and current tobacco use were grouped together or analyzed separately.

COMMENT

This study evaluates several potential risk factors for diabetic foot ulcerations. The results suggest that a dura-

©1998 American Medical Association. All rights reserved.

Downloaded From: http://archinte.jamanetwork.com/ on 01/23/2017

Table 3. Significant Risk Factors for Foot Ulceration: Multivariate Model*

Risk Factor	Odds Ratio	*P*
Loss of protective sensation	15.2	<.001
History of amputation	10.0	<.02
Elevated plantar pressure >65 N/cm^2	5.9	<.001
≥1 Subjective symptoms of neuropathy	5.1	<.02
Hallux rigidus, hallux valgus, toe deformity	3.3	<.03
Poor diabetes control†	3.2	<.03
Duration of diabetes >10 y	3.0	<.04
Male sex	2.7	<.05

*R^2=0.73.
†*Glycosylated hemoglobin >9%.*

Table 4. Cumulative Risk for Ulceration by Foot Risk Category*

Risk Category for Ulceration	Odds Ratio (95% CI)
Category 0	NA
Category 1	1.7 (0.7-4.3)
Category 2†	12.1 (5.2-28.3)
Category 3†	36.4 (16.1-82.3)

**See text for description of categories. CI indicates confidence interval; NA, not applicable.*
†P<.05.

tion of diabetes mellitus of more than 10 years, male sex, poor diabetes control, neuropathy, foot deformity, high plantar foot pressures, and a history of amputation are significantly associated with the presence of foot ulceration. Surprisingly, macrovascular and microvascular disease were not significant risk factors for ulceration in either the univariate or multivariate analysis.

Vascular disease has been strongly associated with diabetes-related lower extremity amputations. That it might also be an important risk factor for foot ulceration, therefore, seemed likely. Other reports on vascular compromise as a risk factor for diabetic foot ulceration provide mixed results. Macrovascular disease, as defined by the ankle-brachial index (<0.80), was not identified as a significant risk factor in either univariate or multivariate models in previous studies.[11,12] In contrast, McNeely et al[11] suggested that transcutaneous oximetry measurements were significantly lower in persons with ulcers. The cutoff point used in this study as the basis to define disease, however, was 60 mm Hg, which is higher than that in most other work in this area. In addition, it has been suggested that patients with peripheral arterial occlusive disease could maintain the integrity of the integument in the presence of low transcutaneous oxygen measurements.[31] Obviously, the evaluation of markers for vascular disease is critical during diabetic foot screening. The results of this investigation suggest, however, that vascular disease is probably a more important risk factor for delayed wound healing and commensurate amputation than ulceration in persons with diabetes.

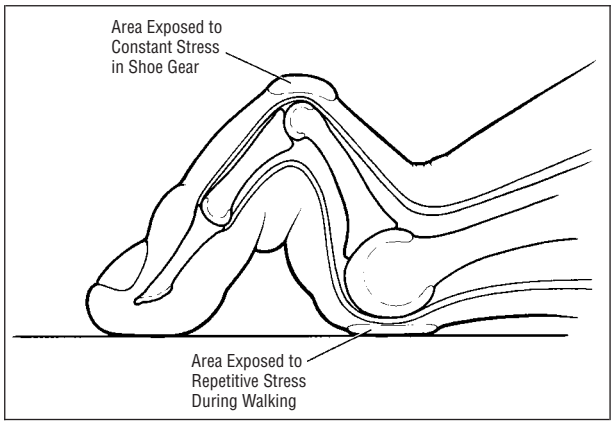
As expected, the presence of peripheral sensory neuropathy was one of the strongest risk factors for foot ulceration in this study. Patients with a loss of protective sensation, as determined by testing the vibratory perception threshold, were more than 15 times as likely to present with an ulceration as patients with intact sensation in the multivariate model. Our results are consistent with other case-control studies that have shown neuropathy to be a pivotal risk factor for both amputation and ulceration in persons with diabetes mellitus.[2,3,11] This reinforces the importance of a neurologic evaluation as one of the initial criteria to screen patients at risk for foot ulceration. Screening for neuropathy can be accomplished in a few minutes with the use of vibratory perception threshold testing. For relatively low cost ($400), the instrument provides reproducible, quantitative sensory information that can be gathered quickly by a physician, nurse, or technician. Because this instrument provides quantitative information, it can also be used to document the progression as well as the presence of neuropathy. Other less expensive instruments such as Semmes-Weinstein monofilaments (Gillis W. Long, Hansen's Disease Center, Filament Project, Carvile, La) have also been shown to be effective screening tools for identifying a high risk for foot ulceration[24,32,33] and may be a practical substitute for many physicians.

In addition to peripheral sensory neuropathy, high-pressure areas on the sole of the foot is one of the classic risk factors for foot ulceration. In a patient with neuropathy, pressure sites exposed to repetitive trauma produced during normal walking are predisposed to injury and ulceration. Brand[34] described the scenario where neuropathy, high plantar pressure, and repetitive trauma formed the principal etiologic factors involved in the development of foot wounds in patients with leprosy. This concept has been incorporated in the treatment of insensate diabetic foot wounds. Subsequent studies attempted to identify a cutoff point for foot pressures below which foot wounds were not likely to develop. For instance, Young et al[20] found that there were no ulcers in patients with peak foot pressures of less than 112 N/cm^2. Patients with ulcers in our study demonstrated a much greater range of foot pressures (10.0-125.0 N/cm^2), and the average peak foot pressure (82 N/cm^2) was much lower than the threshold level described by Young et al. Patients with lower pressures probably require more cycles of repetitive trauma before ulceration than patients with high peak plantar pressures.[35] It seems intuitive that the development of plantar foot ulceration would be a function of both of these variables.

Another mechanism of foot ulceration has been associated with constant as opposed to repetitive pressures. This is common when tight shoes cause constant pressure over bony prominences on the dorsum of the lesser toes, at the medial aspect of the first metatarsal head, or the lateral aspect of the fifth metatarsal. Foot deformities, equinus, limited joint mobility, and previous foot amputation probably contribute to the development of foot wounds because they are more likely to create areas exposed to constant pressure and because they cause biomechanical abnormalities that are related to increased foot pressures, as described earlier. For instance, contrac-

©1998 American Medical Association. All rights reserved.

Downloaded From: http://archinte.jamanetwork.com/ on 01/23/2017



Structural deformity increases the risk for ulceration.

ture, subluxation, or dislocation of a metatarsophalangeal joint causes the metatarsal head to be prominent on the bottom of the foot and the toe to be prominent dorsally (**Figure**). In addition, there is evidence that after a partial foot amputation, patients with diabetes are at greater risk of these types of foot deformities and subsequent foot ulcers developing.[36]

The development of foot deformities and other risk factors for ulcers may be enhanced by the systemic effects of diabetes mellitus. The association between the degree of glucose control, a prolonged duration of diabetes, and the incidence of numerous diabetic complications has been well demonstrated.[37] These include neuropathy[38,39] and soft tissue cross-linking through the production of advanced glycosylation end products.[40] Soft tissue glycosylation also has been reported to contribute substantially to the development of limited joint mobility.[41,42] Peripheral neuropathy also has been associated with atrophy of the intrinsic muscles of the foot and the development of forefoot deformities.[36] All of these factors may decrease the integrity and increase the friability of the soft tissue and predispose patients to ulceration,[43] infection, and subsequent amputation.[3] In our study, poor plasma glucose control was strongly associated with the presence of ulceration.

In this study, male sex was a significant risk factor for ulceration. This finding was not surprising as male sex has been previously associated as a risk factor for a number of diabetes-related lower extremity complications. In general, women seem to have fewer complications and a better prognosis than men. The prevalence of peripheral arterial occlusive disease[22,44-46] and sensory neuropathy[47] is lower in women with diabetes. Likewise, the prevalence and incidence of amputations and mortality associated with amputations of the foot are significantly lower in women.[48,49] In women also, foot ulcers and fractures associated with Charcot arthropathy of the foot have been reported to heal significantly more rapidly than in men.[35,50] Numerous factors may play a role in the effect of gender on lower extremity morbidity. These may include activity level, smoking behavior, hormonal differences, degree of compliance, level of denial, strength of social support mechanisms, and quality of education as well as the prevalence and severity of vascular disease, neuropathy, and diabetes.

The practical value of this study is that it provides criteria to identify high-risk factors for foot ulceration. Several studies have reported the low proportion and poor quality of diabetic foot examinations in general practice and in patients hospitalized specifically for foot disease.[1,51,52] Even though both physicians and patients are well aware of the strong association between diabetes, foot wounds, and lower extremity amputations, this trend still continues. This may be at least partially because many physicians do not have a clear understanding of the most important criteria to include in a screening examination. Most of the criteria to stratify a patient into a high-risk group are straightforward, easy to evaluate, and inexpensive. Several factors can be identified quickly by simply inspecting the foot or by including a few specific questions in the history. For instance, the identification of previous ulceration or partial foot amputation and the presence of foot deformities requires a few moments of focused questioning, inspection, and observation. The only risk factor that cannot be evaluated in the framework of a standard clinical examination is the quantitative evaluation of dynamic foot pressures. A clinical examination, however, including an evaluation for the presence of plantar callosities, may be a practical alternative.[53]

Diabetic foot complications can be prevented, but the process is expensive. With the increasing number of patients with diabetes, it is impractical to provide in-depth preventive foot care for every patient with the disease.[54] In this age of expensive high-tech gadgetry, capitation, and cost-conscious administrators, identifying high-risk factors can be a beneficial strategy for all parties using relatively low-tech, low-cost instrumentation. If high-risk factors are adequately identified and resources appropriately allocated, physicians will have a vehicle to get patients with these factors the treatment they require, and patients will receive appropriate care and cost savings from preventing wounds, amputations, and hospitalizations. Most risk factors identified in this report can be screened by nurses or technicians after a brief period of education and training and verified by an attending physician, if necessary. Resources for special footwear, intensive patient education, and more frequent medical and foot-specific evaluations can then be allocated for patients who fit a high-risk profile.

Accepted for publication May 29, 1997.

Reprints: Lawrence A. Lavery, DPM, MPH, Department of Orthopaedics, University of Texas Health Science Center, 7703 Floyd Curl Dr, San Antonio, TX 78284-7776 (e-mail: lavery@uthscsa.edu).

REFERENCES

1. Edelson GW, Armstrong DG, Lavery LA, Caicco G. The acutely infected diabetic foot is not adequately evaluated in an inpatient setting. *Arch Intern Med.* 1996; 156:2373-2376.
2. Pecoraro RE, Reiber GE, Burgess EM. Causal pathways to amputation: basis for prevention. *Diabetes Care.* 1990;13:513-521.
3. Reiber GE, Pecoraro RE, Koepsell TD. Risk factors for amputation in patients with diabetes mellitus: a case-control study. *Ann Intern Med.* 1992;117:97-105.
4. Apelqvist J, Ragnarson-Tennval G, Persson U, Larsson J. Diabetic foot ulcers in a multidisciplinary setting: an economic analysis of primary healing and healing with amputation. *J Intern Med.* 1994;235:463-471.

©1998 American Medical Association. All rights reserved.

Downloaded From: http://archinte.jamanetwork.com/ on 01/23/2017

5. Levin ME. The diabetic foot: pathophysiology, evaluation, and treatment. In: Levin ME, O'Neal LW, eds. *The Diabetic Foot.* St Louis, Mo: Mosby–Yearbook Inc; 1988: 7-27.
6. Tan JS, Flanagan PJ, Donovan DL, File TM. Team approach in the management of diabetic foot infections. *J Foot Surg.* 1987;(26 suppl 1):S12-S16.
7. Edmonds ME. Experience in a multidisciplinary diabetic foot clinic. In: Connor H, Boulton AJM, Ward JD, eds. *The Foot in Diabetes.* Chichester, NY: John Wiley & Sons; 1987:121-131.
8. Reiber GE. Diabetic foot care: financial implications and practice guidelines. *Diabetes Care.* 1992;15:29-31.
9. Gibbons GW, Marcaccio EJ Jr, Burgess AM, et al. Improved quality of diabetic foot care, 1984 vs 1990: reduced length of stay and costs, insufficient reimbursement. *Arch Surg.* 1993;128:576-581.
10. Crausaz FM, Clavel S, Liniger C, Albenau A, Assal JP. Additional factors associated with plantar ulcers in diabetic neuropathy. *Diabetic Med.* 1988;5:771-775.
11. McNeely MJ, Boyko EJ, Ahroni JE, et al. The independent contributions of diabetic neuropathy and vasculopathy in foot ulceration. *Diabetes Care.* 1995;18: 216-219.
12. Sriussadaporn S, Mekanandha P, Vannasaeng S, et al. Factors associated with diabetic foot ulceration in Thailand: a case-control study. *Diabet Med.* 1997;14: 50-56.
13. Holewski J, Moss K, Stess R, Grunfeld C. Prevalence of foot pathology and lower extremity complications in a diabetic outpatient clinic. *J Rehabil Res Dev.* 1989; 26:35-44.
14. *Second Report on Diabetes Mellitus.* Geneva, Switzerland: World Health Organization; 1980.
15. Harris MI. Classification, diagnostic criteria and screening for diabetes. In: Group NDD, ed. *Diabetes in America.* Bethesda, Md: US Dept of Health and Human Services; 1995. Publication NIH 95-1468.
16. Savage S, Estacio RO, Jeffers B, Schrier RW. Urinary albumin excretion as a predictor of diabetic retinopathy, neuropathy, and cardiovascular disease in NIDDM. *Diabetes Care.* 1996;19:1243-1248.
17. Humphrey LL, Ballard DJ, Frohnert PP, Chu CP, O'Fallon M, Palumbo PJ. Chronic renal failure in non–insulin-dependent diabetes mellitus. *Ann Intern Med.* 1989; 111:788-796.
18. Aldington SJ, Kohner EM, Meuer S, Klein R, Sjolie AK. Methodology for retinal photography and assessment of diabetic retinopathy: the EURODIAB IDDM Complications Study. *Diabetologia.* 1995;38:437-444.
19. Swartz MH. The physical examination. In: Swartz MH, ed. *Physical Diagnosis.* Philadelphia, Pa: WB Saunders Co; 1989:73-82.
20. Young MJ, Breddy JL, Veves A, Boulton AJM. The prediction of diabetic neuropathic foot ulceration using vibration perception thresholds. *Diabetes Care.* 1994; 16:557-560.
21. Rose GA. Diagnosis of ischemic heart pain and intermittent claudication in field study. *Bull World Health Organ.* 1962;27:645-650.
22. Beach KW, Strandness DE. Arteriosclerosis obliterans and associated risk factors in insulin-dependent and non–insulin-dependent diabetes. *Diabetes.* 1980; 29:882-888.
23. Magruder-Habib K, Stevens HA, Alling WC. Relative performance of the MAST, VAST, and CAGE versus *DSM-III-R* criteria for alcohol dependence. *J Clin Epidemiol.* 1993;46:435-441.
24. Birke J, Cornwall MA, Jackson M. Relationship between hallux limitus and ulceration of the great toe. *Sports Phys Ther J Orthop.* 1988;10:172-176.
25. Birke JA, Franks D, Foto JG. First ray joint limitation, pressure, and ulceration of the first metatarsal head in diabetes mellitus. *Foot Ankle.* 1995;16:277-284.
26. Hopson MM, McPoil TG, Cornwall NW. Motion of the first metatarsophalangeal joint: reliability and validity of four measurement techniques. *J Am Podiatr Med Assoc.* 1995;8:198-204.
27. Schlesselmann JJ. *Case-Control Studies: Design, Conduct, Analysis.* New York, NY: Oxford University Press; 1982:chap 2.
28. Walker SH, Duncan DB. Estimation of the probability of an event as a function of several independent variables. *Biometrika.* 1967;54:167-179.
29. Armstrong DG, Lavery LA, Harkless LB. Treatment-based classification system for assessment and care of diabetic feet. *J Am Podiatr Med Assoc.* 1996;86:311-316.
30. Birke JA, Sims DS. The insensitive foot. In: Hunt GC, McPoil TG, eds. *Physical Therapy of the Foot and Ankle.* 2nd ed. New York, NY: Churchill Livingstone Inc; 1995:chap 3.
31. Hauser CJ, Klein SR, Mehringer CM, Appel P, Shoemaker WC. Assessment of perfusion in the diabetic foot by regional transcutaneous oximetry. *Diabetes.* 1984; 33:527-531.
32. Kumar S, Fernando DJS, Veves A, Knowles EA, Young MJ, Boulton AJM. Semmes-Weinstein monofilaments: a simple, effective and inexpensive screening device for identifying diabetic patients at risk of foot ulceration. *Diabetes Res Clin Pract.* 1991;13:63-68.
33. Mueller MJ. Identifying patients with diabetes who are at risk for lower extremity complications: use of Semmes-Weinstein monofilaments. *Phys Ther.* 1996; 76:68-71.
34. Brand PW. The insensitive foot (including leprosy). In: Jahss M, ed. *Disorders of the Foot and Ankle.* 2nd ed. Philadelphia, Pa: WB Saunders Co; 1991:2170-2175.
35. Armstrong DG, Lavery LA, Bushman TR. Peak foot pressures influence healing time of diabetic ulcers treated with total contact casting. *J Rehabil Res Dev.* In press.
36. Lavery LA, Lavery DC, Quebedeaux TL. Increased foot pressures after great toe amputation in diabetes. *Diabetes Care.* 1995;18:1460-1462.
37. Strowig S, Raskin P. Glycemic control and diabetic complications. *Diabetes Care.* 1992;15:1126-1140.
38. Dahl-Jorgensen K, Brinchmann-Hansen O, Hanssen KF, et al. Effect of near normoglycemia for two years on progression of early diabetic retinopathy, nephropathy, and neuropathy: the Oslo Study. *BMJ.* 1986;293:1195-1199.
39. Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med.* 1993;329:966-986.
40. Brownlee M. Glycation products and the pathogenesis of diabetic complications. *Diabetes Care.* 1992;15:1835-1843.
41. Rosenbloom AL. Skeletal and joint manifestations of childhood diabetes. *Pediatr Clin North Am.* 1984;31:569-589.
42. Rosenbloom AL, Silverstein JH, Lexotte DC. Limited joint mobility in childhood diabetics indicates increased risk for microvascular diseases. *N Engl J Med.* 1982; 305:191-194.
43. Moss SE, Klein R, Klein BEK. The prevalence and incidence of lower extremity amputation in a diabetic population. *Arch Intern Med.* 1992;152:610-616.
44. Alcolado JC, Pacy PJ, Beevers M, Dodson PM. Risk factors for peripheral vascular disease in hypertensive subjects with type 2 diabetes mellitus. *Diabet Med.* 1992;9:904-907.
45. Vogt MT, Wolfson SK, Kuller LH. Lower extremity arterial disease and the aging process: a review. *J Clin Epidemiol.* 1992;45:529-542.
46. Melton LJ, Macken KM, Palumbo PJ, Elveback LR. Incidence and prevalence of clinical peripheral vascular disease in a population-based cohort of diabetic patients. *Diabetes Care.* 1980;3:650-654.
47. Franklin GM, Kahn LB, Baxter J, Marshall JA. Sensory neuropathy in non–insulin dependent diabetes mellitus: The San Luis Valley Diabetes Study. *Am J Epidemiol.* 1990;131:633-643.
48. van Houtum WH, Lavery LA, Harkless LB. The impact of diabetes-related lower extremity amputations in the Netherlands. *J Diabetes Complications.* 1996;10: 325-330.
49. Armstrong DG, Lavery LA, van Houtum WH, Harkless LB. The impact of gender on amputation. *J Foot Ankle Surg.* 1997;36:66-69.
50. Armstrong DG, Todd WF, Lavery LA, Harkless LB. The natural history of acute Charcot's arthropathy in a diabetic foot specialty clinic. *Diabet Med.* 1997;14: 357-363.
51. Rosset J, Walker EA, Shamoon H, Engel S, Basch C, Zybert P. Assessment of documented foot examinations for patients with diabetes in inner-city primary care clinics. *Arch Fam Med.* 1990;4:46-50.
52. Cohen SJ. Potential barriers to diabetes care. *Diabetes Care.* 1983;6:499-500.
53. Young MJ, Cavanagh PR, Thomas G, Johnson MN, Murray H. Effect of callous removal on dynamic foot pressures in diabetic patients. *Diabet Med.* 1992;9:75-77.
54. Eckman MH, Greenfield S, Mackey WC, et al. Foot infections in diabetic patients: decision and cost-effectiveness analysis. *JAMA.* 1995;273:712-720.

©1998 American Medical Association. All rights reserved.

Downloaded From: http://archinte.jamanetwork.com/ on 01/23/2017