Classification of Diabetic Foot Wounds

Foot ulcers in persons with diabetes are one of the most common precursors to lower extremity amputation. Appropriate care of the diabetic foot ulceration requires a clear, descriptive classification system that may be used to direct appropriate therapy and possibly predict outcome. Ideally, this system would be used by all participants in a multidisciplinary limb salvage team. We describe a clinical classification system for diabetic foot wounds that evaluates wound depth, the presence of infection, and peripheral arterial occlusive disease in every category of the wound assessment. The goal of this system is to improve communication, leading to a less complex, more predictable treatment course and, ultimately, an improved result. (The Journal of Foot and Ankle Surgery 35(6):528–531, 1996)

Key words: diabetes, foot, ulcer, infection, amputation, neuropathy

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There are several well accepted predisposing factors that place patients with diabetes at high risk for a lower extremity amputation. The most common components in the causal pathway to limb loss include peripheral neuropathy, ulceration, infection, and peripheral vascular disease. Ulceration is the most common single precursor to amputation and has been identified as a component in 84% of lower extremity amputations (1). Systematically recording the characteristics of ulcerations is critical to plan treatment strategies, monitor treatment effectiveness, predict clinical outcomes, and improve communication among health care providers.

The classification systems that have previously been described in the medical literature either do not provide information about some of the most important parameters that would be expected to dictate treatment and predict outcome, or they are incompletely used in the grading scheme. Most classification systems primarily focus on the depth of the ulceration and neglect or inconsistently include infection and peripheral arterial occlusive disease (2–9).

To make a classification system clinically relevant, it should be easy to use, reproducible, and effective to accurately communicate the status of wounds in persons with diabetes mellitus. There are a variety of variables that could be included in such a system, such as faulty wound healing, patient compliance, quality of wound granulation tissue, host immunity, nutritional status, and co-morbidities. However, most of these variables are difficult to measure or categorize. The purpose of this paper is to report a clinical classification system for diabetic foot wounds that evaluates wound depth, the presence of infection, and peripheral arterial occlusive disease in every category of the wound assessment.

Classification Systems

Shea, in 1975, was one of the first to propose a standard wound classification system (3). This scheme was designed to assess decubitus ulcerations and was the model used for formation of subsequent classification systems, including the International Association for Enterostomal Therapy system (4, 5). It was based mainly on wound depth and did not focus on presence or absence of infection. Additionally, the system did not mention ischemia as a co-morbid factor. Because of these limitations, this system has little value for staging diabetic foot wounds.

One of the most commonly cited diabetic wound classification systems was first described by Meggitt in 1976, and popularized by Wagner in 1981 (6, 7). The system is based mainly on wound depth and consists of six wound grades. These include grade 0 (intact skin), grade 1 (superficial ulcer), grade 2 (deep ulcer to tendon, bone, or joint), grade 3 (deep ulcer with abscess or osteomyelitis), grade 4 (forefoot gangrene), and grade 5 (whole-foot gangrene). Infection is included in

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only one of the six Meggitt-Wagner ulcer grades, and vascular disease is only included in the last two classification grades. The system does not allow classification of superficial wounds that are infected or dysvascular. Another limitation of this system is that only the most severe signs of vascular disease (forefoot gangrene and whole foot gangrene) are considered. More subtle and clinically relevant signs of peripheral vascular disease are not included in the wound assessment. Consistent consideration of important risk factors within every grade of an ulcer classification system should allow physicians to quantify wound severity based on the ulcer grade. Sims and coworkers attempted to modify this system by adding additional descriptors to wound grades (10).

Forrest and Gamborg-Nielsen reviewed and attempted to clarify wound assessment with their system published in 1984 (8). It consisted of six grades and attempted to stage wounds based on appearance. Subsequently, patients in each grade were assigned to a medical and surgical treatment protocol. Unfortunately, this system did not include deep or severely infected ulcers, nor did it take into account vascular supply.

Knighton and colleagues developed a system based on their outpatient clinical trial of platelet-derived wound healing factor. The system included several subjective wound descriptors that were arbitrarily assigned weights that were tabulated to produce a "wound score" which the authors postulated would predict outcome (9). The limitations of this system are its inherent difficulties in reproducibly scoring each of the wound variables.

In 1990, Pecoraro developed a 10-stage classification system and compared it with several other published systems in a study of patients hospitalized with diabetic foot wounds that required some form of lower extremity amputation. Their findings indicated that several of the previously published schemes did not provide precise description of between 19 and 49% of the lesions encountered in their study (11). Like other published classification systems, clinical signs of peripheral arterial occlusive disease (necrotic tissue, black eschar, forefoot gangrene) and infection (abscess, inflamed ulcer with exudate) could not be scored with ulcer depth throughout each level of classification. In addition, the terminology used to identify wound characteristics was subjective and open to considerable interpretation.

The University of Texas San Antonio Diabetic Wound Classification System

We have observed that poor outcomes are often associated with wounds of increasing depth, increasing severity of infection, and presence of peripheral vascular disease. These factors have been widely discussed in the literature (12–16). The following classification uses a system of wound grade and stage to categorize wounds by severity. Wounds are graded by depth. Grade 0 represents a pre- or postulcerative site. Grade I ulcers are superficial wounds through the epidermis or epidermis and dermis, but do not penetrate to tendon, capsule, or bone. Grade II wounds penetrate to tendon or capsule. Grade III wounds penetrate to bone or into a joint. Within each wound grade there are four stages: non-ischemic clean wounds (A), non-ischemic infected wounds (B), ischemic wounds (C), and infected ischemic wounds (D).

The criteria for each of the stages are based on clinical and laboratory data. Clean ulcers are defined as wounds without local or systemic signs of infection. Wounds with frank purulence and/or two or more of the following local signs may be classified as "infected." These signs include warmth, erythema, lymphangiitis, lymphadenopathy, edema, pain, and loss of function. Systemic signs of infection may include fever, chills, nausea, vomiting, or generalized malaise (17). The clinical diagnosis of infection in persons with diabetes is often difficult and defined by narrow, subtle parameters. This clinical diagnosis is often obscured by neuropathy and possibly immunopathy. In the insensitive foot, pain and/or loss of function are poor indicators of inflammation and infection (18). Likewise, diabetic subjects have been shown to possess deficiencies in leukocyte adherence, chemotaxis, phagocytosis, and diapedesis (19–21) and often do not have leukocytosis in the presence of acute soft tissue or bone infection (18, 22, 23). Warmth and edema are less than ideal indicators of infection, as ulcerated sites tend to be warmer and more edematous than the corresponding site contralaterally, regardless of the presence of infectious disease (24). However, despite these impediments, diagnosis of a diabetic foot infection remains primarily a clinical one (22, 23). The diagnosis and subsequent treatment of infection may also be assisted by laboratory studies or positive deep tissue cultures or wound-base curettage (25). When osteomyelitis is suspected, bone biopsy with appropriate pathology and culture studies is still the gold standard for diagnosis.

The working diagnosis of lower extremity ischemia is made by a combination of clinical signs and symptoms plus noninvasive vascular studies. Clinical signs and symptoms may include claudication, rest-pain, absent pulses, atrophic integument, absence of pedal hair, dependent rubor, or pallor on elevation. Noninvasive criteria include transcutaneous oxygen measurements of <40 mm. Hg (26), ankle-brachial index (ABI) of <0.80 (14, 27), or absolute toe systolic pressure <45 mm. Hg (28, 29). One or more clinical signs coupled with one or more of the noninvasive values provides a working diagnosis of lower extremity vascular insufficiency.

TABLE 1 The University of Texas Health Science Center, San Antonio, diabetic wound classification system

	Grade			
	0	I	II	III
A	Pre- or postulcerative lesion completely epithelialized	Superficial wound, not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Pre- or postulcerative lesion, completely epithelialized with infection	Superficial wound, not involving tendon, capsule, or bone with infection	Wound penetrating to tendon or capsule with infection	Wound penetrating to bone or joint with infection
C	Pre- or postulcerative lesion, completely epithelialized with ischemia	Superficial wound, not involving tendon, capsule, or bone with ischemia	Wound penetrating to tendon or capsule with ischemia	Wound penetrating to bone or joint with ischemia
D	Pre- or postulcerative lesion, completely epithelialized with infection and ischemia	Superficial wound, not involving tendon, capsule, or bone with infection and ischemia	Wound penetrating to tendon or capsule with infection and ischemia	Wound penetrating to bone or joint with infection and ischemia

Based on this system, outcomes should be worse based on both increasing stage and grade (Table 1).

The Grade 0 Wound

Grade 0-A wounds are pre-ulcerative areas or previous ulcer sites that are now completely epithelialized after debridement of hyperkeratosis and nonviable tissue. The diagnosis of a grade 0 wound can be made only after reduction of any regional hyperkeratosis, as, quite often, frank ulcerations may be hidden by overlying callouses. The grade 0-B wound is defined as a pre- or postulcerative lesion with associated cellulitis. The grade 0-C wound is identical to 0-A, but exists concomitantly with regional signs of ischemia. The grade 0-D wound reveals cellulitis, coupled with a working diagnosis of lower extremity ischemia as previously defined.

While lesions that fall into this grade may not be classically classified as "wounds," the system is important to identify sites "at risk" for frank ulceration, and to monitor newly healed wounds. Since 28 to 50% of diabetics reulcerate within a year of healing their initial wound (30), ulcer classification should allow physicians to follow the progression of wounds over time.

The Grade I Wound

The Grade I-A wound is superficial in nature, with partial or full-thickness skin involvement not including tendon, capsule, or bone. As with any neuropathic lesion, the I-B wound (superficial ulceration coupled with infection) should be examined very carefully. The definition of this wound implies superficial infection without involvement of underlying structures. If the wound shows signs of significant purulence or fluctuance, further exploration to expose a higher grade infection is in order. The I-C wound is complicated by vascular compromise. Grade I-D implies an infected wound with concomitant ischemia.

The Grade II Wound

The II-A wound is marked by deeper involvement than the grade I wound, involving tendon or joint capsule, but *not* bone. II-B is characterized by deep infection, not involving joint or bone. Grade II-C and II-D wounds correspond to ischemic and infected ischemic wounds, respectively.

The Grade III Wound

While by no means pathognomonic, there exists a high correlation between probing to bone and osteomyelitis (31, 32). For this reason, we make a sharp distinction between wounds probing to bone and those without bone or joint involvement. The III-A wound is defined as one probing to bone with no local or systemic signs of acute infection. The III-B wound is infected. Wounds classified as III-C are identical to III-A with concomitant ischemia. The III-D wound is characterized by active infection, exposed bone, and vascular insufficiency.

Conclusion

The multidisciplinary approach in treatment of diabetic pedal ulcerations has resulted in a dramatic reduction in overall amputation rates and hospital length of stay (33–35). This approach, however, requires a simple, clear, and concise method of description and classification of pedal wounds, including the presence or absence of infection and vascular compromise. Communication among members of the team and researchers could be enhanced by a simple, concise method of classifying foot wounds. Further evaluation of this system should help to improve the

working definitions for infection and ischemia, establish wound grade and stage-based treatment recommendations, and validate the premise that increasing wound grade and stage is predictive of outcome.

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