Etiology and Treatment of Pedal Wounds in the Diabetic Patient

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INTRODUCTION

Pedal Wounds secondary to complications related to Diabetes Mellitus

Wounds related to diabetes mellitus are multifactorial in etiology. Primary factors contributing to chronic diabetic foot ulceration include peripheral neuropathy and peripheral vascular disease. Secondary factors including limited joint mobility, neuropathic osteoarthropathy (Charcot foot), and a depressed immune response to infection further complicate treatment. Prompt treatment of diabetic foot wounds with a multidisciplinary approach, coordinating the primary care physician, endocrinologist, vascular surgeon, and podiatrist, can achieve healing, and reduce the chance of amputation.[1]

In the United States, approximately 50% of all nontraumatic lower extremity amputations occur in patients with diabetes mellitus. Limb amputation is not an inevitable fact of diabetes with a controlled, organized approach to wound care. Long-term glycemic control is the goal. Identifying the etiology of the wound and then intervening with techniques to allow for an optimal wound climate for healing must be instituted. This includes optimizing arterial perfusion to the wound site. Removal of unhealthy tissue, thereby reducing bacterial bioburden, via debridement is important for wound bed preparation. Depending upon the depth of the wound, other deeper structures such as tendon, muscle, and bone (in osteomyelitis), may require excisional debridement. In addition, evaluation for a neuropathic component or structural deformity of the foot ie hallux valgus, hammertoes, or pes planus is also necessary to properly remove pressure or offload the wound to optimize healing potential. In certain instances, wounds cannot heal due to structural deformity of the foot and surgical correction of the underlying deformity is required.[2]

Etiology of Diabetic Foot Ulceration

Sensorimotor neuropathy and autonomic neuropathies are primary factors contributing to foot wounds. Sensorimotor neuropathy accounts for reduced or absent reflexes, intrinsic muscle atrophy, resultant musculoskeletal deformity (hammertoes, bunions, prominent metatarsal heads), and sensory loss in a stocking/glove distribution. The insensate foot cannot detect painful stimuli and is more likely to have abnormally high foot pressures because of structural deformity. This predisposes the extremity to injury such as a puncture wound or subsequent plantar ulceration/wound. Autonomic neuropathy is responsible for the decrease or absence of sweating of the lower extremity and arteriovenous shunting resulting in distention of dorsal veins in the foot. The presence of anhidrotic skin leads to cracking, fissuring, and, coupled with abnormally high foot pressures, hyperkeratotic skin or callus, which increases the risk of skin breakdown and development of foot wounds.

Peripheral vascular disease is also a primary causative factor contributing to foot wounds in the diabetic patient. Atherosclerotic occlusive disease of the macrocirculation, especially the distal popliteal and tibial arteries, affects the diabetic foot. Peripheral vascular insufficiency lowers the viability of skin, which reduces the pressure threshold in which ischemia and tissue breakdown occur. In the face of adequate blood supply, neuropathy takes precedence in the pathogenesis of foot ulceration.

Secondary etiologic factors of diabetic foot ulceration play a lesser role overall, but should not be overlooked. Limited joint mobility, caused by non-enzymatic glycosylation of proteins with subsequent rigidity contributes to the development of higher foot pressures and possible ulceration by not permitting adequate compensatory redistribution of high loads. Charcot joint disease compromises structural integrity of the foot causing dislocation with eventual rocker-bottom deformity. This leads to increased plantar midfoot pressures and eventual development of foot ulceration if not heeded. Diabetes is also associated with diminished neutrophil/immune function. The inability to aggressively fight infection allows for necrosis to persist within the wound and prevent healing.[3,4]

History and Clinical Examination of the Foot

Previous foot ulceration or amputation in the patient with diabetes has a strong predictive value for further foot problems. Diabetic complications such as nephropathy and retinopathy are associated with diabetic foot problems/wounds. Smoking and alcohol consumption also increase the risk of the development of foot wounds. Patient education about the causes and prevention of pedal wounds, including the use of proper fitting supportive shoe gear can greatly reduce the risk of diabetic foot ulceration.

Clinical signs of neurological deficit include an impaired sensation to pain, light-touch, cold, hot, and vibration, in addition to reduced or absent ankle and knee reflexes. These can be easily assessed in the office with use of a 5.07g

Table 1. Wagner grading system for diabetic foot infections[7]

Wagner Grade[7]	Wound Severity
0	Intact skin
1	Superficial ulcer of skin or subcutaneous tissue
2	Ulcers extend into tendon, bone, or capsule
3	Deep ulcer with osteomyelitis, or abscess
4	Gangrenous toes or forefoot
5	Gangrenous midfoot or hindfoot

Semmes-Weinstein monofilament or a tuning fork test. Clinical changes of vascular compromise are manifest as atrophic skin, hair loss, cool lower leg and foot, increased capillary filling time and diminished or absent pedal pulses. Dorsalis pedis and posterior tibial pulses can be palpated or assessed with a portable Doppler. If vascular compromise is evident further vascular diagnostic evaluation should be pursued. Revascularization of the extremity should be considered to promote wound healing. Appreciation of any digital deformities, bunion, hammertoe, pes planus, and other palpable bony prominences are important because these are areas of increased risk of foot ulceration. Furthermore, the presence of clinical or radiographic Charcot joint changes, and limited subtalar/ankle range of motion should be documented. Areas of dry skin, callus formation, interdigital maceration, bullae, dystrophic onychomycotic nails, tinea pedis, and skin ulceration require evaluation. Evaluation of pedal ulceration/wounds, should include information regarding location, size, depth, and surrounding soft tissue/ bone/ joint involvement. Classification systems such as Wagner's can be utilized to classify the wound and improve communication between medical disciplines.[5-7] (**Table 1**)

Wound Healing and the Approach to Treatment of Diabetic Foot Wounds

The basics of wound healing are reviewed elsewhere in this issue. Specifically, diabetic wounds display reduced growth factor production, decreased or impaired angiogenic response, macrophage function, collagen accumulation and epidermal barrier function. These all maintain a chronic inflammatory state preventing normal wound healing. Upon determining the extent of neuropathy, structural deformity, and limb perfusion, a wound care algorithm can be initiated. Conservative approaches, surgical intervention, and, at times, a combination of both may be required to achieve complete wound healing. Pressure relief of the wound may be required throughout the treatment protocol.[8]

Initiating a wound environment with proper moisture balance without excessive exudate is the goal. Reduction of lower extremity edema via compression or elevation of the extremity also alleviates unnecessary wound strain and exudate. Topical dressings can be optimized to maintain a moist wound bed and absorb excessive exudative collections of fluid that will macerate adjacent tissue and prevent healing.

Reducing the bacterial bioburden of the wound can also reduce excessive exudate.[9] There is a spectrum of bacterial presence in a wound ranging from contamination and colonization to critical colonization and infection. Chronic nonhealing wounds are usually contaminated and colonized. Recommendations for conservative treatment include cleansing, debridement (surgical, mechanical, enzymatic, and now ultrasonic), exudate management, and topical and oral antibacterial therapy.[10,12]

A critically colonized wound in the presence of unhealthy granulation, malodor, possibly deep sinus tracks to exposed bone, erythema, cellulitis, and systemic signs of infection requires a more aggressive treatment protocol. IV antibiotics with operative staged surgical debridement/surgical correction of foot deformity and hospitalization may be necessary to stabilize the infected wound for the eventual progression to less intensive wound healing therapies. The use of topical growth factors can be applied to a wound to stimulate a wound healing cascade, topical matrix preparations can act as scaffolding for wound healing, and external negative pressure wound therapy can be utilized to enhance granulation tissue and reduce exudate within a stable, noninfected wound.[11]

CASE STUDY

A 38-year-old man with history of diabetes and obesity presented to the emergency room with a large ulcer on his left foot. He stated it began as a blister that he popped and then picked at. A few days later, he noticed the ulceration getting larger, with increasing drainage, and he developed a fever. Exam revealed dorsalis and posterior tibial pulses 2/4 bilateral. He had diminished Semmes Weinstein monofilament 5.07 g thresholds on his toes bilaterally. His left lower extremity was very swollen, especially the foot and ankle. The dorsal aspect of his left foot revealed a large ulceration (**Figure 1).** There was purulent drainage and undermining

Figure 1. The dorsal aspect of his left foot revealed a large ulceration.

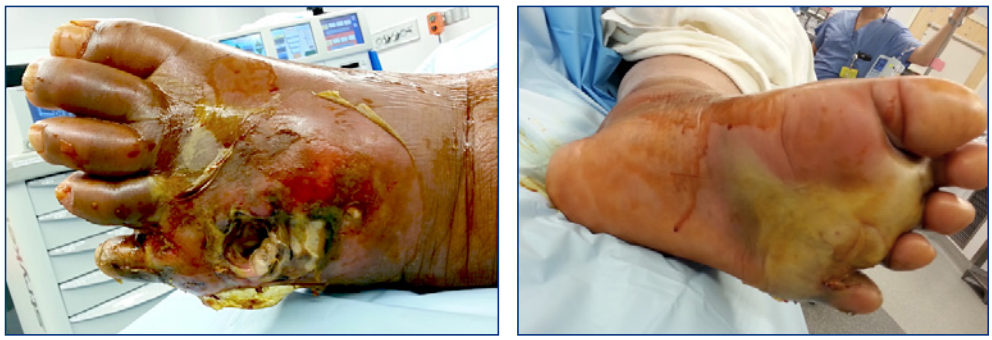


Figure 2: The patient was brought to the operating room immediately for incision and drainage of this wound. This resulted in removal of toes 4, 5, and a significant amount of tissue from the dorsum of his left foot.

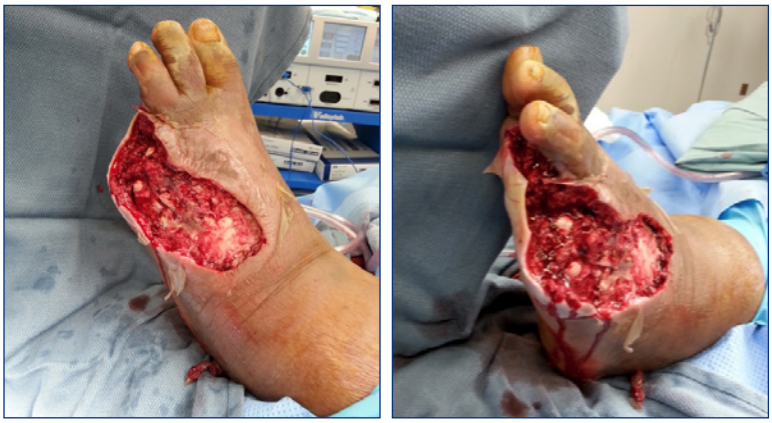


Figure 3: Given the size and depth of the wound, negative pressure therapy was chosen, and carried on for several weeks. When the granulation tissue completely filled in the wound, other modalities to assist in epithelialization could be considered.

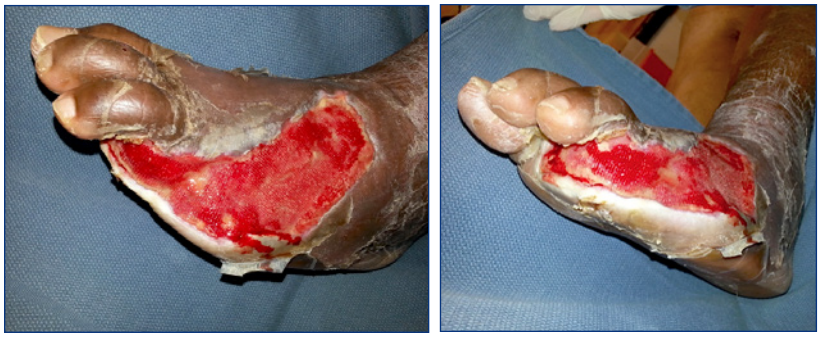
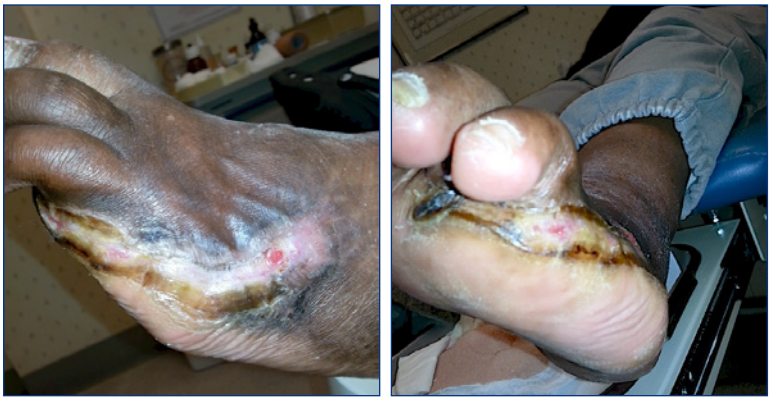


Figure 4: A skin substitute was utilized in this case. The wound subsequently went on to complete closure in four months.



of approximately 5-7 cm, without probe to bone. There was crepitus on palpation in the subcutaneous tissue around the ulceration. X-rays of the foot and ankle revealed gas in the tissue around the ulceration. His lab values revealed a white count of 14.1, and a hemoglobin A1c of 13.1. The patient was brought to the operating room immediately for incision and drainage of this wound. This resulted in removal of toes 4, 5, and a significant amount of tissue from the dorsum of his left foot (**Figure 2**). Then began the challenge of closing this wound.

A few days later, the wound appeared free of infection and any nonviable tissue. Santyl ointment was used during this period to help remove non-viable tissue and promote granulation tissue." At this stage, the wound needed a treatment modality to increase granulation tissue. Options were topical growth factor therapy and negative pressure wound therapy. Given the size and depth of the wound, negative pressure therapy was chosen, and carried on for several weeks. When the granulation tissue completely filled in the wound (**Figure 3**), other modalities to assist in epithelialization could be considered. Skin grafting may play an important role, either autologous or a skin substitute, of which there are many. They typically take longer to heal than the patient's own skin; however, they can obviate the need for another surgery, and another open wound (the donor site). Skin substitutes are expensive, and usually require more than one application.

A skin substitute was utilized in this case. The wound subsequently went on to complete closure in four months (Figure 4).

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Disclosures

None

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