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Review

# Current concepts in the surgical management of acute diabetic foot infections

Javier La Fontaine[a,*], Kavita Bhavan[b], Talal K. Talal[c], Lawrence A. Lavery[a]

[a] University of Texas Southwestern Medical Center, Department of Plastic Surgery, Dallas, TX, United States
[b] University of Texas Southwestern Medical Center, Department of Medicine, Infectious Disease, Dallas, TX, United States
[c] Hamad Medical Corporation, Department of Medicine, Diabetic Foot and Wound Care Center, Doha, Qatar

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ABSTRACT

Diabetic foot complications are common, costly, and difficult to treat. Peripheral neuropathy, repetitive trauma, and peripheral vascular disease are common reasons that lead to ulcers, infection, and hospitalization. Individuals with diabetes presenting with foot infection require optimal medical and surgical management to accomplish limb salvage and prevent amputation; aggressive short-term and meticulous long-term care plans are required. Multiple classification systems have been recommended to ease the understanding and the management of these infections. Multi-disciplinary approach is the mainstay for a successful management. Such teams typically include multiple medical, surgical, and nursing specialties across a variety of public and private health care systems. This article is an overview in how to medically and surgically approach the diabetic foot infection with emphasis in soft tissue infection.



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## 1. Introduction

In the United States alone, there are 23.6 million (7.8% of the population) people affected by diabetes and its attendant increased mortality [1]. Plantar ulceration has been reported as the most frequently common diabetic foot complication with 20%–25% of all hospital admissions owing to foot problems [2]. Approximately 56% of foot wounds become infected and foot complications are associated with approximately one quarter of all hospital days for people with diabetes [3]. Approximately 15% of people with diabetes will develop foot ulceration during their lifetime and two-thirds of the ulcers will be complicated with osteomyelitis [4]. Early diagnosis is the mainstay to successful and to prevent the progression of infection, for instance in cases with resistant bacterial strains and immunocompromised individuals. Although, *Staphylococcus aureus* is the most common infecting organism in diabetic foot infections (DFI), as many as 46% of *S. aureus* isolates are methicillin resistant *Staphylococcus aureus* (MRSA) [5]. This review is an overview in how to approach to the diabetic foot infection with emphasis is soft tissue infection with medical and surgical approach.

* Corresponding author at: University of Texas Southwestern Medical Center, Department of Plastic Surgery, 5323 Harry Hines Boulevard, MSC 8560, Dallas, TX, United States. Tel.: +1 214 648 9103; fax: +1 214 648 2550.
*E-mail address:* javierlafontaine@gmail.com (J. La Fontaine).

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**Table 1**
University of Texas wound classification system.

| | 0 | 1 | 2 | 3 |
|---|---|---|---|---|
| A | Pre- or post ulcerative lesion completely epithelialized | Superficial wound not involving tendon, capsule or bone | Wound penetrating tendon or capsule | Wound penetrating to bone or joint |
| B | With infection | With infection | With infection | With infection |
| C | With ischemia | With ischemia | With ischemia | With ischemia |
| D | With infection and ischemia | With infection and ischemia | With infection and ischemia | With infection and ischemia |

## 2. Risk factors

Neuropathy and immunopathy are the major contributing factors that attribute to patients acquiring an infection [6]. More often than not peripheral vascular disease coexists with neuropathy playing a major role in the healing potential. Neuropathy predisposes the foot to infections while vasculopathy and immunopathy determine the outcomes [7]. About 50% of all patients with diabetes experience lack of sensation which combined with repetitive stress leads to tissue break down and then eventually infection [8]. Patients with diabetic neuropathy alone are 1.7 times more likely to develop pedal ulcerations [9]. The etiology of diabetic neuropathy is not clearly understood, but one major theory has been described as angiopathy of the vasa nervosum causing ischemia of the nerve. Evidence of the metabolic disturbance has been found, including the accumulation of intraneural sorbitol and glycosylation of the nerve protein and reduction of axonal transport. Loss of protective sensation, combined with recurrent trauma, is the primary mechanism of tissue breakdown in the foot [10] Poor glycemic control has been associated with the predisposition of diabetic patients to infections. The presences of high levels of glucose in the bloodstream decrease the ability of leukocyte chemotaxis, and phagocytosis [11]. In general, blood glucose of 250 or more places the patients in a compromised situation to develop an infection.

## 3. Evaluation

Wound infection can be defined as the pathologic presence of bacteria in a wound which elicit an inflammatory response via white blood cells [12]. Knowing that all skin wounds contain microorganisms, infections must be diagnosed clinically rather than microbiologically. Also, patients with an infected foot ulcer may have diminished signs of inflammatory reaction possibly due to peripheral neuropathy or ischemia. Clinical signs of local foot infection include erythema, edema, purulence, warmth, and often pain even in the presence of neuropathy (see Table 2). Systemic signs of toxicity are uncommon in diabetic foot infections. Most patients are afebrile without elevated white blood cell count, or elevated sedimentation rate, or C-reactive protein and report no pain. If any these symptoms are present, then a severe infection most likely is present (see Table 2) [13]. Once there is a suspicion of clinical infection, then microbiology is a useful tool to determine the causative agent once a clinical diagnosis of infection is made. At this time a treatment plan should be implemented. Generally, the treatment option will be dependent upon if the infection is mild, moderate or severe. Assessing the severity of the infection helps to determine the need for hospitalization, the potential necessity and timing of surgery, and the likelihood of amputation. As a general rule, mild diseases can be treated with oral antibiotics in the outpatient setting, whereas moderate and severe disease will usually require intravenous antibiotic therapy and hospitalization [14].

Commonly, patients with a DFI present with laboratory results such as white blood cell count within normal limits even when a severe infection may be present [15]. It has been suggested that patient with longstanding diabetes may not mount an effective immunological response to invading pathogens [13]. According to Kaleta in 2002, he performed a retrospective chart review that revealed patients with a sedimentation rate of 70 or higher were noted to have osteomyelitis and Armstrong found that 82% of the patients with osteomyelitis had normal oral temperatures [15,16]. Among currently available imaging modalities, MRI provides the greatest accuracy (i.e., combined sensitivity and specificity) for the detection of bone infection in the diabetic foot. One recent meta-analysis reported a specificity of 82.5% and 90% sensitivity [17]. Characteristic findings of diabetic foot osteomyelitis on MRI include decreased signal intensity of affected bone on T1-weighted images and increased intensity on T2-weighted. However, it is important to note that MRI is usually not needed as a first-line of imaging in cases of DFI. Initial imaging should include weight-bearing plain radiographs to assess for fractures or dislocations, foreign bodies, subcutaneous emphysema, and associated degenerative changes. CT can be used to further evaluate the bony architecture. Suspicion of osteomyelitis may warrant additional evaluation with MRI. With either of these advanced imaging techniques, consideration must be given to the patient's renal function before administration of contrast material. Nuclear medicine studies, including technetium T c-99m and indium-In111-labeled leukocyte scans can be used in the setting of equivocal findings or relative contraindications to other imaging techniques. However, in DFI involving the soft tissues most of ancillary studies are not helpful [18,19].

In addition, a thorough and careful vascular examination must be performed. At minimum, this should include documentation of dorsalis pedis and tibialis artery pulses, with Doppler ultrasound and ABI assessment as needed. Further imaging, including CT angiography and magnetic resonance angiography, may be of benefit in terms of preoperative planning and does not have the risks inherent in invasive angiography.

**Table 2**
Classification of diabetic foot infection.

| Clinical manifestations of infection | Infection severity | PEDIS grade |
|---|---|---|
| Wound lacking purulence or any manifestations of inflammation. | uninfected | 1 |
| Presence of: 2 manifestations of inflammation (purulence, or erythema, pain, tenderness, warmth, or induration), but any cellulitis/erythema extends, 2 cm around the ulcer, and infection is limited to the skin or superficial subcutaneous tissues; no other local complications or systemic illness. | Mild | 2 |
| Infection (as above) in a patient who is systemically well and metabolically stable but which has: 1 of the following characteristics: cellulitis extending 12 cm, lymphangitic streaking, spread beneath the superficial fascia, deep-tissue abscess, gangrene, and involvement of muscle, tendon, joint or bone. | Moderate | 3 |
| Infection in a patient with systemic toxicity or metabolic instability (e.g., fever, chills, tachycardia, hypotension, confusion, vomiting, leukocytosis, acidosis, severe hyperglycemia, or azotemia). | Severe | 4 |

Adapted from IDSA guidelines: Lipsky et.al.: Diagnosis and treatment of diabetic foot infections, CID 2004:39.