

Diabetic foot infections: a team-oriented review of medical and surgical management

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As the domestic and international incidence of diabetes and metabolic syndrome continues to rise, health care providers need to continue improving management of the long-term complications of the disease. Emergency department visits and hospital admissions for diabetic foot infections are increasingly commonplace, and a like-minded multidisciplinary team approach is needed to optimize patient care. Early recognition of severe infections, medical stabilization, appropriate antibiotic selection, early surgical intervention, and strategic plans for delayed reconstruction are crucial components of managing diabetic foot infections. The authors review initial medical and surgical management and staged surgical reconstruction of diabetic foot infections in the inpatient setting.

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Foot ulceration and infection occur frequently and can deteriorate rapidly in the insensate diabetic patient. Frequently, infections in this patient population are masked by neuropathy and complicated by concomitant metabolic derangements, peripheral arterial disease, and immunocompromise (1, 2). Hence, management of these patients requires a like-minded, multidisciplinary team strategy for medical stabilization and infection control via adequate surgical debridement, antibiotic selection, and delayed reconstruction to achieve functional limb salvage (3–5).

Multiple classification systems exist for diabetic ulceration and diabetic foot syndrome, which inherently overlap. The most widely recognized classification is the Wagner system, which grades ulcers from 0 to 5 based largely on ulcer depth and severity (6). Although easy to remember, this system fails to address peripheral arterial disease, peripheral neuropathy, ulcer dimensions, or the extent of infection.

Other diabetic ulcer descriptors that are commonly used in the literature and have been validated include the University of Texas (UT) Classification and the PEDIS classification. The UT system is easy to use and addresses not only the wound depth, but also the presence or absence of infection and the presence or absence of

ischemia (7). The PEDIS system is even more detailed and was developed by the International Working Group on the Diabetic Foot primarily for research purposes. PEDIS is a detailed classification system that describes each of the following ulceration characteristics on a scale of 1 to 4, depending on severity: Perfusion, Extent (or size), Depth, Infection, and Sensation (8).

Initial evaluation: determination of infection severity

Although the classification of ulceration itself is important, the simple stratification of the diabetic patient's overall clinical status takes obvious precedence in the emergency or inpatient setting. The Infectious Disease Society of America delineates diabetic foot infections into four straightforward categories in their published guidelines in 2004 (9). Infections are described based on the composite of the clinical appearance of the foot and the systemic condition of the patient: uninfected (lacking purulence or inflammation); mild (infection limited to skin/subcutaneous tissue, peri-wound erythema of less than 2 cm, and less than two signs of inflammation); moderate (involvement of muscle, joint, bone, or presence of lymphangitis, peri-wound cellulitis beyond 2 cm, or gangrene); or severe (infection in a patient with systemic

toxicity or metabolic instability) (9). A severe diabetic foot infection, which includes wet gangrene, necrotizing fasciitis, or an abscess resulting in systemic toxicity can quickly become limb- or life-threatening and requires early and appropriate antibiotic selection and surgical debridement. In addition, the authors categorize an infected ulcer with an associated unstable Charcot deformity as a severe infection given the high morbidity associated with this clinical presentation.

Diabetic patients may or may not mount a fever, even in the presence of severe infection, but may manifest other constitutional symptoms. Hypotension, tachycardia, and severe unexplained hyperglycemia are often noted, but greater than 50% of limb threatening infections do not manifest systemic signs or symptoms (10). Initial blood work includes a basic metabolic panel, complete blood count with differential, urinalysis, and blood cultures. A glycosylated hemoglobin, erythrocyte sedimentation rate, and C-reactive protein are often added for a more complete assessment of the glycemic control and degree of systemic response at the time of presentation. Evaluation of the overall nutritional status of the patient via serum albumin and pre-albumin levels is also important to optimize wound healing conditions in the setting of increased metabolic demands.

Additionally, the evaluation of initial radiographs is crucial in determining the severity of the infection. Osteomyelitis, gas in the soft tissues, or the presence of a foreign body implies violation and involvement of deep soft tissue planes. In the neuropathic population specifically, radiographs should be assessed for osseous deformities, fractures, and/or dislocations that could indicate acute Charcot neuroarthropathy.

It goes without saying that the physical evaluation of the foot is paramount for the determination of the severity of infection. Careful palpation for fluctuance or tunneling wounds is important because these imply deep space infections that have the potential to spread more easily along tissue planes (11, 12). Sensation must also be examined closely; pain on palpation of any area of an insensate foot is concerning for more severe infection (12).

The violation of dermal and subcutaneous layers is not uncommon in diabetic foot ulceration and an evaluation of the depth of ulceration is important. If a clinically infected ulceration probes to bone on examination, studies have demonstrated 89–95% positive predictive value (PPV) of this test for contiguous osteomyelitis (13, 14). Other studies involving both infected and non-infected ulcerations have shown a lower PPV but a greater than 91% negative predictive value (14–16). Taken together, these data imply that, in a clinically infected ulcer, a positive probe-to-bone test has a high correlation with underlying osteomyelitis. Importantly though, a negative probe-to-bone test in the setting of a clinically

infected ulcer does not and cannot rule out underlying osteomyelitis (17) (see Fig. 1).

Furthermore, the initial evaluation of the presence or absence of limb ischemia is crucial to categorize the superimposed risk of limb loss. The degree of impaired distal perfusion significantly increases the overall severity of the infection (2). The surgeon should palpate for the presence or absence of pedal and popliteal pulses, and signs of ischemia, necrosis, and gangrene should be noted. A cursory handheld Doppler exam, performed by the surgeon in the emergency department, can give a gross idea of the degree of impairment of distal perfusion without any delay in the progression of treatment. Further vascular workup and intervention are determined once local control of infection via surgical debridement is performed.



Fig. 1. A clinical presentation of a diabetic Charcot foot with plantar ulceration that will require staged osseous and soft tissue reconstruction.

Initial management: antibiotic selection and medical stabilization

In the emergency department, initial parenteral antibiotic selection ought to provide a broad-spectrum coverage of Gram positive, Gram negative, and anaerobic organisms (18–22). Patients with diabetic foot infections ought to receive early consultation with a podiatric surgeon and early cardiac risk stratification by the medicine team so as to determine the severity of the infection and the timing for surgical intervention, when appropriate.

For severe diabetic foot infections, one, or a combination, of the following broad-spectrum antibiotics are recommended: piperacillin-tazobactam, vancomycin, levofloxacin or ciprofloxacin with clindamycin, or imipenem-cilistatin (18). Characteristic odors and personal history of resistant bacterial infections may also contribute to the selection of initial antibiotic agents. Methicillin-resistant *Staphylococcus aureus* (MRSA) is frequently a pathogen in community as well as hospital-acquired infections and has been associated with poor clinical outcomes (23, 24). For this reason, agents with MRSA coverage specific to hospital antibiograms are often started empirically.

Many emergency departments and some specialists immediately obtain superficial soft tissue cultures from diabetic foot wounds. Some literature reports similar findings between superficial swab cultures obtained from chronic wounds and those swabs obtained via deep tissue culture techniques (25–27). Other investigators have postulated that the pathogenic concurrence between swab and biopsy specimens is not perfect but is usually sufficient (28, 29). Conversely, many believe that superficial swab cultures of infected ulcers only complicate the evaluation of the patient, as these cultures may not convey anaerobic and fastidious bacterial presence (25–35).

The swab culture debate is important because severe diabetic foot infections are frequently found to be polymicrobial, with mixed aerobic and anaerobic species of bacteria and occasionally fungus (25, 27, 28). Mild or moderate infections, on the other hand, often have one primary pathogen, which is most frequently *S. aureus* (26, 36, 37). Additionally, the increasing prevalence of MRSA in diabetic foot infections has been associated with wound healing complications and a higher risk of lower extremity amputation (38, 39). Without a doubt, definitive antibiotic therapy is based on culture and sensitivity results from intra-operative cultures and the input of the infectious disease members of the team to determine which organisms are true pathogens.

In the diabetic patient, the degree of end-organ dysfunction frequently affects multiple facets of medical and surgical management during the hospitalization. Antibiotic dosing, cardiac function parameters, metabolic instability, ketoacidosis, distal lower extremity

perfusion, immunosuppression, nutritional status, and healing potential of the lower extremity are all frequently compromised.

The goal of medical management for the patient with a severe diabetic foot infection is to regulate and normalize the metabolic and hemodynamic derangements present and to prevent further decompensation (40, 41). Commonly, severe hyperglycemia, ketoacidosis, hyperosmolality, and azotemia are present at the initial presentation (42), especially in severe diabetic foot infections. Additionally, accompanying osmotic diuresis and/or fluid deprivation from vomiting may cause hyponatremia, hypokalemia, and acute-on-chronic renal insufficiency. Moreover, borderline hypokalemia is often treated so as to prevent the anticipated decrease in serum potassium after correction of hyperglycemia.

The medicine team augments cardiac and renal protection with careful control of blood pressure, initiation of angiotensin-converting enzyme inhibitor therapy, and diligent parental fluid management. Previous records, especially cardiac stress tests and cardiac echography, are comprehensively reviewed and the need for repeat cardiac studies is urgently evaluated if general anesthesia is needed for initial surgical decompression and drainage of the infection. Because infection and gangrene result in increased cardiac demands, a target hematocrit is often established based on the patient's cardiac risk profile. Most diabetic patients with severe infections have anemia of the chronic disease at baseline and will be expected to lose additional heme with repeat surgical debridement, but transfusion needs are assessed on an individualized basis.

As with all diabetic admissions, oral hyperglycemic agents are held and glycemic control is obtained through an insulin correction scale according to the insulin sensitivity factor (ISF). The goal of such a scale is to maintain an inpatient's premeal blood sugar range between 80 and 140 mg/dL and their maximum random blood sugar level below 180 mg/dL. An insulin correction scale considering an ISF is safer, more efficient, and more patient-specific than the standard sliding scale correction and because it is based on the patient's physiologic demand. Because infection typically perpetuates hyperglycemia, the adaptability and ease of dosing adjustment afforded by insulin facilitates tight glycemic control in the inpatient setting.

Additionally, the potential for iodinated contrast administration during the hospitalization, especially in the setting of critical limb ischemia, must be expected. The limb salvage team needs to appreciate and foster the facilitation of metabolic control of the patient through surgical control of the infection, as infection is the primary etiology of the severe metabolic disturbance (10).

Initial surgical decompression and debridement

After the patient is medically stabilized, initial surgical debridement is performed with the goal of resecting all non-viable tissue and decompressing gross abscesses. In severe diabetic foot infections, all members of the team must understand that early decompression and drainage is crucial to successful control of the infection and must occur as soon as the patient's metabolic disturbances have been addressed. Even in mild or moderate diabetic foot infections, the authors advise caution in ordering advanced imaging studies prior to initial surgical intervention, as these may unnecessarily delay surgery.

In mild or moderate diabetic foot infections, local anesthesia may be used, but often, general anesthesia is warranted in severe infections, as the depth of infection and fascial spread may be extensive. Resection of all sloughed and congested skin and the exploration of all sinus tracts are essential and blunt dissection is used to determine the extent of involvement of the fascial planes. Tissue planes that are easily violated with minimal pressure during manual exploration indicate the possibility of necrotizing fasciitis, which has a significantly worse prognosis (43).

After thorough exploration of the affected pedal compartments, the surgeon is able to determine the necessary amputation level or the degree of wide excision needed. All non-viable and infected soft tissue and bone must be excised during the initial debridement to enable wound healing. Additionally, the degree of intra-operative bleeding after resection of non-viable tissue must be assessed (44–47). Exposed tendons should be excised if proximal migration of the infection is suspected and all marginal-appearing tissue should be resected to foster better wound bed granulation. Deep soft tissue and bone intra-operative cultures are sent to microbiology and bone may be sent for histopathological examination if osteomyelitis is suspected (see Fig. 2).



Fig. 2. A clinical presentation of a staged diabetic limb salvage procedure including aggressive initial surgical debridement followed by a partial calcaneectomy.

Definitive surgical management

Many patients with life- or limb-threatening diabetic foot infections have concomitant peripheral arterial disease that complicates their wound healing potential. For this reason, if pedal pulses are non-palpable or mono/biphasic via the handheld Doppler signals, or if minimal bleeding is visualized during the initial surgical debridement, non-invasive vascular studies should be ordered without delay following initial debridement. Ankle and toe brachial indices, pulse volume recordings, and transcutaneous oxygen pressures provide valuable information that ultimately determines the appropriateness of vascular surgery consultation and invasive vascular studies.

The Ankle Brachial Index (ABI), or the ratio of the systolic ankle blood pressure to the standard systolic brachial blood pressure, is a useful screening test because any result less than 1.0 (in a diabetic or non-diabetic patient) strongly suggests significant peripheral arterial compromise. Unfortunately, the ABI may underestimate the severity of arterial insufficiency in the diabetic population, as it is significantly affected by incompressible calcified vessels. Calcification of the tunica media, called Moneckberg's sclerosis, is commonly seen in diabetic patients and results in falsely elevated ABI values. Hence, despite clinical signs of impaired perfusion, the ABI may 'appear' to be within normal limits (48). Furthermore, segmental decreases of 20–30 mmHg between proximal and distal arterial segments may represent occlusive peripheral vascular disease in the affected arterial segment and may warrant further evaluation by the vascular surgeons.

Because of the inherent weakness of the ABI for vascular screening in diabetic patients, qualitative wave forms and toe-brachial pressure indices are typically included in the non-invasive vascular exam (49). Additionally, some institutions also employ transcutaneous oxygen pressure measurements (TCPO₂), which can be useful in predicting the wound healing capacity at different levels in the foot (50–52). The TCPO₂ values greater than 30 mmHg suggest significantly improved chances of healing compared with those less than 30 mmHg (50–52). As with all measurements, the TCPO₂ values should not be evaluated in isolation as an indicator of healing. In fact, the presence of edema and cellulitis affects TCPO₂ readings significantly, and caution must be exercised with interpretation in these situations.

In the severe diabetic foot infection, suboptimal non-invasive study results potentiate timely vascular surgery consultation and, often, angiography (53). Revascularization, if needed, is ideally performed within 1–2 days after the initial surgical debridement (54, 55). Percutaneous transluminal angioplasty is now the typical initial intervention in the salvage of the ischemic diabetic limb, but may be followed, if necessary, by open distal arterial bypass (2, 55).

Regardless of the type of intervention, adequate perfusion is essential before definitive soft tissue reconstruction can occur. If osteomyelitis is confirmed from initial deep cultures or histopathology, further aggressive resection of all affected bone is warranted. Depending on the bone affected, location, and overlying soft tissue envelope, proximal amputation may suffice. When resection of osteomyelitis is more extensive, involving multiple bones, associated with Charcot neuroarthropathy, or results in significant instability in the foot, adjunctive implantation of organism-specific antibiotic beads is often performed. Provisional soft tissue closure over the beads is often obtained with local soft tissue coverage, but may also employ negative pressure wound therapy dressings or external fixation to reduce large soft tissue defects (56).

Parenteral antibiotics are continued in the outpatient setting per infectious disease recommendations. Strict non-weightbearing and biweekly office follow-up visits occur until an explantation of the beads is planned. Antibiotic beads may be left in place for time periods ranging from 2 weeks to permanently, but explantation typically occurs approximately 6 to 9 weeks after insertion of the beads (57, 58).

After eradication of all grossly infected soft tissue and osteomyelitis, staged reconstruction is planned. Significant osseous involvement may potentiate underlying instability and cause further deformity and morbidity in this high-risk patient population. For this reason, adjunctive osseous procedures may be warranted to restore stability and address deformity in the insensate foot in order to minimize ulcer recurrence. The selection of osseous procedures is patient and pathology dependent and may range from simple exostectomy to extended medial column arthrodeses with internal and/or external fixation methods. In diabetic foot infections and ulcerations, soft tissue management is as important as osseous reconstruction. The reconstructive pyramid, an algorithm that details the soft tissue reconstructive options from simplest and most utilized to most complex and least employed, is frequently referred to during preoperative planning.

Often, significant tissue deficits preclude primary closure following aggressive surgical debridement of severe diabetic foot infections. When feasible, the least invasive methods of coverage are employed, such as delayed primary closure or partial closure with wound healing adjuncts such as negative pressure wound therapy. Many wounds are not amenable to delayed primary closure and require plastic surgical techniques including, from least to most complicated, split thickness skin grafting (59), local rotational or advancement flaps (60–63), muscle flaps (64–67), or pedicle flaps (68–70) (see Fig. 3).



Fig. 3. A clinical presentation of a severe diabetic foot infection that necessitates urgent/emergent surgical debridement and/or amputation.

Goals for surgery are discussed in-depth on a patient-by-patient basis, and family presence in these discussions is strongly encouraged. In general, in previously or potentially ambulatory patients, the ultimate goal of both soft tissue and osseous reconstruction is restoration of a functional, plantigrade, shoeable or braceable foot that is free of ulceration. In previously non-ambulatory patients, the goal of surgery is eradication of infection and provision of a stable, ulcer-free foot to aid in transfers.

Discussion

Diabetic limb salvage requires the collaboration of a finely tuned, multidisciplinary team and the implementation of a logical stepwise approach for medical and surgical approaches to the severe infection. The goal of limb salvage is to maintain – or provide – a limb that is functional, plantigrade, durable, stable, and shoeable or braceable. Patient stabilization, medical optimization, aggressive surgical debridement, parenteral antibiotics, vascular assessment/intervention, and delayed soft tissue and osseous reconstruction are all critical components of a successful treatment algorithm for the severe diabetic foot infection.

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