21 Diabetic foot ulcer

21.1 Introduction

Foot ulcers are the most common medical complications of patients with diabetes, with an estimated prevalence of 12-15% among all individuals with diabetes.[1] Diabetic foot ulcers are responsible for more hospitalizations than any other complication of diabetes. Ulcerations can have potential devastating complications as they cause up to 90% of lower extremity amputations in patients with diabetes.

Several factors are involved in the decreased healing potential of a diabetic foot, all of which stem from the metabolic disorders associated with diabetes. The most important of these factors are:

- Level of uncontrolled hyperglycemia
- Reduced circulation and arterial blood flow
- Nutrition status
- Inability to offload the affected region of the foot
- Presence of infection[2]

Failure of the wound healing process, in particular repair of lost extracellular matrix that forms the largest component of the dermal skin layer, is one of the primary culprits in diabetic foot ulcers. The most predictive marker for wound healing is serum albumin, followed by total lymphocyte count (TLC). Commonly accepted levels of these markers for wound healing are a serum albumin of 3.0 g/dL and TLC >1,500/mm³. Additional useful laboratory values to assess wound ulcers include the nutrition marker pre-albumin; the inflammatory markers serum white blood cell count, C-reactive protein, and erythrocyte sedimentation rate; and the glycemic markers serum glucose and hemoglobin A1C.
21.2 Pathophysiology

Diabetic foot ulcers are the result of the combined effects of diabetes-related vascular disease and neuropathy. Patients with diabetes have a higher incidence of atherosclerosis, the thickening of capillary basement membranes, arteriolar hyalinosis, and endothelial proliferation. They may develop atherosclerosis of large and medium-sized arteries, such as aortoiliac and femoropopliteal vessels, but atherosclerosis of the infrapopliteal segments is particularly common. Combined with digital artery disease, ulcers can develop and quickly progress to gangrene in the absence of adequate blood flow.

Peripheral neuropathy is present in 60% of patients with diabetes and 80% of patients with diabetes who have foot ulcers. Neuropathy in these patients is a multifactorial process and is thought to result from a combination of vascular disease occluding the vasa nervorum, endothelial dysfunction, deficiency of myoinositol-altering myelin synthesis and diminishing sodium-potassium adenine triphosphatase activity, chronic hyperosmolarity, and effects of increased sorbitol and fructose.[2] Decreased sensation in the foot predisposes the patient with diabetes to unnoticed injuries and fractures that overload the skin and lead to ulceration.

21.3 Clinical Presentation

Patients with diabetic foot ulcers often present with complaints of increased pain, swelling, discharge, and a foul-smelling odor from the affected foot. In patients with severe neuropathy, however, foot ulcers are often found incidentally by the patient or primary caregiver. On history intake, it is critical to evaluate for previous ulcerations, medical comorbidities, level of diabetic control and monitoring, and tobacco and intravenous drug use.

21.4 Microbiology

Most diabetic foot ulcers present as polymicrobial infections. The most common pathogens seen are aerobic Gram-positive cocci, in particular *Staphylococcus aureus*, and Gram-negative rods such as *Pseudomonas aeruginosa*. Infection with anaerobic organisms such as *Clostridium perfringens* may lead to foot ischemia or gangrene. Deep wound cultures and blood cultures are useful to help direct antibiotic therapy and monitor the presence of early sepsis.

21.5 Physical Examination

The foot examination should begin with noting the condition of the skin involved in and surrounding the ulcer. The key features of the ulcer to note during exam are:

- Depth
- Location
- Drainage
- Signs of infection
- Presence of viable and nonviable gangrenous tissue (Figure 1)
Osteomyelitis is present in approximately 70% of ulcers that can be probed to bone.[4] A comprehensive neurovascular exam should be performed with careful attention to pulses and level of neuropathy, which can be determined with a standardized 10-gauge monofilament. Ulcer classification can be achieved using the Wagner classification system and the Brodsky depth/ischemia classification.

![Figure 1](image1.png)

Figure 1. Lateral (left) and plantar (right) views of a patient with diabetes who presented with severe ulceration over the right foot fifth metatarsal head after wearing tight-fitting shoes for the previous 2 weeks.

## 21.6 Classification

### 21.6.1 Wagner Ulcer Classification System [4]

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Skin intact</td>
</tr>
<tr>
<td>1</td>
<td>Superficial ulcer</td>
</tr>
<tr>
<td>2</td>
<td>Deeper, full-thickness extension of ulcer</td>
</tr>
<tr>
<td>3</td>
<td>Deep abscess or osteomyelitis associated with ulcer</td>
</tr>
<tr>
<td>4</td>
<td>Partial forefoot gangrene with ulcer</td>
</tr>
<tr>
<td>5</td>
<td>Extensive foot gangrene with ulcer</td>
</tr>
</tbody>
</table>

### 21.6.2 Brodsky Depth/Ischemia Classification [5]

Depth
<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>At-risk foot with previous ulcer that may cause new ulcer</td>
<td>Patient education, appropriate shoe wear and insoles</td>
</tr>
<tr>
<td>1</td>
<td>Superficial non-infected ulcer</td>
<td>Custom shoe wear, walking brace, or total contact cast</td>
</tr>
<tr>
<td>2</td>
<td>Deep ulcer with tendon or joint exposed (+/- infection)</td>
<td>Wound care, pressure relief, and surgical incision and debridement as needed; antibiotics if infection present</td>
</tr>
<tr>
<td>3</td>
<td>Extensive ulcer with bone exposed or deep abscess</td>
<td>Surgical incision and debridement, ray or partial foot amputation as needed; intravenous antibiotics</td>
</tr>
</tbody>
</table>

### Ischemia

<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>No ischemia</td>
<td>Adequate blood flow for healing</td>
</tr>
<tr>
<td>B</td>
<td>Ischemia, no gangrene</td>
<td>Vascular studies (Doppler ultrasonography with digital arterial pressures, transcutaneous toe oxygen measurement, arteriography); vascular reconstruction as needed</td>
</tr>
<tr>
<td>C</td>
<td>Partial forefoot gangrene</td>
<td>Vascular studies; vascular reconstruction with proximal or distal bypass/angioplasty; partial forefoot amputation</td>
</tr>
<tr>
<td>D</td>
<td>Total foot gangrene</td>
<td>Vascular studies; proximal vascular reconstruction, major extremity amputation below or above knee</td>
</tr>
</tbody>
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### 21.7 Vascular Studies

The vascular status of the affected extremity is critical in determining the healing potential of foot ulcers and the need for possible surgical intervention. More than 60% of diabetic foot ulcers have decreased arterial blood flow due to concurrent peripheral vascular disease. Further vascular studies are often needed after examination of vascular status of the posterior tibial and dorsalis pedis arteries with palpation and handheld Doppler ultrasound, if palpation is inconclusive. The ankle-brachial index (ABI) can be easy to obtain and helpful in assessing vascular status, but it is often unreliable and falsely elevated in patients with arterial calcification due to noncompressible vessels.[6] This is commonly found in patients with diabetes or renal failure or in those who are heavy smokers.

Doppler ultrasonography with digital arterial pressures and waveform analysis, transcutaneous toe oxygen measurements (TcPO2), and arteriography can be used to further evaluate and determine the extent of peripheral vascular disease. General cutoff values for proper healing of foot ulcer are an ABI >0.45 with toe pressures >40 mm Hg and a transcutaneous toe oxygen measurement >30 mm Hg.[7]

### 21.8 Imaging
Standard anteroposterior, lateral, and oblique radiographs of the affected foot should be obtained to evaluate for cortical bone abnormalities such as demineralization, erosion, periosteal reaction, lucencies, and osteolysis that would be indicated of osteomyelitis (Figure 2). Cortical disruption may not be apparent until 1-2 weeks after the onset of acute clinical osteomyelitis, however, and early radiographic changes can be confused with those seen in Charcot arthropathy.

Given the low sensitivity and specificity of plain radiographs, advanced imaging with nuclear studies such as technetium (Tc) 99m, gallium (Ga) 67, or indium (In) 111 may be needed to differentiate between soft-tissue infection, osteomyelitis, Charcot arthropathy, or a combination of those disorders. MRI with T1- and T2-weighted images may be helpful to determine the extent of bony and soft-tissue disruption, but MRI cannot differentiate between Charcot arthropathy and osteomyelitis with high specificity [8].

**Figure 2.** Anteroposterior (left) and lateral (right) radiographs of the patient in Figure 1 demonstrating subcutaneous gas extension down to bone with cortical disruption.

### 21.9 Conservative Treatment

Non-operative management of diabetic foot ulcers focuses on prevention of future ulcers, wound care, assessment of equinus contracture, total contact casting (TCC), walking braces, and debridement. Foot care education combined with special footwear, increased surveillance, and a focus on strict glucose control can significantly reduce the incidence of ulcer progression by 50% to 60%.[9] Wound care is an essential first step in the management of foot ulcers and should achieve the following goals:

- Off-load pressure
- Provide a protective barrier
- Absorb exudate
- Provide a moist environment
- Provide antibiosis
When local wound care fails, TCC is the gold standard for further off-loading plantar ulcers by redistributing weight-bearing forces across the entire plantar surface of the foot.[10] Prerequisites to TCC are a foot with has an adequate blood supply and the ability to monitor the patient at intervals of 1-2 weeks.

TCC has been shown to significantly increase the healing rate of neuropathic plantar foot ulcers at 12 weeks compared to removable cast walkers and half-shoes.[11] In addition, TCC has been shown to increase time to healing in neuropathic ulcers and ulcers with moderate ischemia or infection.[12] Various methods of cast padding and application are available, depending on physician preference, with a major variable being cast structure at the ulcer site and whether full contact is used versus wound isolation. Forefoot ulcer pressure is optimized when TCC utilizes wound off-loading, with the cast providing total contact everywhere except for the wound site, which is mechanically isolated.[13] Complication rates with TCC range from 11% to 30% in high-risk patients with multiple comorbidities, with the majority of complications being minor and due to dermal abrasions and minor pressure ulcers.[14]

Prior to casting, it is important to assess patients for equinus contractures due to either a tight Achilles or gastrocnemius tendon. Fixed plantarflexion contractures cause increased forefoot pressures and, therefore, a higher likelihood of developing plantar forefoot ulcers. The Silverskiold test can be used to differentiate isolated contractures of the gastrocnemius from the gastro-soleus complex. Patients with Wagner grade 1 and 2 ulcers and isolated gastrocnemius contracture should be treated with gastrocnemius recession (ie, Strayer procedure) followed by TCC to decrease the risk of ulcer recurrence. Compared with TCC alone, the combination of percutaneous tendo-Achilles lengthening and TCC has been shown to result in a significant decrease in recurrence of diabetic plantar ulcers in patients with gastro-soleus contracture.[15,16]

In patients with Wagner grade 3 or greater ulcers, bedside incision and debridement with combined antibiotic therapy is needed prior to TCC application. Casts must be changed every 2-4 weeks, with repeat debridement as needed until erythema, warmth, and edema have decreased to the level of the non-affected or pre-ulcer state. Radiographs of the foot should be taken during treatment with TCC every 4-6 weeks. In total, TCC generally continues for up to 4 months. When the active inflammatory disease phase has resolved, patients can be fitted with an orthotic brace or walker followed by a custom shoe with orthoses.

An alternative to TCC is a prefabricated walking brace that can be used to decrease forefoot and midfoot plantar pressures. Walking braces allow more frequent wound surveys and application of a wider variety of local wound dressings. Braces are ineffective in the presence of severe foot deformities, however, and are often uncomfortable and burdensome for patients to use regularly.

If conservative management fails, more extensive ulcer incision and debridement should be performed to remove all necrotic tissue and leave a healthy tissue base [7]. Progression of the extent of the ulcer can necessitate further debridement in the operating room.

## 21.10 Operative Treatment

Surgical intervention for diabetic foot ulcers begins with appropriate soft-tissue management consisting of:

- Drainage of deep infections
- Removal of necrotic tissue
- Decreasing wound tension [5]
Reduction of internal foot pressures often requires bony correction or realignment arthrodeses. Gastrocnemius recession or Achilles tendon lengthening can assist in further reducing forefoot pressure from fixed plantar contractures. In cases of frank osteomyelitis, operating room culture should be taken via aspiration, curettage, or biopsy rather than swab. Partial or complete amputation often results from severe progression of ulceration into the surrounding soft-tissue and bone.

Overall, diabetes is the predominant etiology for non-traumatic lower extremity amputations in the U.S., with more than 80,000 diabetes-related amputations each year.[17] Half of all non-traumatic amputations are a result of diabetic foot complications, and the 5-year risk of a contralateral amputation is 50%.[18] In patients with diabetes who have peripheral neuropathy and foot ulceration, the recurrence rate is close to 70% and the amputation rate is approximately 12%, even if successful management results in healing of the ulcer.[19]

Once it is determined that an amputation is required, the vascular status of the major arteries (dorsalis pedis, posterior tibial, popliteal) greatly impacts the appropriate level of amputation [20]. The absence of a dorsalis pedis or posterior tibial pulse with an intact popliteal pulse often necessitates a below-the-knee amputation; vascular bypass is not feasible and the healing potential with more distal amputations is significantly compromised. In the combined absence of popliteal, posterior tibial, and dorsalis pedis pulses, it is possible that a proximal vascular lesion is present that may be reconstructable.

Several amputation options exist, ranging from hallux amputation to below-the-knee amputations, and special considerations must be taken into account for each procedure.

- With **hallux amputation**, there is resultant increases pressures under the first metatarsal, lesser metatarsal heads, and remaining toes which can lead to metarsalgia and higher risk of recurrent ulceration.[21]

- **Ray amputations** are generally more functional than transmetatarsal (TMT) amputations, but forefoot stability is compromised when more than two rays are resected.[22]

- **Lateral foot amputations** are better tolerated than medial amputations, and it is important to preserve the bases of the metatarsals during ray amputation to maintain Lisfranc joint integrity.

- Similar to hallux amputations, **first ray amputations** can lead to metatarsalgia, in addition to the added cost of weakening ankle dorsiflexion through loss of the tibialis anterior.

- When ray resections are insufficient, the next step is a **TMT amputation**, which leaves the patient with a residual stump that can bear weight (Figure 3).[22] Assessment of Achilles tightness and overall muscle balance is necessary during TMT to prevent continued plantar flexion and overload of the distal aspect of the weight-bearing stump.
Figure 3. Poorly controlled diabetic patient who underwent TMT amputation which subsequently became re-infected after continued poorly controlled glucose levels. Patient went on to require a Chopart and below the knee amputation during the course of the following year.

- A Chopart amputation has the advantages of keeping the tibiotalar joint and a functional residual limb, but it decreases stability and push-off capability and leads to an equinovarus deformity.[23]

- Syme amputation, the last option before transtibial below-the-knee amputation, has the advantage of retaining a full weight-bearing residual stump for ambulation.[24] Patient selection is important for Syme amputation: Patients must have adequate ambulation potential after surgery, a functional heel pad for anchoring, and adequate vascular and nutritional status for healing.

- Despite the variety of different options available for amputation, many patients with diabetes require revision amputations, often leading to a below-the-knee amputation.

Even with advances in the medical and surgical management of diabetes, the 5-year mortality rate remains poor at approximately 66% after the amputation of a leg — a testament to the debilitating and morbid nature of the disease.

21.11 References


