Diabetic Foot Infections

Laila M. Castellino, MD[a,*], Peter A. Crisologo, DPM[a,b], Avneesh Chhabra, MD[c,d,e], Orhan K. Öz, MD, PhD[c]

KEYWORDS

• Diabetic foot infection • Osteomyelitis • Peripheral vascular disease • Offloading

KEY POINTS

- Diabetes is a growing public health concern, with diabetic foot infections (DFI) being one of the leading causes of lower extremity limb amputation in the United States.
- Accurate diagnosis of DFI requires a combination of clinical, laboratory, and radiologic tests to determine the extent and depth of infection, including the presence of osteomyelitis.
- Increased recognition of the value of multidisciplinary collaborative care can lead to personalized treatment plans, and ultimately improved outcomes and functionality for patients with diabetic foot infection.

INTRODUCTION AND EPIDEMIOLOGY

Diabetes is increasingly recognized as a public health concern worldwide, causing 1.6 million deaths and an increased prevalence of 14% among adults worldwide in 2022, compared to 7% in 1990.[1] In the United States, in 2021, an estimated 29.7 million people—or 8.9% of the population—were diagnosed with diabetes, with variations across race and ethnic groups; prevalence is highest among American Indian and Alaska Native Americans (13.6%), followed by non-Hispanic Blacks (12.1%), Hispanics (11.7%), Non-Hispanic Asians (9.1%), and non-Hispanic Whites (6.9%). Additionally, in 2021, an estimated 97.6 million adults, or more than a third of the US adult population had prediabetes, though only 19% of adults with prediabetes were aware of the diagnosis. The US Centers for Disease Control and Prevention estimates the total direct and indirect costs of diagnosed diabetes in 2022 was $413 billion.[2] Diabetic foot infections (DFI) are the most frequent cause of diabetes-related hospitalization and the most common cause of lower extremity amputation in the general US population.[3,4] The lifetime incidence of foot ulcers among persons with diabetes is between

[a] Division of Infectious Diseases and Geographic Medicine, University of Texas Southwestern Medical Center, Dallas, TX, USA; [b] Department of Plastic Surgery, University of Texas Southwestern Medical Center, Dallas, TX, USA; [c] Department of Radiology, University of Texas Southwestern Medical Center, Dallas, TX, USA; [d] Adjunct faculty, Johns Hopkins University, Baltimore, MD, USA; [e] Walton Center of Neurosciences, Liverpool, UK
* Corresponding author. Division of Infectious Diseases and Geographic Medicine, UT Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390-9113.
E-mail address: Laila.castellino@utsouthwestern.edu

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Abbreviations	
ABI	ankle-brachial index
CNA	Charcot neuroarthropathy
CRP	C-reactive protein
CT	computed tomography
DECT	dual energy CT
DFI	diabetic foot infections
DFO	diabetic foot osteomyelitis
DFU	diabetic foot ulcer
ESR	erythrocyte sedimentation rate
IDSA	Infectious Diseases Society of America
IWGDF	International Working Group on the Diabetic Foot
MRSA	methicillin-resistant *S. aureus*
OM	osteomyelitis
PAD	peripheral arterial disease
PVD	peripheral vascular disease
RCTs	randomized controlled trials
T1W	T1-weighted
WBC	white blood cell

19% and 34%, with widely varying statistics around healing and ulcer recurrence, with some reports of up to 40% recurrence within 1 year.[5] In a prospective series of patients with an infected diabetic foot ulcer (DFU) followed for 12-months, the ulcer healed in only 46% of patients; 17% required a lower extremity amputation, and 15% had died in 1 year.[6] The 5-year mortality rate of patients with a DFI requiring a minor amputation is reported to be as high as 46.2%, in stark contrast to the 5-year pooled mortality of 31% for all reported cancer.[5]

CLINICAL PRESENTATION

Patients with diabetes are prone to develop ulceration due to a combination of factors, including repetitive stress, particularly over bony prominences, foot deformity, peripheral neuropathy, and peripheral artery disease.[7] Most patients develop a superficial wound or ulceration that progresses to cellulitis, abscess, fasciitis, joint infection, and/or osteomyelitis (OM) with or without systemic infection. Patients may not recognize the symptoms of infection, as neuropathy leads to lack of the *gift of pain*, as described in patients with leprosy.[8]

Not all DFU are infected, and diagnosis of DFI is usually made clinically, based on the presence of signs of inflammation such as swelling, erythema, tenderness, warmth, and purulence, as well as presence of systemic symptoms such as fever, tachycardia, and leukocytosis. Several classification systems exist to define the severity of DFI, including systems developed by the International Working Group on the Diabetic Foot (IWGDF) and the Infectious Diseases Society of America (IDSA),[3] the Perfusions, Extent/size, Depth/tissue loss, Infection and Sensation (PEDIS) system;[9] Wagner,[10] University of Texas,[11] and the Society for Vascular Surgery's WIfI (Wound, Ischemia, foot Infection) systems.[12] Classification systems have been used to standardize research methodologies, develop treatment guidance, and prognosticate the risk of amputation and benefit of revascularization. Patients with clinically diagnosed DFI should also be evaluated for predisposing risk factors that led to the development of DFI, such as peripheral vascular disease (PVD), poorly controlled diabetes, pressure ulcer, tinea pedis, maceration, and lymphedema, as these factors contribute to poor healing and risk of recurrence. Additionally, the presence of underlying bone infection

(diabetic foot osteomyelitis, DFO) is important to define, as this has implications for both medical and surgical treatment options.[3]

EVALUATION

Microbiology

DFIs are often polymicrobial with a vast majority having gram-positive organisms particularly *Staphylococcus aureus* and Streptococci. Modern studies report an increase in gram-negative organisms ranging from 30% to 62%.[13–15] The prevalence of pseudomonas varies around the world with tropical and sub-tropical climates reporting higher prevalence compared to northern climates.[16] Even within North America, the prevalence of pseudomonas varies based on climate zones, ranging from 11.6% in hot humid climates to 6.2% in very cold climates.[17] The presence of anaerobes in DFI ranges from 7.7% to as high as 83.8% using newer molecular methods for microbiologic diagnosis, and are usually associated with ischemia, necrosis, deeper lesions, fever, malodor and long duration of ulceration.[18,19] However, the pathogenic role of anaerobes remains poorly understood, as they are present in polymicrobial infections, often in the presence of ischemia and tissue necrosis, which provide a favorable environment. In patients with DFO, bone sample cultures should be obtained to diagnose a pathogen, as soft tissue swab cultures do not reliably corelate to bone culture.[20]

Osteomyelitis

DFO is a much-feared complication and develops in 20% to 50% of patients with DFU.[21,22] Osteomyelitis should be suspected in a patient with ulceration present for many weeks despite proper wound care and offloading, ulcers that are wide, deep, located over bony prominences, with visible bone or presence of a swollen *sausage toe*.[3] Patients with clinically diagnosed DFI, or a long standing DFU that is clinically uninfected, must be evaluated for underlying OM, in order to best determine duration of antimicrobial therapy as well as surgical planning. DFO is usually diagnosed based on a combination of clinical, laboratory, and imaging findings, with multiple tests having varying sensitivity and specificity. The IWGDF/IDSA guidelines suggest starting with a combination of probe-to-bone, plain X-rays, and erythrocyte sedimentation rate (ESR), or C-reactive protein (CRP), all of which are widely available and inexpensive. While the medical community has generally shifted away from the use of ESR for the patient with infection, the body of literature in the diabetic foot space has shown value and use for this peripheral serum biomarker in the diagnosis of foot infection and the stratification of soft tissue infection versus OM.[23–25] Lack of a standard definition of OM, combined with differences in inter-test agreement between various tests and discrepancies in values of ESR and CRP between laboratories can lead to challenges in the diagnostic certainty of DFO. Additionally, imaging alone often cannot distinguish between changes from OM versus Charcot arthropathy. Berendt and colleagues have proposed consensus criteria that assess the probability of DFO, using a combination of tests that are commonly used in the clinical setting (**Table 1**).[26]

Imaging

Anatomic imaging methods

Plain film radiography is the first choice in persons with diabetes and suspected foot infections, as it facilitates identification of bone changes associated with infection as well as changes in soft tissue (**Fig. 1**A). Although conventional radiography is inexpensive, convenient, and fast, there is a delay in reaching the threshold of detection for visualizing OM. Clear evidence of bone erosion in DFO may not occur until 10 to

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Table 1
Proposed consensus criteria for diagnosing osteomyelitis in the diabetic foot

Category	Post-Test Probability of Osteomyelitis	Management Advice	Criteria
Definite ('beyond reasonable doubt')	>90%	Treat for osteomyelitis	Bone sample with positive culture *AND* positive histology *OR* Purulence in bone found at surgery *OR* Atraumatically detached bone fragment removed from ulcer by podiatrist/surgeon *OR* Introsseous abscess found on MRI *OR* Any 2 probably criteria *OR* one probable and 2 possible criteria *OR*, any 4 possible criteria below
Probably ('more likely than not')	51%–90%	Consider treating, but further investigation may be needed	Visible cancellous bone in ulcer *OR* MRI showing bone edema with other signs of osteomyelitis *OR* Bone sample with positive culture but negative or absent histology *OR* Bone sample with positive histology but negative or absent culture *OR* Any two possible criteria below
Possible (but on balance, less rather than more likely)	10%–50%	Treatment may be justifiable, but further investigation usually advised	Plain X-rays show cortical destruction *OR* MRI shows bone edema *OR* cloaca, *OR* Probe to bone positive *OR*, Visible cortical bone *OR* ESR >70 mm/h with no other plausible explanation *OR* Nonhealing wound despite adequate offloading and perfusion for >6 wk *OR* ulcer of >2 wk duration with clinical evidence of infection.
Unlikely	<10%	Usually no need for further investigation or treatment	No signs or symptoms of inflammation *AND* normal X-rays *AND* ulcer present for <2 wk or absent *AND* any ulcer present is superficial *OR* Normal MRI *OR* Normal bone scan

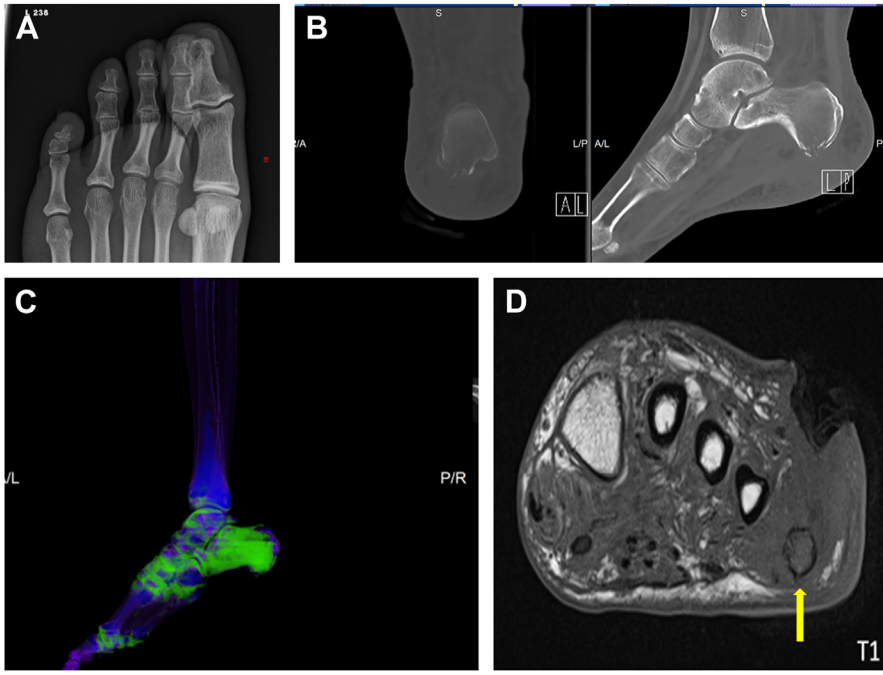


Fig. 1. Anatomic imaging methods. (*A*) Radiograph of osteomyelitis involving the great toe with an osteolytic lesion in the distal phalanx. (*B*) CT images showing soft tissue edema and cortical destruction on the inferior surface of the calcaneus. (*C*) Bone marrow edema in the calcaneus on DECT. (*D*) T1W image showing hypointensity (*arrow*) of bone marrow edema in a metatarsal bone consistent with osteomyelitis.

14 days after infection onset.[27] Visualization of subtle soft tissue changes and non-radiopaque foreign bodies can be challenging.[28]

Computed tomography (CT) imaging offers advantages of 3D and multiplanar reconstructions, can be done relatively quickly, and when properly obtained with intravenous iodinated contrast, can delineate bone changes as well as soft tissue findings, extent of infection, multicompartment involvement, abscess, and air associated with necrotizing fasciitis. Dual energy CT (DECT) and photon-counting CT are new advancements that can create bone marrow 3D maps without bony cortices, showing marrow edema as seen in DFO and inflammatory arthropathies, in addition to conventional CT findings as above.[29–31] DECT also depicts alternative causes of extremity inflammation, for example, gouty tophi deposition in the adjacent soft tissue structures (**Fig. 1**B, C). Additionally, the ability to reduce metal artifact makes this an attractive alternative in patients with contraindications to MRI to assess OM.[29]

Besides X-ray, MRI is the most recommended imaging modality for suspected infection in the diabetic foot.[3,32] The MRI signature of confluent low T1-weighted (T1W) marrow signal intensity alteration in a medullary distribution (**Fig. 1**D) has reported characteristics as high as 95% sensitivity for the prediction of OM, 91% specificity, and 98% negative predictive value for excluding OM,[33] though performance can vary with the experience of the reader.[34] MRI has added advantages of being a radiation-free modality that allows assessment of both cortical and marrow involvement, deep tissue extent of infection, anatomic and functional evaluations (diffusion-weighted imaging and perfusion imaging),[35] and differentiation of alternative diagnoses; for example, gout

and Charcot neuroarthropathy (CNA). However, MRI quality can be degraded in the presence of local metal or may not be possible due to MRI-unsafe devices in-situ. Bone marrow edema is also nonspecific if taken in isolation for characterization of OM, with MRI-scoring systems being developed to standardize the reporting of extremity infections including OM and septic arthritis.[36]

Molecular and cellular imaging methods

Nuclear medicine imaging for suspected foot infection in persons with diabetes includes bone scans, radiolabeled white blood cell (WBC) scans, dual isotope (^{99m}Tc) WBC scan and (^{111}In sulfur colloid) marrow imaging, and 18F-fluorodeoxyglucose PET (FDG PET) imaging.[37]

Routine bone scintigraphy for suspected infection utilizes radiolabeled diphosphonates, which localize to and are retained in bone. The 3-phase bone scan is sensitive for identifying infection but with low specificity. A meta-analysis by Llewellyn reported an average sensitivity of 84.2% and a specificity of 67.7%.[38] In the first or perfusion phase, and in the second phase aka blood pool or tissue phase, both soft tissue and bone infections show greater uptake in the affected foot, while in the third or delay phase, infected bone shows greater uptake than normal bone and surrounding soft tissue (**Fig. 2**A–C).

WBCs can be labeled with either ^{99m}Tc for same day scanning or ^{111}In for delayed scanning or dual radiotracer scanning in the setting of suspected infections in complicated foot problems such as recent trauma, CNA, or orthopedic hardware. WBC scanning is based on the host response of migration of labeled leukocytes to the site of infection. Previous analyses have found that ^{99m}Tc-HMPAO WBC (^{99m}Tc-WBC) scans are highly sensitive (91%) and specific (92%) for the detection of OM.[39] When combined with SPECT/CT ^{99m}Tc-WBC scans show localization of cells to bone in OM versus soft tissue in cases of abscess or cellulitis (**Fig. 2**D–F).

Dual tracer radiolabeled ^{99m}Tc-WBC and ^{111}In-sulfur colloid scanning is used to identify superimposed infection in the setting of CNA or presence of orthopedic hardware that might activate marrow/marrow expansion and cause some low level of leukocyte migration into the site. Diagnoses in the dual isotope dual tracer scan is based on spatial or intensity discordance to identify infection. In the case of active infection, radiolabeled cells will show either uptake in a location different than marrow expansion/activation or much greater intensity than the marrow background (**Fig. 2**G–J).

FDG PET relies on the increased utilization of glucose by inflammatory cells in the site of infection. The higher resolution of PET compared with SPECT combined with CT yields high quality images (**Fig. 2**K–M). In a recently published retrospective study comparing WBC scanning, FDG PET, and MRI in suspected DFI, FDG PET had a specificity of 97.9% for soft tissue infection, and for pedal OM had 69%, 72.4%, and 70.7% for sensitivity, specificity, and accuracy, respectively.[40] However, FDG PET application in complications in the diabetic foot suffers from the accumulation of FDG in sterile inflammation and lack of uniform interpretation criteria for distinguishing OM from CNA.[41]

While many of these imaging techniques offer advantages when the diagnosis of soft tissue versus bone infection is in doubt, the cost and availability of these techniques limits their use to situations where other diagnostic tests such bone biopsy are limited or inconclusive.

TREATMENT

Antimicrobial Therapy

For mild infections, without underlying OM, empiric antibiotic therapy is a reasonable approach, targeting gram-positive organisms, particularly beta-hemolytic streptococci

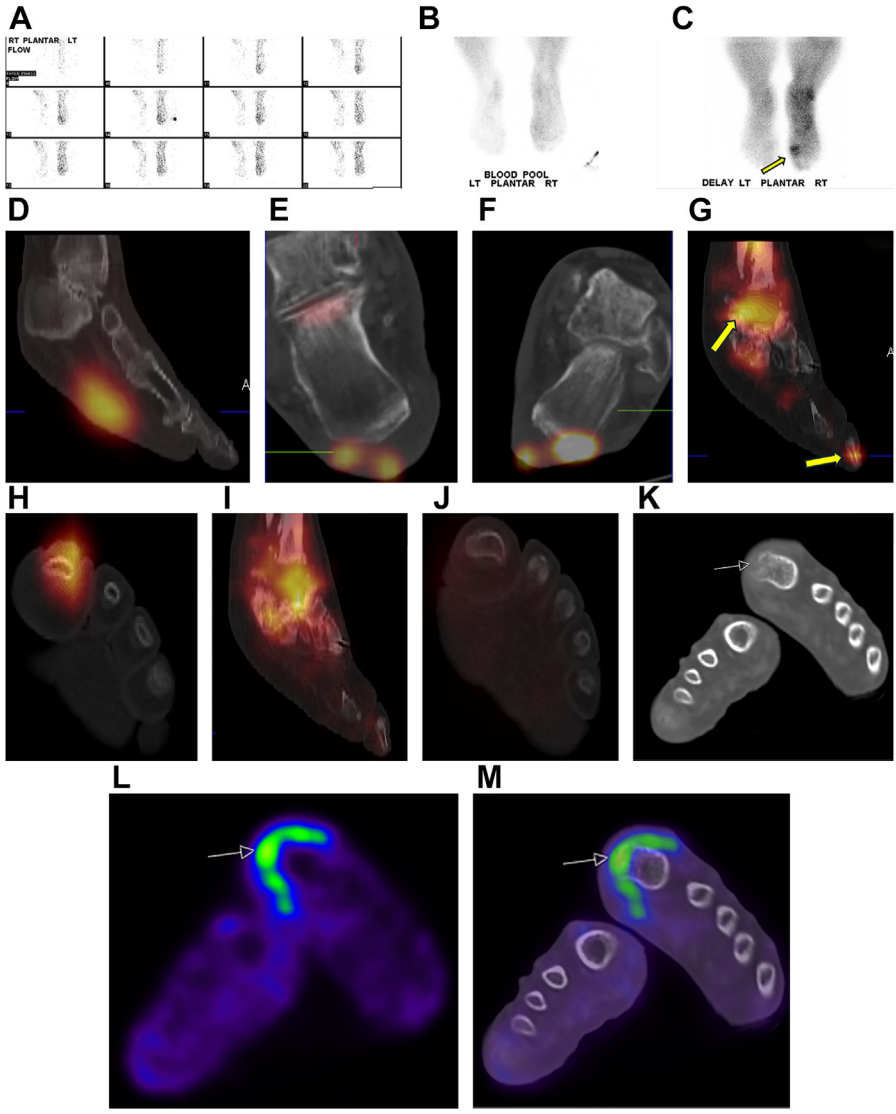


Fig. 2. (*A–C*) Three phase bone scan shows increased flow (*A*) and blood pool (*B*) activity in the left foot and increased uptake in the head of the first metatarsal (*arrow*) on the delay phase (*C*). (*D*) Soft tissue infection of ^{111}In-WBC SPECT/CT (*E, F*) Soft tissue infection and OM on ^{99m}Tc-WBC SPECT/CT. (*G–J*) Dual isotope dual radiotracer ^{111}In-WBC (*G*, *H*) and 99mTc-sulfur colloid scan (*I*, *J*) in a patient with CNA in the hindfoot and ankle, and osteomyelitis in the great toe and posterior aspect (discordant spatial activity, *arrow*) in the ankle. (*K–M*) FDG PET/CT images of osteomyelitis involving the head of the first metatarsal in the left foot incidentally found on a scan obtained for oncology purposes. (*K*) CT showing cortical erosion (*arrow*). (*L*) FDG PET showing FDG accumulation at the erosion site (*arrow*) and in the soft tissue around the joint (*arrowhead*). (*M*) Fused PET/CT image (*arrow points* to focal uptake at the erosion site, *arrowhead* FDG accumulation in soft tissue around the joint).

and *Staphylococcus aureus,* including methicillin-resistant *S. aureus* (MRSA) based on risk factors.[3] For moderate to severe infections based on the IWGDF/IDSA classification, expanding coverage to include gram-negative and anaerobic bacteria is recommended, and should be guided by local antibiogram data. Several studies have compared different antibiotics in the treatment of DFI, with no specific antibiotic class noted to be superior to another, one exception being a trial comparing ertapenem with or without vancomycin versus tigecycline, in which the tigecycline arm had poorer outcomes.[42] Typical recommendations include beta-lactam antibiotics with or without beta-lactamase inhibitors, cephalosporins, carbapenems, and metronidazole for anaerobic coverage in combination with other antibiotics, clindamycin, linezolid, tetracycline, trimethoprim-sulfamethoxazole, daptomycin, fluoroquinolones, or vancomycin[3] (**Table 2**), though knowledge of local antibiogram data should guide empiric regimens. Limited retrospective data exists for the use of long acting lipoglycopeptides.[43] Pseudomonas is not common among North American studies of DFI, and empiric coverage should be considered only in cases with maceration, water exposure, prior antibiotic exposure or *P. aeruginosa* isolated in recent cultures.[3] The prevalence of common drug-resistant organisms, such as MRSA and resistant gram-negative organisms can vary widely and initial empiric antibiotic regimens should take into consideration local rates of these organisms.[44] Many empiric antibiotic regimens cover most anaerobes, though the severity of infection often warrants surgical debridement, which might be sufficient to disrupt the anaerobic environment, thus decreasing the need for antibiotics that specifically target anaerobes.[18] Use of appropriately selected oral antibiotics to treat DFI is supported by the literature,[14,45–47] though it is imperative that patients have close follow-up similar to those on IV antibiotics, as up to one-third of patients being treated with oral antibiotics for orthopedic infections have been reported to develop side effects that warrant additional medical intervention.[48]

Duration of therapy is usually based on the extent of infection with few randomized controlled trials (RCTs) having compared the duration of antibiotic therapy in DFI. In a prospective RCT of 10 days versus 20 days of treatment post-debridement for soft tissue infection, rates of remission and adverse events were similar.[13] OM is typically treated for 6 weeks, and at least 1 RCT has shown no significant differences in outcomes for appropriately selected patients treated nonsurgically, with 6 weeks versus 12 weeks of antibiotics for DFO. An important caveat to note is that patients were excluded from this trial if they had PVD or had other indications for surgical debridement.[49] A recent RCT comparing 3 versus 6 weeks of antibiotics for DFO in patients that had surgical debridement showed no significant differences in treatment outcomes between the groups,[14] leading to a recommendation to treat residual OM for 3 weeks post-debridement.[3] However, this was a pilot study with 90 patients and wide noninferiority margins in which approximately half of all patients had a minor amputation for OM of the toes, with a larger follow-up study underway.[50] Clinicians often use microbiological and/or histopathological data from the proximal margin of amputation to determine if there is residual OM and thus determine the duration of treatment, though data around this approach is conflicting. While some studies have shown that residual OM at the pathologic margin was associated with a higher rate of treatment failure,[51,52] treating with a longer course of antibiotics has not been shown to clearly affect the outcome.[53,54] A more recent study showed no differences in reinfection, amputation, or hospitalization among patients with negative surgical resection margins (negative microbiology and negative pathology).[55] In practice, most clinicians default to a 6-week course of antibiotics if there is concern for residual OM, but this extended course of antibiotics may not lead to improved outcomes.

Table 2
Suggested empiric antibiotics for diabetic foot infections

Infection Severity[a]	Pathogens	Antibiotics	Additional Considerations
Mild	Gram positive cocci	Cephalexin/cefazolin	Add doxycycline or tmp/sx if purulence or MRSA risk factors
	S. aureus, Streptococcus species	Amoxicillin-clavulanate	Add doxycycline or tmp/sx if purulence or MRSA risk factors
		Clindamycin	—
		Trimethoprim-sulfamethoxazole	
		Doxycycline	
Moderate/Severe	Gram-positive cocci	Ampicillin/Sulbactam	—
	Enterobacteriaceae	Piperacillin/tazobactam	
		Ceftriaxone + metronidazole	
	Obligate anaerobes	Cefepime+metronidazone	If risk factors for *Pseudomonas*
		Carbapenems	If risk factors for ESBL producing or ganisms
		Vacomycin/Daptomycin/Linezolid	Added if purulence/MRSA risk factors/hypotension

Abbreviations: ESBL, extended spectrumbeta-lactamase; Tmp/sx, trimethoprim/sulfamethoxazole.
[a] Infection severity based on IDSA/IWGDF classification.

Surgical Approach and Considerations

Surgical management of DFIs can be an unfortunate necessity to preserve a functional limb. Urgent operative intervention within the first 24 to 48 hours after hospital admission is often required to obtain infectious source control in severe foot infections, such as patients with deep abscess, necrotizing soft tissue infection, and/or soft tissue emphysema. To stratify and triage patients, a methodical and repeatable assessment is paramount. Aside from a thorough history and physical, an assessment of this patient population should include proper laboratory and imaging resources. Imaging the diabetic foot is discussed elsewhere in this work, but plays a large role in the surgical decision making.[56] Specifically, the finding of soft tissue emphysema on radiographs, CT, or MRI should increase the clinical suspicion of a gas forming infection, necessitating a more urgent operative intervention. Other clinical assessment tools such as the laboratory risk indicator for necrotizing fasciitis (LRINEC) score may be useful but have shown limited utility in this patient population.[57]

A variety of surgical procedures may be used depending on the severity of the infection, the extent of tissue loss, and associated comorbidities. When approaching the surgical management of the diabetic foot, staged operative management is required, and proper expectations should be conveyed to the patient that multiple procedures will likely be required to preserve a functional limb. The acute operative intervention of the infected foot should first address infectious source control to remove infected, necrotic, and nonviable soft tissue and bone to arrest further progression of the infection. During the index procedure, proper microbiological and pathologic specimens should be obtained to evaluate for the causative pathogen of the soft tissue infection and, if relevant, OM. Obtaining appropriate osseous specimens for microbiology and pathology can improve the success of medical treatment of residual OM by approximately 30%.[58] It is often not known if the entirety of the infected tissues were excised, further necessitating both the intraoperative specimens and staged operative management of the foot infection. This is especially true in cases with OM. In many situations, the patient is brought back to the operating room for the subsequent stage in 48 to 72 hours for resection of any remaining infected or nonviable tissue. The final operative procedure often includes any combination of wound bed preparation, wound closure, coverage of the wound with a bioengineered skin substitute, cellular or tissue-based product, or continuing with wound care.

When specifically addressing the operative management of the patient with OM, additional considerations should be taken. If OM is isolated to a small bone such as a phalanx or a structure like a toe, infection can be completely excised by way of an amputation. This becomes a complex issue when a more structurally important bone becomes infected, such as a metatarsal or any bone proximal to a metatarsal. While OM may be within a defined bone, such as the 5th metatarsal, complete excision is often not preferable when considering the functional status of the patient. In this specific example, the complete excision of an infected 5th metatarsal would cause the loss of the peroneus brevis muscle insertion and a subsequent varus deformity of the foot. This deformity could then cause further ulcerations and infections secondary to this deformity. While the full purview of postamputation pathomechanics is outside the scope of this work, there are many other works on this subject.[59–61] Nonetheless, in clinical situations such as this, the surgeon may decide not to fully excise the OM to preserve a functional limb, opting to treat the residual OM medically. This is especially challenging in the patient with calcaneal OM as complete resection is often not a viable option. A subtotal calcanectomy may be a viable option for resection of much of the infected bone while allowing for additional wound

coverage options.[62,63] The subtotal calcanectomy, in the setting of DFO specifically, has been shown to preserve the functional status of the patient although approximately 25% of patient with DFO of the calcaneus may already be nonambulatory.[64] In the non-ambulatory patient with no reasonable expectation to rehabilitate to an ambulatory status, further consideration should be given to a higher level amputation, the exact extent of which needs to take into consideration the level of infection and the vascular status of the patient.

COMORBIDITIES

Peripheral Vascular Disease

The prevalence of peripheral vascular disease among patients with DFU is as high as 49%.[65] Peripheral arterial disease (PAD) is consistently associated with treatment failure and risk of amputation.[15,66,67] The IWGDF intersocietal PAD guideline recommends all patients with DFU/DFI should be assessed for PVD with palpation of pedal pulses, evaluation of pedal doppler waveforms, ankle-brachial index (ABI) and toe brachial index.[68] Absent pulses, ankle pressure <100 mm Hg, toe pressure <60 mm Hg or monophasic/absent pedal Doppler waveforms warrant further evaluation by vascular surgery. While no single cut-off value can be used to diagnose PVD, a low ankle pressure (<50mmg Hg) or ABI <0.5 is associated with greater risk of major amputation, while toe pressure <30 mm Hg increases probability of major amputation by 20%.[68] Revascularization should ideally be completed prior to surgical debridement to maximize the chance of wound healing, though the severity of infection and practical logistics within a hospital often dictate the timing of revascularization. Altmann and colleagues reviewed 608 patients with ischemia and DFI, comparing patients who had revascularization 2 weeks before or after debridement surgery, and did not show any significant difference in outcome as regards to the sequence or timing of revascularization as related to debridement.[69]

Glycemic Control

Patients with DFU fear major amputation more than death, compared to diabetic patients without ulcers,[70] and oftentimes an episode of DFI serves as a wakeup call for a patient to improve glycemic control. Poor glycemic control is a major risk factor for the development of DFI and is associated with poorer outcomes after surgical management.[71,72] Intensive glycemic control, in which the glycemic levels are maintained as close to the nondiabetic range of Hemoglobin A1c (HbA_{1c}) <6.05% compared to conventional glycemic control without specific glycemic targets, has been shown to reduce the risk of all DFUs. In a retrospective review of 183 patients with diabetes seen at a wound center, HbA_{1c} at baseline was inversely related to change in wound area, such that for each 1% increase in HbA_{1c}, the wound-area healing rate decreased by 0.0028 cm^2 per day.[73] With many newer drugs for diabetes, technologies such as continuous glucose monitors, multimodal diabetes care models, and use of electronic consultation to access specialty care, every effort must be made to ensure patients have access to a holistic plan for diabetes management.[74,75] In addition to an annual comprehensive foot examination, patients should be taught to examine their feet daily for early detection of new foot problems.

Offloading

Offloading is critical to promote wound healing[7] and can be achieved via a combination of offloading devices, surgery, and orthotics. For patients with plantar forefoot or midfoot ulceration a nonremovable knee-high offloading device such as a total contact

cast or nonremovable knee-high device such as a walking boot is recommended. However, in patients with ischemia or ongoing infection, removable knee-high or ankle-high offloading devices should be used, to facilitate frequent inspection and wound care. In general, patient-selected footwear is discouraged due to increased risk of new lesions.[76–78] Surgical strategies such as Achilles tendon lengthening, digital flexor tenotomies etc., are recommended in selected cases that have failed nonsurgical offloading. Patient balance and comfort can be improved by addressing leg-length discrepancy with a shoe lift for the contralateral limb, and/or a walking aid.[79]

Wound Care

In addition to treating infection, addressing comorbidities, and ensuring appropriate offloading, regular wound care is essential to ensure healing of a DFU. Debridement of the ulcer and any surrounding callus results in removal of necrotic or devitalized tissues, in order to promote wound healing and decrease the risk of infection. Debridement can be achieved by surgery, or other mechanical means (wet-to-dry dressing, hydro-debridement, or gaseous debridement), biochemical methods (use of enzymes to liquefy necrotic tissue), or biological methods (use of medical grade larvae). IWGDF guidelines recommend sharp debridement of the ulcer as a standard of care, though sharp debridement should be avoided in case of ischemic or painful ulcers. Enzymatic debridement is suggested as an alternative when sharp debridement is not available or contraindicated.[80] While no recommendation is made on the frequency of debridement, retrospective data suggests that wounds that are debrided with intervals of one week or less heal significantly faster.[81] No specific type of wound dressing has been shown to be superior for DFU, and in general, choice of dressing is often determined by cost and availability. Negative pressure therapy has been shown to decrease the time to healing for postsurgical wounds compared to standard of care dressings,[82,83] though this can be costly and time consuming.

Monitoring treatment

Patients that have been treated for DFI/DFO should be monitored clinically for resolution of infection, recognizing that ulcers might not be completely healed at the end of antibiotic treatment.[6] Routine monitoring of inflammatory markers at the end of treatment has not been shown to predict treatment failure, and similarly radiographs are not recommended as there is often a radiographic lag in improvement.[84,85] Wounds that do not heal appropriately should be evaluated for risk factors that contribute to wound healing—particularly PVD and offloading. All patients should have close clinical follow-up and education to ensure modifiable risk factors for ulcer recurrence, such as off-loading with appropriate foot wear, glucose control, smoking cessation, and PVD are consistently addressed. Foot care should be reevaluated every three to six months for patients who are risk for foot ulceration.[76]

DFI is a complex disease with patients having to cope with multiple recommended interventions simultaneously, with hospitalized patients reporting worse quality of life outcomes as regards to physical function, pain, anxiety, depression,[86] lack of motivation, high costs of care, challenges in limiting activity to achieve appropriate off loading, and difficulty in obtaining timely appointments, described as barriers to care.[87] Potential solutions have been suggested to address barriers to care, particularly as related to social determinants of health.[88] Finally, given the complexity of patients with DFU, multidisciplinary teams and access to specialty care including endocrinology, infectious diseases, podiatry, plastics, vascular surgery, have been shown to improve survival of patients with a longer time to ulcer progression, major amputation, or death.[89,90]

The care of patients with DFI continues to evolve with improved diagnostic studies and increasing literature demonstrating that many patients can be treated without amputation, with improved surgical techniques that support conservative surgery, increasing the use of oral antibiotics, given for a shorter duration, and use of technology to better support patients living with diabetes. Increased recognition of the value of multidisciplinary collaborative care can lead to personalized treatment plans, and ultimately improved outcomes and functionality for patients with DFI.

CLINICS CARE POINTS

- Patients with diabetic foot infection must be evaluated for underlying osteomyelitis, using a combination of clinical, laboratory, and radiographic tests.
- Soft tissue infections can be treated with antibiotics for 10-days, whereas for diabetic foot osteomyelitis, antibiotics for three to six weeks is recommended.
- Knowledge of local microbiology/antibiogram is important to determine empiric antibiotic regimens. Appropriately selected oral antibiotics can be just as effective as intravenous antibiotics.
- Evaluate and address comorbidities that can affect outcomes, especially peripheral vascular disease, offloading, and glucose control. This is ideally done with an interdisciplinary team.

DISCLOSURE

All authors report no conflicts of interest related to this work.

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