A Comprehensive Review of the Pathogenesis, Diagnosis, and Management of Diabetic Foot Infections

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GENERAL PURPOSE: To review an approach to diabetic foot infections (DFIs), including acute osteomyelitis, while also discussing current practices and the challenges in diagnosis and management.

TARGET AUDIENCE: This continuing education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES: After participating in this educational activity, the participant will

1. Identify the risk factors for developing DFIs.
2. Outline diagnostic techniques for assessing DFIs.
3. Select the assessment techniques that support a diagnosis of osteomyelitis.
4. Choose the appropriate pharmacologic and nonpharmacologic treatment options for patients who have DFIs.

ABSTRACT

Diabetic foot ulcers result from a combination of peripheral neuropathy, vascular compromise, and repetitive trauma. Approximately 50% of individuals with diabetic foot ulcers will develop a diabetic foot infection (DFI), and 20% of individuals with a DFI will develop osteomyelitis. Herein, the authors review an approach to DFIs including acute osteomyelitis and discuss current practices and challenges in diagnosis and management.

The diagnosis of a skin and soft tissue DFI is based on clinical criteria. A bone biopsy is considered the criterion standard for diagnosis of osteomyelitis; however, biopsy is not always feasible or available. Consequently, diagnosis can be made using a combination of clinical, biochemical, and radiographic findings. X-ray is the recommended imaging modality for initial evaluation; however, because of its lower relative sensitivity, advanced imaging may be used when clinical suspicion remains after negative initial testing.

The microbiology of skin and soft tissue DFIs and osteomyelitis is similar. *Staphylococcus aureus* and other Gram-positive cocci are the most common pathogens identified. Deep cultures are preferred in both DFI and osteomyelitis to identify the etiologic pathogens implicated for targeted antimicrobial therapy. Management also requires a multidisciplinary approach. Surgical debridement in those with deep or severe infections is necessary, and surgical resection of infected bone is curative in cases of osteomyelitis. Finally, appropriate wound care is critical, and management of predisposing factors, such as peripheral neuropathy, peripheral arterial disease, tinea, and edema, aids in recovery and prevention.

KEYWORDS: diabetes, diabetic foot, infection, osteomyelitis, surgical debridement, ulcer, wound care

ADV SKIN WOUND CARE 2021;34:574–81.
DOI: 10.1097/01.ASW.0000791876.10485.d4

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INTRODUCTION

The prevalence of diabetes is increasing, with an estimated 463 million people living with this condition globally as of 2019. It is estimated that by 2030 and 2045, 578 million and 700 million people, respectively, will be affected worldwide.[1] Complications of diabetes include diabetic nephropathy, retinopathy, neuropathy, and peripheral arterial disease (PAD).[2] Diabetic neuropathy and PAD increase the risk of diabetic foot ulcers (DFUs) with subsequent infections. Importantly, diabetic foot infections (DFIs) remain a leading cause of nontraumatic lower limb amputations with significant associated morbidity and mortality.[3] Thus, the diagnosis and management of DFIs are important in persons living with diabetes. Herein, the authors review an approach to DFIs including acute osteomyelitis and discuss current practices and challenges in diagnosis and management.

EPIDEMIOLOGY

Diabetic foot ulcers are the most common complication of diabetes, with an estimated annual incidence of 2% and a lifetime risk of 15% to 25%.[4–6] The skin breaks down from a combination of peripheral neuropathy, vascular arterial disease, and repetitive trauma.[7] In those with DFU, more than 50% develop soft tissue infections in the ulcerated areas.[8] Subsequently, osteomyelitis can develop in 20% or more of those with DFU-associated soft tissue infections.[9] Severe infections of the soft tissue or bone lead to amputations in an estimated 17% to 36% of individuals.[10–13] The mortality in persons with DFU is significant; the 5-year mortality with new-onset DFUs is 40%. Following a lower extremity amputation, the 5-year mortality increases further to 60%.[11,14–16] Thus, prevention and management of DFUs in persons with diabetes are critically important to reduce morbidity and mortality and improve quality of life.

PATHOGENESIS

Neuropathic changes of the foot leading to foot deformities and loss of protective sensation predispose persons with diabetes to DFU. The presence of foot deformities causes uneven pressure distribution and areas of abnormally high pressure with movement. These high-pressure areas are then prone to damage from repetitive stress and trauma that occur with routine activities of daily living. The loss of protective sensation diminishes the ability of a person to discern or mitigate foot trauma and prevent further injury.[7] Healing is further affected by vascular issues (ie, arterial disease) and sets up a cycle of neuropathy, vascular damage, and repetitive trauma leading to ulceration.[7]

CASE REPORT

A 68-year-old man (M.W.) with a 20-year history of poorly controlled type 2 diabetes mellitus (hemoglobin A_{1c} of 9.1% on insulin therapy) presented with a DFU of 27 months' duration on the plantar aspect of his right great toe. His other comorbidities included dyslipidemia, hypothyroidism, obesity, coronary artery disease with prior myocardial infarction requiring angioplasty, and diabetes-associated chronic kidney disease (stage IIIa).

Examination revealed a plantar ulcer measuring 1.5 × 0.8 × 0.3 cm on the great toe of his right foot. He had palpable dorsalis pedis and posterior tibial pulses and a biphasic waveform on Doppler evaluation (8 MHz). His ankle-brachial indices (ABIs) were 0.9 bilaterally. There was evidence of wound exudate with red friable tissue, and based on the lack of healing over this duration, he was treated with povidone-iodine. To redistribute pressure, M.W. was provided with an offloading boot with custom orthotics.

Days after the assessment, M.W. developed increased right toe swelling and warmth, increased exudate, and fevers and rigors consistent with a soft tissue infection. An X-ray revealed a new area of lucency in the distal phalanx of his right great toe. Bony erosion and fragments were also present in the area. His wound was debrided, and tissue and bone fragments were sent for culture.

He was assessed the next day by the infectious disease consultant and diagnosed clinically with DFU-associated osteomyelitis. Because M.W. was clinically stable, both oral and IV antibiotic options for treatment were discussed, and the patient selected the oral option. He was treated empirically with cefadroxil for a planned 6-week course for his acute infection. Cultures were positive for *Staphylococcus aureus*, which was susceptible to the antibiotics used. M.W. was assessed in follow-up 2 weeks later and had signs of clinical improvement of his right foot. In addition to ongoing care of the DFU and infection, diabetes optimization and lifestyle interventions (ie, diet and exercise) were planned. M.W. provided consent for his case to be included in this article.

RISK FACTORS

The predominant risk factor for a DFI is the presence of a DFU. The loss of skin integrity leads to bacterial invasion into underlying structures and results in infection.[17] Other risk factors include fungal (tinea) infections of the feet, edema, and a history of prior infection. Tinea infections damage skin integrity and lead to secondary bacterial infection.[17,18] Peripheral edema occurs commonly in persons with diabetes because of diabetes-related complications such as heart failure, renal insufficiency, and venous insufficiency.[19,20]

The presence of edema slows DFU healing and increases the risk of infection.[21] History of past infection is also a risk factor for recurrence from inflammatory damage of lymphatic vessels in the affected limb(s) that

occurs with the initial infection. The resultant alterations in lymphatic drainage and pathogen clearance predispose individuals to further infections.[22]

DIAGNOSIS

Diabetic Foot Infections

Infections in persons with diabetes are frequent and can occur in more than 50% of DFUs.[8] The diagnosis of DFI is made on the basis of clinical criteria, because all skin surfaces have bacteria present, and thus wounds may become contaminated and subsequently colonized with varying bacterial burden. The International Working Group on the Diabetic Foot (IWGDF) categorizes and classifies DFIs into four grades found in Table 1.[23] Other grading classifications of DFIs include the University of Texas and the Wagner classification systems. The Wagner system classifies ulcers based on depth in addition to the presence of infection and gangrene.[24] The University of Texas ulcer classification has some advantages because it incorporates the depth of the ulcer separately from the presence of infection, ischemia, or both (Table 2).[25]

Acute Diabetic Foot Osteomyelitis

Infection of the soft tissue and DFUs can extend to involve the bone, otherwise termed *osteomyelitis*. Osteomyelitis is estimated to be present in up to 20% and 60% of individuals with mild and severe DFIs, respectively.[8,9,26] The criterion standard of diagnosis for osteomyelitis is a bone biopsy that reveals a pathogenetic bacterium in addition to tissue evidence of inflammation and infection.[27] Unfortunately, this is not always readily available, done, or feasible, and as a result, surrogate clinical, biochemical, and radiographic methods are often used for diagnosis.

Osteomyelitis is often first suspected based on clinical presentation. Persons presenting with DFU with exposed bone have an increased risk of osteomyelitis because bacteria can reach exposed bone and cause infection.[28] Clinically, the probe-to-bone (PTB) test is commonly used to delineate the presence of osteomyelitis. The test is conducted by placing a sterile probe in a DFU to determine if the ulcer depth reaches bone. Because infection reduces bone quality, the bone encountered by the PTB test may be weak or destroyed.

The positive predictive value is greater than 90% when the pretest probability is 60% or greater. Pretest probabilities of less than 20% yield negative predictive values greater than 95%.[29] In most clinical settings, the negative predictive value is more useful than the positive predictive value. However, the prevalence of osteomyelitis in the population evaluated influences the utility and interpretation of positive and negative predictive values.

In an inpatient setting with high-risk patients, the PTB test can be used to support osteomyelitis diagnosis. Conversely, in low-risk settings such as primary care clinics, a negative PTB test can be used to rule out osteomyelitis.

The size of the ulcer is another finding that can impact the probability of osteomyelitis. Those DFUs with areas larger than 2 cm^2 have a sevenfold increase in the odds of osteomyelitis (positive likelihood ratio, 7.2). An ulcer smaller than 2 cm^2 decreases the likelihood of osteomyelitis by nearly 50% (negative likelihood ratio, 0.48).[28] Exposed bone, the PTB test, and ulcer size must be considered together in the clinical context; however, they serve to enhance initial clinical suspicion for osteomyelitis.

Elevations in white blood cell count, platelets, C-reactive protein, and erythrocyte sedimentation rate (ESR) are neither sensitive nor specific but can supplement clinical suspicion. An ESR greater than 70 mm/h has a positive likelihood ratio of 11 for diabetic foot osteomyelitis. This finding is most useful in differentiating soft tissue infections from osteomyelitis.[28,30,31] When combined with clinical findings, an elevated ESR greater than 70 mm/h will support diagnosis.

Imaging modalities used in the assessment for osteomyelitis include plain radiographs, nuclear bone scans, nuclear white blood cell scans, MRI, positron emission tomography (PET), and single-photon emission computed tomography scans. A summary of imaging modalities, findings of osteomyelitis, and their sensitivity and specificities can be found in Table 3.

Plain radiographs are the quickest and cheapest imaging modality. The IWGDF recommends a combination of the PTB test, ESR, and plain X-rays as the initial studies

Table 1. CLASSIFYING SEVERITY OF INFECTION

Infection Classification	Clinical Description
1	Wound without purulence or manifestations of inflammation without systemic signs of infection
2	Two or more of the following: purulence, erythema, tenderness, warmth, induration, AND cellulitis/erythema ≤2 cm around the ulcer AND infection limited to skin of superficial subcutaneous tissues with no local complications or systemic illness
3	Grade 2 infection with one or more of the following: cellulitis >2 cm, lymphatic streaking, deep tissue involvement, gangrene and involvement of muscle, tendon, joint, or bone AND no signs of systemic toxicity.
4	Any signs of systemic toxicity such as tachycardia, hypotension, vomiting, leukocytosis, acidosis, etc

Adapted from Monteiro-Soares M, Boyko EJ, Jeffcoate W, et al. Diabetic foot ulcer classifications: a critical review. Diabetes Metab Res Rev 2020;36(S1):e3272.

Table 2. WAGNER AND UNIVERSITY OF TEXAS DIABETIC FOOT ULCER CLASSIFICATION SYSTEMS

Wagner DFU Grade Classification System

Assesses ulcer depth and the presence of osteomyelitis or gangrene with the following:

Grade 0	**Intact skin**
Grade 1	Superficial ulcer
Grade 2	Deep ulcer without osteomyelitis or abscess
Grade 3	Ulcer with bone involvement, osteomyelitis, or abscess formation
Grade 4	Gangrene in toes or forefoot
Grade 5	Full-foot gangrene

Advantages	**Disadvantages**
Simple to use	The presence of infection and ischemia are related to poor outcome
Validated	Ischemia in grades 1–3 not considered
Higher grades directly related to increased risk for lower limb amputation	Infection in grades 1, 2, and 4 not considered
	Location of the ulcer is not described
Provides a guide for planning treatment	Patient-related factors not evaluated
Considered the criterion standard	Foot deformities are not evaluated

The University of Texas Diabetic Foot Ulcer Classification System

Grades diabetic foot ulcers by depth and then stages them by the presence or absence of infection and ischemia:

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

Advantages	**Disadvantages**
Simple to use and evaluate	Patient-related factors not evaluated
More descriptive	Foot deformities are not evaluated
Predicts more accurately the outcome of an ulcer (healing or amputation) than Wagner	The location of the ulcer is not described
Cases with infection and/or ischemia are considered	
Provides a guide for planning treatment	

Adapted from Wagner[24] and Lavery et al.[25]

to evaluate for osteomyelitis.[23] However, radiographic changes may not be apparent for at least 2 weeks after the infection has settled into the bone.[32] Thus, MRI and PET scans are recommended when advanced imaging is required.[23]

It is important to note that Charcot arthropathy, a noninfectious but inflammatory neuropathic condition, can mimic osteomyelitis with advanced imaging. Further, this condition can occur in up to 10% of persons with diabetes.[33] The distinction is essential, as management of Charcot arthropathy differs from that of osteomyelitis with immobilization and pressure offloading as the primary treatment recommendations.[34,35]

MICROBIOLOGY

Staphylococcus aureus, along with other Gram-positive bacteria, represent the most common cause of acute DFIs.[36] In addition to Gram-positive organisms, Gram-negative pathogens such as *Escherichia coli*, *Klebsiella pneumoniae*, and *Proteus* species are implicated in infected wounds present for longer than 4 weeks, moderate to severe infections, those with recent (within the past 30 days) antibiotic therapy, and those who reside in warmer climates.[36,37] Anaerobic pathogens contribute to severe and chronic infections and infections complicated by abscess formation.[37] Deep tissue cultures rather than superficial swabs are needed to identify the etiologic pathogen(s), because superficial swabs will often isolate microbes colonizing the skin.[23,37]

The microbiology of diabetic foot osteomyelitis is similar to that of DFIs. *Staphylococcus aureus* is the predominant pathogen (identified in up to 50% of cases) followed by streptococcal species and Gram-negative bacteria.[38] Bone cultures are preferred over superficial cultures as the latter is not always reflective of the etiologic pathogens.[23,37] Cumulative concordance between superficial cultures and bone cultures for all pathogens ranges between 22% and 28%.[38,39] In cases where *S aureus* is identified

Table 3. SUMMARY OF IMAGING MODALITIES FOR OSTEOMYELITIS DIAGNOSIS

Imaging Modality	Suggestive Findings	Pooled Sensitivity, %	Pooled Specificity, %
Plain radiograph	Lytic lesions, periosteal thickening, loss of the trabecular architecture of the bone	54	68
Nuclear bone scan	Focal hyperperfusion, focal hyperemia, increased bone uptake	80	28
Indium-111 tagged WBC scan	Increased WBC uptake	92	75
^{99m}Tc-hexamethyl-propyleneamine oxime tagged WBC scan	Increased WBC uptake	92	91
MRI	Low signal intensity in the medullary space on T1-weighted images, high signal intensity with a surrounding inflammatory process on T2-weighted images	95.6	80.7
PET/CT	Increased bone activity, increased bone uptake	85.1	92.7
SPECT/CT	Increased bone activity, increased bone uptake	95	82

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging; PET, positron emission tomography; SPECT, single-photon emission computed tomography; Tc, technetium; WBC, white blood cell.

on superficial cultures, concordance is increased, ranging from 38% to 44%.[38–40] In the absence of a bone biopsy, *S aureus* isolated from superficial cultures is more likely to equivalate *S aureus* disease in the bone.

ASSESSMENT

Assessment for predisposing factors is critical to the overall management of DFIs. The two most important risk factors to assess and address are PAD and neuropathy, because these factors play a significant role in DFUs and DFIs. Bedside and noninvasive tests used for vascular assessment include palpation and ultrasonography.

The lack of a palpable posterior tibial and dorsalis pedis artery may signify a reduction in blood flow to the region. Doppler ultrasonography can be used to determine the ABI; values between 0.9 and 1.3 are considered normal. Values of less than 0.9 indicate PAD and warrant further investigation with direct visualization imaging. Conversely, values greater than 1.3 are abnormal and typically represent calcified or noncompressible vessels.[41] An alternative to ABI and the audible handheld Doppler ultrasound, toe-brachial indexes (TBIs) can also be used to evaluate for PAD. These can assess for small vessel disease when ABI values are greater than 1.3. Values of less than 0.7 for the TBI are considered abnormal.[42]

Direct visualization of vasculature requires angiography.[41] Contrast angiography is standard; however, it is not commonly used because it is an invasive procedure. Magnetic resonance angiography and computed tomographic angiography are alternatives. Computed tomographic angiography is preferred over magnetic resonance angiography for its superior ability to detect calcification in vasculature, allowing for better planning of revascularization strategies.[41]

Bedside evaluation of peripheral neuropathy is conducted with the monofilament test.[2] The Figure depicts the 10 foot sites to be tested with the monofilament.[43] A full neurologic assessment should accompany the monofilament test to assess for changes in motor and sensory function.

Assessment for tinea and edema is also necessary. Tinea presents as a dry plantar surface mimicking the changes of autonomic neuropathy. With tinea infections, dryness often extends around the sides of the feet in a "moccasin" distribution and may be associated with dryness or maceration in the toe web spaces. Web space maceration can be an entry point for secondary bacterial infection of the lower limb. The toenails can also be affected, with an asymmetrical distribution of distal onycholysis or whole nail plate dystrophy.[44]

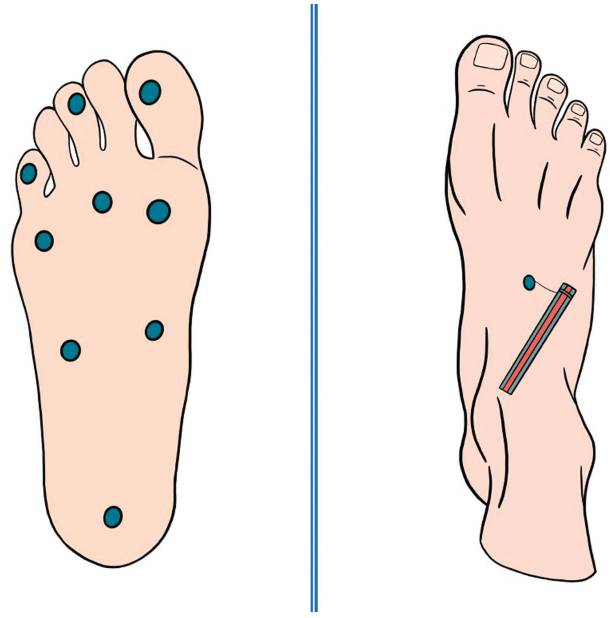
Lower limb edema presents with swelling and occurs when excess fluid accumulates in the interstitial tissue. Local causes of edema include prior infections, venous insufficiency, lymphedema, and lipedema.[45] Evaluation for these causes with appropriate corrective measures improves outcomes in the management DFIs.

MANAGEMENT

In persons with DFIs with or without osteomyelitis, a multidisciplinary approach to therapy is required. Management is aimed at correcting underlying and predisposing risk factors and treatment of the infectious process.

Topical or systemic antifungal therapy may be used in the treatment of tinea, whereas compression therapy remains the mainstay of therapy for edema.[46–48] Management of peripheral neuropathy involves patient education and regular foot checks to assess for puncture wounds, callus formation, or areas of skin breakdown. Referral to a podiatrist or a foot specialist for footwear and consideration of pressure redistribution therapy are indicated. It is possible to manage PAD medically with or without surgical intervention. Medical management focuses on control of dyslipidemia, hypertension, and

Figure. SITES TO BE TESTED WITH THE 10-G MONOFILAMENT TEST
Four or more areas with altered sensation are required for a diagnosis of peripheral neuropathy.



antiplatelet therapy. Lifestyle factors such as exercise and smoking cessation are equally important.[49] Revascularization via surgical intervention is determined based on patient-related factors and the extent and severity of disease.[41]

In addition to managing risk factors, wound care is an important component in the management of DFIs. Wound care involves assessment and monitoring of the wound location, appearance, size, depth, and the periwound area.[50] Bedside debridement and removal of necrotic tissue, eschar, and slough promote wound healing by activating stalled or quiescent cells. It also reduces the bacterial burden in infected wounds.[51] Debridement is also required with chronic nonhealing wounds in which the formation of biofilms by microorganisms allows for enhanced tolerance to antimicrobial therapies. Thus, debridement lowers the biofilm and microbial burden, allowing for improved efficacy of antimicrobials.[52] Optimizing moisture balance with appropriate dressing also promotes wound healing.[51]

Finally, surgical intervention plays a vital role in managing DFIs and osteomyelitis. Severe DFIs or those with abscess formation benefit from surgical debridement;[37] this promotes wound healing in a mechanism equivalent to bedside debridement. Operative surgical debridement also allows for sampling of deep tissue specimens for culture. Cases of osteomyelitis may also benefit from operative intervention. Surgical cure can be achieved with complete resection of infected bone followed by appropriate wound care. Complete surgical resection of necrotic bone, if present, is necessary because antibiotics in isolation are ineffective in nonviable bone.[37]

Between 17% and 36% of those with DFIs will require an amputation.[10–13] The odds of amputation are highest in those with gangrene or necrosis at presentation, IWGDF grade 4 infection, osteomyelitis, and neuroischemic disease.[53] Although amputation can be lifesaving, it contributes to morbidity in persons with diabetes, and providers should recognize the importance of infection prevention via identifying and mitigating risk factors.

The last component of treatment in DFIs is antimicrobial therapy. Both topical and systemic antimicrobials can be prescribed. Topical antimicrobials can be used to treat superficial infections of DFUs. Options include, but are not limited to, iodine, polyhexamethylene biguanide, chlorhexidine, silver, and hypochlorous acid.[54] Some superficial infections and all deep infections require systemic antibiotics. The empiric antibiotic choice involves patient-related factors and the extent and duration of disease. Typical regimens are presented in Table 4. Empiric antibiotics can be modified as new clinical and microbiologic data become available.

Culture-based therapy has become increasingly important with increasing antibiotic resistance found in isolated pathogens. For example, from the late 1990s to present day, an estimated 15% to 30% of DFIs are caused by methicillin-resistant *S aureus* relative to years prior.[55] In addition, there has also been an increase in resistance to antimicrobials seen in Gram-negative organisms causing DFIs.[56] Accordingly, antibiotic therapy

Table 4. TYPICAL ANTIMICROBIAL THERAPY BASED ON DISEASE CLASSIFICATION

Severity	Expected Pathogens	Potential Antimicrobial Agents	Route	Therapy Duration
Mild	*Staphylococcus aureus*, streptococci	First-generation cephalosporins	Oral	1–2 wk
Moderate	*S aureus*, streptococci, Enterobacteriaceae	Combination of first-generation cephalosporin and quinolone, amoxicillin-clavulanate	Typically, oral but may require IV to start	2–3 wk
Severe	Mixed infection with Gram-positives, Gram-negatives, and anaerobes	Piperacillin-tazobactam, carbapenem	IV	2–3 wk, dependent on surgical intervention and wound care
Osteomyelitis	*S aureus*, streptococci, Enterobacteriaceae	Based on bone culture results, if possible	Parenteral or oral	6 wk (if not completely surgically resected)

targeted toward pathogens found on appropriately collected cultures combined with a multidisciplinary approach offers the best chance of success.

CONCLUSIONS

The prevalence of diabetes is increasing worldwide. Diabetic foot ulcers are common and result from peripheral neuropathy and vascular disease in persons with diabetes. Left untreated, up to 50% of DFUs will develop an infectious complication. Osteomyelitis occurs in 20% of DFIs. In addition, diabetes is the leading cause of nontraumatic lower limb amputation, with 20% of those with DFIs requiring amputation. Mortality is also high, with a 5-year mortality of approximately 40%; in those with a history of amputation, mortality is increased to 60%.

Diabetic foot infections are diagnosed based on clinical findings. Although a bone biopsy is considered the criterion standard, osteomyelitis diagnosis can be made via a combination of clinical, biochemical, and radiographic findings. Consequently, management of DFIs and osteomyelitis requires a multidisciplinary approach and is targeted at treating predisposing risk factors along with the infectious process.

PRACTICE PEARLS

- Common in persons with diabetes, DFUs result from neuropathy, vascular damage, and trauma in persons with diabetes and can lead to DFIs.
- To diagnose DFIs, the IWGDF and Infectious Diseases Society of America use a constellation of clinical criteria including local swelling, induration, erythema, tenderness, warmth, and purulent discharge.
- The criterion standard for diabetic foot osteomyelitis diagnosis is bone biopsy; however, this is not always readily available or feasible. A combination of clinical findings such as a positive PTB test and large ulcer size (>2 cm^2) combined with an elevated ESR (>70 mm/h) and positive radiographic findings can suggest a diagnosis instead.
- X-ray is recommended by the IWGDF for use as the initial imaging modality in the evaluation of osteomyelitis; however, in cases where advanced imaging is required, MRI or PET scans are recommended.
- *Staphylococcus aureus* and other Gram-positive cocci remain the predominant bacterial causes of DFIs and diabetic foot osteomyelitis.
- Management of DFIs including osteomyelitis requires a multidisciplinary approach to manage risk factors, provide appropriate wound care, and prescribe antimicrobial therapy.

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