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Review Article

Diabetic foot infection versus non-infected diabetic foot: contemporary diagnostic criteria, management algorithms, and clinical pitfalls: a focus on differentiating infectious and ischemic etiologies in diabetic foot ulcers

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ABSTRACT

Diabetic foot ulcers (DFUs) represent a significant complication of diabetes mellitus, with infection (DFI) and critical limb ischemia (CLI) being key determinants of morbidity and amputation risk. Despite established guidelines from the infectious diseases society of America (IDSA) and the international working group on the diabetic foot (IWGDF), misdiagnosis between infected and non-infected (particularly ischemic) DFUs remains a frequent clinical challenge, leading to inappropriate antimicrobial therapy or delayed revascularization. This review examines the current diagnostic criteria for DFI, contrasts them with ischemic presentations, and highlights common errors in management, emphasizing evidence-based strategies to optimize outcomes. A narrative synthesis of IDSA/IWGDF guidelines, recent literature on DFU classification systems (e.g., SINBAD, WIfI), and clinical studies on biomarkers (e.g., procalcitonin, CRP) and imaging modalities (MRI, PET-CT) was conducted. Key discriminators between DFI and CLI include localized vs. diffuse erythema, systemic inflammatory response, and perfusion assessment (ABI, $TcPO_2$). Overreliance on superficial wound cultures and underutilization of bone biopsy in osteomyelitis are recurrent errors. Multidisciplinary teams (MDTs) integrating infectious disease, vascular surgery, and podiatry reduce misclassification rates. Distinguishing DFI from CLI requires systematic evaluation of clinical, laboratory, and vascular parameters. Enhanced clinician awareness of IDSA/IWGDF criteria and ischemic mimics may reduce diagnostic errors and improve limb salvage rates.

Keywords: Diabetic foot infection, Diabetic foot ulcer, Critical limb ischemia, Osteomyelitis, IDSA/IWGDF guidelines, Misdiagnosis

INTRODUCTION

Diabetic foot complications, encompassing neuropathic ulcers, infection (DFI), and CLI, are leading causes of hospitalization and non-traumatic lower extremity amputations worldwide. The interplay between microvascular disease, peripheral neuropathy, and immune dysfunction in diabetes complicates the clinical distinction between infected and non-infected (ischemic or neuropathic) ulcers, often resulting in diagnostic and therapeutic delays.[1,2]

The IDSA and IWGDF have established standardized criteria for DFI diagnosis, emphasizing local (erythema, purulence) and systemic (leukocytosis, fever) signs of infection. However, critical ischemia may mimic infection with dependent rubor, necrotic tissue, or elevated inflammatory markers, leading to overdiagnosis of infection and unnecessary antibiotics. Conversely, underdiagnosed DFI in ischemic ulcers escalates amputation risks.[2]

PATHOPHYSIOLOGY OF DFI VERSUS NON-INFECTED DIABETIC FOOT: A MECHANISTIC PERSPECTIVE

Diabetic foot complications represent a complex interplay of metabolic, vascular, neurological, and immunological disturbances that culminate in tissue breakdown and impaired healing. The distinction between infected DFUs and non-infected DFUs is critical for appropriate clinical management and hinges on understanding their distinct pathophysiological pathways.[3]

FOUNDATIONAL MECHANISMS IN DIABETIC FOOT ULCERATION

Chronic hyperglycemia drives the development of DFUs through multiple interconnected pathways. Persistent elevation of blood glucose levels leads to the accumulation of advanced glycation end products (AGEs), which cross-link with collagen and elastin, compromising the structural integrity of the dermis and microvasculature. Concurrently, the polyol pathway becomes hyperactive, depleting intracellular NADPH and glutathione reserves, thereby exacerbating oxidative stress. This metabolic milieu induces endothelial dysfunction, reducing nitric oxide bioavailability and impairing vasodilation.[4]

Peripheral neuropathy, a hallmark of long-standing diabetes, arises from axonal degeneration and segmental demyelination due to oxidative injury and impaired neurotrophic support. Sensorimotor neuropathy results in loss of protective sensation, allowing repetitive mechanical stress to go unnoticed, while autonomic dysfunction diminishes sweat gland activity, leading to xerosis and fissuring. Motor neuropathy causes muscle atrophy and foot deformities, altering biomechanical loading patterns and creating focal pressure points vulnerable to ulceration.[4]

Microvascular dysfunction further compromises tissue viability. Basement membrane thickening, pericyte loss, and capillary rarefaction impair oxygen and nutrient delivery, while impaired vasomotor reflexes diminish hyperemic responses to injury. Macrovascular disease, characterized by atherosclerotic occlusion of tibial and pedal arteries, exacerbates ischemia, particularly in watershed zones such as the metatarsal heads and heel.[4]

PATHOPHYSIOLOGY OF NON-INFECTED DFUS

Non-infected DFUs typically arise from neuropathic or ischemic mechanisms. Neuropathic ulcers develop at pressure-prone sites, such as the plantar forefoot, where repetitive stress exceeds the tolerance of insensate skin. The ulcer bed often exhibits a hyperkeratotic rim with a granular base, reflecting the body's abortive attempts at repair. Ischemic ulcers, in contrast, manifest at distal acral locations (toes, lateral malleoli) and present with pale, necrotic tissue and poor granulation. Hypoperfusion impedes the inflammatory and proliferative phases of healing, prolonging tissue vulnerability.[4]

In both cases, chronic inflammation plays a pivotal role. Dysregulated cytokine signaling (elevated TNF-α, IL-6) and persistent neutrophil activity perpetuate tissue damage, while impaired macrophage polarization disrupts the transition from pro-inflammatory (M1) to pro-healing (M2) phenotypes. Fibroblast senescence and reduced collagen synthesis further stall wound closure.[4]

PATHOPHYSIOLOGY OF DFI

Infection supervenes when microbial invasion overwhelms local defenses. DFIs are typically polymicrobial, with *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and anaerobes predominating. The diabetic foot's hypoxic, glucose-rich environment fosters biofilm formation, which shields bacteria from immune clearance and antibiotics.[4]

Bacterial proteases and toxins degrade extracellular matrix components, while endotoxins trigger exaggerated inflammatory responses. Neutrophil dysfunction-due to hyperglycemia-induced impairment of chemotaxis, phagocytosis, and oxidative burst-compromises bacterial killing. The resultant tissue necrosis releases damage-associated molecular patterns (DAMPs), amplifying inflammation and creating a vicious cycle of injury and infection.[4]

Osteomyelitis, a feared complication, arises from contiguous spread or hematogenous seeding. Bacteria adhere to bone matrix via adhesins (e.g., *S. aureus* fibronectin-binding proteins), while biofilm formation within Haversian canals renders eradication difficult. Osteoclast activation and cytokine-mediated bone resorption (driven by RANKL, IL-1β) lead to progressive destruction.[5]

DISTINGUISHING INFECTIOUS FROM ISCHEMIC ETIOLOGIES

The IWGDF and IDSA criteria emphasize clinical signs of infection (purulence, erythema, warmth, tenderness) and systemic inflammation (leukocytosis, fever). However, ischemia can mask typical infectious signs due to impaired leukocyte trafficking. Hypoxia-inducible factor (HIF)-1α-driven angiogenesis in ischemia produces friable, hemorrhagic granulation tissue, whereas infection often yields malodorous, exudative wounds with undermining edges.[6]

Procalcitonin and presepsin may aid in discerning occult infection, while imaging (MRI, PET) differentiates osteomyelitis from Charcot neuroarthropathy. Angiography or transcutaneous oximetry quantifies perfusion deficits, guiding revascularization decisions.[6]

Background

Diabetic foot syndrome (DFS) represents one of the most debilitating and economically burdensome complications of diabetes mellitus (DM), with a lifetime incidence of DFUs approaching 19-34% among diabetic patients. The pathophysiological triad of peripheral neuropathy, micro- and macrovascular disease, and impaired immune response creates a fertile ground for ulceration, infection, and tissue necrosis, culminating in high rates of hospitalization, limb amputation, and mortality. Among the most critical clinical dilemmas in DFS management is the accurate differentiation between DFI and non-infected yet critically ischemic (CLI) ulcers, as misclassification frequently leads to inappropriate antibiotic use, delayed revascularization, and preventable amputations.[4,5]

PATHOPHYSIOLOGICAL UNDERPINNINGS OF DIAGNOSTIC COMPLEXITY

Neuropathic vs. ischemic vs. infective etiologies

Neuropathic ulcers arise from sensory-motor-autonomic neuropathy, leading to repetitive trauma, unrecognized pressure points, and dry, fissured skin. These wounds are typically painless, well-circumscribed, and non-inflammatory unless secondarily infected.[5]

Ischemic ulcers result from peripheral arterial disease (PAD), often presenting with punched-out necrotic edges, absent pulses, and dependent rubor mimicking infection. Hypoperfusion impairs wound healing and masks typical infectious signs due to diminished leukocyte recruitment.[6]

DFI is characterized by microbial invasion and host inflammatory response, with clinical manifestations ranging from local cellulitis to life-threatening necrotizing fasciitis. The IDSA/IWGDF classification stratifies DFI by severity (mild, moderate, severe), yet ischemia can obscure classic infectious signs and complicating diagnosis.[6]

Overlapping clinical and biochemical markers

Both DFI and CLI may exhibit erythema, warmth, edema, and elevated inflammatory markers (CRP, ESR, leukocytosis), leading to diagnostic overlap.[6]

Procalcitonin (PCT) has emerged as a potential discriminator, with higher specificity for bacterial infection than CRP, though its utility in chronic wounds remains debated.[7]

Hypoxia-driven inflammation in CLI can mimic infection on advanced imaging (e.g., bone marrow edema on MRI), increasing false-positive osteomyelitis diagnoses.[7]

Current guidelines and their limitations

The IDSA and IWGDF provide evidence-based criteria for DFI diagnosis:

IDSA definite DFI: ≥2 classic signs (purulence, erythema, warmth, tenderness, induration) or one sign plus systemic inflammation (fever, leukocytosis).

IWGDF probable DFI: Ulcer with surrounding inflammation plus either exudate or foul odor.[8]

However, critical ischemia blunts these signs: Dependent rubor may be mistaken for cellulitis. Necrotic tissue in CLI is often misattributed to infection rather than hypoxia. False-negative cultures occur in ischemic ulcers due to poor bacterial load from impaired perfusion.[8]

Common diagnostic and therapeutic pitfalls

Overdiagnosis of infection in CLI

Empiric antibiotics are frequently initiated for non-infected ischemic ulcers, contributing to antimicrobial resistance and *Clostridioides difficile* infections.[9]

Misinterpretation of imaging: MRI findings of bone marrow edema may reflect Charcot neuroarthropathy or ischemia rather than osteomyelitis.[6]

Underdiagnosis of infection in ischemic ulcers

"Silent" DFI: Neuropathy and ischemia may mask pain and fever, delaying treatment.[9]

Inadequate sampling: Superficial swabs often yield colonizers (e.g. Staphylococcus epidermidis), missing deep-tissue pathogens (e.g., *S. aureus Pseudomonas*).[9]

Failure to assess vascular status

Ankle-brachia, l index (ABI) is often falsely elevated due to medial arterial calcification.[9]

Toe pressures (<30 mmHg) or $TcPO_2$ (<25 mmHg) better predict healing potential in diabetic patients.[10]

The imperative for multidisciplinary care

Optimal management requires: Vascular surgery evaluation for revascularization in CLI.[10]

Infectious disease consultation to guide antibiotic duration (e.g., 6 weeks for osteomyelitis vs. 2 weeks for soft-tissue infection).[10]

Podiatric and wound care specialists for offloading and debridement.

The interplay between infection, ischemia, and neuropathy in DFUs demands a systematic, guideline-driven approach to avoid diagnostic errors. Enhanced clinician awareness of IDSA/IWGDF criteria, combined with vascular assessment and judicious imaging, is essential to reduce unnecessary antibiotic use and improve limb salvage rates. Future research should focus on novel biomarkers and advanced imaging techniques to further refine diagnostic accuracy.[10]

CURRENT CONSIDERATIONS IN THE MANAGEMENT

Diagnostic challenges in diabetic foot pathology

The diabetic foot represents a complex clinical entity where the interplay of microvascular complications, peripheral sensory neuropathy, and immunocompromised status creates a perfect storm for tissue breakdown and impaired healing. Current diagnostic paradigms must account for:

Neuro-ischemic vs. pure infectious presentations: While neuropathic ulcers typically present with well-demarcated edges, calloused margins, and minimal exudate, ischemic lesions demonstrate punched-out appearance with necrotic bases and poor granulation tissue formation. The infectious component superimposes erythema extending >0.5 cm from ulcer margins, purulent discharge, and malodorous secretions.[10]

Biomarker ambiguity: Traditional markers like C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) show poor specificity, with elevations seen in both infection (DFI) and CLI. Emerging evidence suggests:

Procalcitonin >0.3 ng/mL has 85% specificity for bacterial infection, presepsin (sCD14-ST) shows promise in distinguishing bacterial sepsis and matrix metalloproteinase-9 (MMP-9) elevations correlate with wound chronicity rather than infection status.[10]

CURRENT CLASSIFICATION SYSTEMS AND THEIR LIMITATIONS

The IDSA/IWGDF classification system for DFI provides a clinically useful framework but presents several practical challenges:

Severity stratification

Mild DFI: Local infection with ≥2 classic signs (erythema, warmth, pain, induration) but no systemic involvement.[10]

Moderate DFI: More extensive local infection or patient with metabolic instability (e.g., hyperglycemia, acidosis).[10]

Severe DFI: Systemic inflammatory response syndrome (SIRS) or hemodynamic instability.[10]

Critical limitations

Ischemic confounding: CLI patients may present with pseudo-infection signs including: Dependent rubor mimicking cellulitis, non-purulent necrosis misclassified as wet gangrene, elevated inflammatory markers secondary to tissue hypoxia.[7,10]

Neuropathic masking: Loss of protective sensation may obscure pain, while autonomic dysfunction causes dry, fissured skin that can be mistaken for infection

Vascular assessment imperatives

The WIfI (Wound, ischemia, foot infection) classification system from the society for vascular surgery provides a more comprehensive assessment framework:[10]

Key vascular parameters

Ankle-brachial index (ABI): Often unreliable due to medial calcinosis (consider toe-brachial index <0.7 as alternative).[11]

Transcutaneous oxygen pressure ($TcPO_2$): Values <30 mmHg predict poor healing.[11]

Angiosome concept: Direct vs. indirect revascularization impacts healing rates.[7]

Common vascular evaluation errors

Overreliance on palpable pulses (insensitive in diabetics).[11]

Failure to perform pre-and post-exercise ABI in borderline cases.[8]

Misinterpretation of monophasic waveforms on arterial duplex as "adequate" flow.[11]

Microbiological considerations

Optimal culture techniques remain controversial:

Sampling methodologies

Deep tissue biopsy (gold standard) vs. superficial swabs (high contamination risk), bone culture vs. histopathology for osteomyelitis diagnosis, molecular techniques (PCR, mass spectrometry) vs. conventional cultures.[8,11]

ANTIMICROBIAL STEWARDSHIP CHALLENGES

Overuse of broad-spectrum agents (e.g., piperacillin-tazobactam) for colonization. Inadequate coverage of biofilm-forming organisms (e.g., *P. aeruginosa*).[12] Prolonged durations for soft tissue infections without osteomyelitis.[8]

Imaging modalities

Appropriate use and interpretation-Advanced imaging presents both opportunities and pitfalls.

Modality-specific considerations

Plain radiography: Early osteomyelitis signs (periosteal reaction, cortical erosion) take 10-14 days to appear.

Sensitivity <60% for early bone infection.[12]

Magnetic resonance imaging (MRI): T1 hypointensity + T2 hyperintensity + contrast enhancement suggests osteomyelitis.[12]

Diffusion-weighted imaging (DWI) helps differentiate infection from Charcot.[12]

Pitfall: Ischemic bone marrow edema mimics infection.

Nuclear medicine: White blood cell (WBC) scintigraphy remains gold standard but lacks availability.[12]

FDG-PET/CT shows 90% accuracy but high cost.

THERAPEUTIC DECISION-MAKING

Current management algorithms must integrate:

MDTs approach

Vascular surgery (revascularization timing), infectious disease (antibiotic selection/duration), podiatry (offloading strategies) and endocrinology (glycemic control optimization).[13]

Common management errors

Delaying revascularization to "clear infection first" in ischemic DFI, overlooking nutritional deficiencies (albumin <3.0 g/dL impairs healing), inadequate surgical debridement of non-viable tissue and failure to address biomechanical abnormalities contributing to ulceration.[10,13]

Emerging diagnostic technologies

Innovative approaches show promise: Fluorescence imaging (MolecuLight i:X) for bacterial detection, point-of-care PCR for rapid pathogen identification, artificial intelligence algorithms for wound assessment and MicroRNA signatures for infection prediction.[14]

Quality metrics and outcome optimization

Performance indicators for DFU management: Time-to-antibiotics for severe DFI (<1 hour), vascular assessment completion rate (goal >90%), amputation-free survival at 1 year and antimicrobial days of therapy per DFI episode.[14]

CONCLUSION TOWARDS PRECISION MEDICINE IN DIABETIC FOOT CARE

The contemporary management of diabetic foot complications demands: Strict adherence to IDSA/IWGDF criteria while recognizing their limitations in ischemic presentations. Comprehensive vascular assessment before attributing inflammatory signs to infection. Judicious use of advanced diagnostics with understanding of modality-specific pitfalls. Multidisciplinary collaboration to address the multifactorial nature of DFUs.[15]

CONCLUSION

The management of diabetic foot pathology represents one of the most clinically challenging scenarios in contemporary medicine, where the intricate interplay between microvascular compromise, peripheral neuropathy, and immune dysfunction creates a diagnostic and therapeutic labyrinth. This review has systematically examined the critical distinctions between infected diabetic foot (DFI) as defined by IDSA/IWGDF criteria and non-infected yet critically ischemic (CLI) presentations, highlighting both current standards and persistent challenges in clinical practice. The overlapping clinical manifestations of DFI and CLI-including erythema, tissue necrosis, and systemic inflammatory responses-demand a methodical, evidence-based approach to differentiation. While IDSA/IWGDF criteria provide a valuable framework for infection diagnosis, their limitations in ischemic contexts necessitate concomitant vascular assessment using advanced modalities ($TcPO_2$, angiosome-directed angiography), judicious interpretation of biomarkers (recognizing that procalcitonin >0.5 ng/mL increases the probability of true infection by 3-fold compared to CRP alone), and multimodal imaging integration, particularly MRI with diffusion-weighted sequences to discriminate osteomyelitis from neuropathic osteoarthropathy. Current management strategies must balance antimicrobial stewardship (avoiding prolonged courses for colonized ischemic ulcers), revascularization urgency (endovascular-first approaches for Rutherford class 4-6 ischemia), and wound base preparation (enzymatic vs. sharp debridement based on perfusion status), with emerging data supporting 72-hour antibiotic timeouts when infection markers fail to correlate with clinical findings in suspected CLI mimics. Optimal outcomes correlate strongly with structured MDT involvement (vascular surgery, infectious disease, podiatry), standardized protocols for wound classification using both WIfI and SINBAD systems, and real-time telemedicine consultations in resource-limited settings. Despite advances, several areas require continued focus, including diagnostic uncertainty in "borderline" cases (approximately 15-20% of DFUs present with equivocal features, necessitating advanced techniques like 18F-FDG PET/CT or bone biopsy with histopathology), antibiotic resistance patterns (the rise of ESBL-producing organisms and MRSA in DFIs necessitates culture-directed therapy, yet <40% of centers routinely perform deep tissue sampling), and health systems barriers (disparities in access to hyperbaric oxygen therapy, advanced wound care products, and MDT clinics persist globally, disproportionately affecting outcomes in low-resource settings). The next frontier in diabetic foot management lies in molecular diagnostics (rapid PCR panels for *S. aureus*/*Pseudomonas* genotyping, microRNA signatures predicting infection severity), advanced imaging analytics (AI-assisted MRI interpretation reducing inter-reader variability, optical coherence tomography for real-time biofilm detection), and personalized medicine approaches (cytokine profiling to guide immunomodulatory therapies, microbiome mapping for targeted antimicrobial therapy), alongside global standardization efforts (consensus definitions for "infection vs. inflammation" in ischemic wounds, international registries tracking amputation prevention strategies). Final recommendations for clinical practice include adopting a "vascular-first" mentality (performing ankle-brachial index with waveform analysis on all DFU patients, considering angiosome-guided revascularization when $TcPO_2$ <30 mmHg), refining infection diagnosis (applying modified IDSA criteria in CLI patients requiring ≥3 signs for DFI diagnosis, utilizing bone culture-directed antibiotic regimens for osteomyelitis), implementing structured follow-up (weekly wound measurements with standardized photography, serum albumin/prealbumin monitoring to address catabolic states), and enhancing prevention strategies (custom therapeutic footwear for all patients with LOPS, community-based foot surveillance programs targeting high-risk populations). Diabetic foot epidemic demands nothing less than a paradigm shift from reactive to proactive care, where early vascular assessment, precision diagnostics, and collaborative care models converge to prevent the tragic trajectory from ulceration to amputation. While current guidelines provide essential scaffolding, their intelligent application-tempered by clinical wisdom and augmented by emerging technologies-will define next era of limb salvage success. As we stand at intersection of evidence-based medicine and innovation, our collective challenge remains: to transform the diabetic foot from costly complication into a preventable condition through relentless focus on accurate differentiation, timely intervention, and systems-based solutions.

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