



Complexities in Managing Diabetic Foot Ulcers: A Comprehensive Review

Nisarga HS^{1*}, Pradeepa MS², Sinjid R³

DOI:10.21760/jaims.11.8.26

^{1*} Nisarga HS, Internee, Rajeev Institute of Ayurvedic Medical Sciences and Research Centre, Hassan, Karnataka, India.² Pradeepa MS, Professor and Principal of Cauvery Ayurveda Medical College and Hospital, Mysuru, Karnataka, India.³ Sinjid R, Associate Professor, Department of Shalya Tantra, Rajeev Institute of Ayurvedic Medical Sciences and Research Centre, Hassan, Karnataka, India.

Diabetic foot ulcers over the heel region are particularly most challenging due to their anatomical location, compromised vascularity, neuropathy and high susceptibility to infection. In patients with Diabetes Mellitus, chronic hyperglycemia results in neuropathy, microangiopathy and impaired immune response, all of which delay wound healing. The heel's weight-bearing nature and limited soft tissue coverage predispose to deep tissue necrosis and osteomyelitis. Secondary infection and sinus formation further complicates management and increase the risk of amputation. This paper reviews the anatomical and pathophysiological factors contributing to these challenges.

Keywords: Diabetes mellitus, Diabetic foot ulcer, Diabetic heel ulcer

Corresponding Author

Nisarga HS, Internee, Rajeev Institute of Ayurvedic Medical Sciences and Research Centre, Hassan, Karnataka, India.

Email: hsnisarga4@gmail.com

How to Cite this Article

Nisarga HS, Pradeepa MS, Sinjid R, [Complexities in Managing Diabetic Foot Ulcers: A Comprehensive Review](#). J Ayu Int Med Sci. 2026;11(8):194-200.

Available From

<https://jaims.in/jaims/article/view/6600/>

To Browse



Manuscript Received
2026-05-19

Review Round 1
2026-06-04

Review Round 2
2026-06-20

Review Round 3
2026-07-06

Accepted
2026-07-21

Conflict of Interest
None

Funding
Nil

Ethical Approval
Not required

Plagiarism X-checker
11.36

Note



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Introduction

Diabetic foot ulcers are a major complication of Diabetes Mellitus and a leading cause of non-traumatic lower limb amputation worldwide. Among them, heel ulcers represent a high-risk subgroup due to their unique anatomical and biomechanical characteristics. The heel is a primary weight-bearing area with limited soft tissue coverage over the calcaneus, making it prone to deep tissue involvement and early osteomyelitis. In diabetes, peripheral neuropathy, microangiopathy and immune dysfunction further impair wound healing. Reduced perfusion, poor wound contraction and increased susceptibility to infection contribute to chronicity and sinus formation. Because of these combined anatomical and metabolic factors, diabetic heel ulcers are more difficult to treat and are associated with prolonged healing time and higher amputation rates compared to other foot ulcers. Understanding these challenges is essential for improving management strategies in wound healing.[1]

Anatomy of Heel and its Impact on Delayed Wound Healing

The heel is a specialized weight-bearing structure whose unique anatomy significantly complicates ulcer management. It is composed of thick keratinized skin tightly adherent to the underlying structures, limiting tissue mobility and making wound contraction and surgical closure difficult. Beneath the skin lies a dense fibrofatty subcutaneous heel pad, divided by fibrous septae, which functions as a shock absorber during gait. Deep to this structure lies the calcaneus, the largest tarsal bone of the foot, which bears a substantial portion of body weight. The plantar fascia originating from the calcaneus provides longitudinal arch support and further contributes to the biomechanical load transmitted through the heel. The vascular supply of the heel is derived primarily from branches of the tibial and peroneal arteries. The relatively superficial location of the calcaneus and the limited soft tissue coverage overlying it increase the risk of rapid spread of infection to bone, predisposing to osteomyelitis. Its biomechanical role as a primary weight-bearing surface increases mechanical stress thereby tissue regeneration is repeatedly hampered and delays healing process.[2]

Weight bearing biomechanics

Weight-bearing biomechanics plays a critical role in delayed healing of diabetic heel ulcers. Constant pressure during ambulation and compression while lying in supine position impair local tissue perfusion, resulting in ischemia and delayed granulation tissue formation. Elevated plantar pressures during gait further compromise wound healing. Unlike forefoot ulcers, heel ulcers are subjected to continuous axial loading during standing and walking with shear stress during movement.[3] Even minimal mechanical stress can disrupt fragile granulation tissue, thereby prolonging the inflammatory phase of wound healing.[4] Furthermore, effective offloading of the heel is more challenging than the forefoot due to anatomical and biomechanical factors, leading to persistent mechanical trauma. Consequently, heel ulcers demonstrate slower healing rates and have poor outcomes compared to plantar forefoot ulcers, primarily due to sustained pressure, impaired vascularity, and difficulty in pressure redistribution.[5,6]

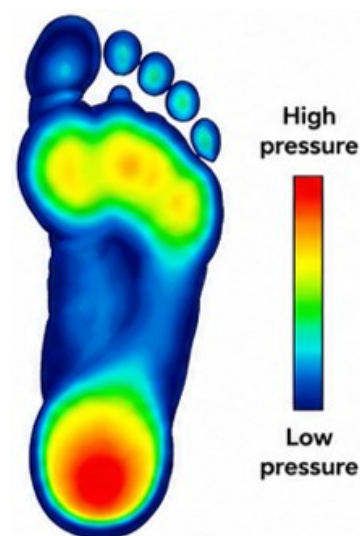


Fig. 1: High pressure regions of the foot

Limited soft tissue coverage

The heel has minimal soft tissue coverage between the skin and the underlying calcaneus. Although a dense fibrofatty heel pad overlies the calcaneus and functions as a shock absorber, it provides limited muscular cushioning and restricted tissue mobility. This anatomical arrangement predisposes heel ulcers to rapid deep tissue involvement. Because the calcaneus lies relatively superficial, infection can quickly extend to bone, leading to early calcaneal osteomyelitis.

Furthermore, the inherent anatomical rigidity of the heel limits natural mechanical wound contraction and compromises tissue adaptability during healing. In diabetic patients, this limited soft tissue coverage significantly increases the risk of early bone involvement, delayed granulation tissue formation, and poor epithelialization. Consequently, diabetic heel ulcers demonstrate prolonged healing time, chronicity, and a higher risk of amputation.[7]

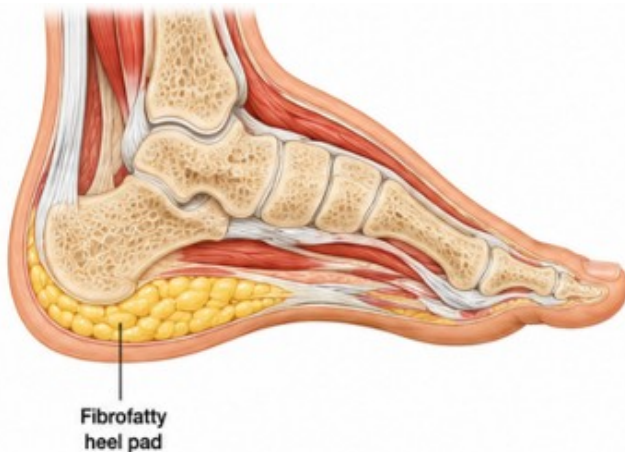


Fig. 2: Fibrofatty heel Pad

Impaired wound contraction in diabetes mellitus

Wound contraction is a crucial phase of healing mediated by myofibroblasts, which generate contractile forces to draw wound edges inward and reduce wound size. In Diabetes Mellitus, this process is markedly impaired due to multiple pathophysiological mechanisms. Hyperglycemia negatively affects fibroblast function by reducing cellular proliferation, decreasing collagen synthesis, and impairing extracellular matrix deposition. Additionally, there is diminished transformation of fibroblasts into myofibroblasts, resulting in reduced contractile force generation.

Persistent hyperglycemia also prolongs the inflammatory phase of wound healing, characterized by sustained inflammatory cytokine activity and delayed transition to the proliferative phase. This chronic inflammatory state further reduces tissue elasticity and compromises effective wound contraction.[8,9]

Impaired Blood supply

The heel belongs to a distinct Angiosome[10] with limited collateral circulation, making revascularization particularly challenging.

The calcaneal region receives its primary blood supply from the medial calcaneal branches of the posterior tibial artery and the lateral calcaneal branches of the peroneal artery. Because heel lesions lie within this relatively isolated vascular territory, they are highly vulnerable to ischemia. Peripheral arterial disease (PAD) commonly coexists in diabetic foot ulcer individuals and further compromises perfusion. Reduced oxygen delivery impairs fibroblast function, collagen synthesis, and overall tissue repair. Ischemia also promotes tissue necrosis and limits effective antibiotic penetration into infected tissues. Consequently, impaired blood supply significantly delays healing and increases the risk of amputation in diabetic heel ulcers.

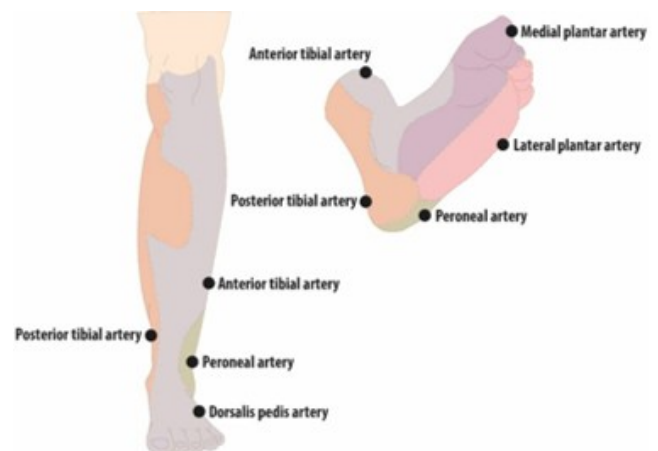


Fig. 3: Angiosome distribution in the foot

Diabetic Mellitus[11]

Diabetes Mellitus is characterized by chronic hyperglycemia, which adversely affects wound healing through multiple mechanisms:

Increased glucose in the tissue precipitates infection.

Elevated tissue glucose level in diabetes mellitus creates a favorable environment for microbial growth and significantly increase the risk of infection in diabetic foot ulcers. Approximately 15-25% of diabetic patients will develop a diabetic foot ulcer during their lifetime. The annual risk of developing diabetic foot ulcer is estimated to be about 2% among diabetic patient.[12]

Diabetic microangiopathy

Diabetic microangiopathy which affects microcirculation leading to hypoxia reduces nutrient supply to tissues. Peripheral arterial occlusive disease is 20% common in diabetic foot.

It may be atherosclerosis of major vessels like dorsalis pedis or posterior tibial arteries (macroangiopathy) leading into severe ischemia of foot or it may be thrombotic blockage of small vessels and arterioles causing thickening of the capillary basement membrane (microangiopathy). Collateral vessels are also involved in diabetics very early, aggravating the ischemic events.

Diabetic atherosclerosis

Diabetic atherosclerosis itself reduces the blood supply and leads to gangrene. Thrombosis can be precipitated by infection causing infective gangrene. Blockage occurs at plantar, tibial, and dorsalis pedis vessels.

Increased glycosylated hemoglobin in blood

It causes defective oxygen dissociation leading to more hypoxia. At tissue level there will be increased glycosylated tissue proteins, which prevents proper oxygen utilization and so aggravates hypoxia.

Peripheral Neuropathy

Diabetic neuropathy in foot involves all sensory, motor and autonomous components. Due to sensory neuropathy, minor injuries are not noticed and hence chronic infection occurs. Due to motor neuropathy, dysfunction of muscles, arches of foot and joints occurs. And loss of reflexes of foot occurs causing more prone for trauma and abscess. Due to autonomic neuropathy, skin will be dry, causing defective skin barrier and so more prone for infection. Tibial, medial and lateral plantar nerves are involved. Tibial nerve involvement causes intrinsic muscle paralysis leading onto claw and hammer toes. Plantar nerve involvement leads into loss of sensation in the sole causing recurrent multiple ulcers. Foot is felt like dead, senseless with walking like in the sand. Charcot's neuropathic joint is hypermobile unstable joints of the foot often with high vascularity. Muscle strength, functions of the arches of the foot, joint function and skin elasticity are lost.

Polymicrobial Infections in Diabetic Foot Ulcer

Polymicrobial infection is a common and serious complication of diabetic foot ulcers, significantly delaying wound healing. In patients with Diabetes Mellitus, hyperglycemia impairs neutrophil function and reduces local immunity, predisposing ulcers to bacterial colonization.

Initially, gram-positive organisms such as *Staphylococcus aureus* are commonly isolated. With chronicity, prolonged wound exposure, or prior antibiotic use, gram-negative organisms such as *Klebsiella* species may emerge. This shift indicates a more complex and potentially resistant infection. Secondary infections are frequent in chronic heel ulcers. Infection promotes persistent inflammation, tissue necrosis, and sinus formation. Culture and sensitivity testing is essential for targeted antibiotic therapy. Early identification and appropriate management are crucial to prevent progression to limb-threatening complications.[13,14]

Antibiotic Resistance in Diabetic Foot Ulcers

Antibiotic resistance is an emerging and significant challenge in the management of diabetic foot ulcers (DFUs). Chronic ulcers in patients with Diabetes Mellitus are frequently exposed to repeated and empirical antibiotic therapy, promoting development of resistant organisms. Common pathogens such as *Staphylococcus aureus*, including MRSA strains, and gram-negative bacteria like *Klebsiella* & *Pseudomonas* species increasingly demonstrate multidrug resistance. Biofilm formation within chronic wounds further reduces antibiotic penetration and efficacy. Poor vascularity in ischemic limbs also limits drug delivery to infected tissues. Inadequate dosing, incomplete treatment courses, and irrational antibiotic use also contribute to resistance. Multidrug-resistant infections are associated with prolonged hospital stay, higher treatment costs, and increased amputation risk. Therefore, culture-guided therapy, rational use of antibiotics, and strict glycemic control are essential to improve outcomes and limit resistance development.[15,16]

Bone Exposure, Osteomyelitis and Risk of Amputation

Bone exposure in diabetic heel ulcers markedly increases the risk of calcaneal osteomyelitis and subsequent amputation. The calcaneus lies superficially with minimal soft tissue coverage and sinus tracts frequently communicate directly with infected bone, facilitating rapid spread of infection. Osteomyelitis is difficult to eradicate due to poor vascularity of infected bone, sequestrum formation and limited antibiotic penetration, often requiring prolonged antibiotic therapy or surgical intervention. The presence of osteomyelitis significantly reduces the probability of wound healing and worsens overall prognosis.

Diabetic patients are particularly susceptible to infection due to impaired immune defenses, including reduced leukocyte function, defective chemotaxis, phagocytosis and altered inflammatory responses.[17] Gram-positive cocci, especially *Staphylococcus aureus*, are the most common pathogens in diabetic foot infections.[18] Peripheral neuropathy, angiopathy, delayed presentation & poor foot care further contribute to infection progression. Clinical diagnosis of osteomyelitis relies on signs of inflammation, probe-to-bone testing[19] and plain radiographs for assessment of osseous involvement and surgical planning.

Sinus Formation in Diabetic Foot Ulcers

A sinus tract is a blind-ending abnormal channel extending from a deep focus of infection, commonly bone or deep soft tissue, or to skin surface, allowing persistent discharge of pus or serous fluid. In diabetic heel ulcers, sinus formation usually represents advanced and chronic infection associated with deep tissue necrosis & underlying calcaneal osteomyelitis. Chronic infection, persistent abscess cavities, necrotic bone, and inadequate drainage contribute to development of these tracts. The pathophysiology is multifactorial, involving sustained infection, ischemia due to poor vascular supply and impaired immune response in Diabetes Mellitus. Sinus tracts often have deep, narrow openings in heel, making underlying infection difficult to assess clinically. They serve as reservoirs for bacteria, limit effective antibiotic penetration & maintain continuous purulent discharge. Because of hidden pockets of infection, medical therapy alone is frequently inadequate, necessary surgical exploration & aggressive debridement required. Sinus formation therefore indicates advanced disease, prolonged healing time, high recurrence rates & increased risk of amputation. Furthermore, heel ulcers with sinus formation demonstrate significantly longer healing durations compared to forefoot ulcers, reflecting complexity of infection & compromised local tissue conditions.[20]

Conclusion

The management of diabetic foot ulcers is significant clinical challenge due to their multifactorial pathogenesis, anatomical location, results in delayed recovery and high risk of complications such as osteomyelitis and amputation. Effective management requires multidisciplinary approach integrating meticulous wound debridement,

Strict glycemic control, appropriate antimicrobial therapy, pressure offloading, and advanced wound care strategies. Early identification of complications such as sinus formation and deep tissue infection is crucial to achieve optimal outcomes. Combined therapies, including both modern and traditional modalities, can aid in successful wound healing. Patient education, regular follow-up, and adherence to treatment protocols play vital role in preventing recurrence. Diabetic heel ulcers demand an individualized and evidence-based approach along with comprehensive understanding of anatomical and pathological complexities which prevent delay in wound healing, minimize complications, reduce recurrence and improving overall quality of life of affected individuals.

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