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Effect of Nutrition, Nursing Care, and Physical Rehabilitation on Recovery After Diabetic Foot-An Updated Review

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Abstract

Background: Diabetic foot ulcers (DFUs) represent one of the most severe complications of diabetes mellitus, contributing significantly to global morbidity, mortality, and healthcare burden. Their pathogenesis is multifactorial, involving peripheral neuropathy, peripheral arterial disease, impaired wound healing, and infection. DFUs remain the leading cause of non-traumatic lower-limb amputations and are associated with high recurrence and mortality rates.

Aim: This review aims to provide an updated and comprehensive analysis of the effects of nutrition, nursing care, and physical rehabilitation on the recovery of patients with diabetic foot ulcers.

Methods: A narrative review methodology was used, synthesizing current evidence on DFU etiology, epidemiology, pathophysiology, diagnostic evaluation, classification systems, and multidisciplinary management approaches. Special emphasis was placed on postoperative care, nutritional optimization, and rehabilitation strategies.

Results: Effective DFU management requires integrated multidisciplinary care, including early diagnosis, offloading, sharp debridement, infection control, revascularization when indicated, and glycemic optimization. Nursing care is essential for wound monitoring, infection prevention, patient education, and glycemic stabilization. Nutrition therapy improves wound healing by supporting tissue repair, immune function, and metabolic control. Physical therapy enhances mobility, improves circulation, reduces pressure-related injury through offloading strategies, and lowers recurrence risk. Coordinated care significantly reduces amputation rates and improves functional recovery.

Conclusion: Recovery from diabetic foot ulcers improves markedly when nursing care, nutrition support, and physical therapy are incorporated into a unified interdisciplinary treatment plan. This integrated approach enhances healing, reduces complications, and improves long-term outcomes.

Keywords: Diabetic foot ulcer, neuropathy, peripheral arterial disease, nutrition, nursing care, physical therapy, wound healing, rehabilitation.

Introduction

Diabetes mellitus represents a critical and escalating global health concern, exerting a profound impact on morbidity, mortality, and healthcare expenditures worldwide. Among its diverse spectrum of complications, diabetic foot ulcers constitute one of the most debilitating and clinically challenging conditions, significantly impairing patient quality of life while contributing disproportionately to disease burden and adverse outcomes. Epidemiological evidence indicates that approximately one-third of individuals diagnosed with diabetes will develop a foot ulcer during their lifetime, underscoring its high prevalence and clinical significance. Notably, diabetic foot ulcers remain the primary cause of nontraumatic lower-limb amputations on a global scale, reflecting both the severity of the condition and gaps in preventive and therapeutic strategies [1][2]. The pathogenesis of diabetic foot ulcers is complex and multifactorial, involving an interplay of peripheral neuropathy, peripheral arterial disease (PAD), and impaired wound healing capacity, often compounded by superimposed infection. Peripheral neuropathy contributes to loss of protective sensation, predisposing patients to unnoticed repetitive trauma, while PAD compromises tissue perfusion and oxygenation, thereby delaying healing processes. In addition, metabolic dysregulation associated with

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diabetes further impairs cellular repair mechanisms and immune function, increasing susceptibility to infection and chronic ulceration [3]. Importantly, diabetic foot ulcers should not be viewed solely as localized lesions but rather as clinical manifestations of systemic vascular pathology, frequently associated with widespread atherosclerotic disease. Consequently, their presence is strongly linked to an elevated risk of cardiovascular morbidity and premature mortality, emphasizing their prognostic significance beyond the affected limb [4][5].

The clinical trajectory of patients with diabetic foot ulcers is often complicated by a range of factors that influence outcomes. Ulcer severity, duration, presence of infection, and underlying PAD are critical determinants of healing potential. Additionally, comorbid conditions such as chronic kidney disease and cardiovascular disease, along with lifestyle factors including smoking and demographic variables such as male gender, further exacerbate the risk of poor prognosis. These elements collectively affect rates of wound healing, recurrence, and progression to major lower-limb amputation or death [1][7][8][9][10]. The consequences of major amputation are particularly severe, as they are associated with substantial functional impairment and a markedly reduced survival rate, with five-year mortality exceeding 50% among affected individuals [6]. Given the multifaceted nature of diabetic foot ulcers, their effective management necessitates a comprehensive and evidence-based approach that addresses both local wound care and systemic factors. Core therapeutic strategies include pressure offloading to minimize mechanical stress, prompt and appropriate infection control, revascularization procedures to restore adequate blood flow, and timely surgical assessment when indicated. Optimization of glycemic control and rigorous management of comorbidities are equally essential components of care. Due to the complexity of the condition, outcomes are significantly improved when patients are managed within a coordinated, multidisciplinary framework that integrates the expertise of healthcare professionals across various specialties, thereby ensuring a holistic and patient-centered approach to treatment [11][12][13][14].

Etiology

Ulcer Classification

Diabetic foot ulcers arise from complex and interrelated pathophysiological mechanisms, most commonly involving peripheral neuropathy, peripheral arterial disease, or a combination of both processes. These mechanisms underpin the classification of diabetic foot ulcers into three principal categories, namely neuropathic, ischemic, and neuro-ischemic ulcers. This classification is clinically relevant, as it guides both diagnostic evaluation and therapeutic decision-making, while also reflecting the underlying disease processes contributing to tissue breakdown and impaired healing. Neuropathic ulcers are the most frequently encountered subtype and are primarily attributed to peripheral neuropathy, which affects a substantial proportion of individuals with diabetes. These ulcers typically develop on the plantar surface of the foot, particularly at areas subjected to repetitive mechanical stress such as the metatarsal heads and the heel. Clinically, they are characterized by well-demarcated, punched-out lesions that are often surrounded by hyperkeratotic callus tissue. A defining feature of neuropathic ulcers is the absence of pain, which results from the loss of protective sensation. The pathogenesis involves both sensory and motor neuropathy, where sensory deficits prevent the perception of trauma, and motor dysfunction leads to altered foot biomechanics and abnormal pressure redistribution. Consequently, minor repetitive injuries remain unrecognized and untreated, ultimately progressing to ulceration. Peripheral neuropathy alone contributes to approximately 60% to 70% of diabetic foot ulcers, highlighting its dominant etiological role [1][11].

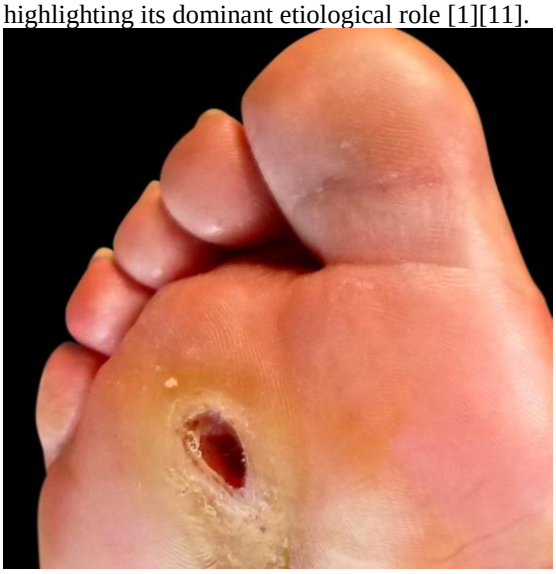


Fig. 1: Diabetic Ulcer.

In contrast, ischemic ulcers develop as a direct consequence of peripheral arterial disease, which compromises blood flow to the lower extremities. These ulcers are commonly located at the distal aspects of the toes or along the lateral margins of the foot, areas particularly vulnerable to reduced perfusion. Unlike neuropathic ulcers, ischemic lesions are typically painful and present with irregular borders, pale or necrotic tissue, and surrounding skin that appears cool to the touch. The absence or diminution of peripheral pulses is a key clinical finding, reflecting underlying arterial insufficiency. Peripheral arterial disease accounts for up to 50% of diabetic foot ulcers and significantly impairs wound healing due to inadequate oxygen and nutrient delivery to affected tissues [7][11][15].

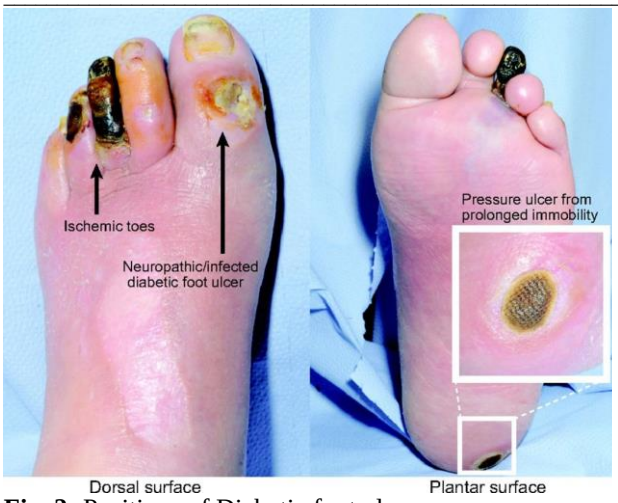


Fig. 2: Positions of Diabetic foot ulcer.

Neuro-ischemic ulcers represent a combination of both neuropathic and ischemic processes and are increasingly prevalent among older individuals with long-standing diabetes and multiple comorbid conditions. The clinical presentation varies depending on the relative contribution of neuropathy and ischemia, with ulcer location and pain intensity differing among patients. These ulcers are often more severe and demonstrate poorer healing outcomes due to the coexistence of impaired sensation and compromised vascular supply, making their management particularly challenging [11][16][17].

Table 3. Types of foot ulceration

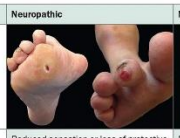
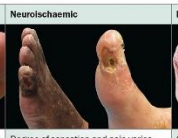
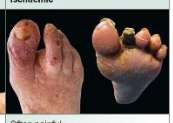
Characteristics	Neuropathic	Neuroischaemic	Ischaemic
Appearance			
Neurological symptoms	Reduced sensation or loss of protective sensation	Degree of sensation and pain varies	Often painful
Pressure	Periwound callus	Often minimal callus	Minimal or no callus
Vascular symptoms	Pedal pulses present	One or both pedal pulses absent	One or both pedal pulses absent
Sites	Weight-bearing surfaces (i.e. plantar foot, digital apices)	Digits and margins of the foot	Digits and periungual and interdigital areas

Fig. 3: Types of diabetic foot ulcers.

A wide range of risk factors contributes to the development of diabetic foot ulcers, with peripheral neuropathy representing the most significant determinant. Motor neuropathy alters the structural integrity of the foot, leading to deformities such as claw toes, hammer toes, and prominent metatarsal heads. These anatomical changes result in abnormal pressure distribution and increased callus formation, which predisposes the skin to breakdown and ulceration. Sensory neuropathy further exacerbates this risk by eliminating protective sensations, thereby preventing patients from recognizing pain, pressure, or temperature changes. As a result, repetitive micro-trauma accumulates over time, ultimately leading to tissue damage and ulcer formation [18][19]. Autonomic neuropathy also plays a contributory role by reducing sweat production, which leads to dry, fissured skin and impaired microvascular regulation. These changes increase susceptibility to both ulceration and infection [17]. Peripheral arterial disease significantly amplifies the risk of ulcer development and adversely affects healing outcomes. Its prevalence is markedly higher among individuals with diabetes compared to the general population, and it serves as an independent predictor of both ulcer occurrence and progression. Reduced perfusion to the lower extremities limits tissue viability and impairs the delivery of essential nutrients required for wound repair. The coexistence of peripheral arterial disease and neuropathy further compounds the risk, creating a synergistic effect that accelerates tissue breakdown. Current intersocietal guidelines emphasize the importance of routine screening for peripheral arterial disease and risk-based monitoring to facilitate early detection and intervention [20][21].

Chronic kidney disease represents another critical risk factor, independently associated with an increased incidence of diabetic foot ulcers. A substantial proportion of individuals with diabetes develop chronic kidney disease, and among these patients, the prevalence of foot ulceration is notably high. The underlying mechanisms include an increased burden of neuropathy, higher rates of peripheral arterial disease, and compromised immune function, all of which contribute to impaired wound healing. The risk is further elevated among patients undergoing dialysis, with a significantly higher prevalence of ulceration compared to those not receiving dialysis. Advanced stages of chronic kidney disease are associated with a markedly increased risk of major lower-limb amputation, emphasizing the need for vigilant monitoring and preventive care. Regular foot examinations at intervals of one to three months are recommended for individuals with end-stage renal disease to facilitate early detection and management [22][23][24][25]. Additional contributing factors include prolonged duration of diabetes, suboptimal glycemic control, prior history of ulceration or amputation, advanced age, male sex, elevated body mass index, hypertension, and dyslipidemia. Each of these factors independently and collectively increases the likelihood of ulcer development while simultaneously impairing the healing process. The cumulative effect of these comorbidities underscores the necessity for comprehensive, multidisciplinary prevention strategies that address both systemic and local determinants of disease progression [26][18].

Epidemiology

Diabetic foot ulcers constitute a prevalent and clinically significant complication of diabetes mellitus, representing a major global health concern with substantial implications for patient outcomes and healthcare systems. Epidemiological data demonstrate that foot ulceration is not only common but also associated with considerable morbidity and mortality. A systematic review conducted in 2017, encompassing data from 16 countries, estimated the global prevalence of diabetic foot ulcers at approximately 6.3%, highlighting the widespread nature of this

condition across diverse populations. Furthermore, lifetime risk estimates indicate that between 19% and 34% of individuals with diabetes will develop a foot ulcer, underscoring the high cumulative burden of this complication over the disease course [1]. The distribution of diabetic foot ulcer prevalence exhibits notable regional variability, reflecting differences in healthcare access, socioeconomic factors, and disease management practices. North America reports the highest prevalence at approximately 13%, whereas Oceania demonstrates the lowest rate at around 3%. In Africa, the prevalence is estimated at 7.2%, exceeding the 5.5% reported in Asia, which may be attributed to disparities in early detection, preventive strategies, and availability of specialized care. Within the United States, analysis of Medicare fee-for-service data indicates a point prevalence of 8% among individuals with diabetes. This prevalence is not uniform across populations, as significant variations exist based on geographic location and ethnicity. Higher rates have been documented among American Indian, Black, and Hispanic populations, suggesting the influence of social determinants of health, healthcare accessibility, and potential genetic predispositions [2].

Sex-based differences are also evident in the epidemiology of diabetic foot ulcers, with a higher prevalence observed among men compared to women, reported at 4.5% and 3.5%, respectively. Additionally, the type of diabetes appears to influence ulcer prevalence, as individuals with type 2 diabetes exhibit a slightly higher prevalence rate of 6.4% compared to 5.5% in those with type 1 diabetes. These differences may be explained by variations in disease duration, comorbidity profiles, and the degree of metabolic control, all of which contribute to the risk of ulcer development [2]. A particularly concerning aspect of diabetic foot ulcer epidemiology is the high rate of recurrence following initial healing. Evidence indicates that recurrence occurs in approximately 42% of patients within one year and increases to 65% within five years, reflecting the chronic and relapsing nature of the condition. This pattern emphasizes the need for sustained preventive measures and long-term patient monitoring even after apparent clinical resolution [1]. Mortality associated with diabetic foot ulcers is substantial and represents a critical dimension of the disease burden. Meta-analytical data reveal that the five-year mortality rate approaches 50%, a figure comparable to or exceeding that of several major malignancies. The prognosis is further worsened in patients who undergo major lower-limb amputation, as this intervention is associated with significantly increased mortality risk. These findings highlight the severe systemic implications of diabetic foot ulcers and reinforce their role as a marker of advanced disease and poor overall health status [6][27]. As the global prevalence of diabetes continues to rise, the incidence and total number of diabetic foot ulcer cases are expected to increase correspondingly. This trend is particularly concerning in low- and middle-income regions, where access to multidisciplinary foot care services and early intervention strategies remains limited. Consequently, addressing the epidemiological burden of diabetic foot ulcers requires not only improved clinical management but also enhanced preventive strategies, public health initiatives, and equitable access to specialized care.

Pathophysiology

Diabetic foot ulcers arise from a multifactorial and progressive pathophysiological process in which metabolic, vascular, neurological, and mechanical abnormalities converge to produce tissue breakdown. Chronic hyperglycemia plays a central role in initiating and sustaining this process through its deleterious effects on both macrovascular and microvascular structures, as well as peripheral nerves. Sustained elevation of blood glucose levels leads to oxidative stress, accumulation of advanced glycation end products, and activation of inflammatory pathways, all of which contribute to structural and functional damage in multiple tissues. Peripheral neuropathy emerges as a key consequence, characterized by sensory, motor, and autonomic dysfunction. Sensory neuropathy results in the loss of protective sensation, preventing patients from detecting pain, pressure, or thermal injury, thereby allowing repetitive trauma to occur unnoticed [28]. Motor neuropathy further exacerbates this condition by inducing muscular imbalance and atrophy, leading to structural deformities such as claw toes, hammer toes, and prominent metatarsal heads. These deformities alter normal foot biomechanics and redistribute plantar pressure to focal areas, significantly increasing the risk of skin breakdown. Concurrently, autonomic neuropathy impairs sweat gland function, resulting in dry, inelastic skin that is prone to fissuring and ulceration. The combination of increased plantar pressure and callus formation creates a localized environment highly susceptible to tissue injury, where repetitive stress exceeds the skin's capacity for repair [29].

Peripheral arterial disease is another critical contributor to the pathophysiology of diabetic foot ulcers. It is highly prevalent among individuals with diabetes and is characterized by arterial narrowing, reduced elasticity, and impaired blood flow to the lower extremities. This compromised perfusion limits oxygen and nutrient delivery to tissues, thereby impairing cellular metabolism and reducing the capacity for wound healing. In addition, vascular insufficiency promotes ischemia, which accelerates tissue necrosis and increases the likelihood of complications such as gangrene and amputation. The coexistence of neuropathy and ischemia creates a particularly high-risk environment in which both injury and impaired healing occur simultaneously

[7][30]. Impaired wound healing represents a defining feature of diabetic foot ulcer pathophysiology. The normal sequence of wound repair is disrupted, with a prolonged inflammatory phase and inadequate progression to tissue regeneration. Chronic low-grade inflammation persists due to dysregulated cytokine activity, while angiogenesis is defective, limiting the formation of new blood vessels required for tissue repair. Fibroblast dysfunction further compromises extracellular matrix production, and excessive activity of matrix metalloproteinases leads to degradation of essential structural proteins. The accumulation of advanced glycation end products interferes with cellular signaling and collagen synthesis, collectively hindering effective healing and prolonging ulcer duration [31][32]. Infection introduces an additional layer of complexity, often transforming a localized lesion into a severe and potentially limb-threatening condition. Diabetic foot ulcers frequently harbor polymicrobial communities that form structured biofilms on the wound surface. These biofilms provide a protective environment for microorganisms, reducing their susceptibility to host immune responses and antibiotic therapy. As a result, infections become persistent and difficult to eradicate, maintaining a cycle of inflammation and tissue destruction. The presence of biofilms not only delays wound healing but also increases the risk of systemic spread and severe complications, including sepsis and amputation [33].

Histopathology

Histopathological examination of diabetic foot ulcers reveals a spectrum of structural and cellular alterations that reflect the chronic and multifactorial nature of the disease process. At the epidermal level, there is typically clear evidence of ulceration, characterized by a loss of normal epithelial continuity and disruption of the stratified squamous architecture. The edges of the ulcer often display hyperkeratosis and acanthosis in the surrounding skin, changes that arise from chronic mechanical stress and repetitive pressure. Despite attempts at repair, re-epithelialization is frequently incomplete and disorganized, with impaired keratinocyte migration and differentiation contributing to delayed wound closure [34]. Within the dermis, significant abnormalities are observed in both the cellular and extracellular components. The dermal layer is often composed of dense fibrocollagenous tissue, reflecting chronicity and repeated cycles of injury and repair. However, the formation of functional granulation tissue is markedly impaired. Fibroblasts, which play a central role in collagen synthesis and tissue remodeling, are reduced in number and exhibit diminished proliferative capacity. This dysfunction limits the deposition of new extracellular matrix and compromises the structural integrity of the healing tissue. Additionally, angiogenesis is defective, resulting in reduced capillary density. The small blood vessels that are present often demonstrate pathological changes such as basement membrane thickening, endothelial cell swelling, and hyaline degeneration, all of which are characteristic of diabetic microangiopathy and contribute to localized hypoxia [35].

The inflammatory component of diabetic foot ulcers is typically chronic and heterogeneous. Histological analysis reveals a mixed inflammatory infiltrate composed predominantly of lymphocytes, plasma cells, and macrophages, with variable involvement of neutrophils, particularly in the presence of secondary infection. These immune cells often exhibit functional impairment, leading to an ineffective and prolonged inflammatory response that fails to transition into the proliferative phase of wound healing. Persistent inflammation promotes further tissue damage and delays repair. Areas of necrosis and fibrin deposition are commonly observed, reflecting ongoing tissue injury and impaired clearance of damaged material [36]. In advanced or long-standing ulcers, pathological changes extend into deeper tissues, including muscle, adipose tissue, and bone. Myonecrosis and fat necrosis may be evident, indicating severe ischemic injury and metabolic dysfunction. In cases complicated by infection, osteomyelitis may develop, characterized by inflammatory destruction of trabecular bone, accompanied by reactive sclerosis and attempts at bone remodeling. These deep tissue changes significantly increase the risk of limb loss and complicate clinical management. Overall, the histopathological features of diabetic foot ulcers illustrate a failure of coordinated tissue repair, marked by persistent inflammation, impaired vascularization, and defective cellular function, which together underpin the chronicity and high complication rate associated with this condition.

History and Physical

Clinical History

A comprehensive and structured clinical history forms the cornerstone of effective evaluation and management of patients with diabetes presenting with foot ulcers. The assessment begins with detailed documentation of the ulcer itself, including its anatomical location, duration, and any identifiable precipitating factors such as trauma, ill-fitting footwear, or walking barefoot. Associated local symptoms must be carefully explored, including the presence of discharge, malodor, and pain, although pain may be absent in neuropathic ulcers. Systemic features such as fever, rigors, and malaise should also be assessed, as they may indicate spreading infection or systemic involvement. A thorough history must include prior episodes of foot ulceration, previous antibiotic use, and any history of minor or major amputations. In addition, prior vascular interventions such as angioplasty or bypass surgery should be recorded, as these have implications for both prognosis and management. A detailed diabetes history is essential and should include the type and

duration of the disease, as well as the presence of associated microvascular and macrovascular complications. Microvascular complications include diabetic retinopathy and neuropathy, while macrovascular disease encompasses cerebrovascular events such as transient ischemic attacks and strokes, carotid artery disease, ischemic heart disease, and peripheral arterial disease. Glycemic control must be critically reviewed, including the frequency of hypoglycemic episodes, symptoms of hyperglycemia, and any previous episodes of diabetic ketoacidosis or hyperosmolar hyperglycemic state. Medication history should be carefully documented, with particular attention to agents such as sodium-glucose cotransporter 2 inhibitors, antiplatelet drugs, statins, and anticoagulants, in addition to adherence to prescribed therapeutic footwear and offloading strategies. The history should also address symptoms suggestive of peripheral neuropathy, including hypoesthesia, paresthesia, dysesthesia, or complete loss of sensation, as well as features of peripheral arterial disease such as intermittent claudication, rest pain, and nonhealing wounds. Lifestyle factors play a significant role and must be documented, including smoking status with quantification in pack-years, alcohol consumption, occupation, mobility status, and use of walking aids. Visual impairment should be assessed, as it may affect self-care and ulcer detection. Comorbid conditions such as congestive heart failure and chronic kidney disease are important contributors to lower limb edema and impaired healing. The clinical history should conclude with an exploration of the patient's perspectives, concerns, and expectations, which are critical for ensuring adherence and delivering patient-centered care [31][33][35].

Clinical Examination

A meticulous and systematic physical examination is required to complement the clinical history. Both lower limbs should be fully exposed from the knees to the toes and assessed comparatively. Inspection should focus on identifying signs of vascular insufficiency and tissue compromise, including pallor, cyanosis, dependent rubor, mottling, edema, and gangrene. Dermatological findings such as calluses, corns, fissures, fungal infections, and areas of skin breakdown must be noted. Previous surgical scars from amputations or revascularization procedures should be documented. Trophic changes, including hair loss, atrophic skin, muscle wasting, and nail abnormalities, provide additional evidence of chronic vascular and neuropathic disease. Particular attention must be given to structural deformities such as claw toes, hammer toes, hallux valgus, pes cavus, and pes planus, all of which contribute to abnormal pressure distribution. Signs of Charcot neuroarthropathy, including midfoot collapse, rocker-bottom deformity, erythema, and increased local temperature, should be actively sought. The ulcer itself requires detailed characterization, including its location, size, shape, depth, and the nature of its edges and base. The presence of granulation tissue, slough, necrosis, or exposed deeper structures such as tendon or bone should be documented. Surrounding skin changes, including erythema, induration, warmth, and edema, provide important clues to infection. The type and quantity of exudate should also be assessed. The probe-to-bone test is a valuable bedside tool for detecting underlying osteomyelitis, indicated by a firm or gritty resistance when probing the ulcer. Neurological assessment should include evaluation of light touch, pinprick sensation, vibration sense, joint position, and monofilament testing. Palpation is essential to assess local temperature, tenderness, and the extent of soft tissue swelling, distinguishing between localized and generalized edema. Peripheral pulses, including dorsalis pedis, posterior tibial, popliteal, and femoral arteries, must be palpated bilaterally to assess vascular status. Examination of the abdomen may reveal an expansile pulsation suggestive of an abdominal aortic aneurysm [28][29] . Assessment of footwear and offloading devices is an integral component of the examination, as improper use may contribute to ulcer formation or delayed healing. A broader systemic examination, including cardiovascular, renal, and hepatic evaluation, is necessary to identify contributing factors such as heart failure or systemic causes of edema. Charcot neuroarthropathy typically presents as a warm, swollen, erythematous foot with preserved or bounding pulses and underlying neuropathy, often without ulceration in early stages, whereas chronic cases demonstrate deformity and sensory loss.

Diabetic Ulcer Classification Systems

Standardized classification systems are essential for assessing ulcer severity, guiding management, and facilitating communication among healthcare providers. The International Working Group on the Diabetic Foot and Infectious Diseases Society of America classification focuses on the severity of infection. It categorizes ulcers into four grades, ranging from uninfected wounds with no local or systemic signs to severe infections characterized by systemic inflammatory response [37]. This system is widely used in clinical practice due to its simplicity and relevance in guiding antimicrobial therapy. The SINBAD scoring system provides a practical and structured approach to ulcer assessment at the bedside. It evaluates six domains, including site, ischemia, neuropathy, bacterial infection, area, and depth, assigning a binary score to each parameter. The cumulative score reflects ulcer severity, with higher scores associated with poorer healing outcomes and increased risk of amputation. Its simplicity makes it particularly useful for routine documentation and clinical audits [38]. The WIfI classification system offers a more comprehensive evaluation by integrating

wound characteristics, degree of ischemia, and severity of infection. Each component is graded on a scale, and the combined score is used to stratify patients into stages that predict the risk of major amputation within one year. This system also helps determine the potential benefit of revascularization, thereby supporting clinical decision-making in complex cases. Higher WIfI stages are strongly associated with adverse outcomes, making it a valuable tool in risk stratification and treatment planning [39].

Evaluation

Laboratory Studies

Laboratory investigations constitute an essential component in the comprehensive evaluation of diabetic foot ulcers, particularly in identifying infection, assessing systemic involvement, and guiding therapeutic decisions. A complete blood count is routinely performed and may demonstrate leukocytosis with neutrophilia in the presence of infection. However, this response may be attenuated or absent in individuals with diabetes due to underlying immune dysfunction, thereby limiting its sensitivity in detecting severe infections. Inflammatory markers provide additional supportive information. C-reactive protein is widely used as a nonspecific indicator of inflammation or infection and is frequently elevated in diabetic foot infections, although normal values do not exclude the presence of underlying pathology. Similarly, erythrocyte sedimentation rate is commonly elevated in inflammatory states, and values exceeding 70 mm per hour raise strong suspicion for underlying osteomyelitis. Procalcitonin has emerged as a more specific biomarker for bacterial infection and may assist in differentiating infectious from noninfectious causes of inflammation. Despite their utility, these markers should be interpreted in conjunction with clinical findings and imaging studies rather than relied upon in isolation [33][40][41][42]. Assessment of metabolic and organ function is equally important. Renal and hepatic function tests are necessary to identify baseline abnormalities or complications arising from infection, sepsis, or pharmacological interventions. Glycemic control must be evaluated through measurement of glycated hemoglobin, as poor glycemic control is strongly associated with impaired wound healing and increased susceptibility to infection. Acute illness and infection may further destabilize glucose homeostasis, necessitating close monitoring and potential adjustment of antidiabetic therapy, including escalation to insulin where appropriate [43][44]. Microbiological evaluation is a critical step in cases of suspected infection. Tissue specimens obtained through curettage or biopsy are preferred for Gram staining and culture, as they provide more accurate identification of causative organisms compared to superficial swabs, which are often contaminated by colonizing flora. In cases where osteomyelitis is suspected, bone biopsy remains the gold standard for definitive microbiological diagnosis when feasible [33].

Imaging Studies

Imaging plays a pivotal role in the diagnostic workup and management planning of diabetic foot ulcers. Plain radiography is typically the first-line imaging modality and is indicated in ulcers that extend beyond superficial layers, when there is suspicion of gas within soft tissues, exposed bone, or a positive probe-to-bone test. Radiographs can identify structural abnormalities such as fractures, foreign bodies, osteolysis, and features suggestive of osteomyelitis or Charcot neuroarthropathy. However, early stages of bone infection may not be apparent on plain films, necessitating further evaluation with advanced imaging techniques [45].

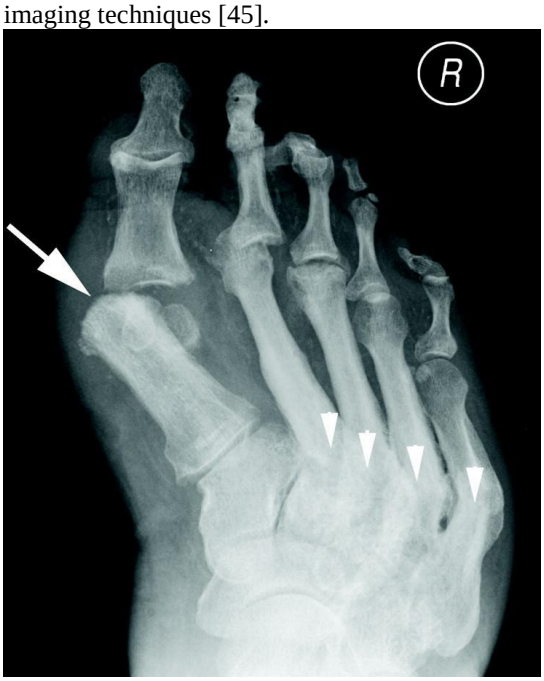


Fig. 4: X-ray evaluation of diabetic ulcer.

Magnetic resonance imaging provides superior soft tissue contrast and detailed visualization of bone marrow changes, making it the modality of choice for detecting osteomyelitis, deep soft tissue infections, abscess formation, septic arthritis, and Charcot neuroarthropathy when radiographic findings are inconclusive. In selected cases, additional modalities such as radionuclide imaging or positron emission tomography may be employed to confirm the diagnosis and delineate the extent of infection [33][45].

Evaluation of Peripheral Artery Disease

Assessment of peripheral arterial disease is fundamental in patients with diabetic foot ulcers, as vascular insufficiency significantly influences healing outcomes and risk of amputation. Initial evaluation relies on noninvasive techniques, including pedal Doppler waveform analysis, ankle-brachial index, and toe-brachial index measurements. Peripheral arterial disease is considered less likely when the ankle-

brachial index ranges between 0.9 and 1.3, the toe-brachial index is 0.70 or greater, and pedal waveforms are triphasic or biphasic. Conversely, severe arterial insufficiency is suggested by ankle pressures below 50 mm Hg or an ankle-brachial index less than 0.5, both of which are associated with poor healing potential and increased amputation risk. Toe pressure measurement using pneumatic plethysmography provides valuable prognostic information regarding wound healing. Pressures of 30 mm Hg or higher are associated with improved healing likelihood, whereas lower values indicate a significantly increased risk of limb loss. Transcutaneous oxygen pressure measurement offers an alternative assessment of tissue perfusion, particularly when toe pressures cannot be obtained. Values of 25 mm Hg or higher are associated with improved healing probability, while lower values correlate with increased risk of major amputation [7].

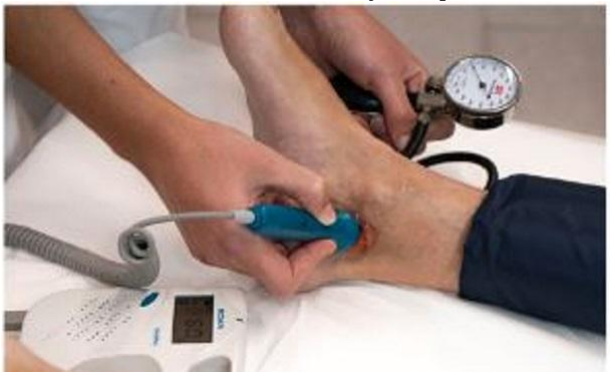


Fig. 5: ankle-brachial index measurement.

It is important to note that ankle-brachial index measurements may be falsely elevated in individuals with diabetes due to arterial calcification, reducing their reliability. In such cases, toe pressures, toe-brachial index, and transcutaneous oxygen measurements are preferred. When revascularization is under consideration, detailed anatomical imaging of the arterial system using computed tomography angiography, magnetic resonance angiography, or catheter-based digital subtraction angiography is essential to guide interventional planning and optimize clinical outcomes [46][47].

Treatment / Management

Management of diabetic foot ulcers requires a structured, evidence-based strategy implemented by an interprofessional diabetic foot team. Early referral following systematic assessment is crucial, particularly in cases of moderate-to-severe infection, critical limb ischemia, or rapidly progressing ulcers, where urgent intervention may prevent major complications [33]. Coordinated care ensures that medical, surgical, vascular, and rehabilitative aspects are addressed simultaneously, improving healing rates and reducing recurrence. Offloading remains the central component of therapy for neuropathic plantar ulcers. Nonremovable knee-high devices, such as total contact casts or nonremovable walkers, provide consistent pressure relief for forefoot and midfoot ulcers, promoting tissue recovery [25][29]. Sharp debridement of callus and necrotic tissue should be performed regularly to optimize wound healing. Selection of dressings should be individualized, based on exudate level, wound depth, local infection, patient comorbidities, and cost-effectiveness [28]. Appropriate offloading combined with meticulous wound care has been shown to accelerate closure and reduce ulcer recurrence.

Antibiotic therapy is indicated only in the presence of clinical infection. Mild soft-tissue infections generally require 1 to 2 weeks of oral antibiotics, whereas moderate-to-severe infections often necessitate 2 to 3 weeks of intravenous therapy in a hospital setting. Severe or chronic infections are typically polymicrobial, frequently including gram-negative rods and anaerobes, while mild infections are predominantly caused by Staphylococcus aureus. Empiric antibiotic selection should cover gram-positive cocci for mild cases, with broader-spectrum regimens used for moderate-to-severe or ischemic ulcers [33]. Osteomyelitis complicating foot ulcers may be managed conservatively in selected patients, particularly forefoot infections without peripheral artery disease, exposed bone, or abscess requiring drainage, using antibiotic therapy for approximately six weeks [33][48]. Surgical intervention, including minor amputations such as toe, ray, or transmetatarsal procedures, is indicated when infection involves metatarsal bones, necrotic tissue, or septic joints. Postoperative antibiotic duration varies from 2 to 5 days following minor amputation with negative bone margins to up to three weeks if margins are culture-positive. Urgent surgical consultation is warranted for necrotizing fasciitis, deep abscesses, compartment syndrome, severe ischemia, or gangrene, with early debridement within 24 to 48 hours significantly improving outcomes [33].

Reconstructive procedures, including Achilles tendon lengthening, gastrocnemius recession, and metatarsal head resection, reduce recurrence of plantar ulcers. Targeted interventions such as flexor tenotomy or hallux osteotomy address apical ulceration. Protective footwear and custom orthoses remain critical to prevent re-ulceration [29]. Management of peripheral artery disease is integral to ulcer healing and limb preservation. Any infected ulcer with suspected ischemia warrants urgent vascular evaluation [33]. Revascularization through endovascular angioplasty or bypass surgery is indicated for significant stenosis or critical limb ischemia, guided by vascular anatomy and patient comorbidities [7][46]. Major below-knee amputation may be necessary when infection and ischemia persist despite optimal medical, surgical, and vascular interventions. Optimization of glycemic control, blood pressure management, and initiation of statin and

antiplatelet therapy are essential adjuncts, reducing ulcer recurrence and improving long-term outcomes.

Postoperative and Rehabilitation Care

Postoperative and rehabilitation care for patients with diabetic foot ulcers requires a coordinated, interprofessional approach, with nursing, nutrition, and physical therapy playing pivotal roles in optimizing outcomes. Nursing staff are central to ongoing monitoring and wound management. They perform frequent assessments of the surgical site, including inspection for erythema, exudate, necrosis, or signs of infection, and regularly measure vital signs to detect early systemic complications. Nurses administer medications, including antibiotics when clinically indicated, and ensure precise glycemic control by monitoring blood glucose and adjusting insulin or oral hypoglycemics in collaboration with the endocrinology team. Education is a core nursing responsibility, guiding patients on wound care, offloading techniques, and early warning signs of infection or ischemia to prevent re-ulceration [33][28]. Nutrition support is equally critical in the postoperative period. Dietitians assess the patient's caloric and protein needs to support tissue repair and immune function. Optimizing glycemic control through individualized meal planning, carbohydrate monitoring, and supplementation with vitamins and micronutrients enhances wound healing. Specific attention to protein intake, amino acid balance, and micronutrients such as vitamin C, zinc, and arginine supports collagen synthesis and fibroblast activity. Nutrition interventions also focus on reducing cardiovascular risk, incorporating weight management, and improving lipid profiles. Collaboration between the dietitian, endocrinologist, and primary care provider ensures that pharmacologic therapy, including GLP-1 agonists and SGLT2 inhibitors, complements dietary strategies to enhance glycemic and cardiovascular outcomes.

Physical therapy is essential to restore mobility, improve circulation, and prevent complications such as muscle atrophy or joint stiffness. Early mobilization, when safe, promotes tissue perfusion, reduces edema, and enhances functional recovery. Therapists design individualized exercise programs tailored to neuropathy, ischemia, and musculoskeletal limitations, incorporating strength training, range-of-motion exercises, and gait retraining. Offloading strategies, including the use of total contact casts, postoperative shoes, or custom orthoses, are implemented in collaboration with physical therapists to protect vulnerable areas and redistribute plantar pressures. Education on safe ambulation, progressive weight bearing, and balance training reduces fall risk and prevents recurrent ulceration [29][28]. Interdisciplinary coordination ensures continuity of care, particularly during transitions from inpatient to outpatient settings. Nurses, dietitians, and physical therapists provide patient-centered education and follow-up, reinforcing adherence to wound care protocols, nutrition plans, and exercise regimens. Periodic reassessment of healing potential using toe pressures or TcPO2 measurements guides further interventions, including potential revascularization if perfusion remains inadequate [7][28]. Overall, postoperative and rehabilitation care for diabetic foot ulcers is most effective when nursing, nutrition, and physical therapy are integrated into a structured, interprofessional framework. Each discipline contributes uniquely to wound healing, infection prevention, metabolic optimization, and functional recovery, collectively reducing the risk of recurrence, major amputation, and cardiovascular complications.

Conclusion:

Diabetic foot ulcers remain a major global health challenge due to their complex pathogenesis, high recurrence rates, and strong association with morbidity, amputation, and mortality. This review highlights that optimal DFU management extends far beyond traditional wound care; instead, it requires a comprehensive, interdisciplinary strategy that addresses both local wound factors and systemic health determinants. Nursing care plays a central role through continuous wound monitoring, infection surveillance, glycemic management, patient education, and support for adherence to offloading and medication protocols. Nutrition is equally vital, as adequate protein, micronutrients, and glycemic control are fundamental to effective tissue repair, immune competency, and reduction of cardiovascular risk. Physical rehabilitation further enhances healing by improving circulation, restoring mobility, preventing joint stiffness, reducing plantar pressures, and minimizing fall and re-ulceration risks. When these domains—nursing, nutrition, and physical therapy—are coordinated within a multidisciplinary team, patient outcomes improve significantly. Healing rates increase, hospitalization and amputation rates decline, and long-term functional independence becomes more achievable. Ultimately, comprehensive care not only promotes wound closure but also addresses underlying metabolic, vascular, and biomechanical factors, offering patients a more sustainable path to recovery and improved quality of life.

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