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Review Article

Motor Diabetic Neuropathy and the Diabetic Foot: Pathophysiology, Clinical Features, and the Role of Multidisciplinary Rehabilitation-A Narrative Review

Benazir S[1]*, Manoj Abraham[2] and Kannan[2]

[1]Professor, Department of Physiotherapy, The Tamil Nadu Dr. M.G.R. Medical University, Tamilnadu, India

[2]Principal, Department of Physiotherapy, The Tamil Nadu Dr. M.G.R. Medical University, Tamilnadu, India

***Corresponding Author:** Benazir S, Professor, Department of Physiotherapy, The Tamil Nadu Dr. M.G.R. Medical University, Tamilnadu, India.

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Abstract

Diabetic foot syndrome is a severe incapacitating consequence of diabetes mellitus, emerging from a complex interplay of vascular insufficiency, biomechanical stress, infection, and peripheral neuropathy. Historically, clinical focus has heavily favoured sensory nerve degradation, as the loss of protective pain perception allows repeated micro-traumas to go unnoticed. However, motor neuropathy remains a critically underappreciated yet plays an equal role as destructive catalyst in the ulceration cascade.

The insidious denervation and subsequent neurogenic atrophy of the foot's intrinsic musculature—specifically the lumbricals, interossei, and short flexors—disrupt the delicate balance between flexor and extensor muscle groups. This structural deterioration directly precipitates severe architectural abnormalities of foot, including pes cavus, claw-toe, and hammer-toe deformities. Consequently, natural gait mechanics and dynamic balance are compromised, forcing abnormal plantar pressure onto vulnerable areas like the metatarsal heads and the apex of the great toe. These mechanical shifts create an ideal environment for reactive hyperkeratosis (callus formation), subcutaneous haemorrhage, and inevitable full-thickness epidermal breakdown.

This comprehensive narrative review explores the underlying cellular mechanisms and clinical trajectory of motor diabetic neuropathy. It highlights the importance of early diagnostic modalities, transitioning from bedside clinical signs such as impaired heel-walking and absent calcaneal reflexes to advanced assessments like nerve conduction studies (NCS) and quantitative pedobarographic (plantar pressure mapping). Furthermore, the review evaluates established wound grading frameworks, including the Wagner, University of Texas, and PEDIS classification systems, which guide systematic therapeutic interventions.

Crucially, this analysis underscores the profound efficacy of proactive, rehabilitation-focused management strategies delivered through a multidisciplinary framework. Instead of relying solely on reactive wound care, evidence heavily supports early biomechanical interventions. The integration of rigorous off-loading techniques such as Total Contact Casting (TCC), targeted physiotherapy, progressive resistance training, and gait re-education is paramount. By addressing the root biomechanical imbalances caused by motor nerve damage, structured team-based care can successfully halt the cycle of chronic ulcer recurrence, preserve lower-limb function, and drastically minimise the incidence of preventable non-traumatic amputations.

Keywords: Diabetic Foot; Motor Neuropathy; Plantar Pressure; Off-Loading; Multidisciplinary Rehabilitation; Intrinsic Muscle Atrophy; Gait Dysfunction

Introduction

Diabetes mellitus is a major public health challenge of the current century, affecting a huge population of more than half a billion individuals globally and it imposes devastating burden on health systems in both high- and low-income countries. Among its numerous chronic complications, diabetic foot disease is unique and distinguished by an exceptional, intricate complex pathophysiology, encompassing the triad of peripheral neuropathy, peripheral arterial disease, structural foot deformity, soft-tissue ulceration, limb-threatening infection, and non-traumatic lower-limb amputation [1].

Epidemiological evidence indicates that approximately one in every four individuals with diabetes will experience at least one foot ulcer during their lifetime [2]. Globally, diabetic foot ulcers are among the most frequent reasons for diabetes-related hospitalisation and account for a disproportionate share of non-traumatic lower-limb amputations [3]. The pathogenesis of diabetic foot ulceration follows a sequential progression initiated by peripheral neuropathy and structural deformity. Initially, sensory impairment compromises nociception, rendering the patient unaware of repetitive microtrauma to the insensate extremity. Motor neuropathy causes muscle imbalances that progressively alter and distort the biomechanical architecture of the foot. When persistent mechanical stress is exerted upon these structurally compromised tissues, tissue breakdown and epidermal disruption occurs inevitably. The subsequent superimposition of peripheral ischemia combined with microbial infection causes secondary pathophysiological complications, which severely impair the wound-healing and exponentially elevates the risk of lower extremity amputation.

The consequences of diabetic foot disease have more delirious effect beyond the wound itself. Chronic ulceration impairs mobility, undermines personal independence, and is associated with a clinically meaningful reduction in health-related quality of life [4]. The direct and indirect costs of care are comparable to those of major malignancies — a burden felt most acutely in resource-limited settings, where access to specialist care remains inconsistent [5]. Yet up to 80% of diabetes-related amputations are considered preventable through early risk identification, structured patient education, appropriately fitted protective footwear, and timely treatment of pre-ulcerative lesions [6].

International clinical guidelines, including those of the International Working Group on the Diabetic Foot (IWGDF), advocate for structured multidisciplinary team care incorporating diabetologists, podiatrists, vascular surgeons, physiotherapists, and orthotists — an approach consistently associated with reduced amputation rates [7]. Within this clinical framework, rehabilitation has transitioned from a supportive intervention to a primary cornerstone of therapeutic management.

Targeted exercise regimens, incorporating balance training, foot musculature strengthening for pressure off-loading, and motor neuropathy-specific interventions along with orthotic modalities (e.g., protective insoles), gait re-education, and biofeedback for sensory-deficient extremities, synergistically improve functional outcomes and mitigate ulcer recurrence rates. This review specifically examines motor diabetic neuropathy as a primary driver of structural foot deformities and subsequent ulceration [7]. Furthermore, it evaluates evidence-based, rehabilitation-centric strategies for both sensory and motor neuropathies designed to facilitate wound healing and interrupt this degenerative pathophysiological cycle.

Motor diabetic neuropathy

Diabetic neuropathy encompasses a wide spectrum of nerve fibre involvement affecting peripheral somatic and autonomic pathways. Distal symmetric polyneuropathy (DSPN) is its most common prevalent form, which includes simultaneous impairment of sensory, motor, and autonomic fibres, though it is sensory loss that typically dominates the early clinical picture [8]. The disease progression involves advancing motor involvement manifests primarily through distal extremity muscle weakness, structural foot deformities, altered gait mechanics, and postural instability. These multiple pathological changes synergistically alter the biomechanical profile of the lower extremity, establishing the situations that causes skin breakdown and recurrent plantar ulceration.

Diabetic foot syndrome is the ultimate cumulative anatomical and functional consequences of all parallel processes converging as one. Sensory impairment renders the insensate foot unable to perceive the constant repetitive micro-trauma; motor neuropathy produces weakness of the intrinsic foot musculature, toe deformity, and abnormal plantar pressure distribution; and autonomic dysfunction results in anhidrosis, xerosis, and skin fissuring that facilitate bacterial colonisation. Superimposed ischaemia from peripheral arterial disease retards healing, and the immunological impairment characteristic of diabetes attenuates the normal response to infection. Structural complications such as Charcot neuroarthropathy further distort pressure distribution, compounding ulceration risk [9].

On electrophysiological evaluation, sensory and motor fibre abnormalities frequently appear concurrently, although overt clinical motor signs such as, the inability to walk on the heels, classified as stage 2b DSPN by Dyck and colleagues — tend to manifest at a later stage [10]. An intrinsic diabetic myopathy may further potentiate neuropathy-driven weakness, and structured exercise intervention has demonstrated measurable benefit in preserving motor function in patients with DSPN [11]. The histological and molecular level, motor diabetic neuropathy involves progressive axonal degeneration, segmental demyelination, impaired axonal transport, and selective loss of large myelinated fibres. These changes are mediated by hyperglycaemia-driven mechanisms including enhanced polyol pathway flux, oxidative stress, reduced neurotrophic factor availability, and endoneural microvascular dysfunction all of which compromise both axonal integrity and Schwann cell function, reducing nerve conduction velocity and amplitude [12].

Risk factors for diabetic foot ulceration

The most robust predictors of foot ulcer development include a previous history of ulceration or amputation, peripheral neuropathy, structural foot deformity, peripheral arterial disease, limited joint mobility, elevated plantar pressure, and the use of ill-fitting footwear [13]. Additional contributory factors encompass peripheral oedema, obesity, inadequate glycaemic control, visual impairment, and several modifiable mechanical determinants particularly callus formation, unrelieved repetitive loading, and minor trauma that escapes detection owing to sensory deficits [14]. Lower-limb hygiene, combined with a lack of patient awareness regarding the therapeutic benefits of foot mobility and targeted exercises, often delays clinical presentation and further exacerbates individual risk profiles. A comprehensive understanding of the complex interplay among these behavioural, educational, and physiological variables is critical for accurate patient risk stratification and for the efficient implementation of targeted preventive interventions.

Clinical presentation

The clinical manifestations of diabetic motor neuropathy typically follow an insidious onset, evolving over months to years. Early symptom is characterised by lower extremity fatigue during ambulation, impaired forefoot and digit dorsiflexion during the swing phase of gait resulting in sluggish clearance and recurrent fall episodes and a subjective perception of postural instability. Additionally, patients frequently report nocturnal lower limb cramping, predominantly within the calf musculature, while advanced stages may present with fasciculations detectable via electrodiagnostic testing.

Upon physical examination, findings include an inability to perform heel-walking or maintain weight-bearing on the heels, weakness of the ankle dorsiflexors and digit extensors, and diminished or absent calcaneal (ankle) deep tendon reflexes. Chronic, protracted cases often exhibit a pes cavus deformity. As denervation advances, pathology extends proximally to affect the tibialis anterior and peroneal muscle groups. In rare instances of severe, long-standing disease, upper extremity involvement may occur, illustrating the classic length-dependent, dying-back gradient of neuropathic degeneration.

Motor neuropathy preferentially affects the distal lower-limb musculature, most notably the intrinsic foot muscles, ankle dorsiflexors, and toe extensors [14]. The imbalance between extrinsic and intrinsic foot muscle groups leads progressively to claw-toe and hammer-toe deformity, prominence of the metatarsal heads, and redistribution of plantar loading towards pressure-vulnerable sites [15]. Gait analysis confirms a reduction in ankle power generation during the push-off phase, decreased walking speed, and impaired static and dynamic balance changes collectively shown to elevate fall risk in this population [16]. Electrophysiological testing further confirms that this motor fibre involvement, quite independent of sensory loss, exerts and establishes a clinically meaningful destabilising effect on both gait mechanics and plantar loading distribution.

Contractures and reduced joint mobility most commonly affect the first metatarsophalangeal joint, the subtalar joint, and the ankle, which further restricts functional mobility and alters foot-floor contact patterns, compounding the biomechanical vulnerability created by muscle imbalance [15]. Collectively, these structural alterations exacerbate the frequency of high-risk falls, curtail participation in community-based activities, and adversely impact psychological well-being. Consequently, these compounding deficits severely compromise health-related quality of life (HRQoL) and independence in activities of daily living (ADLs).

Modern imaging and muscle structural analysis

Although nerve conduction studies are valuable for assessing functional electrical activity, state-of-the-art imaging methods have become essential for measuring the physical breakdown of the affected muscles. Tools such as magnetic resonance imaging (MRI) and high-definition ultrasound enable accurate assessments of muscle structure. These technologies can uncover significant muscle shrinkage and fat replacement (steatosis) in the intrinsic muscles of the foot before obvious, severe deformities appear [17].

These imaging scans demonstrate that the buildup of fat within the muscles is strongly linked to the severity of the motor neuropathy, serving as an effective, non-invasive indicator of tissue damage and disease worsening [18]. Additionally, techniques like ultrasound elastography can identify early increases in muscle stiffness and changes in tendon behaviour. This provides a detailed look at the microscopic alterations that occur before large-scale joint stiffness and soft-tissue contractures develop [19].

Pressure redistribution and surgical management

To protect against the mechanical weaknesses caused by nerve loss, specialised treatments are necessary to stabilise the lower limbs and prevent tissue damage. Medical professionals often prescribe tailored orthotic devices, including total contact insoles and ankle-foot orthoses (AFOs). These aids are designed to support permanent deformities, regulate irregular joint movements, and artificially recreate a safer, more balanced spread of pressure across the sole of the foot during walking [20].

If non-invasive pressure-relief methods are insufficient to reduce areas of high physical stress—especially in patients with stiff toe deformities or severe equinus, preventative surgery may be recommended [21]. Operations such as lengthening the Achilles tendon, gastrocnemius recession, or specific flexor tenotomies are designed to help the foot achieve a flatter, more natural resting position on the ground. By correcting the damaging forces caused by muscle imbalances, these surgical approaches greatly lower the chances of developing new or recurring plantar ulcers [22].

Pathophysiology

The diabetic foot represents the clinical endpoint of several pathological processes: peripheral neuropathy, peripheral arterial disease, biomechanical abnormality, susceptibility to infection, and impaired wound healing [17]. Chronic hyperglycaemia and dyslipidaemia concurrently drive these pathological processes through distinct, multifactorial mechanisms. These include progressive axonal degeneration with resultant denervation and neurogenic muscular atrophy, as well as segmental demyelination that impairs nerve conduction velocity. Furthermore, there is increasing recognition of a concomitant diabetic myopathy characterised by abnormal intramyocellular lipid accumulation, muscle fibre-type transformation, and mitochondrial dysfunction. Because these neurogenic and myopathic alterations occur in parallel, the resulting functional weakness frequently exceeds the severity predicted by axonal loss alone.

Histological and imaging studies of peripheral nerves reveal axonal atrophy, segmental demyelination, endoneural microvascular changes, and markedly reduced regenerative capacity to proportionally overcome damage – the findings that correlate positively with suboptimal glycaemic control, longer diabetes duration, and the presence of coexisting microvascular complications [12]. Exercise-based rehabilitation programmes and tighter long-term metabolic control may attenuate the rate of progression and preserve residual motor function [11].

At the level of the foot, the interplay between the three neuropathic subtypes creates a highly vulnerable tissue environment. Sensory loss abolishes the physiological warning signal of pain so that harmful repetitive mechanical forces act without prompting any protective behavioural response. Motor neuropathy distorts foot architecture through progressive muscle strength imbalance. Autonomic neuropathy eliminates sympathetic sudomotor activity, producing dry, rigid skin with impaired barrier function and increased susceptibility to damage, fissuring and bacterial invasion. These changes converge with arteriolar microvascular dysfunction and thickening of capillary basement membranes to reduce effective tissue perfusion so that even a modest degree of ischaemia can tip the foot from a state of compensated risk to one of active tissue breakdown [17].

The intrinsic foot muscles, namely the lumbricals, interossei, and short flexors, are disproportionately vulnerable to diabetic denervation because of their distal anatomical location and their dependence on the smallest-diameter myelinated fibres. As these muscles undergo neurogenic muscular atrophy, the dynamic balance between long flexors and extensors is lost, producing the characteristic deformities of motor neuropathy: claw toes, hammer toes, prominent and exposed metatarsal heads, and an elevated longitudinal arch. These structural changes redirect plantar loading onto anatomically vulnerable points, most commonly the plantar surfaces of the metatarsal heads and the apex of the great toe, creating conditions favourable to callus formation, subcutaneous haemorrhage, and ultimately, ulceration [15].

The progression of diabetic foot disease relies heavily on the triad of peripheral neuropathy (sensory, motor, and autonomic), combined with vascular compromise. While sensory loss masks injury, motor neuropathy alters the physical architecture of the foot.

Metabolic and cellular triggers of neuromuscular deterioration

Long-term systemic issues—chiefly elevated blood sugar and abnormal cholesterol levels—serve as the primary metabolic catalysts for the breakdown of peripheral muscles and nerves. These harmful elements operate through a complex variety of pathways that damage both the structure of muscle fibres and the transmission of nerve signals. Microscopic and imaging assessments consistently reveal a severely impaired ability for cellular repair; a finding strongly linked to a longer history of diabetes and poor metabolic management [12].

- **Nerve Fibre and Myelin Damage:** The progressive shrinking of nerve axons and patchy loss of their protective myelin sheaths drastically slow down nerve signal speeds, leading to nerve-induced muscle wasting [12].
- **Diabetic Myopathy:** A simultaneous muscular disease characterised by abnormal fat buildup within muscle cells, altered muscle fibre types, and impaired mitochondrial function.
- **Small Blood Vessel Alterations:** Changes in the tiny blood vessels that supply the nerves restrict essential nutrient delivery. However, rigorous blood sugar control and targeted physical therapy can help safeguard the remaining muscle strength [11].

The Three-Part Neuropathic Threat and Tissue Fragility

The overlapping effects of damaged sensory nerves, impaired autonomic functions, and poor circulation create an extremely vulnerable tissue environment that is highly susceptible to infection and breakdown. Because of this compounded risk, even a minor drop in blood supply can quickly push the foot from a stable state into one of active ulceration [17].

Disease Component	Clinical Outcome
Sensory Nerve Damage	Silences the body's natural pain warnings, permitting repeated minor physical injuries to happen without the patient noticing or adjusting their step [17].
Autonomic Nerve Damage	Halts normal sweating mechanisms, leading to severely dry, rigid skin that easily cracks and invites bacterial infections [17].
Small Vessel Dysfunction	The thickening of tiny capillary walls and impaired arterioles severely restrict healthy blood flow to the tissues, drastically slowing the healing process [17].

Table 1: Component and clinical out come.

Local muscle nerve loss and mechanical breakdown

The small, intrinsic muscles within the foot (such as the short flexors, interossei, and lumbricals) are exceptionally prone to nerve-induced wasting due to their location at the body's far extremities and their reliance on fine myelinated nerve fibres. This localised muscle deterioration severely destabilises the foot's structural integrity and physical mechanics.

- **Disruption of Muscular system:** The normal counterbalance between the long flexing and extending muscles is ruined, which directly causes the physical foot distortions typical of this condition [15].
- **Physical Malformations:** Over time, individuals develop pronounced structural changes, including a highly arched foot, prominent metatarsal bones, hammer toes, and claw toes [15].
- **Dangerous Pressure Shifts:** These architectural changes force the mechanical weight of walking away from robust

areas onto highly fragile spots (especially the balls of the feet and the tips of the big toes). This unnatural pressure distribution sets the perfect stage for heavy calluses, bleeding under the skin, and eventually, open ulcers [15].

Neuropathy Subtype	Physiological/Histological Changes	Direct Contribution to Foot Pathomechanics	Secondary Complications
Sensory	Distal symmetric polyneuropathy (DSPN), loss of large and small myelinated fibers.	Abolishes the baseline warning signal of pain; leads to an insensate foot.	Unperceived repetitive micro-trauma, minor mechanical trauma escaping detection.
Motor	Progressive axonal degeneration, segmental demyelination, selective loss of large myelinated fibers.	Denervation and neurogenic muscular atrophy of intrinsic foot muscles (lumbricals, interossei, short flexors).	Muscle imbalance, claw-toe/hammer-toe deformity, prominent metatarsal heads, elevated longitudinal arch, altered plantar loading.
Autonomic	Damage to sympathetic pathways.	Eliminates sympathetic sudomotor activity.	Anhidrosis, xerosis, rigid skin with impaired barrier function, skin fissuring, and bacterial colonization

Table 2: Comparative Triad of Peripheral Neuropathy Subtypes.

Diagnostic approach

Clinical assessment serves as the primary, most accessible initial tier of diagnosis. In patients with established diabetes, the presence of cardinal bedside indicators specifically, an inability to heel-walk, ankle dorsiflexion weakness, diminished or absent calcaneal reflexes, and prominent intrinsic muscle atrophy provides strong clinical grounds for further assessments and neurophysiological evaluation.

Nerve conduction studies (NCS) remain the diagnostic gold standard for diabetic motor neuropathy. Characteristic electrophysiological findings include reduced compound muscle action potential (CMAP) amplitudes secondary to axonal loss, mild-to-moderate deceleration of motor conduction velocity indicating segmental demyelination, and prolonged or absent F-wave latencies. Notably, altered F-wave latency serves as a highly sensitive, early marker of motor fibre involvement, particularly within proximal nerve segments. These electrophysiological abnormalities demonstrate broad consistency across both type 1 and type 2 diabetes, suggesting a shared pathophysiological mechanism irrespective of the primary metabolic subtype.

Plantar pressure mapping using pedobarographic platforms provides quantitative documentation of abnormal loading patterns and is an invaluable tool for identifying pre-ulcerative high-pressure zones before skin breakdown occurs [16]. Semmes–Weinstein monofilament testing and biothesiometry remain standard methods for detecting concurrent sensory loss and grading its severity. Advanced neuroimaging modalities including muscle magnetic resonance imaging (MRI) for the quantification of intrinsic muscle atrophy and diffusion tensor imaging (DTI) for peripheral nerve assessment—are currently confined primarily to research settings, though their translational clinical utility remains under active investigation.

A systematic, well-structured foot examination encompassing aspects such as inspection, neurological assessment, vascular evaluation, and biomechanical analysis should be performed mandatorily at least annually in all patients with diabetes and more frequently in those with established prolonged neuropathy, prior ulceration, or other high-risk features [7]. Pre-ulcerative diagnosis and targeted preventive strategies significantly mitigate the risk of lower extremity amputation. Early identification of motor neuropathy enables the timely implementation of targeted exercise interventions for insensate, well-vascularized feet. These therapeutic regimens preserve intrinsic muscle mass, prevent the collapse of the plantar arch, and optimise the distribution of plantar pressure loads, thereby averting irreversible structural deformities prior to the onset of ulceration.

Diagnostic Tool	Assessment Targets	Gold Standard/Clinical Findings	Clinical Utility
Nerve Conduction Studies (NCS)	Motor and sensory nerve fibers.	Diagnostic Gold Standard: Reduced CMAP amplitude (axonal loss), slowed conduction velocity (demyelination), prolonged/absent F-wave latencies.	Quantifies underlying fiber degeneration irrespective of metabolic diabetes subtype.
Plantar Pressure Mapping (Pedobarographic)	Biomechanical loading and force distribution.	Identifies abnormal weight-bearing, metatarsal head overloading, and pre-ulcerative high-pressure zones.	Allows for prospective risk-profiling and objective monitoring of orthotic performance.
Semmes–Weinstein Monofilament & Biothesiometry	Cutaneous sensation and vibration perception thresholds.	Loss of protective sensation grading.	Standard bedside method for stratifying concurrent sensory loss.
Advanced Neuroimaging (Muscle MRI / DTI)	Muscle volume and peripheral nerve architecture.	Quantifies deep intrinsic muscle atrophy and tracks nerve pathway structural updates.	Currently limited to clinical research applications.

Table 3: Diagnostic Modalities for Motor and Sensory Deficits.

Ulcer formation: The Edmonds mechanical pathway

Edmonds' mechanical pathway model provides a structured conceptual framework explaining the sequence by which neuropathy-induced deformity progresses to established ulceration [18]. The model describes a stepwise cascade:

- Loss of protective plantar sensation
- Progressive structural foot deformity secondary to motor imbalance
- Redistribution of plantar loading with abnormal pressure concentration
- Focal elevation of plantar pressure at vulnerable anatomical sites
- Reactive callus formation over high-pressure areas
- Subcutaneous haemorrhage beneath thickened callus
- Epidermal breakdown and ulceration

Classification System	Core Assessment Domains	Clinical Advantages	Limitations
Wagner System	Lesion depth, tissue necrosis, and presence/extent of gangrene (6 grades).	Simple, rapid clinical application.	Lacks explicit integration of independent vascular (ischaemia) or infection markers. Most commonly applied tool in Indian clinical practice.
University of Texas (UT) System	Wound depth, integrated alongside independent variables of infection and ischaemia.	Significantly higher prognostic granularity and accuracy regarding amputation risk.	Gaining substantial ground in tertiary and academic Indian medical centers.
PEDIS System	5 specific domains: Perfusion, Extent, Depth, Infection, Sensation.	Highly standardized outcome reporting.	Developed explicitly for academic research and clinical trials by the IWGDF.

Table 4: Classification system of ulcer.

The pathological role of plantar calluses

Plantar calluses are among the most clinically significant findings in individuals with diabetic feet at risk. While their initial formation may represent a physiological adaptive response to localised mechanical stress, unchecked progression converts them into direct precursors of ulceration [19]. Calluses in diabetic feet arise through accelerated keratinocyte proliferation in response to excessive shear and compressive forces that are amplified by the claw-toe deformity and intrinsic muscle atrophy with decreased heel pad, a characteristic feature of motor neuropathy. As sensory loss prevents the patient from perceiving abnormal loading, normal weight-bearing mechanics are not modulated, leading to progressive metatarsal head overloading and relative unloading through the toes during the propulsive phase of gait and during high-demand functional activity of lower limbs.

As the callus thickens, its transition from a pressure-buffering dissipating structure is transformed to a pressure-concentrating one, which can cause forces to be transmitted from the underlying bone to progressively transmit weight to the thinning layer of viable soft tissue. This generates shear stress vectors within the dermis and sub dermis, leading to mechanical microhaemorrhage, pathological inflammatory tissue breakdown, and thus ultimately full-thickness ulceration. The majority of neuropathic plantar ulcers arise through this mechanism from chronic, repetitive, moderate loading rather than from a single acute traumatic event because the absence of protective sensation prevents the reflex gait modifications that would otherwise interrupt the process [20].

In high-risk patient populations, calluses have been associated with approximately one-fifth of all observed structural foot deformities, with most ulcers attributable to sustained plantar stress rather than acute injury [19]. Preventive management, therefore, centres on patient self-inspection education, with biofeedback and strengthening exercises to prevent arch collapse and maintain structural and functional integrity of the foot duly focused on; regular structured foot reviews; pressure-redistributing insoles and therapeutic footwear; and routine callus debridement interventions collectively shown to reduce incident ulcer rates compared with standard care advice alone [20]. Plantar pressure mapping and Semmes–Weinstein monofilament testing facilitates prospective identification of feet at imminent risk before ulceration develops.

Classification and staging of diabetic foot ulcers

Several validated classification systems are used to grade diabetic foot ulcers systematically and to guide therapeutic decision-making. The Wagner grading system acts as one of the earliest and most widely used. It stratifies ulcers across six grades according to lesion depth and the presence and extent of gangrene [21]. While its simplicity facilitates rapid clinical application, the system does not explicitly incorporate ischaemia or infection status, which limits its prognostic granularity.

The University of Texas (UT) wound classification system addresses this limitation by integrating infection and ischaemia as independent variables alongside wound depth, thereby substantially improving prognostic accuracy and providing more nuanced guidance for management decisions [22]. The PEDIS system, which categorises ulcers according to five domains: Perfusion, Extent, Depth, Infection, and Sensation, was developed specifically to standardise outcome reporting in diabetic foot research and is recommended by the IWGDF (International Working Group of Diabetic Foot) for academic and clinical trial purposes [23]. In Indian clinical practice, the Wagner system remains the most commonly applied grading tool, though increasing adoption of the UT classification is observed in tertiary and academic centres.

Infection and peripheral arterial disease

Diabetic foot infections are characteristically polymicrobial, involving a mixture of Gram-positive cocci — most importantly Staphylococcus aureus and streptococcal species — Gram-negative enteric bacilli, and obligate anaerobes in deep or necrotic wounds [24]. Infectious progression is accelerated in patients with diabetes by a complex interplay of multifactorial mechanisms. These include various factors such as impaired neutrophil chemotaxis and phagocytic activity, reduced local tissue oxygenation, and the formation of chronic wound biofilms, highly organised, polymicrobial communities that exhibit heightened resistance to conventional antimicrobial therapies and impede the physiological healing cascade. Consequently, infected ulcers necessitate prompt clinical evaluation, rigorous microbiological sampling, and standardised classification utilising established severity frameworks, such as those promulgated by the Infectious Diseases Society of America (IDSA), to optimise empirical antibiotic selection and establish appropriate thresholds for surgical intervention.

Peripheral arterial disease (PAD) is a critical determinant of wound healing potential and is associated with the most adverse clinical outcomes in diabetic foot disease [25]. The distribution of atherosclerotic occlusive disease in diabetes characteristically involves infrapopliteal vessels — the anterior and posterior tibial and peroneal arteries, which are frequently heavily calcified. Non-invasive haemodynamic measures — most notably toe systolic blood pressure and transcutaneous oxygen tension ($TcPO_2$) — are accordingly recommended to assess ischaemia severity and to identify patients who may benefit from endovascular or surgical revascularisation [25].

Management	Primary Interventions and Gold Standards	Biomechanical/Physiological Mechanisms
Mechanical Off-loading	* Gold Standard: Total Contact Casting (TCC). * Alternatives: Modified non-removable walker boots, customized therapeutic footwear, pressure-redistributing accommodative insoles, and forefoot-relief shoes.	Redistributes plantar forces evenly across the entire foot surface, reduces pressure focalization, and ensures mandatory patient compliance.
Wound Care & Infection Control	* Systematic debridement of calluses/slough. * Moist wound environment selection. * Empirical or targeted culture-guided antibiotics. * Urgent surgical revascularization for underlying PAD.	Direct debridement reduces local pressure and removes devitalized tissue; antibiotic regimens disrupt polymorphic biofilm communities that resist standard healing.
Physiotherapy & Exercise Rehabilitation	* Supervised progressive resistance training. * Aerobic and proprioceptive balance training. * Gait re-education (restoring heel-to-toe progression). * Real-time foot offloading pressure visual biofeedback.	Safely induces type II muscle fiber hypertrophy and increases neural recruitment efficiency. Corrects gait parameters to reduce forefoot over-loading, prevents arch collapse, preserves residual motor function, and improves balance stability.

Table 5: Multidisciplinary Treatment Pillars.

Multidisciplinary management

The management of diabetic foot disease is most effective when delivered through a coordinated, integrated multidisciplinary team comprising diabetologists, podiatrists, vascular surgeons, orthotists, physiotherapists, clinical psychologists, and specialist wound-care nurses, built around three fundamental pillars of care [7].

Off-loading

Pressure relief from the ulcer site is widely regarded as the single most impactful therapeutic intervention for neuropathic plantar ulcers and constitutes the cornerstone of conservative management [26]. Total contact casting (TCC) remains the definitive therapeutic gold standard for off-loading neuropathic ulcers, as it effectively redistributes plantar forces across the entire plantar surface. When TCC is contraindicated or poorly tolerated, viable alternatives include prefabricated removable walker boots, customised therapeutic footwear equipped with accommodative orthotic insoles, and specialised half-shoes or forefoot-relief orthoses. Device selection must be assessed and individualised, balancing ulcer topography, patient mobility, localised skin integrity, and specific functional demands and goals. Furthermore, continuous plantar pressure monitoring enables the objective assessment of off-loading efficacy and facilitates precise, real-time tracking of high-pressure zones.

Wound care and infection control

The following essential aspects are included in wound care and infection control such as:

- **Routine Tissue Removal:** Regularly clearing away thickened calluses and dead tissue.
- **Optimal Hydration:** Selecting specific dressings designed to keep the wound bed adequately moist.
- **Tailored Antimicrobial Therapy**: Administering either broad-spectrum or specific, culture-based antibiotics.
- **Emergency Blood Flow Restoration:** Rapidly performing surgical procedures to correct underlying peripheral arterial disease.

By directly clearing away dead tissue, healthcare providers can immediately alleviate localised pressure. Concurrently, targeted antibiotic treatments are administered for breaking down the stubborn, multi-layered bacterial biofilms that typically block the body's natural healing processes.

Exercise, physiotherapy, and rehabilitation

Optimal local wound management requires systematic debridement of hyperkeratotic callus, slough, and necrotic tissue, an intervention that inherently reduces localised plantar pressures and promotes a viable, granulating wound bed. This is supplemented by maintaining a physiologically moist wound environment, selecting dressings judiciously based on exudate volume and wound morphology, and actively managing biofilms to reduce microbial colonisation.

When infection is confirmed or suspected, therapeutic management is stratified by clinical severity. Mild soft-tissue infections are typically amenable to empirical oral antibiotic regimens targeting Gram-positive pathogens. Conversely, moderate-to-severe infections particularly those with systemic manifestations or deep-tissue involvement necessitate parenteral antibiotic therapy guided by definitive wound culture and sensitivity profiles, often in conjunction with urgent surgical drainage or debridement. Concurrently, peripheral revascularisation must be expedited in all patients presenting with significant ischemia that impairs the wound-healing sequence**.**

No currently available pharmacological agent has demonstrated disease-modifying efficacy specifically for motor diabetic neuropathy, and intensive glycaemic control – while fundamental to the overall management of diabetes – has limited capacity to reverse established motor deficits [11]. Structured exercises therefore constitute the primary rehabilitative intervention. Supervised progressive resistance training safely increases lower-limb muscle strength, promotes type II muscle fibre hypertrophy, and enhances neural recruitment efficiency, even in patients with established DSPN and existing foot complications [11]. Earlier concerns that weight-bearing exercise might increase ulcer incidence in neuropathic feet have not been substantiated by clinical evidence, provided appropriate footwear and monitoring are in place.

Aerobic exercise and structured balance training programmes improve gait stability, reduce fall risk, and attenuate the functional consequences of sensorimotor impairment [16]. Combined exercise programmes that integrate aerobic, resistance, and proprioceptive components confer additional metabolic benefits improving glycaemic control, insulin sensitivity, and lipid profiles along with their neurological and biomechanical effects. Physiotherapy-directed gait re-education, targeted at restoring heel-to-toe progression, reducing excessive forefoot loading, and improving ankle dorsiflexion range, directly addresses the mechanical drivers of callus formation and plantar ulceration. Occupational therapy assessment of footwear and assistive device needs, combined with patient education regarding daily foot inspection and self-care, completes the rehabilitative package [7].

Foot offloading pressure feedback is effective in patients with diabetic neuropathy. The system provides visual alerts to teach patients how to change their loading pattern and to prevent ulcerative areas.

Outcomes and prognosis

The clinical prognosis of diabetic foot ulcers varies substantially according to wound characteristics, depending upon the presence of infection and ischaemia, ulcer grade at presentation, and patient-level factors which includes glycaemic control, nutritional status, and comorbidities [27]. Purely neuropathic ulcers in a well-perfused foot generally carry a favourable prognosis if appropriately off-loaded and managed; healing rates in this subgroup are high with adherent, structured care. Neuroischaemic or infected ulcers present more challenge in clinical picture, with slower healing, higher complication rates, and substantial greater risk of major amputation.

Timely multidisciplinary intervention has been demonstrated to reduce major lower-limb amputation rates by 50 to 85% in structured programmes, underscoring the potential of coordinated care systems to transform outcomes in this population [28,29].

Within the Indian healthcare ecosystem, achieving this benchmark is complicated due to socioeconomic and systemic challenges which includes inability to access heterogeneous specialised podiatric and vascular services, prolonged delays in clinical presentation, inability or ignorance to implement appropriate physiotherapy exercise interventions and financial constraints that impede sustained compliance with structured foot care programs. Ultimately, optimal long-term clinical outcomes are correlated with consistent adherence to multidisciplinary follow-up, sustained glycaemic optimisation, and strict compliance with prescribed off-loading and therapeutic footwear regimens.

Conclusion

Diabetic foot disease arises from a complex combination of metabolic dysregulation, vascular insufficiency, neuro-structural deformities, and increased susceptibility to infection. Historically, the clinical significance of motor neuropathy has been overshadowed by the prominence of sensory loss; however, it plays a critical role in driving the architectural deformities and gait instabilities that culminate in plantar ulceration. Its pathological footprint characterised by progressive intrinsic muscle atrophy, digital deformities, abnormal plantar pressure distribution, and compromised dynamic postural stability warrants explicit integration into both diagnostic algorithms and systematic therapeutic planning.

Structured exercise rehabilitation protocol, targeted biomechanical off-loading, and coordinated multidisciplinary intervention constitute the most robust, evidence-based strategies to disrupt the cycle of recurrent ulceration and mitigate the burden of lower extremity amputation. Integrating motor neuropathy screening earlier into routine diabetic foot assessments, coupled with vast expanding role of physiotherapy within specialist wound care teams, represents a critical paradigm shift: moving from reactive local wound management to the proactive preservation of lower-limb biomechanics and patient quality of life and as well as in prevention of foot amputations which is the major concern and challenge of Health sector.

Conflict of Interest

The authors declare no conflicts of interest pertaining to this review.

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