Review Article

From Deficiency to Recovery: Vitamin D's Role in Diabetic Foot Ulcers

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ABSTRACT

Background: Diabetic foot ulcers (DFUs) are among the most serious complications of diabetes mellitus, affecting up to one-third of patients during their lifetime. They are a leading cause of hospitalization, infection, and lower-limb amputation. Emerging evidence suggests that vitamin D deficiency may contribute to impaired wound healing, vascular dysfunction, and immune dysregulation in DFU patients.

Objective: This systematic review evaluates the role of vitamin D in the prevention and management of DFUs, with emphasis on its pathophysiological mechanisms, clinical associations, and therapeutic potential.

Methods: A comprehensive search was conducted across PubMed, Scopus, Springer, ADA, NIH, and other databases without language or year restrictions. Studies examining the correlation between vitamin D levels and DFUs, as well as interventional trials assessing supplementation, were included. Case reports, editorials, and animal studies were excluded. The available study-selection information was organized using a PRISMA-style flow diagram.

Results: Evidence consistently demonstrates that DFU patients exhibit significantly lower serum vitamin D levels compared to controls. Meta-analyses confirm associations between deficiency and peripheral arterial disease, neuropathy, and delayed wound healing. Randomized controlled trials further support its role in improving glycaemic control and wound healing outcomes. Thus, supplementation is a potentially useful adjunct in patients with documented deficiency, but evidence is insufficient to establish vitamin D as a stand-alone DFU treatment.

Conclusion: Vitamin D deficiency is a modifiable risk factor in DFU pathogenesis. Supplementation appears to improve healing, reduce complications, and strengthen immune responses. Future large-scale randomized trials are warranted to establish standardized protocols for vitamin D use in DFU management.

Keywords: *Diabetic foot ulcer (DFU), Vitamin D deficiency, wound healing, peripheral arterial disease, Supplementation*

1. INTRODUCTION

Diabetic foot ulcers (DFUs) affect 15-25% of patients with diabetes during their lifetime and remain a leading cause of hospitalization and lower-limb amputation [1]. Globally, diabetes mellitus (DM) affects around 550 million people, projected to rise to 700 million by 2045 [2]. Vitamin D, a steroid hormone essential for calcium homeostasis, has immunomodulatory and wound-healing properties. Deficiency is linked to cardiovascular disease, hypertension, autoimmune disorders, fractures, and diabetes complications [3, 4]. Its receptors are expressed in endothelial cells, immune cells, macrophages, T lymphocytes and pancreatic beta cells, influencing vascular function, immune

modulation, and insulin secretion [5]. These receptors help vitamin D regulate glucose use, immune function, blood vessel growth, collagen production, and defence against infections. Therefore, low vitamin D levels might lead to slower wound healing, a higher chance of infections, and worsening of diabetic foot infections. With many diabetic patients worldwide having low vitamin D, it's crucial to understand how vitamin D affects DFU prevention and care.

1.1 Pathophysiology of Diabetic Foot Ulcers: DFUs arise from neuropathy, peripheral arterial disease (PAD), and immune dysfunction, all connected to hyperglycaemia. Mechanisms include polyol pathway dysfunction, oxidative stress, non-enzymatic glycosylation, and microvascular ischemia [6]. Structural foot anomalies further increase risk of infection [7-10] (Figure.1).

1.2 Vitamin D deficiency and Neuropathy: Peripheral neuropathy is a key risk factor for developing DFUs. Ongoing high blood sugar levels damage peripheral nerves through oxidative stress, harmful compounds known as advanced glycation end products, and poor blood flow. A lack of vitamin D has been linked to less production of growth factors and more inflammation in nerves. Research shows that diabetic patients with low vitamin D levels are more likely to have sensory neuropathy, slower nerve signal speeds, and a greater risk of foot ulcers. Raising vitamin D levels may help improve nerve function and lessen neuropathic symptoms [11-14]. These findings support an association but do not establish that vitamin D deficiency independently causes neuropathy. Neuropathic pain should be considered separately from neuropathy itself. Some clinical studies have reported improvement in painful diabetic neuropathy after vitamin D correction or supplementation, but pain response is a different outcome from restoration of nerve function or prevention of DFUs. Vitamin D may therefore be considered a potential adjunct when deficiency is present, rather than a replacement for established neuropathy management.

1.3 Vitamin D and Pain Perception: Neuropathic pain can greatly reduce the quality of life for diabetic patients. New research suggests that vitamin D plays a role in how pain is felt by influencing inflammatory substances in the body and how calcium works in neurons. Low vitamin D has been linked to higher pain scores, burning feelings, and tingling sensations. Some clinical studies have shown that vitamin D supplementation can reduce neuropathic pain, highlighting its potential as a helpful addition to other treatments 12, 15].

1.4 Vitamin D and Peripheral Arterial Disease: Peripheral arterial disease is a major factor in the formation of DFUs and slow healing. A lack of vitamin D can lead to poor blood vessel function, thickening of blood vessels, and inflammation, all of which reduce blood flow. Low vitamin D levels are associated with worse ankle-brachial index readings and a higher risk of reduced blood flow to the legs. Proper levels of vitamin D may help improve blood vessel health and increase blood flow in diabetic patients [16-18]. The biological mechanisms proposed include endothelial dysfunction, inflammatory signaling, and effects on vascular homeostasis. However, these observations are subject to confounding by age, diabetes duration, renal disease, obesity, physical activity, and other vascular risk factors. Therefore, the present evidence supports an association between low vitamin D status and PAD rather than establishing vitamin D deficiency as an independent causal driver of PAD.

1.5 Vitamin D and Wound Healing Mechanisms: Wound healing involves three stages: inflammation, proliferation, and remodelling. Vitamin D plays a role in all these stages. During the inflammatory phase, vitamin D helps control the amount of inflammatory substances and thus affects the macrophages. In the proliferative stage, it encourages keratinocyte movement, fibroblast growth, collagen formation, and the creation of the extracellular matrix. Additionally, vitamin D boosts the expression of vascular endothelial growth factor (VEGF), which supports blood vessel growth and oxygen supply to healing tissues. These processes work together to speed up wound closure and tissue regeneration [19-22]. However

the available evidence does not justify attributing human DFU healing to a single VEGF-mediated pathway.

1.6 Vitamin D and Infection: Control Infections can lead to hospitalization and amputations in patients with DFUs. Vitamin D promotes the production of antimicrobial peptides like cathelicidin, which fight against various bacteria, viruses, and fungi. Low vitamin D levels have been associated with higher infection rates, longer hospital stays, and slower healing. Recent research shows that patients who take vitamin D supplements may have fewer infections and better overall health outcomes [23-25].

1.7 Vitamin D and Ulcer Severity: Observational studies have shown a negative relationship between serum 25-hydroxyvitamin D levels and ulcer severity. Patients with worse Wagner grades often have much lower vitamin D levels as compared to those with mild ulcers. Vitamin D deficiency is also linked to larger ulcers, longer healing times, more frequent recurrences, and higher amputation risks. Wagner grading has historically been used to describe DFUs, but contemporary IWGDF guidance recommends the SINBAD system for communication and audit and allows WIfI classification where appropriate, particularly when PAD and limb-threat assessment are important. Wagner grade should therefore not be used as the sole or primary modern measure of DFU severity. These results indicate that vitamin D levels might be a valuable indicator for managing DFUs [26-29].

1.8 Future Perspectives: While there is increasing evidence supporting vitamin D's role in preventing and treating DFUs, there are still significant gaps in knowledge. Cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2) are established forms used for correction of vitamin D deficiency. There is currently no universally accepted DFU-specific vitamin D preparation, dose, duration, or target serum 25(OH)D concentration. In adults with documented deficiency, vitamin D2 or D3 may be used according to established deficiency-treatment protocols, with dose selection and monitoring determined clinically. Importantly, doses used in DFU trials have varied, and the available evidence does not support prescribing a particular high-dose regimen solely because a patient has a DFU. Vitamin D replacement should be considered an adjunct to standard DFU management, not a substitute for off-loading, vascular assessment/revascularization when indicated, glycaemic management, wound care, and infection management Future large-scale randomized controlled trials will be essential to determine the best dosage, treatment length, and target vitamin D levels [30, 31]. So, Research analyzing vitamin D alongside other therapies, like antimicrobial agents, growth factors, stem cell treatments, and advanced wound dressings, may also enhance healing outcomes [32]. Regular vitamin D screening should be part of managing diabetic foot care, as it could be an effective way to lower complications and amputation rates.

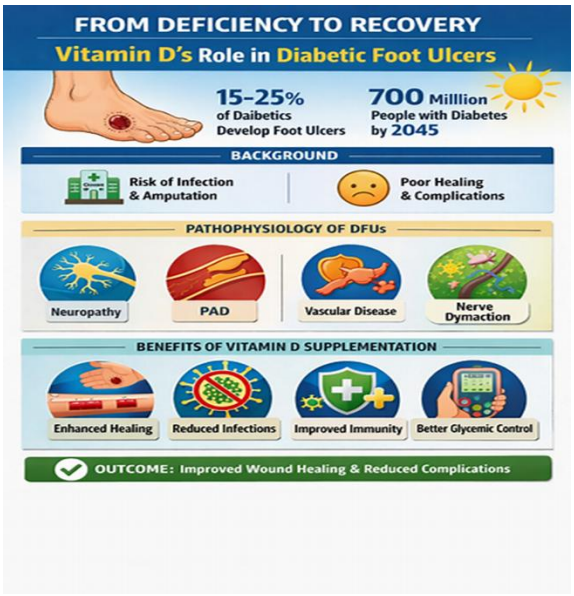


Figure 1. Pathophysiology and wound healing effectiveness of vitamin D in DFU's

2. MATERIALS AND METHODS

Searches were conducted in PubMed, Scopus, Springer, ADA, NIH, and Nature databases using combinations of "Diabetic Foot Ulcer," "Vitamin D," and "Prophylactic Management" (Figure.2).

2.1 Inclusion criteria:

I. Peer-reviewed studies from high-index journals
II. Studies on prophylactic vitamin D in DFUs
III. Correlation between vitamin D levels and DFUs

2.2 Exclusion criteria:

I. Case reports, editorials
II. Animal studies

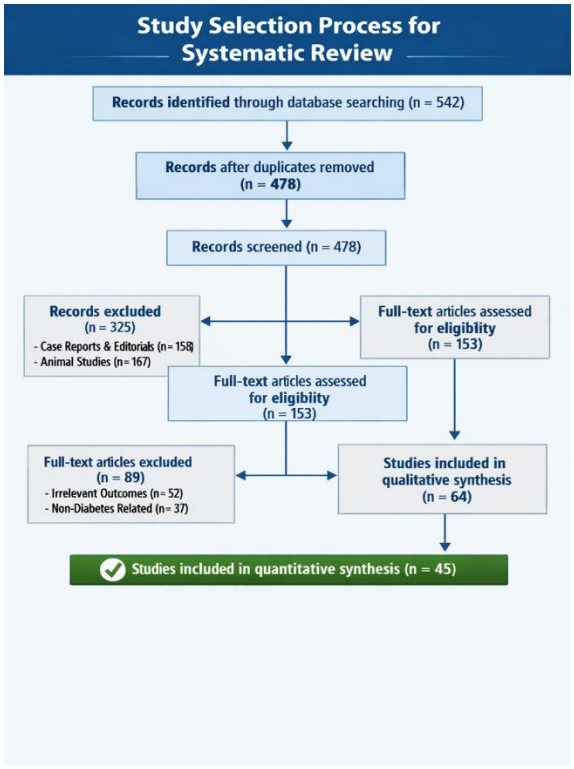


Figure 2: Overview and baseline characteristics of the included studies for systematic reviews (PRISMA) flow diagram.

3. REVIEW OF LITERATURE

Vitamin D and PAD: Vitamin D deficiency has become an important and possibly changeable risk factor in the development and progression of diabetic foot ulcers (DFUs). Besides its known role in calcium balance, vitamin D has various effects on immune function, blood vessel growth, glucose metabolism, and tissue repair. These processes are essential for healing wounds in patients with diabetes. Several studies have found a strong link between vitamin D deficiency and peripheral arterial disease (PAD), which is a major cause of DFU development. Iannuzzo et al. showed that patients with PAD had much lower serum vitamin D levels compared to healthy individuals. This suggests that deficiency may lead to problems with blood flow and blood vessel function [33].

Racial Differences: Reis et al. showed that racial variation in vitamin D status explained nearly one-third of excess Peripheral arterial disease risk in Black adults, pointing out the role of vitamin D as a factor in vascular health [34].

Neuropathy: Diabetic peripheral neuropathy is another significant risk factor for DFUs as Qu et al. found Asian type 2 diabetics with deficiency were 1.22 times more likely to develop neuropathy. He also showed that vitamin D-deficient patients with type 2 diabetes had a higher risk of developing neuropathy [35]. More recently, Tang et al. found that low vitamin D levels were linked to worse neuropathic symptoms and a greater likelihood of diabetic foot issues [36].

Wound Healing Mechanisms: The role of vitamin D in wound healing has been studied extensively. Vitamin D receptors are present in keratinocytes, fibroblasts, macrophages, and endothelial cells, indicating its direct role in tissue repair. Vitamin D boosts the expression of antimicrobial peptides, especially cathelicidin, which improves innate immunity and protects wounds from infections. Recent studies also revealed that vitamin D encourages keratinocyte growth, fibroblast movement, collagen formation, and blood vessel creation through VEGF-mediated pathways. Clinical studies have reinforced the connection between vitamin D levels and ulcer severity thus vitamin D enhances healing by upregulating antimicrobial peptides, promoting keratinocyte migration, fibroblast collagen synthesis, and VEGF-mediated angiogenesis [37].

Clinical Correlations: Clinical studies have reinforced the connection between vitamin D levels and ulcer severity. Liu *et al.*, (2024) discovered a notable negative association between serum 25-hydroxyvitamin D concentrations and the severity of DFUs [38]. Similarly, Hu et al. (2023) conducted a systematic review and meta-analysis, concluding that vitamin D deficiency significantly raised the risk of DFU development and progression [39]

Supplementation Benefits: The potential benefits of vitamin D supplementation have attracted growing attention. Gao et al. (2025) reported reduced infection rates and improved healing with vitamin D supplementation [40].

Adjunct Therapy: Al-Rawaf & Gabr (2021) showed vitamin D combined with ginger improved glycaemic control and wound healing at the molecular level [41]. Infection continues to

be a major problem for patients with DFUs. Recent findings suggest that vitamin D strengthens the body's defence by boosting the production of antimicrobial peptides and regulating macrophage activity.

Preventive Strategies: Frykberg *et al.,* (2006) emphasized that while not all DFU complications can be prevented, incidence can be reduced through appropriate management programs [42].

Recent Meta-Analyses: Liu *et al.,* (2024) umbrella review confirmed consistent associations between deficiency and DFU severity [38]. Reveendran *et al.,* (2025) found that DFU patients had significantly lower serum vitamin D levels compared to diabetic individuals without foot ulcers. Lifestyle factors also affect vitamin D status and may indirectly influence DFU risk [43]. Lin *et al.,* (2022) illustrated that vitamin D deficiency was significantly linked to delay wound healing and worse clinical outcomes in DFU patients linked deficiency to poor healing outcomes [44]. An umbrella review by Liu *et al.,* (2024) validated consistent connections between low vitamin D levels, ulcer severity, neuropathy, and poor healing across various systematic reviews.

Lifestyle Factors: Benson *et al.,* (2025) reported that patients with lower vitamin D levels had more frequent diabetic foot infections and longer healing times. Recent meta-analyses have further confirmed these findings [45]. Bashir *et al.,* reported much lower vitamin D levels among inactive females compared to those who worked outdoors in sunlight. This highlights the importance of outdoor activity and adequate sun exposure in maintaining healthy vitamin D level [46].

Overall, current evidence indicates that vitamin D deficiency plays a role in the development of DFUs through its effects on blood vessel function, neuropathy, immune response, and tissue repair. While existing literature supports the positive effects of vitamin D supplementation, more large-scale randomized controlled trials are necessary to determine the best dosages and assess its long-term effects on ulcer recurrence, amputation rates, and quality of life (Table.1).

Table1: Showing the comparison of DFU's and Vitamin D in different studies

Study (Author, Year)	Study Focus	Population	Key Findings	Clinical Relevance
Iannuzzo *et al.,* (2019)	Vitamin D & PAD	PAD patients	Lower vitamin D linked to higher disease risk	Vascular compromise increases DFU risk
Reis *et al.,* (2008)	Racial differences	Black adults	~1/3 PAD risk explained by vitamin D variation	Demographic variability in DFU risk
Qu *et al.,* (2017)	Neuropathy risk	Asian T2DM patients	1.22× higher neuropathy risk with deficiency	Neuropathy predisposes to DFU
Liu *et al,.* (2024)	Clinical correlation	DFU patients	Inverse relation between vitamin D and severity	Lower vitamin D → severe DFU
Gao *et al.,* (2025)	Supplementation	DFU patients	Reduced infection, improved healing	Supports vitamin D therapy
Al-Razzaf & Gabr (2021)	Adjunct therapy	DFU patients	Vit D + ginger improved outcomes	Combination therapy benefit
Lin *et al.,* (2024)	Meta-analysis	Multiple studies	Strong association with DFU severity	High-level evidence
Raveendran *et al.,* (2025)	Observational	DFU patients	Lower vitamin D levels	Confirms deficiency link
Lin *et al.,* (2022)	Healing outcomes	DFU patients	Poor healing with deficiency	Prognostic role
Bashir *et al.,* (2019)	Lifestyle factors	Sedentary vs laborers	Lower Vit D in sedentary females	Lifestyle affects DFU risk
Frykberg *et al.,* (2006)	Prevention	General population	Proper management reduces incidence	Prevention strategies important

4. CONCLUSION

Vitamin D deficiency is common in diabetes and contributes to vascular disease, neuropathy, and impaired wound healing. Evidence supports its role in DFU prevention and recovery, with supplementation improving immune response, infection control, and tissue repair. Future randomized controlled trials are needed to establish standardized protocols for vitamin D use in DFU management.

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CONFLICT OF INTEREST:
The authors declare no conflicts of interest.

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Not applicable

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