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Original Article

Prevalence and Risk Factors of Diabetic Foot Syndrome in Patients with Poorly Controlled Type 2 Diabetes Mellitus: A Cross-Sectional Analytical Study

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ABSTRACT

Background: Diabetic foot syndrome is a disabling and potentially preventable complication of type 2 diabetes mellitus, particularly among patients with poor glycemic control, where neuropathic, vascular, metabolic, and infectious factors contribute to ulceration and limb-threatening complications. Objective: To determine the prevalence of diabetic foot syndrome and identify associated risk factors among patients with poorly controlled type 2 diabetes mellitus. Methods: A hospital-based cross-sectional analytical study was conducted among 320 adults with poorly controlled type 2 diabetes mellitus. Demographic, clinical, anthropometric, and biochemical data were collected using a structured proforma. All participants underwent diabetic foot assessment, including evaluation for peripheral neuropathy, peripheral arterial disease, deformity, ulceration, and infection. Associations were assessed using chi-square tests and independent-samples t-tests, while independent predictors were identified through multivariable logistic regression. Results: The prevalence of diabetic foot syndrome was 28.8% (92/320). Peripheral neuropathy was present in 41.3% and peripheral arterial disease in 29.1% of participants. Patients with DFS had higher age, longer diabetes duration, higher HbA1c, and greater BMI than non-DFS patients. Independent predictors of DFS were peripheral arterial disease (AOR: 6.44, 95% CI: 3.39–12.2), peripheral neuropathy (AOR: 5.82, 95% CI: 3.21–10.5), HbA1c ≥8% (AOR: 3.76, 95% CI: 2.01–7.02), diabetes duration >10 years (AOR: 2.89, 95% CI: 1.54–5.42), and smoking (AOR: 1.92, 95% CI: 1.05–3.54). Conclusion: Diabetic foot syndrome is highly prevalent among poorly controlled T2DM patients and is strongly associated with neuropathy, PAD, poor glycemic control, prolonged diabetes duration, and smoking. Early screening, metabolic optimization, and multidisciplinary diabetic foot care are essential to prevent ulceration, infection, and amputation. Keywords: Diabetic foot syndrome; type 2 diabetes mellitus; peripheral neuropathy; peripheral arterial disease; HbA1c; risk factors.

INTRODUCTION

Diabetes mellitus (DM) is one of the fastest-growing metabolic diseases globally and has become a major public health crisis of the 21st century. Global estimates suggest that the prevalence of diabetes among adults continues to rise in response to urbanization, physical inactivity, dietary changes, and increasing obesity. Type 2 diabetes mellitus (T2DM) accounts for most diabetes cases and is closely linked with insulin resistance, progressive β-cell dysfunction, and chronic metabolic disturbance (1). Long-standing hyperglycemia in T2DM contributes to multisystem injury through both microvascular and

macrovascular complications, resulting in increased morbidity, mortality, disability, and health-care costs.

Diabetic foot syndrome (DFS) is among the most disabling and preventable complications of T2DM. It represents a complex clinical condition involving peripheral neuropathy, peripheral arterial disease (PAD), foot deformity, infection, ulceration, and tissue destruction. Rather than being a single disease entity, DFS reflects the interaction of metabolic, vascular, neurological, mechanical, and infectious processes affecting the lower limb. The International Working Group on the Diabetic Foot defines diabetic foot disease as infection, ulceration, or destruction of foot tissues associated with neurological abnormalities and varying degrees of peripheral vascular disease (2). This multifactorial pathogenesis makes DFS difficult to manage once established and emphasizes the importance of early identification of high-risk patients.

The burden of diabetic foot complications is substantial. Previous evidence suggests that approximately 19–34% of people with diabetes may develop a foot ulcer during their lifetime, while annual incidence is higher among patients with poor glycemic control, longer diabetes duration, neuropathy, and vascular disease (3). Diabetic foot ulcers are also among the leading causes of non-traumatic lower-limb amputation worldwide, with infection, ischemia, and delayed presentation contributing to poor outcomes (4). Beyond clinical consequences, DFS imposes considerable financial and psychosocial burdens due to prolonged hospital admissions, surgical interventions, antibiotic therapy, rehabilitation, immobility, dependency, and reduced quality of life.

The pathophysiology of DFS is driven largely by chronic hyperglycemia and its downstream effects. Persistent elevation of blood glucose promotes oxidative stress, inflammation, endothelial dysfunction, impaired nitric oxide availability, and formation of advanced glycation end-products, all of which contribute to vascular and neural injury (5). Peripheral neuropathy is a central component of DFS and includes sensory, motor, and autonomic dysfunction. Sensory neuropathy causes loss of protective sensation and allows repeated unnoticed trauma; motor neuropathy produces intrinsic foot-muscle imbalance and deformity; and autonomic neuropathy reduces sweating and skin integrity, increasing fissuring and susceptibility to infection (6).

PAD further increases the risk and severity of diabetic foot complications by reducing lower-limb perfusion, oxygen delivery, and wound-healing capacity. When neuropathy and ischemia coexist, the risk of ulceration, infection, gangrene, and amputation increases substantially. Hyperglycemia also impairs immune function, including neutrophil chemotaxis, phagocytosis, and bacterial clearance, thereby facilitating infection and progression of tissue damage (7). The combined effects of neuropathy, ischemia, and infection form the major pathological triad underlying DFS severity.

Poor glycemic control remains one of the most important modifiable risk factors for DFS. Glycated hemoglobin (HbA1c) reflects chronic glycemic exposure and is strongly associated with microvascular and macrovascular complications in T2DM (8). Patients with elevated HbA1c are more likely to develop neuropathy, vascular disease, impaired wound healing, and recurrent ulceration. In addition to hyperglycemia, older age, longer diabetes duration, smoking, obesity, hypertension, dyslipidemia, nephropathy, and retinopathy have been associated with increased diabetic foot risk (9,10). Smoking is particularly important because it accelerates endothelial dysfunction, vasoconstriction, atherosclerosis, and tissue hypoxia.

Despite extensive global evidence, region-specific data on the prevalence and predictors of DFS among patients with poorly controlled T2DM remain limited, particularly in South Asian clinical settings where diabetes burden is increasing rapidly. Local disease patterns may be influenced by delayed health-seeking behavior, limited foot-screening services, socioeconomic barriers, footwear practices, walking barefoot, and inconsistent access to multidisciplinary diabetic care. Therefore, local epidemiological

evidence is required to quantify disease burden, identify high-risk subgroups, and support context-specific prevention strategies.

Preventive care remains the most effective approach to reducing diabetic foot complications. Routine foot examination, patient education, appropriate footwear, smoking cessation, strict metabolic control, and early vascular and neurological assessment are essential components of prevention. Simple clinical tools such as the 10 g monofilament test, vibration assessment, pulse examination, and ankle-brachial index can identify high-risk patients before ulceration occurs. Multidisciplinary care involving endocrinologists, podiatrists, vascular specialists, wound-care teams, and rehabilitation professionals has been shown to reduce ulcer recurrence and amputation risk (11,12).

Therefore, the present study was conducted to determine the prevalence of diabetic foot syndrome and identify associated risk factors among patients with poorly controlled type 2 diabetes mellitus. The study specifically aimed to assess the association of DFS with peripheral neuropathy, PAD, glycemic control, diabetes duration, smoking, and other cardiometabolic factors in order to support early screening, risk stratification, and preventive diabetic foot care in this high-risk population (13).

MATERIALS AND METHODS

This hospital-based cross-sectional analytical study was conducted to determine the prevalence of diabetic foot syndrome and identify its associated risk factors among patients with poorly controlled type 2 diabetes mellitus. A cross-sectional design was selected because it allows simultaneous assessment of disease burden and associated clinical exposures within a defined population, making it suitable for estimating prevalence and examining risk-factor associations in routine clinical settings.

The study was conducted in the outpatient diabetes and endocrinology clinics of a tertiary care hospital that receives patients from both urban and rural communities. Data were collected over a defined study period of 6–12 months to ensure adequate recruitment and representation of patients attending diabetic outpatient services. The study population comprised adult patients with established T2DM and poor glycemic control who presented for outpatient diabetic care during the study period.

Patients were eligible for inclusion if they had diagnosed T2DM, were aged 30 years or older, had poor glycemic control defined as HbA1c ≥7%, and were able to provide voluntary informed consent. For risk stratification within the poorly controlled cohort, HbA1c ≥8% was additionally analyzed as a higher-risk glycemic category. Patients with type 1 diabetes mellitus, gestational diabetes, traumatic or non-diabetic foot injury, active malignancy, serious systemic illness such as end-stage renal disease on dialysis, or lower-limb amputation due to non-diabetic causes were excluded.

A non-probability consecutive sampling technique was used. All eligible patients attending the diabetic clinic during the study period were approached consecutively until the required sample size was achieved. This approach improved feasibility and allowed recruitment of a real-world clinical population, although selection bias related to hospital-based sampling was minimized as far as possible through consecutive enrolment of eligible participants.

The sample size was calculated using the Cochran formula for prevalence studies, based on an expected prevalence of diabetic foot complications from previous literature, a 95% confidence level, and a 5% margin of error. A 10% allowance for non-response was added to ensure adequate statistical power. The final sample size was considered sufficient for prevalence estimation and multivariable logistic regression analysis of major risk factors.

Data were collected using a structured proforma developed after review of relevant literature and diabetic foot assessment guidance. The proforma recorded socio-demographic information including age, gender, occupation, educational status, and smoking history; clinical history including duration of diabetes, medication use, hypertension, dyslipidemia, previous foot ulcer or infection, and physical

activity level; and anthropometric and clinical measurements including height, weight, body mass index, and blood pressure. HbA1c was measured using standard laboratory procedures and was used as the principal biochemical marker of long-term glycemic control.

All participants underwent a detailed diabetic foot assessment performed by trained clinical personnel using standardized examination procedures. Peripheral neuropathy was assessed using the 10 g Semmes-Weinstein monofilament test, 128 Hz tuning fork vibration assessment, and pinprick or temperature sensation where applicable. Peripheral neuropathy was diagnosed by loss of protective sensation on clinical assessment. Peripheral arterial disease was assessed through palpation of dorsalis pedis and posterior tibial pulses, capillary refill time, and ankle-brachial index where available; an ankle-brachial index below 0.90 was considered suggestive of PAD. Foot deformities including claw toes, hammer toes, Charcot changes, and prominent metatarsal heads were documented through clinical inspection.

The primary outcome variable was the presence or absence of diabetic foot syndrome. DFS was operationally defined as the presence of neuropathic ulceration, ischemic ulceration, infected foot lesion, or Charcot neuroarthropathy with structural damage. Independent variables included age, gender, diabetes duration, HbA1c level, smoking status, body mass index, hypertension, dyslipidemia, peripheral neuropathy, peripheral arterial disease, and microvascular complications including retinopathy and nephropathy.

To improve data quality and reduce measurement error, data collectors were trained before data collection, the proforma was pre-tested on a small proportion of the target population, and clinical examination procedures were standardized. Data were checked for completeness before entry, and double entry was used to reduce data-entry errors. Potential confounding was addressed analytically by including clinically relevant variables and variables with $p < 0.20$ in univariate analysis in the multivariable logistic regression model.

Data were analyzed using SPSS version 27. Continuous variables were summarized as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The chi-square test was used to assess associations between categorical variables, and the independent-samples t-test or Mann–Whitney U test was applied for continuous variables depending on distribution. Multivariable logistic regression was performed to identify independent predictors of DFS, and results were reported as adjusted odds ratios with 95% confidence intervals. Multicollinearity among predictor variables was assessed before final model interpretation, and statistical significance was set at $p < 0.05$.

The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the institutional review board of the relevant institution before data collection. Written informed consent was obtained from all participants. Confidentiality and anonymity were maintained throughout the study, and participants identified with diabetic foot complications were referred for appropriate clinical management.

RESULTS

A total of 320 patients with poorly controlled type 2 diabetes mellitus were included in the analysis. The mean age of the participants was 56.8 ± 11.4 years, and the sample included 188 males (58.8%) and 132 females (41.2%). The mean duration of diabetes was 11.6 ± 6.2 years, while the mean HbA1c was 9.2 ± 1.4%, confirming poor glycemic control in the study population. Overall, 231 participants (72.2%) had HbA1c ≥8%. Hypertension was present in 198 patients (61.9%), dyslipidemia in 174 (54.4%), obesity in 146 (45.6%), and smoking history in 102 (31.9%) (Table 1).

Table 1. Baseline Characteristics of Participants with Poorly Controlled Type 2 Diabetes Mellitus

Variable	Overall Sample (n = 320)
Age, years	56.8 ± 11.4
Male sex	188 (58.8%)

Variable	Overall Sample (n = 320)
Female sex	132 (41.2%)
Duration of diabetes, years	11.6 ± 6.2
HbA1c, %	9.2 ± 1.4
HbA1c ≥8%	231 (72.2%)
Hypertension	198 (61.9%)
Dyslipidemia	174 (54.4%)
Obesity	146 (45.6%)
Smoking history	102 (31.9%)

The overall prevalence of diabetic foot syndrome was 28.8%, affecting 92 of 320 patients. Among patients with DFS, neuropathic ulcers were the most frequent presentation, accounting for 46.7% of cases, followed by neuro-ischemic ulcers in 29.4% and ischemic ulcers in 23.9%. Active infected ulcers were present in 18.1% of DFS cases, while gangrenous changes were observed in 6.5%, indicating that a clinically important proportion of patients presented with advanced diabetic foot complications (Table 2).

Table 2. Prevalence and Clinical Pattern of Diabetic Foot Syndrome

Variable	Frequency / Percentage
Total participants	320
Diabetic foot syndrome	92 (28.8%)
No diabetic foot syndrome	228 (71.2%)
Neuropathic ulcers among DFS cases	46.7%
Ischemic ulcers among DFS cases	23.9%
Neuro-ischemic ulcers among DFS cases	29.4%
Active infected ulcers among DFS cases	18.1%
Gangrenous changes among DFS cases	6.5%

Peripheral neuropathy was identified in 132 participants (41.3%) and was markedly more frequent among patients with DFS than among those without DFS (78.3% vs 26.4%, $p < 0.001$). Peripheral arterial disease was present in 93 participants (29.1%) and was also significantly more common in the DFS group (65.2% vs 14.8%, $p < 0.001$). Foot deformities were observed in 78 participants (24.4%), with claw toes and Charcot-related changes being the most frequently documented structural abnormalities (Table 3).

Table 3. Clinical Risk Profile of Study Participants

Clinical Variable	Overall Frequency	DFS Group	Non-DFS Group	p-value
Peripheral neuropathy	132 (41.3%)	78.3%	26.4%	<0.001
Peripheral arterial disease	93 (29.1%)	65.2%	14.8%	<0.001
Foot deformities	78 (24.4%)	Not specified	Not specified	Not reported

Patients with DFS were older, had longer diabetes duration, and had poorer glycemic control than patients without DFS. The mean age was 61.2 ± 10.8 years in the DFS group compared with 54.9 ± 11.1 years in the non-DFS group ($p < 0.001$). Diabetes duration was also substantially longer among DFS patients (15.3 ± 6.1 vs 9.9 ± 5.4 years, $p < 0.001$), while HbA1c was higher in the DFS group (10.1 ± 1.3% vs 8.8 ± 1.1%, $p < 0.001$). Smoking, hypertension, and higher BMI were also significantly associated with DFS (Table 4).

Table 4. Comparison Between DFS and Non-DFS Groups

Variable	DFS (n = 92)	Non-DFS (n = 228)	Mean Difference / Difference	p-value
Age, years	61.2 ± 10.8	54.9 ± 11.1	+6.3 years	<0.001
Diabetes duration, years	15.3 ± 6.1	9.9 ± 5.4	+5.4 years	<0.001
HbA1c, %	10.1 ± 1.3	8.8 ± 1.1	+1.3%	<0.001
Smoking	45.7%	26.3%	+19.4 percentage points	0.001
Hypertension	73.9%	57.0%	+16.9 percentage points	0.005
BMI, kg/m²	30.4 ± 5.2	28.1 ± 4.7	+2.3 kg/m²	0.003

Univariate analysis showed that diabetes duration greater than 10 years, HbA1c ≥8%, peripheral neuropathy, PAD, and smoking were significantly associated with DFS. PAD showed the strongest crude association with DFS (OR: 7.54, 95% CI: 4.12–13.8, $p < 0.001$), followed by peripheral neuropathy (OR:

6.91, 95% CI: 4.01–11.9, p < 0.001) and HbA1c ≥8% (OR: 4.18, 95% CI: 2.31–7.56, p < 0.001). Diabetes duration greater than 10 years and smoking were also significant risk factors (Table 5).

Table 5. Univariate Analysis of Risk Factors Associated with Diabetic Foot Syndrome

Risk Factor	Crude OR	95% CI	p-value
Diabetes duration >10 years	3.42	2.01–5.84	<0.001
HbA1c ≥8%	4.18	2.31–7.56	<0.001
Peripheral neuropathy	6.91	4.01–11.9	<0.001
Peripheral arterial disease	7.54	4.12–13.8	<0.001
Smoking	2.14	1.28–3.59	0.004

On multivariable logistic regression analysis, peripheral arterial disease remained the strongest independent predictor of DFS (AOR: 6.44, 95% CI: 3.39–12.2, p < 0.001), followed by peripheral neuropathy (AOR: 5.82, 95% CI: 3.21–10.5, p < 0.001). Poor glycemic control, defined as HbA1c ≥8%, independently increased the odds of DFS by nearly four times (AOR: 3.76, 95% CI: 2.01–7.02, p < 0.001). Diabetes duration greater than 10 years was also independently associated with DFS (AOR: 2.89, 95% CI: 1.54–5.42, p = 0.001), while smoking remained a weaker but statistically significant predictor (AOR: 1.92, 95% CI: 1.05–3.54, p = 0.032) (Table 6).

Table 6. Multivariable Logistic Regression Analysis of Independent Predictors of Diabetic Foot Syndrome

Predictor	Adjusted OR	95% CI	p-value
Peripheral neuropathy	5.82	3.21–10.5	<0.001
Peripheral arterial disease	6.44	3.39–12.2	<0.001
HbA1c ≥8%	3.76	2.01–7.02	<0.001
Diabetes duration >10 years	2.89	1.54–5.42	0.001
Smoking	1.92	1.05–3.54	0.032

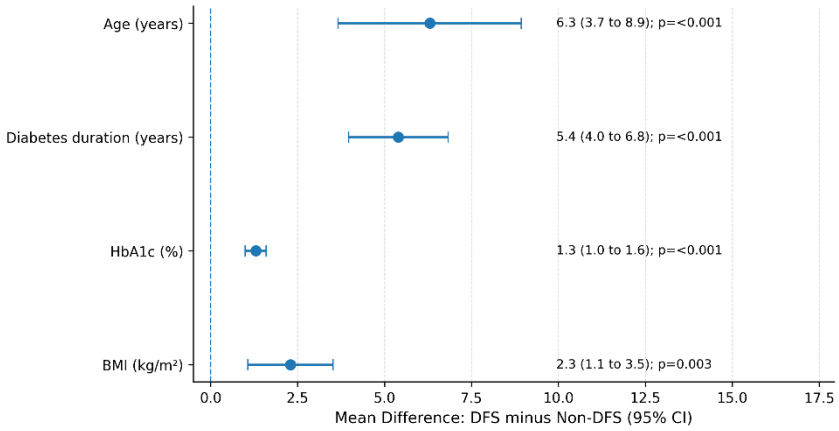

Figure 1 Clinical and Metabolic Differences Associated with Diabetic Foot Syndrome

Among patients with DFS, mild neuropathy was observed in 34.8%, active ulcers in 39.1%, infected ulcers in 18.1%, and gangrene in 8.0%. These findings indicate that although a considerable proportion of patients had early or moderate neuropathic involvement, more than one-fourth had infected ulcers or gangrenous changes, reflecting clinically advanced disease. Overall, peripheral neuropathy and PAD emerged as the two most influential independent predictors, supporting the neuro-ischemic pathway as a central mechanism in the development of diabetic foot syndrome in patients with poorly controlled T2DM (Table 7).

Table 7. Severity Profile Among Patients with Diabetic Foot Syndrome

Severity Indicator Among DFS Cases	Percentage
Mild neuropathy	34.8%
Active ulcers	39.1%
Infected ulcers	18.1%
Gangrene	8.0%

Figure 1, Patients with diabetic foot syndrome showed consistently higher clinical and metabolic risk profiles than non-DFS patients, with greater mean age by 6.3 years, diabetes duration by 5.4 years, HbA1c by 1.3%, and BMI by 2.3 kg/m²; all confidence intervals remained above zero, indicating statistically significant differences, with the strongest gradients observed for age and diabetes duration.

DISCUSSION

This study demonstrates a high prevalence of diabetic foot syndrome among patients with poorly controlled type 2 diabetes mellitus, with DFS identified in 28.8% of the study population. This finding is clinically important because the included participants already represented a high-risk group with poor glycemic control, long diabetes duration, and a high burden of cardiometabolic comorbidities. The observed prevalence falls within the broad range reported internationally for diabetic foot complications, where lifetime risk of foot ulceration among people with diabetes has been estimated at approximately 19–34% (14). The findings therefore support the view that patients with persistent hyperglycemia represent a vulnerable subgroup requiring systematic foot screening and early preventive care.

Peripheral neuropathy emerged as one of the strongest independent predictors of DFS. Patients with neuropathy had markedly higher odds of DFS even after adjustment for other clinical factors, confirming the central role of sensory, motor, and autonomic nerve dysfunction in diabetic foot pathogenesis. Loss of protective sensation allows repetitive unnoticed trauma, while motor neuropathy contributes to deformity and abnormal plantar pressure distribution. Autonomic dysfunction further compromises skin integrity through dryness and fissuring, creating an entry point for infection (15,16). These mechanisms explain why neuropathy was substantially more frequent among DFS patients than non-DFS patients in the present study.

Peripheral arterial disease was the strongest independent predictor in the multivariable model, highlighting the vascular component of diabetic foot disease. PAD reduces tissue perfusion, oxygen delivery, and wound-healing capacity, thereby increasing the risk of ulcer persistence, infection, gangrene, and eventual amputation. The coexistence of PAD and neuropathy produces a neuro-ischemic pattern that is particularly dangerous because patients may not perceive injury while simultaneously having impaired tissue repair capacity (17,18). These findings reinforce the need for routine vascular assessment, including pulse examination and ankle-brachial index testing where available, particularly in patients with long-standing diabetes and poor glycemic control.

Poor glycemic control was also independently associated with DFS. Participants with HbA1c ≥8% had significantly higher adjusted odds of DFS, supporting the role of chronic hyperglycemia in accelerating microvascular injury, endothelial dysfunction, oxidative stress, impaired immune response, and delayed wound healing. This finding is consistent with previous evidence linking higher HbA1c levels with diabetic complications and ulcer risk (19,20). In clinical practice, this suggests that diabetic foot prevention should not be limited to local foot care alone but must include strict metabolic control and regular monitoring of glycemic status.

Longer diabetes duration was another significant predictor of DFS. Patients with diabetes duration greater than 10 years had increased odds of DFS, reflecting cumulative exposure to hyperglycemia and progressive vascular and neurological injury over time. This relationship is biologically plausible because chronic diabetes produces progressive nerve damage, endothelial dysfunction, arterial stiffness, and tissue vulnerability. The finding supports the need to begin preventive foot assessment early in the disease course rather than waiting until patients develop symptoms or visible ulceration.

Smoking remained a statistically significant predictor of DFS, although its effect size was lower than that of neuropathy, PAD, and glycemic control. Smoking promotes vasoconstriction, endothelial dysfunction, atherosclerosis, tissue hypoxia, and impaired immune response, all of which can worsen diabetic foot risk and delay wound healing (21). Because smoking is modifiable, its association with DFS carries direct

public health relevance. Smoking cessation counseling should therefore be integrated into diabetic foot prevention strategies, especially for patients with vascular compromise.

The study also found that DFS patients had higher mean age, longer diabetes duration, higher HbA1c, and greater BMI compared with non-DFS patients. These differences suggest that DFS develops through the combined effect of metabolic, vascular, neurological, and mechanical risk factors. Obesity may contribute through increased plantar loading, insulin resistance, systemic inflammation, and reduced mobility. Similarly, hypertension and dyslipidemia may contribute to vascular injury and PAD, further increasing susceptibility to ulceration and delayed healing (22).

A concerning finding was the presence of infected ulcers and gangrenous changes among DFS patients. These advanced presentations suggest possible delays in diagnosis, inadequate routine screening, limited patient awareness, or late referral to specialized care. In low- and middle-income settings, diabetic foot complications often progress silently until infection or gangrene develops, increasing the risk of hospitalization and amputation (23,24). The present findings therefore support implementation of structured diabetic foot screening at outpatient diabetic clinics, including monofilament testing, vascular assessment, footwear advice, self-care education, and timely referral of high-risk patients.

This study has several strengths. It focused specifically on poorly controlled T2DM patients, a clinically important high-risk population. It assessed both neurological and vascular components of DFS and used multivariable logistic regression to identify independent predictors. However, the study also has limitations. Its cross-sectional design prevents causal inference, and the temporal relationship between risk factors and DFS cannot be established. The hospital-based single-center setting may limit generalizability to community populations. ABI assessment was performed where available, which may introduce measurement variability. Residual confounding may also remain despite adjustment for major clinical predictors. Future multicenter prospective studies are recommended to validate these findings and clarify causal pathways (25,26).

Overall, the findings confirm that diabetic foot syndrome in poorly controlled T2DM is strongly associated with neuropathy, peripheral arterial disease, poor glycemic control, longer disease duration, and smoking. These results emphasize that DFS is largely preventable through early detection, structured risk stratification, metabolic optimization, vascular assessment, patient education, and multidisciplinary diabetic foot care.

CONCLUSION

Diabetic foot syndrome was highly prevalent among patients with poorly controlled type 2 diabetes mellitus, affecting nearly one-third of the study population. Peripheral arterial disease and peripheral neuropathy were the strongest independent predictors, while HbA1c ≥8%, diabetes duration greater than 10 years, and smoking also significantly increased the likelihood of DFS. These findings highlight the multifactorial nature of diabetic foot complications and support the need for routine foot screening, early neurological and vascular assessment, strict glycemic control, smoking cessation, patient education, and multidisciplinary diabetic foot care to reduce ulceration, infection, gangrene, amputation risk, and long-term disability.

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