DOI: 10.7860/JCDR/2026/80723.23595

Original Article

Internal Medicine Section

Prevalence and Predictors of Peripheral Arterial Disease in Type 2 Diabetes Mellitus Patients at a Tertiary Care Centre in West Bengal, India: A Cross-sectional Study

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ABSTRACT

Introduction: Peripheral Arterial Disease (PAD) is characterised by the obstruction of arteries in the extremities. PAD in diabetes is often asymptomatic and underdiagnosed, posing a significant risk of morbidity and mortality due to associated cardiovascular events.

Aim: To assess the prevalence and predictors of PAD in patients with Type 2 Diabetes Mellitus (T2DM).

Materials and Methods: It was a single-centre, cross-sectional, hospital-based study conducted in the Department of General Medicine, Midnapore Medical College and Hospital, West Bengal, India, over a period of 12 months (January 2019 -December 2019. A total of 100 patients with T2DM aged 18-65 years were included. Data collection included demographic details, clinical history, anthropometric measurements {Body Mass Index (BMI), waist circumference, waist-hip ratio}, blood pressure and relevant laboratory investigations. Ankle-Brachial Index (ABI) was measured for PAD screening and duplex ultrasound was used to confirm diagnosis. Factors assessed as potential predictors of PAD included age, duration of diabetes, glycaemic status, blood pressure, lipid profile, renal function, Coronary Artery Disease (CAD) and microvascular complications. Chi-square test for categorical variables and ANOVA for multiple group comparisons. Regression analysis was performed to identify independent predictors for PAD. A p-value <0.05 was considered statistically significant.

Results: Of the 100 participants, 54% were male and 46% female, with a mean age of 48.3±8.5 years. The mean duration of diabetes was 5.7±3.2 years. The prevalence of PAD, defined as ABI ≤ 0.90, was 19% (n=19). Compared to non PAD patients, those with PAD had significantly higher mean Fasting Plasma Glucose (FPG) (155.4±18.6 vs. 113.4±18.9 mg/dL, p<0.001), Postprandial Plasma Glucose (PPPG) (247.1±47.6 vs. 152.1±44.3 mg/dL, p<0.001), duration of diabetes (9.4±3.8 vs. 4.8±2.4 years, p<0.001), Systolic Blood Pressure (SBP) (139.6±17.0 vs. 122.6±15.4 mmHg, p<0.001), Total Cholesterol (TC) (191.4±50.8 vs. 155.7±30.4 mg/dL, p<0.001), triglycerides (161.0±93.0 vs. 107.7±40.3 mg/dL, p<0.001) and LDL cholesterol (115.5±50.1 vs. 90.0±26.3 mg/dL, p=0.002). Conversely, estimated Glomerular Filtration Rate (eGFR) was significantly lower in PAD patients (58.7±27.7 vs. 103.2±36.4 mL/min/1.73 m², p<0.001).

Conclusion: PAD is a common but underdiagnosed complication among Indian patients with T2DM. Emphasis on early screening using simple tools like ABI, along with timely intervention for modifiable risk factors, may help reduce cardiovascular morbidity, prevent diabetic foot complications and improve overall outcomes.

Keywords: Ankle-brachial index, Diabetes complications, Epidemiology, Risk factors

INTRODUCTION

The PAD is defined as acute or chronic obstruction of arteries supplying the extremities, which may lead to ischemia and tissue loss when severe [1]. Atherosclerosis is the most common cause. Clinical manifestations vary from asymptomatic disease to intermittent claudication, rest pain and non healing ulcers or gangrene. The true prevalence of PAD in diabetes is difficult to estimate, as many patients are asymptomatic or have blunted pain perception due to peripheral neuropathy. Most epidemiological studies [2,3] rely on the ABI, a simple and reliable non invasive test. Globally, PAD prevalence is about 6% in adults over 40 years, increasing to 15-20% in those above 65 years [4]. Approximately, 10-30% of patients develop claudication. The incidence of critical limb ischaemia is 22 per 100,000 annually, with amputation rates of 10-30% at 30 days and a 1-year mortality of around 15% [5-7].

The PAD is associated with high cardiovascular risk. Nearly half of patients have coexistent CAD and a quarter have carotid stenosis [8-10]. Mortality rises with worsening ABI; five-year mortality is 10% with ABI <0.85 and up to 50% annually when ABI <0.40 [11]. PAD is a major contributor to diabetic foot ulceration, increasing risk by 14-24% [12]. Diabetes accelerates atherosclerosis, affecting coronary, carotid and lower extremity vessels and PAD is often silent until advanced disease develops [13]. Diabetic patients more commonly develop distal (femoro-popliteal and tibial) disease, whereas smoking and hypertension are associated with proximal (aorto-iliac) disease.

The pathogenesis of PAD in diabetes is multifactorial. Chronic hyperglycaemia induces endothelial dysfunction, vascular smooth muscle changes and platelet hyperactivity [14,15]. Metabolic abnormalities such as hyperglycaemia, elevated free fatty acids and insulin resistance contribute to vascular injury by reducing nitric oxide, increasing oxidative stress and activating pro-inflammatory and pro-thrombotic pathways [16-20]. These mechanisms promote accelerated atherosclerosis in diabetes.

In India, considerable attention has been paid to diabetic complications like CAD, retinopathy, nephropathy and neuropathy, but PAD has received less focus despite its significant burden. Early detection is essential since PAD often remains clinically silent yet markedly increases the risk of cardiovascular events, amputations and mortality. Hence, the present study was conducted to estimate the prevalence of PAD in patients with T2DM attending a tertiary care centre in West Bengal and to identify the predictors of PAD, including demographic, clinical and biochemical factors. Understanding these

predictors may aid in earlier diagnosis, improved risk stratification and targeted management strategies to reduce diabetes-related morbidity and mortality. The primary objective was to estimate the prevalence of PAD in patients with T2DM attending a tertiary care centre in West Bengal. The secondary objectives was to identify the clinical, demographic and biochemical predictors of PAD and to evaluate the association of PAD with microvascular complications (retinopathy, nephropathy, neuropathy) and macrovascular co-morbidities such as CAD.

MATERIALS AND METHODS

It was a single-centre, cross-sectional, hospital-based study conducted in the Department of General Medicine, Midnapore Medical College and Hospital, West Bengal, India, over a period of 12 months (January 2019 -December 2019) Ethical clearance was obtained from the Institutional Ethics Committee (Memo No. MMC/IEC-2019/193, dated 28/01/2019) and written informed consent was taken from all participants.

Sample size calculation: It was calculated using the formula,

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Assuming a PAD prevalence (p) of 6% in type 2 diabetes, with 95% confidence (Z=1.96) and 5% absolute precision (d=0.05), the minimum required sample size was 87, which increases to 97 after adjusting for 10% attrition. Due to feasibility, 100 patients were finally enrolled [4].

Inclusion criteria: Patients with T2DM, aged between 18-65 years, attending outpatient or inpatient services were included.

Exclusion criteria: Patients with leg ulcer, trauma, prior leg or ankle surgery, lower limb filariasis, deep vein thrombosis, renal impairment, or history of smoking were excluded from the study.

Study Procedure

The screened patients, 100 fulfilled the eligibility criteria and were included in the study. Demographic details, duration of diabetes and symptoms suggestive of PAD (claudication, rest pain, tissue loss) were recorded. History of ischaemic heart disease was noted. Clinical assessment included BMI, waist and hip circumference, blood pressure, peripheral pulses, dependent rubor/pallor and detailed foot examination (temperature, hair, nail, sweating, venous prominence, fissures, ulceration, deformities, callosities).

For ABI measurement, patients rested for 5-10 minutes in supine position. Blood pressure was measured using an appropriate sphygmomanometer cuff and a handheld 5 MHz Doppler probe. The probe was angled at 45-60° in the direction of the blood flow over the brachial pulse to detect a signal. The sphygmomanometer cuff was inflated until the signal disappears; then, 30 mmHg higher and deflated slowly until the signal returns in order to obtain brachial SBP. The procedure was repeated with the other arm and the higher of the two values is recorded to calculate the ABI. Sphygmomanometer cuff was placed around the patient's ankle immediately above the malleoli. Dorsalis pedis pulsation is palpated where possible at the dorsum of the feet between the proximal portions of the first and second metatarsal. The posterior tibial pulse was palpated in the groove posterior (behind) and slightly inferior (below) to the medial malleolus. If unable to palpate, ultrasound gel is applied and the pulsation located with doppler probe signal. The cuff was inflated and the same procedure was repeated as for the arms to obtain systolic ankle blood pressure. The same procedure was repeated for the other foot. Higher of the two values (among dorsalis pedis and posterior tibial) for each foot is used to calculate ABI. ABI was calculated as the ratio of ankle systolic pressure (higher of dorsalis pedis or posterior tibial) to brachial systolic pressure (higher of two arms). ABI ≤0.90 was considered diagnostic for PAD [21].

Patients with abnormal ABI underwent duplex sonography in the Department of Radiology for confirmation of PAD, assessing both anatomical characteristics and functional significance of arterial stenosis. Doppler waveform analysis was used to grade severity based on Peak Systolic Velocity (PSV) and flow pattern.

Doppler waveform becomes altered if the probe is placed distal on an arterial stenosis and characterised by deceleration of systolic flow, loss of early diastolic reversal and diminished PSV frequencies. The normal PSV in peripheral lower limb arteries varies from 45-180 cm/s. Mild disease results in biphasic waveform with loss of late diastolic forward flow. With increasing severity- the narrowing of waveform and then becomes monophasic and loss of flow reversal, slow systolic upstroke results in 'parvus tardus' appearance (decreased PSV and delayed rise). Severe arterial disease manifests as PSV >200 cm/s, mono phasic waveform and spectral broadening of the doppler waveform [22].

Screening for diabetic retinopathy was done by direct ophthalmoscopy, with positive cases confirmed by indirect ophthalmoscopy in ophthalmology. Peripheral neuropathy was assessed using 10g Semmes-Weinstein monofilament, vibration sense and ankle reflex. Laboratory tests included FPG, PPPG, fasting lipid profile and serum creatinine. The eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula, which estimates GFR-based on serum creatinine, age, gender and race. Compared to the Modification of Diet in Renal Disease (MDRD) formula, the CKD-EPI equation provides a more accurate estimation, especially at higher GFR values [23]. CAD was assessed using the Rose questionnaire, a standardised, validated tool used for epidemiological assessment of CAD. It identifies angina pectoris and related chest pain symptoms through a structured set of questions regarding exertional chest discomfort, it's location and relation to physical activity [24]. The Minnesota codes, the classification system provides a standardised method for coding Electrocardiogram (ECG) findings in epidemiological and clinical studies. It allows objective identification of ischaemic changes, myocardial infarction patterns and other ECG abnormalities related to CAD [25].

STATISTICAL ANALYSIS

Data were entered in Microsoft Excel 2021 and analysed using IBM SPSS version 24.0. Descriptive statistics were applied for demographic and clinical variables. Patients were divided into PAD and non PAD groups based on ABI classification [21]. Group comparisons were performed using unpaired student's t-test for continuous variables, Chi-square test for categorical variables and ANOVA for multiple group comparisons. Regression analysis was done to identify independent predictors for PAD. A p-value <0.05 was considered statistically significant.

RESULTS

Among the total 100 participants, 54 were males and 46 were females, with a mean age of 48.3±8.5years. A total of 59 patients (28 males and 31 females) were in the 30-50 years group (47.5% and 52.5%, respectively). The 50-65 years group comprised 40 patients (26 males and 14 females) (65.0% and 35.0%, respectively). Among PAD patients, males were 11 and females 8 is presented in [Table/Fig-1].

Considering ABI ≤ 0.90 (either in both or any one of the lower limbs) as diagnostic cut-off of PAD, 19 patients had PAD. Out of those 19 patients, 8 patients had bilateral PAD. Among the PAD patients

Age groups (years)	Male n (%) (n=11)	Female n (%) (n=8)	Total n (%) (n=19)
30-50	4 (36.4)	1 (12.5)	5 (26.3)
50-65	7 (63.6)	7 (87.5)	14 (73.7)

[Table/Fig-1]: Age group and gender distribution in the PAD group (n=19).

mild (ABI 0.7-0.9), moderate (ABI 0.4-0.69) PAD were 12 and seven patients, respectively. The majority, 73.7% were in the 50-65 years age group. This indicates significantly higher prevalence of PAD in the older age group, with a relatively higher proportion of females in the 50-65 years age group compared to males [Table/Fig-2].

Age groups (years)	PAD n (%) (n=19)	No PAD n (%) (n=81)	Pearson's Chi-square (χ^2)	p-value
18-30	0	1 (1.2)	11.135	0.004
30-50	5 (26.3)	54 (66.7)		
50-65	14 (73.7)	26 (32.1)		

[Table/Fig-2]: Age group distribution between PAD and non PAD group.

Mean BMI of males (23.1 kg/m^2) were in overweight group as per World Health Organisation (WHO) Asian BMI classification criteria (>23.0 kg/m^2), whereas mean BMI of females were 22.7 kg/m^2. Mean waist circumference (representing abdominal obesity) of males (82.9 cm) were in normal group, whereas females were having abdominal obesity group (80.4 cm) as per WHO Asian classification criteria (male <90 cm, female <80 cm). Mean waist hip ratio (representing central obesity) of males (0.90) in non obese and females (0.86) are in obese group as per WHO Asian classification criteria (male >0.9, female >0.85) [Table/Fig-3].

The mean duration of diabetes was 5.7±3.2 years. Average duration of diabetes in whole study group was 5.7 years and males having mean duration of diabetes slightly higher than females (6.0 years vs. 5.2 years). The current study showed that PAD patients had significantly higher mean values in FPG, PPPG, duration of diabetes, SBP, Diastolic Blood Pressure (DBP), TC, triglycerides and Low Density Lipoprotein Cholesterol (LDL-C). Conversely, the eGFR was significantly lower in PAD patients {58.65 mL/min/1.73m^2 Body Surface Area (BSA) vs.103.16 mL/min/1.73m^2 BSA}. The differences were statistically significant (p-value <0.001) for most parameters, indicating a strong association between PAD and adverse health indicators [Table/Fig-4].

Patients with PAD demonstrated a markedly higher prevalence of claudication, reduced peripheral pulses and co-existing CAD, reflecting more advanced systemic atherosclerosis. Microvascular complications such as diabetic retinopathy and nephropathy were also significantly more frequent among PAD patients, indicating a strong association between PAD and generalised vascular involvement [Table/Fig-5].

Regression analysis identified multiple clinical and biochemical parameters as independent predictors of PAD. Advancing age, abdominal adiposity, longer diabetes duration, poor glycaemic control, hypertension (especially SBP), higher LDLc, existing CAD and presence of microvascular complications significantly contribute to the development of PAD in diabetic patients [Table/Fig-6].

- SBP: Systolic blood pressure; DBP: Diastolic blood pressure; LDL-C: Low density lipoprotein cholesterol; eGFR: Estimated glomerular filtration rate
- Each independent variable was analysed separately using simple linear regression with PAD as the dependent variable.
- Significance was determined at p<0.05.
- Significant predictors of PAD indicates a statistically meaningful association with Peripheral Arterial Disease (PAD).

Parameters		Mean±SD	Std. error mean	t	p-value	95%CI	
						Upper	Lower
Waist circumference (cm)	PAD	87.5±7.6	1.7	3.902	<0.001	10.77	3.51
	No PAD	80.4±7.1	0.8				
Waist-hip ratio	PAD	0.92±0.08	0.02	2.823	0.006	0.09	0.02
	No PAD	0.87±0.07	0.01				
BMI (kg/m^2)	PAD	24.3±2.6	0.6	3.018	0.003	2.82	0.58
	No PAD	22.6±2.1	0.2				

[Table/Fig-3]: Waist circumference, waist hip ratio, Body Mass Index (BMI) distribution in PAD & non-PAD group.

Parameters		Mean±SD	Std. error mean	t	p-value	95%CI	
						Upper	Lower
FPG (mg/dL)	PAD	155.4±18.6	4.3	8.642	<0.001	51.13	32.03
	No PAD	113.4±18.9	2.1				
PPPG (mg/dL)	PAD	247.1±47.6	10.9	8.291	<0.001	117.71	72.75
	No PAD	152.1±44.3	4.9				
Duration of diabetes (years)	PAD	9.4±3.8	0.9	6.528	<0.001	5.97	3.19
	No PAD	4.8±2.4	0.3				
SBP (mmHg)	PAD	139.6±17.0	3.9	4.420	<0.001	9.0	24.7
	No PAD	122.6±15.4	1.7				
DBP (mmHg)	PAD	83.5±7.2	1.6	2.930	0.004	1.4	7.5
	No PAD	79.0±5.7	0.6				
Total Cholesterol (TC) (mg/dL)	PAD	191.4±50.8	11.6	3.998	<0.001	18.0	53.5
	No PAD	155.7±30.4	3.4				
Triglyceride (TG) (mg/dL)	PAD	161±93	21.3	3.873	<0.001	26.0	80.6
	No PAD	107.7±40.3	4.5				
LDL-C (mg/dL)	PAD	115.5±50.1	11.5	3.122	0.002	9.3	41.7
	No PAD	90±26.3	2.9				
eGFR (mL/min/1. 73m^2 BSA)	PAD	58.65±27.69	6.35	-4.998	<0.001	-62.19	-26.84
	No PAD	103.16±36.37	4.04				

[Table/Fig-4]: Duration of diabetes, glycaemic status, blood pressure, lipid profile in PAD and non PAD group.
SBP: Systolic blood pressure; DBP: Diastolic blood pressure; LDL-C: Low density lipoprotein cholesterol; eGFR: Estimated glomerular filtration rate

Parameters		PAD (%) (n=19)	No PAD (%) (n=81)	Pearson's Chi-square (χ^2)	p-value
Claudication history	Claudication present	8 (42.1)	1 (1.2)	31.389	<0.001
	Claudication absent	11 (57.9)	80 (98.8)		
Pulse palpability	Palpable	5 (26.3)	81 (100)	69.400	<0.001
	Not Palpable/ diminished	14 (73.7)	0		
Diabetic neuropathy	Neuropathy present	3 (15.8)	3 (3.7)	3.986	0.05
	Neuropathy absent	16 (84.2)	78 (96.3)		
Coronary Artery Disease (CAD)	CAD Present	10 (52.6)	5 (6.2)	26.053	<0.001
	CAD Absent	9 (47.4)	76 (93.8)		
Diabetic retinopathy	Retinopathy present	15 (78.9)	8 (9.9)	41.458	<0.001
	Retinopathy absent	4 (21.1)	73 (90.1)		
Diabetic nephropathy	Nephropathy present	15 (78.9)	15 (18.5)	26.494	<0.001
	Nephropathy absent	4 (21.1)	66 (81.5)		

[Table/Fig-5]: Distribution of claudication history, pulse palpability, Coronary Artery Disease (CAD), diabetic neuropathy, retinopathy, nephropathy in PAD and non PAD group.

Study (CUPS) study and Mohan V et al., also identified age as a significant predictors of PAD [26-29]. International studies by Fowkes FGR et al., (Edinburgh, UK), Beach KW et al., (Washington, DC) and Beks PJ et al., (Amsterdam, Netherlands) similarly reported higher prevalence of PAD in patients aged 50-74 years, highlighting age above 50 years as an important risk factor [4,30,31].

Duration of diabetes was also a significant predictor, with an average disease duration of 9.4±3.8 years among PAD patients. This finding aligns with the proposed pathophysiological model of microvascular and macrovascular complications in diabetes described by Fowler MJ [32]. The Fremantle, CUPS and Mohan V et al., studies also support duration of diabetes as a predictor of PAD. Most studies have shown that a duration greater than 10 years increases PAD prevalence, whereas the present study demonstrated significance even below 10 years [26,28,29].

The BMI was not identified as a predictor of PAD in this cohort, although 47.4% of PAD patients had BMI >23, consistent with overweight/obese categories in Asian classification. The average BMI among PAD patients was 24.3±2.6. Yakubu PD et al., reported higher PAD prevalence among obese patients compared to normal or underweight groups [33]. However, the lack of association in the present study may be explained by the fact that PAD predominantly affects older age groups and BMI is an imperfect measure of obesity in these populations due to reductions in lean tissue and muscle mass.

Abdominal obesity, measured by waist circumference, was a significant predictor of PAD in diabetic patients, with higher

Independent variables	B	Beta	t	p-value	R^2	Adjusted R^2	Interpretation
Age	-0.008	-0.493	-5.613	<0.001	0.243	0.236	Significant predictor of PAD
Gender	0.018	0.063	0.627	0.532	0.004	-0.006	Not a predictor of PAD
BMI	-0.009	-0.153	-1.529	0.129	0.023	0.013	Not a predictor of PAD
Waist circumference	-0.005	-0.283	-2.918	0.004	0.080	0.071	Significant predictor of PAD
Waist-hip ratio	-0.375	-0.202	-2.040	0.054	0.041	0.031	Not a significant predictor of PAD
Duration of diabetes	-0.026	-0.597	-7.373	<0.001	0.357	0.350	Significant predictor of PAD
Coronary Artery Disease (CAD)	0.210	0.535	6.261	<0.001	0.286	0.278	Significant predictor of PAD
Hypertension	0.163	0.479	5.408	<0.001	0.230	0.222	Significant predictor of PAD
Systolic Blood Pressure (SBP)	-0.005	-0.636	-8.153	<0.001	0.404	0.398	Significant predictor of PAD
Diastolic Blood Pressure (DBP)	-0.010	-0.012	0.139	0.890	0.191	0.183	Not a predictor of PAD
Fasting Blood Sugar (FBS)	-0.004	-0.630	-8.027	<0.001	0.397	0.391	Significant predictor of PAD
Postprandial Blood Sugar (PPBS)	-0.002	-0.645	-8.350	<0.001	0.416	0.410	Significant predictor of PAD
eGFR	0.002	0.499	5.701	<0.001	0.249	0.241	Significant predictor of PAD
TC	0.001	0.037	0.119	0.906	0.092	0.083	Not a predictor of PAD
Triglyceride	0.001	-0.114	-0.970	0.335	0.093	0.084	Not a predictor of PAD
LDL-C	-0.001	-0.236	-2.403	0.018	0.056	0.046	Significant predictor of PAD
HDL-C	-8.897	-0.005	-0.052	0.958	0.000	-0.010	Not a predictor of PAD
Liver Function Tests (LFT)	-0.007	-0.009	-0.092	0.927	0.000	-0.010	Not a predictor of PAD
Nephropathy	0.155	0.504	5.773	<0.001	0.254	0.246	Significant predictor of PAD
Retinopathy	0.215	0.644	8.338	<0.001	0.415	0.409	Significant predictor of PAD
Neuropathy	0.104	0.176	1.768	0.080	0.031	0.021	Not a predictor of PAD

[Table/Fig-6]: Simple linear regression analysis of individual independent variables associated with PAD.

- R^2 and adjusted R^2 values represent the proportion of variance explained by each variable individually.

DISCUSSION

In the current study, age emerged as a significant predictor of PAD in patients with diabetes. The mean age of patients with PAD was 55 years compared to 46.8 years in those without PAD and 73.7% of patients with PAD were in the 50-65 years category. The Fremantle Diabetes Study, Agarwal AK et al., the Chennai Urban Population prevalence in females (25%) compared to males (0%). However, central obesity defined by waist-hip ratio was not predictive, though present in 72.7% of males and 50% of females. Similar findings have been reported by Yakubu PD et al., and Ix JH et al., [33,34].

The PPPG showed significant association with PAD. This is explained by its role as an indirect marker of insulin resistance, which is central to the pathogenesis of PAD in T2D. Yakubu PD et al., and Ikem R et al., suggested that FPG is less likely to predict PAD [33,35].

Although dyslipidaemia is a well-established risk factor for atherosclerosis, its association with PAD is inconsistent. In the present study, LDL-C was identified as a predictor, while TC, triglycerides and HDL-C were not associated with PAD. Shukla V et al., reported similar findings [36].

The CAD was strongly associated with PAD, with 52.6% prevalence among PAD patients. Previous studies [27,36] also identified CAD as a predictor of PAD. Symptomatic PAD (claudication) was significantly more common in the PAD group (42.1%) compared to non PAD (1.2%). However, asymptomatic PAD (57.9%) was more frequent, likely due to concurrent neuropathy blunting pain perception. Tyagi V et al., similarly reported that 41.4% of PAD patients were asymptomatic [37].

Among diabetic complications, nephropathy (as evidenced by reduced eGFR) showed a strong association with PAD, with 78.9% prevalence in the PAD group. Retinopathy was also significantly associated, with 78.9% of PAD patients affected, consistent with findings by Fowler MJ [32]. In contrast, diabetic neuropathy, though present in 15.8% of PAD patients, was not a predictor, which aligns with observations by Agarwal AK et al., and Shukla V et al., [27,36]. Among complications diabetic retinopathy was found as a predictor of PAD. A 78.9% of patients among PAD group had diabetic retinopathy. Fowler MJ also found retinopathy as a predictor [32].

Simple linear regression analysis identified several significant predictors of PAD in type 2 diabetes patients. Age, waist circumference, duration of diabetes, CAD, hypertension, SBP, FPG and PPPG, reduced eGFR, elevated LDL cholesterol, nephropathy and retinopathy were found to be significant predictors ($p<0.05$). Among these, SBP, PPPG and retinopathy showed the strongest associations with PAD, as indicated by higher beta values and explanatory power (R^2). In contrast, gender, BMI, waist-hip ratio, DBP, TC, triglycerides, HDL-C, liver function tests and neuropathy were not significant predictors.

Limitation(s)

The present cross-sectional, single centred study was done with limited sample size (n=100), in a time-frame of 1 year. Nephropathy was assessed by eGFR, spot urine albumin-creatinine ratio or 24 hours urinary albumin estimation facility was not available in our set up during study period. HbA1c level measuring facility was also not available for assessing long term glycaemic status. Exclusion of smokers limits generalisability.

CONCLUSION(S)

The PAD in diabetes is more commonly associated than is generally believed. Increasing age, duration of diabetes, abdominal obesity, CAD, retinopathy, eGFR, glycaemic control status, LDL cholesterol, hypertension in general, SBP are important independent predictors of PAD in diabetes. Hence, early detection of PAD should be attempted for early initiation of treatment and to decrease mortality and morbidity in diabetic patients. Future multicentric follow-up studies using larger cohorts of patients are necessary to validate these results and to extend these findings among PAD patients without T2DM.

Authors' contribution: BCS: Concept and design of the study; TM: Collection of samples done; RM, UM, DH: Acquisition, analysis and interpretation of data; RM, UM: Drafting of the article; BCS, UM: Supervision; Final approval was done by all authors.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: May 20, 2025
- Manual Googling: Nov 19, 2025
- iThenticate Software: Nov 22, 2025 (1%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **May 19, 2025**
Date of Peer Review: **Sep 15, 2025**
Date of Acceptance: **Nov 26, 2025**
Date of Publishing: **Jun 01, 2026**