RESEARCH ARTICLE

A cross-sectional study evaluating the Ankle-Brachial index in type 2 diabetic neuropathy patients in a tertiary care hospital

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ABSTRACT

Background: Diabetic peripheral neuropathy (DPN) is a prevalent and disabling consequence of type 2 diabetes mellitus (T2DM) that frequently coexists with peripheral artery disease (PAD). We hypothesized that early diagnosis of subclinical vascular abnormalities using the ankle-brachial index (ABI) may help identify individuals at increased risk of neuropathy.

Aim: Our present study aimed to investigate the relationship between the ABI and peripheral neuropathy, as assessed by vibration perception threshold, in type 2 diabetes mellitus patients, and to explore the predictive utility of ABI for neuropathy among T2DM patients.
Methods: A cross-sectional study was conducted on 240 patients with T2DM who visited the outpatient department. Participants were separated into two groups based on vibration perception threshold (VPT)

scores: Neuropathy (VPT > 15V) and no neuropathy (VPT < 15V). Anthropometric marker (Body Mass Index) and biochemical markers (FBS, PPBS, and HbA1c) were measured. The ABI was measured with a handheld Doppler equipment. Data were analyzed using the Mann-Whitney t-test, and results were reported as Median (Interquartile range), followed by Spearman correlation and binomial regression analyses.

Results: Patients with neuropathy exhibited significantly higher HbA1c p = 0.046), higher PPBS (p = 0.004), and significantly longer duration of diabetes (p < 0.001) as compared to the no neuropathy group. The neuropathy group showed significantly higher ABI (p = 0.036) as compared to patients with no neuropathy. Binomial logistic regression analysis found diabetes duration (p < 0.001) and ABI (p = 0.039) as significant independent predictors of neuropathy with an accuracy of 45%, sensitivity of 85%, specificity of 50%, and area under the curve (AUC) of 0.718.

Conclusion: Neuropathy assessed by VPT in T2DM patients is significantly correlated with longer diabetes duration and vascular alterations revealed by ABI. Hence, regular screening with ABI and VPT can be used for identifying neuropathy and vascular problems at an early stage, facilitating the preventive treatment for diabetic neuropathy.

Keywords: Type 2 Diabetes Mellitus, Ankle–Brachial Index, Peripheral Neuropathy, Glycemic Control, Vibration Perception Threshold

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INTRODUCTION

Globally, diabetes is becoming a burden, with numbers exceeding 590 million, as reported. Sedentary lifestyle and obesity are giving a boost to the prevalence of Type 2 diabetes mellitus (T2DM).[1] Chronic diabetes mellitus leads to peripheral nervous system disruption, with sensory loss in the lower limbs, followed gradually by the upper limbs. Diabetes may lead to the development of atherosclerotic changes in the lower extremities, progressing to peripheral arterial disease (PAD), increasing the risk of ulceration and amputation. If PAD and DPN develop together, it will lead to subclinical limb ischemia, which should be diagnosed and treated in the early stage for the clinical management of diabetes.[3] Ankle-Brachial Index (ABI) is one of the affordable and accurate tools for early diagnosis of subclinical atherosclerosis and lower-limb symptoms in patients with T2DM.[4] Previous research by Hedayati et al. in 2025 highlighted the potential role of ABI as a simple, non-invasive, and accessible screening tool for identifying patients at increased risk of diabetes-induced kidney disease.[5] Chevtchouk *et al*., in 2017, investigated neuropathic pain and peripheral vascular disease in diabetes and compared this with the duration of type 1 and type 2 diabetes.[6] In both studies, there was no specific information regarding ABI as a diagnostic predictor of neuropathy assessed by vibration perception threshold in T2DM. Ravidas *et al*., in 2020, evaluated ABI in 105 T2DM patients with diabetic foot ulcers and found ABI to be a

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reliable and significant tool for duration of wound healing in patients.[7] Another study done by Zhang *et al*. in 2020 found that glycemic variability relates to lower extremity arterial disease and diabetic peripheral neuropathy in patients with T2DM.[8] In that study, they showed that ABI significantly correlated with glycemic indices, but no predictive utility of ABI to assess neuropathy was elucidated. Hence, limited data are available in India examining the combined role of ABI and VPT in determining neuropathy in T2DM patients. Therefore, we aimed to evaluate ABI's ability to predict peripheral neuropathy in patients with T2DM.

Materials and methods

A cross-sectional observational study was conducted among patients diagnosed with T2DM to assess ABI and its association with DPN, as assessed by VPT. The study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Ethics Committee (IEC/AIIMS/Kalyani/Meeting/2023/029). Patients with T2DM aged 30–70 years attending the diabetic outpatient clinic of a tertiary care hospital were screened from September 2023 to February 2025. The sample size was determined based on previously reported prevalence rates of DPN among patients with T2DM in Indian populations, which range from 30% to 60% across studies.[9-11] Considering an expected prevalence of 45%, a 95% confidence level (Z = 1.96), and a margin of error of 6% (d = 0.06). Substituting these values yielded a minimum sample size of 200 participants. A total of 280 participants were screened for eligibility in the Neurophysiology Laboratory, Department of Physiology, AIIMS, Kalyani. Among them, 30 patients were excluded, as mentioned in Figure 1. Finally, 240 T2DM patients were included in the analysis after excluding 10 patients (Figure 1). Among 240 patients, 103 patients were a part of a previously published cohort[12]. Participants were selected using convenience sampling during routine outpatient visits, after obtaining written informed consent.

The study included patients with a minimum diabetes duration of one year and the ability to provide informed

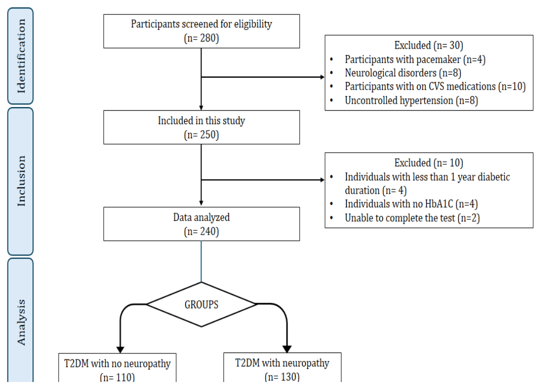

Figure 1: Flowchart of participant recruitment, identification, inclusion, and analysis

consent. Whereas, exclusion criteria were: history of stroke or other central nervous system disorders, history of peripheral artery disease, chronic alcohol use, chronic kidney disease (Stage 3 and above), current foot ulcers or infections, and any diagnosed non-diabetic neuropathy or systemic disease affecting peripheral nerves

Screening test for diabetic neuropathy

All patients were screened with the Michigan Neuropathy Questionnaire and the 10-g monofilament test according to standard protocol and recommendations.[8,9]

Anthropometric Assessment

Height was measured using a SECA 213 portable stadiometer, and body weight was recorded using a SECA 803 digital weighing scale (SECA GmbH & Co. KG., Hamburg, Germany). For each participant, equipment was calibrated before data collection as per the manufacturer's instructions.

Measurement of Vibration Perception Threshold (VPT)

In the present study, we measured VPT to assess neuropathy status using our recently published 2024 protocol.[12] After obtaining all data, the VPT values were graded as normal (<15V), mild (15-20V), moderate (20-25V), and >25 V as severe neuropathy.[13]

Measurement of Ankle-Brachial Index (ABI)

A handheld Doppler device (VD8M Mini Vascular Doppler) with a vascular probe was used to measure ABI. The patient was allowed to rest for 5 minutes in the supine position. A Riva Rocci cuff was placed on the arm, and the brachial pulse was palpated. Conductivity gel was applied, and the probe's tip was placed into the gel at a 45 to 60° angle until a clear arterial pulse was heard. The cuff was inflated to the point that the pulse sound disappeared. Then, above 20 mmHg, the cuff was deflated at a rate of 2 mmHg per second. When the arterial pulse sound resumed, that was marked as systolic pressure. Systolic blood pressure was determined in both arms and both ankles. The higher value of the brachial systolic pressure and the higher value of the two ankle readings for each leg (posterior tibial and dorsalis pedis) were selected. Then the higher of the two systolic pressures in the legs was divided by the higher of the two systolic pressures in the arms to obtain the ABI values.[15]

Assessment of blood glucose indices

Fasting blood glucose (FBG), postprandial blood glucose (PPBG), glycated haemoglobin (HbA1c), and duration of diabetes were recorded and compared between the neuropathy and non-neuropathy groups. Venous blood samples were collected in the morning after an overnight fast of at least 8 hours for estimation of FBG and HbA1c, while PPBG was measured 2 hours after a standardised meal on the same day. FBG and PPBG concentrations, and HbA1C, were determined using an automated hematoanalyzer (Thermo Fisher Scientific, Model: GENESYS 1XX).

Quality Control Measures

To confirm the quality of collected data, (a) testing was performed by trained personnel blinded to the clinical scores, (b) a standard script was used to instruct patients to minimise bias, (c) inter- and intra-observer variability was minimised by repeating 10% of tests under supervision, (d) all instruments were tested for technical error of measurement and performed calibration before study initiation, (e) the room temperature was maintained at 22 to 25°C during the testing period, (f) The skin temperature of the patient should be in between 33 to 37°C, and (g) patients were asked to take rest for 15 to 20 minutes in supine position before starting the test.

Statistical analyses

Data from 240 patients were analysed using Jamovi (Version 2.5.5). Because the data were not normally distributed, a nonparametric test was used to compare all recorded parameters between the neuropathy and no-neuropathy groups. A Mann-Whitney U-test was performed to assess all recorded parameters between the two groups. Spearman's correlation and binomial regression analyses were used to examine the associations among VPT, ABI, and diabetes duration. The diagnostic performance was evaluated using the Receiver Operating Characteristic (ROC) curve. Area under the curve (AUC), accuracy, specificity, and sensitivity were obtained from ROC analysis combining two predictors (ABI and duration of diabetes). Data visualization and graphical representations were generated using R software (version 4.4.3). The *p-value* less than 0.05 was considered statistically significant.

RESULTS

The study comprised 240 individuals with T2DM, who were divided into two groups based on VPT results: those with peripheral neuropathy and those without. Of all, 110 patients did not have neuropathy, and 130 did. The duration of diabetes was significantly longer in patients with neuropathy than in those without neuropathy. Postprandial blood glucose (PPBS) and HbA1c were also significantly higher in the neuropathy group, suggesting worse glycemic management. FBS and BMI differences were not statistically significant (Table 1).

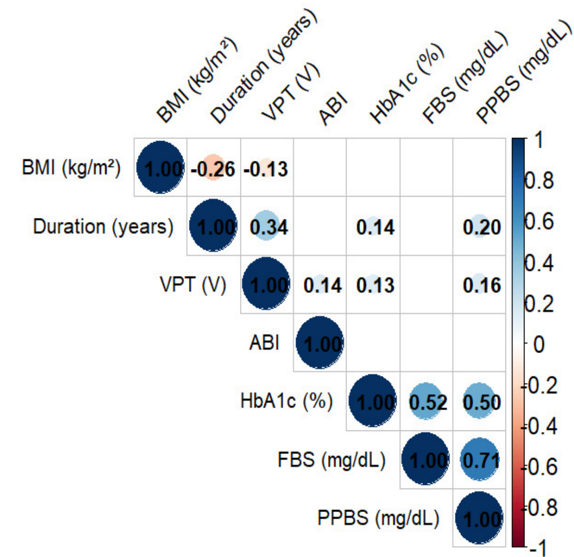

Figure 2: Correlation matrix of clinical, biochemical, and neuropathy-related parameters**.** The figure illustrates correlation coefficients (r value) among body mass index (BMI), duration of diabetes, vibration perception threshold (VPT), ankle–brachial index (ABI), glycated hemoglobin (HbA1c), fasting blood sugar (FBS), and postprandial blood sugar (PPBS). Circle size and colour intensity indicate the strength of the correlation, with blue indicating positive correlations and red indicating negative correlations (on a scale from −1 to +1).

Spearman's rho correlation analysis revealed that there was a significant association of ABI (r=0.14; p=0.041), duration of diabetes (r=0.34; p <0.001), PPBS (r=0.16; p =0.050), and BMI (r=-0.13; p=0.040) with neuropathy status marker VPT (Figure 2). VPT shows a moderate positive correlation with the duration of diabetes and ABI. Negative correlation was found between BMI and neuropathy-related measures.

Binary logistic regression analysis was performed to identify independent predictors of elevated VPT, a marker of peripheral neuropathy, in patients with T2DM (Table 2). The model demonstrated that both ABI and diabetes duration were significant predictors of neuropathy status, along with glycemic and metabolic markers. Binomial logistic regression analysis showed a significant inverse association of ABI with VPT (β = –2.928, SE = 1.45, p = 0.024), indicating that lower ABI values were associated with increased VPT

Table 1: Comparison of clinical, glycemic, and vascular parameters between the neuropathy and no neuropathy groups

Parameter	*No neuropathy (n=110) Median (IQR)*	*Neuropathy (n=130) Median (IQR)*	*Effect size (Rank Biserial correlation)*
Body Mass Index	24.90 (22.10-26.10)	24.40 (22.10-28.50)	0.13
Glycated haemoglobin (%)	7.00 (6.20-8.10)	7.50 (6.40-8.80)*	-0.15
Fasting Blood Sugar (mg/dL)	128 (113-157)	137(113-170)	-0.07
Postprandial Blood Sugar (mg/dL)	193 (152-257)	224 (179-296)*	-0.22
Duration of Diabetes (in years)	5.00 (3.00-8.00)	9.00 (5.00-13.50)*	-0.38

Ankle-Brachial Index	1.05 (0.98-1.11)	1.08 (1.00-1.15)*	-0.03
Vibration Perception Threshold (V)	9.25 (7.35-11.40)	22.5(17.30-29.50)*	-0.99

Values are expressed as Median (Interquartile Range). Independent samples Mann-Whitney U test applied; * indicates significant differences between the groups ($p < 0.05$).

Table 2: Binary logistic regression and linear regression analysis identifying predictors of elevated vibration perception threshold in patients with type 2 diabetes mellitus.

Model coefficients: Groups						95% CI	
Predictors	Estimate	SE	Z	p-value	Odds ratio	Lower	Upper
Intercept	3.936	1.5771	2.50	0.013	51.2272	2.32855	1126.98
ABI	-2.928	1.4524	-2.02	0.044	0.0535	0.00310	0.92
Duration	-0.124	0.0282	-4.40	<.001	0.8835	0.83607	0.93
Linear regression							
Predictors	Estimate	SE	t	R	R2	P value	
ABI	-0.03	0.012	-2.27	0.146	0.021	0.024	
Duration	-3.70	0.749	-4.94	0.305	0.09	<0.001	

Note: Dependent variable: Vibration perception threshold, ABI: Ankle Brachial Index, regression coefficients represent log odds of belonging to the neuropathy group. Odds ratios (OR) with 95% confidence intervals (CI) are reported. SE = Standard Error; t = t-test value for each regression coefficient; R = Regression coefficient; Duration = Duration of Diabetes (in years)

Table 3: Predictive measures and Receiver Operating Curve analysis of predictors

Predictors	Accuracy	Specificity	Sensitivity	AUC	R^2McF	R^2CS
ABI	45%	50%	85%	0.72	0.09	0.12
Duration of Diabetes						

Note: Combined Model estimated using sample size of N=240; AUC: Area under the curve; R^2McF : Mcfadden's R^2; R^2CS : Cox and Snell's R^2

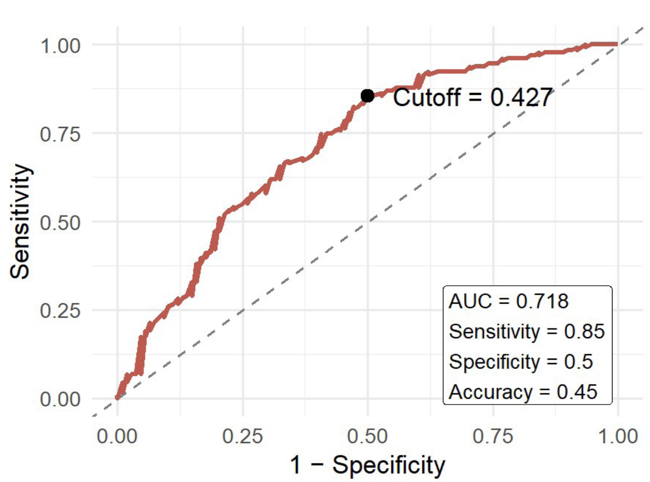

Figure 3: Receiver Operating Curve reflects a combined model of Ankle Brachial Index and duration of diabetes as significant diagnostic predictors, showing sensitivity, specificity, and AUC to predict neuropathy in type 2 diabetes mellitus patients

scoring (Table 2). Duration of diabetes also emerged as a significant independent predictor of elevated VPT, indicating the cumulative effect of chronic hyperglycemia on peripheral nerve function (Table 2). Linear regression analysis was performed separately for ABI and Duration of diabetes, and both showed a significant inverse association with VPT (Table 2).

Receiver operating characteristic (ROC) analysis was conducted to assess the discriminative ability of the predictors – ABI and duration of diabetes (Table 3). The model explained 9% of variance according to McFadden's R^2 and 12% according to Cox and Snell's R^2 (Table 3). The cut-off value was 0.427. Both predictors demonstrated significant predictive capacity with an accuracy of 45%, sensitivity of 85%, specificity of 50%, and an AUC of 0.718 (Figure 3).

DISCUSSION

In this study, we have measured the ABI and glycemic parameters in individuals with type 2 diabetes mellitus (T2DM) to determine their relationship with peripheral neuropathy. We found that patients having neuropathy had

a significantly longer duration of diabetes, higher HbA1c, and higher postprandial glucose levels than those without neuropathy. Although in our very recent 2024 article by Shaikh *et al*.,[12] the duration of diabetes showed no significant difference between the neuropathy and no neuropathy groups, we observed a significant effect of duration of diabetes, which may be due to the large sample size.[12] These findings are consistent with the well-known effect of chronic hyperglycemia in developing vascular complications in diabetic patients.

The duration of diabetes is a significant predictor of neuropathy, as found in logistic regression analysis ($p < 0.001$). This aligns with the findings of Bansal *et al*.,[10] who assessed 2,006 patients with T2DM, comprising 1,637 known cases and 369 recently identified cases, to ascertain the prevalence and risk factors of DPN. It was evaluated by using the ankle reflex examination, pinprick feeling, 10 g monofilament testing, and VPT grading. The prevalence of DPN was 29.2% overall, with known diabetics having a higher frequency (33.7%) than newly diagnosed cases (9.2%; $p < 0.001$). The duration of diabetes, dyslipidemia, and poor glycemic control (high HbA1c) were found to be independent risk factors using regression analysis. This study showed that the main predictors of neuropathy include prolonged disease duration and chronic hyperglycemia. Additionally, the time to development of diabetic neuropathy and its determinants were examined in a retrospective study by Tantigegn *et al*.[16] They took 669 patients in Amhara, Ethiopia, who had just received a type 2 diabetes diagnosis. They concluded that T2DM patients, especially those with poor glycemic control and vascular comorbidities, acquire neuropathy earlier. These findings support our results, demonstrating an association between neuropathy and diabetes duration and elevated fasting blood sugar levels, as well as the importance of early detection and management of risk factors. A longer disease duration indicates exposure to metabolic and vascular stress, which increases the risk of neural and arterial damage.[17]

The neuropathy patients group showed higher HbA1c and postprandial glucose levels, indicating poor glycemic control, which, in turn, contributed to nerve ischemia through oxidative stress, advanced glycation end products (AGEs), and endothelial dysfunction. A comprehensive review done by Tesfaye and Selvarajah in 2012 emphasized that up to 50% of people with diabetes have DPN, which makes it a significant contributor to morbidity, disability, and even death.[18] They compiled the data showing that the main causes of neuropathy development include poor glycemic control, longer-term diabetes, hyperlipidemia, obesity, and vascular dysfunction. They explained how oxidative stress and nerve microvascular disease play pivotal roles in DPN. They also emphasized the importance of managing cardiovascular risk factors and achieving glycemic control to prevent the progression of neuropathy. Treatments for symptoms include alpha-lipoic acid, pregabalin, and duloxetine. These observations support the results of our study, which gave significant correlations between increased HbA1c, long-term diabetes, and neuropathy, highlighting the importance of vascular and metabolic regulation in preventing brain ischemia.

The ABI is a widely used screening tool for PAD. In our data, mean ABI values were lower among patients without neuropathy but remained within the normal range in both groups. In logistic regression results, ABI was identified as a significant predictor of neuropathy, showing a relationship between subclinical vascular insufficiency and dysfunction of nerves. In this context, Chevtchouk *et al*. investigated the relationship between ABI and diabetic neuropathy in a cross-sectional study comprising 225 patients with type 2 diabetes mellitus. In their study, a Doppler instrument was used to quantify ABI, while nerve conduction studies and neuropathic pain questionnaires were used to evaluate neuropathy.[6] According to this study, patients with deviating ABI (< 0.9 or > 1.3) exhibited lower sensory conduction velocities and higher neuropathic pain levels. The authors concluded that ABI is a straightforward, simple indicator of vascular involvement and that peripheral artery disease and neuropathy persistently coexist in diabetes. These results verify our finding that, even when values are within the normal range, ABI correlates with the presence of neuropathy, indicating early subclinical vascular damage. Potier *et al*. compiled information from several studies investigating the application and diagnostic use of ABI in patients with diabetes in a review of the clinical evidence[19]. They pointed out that peripheral artery disease can be diagnosed with an ABI of less than 0.9, but arterial calcification in people with chronic diabetes may produce high values (> 1.3).[19] They have stressed that ABI provides important prognostic information on cardiovascular and neuropathic outcomes and have suggested it as a crucial screening tool for limb ischemia. These results support the use of ABI as a non-invasive indicator of vascular dysfunction in the current investigation, which also reflects the same mechanisms causing neuropathy and nerve ischemia. However, our study's findings elucidate the predictive utility of ABI for assessing peripheral neuropathy in T2DM patients, a question not addressed by the above-mentioned studies.[9-11] Moreover, endothelial dysfunction, oxidative stress, and vascular damage are common pathophysiological factors that explain the correlation between reduced ABI and neuropathy in individuals with T2DM.[17] Nitric oxide-mediated vasodilation is retarded by chronic hyperglycemia because it produces advanced glycation end products and activates the polyol pathway, which, in turn, increases reactive oxygen species. Axonal degeneration and demyelination result from the thickening of the capillary basement membrane and endoneurial hypoxia, which impedes blood flow to peripheral neurons.[20] Therefore, even a slight reduction in ABI could be

a sign of early angiopathic alteration that correlates with the functional loss and nerve ischemia, which is seen in diabetic neuropathy.

Therefore, we conclude that the ABI is correlated with the occurrence of peripheral neuropathy in individuals with neuropathy. Frequent screening with straightforward, non-invasive methods like VPT and ABI measurement can help in early identification of neural and vascular impairment, allowing for treatment and lowering the risk of long-term diabetic complications like limb ischemia and foot ulcers.

This study has certain limitations. First, participants were recruited through convenience sampling, which may limit the generalizability of the findings to the broader population. The study was conducted at a single tertiary care centre; future studies involving larger, more diverse populations across different regions of India would help strengthen external validity. Moreover, some factors, such as adiposity measures and lipid profile, were not evaluated in the present study. Here, we assessed vibration perception threshold for neuropathy, whereas advanced techniques such as quantitative sensory testing could be incorporated for a more comprehensive evaluation. Furthermore, the inclusion of circulating blood biomarkers related to inflammation and vascular dysfunction could help elucidate the underlying pathophysiological mechanisms.

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PEER-REVIEWED CERTIFICATION

During the review of this manuscript, a double-blind peer-review policy has been followed. The author(s) of this manuscript received review comments from a minimum of two peer-reviewers. Author(s) submitted revised manuscript as per the comments of the assigned reviewers. On the basis of revision(s) done by the author(s) and compliance to the Reviewers' comments on the manuscript, Editor(s) has approved the revised manuscript for final publication.