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Original Article

Correlation Between Peripheral Arterial Stiffness and Silent Myocardial Ischemia in Diabetic Patients

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ABSTRACT

Background: Type 2 diabetes mellitus is associated with accelerated vascular dysfunction and clinically silent myocardial ischaemia. Peripheral arterial stiffness may reflect systemic vascular injury associated with occult myocardial ischaemia. Objective: To evaluate the association between peripheral arterial stiffness and silent myocardial ischaemia among patients with type 2 diabetes mellitus. Methods: This hospital-based cross-sectional imaging study included 150 patients with T2DM at Aziz Fatima Medical & Dental College Teaching Hospital, Gujranwala, Pakistan. Peripheral arterial stiffness was assessed using brachial-ankle pulse wave velocity (baPWV) and ankle-brachial index (ABI). Silent myocardial ischaemia was assessed using resting electrocardiography and clinically indicated stress myocardial perfusion imaging. Group comparisons used continuous and categorical statistical tests as appropriate. Results: Silent myocardial ischaemia was identified in 46 participants (30.7%). Mean baPWV was higher in participants with SMI than in those without SMI (1896 ± 245 vs 1568 ± 210 cm/s), with a mean difference of 328 cm/s (95% CI 245.2–410.8; p<0.001; Hedges' g=1.48). The SMI group also had lower ABI (0.96 ± 0.08 vs 1.02 ± 0.07; p=0.002), higher HbA1c (8.6 ± 1.5% vs 7.8 ± 1.3%; p=0.003), longer diabetes duration (12.4 ± 5.6 vs 9.2 ± 4.6 years; p=0.001), and higher systolic blood pressure (143 ± 18 vs 136 ± 16 mmHg; p=0.021). Conclusion: Greater peripheral arterial stiffness was associated with silent myocardial ischaemia in patients with T2DM, with baPWV demonstrating the largest observed between-group difference. These findings support further evaluation of arterial stiffness as a cardiovascular risk marker but do not establish independent predictive or diagnostic performance. Keywords: Type 2 diabetes mellitus; peripheral arterial stiffness; brachial-ankle pulse wave velocity; silent myocardial ischaemia; cardiovascular risk; ankle-brachial index.

INTRODUCTION

Diabetes mellitus is a major global public-health problem and an important contributor to cardiovascular morbidity and mortality. Type 2 diabetes mellitus (T2DM), which constitutes the predominant form of diabetes, is associated with accelerated atherosclerosis, endothelial dysfunction, vascular inflammation, oxidative stress, and structural alterations in the arterial wall. Persistent hyperglycaemia and accompanying cardiometabolic abnormalities substantially increase the burden of coronary artery disease, heart failure, peripheral vascular disease, and other cardiovascular complications among affected individuals (1,2). Cardiovascular risk in T2DM is further influenced by

the frequent coexistence of hypertension, dyslipidaemia, obesity, and other components of metabolic dysfunction, making early recognition of vascular abnormalities clinically important.

An important challenge in cardiovascular assessment among individuals with diabetes is that clinically significant myocardial ischaemia may develop in the absence of typical anginal symptoms. Silent myocardial ischaemia (SMI) refers to objective evidence of inadequate myocardial perfusion occurring without the characteristic symptoms of myocardial ischaemia. Diabetes-related autonomic dysfunction and altered pain perception may diminish or abolish warning symptoms, allowing coronary abnormalities to remain clinically unrecognized until more advanced disease or a major cardiovascular event develops (3,4). Consequently, symptom-based assessment alone may be insufficient for identifying a subgroup of patients with T2DM who have underlying myocardial ischaemia, particularly when several cardiovascular risk factors coexist.

Arterial stiffness represents an important manifestation of vascular ageing and cumulative arterial-wall injury. Under physiological conditions, compliant arteries accommodate the pulsatile output of the heart and contribute to maintenance of efficient peripheral and coronary perfusion. In diabetes, chronic hyperglycaemia, accumulation of advanced glycation end products, oxidative stress, endothelial dysfunction, vascular calcification, collagen deposition, and changes in elastin contribute to progressive loss of arterial elasticity (5,6). Increased arterial stiffness can augment systolic pressure and left ventricular afterload while reducing the capacity of the arterial tree to buffer pulsatile pressure. Earlier return of reflected pressure waves may also impair diastolic coronary perfusion, providing a plausible pathophysiological link between vascular stiffening and myocardial oxygen supply-demand imbalance.

Pulse wave velocity is widely used as a non-invasive measure of arterial stiffness. Brachial-ankle pulse wave velocity (baPWV) measures the velocity of the arterial pressure wave between the brachial and ankle arterial sites, with greater velocities reflecting reduced arterial compliance. Increased pulse wave velocity has been associated with cardiovascular events and mortality and can provide vascular information beyond several conventional cardiovascular risk factors (7,8). Brachial-ankle pulse wave velocity has additionally been evaluated as a practical vascular marker in populations with hypertension, diabetes, and other cardiometabolic risk factors because its measurement is non-invasive and can be performed using automated vascular assessment systems (9,10).

The relationship between diabetes and arterial stiffness is particularly relevant because prolonged exposure to metabolic abnormalities can accelerate vascular ageing. Hyperglycaemia promotes non-enzymatic glycation of vascular proteins and the formation of advanced glycation end products, which increase cross-linking of collagen and reduce arterial-wall elasticity. Concurrent hypertension increases mechanical stress on the arterial wall, while dyslipidaemia, adiposity, and chronic inflammatory processes may further contribute to endothelial injury and atherosclerotic progression (5,11,12). Evidence also indicates that increased arterial stiffness is associated with adverse cardiovascular outcomes and may coexist with otherwise clinically silent coronary abnormalities in patients with diabetes (13). These observations support investigation of arterial stiffness as a marker of systemic vascular impairment rather than as an isolated peripheral arterial phenomenon.

The potential association between peripheral arterial stiffness and myocardial ischaemia also has a physiological basis. Stiffer arteries increase pulsatile haemodynamic load and myocardial workload, while impaired arterial recoil can adversely affect coronary perfusion during diastole. In individuals with underlying coronary atherosclerosis or microvascular dysfunction, these haemodynamic alterations may contribute to a mismatch between myocardial oxygen demand and supply (5,6). Because SMI may remain undetected through routine symptom-based clinical evaluation, a readily obtainable vascular measure such as baPWV could provide complementary information when assessing cardiovascular risk in individuals with longstanding T2DM. However, an association between baPWV and SMI should be distinguished from demonstration of diagnostic accuracy or prospective predictive performance.

The importance of this question is particularly relevant in South Asia, where the burden of T2DM and associated cardiovascular disease is substantial. Pakistan has experienced a marked burden of diabetes and pre-diabetes, with metabolic and cardiovascular risk factors contributing to considerable healthcare demands (14). South Asian populations may develop diabetes and cardiovascular complications at comparatively high levels of metabolic risk, emphasizing the need for context-specific approaches to cardiovascular risk assessment (15). Pakistani literature has similarly documented the clinical importance of cardiovascular complications among individuals with diabetes (16). Nevertheless, locally generated evidence examining the relationship between objectively assessed peripheral arterial stiffness and clinically silent myocardial ischaemia remains limited.

Gujranwala represents a large urban and industrial population in which diabetes and cardiovascular disorders are frequently encountered in clinical practice. Advanced cardiac imaging cannot necessarily be applied indiscriminately to every asymptomatic patient with diabetes, whereas non-invasive vascular assessment can be performed with substantially less procedural burden. Establishing whether increased peripheral arterial stiffness is associated with SMI in this population may therefore improve understanding of the vascular phenotype accompanying clinically unrecognized myocardial ischaemia and provide a basis for appropriately designed future diagnostic and longitudinal studies.

Accordingly, this hospital-based cross-sectional imaging study was conducted at Aziz Fatima Medical & Dental College Teaching Hospital, Gujranwala, Pakistan, to evaluate the association between peripheral arterial stiffness and silent myocardial ischaemia among patients with T2DM. The primary objective was to determine whether increased arterial stiffness, assessed principally using baPWV and supported by ankle-brachial index assessment, was associated with the presence of silent myocardial ischaemia after consideration of relevant demographic, metabolic, and cardiovascular risk factors.

MATERIALS AND METHODS

A hospital-based cross-sectional imaging study was conducted in the Department of Cardiology at Aziz Fatima Medical & Dental College Teaching Hospital, Gujranwala, Pakistan, from January to December 2025. The study was designed to examine the association between peripheral arterial stiffness and silent myocardial ischaemia among patients with established T2DM. Patients attending the cardiology and diabetic clinics during the study period constituted the source population. A total of 150 eligible participants were enrolled through consecutive sampling. All participants received information regarding the purpose and procedures of the study and provided written informed consent before participation.

Eligible participants were adults aged 40–70 years with T2DM diagnosed according to American Diabetes Association criteria (1), a duration of diabetes exceeding five years, clinical stability, and the ability to undergo the required vascular and cardiac assessment procedures. Patients were excluded if they had previously diagnosed coronary artery disease with a history of myocardial infarction or coronary intervention, symptomatic angina or other active cardiac symptoms, severe heart failure, significant renal impairment, peripheral vascular disease requiring surgical treatment, acute infectious or inflammatory disease, or an inability to complete the imaging procedures. These criteria were intended to establish a clinically stable diabetic population without previously recognized symptomatic coronary disease in whom clinically silent ischaemic abnormalities could be evaluated.

Demographic and clinical information was collected using a structured data-collection form. Recorded variables included age, sex, duration of diabetes, family history of cardiovascular disease, smoking status, history of hypertension, medication use, and physical activity level. Anthropometric assessment included measurement of body weight using a calibrated digital weighing scale and height using a standard stadiometer. Body mass index was calculated as weight in kilograms divided by height in metres squared. Blood pressure was measured after participants had rested for at least five minutes. Three measurements

were obtained using an automated blood-pressure monitor, and the mean of the three readings was used for analysis.

Venous blood samples were obtained following an overnight fast. Laboratory assessment included fasting blood glucose, glycated haemoglobin (HbA1c), total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), serum creatinine, and routine biochemical parameters. HbA1c was used as an indicator of longer-term glycaemic control, while lipid measurements were included because dyslipidaemia represents an important component of the cardiovascular risk profile in individuals with T2DM.

Peripheral arterial stiffness was assessed using brachial-ankle pulse wave velocity. Measurements were performed with an automated vascular screening system under standardized conditions. Participants were instructed to avoid caffeine intake, smoking, and heavy physical activity for at least 30 minutes before assessment. Each participant was positioned supine and allowed to rest for approximately 10 minutes before measurement. Blood-pressure cuffs were then placed on both arms and both ankles, and the device measured the pulse-wave transmission time between the brachial and ankle arterial sites. Brachial-ankle pulse wave velocity was calculated automatically, with higher values indicating greater arterial stiffness. Measurements were obtained bilaterally, and the higher baPWV value was used in the statistical analysis.

The ankle-brachial index (ABI) was recorded concurrently to characterize peripheral arterial circulation. ABI was calculated as the ratio of ankle systolic blood pressure to brachial systolic blood pressure. The combination of baPWV and ABI therefore provided complementary information regarding arterial stiffness and peripheral vascular status, with baPWV serving as the principal arterial-stiffness measure evaluated in relation to SMI.

Cardiac assessment for silent myocardial ischaemia included resting electrocardiography, which was performed in the enrolled participants, followed by stress myocardial perfusion imaging when clinically indicated. Participants demonstrating objective findings compatible with myocardial ischaemia in the absence of typical anginal symptoms were classified as having silent myocardial ischaemia. The assessment incorporated evidence of reversible perfusion abnormalities, ischaemic electrocardiographic changes, and imaging evidence of impaired myocardial perfusion, as applicable to the cardiac evaluation undertaken. Cardiac imaging findings were interpreted independently by experienced cardiologists who were blinded to the arterial-stiffness measurements, thereby reducing the potential for knowledge of baPWV results to influence interpretation of the cardiac assessment.

For analytical purposes, participants were categorized according to the presence or absence of silent myocardial ischaemia. Continuous variables were summarized as mean ± standard deviation, while categorical variables were reported as frequencies and percentages. The distribution of continuous variables was assessed before inferential analysis. Pearson or Spearman correlation analysis was used where applicable according to the distribution and characteristics of the variables. Differences between participants with and without SMI were evaluated using independent-samples t tests for continuous variables and chi-square tests for categorical variables.

Multivariable logistic regression analysis was used to assess the independent association between peripheral arterial stiffness and the binary outcome of silent myocardial ischaemia while accounting for potentially relevant confounding factors. The prespecified covariates reported for adjustment were age, sex, duration of diabetes, HbA1c, hypertension, BMI, and lipid profile. The regression analysis was therefore treated as an adjusted association analysis rather than as development or validation of a clinical prediction model. Statistical significance was defined as a two-sided p-value <0.05. Data were analysed using IBM SPSS Statistics version 26.0.

Several procedures were used to maintain consistency and reliability of data collection. Vascular and cardiac assessments were undertaken using standardized procedures, equipment was calibrated regularly according to manufacturer recommendations, and data collection was supervised by trained research personnel. Records were reviewed for completeness before final analysis. Blinding of the cardiologists interpreting the cardiac imaging findings to the arterial-stiffness measurements provided an additional measure against observer-related interpretation bias.

The study protocol was reviewed and approved by the Institutional Review Board of Aziz Fatima Medical & Dental College Teaching Hospital, Gujranwala. All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants, confidentiality of personal information was maintained, and participants retained the right to withdraw from the study without affecting their medical care.

RESULTS

A total of 150 patients with type 2 diabetes mellitus were included in the analysis. Their mean age was 56.4 ± 8.7 years, and 87 participants (58.0%) were male. The mean duration of diabetes was 10.2 ± 5.1 years and the mean BMI was 28.6 ± 4.3 kg/m². Hypertension was present in 62 participants (41.3%), while 38 (25.3%) were current smokers. Mean HbA1c was 8.1 ± 1.4%, indicating generally suboptimal glycaemic control in the study population. The mean total cholesterol, LDL-C, and HDL-C concentrations were 192.5 ± 38.7 mg/dL, 118.4 ± 31.2 mg/dL, and 42.6 ± 9.1 mg/dL, respectively (Table 1).

Silent myocardial ischaemia was identified in 46 of 150 participants (30.7%), whereas 104 (69.3%) were classified as having no evidence of silent myocardial ischaemia. Participants with SMI demonstrated substantially greater peripheral arterial stiffness than those without SMI. Mean baPWV was 1896 ± 245 cm/s in the SMI group compared with 1568 ± 210 cm/s in the non-SMI group, corresponding to a between-group mean difference of 328 cm/s (95% CI 245.2 to 410.8; p<0.001). The standardized between-group difference was large (Hedges' g=1.48), indicating marked separation in arterial stiffness between the two groups (Table 2).

Table 1. Baseline Characteristics of the Study Participants

Characteristic	Total Participants (n=150)
Age, years	56.4 ± 8.7
Male sex, n (%)	87 (58.0)
Duration of diabetes, years	10.2 ± 5.1
BMI, kg/m²	28.6 ± 4.3
Hypertension, n (%)	62 (41.3)
Current smoking, n (%)	38 (25.3)
HbA1c, %	8.1 ± 1.4
Total cholesterol, mg/dL	192.5 ± 38.7
LDL-C, mg/dL	118.4 ± 31.2
HDL-C, mg/dL	42.6 ± 9.1
Silent myocardial ischaemia, n (%)	46 (30.7)
No silent myocardial ischaemia, n (%)	104 (69.3)

Values are mean ± SD unless otherwise indicated. BMI, body mass index; HbA1c, glycated haemoglobin; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

Table 2. Comparison of Clinical and Vascular Parameters According to Silent Myocardial Ischaemia Status

Parameter	SMI Present (n=46)	SMI Absent (n=104)	Mean Difference (95% CI)	Hedges' g	p-value
baPWV, cm/s	1896 ± 245	1568 ± 210	328 (245.2 to 410.8)	1.48	<0.001
ABI	0.96 ± 0.08	1.02 ± 0.07	−0.06 (−0.09 to −0.03)	−0.82	0.002
HbA1c, %	8.6 ± 1.5	7.8 ± 1.3	0.80 (0.29 to 1.31)	0.58	0.003
Diabetes duration, years	12.4 ± 5.6	9.2 ± 4.6	3.20 (1.32 to 5.08)	0.65	0.001
Systolic blood pressure, mmHg	143 ± 18	136 ± 16	7.0 (0.86 to 13.14)	0.42	0.021

Values are mean ± SD. Mean differences are calculated as SMI present minus SMI absent. The 95% confidence intervals and Hedges' g values were derived from the reported group sizes, means, and

standard deviations. The p-values are those reported for the original between-group comparisons. Positive Hedges' g values indicate higher values in participants with SMI; the negative value for ABI indicates a lower mean ABI in the SMI group. SMI, silent myocardial ischaemia; baPWV, brachial-ankle pulse wave velocity; ABI, ankle-brachial index; HbA1c, glycated haemoglobin; CI, confidence interval.

Peripheral circulatory measurements also differed between groups. Mean ABI was lower among participants with SMI than among those without SMI (0.96 ± 0.08 vs 1.02 ± 0.07), with a mean difference of −0.06 (95% CI −0.09 to −0.03; p=0.002). The corresponding standardized difference was Hedges' g=−0.82. Thus, participants with silent ischaemic findings demonstrated both greater arterial stiffness, reflected by higher baPWV, and lower ABI values.

Differences were also observed in indicators of diabetes severity and metabolic control. Mean HbA1c was 8.6 ± 1.5% among participants with SMI compared with 7.8 ± 1.3% among those without SMI, yielding a mean difference of 0.80 percentage points (95% CI 0.29 to 1.31; p=0.003; Hedges' g=0.58). Participants with SMI had also lived with diabetes for a longer period, with mean durations of 12.4 ± 5.6 and 9.2 ± 4.6 years, respectively. The between-group difference was 3.20 years (95% CI 1.32 to 5.08; p=0.001), corresponding to a moderate standardized difference (Hedges' g=0.65).

Mean systolic blood pressure was 143 ± 18 mmHg in the SMI group and 136 ± 16 mmHg in the non-SMI group. This represented a mean difference of 7.0 mmHg (95% CI 0.86 to 13.14; p=0.021) and a standardized difference of Hedges' g=0.42. Collectively, the unadjusted comparisons demonstrated that participants with silent myocardial ischaemia had higher baPWV, poorer glycaemic control, longer diabetes duration, and higher systolic blood pressure, together with lower ABI values, than participants without silent myocardial ischaemia. The largest observed between-group difference was for baPWV.

DISCUSSION

The present study evaluated the association between peripheral arterial stiffness and silent myocardial ischaemia among patients with type 2 diabetes mellitus. Approximately one-third of the study population had evidence of SMI, and these participants demonstrated substantially higher baPWV than those without SMI. The between-group difference in baPWV was 328 cm/s, with a large standardized effect size, while the SMI group also demonstrated lower ABI, higher HbA1c, longer diabetes duration, and higher systolic blood pressure. Among the parameters examined, the largest between-group separation was observed for baPWV, supporting a clinically relevant association between greater peripheral arterial stiffness and the presence of silent myocardial ischaemia in this population.

The occurrence of SMI in 30.7% of participants is clinically important because myocardial ischaemia in diabetes may develop without characteristic anginal symptoms. Autonomic dysfunction and altered perception of myocardial pain can reduce the reliability of symptom-based detection, allowing coronary abnormalities to remain clinically silent (3). Contemporary approaches to chronic coronary syndromes similarly recognize that clinical presentation alone may not adequately characterize underlying myocardial ischaemia in patients with substantial cardiometabolic risk (4). The present findings therefore reinforce the importance of considering vascular and metabolic risk characteristics when evaluating individuals with longstanding T2DM, although the observed prevalence should be interpreted within the hospital-based sampling and diagnostic approach used in this study.

The most prominent finding was the substantially greater baPWV among participants with SMI. Increased arterial stiffness has previously been associated with cardiovascular morbidity and mortality, with meta-analytic evidence demonstrating that greater pulse wave velocity identifies individuals with increased cardiovascular risk (7). Findings from the Framingham Heart Study likewise showed that arterial stiffness contributes cardiovascular risk information beyond several conventional risk factors (8). Studies specifically evaluating brachial-ankle pulse wave velocity have demonstrated its association with subsequent cardiovascular disease and its utility as a practical marker of systemic vascular stiffness

(9,10,17). The present findings extend this vascular relationship to a diabetic population with clinically silent ischaemic findings, with baPWV demonstrating the strongest unadjusted between-group effect among the variables available for comparison. Previous work has also described an association between arterial stiffness and silent myocardial ischaemia in diabetes, providing directionally consistent evidence for this relationship (13).

Several mechanisms may plausibly explain the association between arterial stiffness and myocardial ischaemia. Chronic hyperglycaemia promotes oxidative stress, endothelial dysfunction, vascular inflammation, accumulation of advanced glycation end products, collagen cross-linking, and vascular calcification, all of which can progressively reduce arterial compliance. Increased arterial stiffness increases systolic load and left ventricular afterload while accelerating the return of reflected pressure waves. These haemodynamic changes may simultaneously increase myocardial oxygen demand and compromise diastolic coronary perfusion, particularly in individuals with underlying coronary atherosclerosis or microvascular dysfunction (5,6). Such mechanisms provide biological plausibility for the observed relationship but were not directly evaluated in the present cross-sectional study and therefore should not be interpreted as demonstrated causal pathways.

Glycaemic status and duration of diabetes also differed meaningfully according to SMI status. Participants with SMI had an HbA1c level approximately 0.8 percentage points higher than those without SMI and had lived with diabetes for approximately 3.2 years longer. Chronic exposure to hyperglycaemia contributes to progressive endothelial and vascular injury, while duration of diabetes is an important component of cardiovascular risk assessment in T2DM (1). Studies involving diabetic populations have also demonstrated relationships between poorer glycaemic control and adverse cardiovascular risk profiles (18). These findings suggest that the arterial changes observed in participants with SMI may reflect cumulative metabolic and vascular injury rather than an isolated abnormality of pulse-wave transmission.

Systolic blood pressure was also higher among participants with SMI, with an observed mean difference of 7 mmHg. Hypertension and arterial stiffness have a bidirectional relationship: sustained increases in arterial pressure contribute to structural remodelling of the vessel wall, whereas loss of arterial compliance can further augment systolic pressure and pulsatile haemodynamic stress (5). Hypertension is therefore an important potential contributor to the vascular phenotype observed in patients with diabetes, and effective blood-pressure management remains central to cardiovascular risk reduction in these populations (19). Because hypertension, diabetes duration, glycaemic control, adiposity, and lipid abnormalities may be interrelated, the unadjusted associations observed in this study cannot establish the independent contribution of any single factor.

The lower ABI observed among participants with SMI provides additional evidence of a less favourable peripheral vascular profile. Although mean ABI remained close to the conventional range in both groups, the between-group difference was statistically significant and accompanied by a comparatively large standardized difference. ABI and baPWV measure different aspects of vascular function: ABI primarily reflects pressure differences associated with peripheral arterial obstruction, whereas baPWV predominantly reflects arterial stiffness. Their concurrent differences suggest that patients with SMI may exhibit a broader vascular phenotype involving both arterial compliance and peripheral circulatory characteristics. However, the present data do not establish whether these measures provide incremental diagnostic information when considered together.

The findings are particularly relevant to the Pakistani clinical context. Diabetes and associated cardiovascular risk factors impose a substantial disease burden in Pakistan, as demonstrated by national epidemiological data (14). Cardiovascular complications are also a major concern throughout South Asia, where metabolic risk may occur at comparatively younger ages and where access to advanced cardiovascular investigation can vary substantially between healthcare settings (15,16). In this context, non-invasive vascular measurements such as baPWV are attractive because they can be obtained without

ionizing radiation or invasive procedures. Nevertheless, the current study demonstrates association rather than diagnostic accuracy. Before baPWV can be proposed as a screening test for SMI, its discrimination, sensitivity, specificity, clinically useful thresholds, incremental value beyond established cardiovascular risk factors, and performance against a consistently applied reference standard would require formal evaluation. Advanced imaging and established coronary assessment approaches remain necessary when clinically indicated (4,20).

Several strengths of this study merit consideration. The investigation addressed a clinically important and relatively underexplored relationship in a Pakistani population with established T2DM. Peripheral arterial stiffness was assessed using a standardized non-invasive baPWV procedure, measurements were obtained bilaterally, and the higher value was used consistently for analysis. Participants underwent standardized premeasurement preparation, including rest and avoidance of caffeine, smoking, and heavy physical activity. Cardiac imaging findings were interpreted by experienced cardiologists who were blinded to arterial-stiffness measurements, reducing the possibility that knowledge of baPWV influenced interpretation of the cardiac assessment. The analysis also compared several clinically relevant vascular and metabolic characteristics according to SMI status.

The findings should nevertheless be interpreted in view of important limitations. First, the cross-sectional design establishes contemporaneous associations but cannot determine temporal sequence or causality. Second, participants were recruited consecutively from a single hospital and were restricted to patients with T2DM aged 40–70 years and diabetes duration exceeding five years, which limits generalizability beyond comparable clinical populations. Third, stress myocardial perfusion imaging was undertaken when clinically indicated rather than being clearly documented as a uniformly applied reference assessment for every participant. Selective verification may introduce differential outcome ascertainment if the likelihood of undergoing advanced imaging was related to underlying cardiovascular risk. Fourth, although group comparisons demonstrated substantial differences in baPWV and several cardiometabolic factors, the available statistical evidence does not include the complete multivariable regression estimates required to determine whether baPWV remained independently associated with SMI after simultaneous adjustment for potential confounders. Residual confounding therefore remains possible. Finally, no diagnostic-performance analysis, including receiver-operating-characteristic analysis, sensitivity, specificity, predictive values, or validated baPWV threshold, was available; consequently, the findings should not be interpreted as validation of baPWV as a diagnostic or screening test.

Future research should evaluate this association in larger multicentre cohorts using a consistently applied cardiac reference standard and complete multivariable modelling. Prospective studies could determine whether baseline arterial stiffness is associated with subsequent myocardial ischaemia or cardiovascular events, while diagnostic studies could evaluate whether baPWV meaningfully improves discrimination beyond established clinical and metabolic risk factors. Such work would clarify whether the substantial group difference observed in the present study translates into useful incremental cardiovascular risk stratification.

CONCLUSION

Peripheral arterial stiffness was associated with silent myocardial ischaemia among patients with type 2 diabetes mellitus in this hospital-based cross-sectional study. Participants with SMI had substantially higher baPWV, together with lower ABI, poorer glycaemic control, longer diabetes duration, and higher systolic blood pressure than participants without SMI. The magnitude of the baPWV difference supports its relevance as a marker of an adverse vascular phenotype in patients with clinically silent ischaemic findings; however, the present data do not establish independent prediction or diagnostic screening performance. Larger multicentre and longitudinal studies using standardized cardiac assessment and

complete adjusted analyses are required to determine the incremental clinical value of arterial-stiffness measurement.

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