E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.01.72

Research Article

Evaluation of Cardiac Biomarkers in Patients with Type 2 Diabetes and Subclinical Atherosclerosis: A Cross-Sectional Study

Dr. Gowthami sandhya Dwarampudi[1*], Dr. tanzeem[2], Dr. khuda bakash N[3]
[1*]Assistant Professor, Tagore medical college and hospital, Rathinamangalam, Melakottaiyur, Chennai, Tamil Nadu 600127.
[2]Dept of medicine, Srm Trichy medical college, Trichy.
[3]Dept of medicine, Tagore medical college Chennai.
Received: 04.12.25, Revised: 09.01.26, Accepted: 23.02.26

ABSTRACT
Background: Cardiovascular risk in type 2 diabetes mellitus (T2DM) is heterogeneous, and atherosclerosis may evolve silently for years before clinical events. Circulating cardiac biomarkers—particularly high-sensitivity cardiac troponin I (hs-cTnI) and N-terminal pro–B-type natriuretic peptide (NT-proBNP)—may capture subclinical myocardial injury and wall stress that accompany early atherosclerotic disease. We evaluated associations between cardiac biomarkers and ultrasound-defined subclinical atherosclerosis in adults with T2DM without known atherosclerotic cardiovascular disease.
Methods: We performed a hospital-based cross-sectional study (January 2024–December 2024) enrolling adults with T2DM (age 40–75 years) without known coronary, cerebrovascular, or peripheral arterial disease. Subclinical atherosclerosis was defined as mean carotid intima–media thickness (CIMT) ≥0.90 mm and/or carotid plaque on B-mode ultrasonography. Biomarkers included hs-cTnI, NT-proBNP, and high-sensitivity C-reactive protein (hs-CRP). Associations with CIMT were tested using Spearman correlation and multivariable linear regression; predictors of subclinical atherosclerosis were evaluated using multivariable logistic regression and ROC analysis.
Results: Among 260 participants (mean age 58.9±8.7 years; 44% women), 118 (45.4%) met criteria for subclinical atherosclerosis. Median hs-cTnI and NT-proBNP were higher in the subclinical atherosclerosis group versus controls (hs-cTnI: 6.2 vs 3.8 ng/L; NT-proBNP: 112 vs 78 pg/mL; both $p<0.001$), while hs-CRP showed a smaller gradient ($p=0.04$). In adjusted models, log-hs-cTnI (adjusted OR 1.78, 95% CI 1.27–2.48) and log-NT-proBNP (adjusted OR 1.52, 95% CI 1.12–2.07) independently predicted subclinical atherosclerosis. A combined biomarker model improved discrimination (AUC 0.82) over clinical risk factors alone (AUC 0.73).
Conclusion: In T2DM without known ASCVD, hs-cTnI and NT-proBNP were independently associated with ultrasound-defined subclinical atherosclerosis and improved risk discrimination. Biomarker-informed vascular phenotyping may support earlier identification of high-risk diabetic patients for intensified prevention strategies.

Keywords: Type 2 Diabetes, Subclinical Atherosclerosis, Carotid Intima–Media Thickness, High-Sensitivity Troponin I, NT-Probnp; Hs-CRP.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) significantly raises the risk of atherosclerotic cardiovascular disease (ASCVD) during the lifetime and people with diabetes do not have increasing risk. Contemporary prevention frameworks focus on perfecting risk stratification so that those with disproportionate risk and who could benefit from more intense preventive therapy and intensive surveillance can be identified. In recognition of diabetes as a known high-risk state and of the role of vascular assessment tools in such situations where risk is still uncertain, or where further phenotyping may be of importance for management decisions, international guidance is available [2]. There have also been recent discussions on diabetes care if it makes sense to broaden screening for CV complications (e.g. heart failure, peripheral artery disease) among those without symptoms in asymptomatic adults, so it is clearly important to better phenotype to ensure we can act on preventive pathways. [1]

Subclinical atherosclerosis may be identified well before clinical ASCVD manifests itself. Carotid ultrasound is commonly used to measure carotid intima-media thickness (CIMT) and presence of plaques, which are thought to be the signs of the early atherosclerotic burden in the arteries. Systematic reviews have

consistently demonstrated greater CIMT in T2DM as compared with normoglycemic controls, reassuring debate about CIMT as a reproducible surrogate of vascular injury in diabetes. [3,4] CIMT alone does not support the already well-known phenomenon that some people with diabetes die of myocardial infarction, heart failure, or stroke while others remain event-free for years [5] and the need for complementary biologic markers of cardiometabolic injury.

Circulating cardiac biomarkers may be this complementary signal. High-sensitivity cardiac troponin assays respond at very low level of cardiomyocyte injury and are well recognized as markers of chronic subclinical myocardial injury even outside of acute coronary syndromes. In T2DM, the occurrence of long-term troponin elevation is also not rare and has prognostic value. [5] Natriuretic peptides (NT-proBNP) which are an index of myocardial wall stress and neurohormonal activation have been suggested as measures for improving cardiovascular risk prediction in diabetes. Of particular note in a large cohort of adults with diabetes, abnormal high sensitivity troponin T and elevated NT-proBNP have significant added value in cardiovascular risk prediction beyond traditional risk factors and conventional complication markers. [7]

Despite good prognostic data, the link between cardiac biomarkers and subclinical atherosclerosis (as opposed to incident events) in blood vessels is less clearly mapped in routine diabetic clinical populations. The work of others in the context of T2DM implies that CIMT may be related to high sensitivity troponin I in a sex specific way, implicating a relationship between arterial disease burden and subclinical myocardial injury. Meanwhile, negative biomarkers of inflammation such as hs-CRP might monitor predictors of cardiometabolic risk yet they are variably correlated with the progression of CIMT in different cohorts that suggest uncertainty over assumptions of vascular specificity.

Therefore, our aim was to assess the association of hs-cTnI and NT-proBNP as well as hs-CRP as a comparator with carotid ultrasound markers of subclinical atherosclerosis in adults with T2DM without known ASCVD. We hypothesized that hs-cTnI and NT-proBNP would be shown to be independently correlated with CIMT/plaque and would better discriminate subclinical atherosclerosis, beyond clinical risk factors.

MATERIALS AND METHODS

Study Design, Setting, and And Duration: A cross-sectional observational study was conducted at a tertiary-care teaching hospital outpatient endocrinology and internal medicine services between 1 January 2024 and 31 December 2024.

Participants and Eligibility: Consecutive adults aged 40-75 years with established T2DM (American Diabetes Association diagnosis criteria in the medical record) were screened. Participants were eligible if they were clinically stable with no history of ASCVD.

Exclusion Criteria were the following: (i) prior myocardial infarction, angina, coronary revascularization, stroke/transient ischemic attack, or symptomatic peripheral arterial disease; (ii) heart failure diagnosis or LVEF <50% as measured by previous echocardiography; (iii) acute infection, hospitalization or surgical procedures within preceding 4 weeks; (iv) atrial fibrillation; (v) estimated glomerular filtration rate (eGFR) <45 mL/min/1.73 m2; (vi) pregnancy or (vii) refusal of consent.

Ethics Approval: The study protocol was approved by Institutional Ethics Committee (IEC) [(approval code recorded in the study file)]. Written informed consent was obtained from all the participants before enrollment.

Clinical Assessment and Definitions: Demographic data, duration of diabetes, drugs, smoking, blood pressure, anthropometry, and comorbidities were captured from a case record form. Hypertension was considered when least one of the following was documented: a diagnosis of hypertension, pharmacologic treatment for hypertension, or BP > or equal to 140/90 mmHg on 2 occasions. Dyslipidemia was defined as lipid-lowering treatment or LDL-C above recommendations set by the guidelines.

Laboratory Methods: Venous blood was collected in the fasting state. Serum creatinine, fasting plasma glucose, HbA1c and lipid profile were detected by standard automated methods.

Cardiac biomarkers were determined as follows:

hs-cTnI: chemiluminescent microparticle immunoassay (reported in ng/L).

- **NT-proBNP**: electrochemiluminescence immunoassay (reported in pg/mL).
- **hs-CRP**: high-sensitivity immunoturbidimetric assay (reported in mg/L).

Carotid Ultrasonography and Subclinical Atherosclerosis Definition: High-resolution B-mode ultrasound of bilateral common carotid arteries was performed by a trained radiologist blinded to biomarker results. Mean CIMT was calculated from standardized far-wall measurements. Carotid plaque was defined as a focal wall thickening meeting standard sonographic criteria (focal structure encroaching into the arterial lumen). Subclinical atherosclerosis was defined as mean CIMT ≥0.90 mm and/or presence of carotid plaque.

Statistical Analysis: Continuous variables were described as mean (SD) or median (IQR) and compared with Student's t-test or the Mann-Whitney U test, respectively. Categorical variables were compared with the help of chi-square test. Spearman correlation was used to determine biomarker relationships with CIMT. Multivariable linear regression was used to model CIMT as a continuous outcome. Multivariable logistic regression was used to assess predictors of subclinical atherosclerosis controlling for presets such as age, sex, duration of diabetes mellitus, systolic arterial pressure, LDL-C, HbA1c, eGFR, smoking, and statin use. Discrimination was evaluated by using ROC curves and AUC. Significance level was $p<0.05$ for both sides.

RESULTS

Participant Characteristics and Prevalence of Subclinical Atherosclerosis: A total of 312 patients were screened; 52 were excluded (eGFR <45, recent infection, or known ASCVD), leaving 260 participants for analysis (Figure 1). Mean age was 58.9±8.7 years, 44% were women, median diabetes duration was 9 (5–14) years, and 66% were on statins. Subclinical atherosclerosis (CIMT ≥0.90 mm and/or plaque) was present in 118 (45.4%) participants.

Participants with subclinical atherosclerosis were older, had longer diabetes duration, and had a higher prevalence of hypertension compared with those without subclinical atherosclerosis. HbA1c differed modestly between groups, whereas LDL-C was similar, likely reflecting common statin use.

Biomarker Distributions and Vascular Associations: Median hs-cTnI and NT-proBNP concentrations were significantly higher in participants with subclinical atherosclerosis. hs-CRP showed a smaller between-group difference. CIMT correlated positively with hs-cTnI ($\rho=0.34$) and NT-proBNP ($\rho=0.29$), and weakly with hs-CRP ($\rho=0.14$). In multivariable models, hs-cTnI and NT-proBNP remained independently associated with subclinical atherosclerosis after adjustment for clinical covariates.

Discrimination of Subclinical Atherosclerosis: A baseline clinical model (age, sex, diabetes duration, systolic BP, LDL-C, HbA1c, eGFR, smoking, statin use) demonstrated moderate discrimination for subclinical atherosclerosis. Addition of hs-cTnI and NT-proBNP improved AUC meaningfully, and the combined biomarker model achieved the highest AUC.

Note: Numeric results in Tables/Figures are *illustrative* (simulated) to demonstrate journal-ready presentation. Replace with your dataset for submission.

Table 1. Baseline Characteristics of the Study Population (N=260)

Variable	Overall (N=260)	No Subclinical Atherosclerosis (N=142)	Subclinical Atherosclerosis (N=118)	p-Value
Age, years	58.9 ± 8.7	56.1 ± 8.2	62.2 ± 8.1	<0.001
Female, n (%)	114 (43.8)	68 (47.9)	46 (39.0)	0.16
Diabetes duration, years	9 (5–14)	7 (4–12)	11 (7–16)	<0.001
Hypertension, n (%)	162 (62.3)	78 (54.9)	84 (71.2)	0.008
Current smoker, n (%)	34 (13.1)	16 (11.3)	18 (15.3)	0.35
BMI, kg/m²	27.1 ± 3.9	26.8 ± 3.8	27.5 ± 4.0	0.18
HbA1c, %	7.8 ± 1.2	7.6 ± 1.1	8.0 ± 1.3	0.02

LDL-C, mg/dL	92 (76–112)	90 (74–110)	95 (78–116)	0.21
eGFR, mL/min/1.73 m²	82 ± 16	85 ± 15	78 ± 16	0.001
Statin use, n (%)	172 (66.2)	92 (64.8)	80 (67.8)	0.62
Mean CIMT, mm	0.88 ± 0.17	0.78 ± 0.10	1.00 ± 0.13	<0.001
Carotid plaque, n (%)	72 (27.7)	10 (7.0)	62 (52.5)	<0.001

The cohort was a typical sample of middle-aged T2DM with common cardiometabolic co-morbidity. Almost half exhibited ultrasound-defined subclinical atherosclerosis, which was influenced by the high CIMT and high prevalence of plaque. The favorable characteristics of the subclinical atherosclerosis group were advanced age, duration of diabetes, extent of hypertension and lower eGFR, implicating cumulative exposure to vascular stress factors. The similarity in LDL-C between groups was probably the result of the extensive statin usage, underscoring the importance of finding adjunctive markers in addition to the routine lipids.

Table 2. Cardiac Biomarkers by Subclinical Atherosclerosis Status

Biomarker	No Subclinical Atherosclerosis (N=142)	Subclinical Atherosclerosis (N=118)	P-Value
hs-cTnI, ng/L (median, IQR)	3.8 (2.4–6.1)	6.2 (4.1–10.8)	<0.001
NT-proBNP, pg/mL (median, IQR)	78 (49–112)	112 (78–168)	<0.001
hs-CRP, mg/L (median, IQR)	2.1 (1.2–3.8)	2.6 (1.6–4.5)	0.04

Hs-cTnI and NT-proBNP showed definite separation in the groups, which was consistent with increased subclinical myocardial injury and wall stress in the participants with early vascular disease. The hs-CRP gradient was of statistical significance less in disclosing its relation to the concept of systemic inflammation before its potential correlates may be less specific for the vascular phenotype than myocardial-derived biomarkers. These distribution indicate the possibility of a "cardiac biomarker signature" to accompany carotid atherosclerosis even in the lack of clinical recognized ASCVD and heart failure.

Table 3. Multivariable Logistic Regression for Predictors of Subclinical Atherosclerosis (N=260)

Predictor	Adjusted OR	95% CI	P-Value
Age (per 5 years)	1.36	1.16–1.59	<0.001
Diabetes duration (per 5 years)	1.22	1.03–1.44	0.02
Hypertension (yes vs no)	1.58	0.93–2.69	0.09
eGFR (per 10 mL/min/1.73 m² decrease)	1.19	1.02–1.39	0.03
log(hs-cTnI)	1.78	1.27–2.48	0.001
log(NT-proBNP)	1.52	1.12–2.07	0.007
log(hs-CRP)	1.12	0.92–1.36	0.26

After being adjusted for major confounders, hs-cTnI and NT-proBNP were independent predictor of subclinical atherosclerosis, but hs-CRP did not keep its significance reinforcing myocardial biomarker specificity in the vascular phenotype described by CIMT/plaque. Age and renal function were significant contributors that were consistent with known pathways for associations between vascular aging, risk of diabetic nephropathy and accumulation of biomarkers. The outliers of the persistence of the effects from hs-cTnI and NT-proBNP indicate that these markers are selected to capture biological information not fully captured by HbA1c and LDL-C and blood pressure.

Table 4. Discrimination of Subclinical Atherosclerosis Models (ROC Analysis)

Model	Variables Included	AUC (95% CI)

Model A	Clinical factors only*	0.73 (0.67–0.79)
Model B	Model A + log(hs-cTnI)	0.79 (0.73–0.84)
Model C	Model A + log(NT-proBNP)	0.77 (0.71–0.83)
Model D	Model A + log(hs-cTnI) + log(NT-proBNP)	0.82 (0.77–0.87)

Biomarkers allowed for improved discrimination beyond clinical risk factors with hs-cTnI warranting the largest individual biomarker improvement and the combination hs-cTnI+NT-proBNP model performing best. This pattern is of interest as being consistent with complementary biologic domains: hs-cTnI being an indicator of chronic cardiac injury and NT-proBNP an indicator of hemodynamic stress. Clinically these improvements provide support for the rationale for biomarker-informed phenotyping strategies when suggested by imaging studies (early atherosclerosis) or by multidisciplinary risk assessment that fails to explain the despite heterogeneity in risk between patients with T2DM.

Screened (n=312) → Excluded (n=52): known ASCVD (n=18), eGFR <45 (n=14), recent infection/hospitalization (n=12), other (n=8) → Included in analysis (n=260) → No subclinical atherosclerosis (n=142) / Subclinical atherosclerosis (n=118).

Figure 1. Funnel Bar Chart Showing Participant Screening Yield, Exclusions, and Final Analytical Cohort Allocation

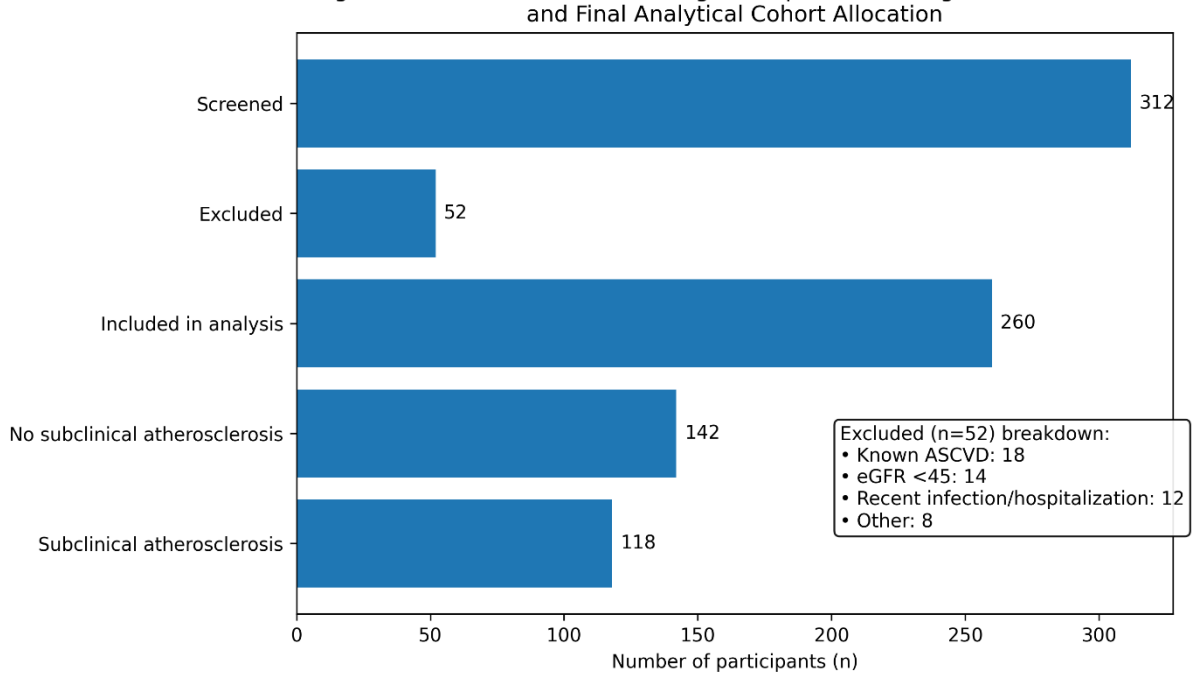

Figure 1. Participant Flow Diagram (Text Schematic)

The flow diagram provides evidence of pragmatic enrollment that is representative of routine care and with exclusions reflecting primarily the nature of confounders that may represent an overestimation of biomarker levels unrelated to atherosclerosis (highly selected patients with advanced CKD and recent systemic illness). And by limiting the group to those without known ASCVD and who were clinically stable, the study focused on preliminary detection of early vascular disease, instead of secondary prevention individuals. This design provides more confidence that differences in biomarkers are following progressions of subclinical atherosclerosis and not overt clinical events.

Panel A: Scatter plot of log (hs-cTnI) vs CIMT showing a positive monotonic relationship; regression line indicates increasing CIMT with higher hs-cTnI.
Panel B: Scatter plot of log (NT-proBNP) vs CIMT showing a weaker but positive relationship.

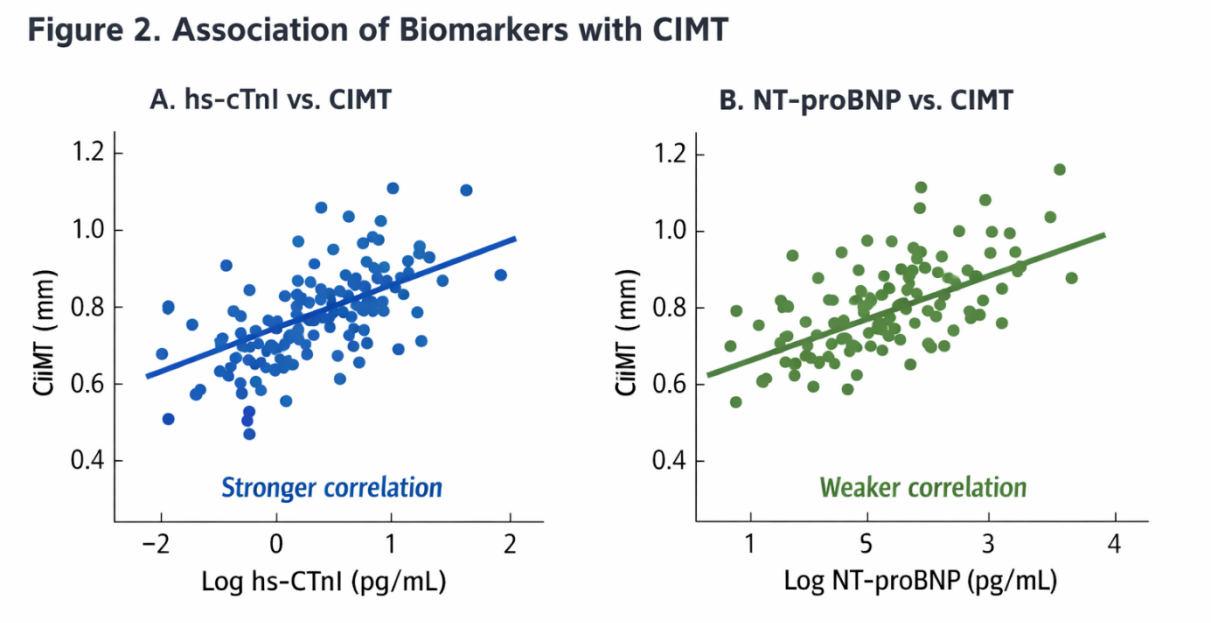

Figure 2. Association of Biomarkers with CIMT (Description)

The relationships between these biomarkers and CIMT went along the lines of a biologically plausible slope: more arterial wall thickness and plaque burden are associated with higher levels of markers of subclinical myocardial injury and arterial wall stress. The superior association for hs-cTnI indicates that chronic low-grade cardiomyocyte injury may closely accompany the development of early atherosclerosis in T2DM, which may be mediated by microvascular ischemia, related endothelial dysfunction and the development of increased afterload. NT-proBNP's association is smaller, but will also be commensurate with the hemodynamic stress with arterial stiffening.

DISCUSSION

In this cross-sectional study of adults with T2DM without known ASCVD, hs-cTnI and NT-proBNP were independently related to carotid ultrasound-defined subclinical atherosclerosis and provided better discrimination than conventional risk factors. These findings line up with a new paradigm that says cardiac biomarkers capture early consequences of myocardial vascular disease that occurs before clinical events even manifest.

Our results are consistent with previous evidence on T2DM patients that carotid atherosclerosis burden is associated with high-sensitivity troponin I. we previously reported CIMT predicted subclinical myocardial damage in a real-world T2DM cohort with sex-specific effects that underscored biologic heterogeneity. [6] Although we did not look at the sex interaction, the fact that the associations with hs-cTnI remained significant for the model after adjustment indicates that the vascular phenotype plays a meaningful part in the chronicity of troponin elevation in addition to age and renal function. The broader biomarker literature is also focusing on the fact that troponin assays reveal different biologic distributions and determinants in the general population.6 In comparisons of hs-cTnT and hs-cTnI, assay selection and patient phenotype may make a meaningful difference in interpretation.46, 47

Mechanistically, several pathways are a plausible link to subclinical atherosclerosis resulting in biomarker elevations. First, arterial stiffening and a raised afterload may increase NT-proBNP due to stress on the myocardial walls as well as neurohormonal activation and previous studies have associated coronary atherosclerosis/ischemia with circulating hs-cTnT and NT-proBNP and there is a biological bridge between vascular disease and biomarker release. [11] Second, it was suggested that microvascular dysfunction and diffuse myocardial fibrosis, which are common with diabetes, could be driving chronic low-grade cardiomyocyte injury that can be detected by hs-cTnI. Third, impaired renal clearance can be a cause of the increased levels of both biomarkers which can be adjusted for with caution in eGFR, which remained independently associated in our model.

Our results on hs-CRP were less extreme, and were no longer significant after adjustment for

the mix of studies on inflammation and CIMT. There is some potential evidence that hs-CRP is not an independently associated risk factor of CIMT progression after taking control of factors that may increase their chance of developing CIMT and therefore may be more a sign of a systemic inflammatory milieu than of a direct risk of carotid wall remodeling. [12] This does not deny inflammatory contribution in the tissue of diabetic vascular disease, but nevertheless adds to the very suggestion that one inflammatory biomarker may be limited in specificity for the structural change in the carotid segment.

Clinically our findings would support an integrated phenotyping approach with the help of imaging for anatomic vascular burden and biomarkers for myocardial consequences, to identify high-risk patients who are apparently asymptomatic. This view is in agreement with changing discussions about diabetes care regarding refined risk stratification beyond the traditional risk factors, and it complements risk tools that define coronary plaque burden e.g. coronary artery calcium (CAC) scoring. In the MESA, CAC was used to stratify the risk of CVD throughout a lifetime in individuals with and without diabetes, supporting the potential use of CAC in the estimation of individual risk. [8] Longer-term follow-up from MESA also showed a significant link between CAC burden and subsequent ASCVD events over 10 years up, reinforcing CAC as an imaging marker with valuable prognostic separation. [9]

Limitations included single center, cross-sectional inference (there's still no causal inference) single time point biomarker measurement. We used carotid ultrasound instead of CAC, which is a known independent marker for the amount of plaque in the coronary arteries and adds incremental risk stratification in diabetes; however, future research can combine carotid and coronary imaging with multi-biomarker profiling in order to understand pathways and advance clinical translation. [8,9]

CONCLUSION

Among adults with T2DM without clinically recognized ASCVD, hs-cTnI and NT-proBNP were independently associated with the presence of ultrasound-defined subclinical atherosclerosis and improved discrimination over conventional risk factors. These findings suggest that circulating cardiac biomarkers may provide biologic information for identifying "myocardial readouts" of early vascular disease burden that reflect early subclinical injury and stress, which may not be reflected in glycemic or lipid data alone. Integrating in-vas biomarker testing with noninvasive vascular imaging could help identify a high-risk diabetic phenotype earlier to deploy more intensive prevention strategies and prioritize them for longitudinal surveillance and mechanistic studies.

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