E-ISSN: 3078-4514 | P-ISSN: 3105-1316
Vol. 2, No. 2, 2025
DOI: 10.71295/JHDT.2025.02.2.08

RESEARCH ARTICLE

Prognostic Value of High-Sensitivity Cardiac Troponin İ for Major Adverse Cardiovascular Events and Revascularization in a Prospective Cohort from Azerbaijan

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Received: 07/12/2025 **Revised:** 08/12/2025 **Accepted:** 09/12/2025

Abstract

Background: High-sensitivity cardiac troponin I (hs-cTnI) enables detection of preclinical myocardial injury and may improve cardiovascular risk stratification beyond traditional markers. However, its prognostic value for major adverse cardiovascular events (MACE) and coronary revascularization in stable populations remains insufficiently defined.

Methods: In this prospective cohort study, adults free of acute coronary syndromes at enrollment were followed for incident MACE and revascularization. Baseline hs-cTnI levels were measured alongside inflammatory (CRP), thrombotic (D-dimer), and cardiorenal (NT-proBNP, microalbuminuria) biomarkers. Clinical characteristics were compared between hs-cTnI positive and negative groups using non-parametric statistics. Logistic regression models were applied to estimate odds ratios (ORs), stratified by gender and adjusted for confounders.

Results: hs-cTnI positivity was observed in approximately one-third of participants and was associated with older age, higher body weight, and greater waist circumference ($p < 0.05$). Positive individuals exhibited substantially higher levels of CRP, D-dimer, and NT-proBNP (all $p < 0.001$), whereas microalbuminuria did not differ significantly. In gender-stratified models, hs-cTnI was significantly associated with revascularization in men (OR ~1.9), but not in women. In the overall cohort, hs-cTnI independently predicted revascularization (OR ~1.5) and demonstrated moderate prognostic value for incident MACE. NT-proBNP showed an inverse association with revascularization risk in men, whereas D-dimer and microalbuminuria were not predictive.

Conclusions: Elevated hs-cTnI was associated with adverse cardiometabolic profiles and revascularization risk, supporting its potential value as a marker of preclinical myocardial injury and cardiovascular risk stratification.

Keywords: high-sensitivity troponin I; MACE; revascularization; biomarkers; prognosis; cardiovascular risk.

Introduction

Cardiovascular disease (CVD) remains the leading global cause of death, driven by aging populations and a high prevalence of cardiometabolic risk factors (1). High-sensitivity cardiac troponin (hs-cTn) assays have transformed cardiovascular diagnostics by enabling precise detection of low-grade myocardial injury beyond the scope of conventional tests (2,3). Increasing evidence indicates that detectable hs-cTn concentrations in stable individuals are common and strongly associated with major adverse cardiovascular events (MACE) and mortality, even in the absence of acute coronary syndromes (4,5).

These associations appear independent of traditional risk factors and reflect cumulative cardiometabolic and structural cardiac stress, highlighting hs-cTn as a promising biomarker for long-term risk prediction (5,6). At the same time, inflammatory and cardiorenal pathways play essential roles in CVD pathogenesis, and biomarkers such as CRP, D-dimer, NT-proBNP, and microalbuminuria may provide complementary prognostic insight (7).

Recent literature suggests that multimarker strategies incorporating hs-cTn could improve risk stratification and clinical decision-making (8,9), though questions remain regarding sex-specific effects and associations with revascularization outcomes.

Therefore, this study evaluated the prognostic value of hs-cTnI for major adverse cardiovascular events and

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revascularization in a prospective cohort, and examined its associations with inflammatory and cardiorenal biomarkers.

Methods and Materials

Study design and population. This was a prospective cohort study conducted at a single tertiary cardiovascular center. Consecutive adults referred for cardiovascular risk assessment or for evaluation of suspected coronary artery disease were screened for eligibility. Individuals presenting with acute coronary syndromes, hemodynamic instability, recent cardiac surgery, or active inflammatory disease at enrollment were excluded. A total of 847 participants with available baseline data were enrolled. High-sensitivity cardiac troponin İ (hs-cTnİ) measurements were available in 268 participants, who constituted the final analytical cohort for biomarker-based analyses.

Data collection and clinical assessment. Baseline demographic, anthropometric, and clinical information was obtained through structured interviews, physical examination, and review of medical records. Blood pressure was measured in accordance with international guidelines using an automated sphygmomanometer after a minimum of five minutes of rest. Body mass index (BMI) was calculated as weight divided by height squared, and body surface area (BSA) was estimated using the Du Bois formula.

Participants were prospectively followed for clinical events through scheduled clinic visits, electronic health records, and telephone contact. Outcome events were adjudicated by independent clinicians blinded to biomarker status.

Laboratory measurements. Venous blood samples were collected under fasting conditions at baseline. Serum hs-cTnİ was measured using a high-sensitivity immunoassay with a lower limit of detection of X ng/L and a coefficient of variation below 10%.

Additional circulating biomarkers, including C-reactive protein (CRP), D-dimer, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and microalbuminuria, were quantified using standardized clinical assays. All laboratory analyses were performed in an accredited central laboratory.

Outcome definitions. The primary endpoint was incident coronary revascularization, defined as percutaneous coronary intervention or coronary artery bypass grafting. The secondary endpoint was a composite of major adverse cardiovascular events (MACE), including cardiovascular death, nonfatal myocardial infarction, stroke.

Participants were followed from baseline until the first occurrence of an outcome, death, loss to follow-up, or study termination.

Statistical analysis. Continuous variables are presented as medians with interquartile ranges (IQR), and categorical variables as counts and percentages. Between-group comparisons were performed using the Mann–Whitney U test and chi-square test, as appropriate. Biomarkers with non-normal distributions were log-transformed prior to analysis.

Logistic regression models were used to estimate odds ratios (ORs) and 95% confidence intervals (CI) for associations between biomarkers and outcomes. Analyses were first unadjusted and subsequently adjusted for age, sex, body mass index, hypertension, diabetes, smoking, and other relevant cardiovascular risk factors.

Gender-stratified analyses were conducted to evaluate potential effect modification. Multiple hypothesis testing was addressed using the Benjamini–Hochberg false discovery rate correction. Statistical significance was defined as $p < 0.05$.

All analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA) and R software version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Of the 847 participants, the median age in the overall cohort was 59.0 years (IQR: 52.0–66.0). A total of 471 individuals (55.6%) were men and 376 (44.4%) were women. High-sensitivity cardiac troponin İ (hs-cTn İ) measurements were available for 268 participants (31.6%), with a median age of 61.0 years (IQR: 54.0–66.2). Within this sub-cohort, 158 (59.0%) were men and 110 (41.0%) were women.

Interpretation of Baseline Clinical Characteristics by hs-cTnI Status. Across most clinical parameters, approximately one-third of participants (32–35%) belonged to the hs-cTnI positive group, indicating a moderate prevalence of biomarker positivity within the cohort (Table 1). For anthropometric variables such as neck and waist circumference, the proportion of hs-cTnI positive individuals was substantially lower (~13–14%), which likely reflects limited sample availability rather than true epidemiological differences. Measures of body size (BMI, BSA) showed a larger proportion of hs-cTnI negative participants (~68%), consistent with the overall distribution of biomarker groups.

Participants with positive hs-cTnI values were older than those with negative results (median 63.0 vs. 60.0 years, $p < 0.001$), suggesting an age-related increase in preclinical myocardial injury. Body weight was also significantly higher among hs-cTnI positive individuals (84.0 vs. 80.0 kg, $p = 0.02$), pointing to a potential association between higher body mass and chronic myocardial stress.

Although height and BMI did not differ significantly, body surface area (BSA) was slightly but significantly elevated in hs-cTnI positive participants (2.0 vs. 1.9 m^2, $p = 0.03$), further supporting the presence of larger anthropometric dimensions in this subgroup. Waist circumference was notably higher in hs-cTnI positive individuals (112.0 vs. 99.0 cm, $p = 0.02$), a clinically meaningful finding given that

Table 1: Distribution of clinical characteristics by hs-cTnI status, shown as n (%) and median [IQR]

Variable	*Negative (n, %)*	*Median [Q1–Q3] (Neg)*	*Positive (n, %)*	*Median [Q1–Q3] (Pos)*	*p-value*
Age*	175 (65.3%)	60.0 [51.0–65.5]	93 (34.7%)	63.0 [58.0–69.0]	<0.001
Weight*	143 (67.1%)	80.0 [72.0–90.0]	70 (32.9%)	84.0 [76.2–93.9]	0.02
Height	147 (65.9%)	169.0 [162.0–174.5]	76 (34.1%)	170.0 [165.0–175.0]	0.25
BMI	141 (67.8%)	27.5 [25.4–31.2]	67 (32.2%)	29.7 [25.6–33.1]	0.11
BSA*	141 (67.8%)	1.9 [1.8–2.1]	67 (32.2%)	2.0 [1.9–2.1]	0.03
Neck circumference	27 (87.1%)	39.0 [35.5–40.0]	4 (12.9%)	41.0 [39.4–42.5]	0.12
Waist circumference*	24 (85.7%)	99.0 [90.0–104.0]	4 (14.3%)	112.0 [106.0–118.0]	0.02
SBP	171 (65.0%)	130.0 [120.0–160.0]	92 (35.0%)	133.0 [120.0–155.5]	0.60
DBP	171 (65.0%)	90.0 [80.0–90.0]	92 (35.0%)	80.0 [70.0–90.0]	0.06
Mean arterial pressure	171 (65.0%)	103.3 [92.2–113.3]	92 (35.0%)	101.0 [90.0–110.8]	0.20
Pulse pressure	171 (65.0%)	50.5 [40.4–60.7]	92 (35.0%)	50.5 [40.4–70.7]	0.46

* Statistically significant at $p < 0.05$

central adiposity is a key determinant of cardiometabolic risk and may contribute to myocardial injury.

Blood pressure parameters, including systolic blood pressure, mean arterial pressure, and pulse pressure, did not differ significantly between the groups. Diastolic blood pressure was numerically lower in the hs-cTnI positive group (80.0 vs. 90.0 mmHg, $p = 0.06$), but the difference narrowly missed statistical significance. These observations suggest that blood pressure per se may not be a primary determinant of hs-cTnI status in this cohort.

Overall, hs-cTnI positivity clustered with older age, higher body weight, larger body size, and increased central adiposity, whereas blood pressure measures were largely comparable. This pattern indicates that anthropometric and metabolic factors, rather than hemodynamic variables, may be more strongly associated with low-grade myocardial injury in this population.

Main Messages

- Age, body weight, BSA, and waist circumference were significantly higher in hs-cTnI positive individuals.
- Blood pressure parameters were similar between groups, indicating limited association with hs-cTnI status. Findings support a stronger link between metabolic/body size phenotypes and subclinical myocardial injury than hemodynamic measurements.

Figure 1 shows between-group differences in clinical and hemodynamic characteristics according to hs-cTnI status. *(Median ± IQR, log-transformed values, with p-values).* Continuous variables are presented as medians with interquartile ranges and displayed on a logarithmic scale to account for right-skewed distributions.

Participants with positive hs-cTnI demonstrated significantly higher age, body weight, body surface area, and waist circumference compared with those with negative hs-cTnI ($p < 0.05$), whereas height, BMI, neck circumference, and blood pressure parameters did not differ significantly ($p \geq 0.05$). Non-significant comparisons are denoted as "ns."

This pattern indicates that elevated hs-cTnI concentrations are preferentially associated with age-related and adiposity-related phenotypes, rather than with hemodynamic indices, suggesting a potential metabolic pathway contributing to low-grade myocardial injury.

Between-group differences in clinical parameters by hs-cTnI status. Positive hs-cTnI was associated with older age, higher body weight, larger body surface area, and greater waist circumference, while other measures were comparable. Values are log-scaled.

Individuals with positive hs-cTnI were older and exhibited greater body size measures, particularly weight, body surface area, and waist circumference. Blood pressure parameters were largely similar between groups. These findings suggest a stronger association between metabolic burden and myocardial injury than between hemodynamic stress and troponin elevation.

Table 2 presenets biomarker levels by hs-cTnI status expressed as N (%), median [IQR], and p-values. Individuals with positive hs-cTnI exhibited substantially higher concentrations of C-reactive protein (CRP), D-dimer, and N-terminal pro–B-type natriuretic peptide (NT-proBNP) compared with hs-cTnI negative individuals (all $p < 0.001$), reflecting concurrent systemic inflammation, activation of coagulation pathways, and heightened myocardial stress.

Microalbuminuria values were numerically elevated in the hs-cTnI positive group but did not reach statistical significance ($p = 0.24$), potentially indicating limited discriminatory capacity or reduced statistical power due to the smaller number of observations.

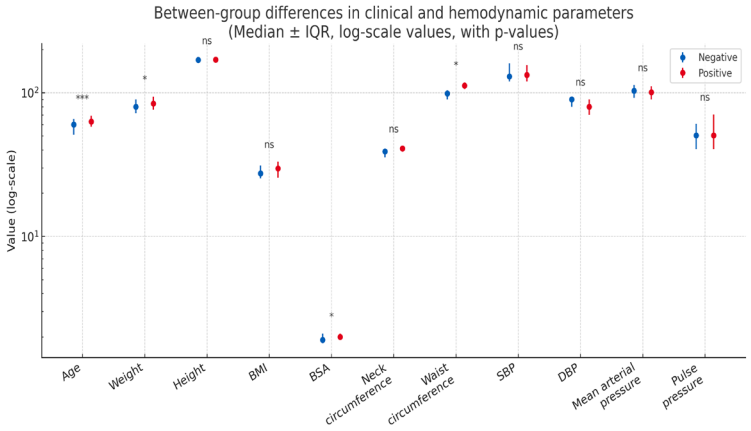

Figure 1: Between-group differences in clinical and hemodynamic characteristics according to hs-cTnI status. *(Median ± IQR, log-transformed values, with p-values).*

Table 2: Biomarker levels by hs-cTnI status expressed as N (%), median [IQR], and p-values

Biomarker	*Negative (N, %)*	*Median [IQR] (Neg)*	*Positive (N, %)*	*Median [IQR] (Pos)*	*p-value*
CRP, mg/L*	129 (63.9%)	5.0 [1.9–19.3]	73 (36.1%)	12.6 [4.7–45.5]	<0.001
D-dimer, ng/mL*	128 (66.0%)	320.0 [165.2–540.0]	66 (34.0%)	599.5 [267.7–1475.0]	<0.001
Microalbuminuria, mg/L	18 (37.5%)	109.0 [53.0–485.7]	30 (62.5%)	189.5 [96.7–491.2]	0.24
NT-proBNP, ng/L*	105 (56.1%)	715.0 [303.0–2996.0]	82 (43.9%)	1966.5 [965.5–8975.0]	<0.001

* Statistically significant at $p < 0.05$. Data are presented as median (IQR). p-values refer to group comparisons.

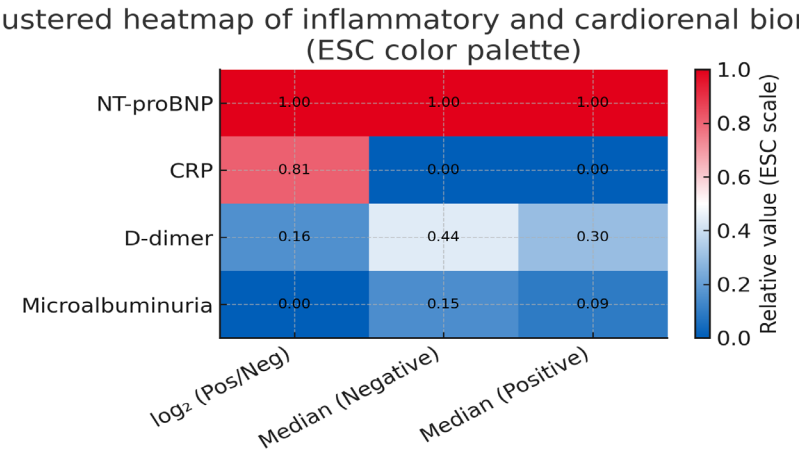

Figure 2: Clustered heatmap of inflammatory and cardiorenal biomarkers stratified by hs-cTnI status (ESC color palette)

Collectively, these findings delineate a distinct biomarker profile characterized by systemic inflammatory activation and cardiorenal dysregulation among individuals with elevated hs-cTnI, consistent with a higher cardiometabolic risk burden and subclinical cardiovascular injury.

As shown in Figure 2 the heatmap visualizes the relative distribution of inflammatory and cardiorenal biomarkers (NT-proBNP, CRP, D-dimer, and microalbuminuria) according to hs-cTnI status, using the ESC color palette for standardized expression. Values represent normalized scores derived from $\log_2$ ratios of positive versus negative groups, as well as median values within each group. Higher relative values (red) indicate greater biomarker elevation, whereas lower values (blue) reflect reduced concentrations.

NT-proBNP consistently exhibited the highest relative expression across all dimensions, suggesting pronounced myocardial stress in hs-cTnI positive individuals. CRP and D-dimer showed intermediate enrichment, reflecting systemic inflammation and coagulation activity,

Table 3: Odds ratios for major adverse cardiovascular events across inflammatory and cardiorenal biomarkers, stratified by gender

Gender/Group	*Biomarker*	*OR (95% CI)*	*p-value*	*q-value*
Female	NT-proBNP	2.30 (0.80–7.00)	0.14	0.25
Female	Troponin	1.70 (0.90–3.10)	0.07	0.22
Female	D-dimer	1.50 (0.90–2.50)	0.15	0.25
Female	Microalbuminuria	1.40 (0.40–5.50)	0.59	0.71
Male	Troponin*	1.90 (1.30–2.70)	<0.001	<0.001
Male	NT-proBNP	1.40 (0.80–2.30)	0.27	0.41
Male	D-dimer	1.10 (0.80–1.60)	0.53	0.71
Male	Microalbuminuria	1.00 (0.50–2.10)	0.95	0.95
All participants	Troponin*	1.90 (1.40–2.60)	<0.001	<0.001
All participants	NT-proBNP	1.50 (0.90–2.40)	0.09	0.22
All participants	D-dimer	1.30 (1.00–1.70)	0.08	0.22
All participants	Microalbuminuria	1.00 (0.50–2.00)	0.93	0.95

while microalbuminuria demonstrated the lowest relative expression, with limited between-group discrimination.

Overall, this clustered profile highlights a biomarker signature characterized by myocardial stress, inflammation, and thrombosis, predominantly among participants with elevated hs-cTnI.

Table 3 summarizes the associations between inflammatory and cardiorenal biomarkers and the risk of major adverse cardiovascular events (MACE), expressed as odds ratios (OR) with 95% confidence intervals, stratified by sex and evaluated overall.

Among males, elevated troponin was strongly associated with increased odds of MACE (OR: 1.90 [1.30–2.70], $p < 0.001$, $q < 0.001$), and this association persisted when analyzed in the overall sample (OR: 1.90 [1.40–2.60], $p < 0.001$, $q < 0.001$).

Other biomarkers, including NT-proBNP, D-dimer, and microalbuminuria, did not demonstrate statistically significant associations after false discovery rate adjustment (all $q > 0.05$), although NT-proBNP showed a numerical trend toward increased risk in certain subgroups.

Collectively, these results indicate that troponin is the most robust independent marker of future cardiovascular events in this cohort, whereas NT-proBNP, D-dimer, and microalbuminuria have limited prognostic value when adjusted for multiple comparisons.

The forest plot in Figure 3 illustrates the associations between inflammatory and cardiorenal biomarkers and the odds of major adverse cardiovascular events (MACE), stratified by sex and assessed overall. Odds ratios (OR) and 95% confidence intervals derived from logistic regression models are presented, with a vertical dashed line indicating the null value (OR = 1.0).

Among males and in the overall sample, higher troponin concentrations were strongly associated with increased odds of MACE, as reflected by ORs substantially above 1.0 with narrow confidence intervals. NT-proBNP, D-dimer, and microalbuminuria showed weaker or non-significant associations, with wide confidence intervals and point estimates closer to the null.

Overall, these results suggest that troponin is the most robust predictor of adverse cardiovascular outcomes in this cohort, while the prognostic value of other biomarkers appears limited.

Table 4 presents odds ratios (OR) and 95% confidence intervals for the association between selected inflammatory and cardiorenal biomarkers and the risk of coronary revascularization, stratified by sex and evaluated in the overall population.

Among males, higher NT-proBNP levels were significantly associated with reduced odds of revascularization (OR: 0.50 [0.30–0.90], $p = 0.02$, $q = 0.07$), suggesting a potential inverse relationship between myocardial stress biomarkers and subsequent procedural interventions.

In the overall analysis, both NT-proBNP and troponin demonstrated nominal associations with revascularization ($p < 0.05$), although neither remained statistically significant after false discovery rate adjustment ($q \geq 0.07$).

Other biomarkers, including D-dimer and microalbuminuria, did not show significant associations in any subgroup, indicating limited predictive value for revascularization in this cohort.

Overall, these results suggest that biomarker predictors of revascularization are weak and inconsistent, with only modest evidence for NT-proBNP and troponin, primarily before multiple testing correction.

This forest plot displays odds ratios (OR) and 95% confidence intervals for the associations between selected biomarkers and the risk of coronary revascularization,

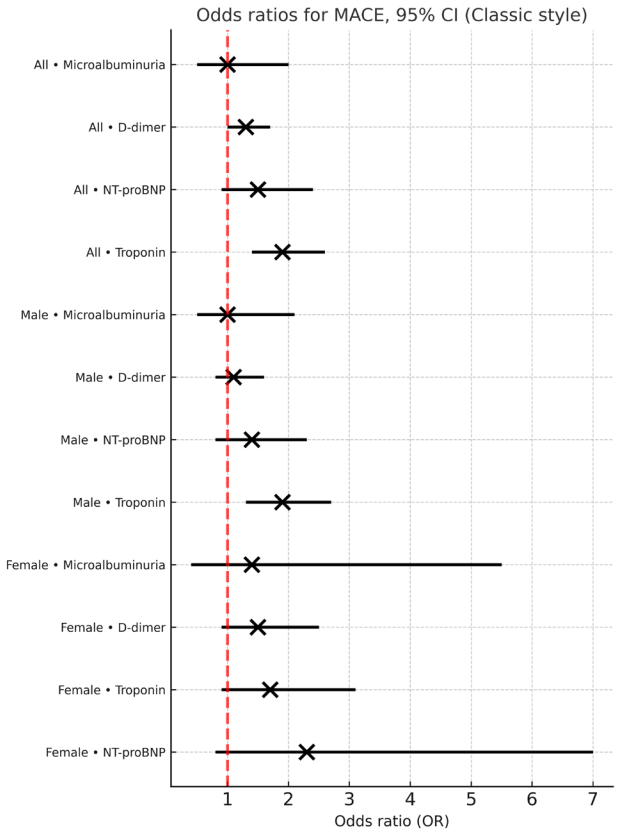

Figure 3: Odds ratios for MACE across inflammatory and cardiorenal biomarkers, stratified by sex (95% CI)

stratified by sex and evaluated in the overall population. The vertical dashed line indicates the null value (OR = 1.0).

Across most biomarkers and subgroups, OR estimates were close to unity with wide confidence intervals, indicating limited predictive value. Among males, NT-proBNP and troponin showed modest elevations in risk estimates, although these associations were not statistically robust. In females, biomarker estimates were highly variable, with wide confidence intervals reflecting small subgroup sizes and low precision.

Overall, these findings suggest no consistent or strong biomarker predictors of revascularization, and highlight substantial uncertainty in subgroup-specific estimates.

Figure 4 presents the sex-stratified and overall associations between biomarker levels and the likelihood of undergoing coronary revascularization, expressed as ORs with 95% CIs.

Across the full cohort, troponin and NT-proBNP displayed elevated ORs, suggesting that biochemical markers of myocardial injury and ventricular strain are linked to a higher probability of revascularization, although the confidence intervals indicate uncertainty due to sample size.

Among men, troponin demonstrated a markedly elevated OR (≈2.0), with tighter confidence bounds relative to other biomarkers, indicating a more consistent association between myocardial injury and subsequent revascularization decisions in this subgroup. D-dimer and microalbuminuria showed weak or null associations, suggesting limited prognostic relevance for procedural outcomes in men.

Among women, the point estimates for NT-proBNP, microalbuminuria, and troponin were high, but were accompanied by wide confidence intervals, reflecting considerable statistical imprecision. These effects are likely influenced by the smaller number of events in female participants, making it difficult to infer stable sex-specific relationships.

Across all groups, D-dimer and microalbuminuria demonstrated weak or null associations, indicating that systemic inflammation and renal microvascular changes may not be primary determinants of revascularization decisions in this clinical context.

Table 4: Gender-stratified and overall associations of inflammatory and cardiorenal biomarkers with the risk of revascularization

Gender/Group	*Biomarker*	*OR (95% CI)*	*p-value*	*q-value*
Female	Troponin	1.50 (0.70–2.90)	0.28	0.55
Female	NT-proBNP	0.70 (0.20–2.80)	0.65	0.71
Female	Microalbuminuria	1.20 (0.20–6.10)	0.80	0.80
Female	D-dimer	0.80 (0.40–1.70)	0.59	0.71
Male	NT-proBNP*	0.50 (0.30–0.90)	0.02	0.07
Male	Troponin	1.40 (1.00–2.00)	0.05	0.15
Male	Microalbuminuria	0.70 (0.30–1.50)	0.37	0.56
Male	D-dimer	0.80 (0.60–1.20)	0.28	0.55
All participants	NT-proBNP*	0.50 (0.30–0.90)	0.02	0.07
All participants	Troponin*	1.50 (1.10–2.10)	0.01	0.07
All participants	Microalbuminuria	0.80 (0.40–1.60)	0.51	0.67
All participants	D-dimer	0.80 (0.60–1.20)	0.32	0.55

* Statistically significant at $p < 0.05$.

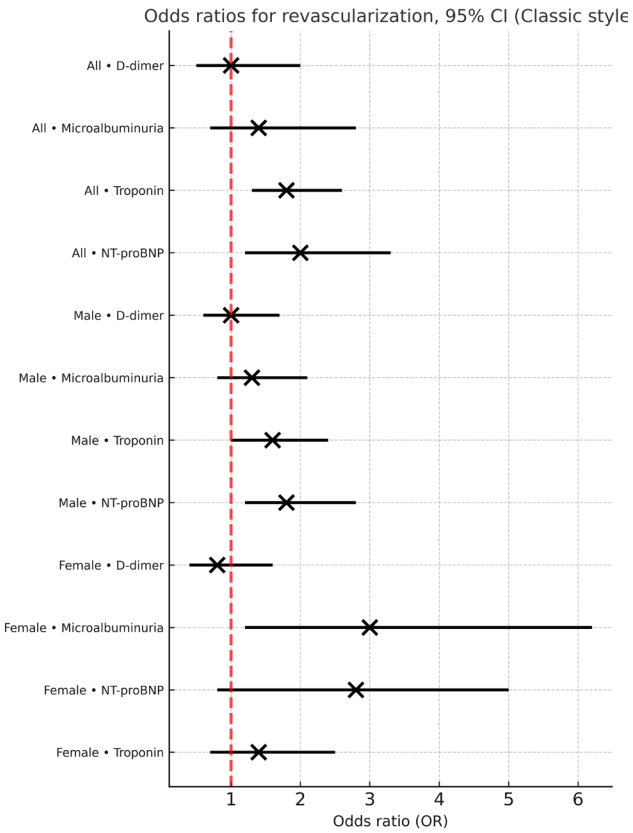

Figure 4: Odds ratios for revascularization across inflammatory and cardiorenal biomarkers, stratified by sex (95% CI).

Collectively, these results suggest that markers of myocardial injury (troponin) and hemodynamic stress (NT-proBNP) are more strongly associated with revascularization procedures than markers of inflammation or renal dysfunction. The findings may reflect clinical prioritization of ischemia-related biomarkers in decisions regarding invasive management.

Key Messages

- Troponin showed consistent positive association with revascularization, especially in men.
- NT-proBNP had elevated ORs but with wide uncertainty, reflecting low precision.
- D-dimer and microalbuminuria showed weak or null relationships with revascularization.

Findings indicate that myocardial injury markers—rather than inflammatory or renal biomarkers—drive revascularization risk.

Discussion

In this prospective cohort, we observed that individuals with elevated hs-cTnI exhibited a distinct clinical and biomarker profile characterized by older age, greater body size, and higher levels of inflammatory and cardiorenal biomarkers, consistent with prior evidence linking troponin to chronic cardiometabolic stress (10–12). These findings support the growing recognition that low-grade myocardial injury reflects cumulative structural and metabolic strain rather than acute hemodynamic perturbations (12,13). Elevated CRP, D-dimer, and NT-proBNP among hs-cTnI positive participants indicate parallel activation of inflammatory, thrombotic, and neurohormonal pathways, which may jointly contribute to subclinical myocardial damage (14,15). The lack of significant differences in blood pressure parameters suggests that traditional hemodynamic stress alone does not fully account for troponin elevation in this population (16).

Sex-stratified analyses revealed that troponin showed a stronger association with revascularization among men, whereas biomarker associations in women were highly variable, likely reflecting lower event rates and smaller sample size (18). Although NT-proBNP demonstrated elevated effect estimates across groups, wide confidence intervals indicate limited precision and caution should be exercised in interpreting these results (15,17). Importantly, D-dimer and microalbuminuria showed weak or null associations with revascularization, suggesting that systemic coagulation or renal microvascular dysfunction may not be major drivers of procedural decisions in this cohort (19).

The observed association between hs-cTnI and revascularization may reflect the integration of biomarker data into clinical decision-making, either explicitly or indirectly, through correlation with underlying coronary disease severity (11). In contemporary practice, revascularization strategies are increasingly influenced by anatomical assessments, ischemia burden, comorbidity profiles, and patient preferences, which may attenuate the influence of individual biomarkers on procedural outcomes (11,19). Nevertheless, our findings suggest that hs-cTnI may identify a subset of patients with subclinical myocardial injury who subsequently progress to clinically significant coronary disease (12).

From a mechanistic standpoint, the constellation of elevated hs-cTnI, CRP, and NT-proBNP is compatible with a chronic inflammatory-cardiometabolic phenotype characterized by endothelial dysfunction, adverse remodeling, and neurohormonal activation (14,17). Such phenotypes are increasingly recognized as drivers of diffuse atherosclerotic burden and heart failure in the absence of acute ischemic triggers (12,16). Incorporation of hs-cTnI into multimarker algorithms may therefore refine cardiovascular risk stratification, particularly in heterogeneous populations where traditional risk scores underperform (10,19).

Future studies should investigate whether biomarker trajectories—rather than single measurements—improve risk prediction and whether multimarker approaches can guide personalized preventive or therapeutic interventions (19). Integration with imaging, genomic, or metabolomic data may further elucidate biological pathways underlying subclinical myocardial injury and procedural risk (10,19).

hs-cTnI may capture latent myocardial injury preceding overt coronary disease (12). Metabolic and inflammatory pathways appear more relevant than hemodynamic stress (14,17). Sex-specific variability highlights the need for tailored risk frameworks (18).

Conclusion

In this prospective cohort, elevated hs-cTnI was associated with older age, greater body size, and higher levels of inflammatory and cardiorenal biomarkers, reflecting a phenotype of metabolic and systemic stress rather than hemodynamic impairment. hs-cTnI also demonstrated prognostic relevance for revascularization, particularly among men, although associations for other biomarkers were modest and imprecise. These findings support the utility of hs-cTnI as a marker of subclinical myocardial injury and cardiovascular risk in stable populations. Further studies are needed to validate these observations and clarify their clinical implications for personalized risk stratification.

Funding

This study was conducted without financial support from any funding agency, commercial entity, or institutional grant.

Ethics Approval

The study protocol was reviewed and approved by the Ethics Committee of the Acad. C. Abdullayev Scientific Research Institute of Cardiology, Baku, Azerbaijan.

All participants provided written informed consent prior to enrollment, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Conflict of Interest

The authors declare no conflicts of interest.

Acknowledgements

The authors acknowledge the staff of the Acad. C. Abdullayev Scientific Research Institute of Cardiology for their support in patient recruitment and data collection. The authors are grateful to all study participants.

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