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Glycemic control as a predictor of myocardial stress in patients with type 2 diabetes mellitus undergoing maintenance hemodialysis

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Abstract:

Aim. Patients with type 2 diabetes mellitus (T2DM) undergoing maintenance hemodialysis represent a population at extremely high cardiovascular risk. Chronic hyperglycemia contributes to myocardial injury through inflammation, oxidative stress, endothelial dysfunction, and pathological cardiac remodeling. N-terminal pro-brain natriuretic peptide (NT-proBNP) is a well-established biomarker of myocardial stress and cardiovascular risk; however, its relationship with glycemic control in patients undergoing hemodialysis remains insufficiently studied.

Objective. To assess the relationship between glycemic control and NT-proBNP levels in patients with T2DM and diabetic nephropathy undergoing maintenance hemodialysis.

Materials and Methods. This cross-sectional study included 60 patients with T2DM and diabetic nephropathy receiving maintenance hemodialysis (CKD stage 5D). According to glycated hemoglobin (HbA1c) levels, patients were divided into two groups: HbA1c $< 7.0\%$ (n=26) and HbA1c $\geq 7.0\%$ (n=34). NT-proBNP levels were assessed and compared between groups. Spearman correlation analysis was used to evaluate the association between HbA1c and NT-proBNP. Logistic regression analysis was performed to identify independent predictors of elevated NT-proBNP.

Results. Patients with HbA1c $\geq 7.0\%$ had significantly higher NT-proBNP levels compared with those with HbA1c $< 7.0\%$ (628.3 vs. 549.5 pg/mL; p=0.04). In the subgroup with poor glycemic control, a moderate positive correlation was observed between HbA1c and NT-proBNP (r=0.34; p=0.049). Logistic regression analysis identified HbA1c as an independent predictor of elevated NT-proBNP (OR=1.45; 95% CI 1.02–2.05; p=0.04). Based on these findings, a three-level cardiovascular risk stratification algorithm using HbA1c and NT-proBNP was proposed.

Conclusion. Poor glycemic control is associated with elevated NT-proBNP levels in patients with T2DM undergoing maintenance hemodialysis. HbA1c may serve as an independent predictor of myocardial stress and cardiovascular risk in this population. Combined assessment of HbA1c and NT-proBNP may improve cardiovascular risk stratification and support optimization of treatment strategies.

Keyword: cardiovascular risk, chronic kidney disease, HbA1c, NT-proBNP

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Introduction

Diabetes mellitus (DM) remains one of the most significant medical and social challenges in modern healthcare. According to the 10th edition of the International Diabetes Federation Diabetes Atlas, more than 537 million people worldwide were living with diabetes in 2021, and the global prevalence of the disease exceeded 10% among adults aged 20–79 years [1]. The prevalence of

diabetes is expected to continue increasing in virtually all regions of the world, making DM one of the major contributors to the global burden of non-communicable diseases.

Diabetic nephropathy is one of the most severe chronic complications of diabetes mellitus and remains the leading cause of end-stage chronic kidney disease (CKD). According to global epidemiological data, CKD affects approximately 15% of the general population and nearly every second patient with arterial hypertension and diabetes mellitus [2]. Diabetic kidney disease remains the leading cause of end stage renal disease (ESRD), accounting for approximately 50% of all ESRD cases in developed countries [3].

A similar upward trend in CKD prevalence has also been observed in the Republic of Uzbekistan. According to the national registry data for 2018, 107,659 patients with CKD (3,272.3 per 1 million population) were registered in the country, of whom 23,261 (21.6%; 707.0 per 1 million population) had diabetes-associated CKD. End-stage CKD was registered in 3,210 patients (13.8%; 97.6 per 1 million population), while 2,753 patients (11.8%; 83.7 per 1 million population) were receiving maintenance hemodialysis [4].

Patients with type 2 diabetes mellitus (T2DM) and end-stage CKD undergoing maintenance hemodialysis represent a population at extremely high cardiovascular risk. Cardiovascular disease remains the leading cause of mortality in patients with ESRD [5], despite advances in renal replacement therapy. The development of cardiovascular complications in this population is multifactorial and is driven by chronic inflammation, metabolic disturbances, arterial hypertension, anemia, oxidative stress, and the accumulation of uremic toxins [6]. In patients with T2DM, these pathophysiological mechanisms are further aggravated by chronic hyperglycemia, diabetic cardiomyopathy, impaired autonomic cardiac regulation, and increased formation of advanced glycation end products (AGEs), all of which contribute to progressive myocardial injury [7].

N-terminal pro–B-type natriuretic peptide (NT-proBNP) is one of the most informative biomarkers of myocardial stress, reflecting increased ventricular filling pressure and myocardial dysfunction. In patients undergoing maintenance hemodialysis, NT-proBNP concentrations are predictably elevated because the peptide is actively cleared by the kidneys, whose function is severely impaired in dialysis patients, complicating the interpretation of NT-proBNP levels solely as a marker of cardiac pathology [8]. Nevertheless, prospective studies have demonstrated that elevated NT-proBNP levels retain independent prognostic value and are associated with both cardiovascular and all-cause mortality in patients receiving chronic dialysis [9].

Several studies have also demonstrated that, in patients with diabetes, the combination of elevated NT-proBNP levels and poor glycemic control is independently and jointly associated with adverse cardiovascular outcomes and all-cause mortality [10].

Glycated hemoglobin (HbA1c) remains the standard marker of long-term glycemic control; however, its interpretation in patients with end-stage CKD undergoing dialysis is substantially limited. Erythropoiesis-stimulating agents, used in more than 90% of hemodialysis patients, may artificially lower HbA1c levels by accelerating erythrocyte turnover, while hemolysis induced by uremia or the dialysis procedure itself may further reduce HbA1c values, making conventional HbA1c targets less reliable in the dialysis population [11].

Furthermore, in patients with diabetic nephropathy, monitoring lipid metabolism parameters is of additional clinical importance, as dyslipidemia contributes to nephropathy progression both through atherosclerotic damage to renal vessels and direct nephrotoxic effects on vascular endothelium. This category of patients also remains at high risk for cardiovascular events [13].

At present, the relationship between glycemic control parameters, particularly HbA1c, and biomarkers of myocardial stress such as NT-proBNP in patients with T2DM and diabetic nephropathy undergoing maintenance hemodialysis remains insufficiently studied, while the available evidence remains largely fragmented. This highlights the need for further investigation into the role of HbA1c as a potential predictor of elevated NT-proBNP and, consequently, cardiovascular risk in this patient population, which formed the basis for the present study.

The aim of this study was to evaluate the association between glycemic control parameters and myocardial stress, assessed by NT-proBNP, in patients with type 2 diabetes mellitus undergoing maintenance hemodialysis.

Materials and Methods

This cross-sectional comparative study was conducted at the Republican Specialized Scientific and Practical Medical Center of Nephrology and Kidney Transplantation. The study included 60 patients with T2DM undergoing maintenance hemodialysis. According to glycated hemoglobin (HbA1c) levels, patients were stratified into two groups: Group 1 — HbA1c $< 7.0\%$ (n=26) and Group 2 — HbA1c $\geq 7.0\%$ (n=34).

The inclusion criteria were a confirmed diagnosis of T2DM, diabetic nephropathy as the cause of end-stage chronic kidney disease (CKD stage 5D), maintenance hemodialysis for at least 3 months, age > 18 years, and availability of complete clinical, laboratory, and echocardiographic data. Exclusion criteria included acute coronary syndrome within the preceding 3 months, decompensated chronic heart failure, severe cardiac arrhythmias, hemodynamically significant valvular heart disease, and concomitant conditions that could affect the interpretation of study results.

All patients received standard bicarbonate-based hemodialysis therapy three times weekly in accordance with current clinical practice guidelines. All participants underwent comprehensive clinical, laboratory, and instrumental evaluation. The assessed parameters included markers of glycemic control (HbA1c and glycated albumin), cardiac biomarkers (NT-proBNP and CK-MB), as well as routine laboratory parameters including hemoglobin, total protein, and serum albumin levels. Echocardiographic examination was performed according to a standardized protocol and included assessment of left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), stroke volume (SV), left ventricular ejection fraction (LVEF), cardiac chamber dimensions, and mean pulmonary artery pressure.

Statistical analysis was performed using IBM SPSS Statistics version 26.0. Normality of data distribution was assessed using the Shapiro–Wilk test. Quantitative variables are presented as median and interquartile range [Me (IQR)]. Between-group comparisons were performed using the Mann–Whitney U test. Associations between continuous variables were assessed using Spearman's rank correlation analysis. Binary logistic regression analysis was performed to identify independent predictors of elevated NT-proBNP levels. Results are presented as odds ratios (ORs) with 95% confidence intervals (95% CIs). A two-sided p-value < 0.05 was considered statistically significant.

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. The study protocol was approved by the local Ethics Committee (Approval No. 1/24-2437). Written informed consent was obtained from all participants prior to enrollment.

Results

A total of 60 patients with type 2 diabetes mellitus undergoing maintenance hemodialysis were included in the final analysis. According to HbA1c levels, patients were stratified into two groups: HbA1c $< 7.0\%$ (n=26) and HbA1c $\geq 7.0\%$ (n=34). The demographic, hemodynamic, laboratory, and echocardiographic characteristics of the study participants according to HbA1c levels are presented in Table 1.

The two groups were comparable in terms of demographic characteristics, including age, duration of diabetes, and duration of hemodialysis (all $p > 0.05$). Analysis of hemodynamic parameters revealed significant differences in blood pressure levels. Patients with HbA1c $< 7.0\%$ had significantly higher systolic blood pressure compared with those with HbA1c $\geq 7.0\%$ [160 (150–170) vs. 150 (140–160) mmHg, $p = 0.04$]. Similarly, diastolic blood pressure was significantly higher in the HbA1c $< 7.0\%$ group [100 (90–110) vs. 90 (80–100) mmHg, $p = 0.01$]. Heart rate was comparable between the groups ($p = 0.38$).

In contrast, parameters reflecting glycemic control differed significantly, with patients in the HbA1c $\geq 7.0\%$ group exhibiting significantly higher HbA1c and glycated albumin levels compared with those in the HbA1c $< 7.0\%$ group (both $p < 0.001$).

Among cardiac biomarkers, NT-proBNP concentrations were significantly higher in patients with HbA1c $\geq 7.0\%$ compared with those with HbA1c $< 7.0\%$ [628.3 (484–747) vs. 549.5 (433–652) pg/mL, $p = 0.04$]. In contrast, CK-MB levels did not differ significantly between the groups ($p = 0.79$).

No significant differences between-group differences were observed in echocardiographic parameters, including left ventricular end-diastolic volume, left ventricular end-systolic volume, stroke volume, and left ventricular ejection fraction (all $p > 0.05$).

Table 1. Baseline characteristics of patients according to HbA1c levels

Variable	**HbA1c** $<$ 7,0% **(n=26)**	**HbA1c** $\geq$ 7,0% **(n=34)**	**p**
Demographic characteristics			
Age, years	62,5 (55,8–68,4)	58,0 (53,8–64,0)	0,13
Duration of diabetes, years	12 (9,8–15,0)	14 (11,8–17,0)	0,12
Duration of hemodialysis, months	14,0 (8,4–19,4)	12,0 (8,0–14,0)	0,13
Hemodynamic parameters			
Systolic blood pressure, mmHg	160 (150–170)	150 (140–160)	0,04*
Diastolic blood pressure, mmHg	100 (90–110)	90 (80–100)	0,01*
Heart rate, beats/min	89 (80–90)	90 (80–97)	0,38
Markers of glycemic control			
HbA1c, %	6,4 (6,1–6,7)	8,3 (7,6–9,1)	$<$ 0,001*
Glycated albumin, %	16,0 (15,0–16,2)	23,0 (19,0–24,0)	$<$ 0,001*
Cardiac biomarkers			
NT-proBNP, pg/mL	549,5 (433–652)	628,3 (484–747)	0,04*
CK-MB, U/L	42,4 (32,5–47,9)	36,0 (32,5–47,9)	0,79
Echocardiographic parameters			
Left ventricular end-diastolic volume (LVEDV), mL	178 (165,8–189)	178 (167–186)	0,88
Left ventricular end-systolic volume (LVESV), mL	75,2 (62,1–78,8)	75,7 (66,9–78,5)	0,75
Stroke volume (SV), mL	107,3 (93,6–119,3)	102,3 (98,6–110,6)	0,65
Left ventricular ejection fraction (LVEF), %	57 (55–57)	54 (48–57)	0,56

*Note: Data are presented as median (interquartile range, IQR). *$p < 0.05$; ***$p < 0.001$. Between-group comparisons were performed using the Mann–Whitney U test.*

A moderate positive correlation between HbA1c and NT-proBNP levels was observed exclusively in patients with HbA1c $\geq$ 7.0% ($r = 0.34$, $p = 0.049$; Table 2), indicating that worsening glycemic control was associated with higher levels of myocardial stress in this subgroup. In contrast, no significant association was detected in patients with HbA1c $<$ 7.0% ($r = 0.15$, $p = 0.45$), suggesting the absence of a meaningful relationship between glycemic status and NT-proBNP under conditions of adequate glycemic control.

Table 2. Correlations between markers of glycemic control and cardiac biomarkers

Variable pair	**HbA1c** $\leq$ 7%	**p**	**HbA1c** $>$ 7%	**p**
HbA1c – NT-proBNP	r=0,15	0,45	r=0,34*	0,049
HbA1c – CK-MB	r=$-0,28$	0,16	r=$-0,10$	0,56
Glycated albumin – NT-proBNP	r=0,03	0,90	r=0,01	0,97
Glycated albumin – CK-MB	r=$-0,23$	0,26	r=$-0,06$	0,74

*Note: Data are presented as Spearman's rank correlation coefficients (r). *$p < 0.05$.*

As shown in Figure 1, binary logistic regression analysis identified HbA1c as the only independent predictor of elevated NT-proBNP levels (OR=1.45; 95% CI 1.02–2.05; $p = 0.04$). Systolic blood pressure demonstrated borderline significance (OR=1.03; 95% CI 1.00–1.06; $p = 0.05$), whereas age and duration of hemodialysis were not significantly associated with elevated NT-proBNP levels (all $p > 0.05$).

The overall model fit was acceptable (Hosmer–Lemeshow $\chi^2 = 8.42$; $p = 0.04$) .

The predictive performance of HbA1c for identifying elevated NT-proBNP levels was characterized by an AUC of 0.80 [95% CI: –], with a sensitivity of 78.3% and specificity of 61.5% at the optimal cut-off, yielding an overall accuracy of 68.3%.

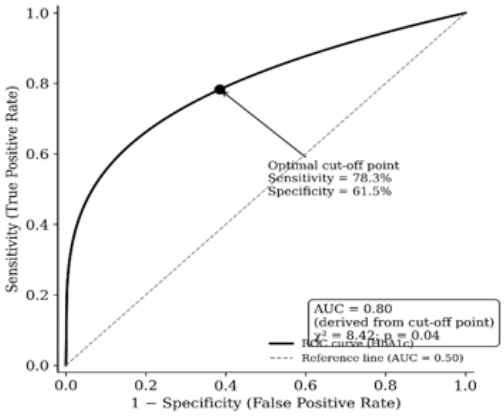


Figure 1. ROC curve of HbA1c as a predictor of elevated NT-proBNP levels

Proposed model for cardiovascular risk stratification

Based on the obtained findings, a three-level model for cardiovascular risk stratification in patients with type 2 diabetes mellitus undergoing maintenance hemodialysis was proposed (Table 3). The model incorporates sequential assessment of HbA1c and NT-proBNP levels using the median NT-proBNP value observed in the study population (590 pg/mL) as the threshold for risk categorization.

Table 3. Proposed model for cardiovascular risk stratification in patients with type 2 diabetes mellitus undergoing maintenance hemodialysis

Risk category	Criteria	Suggested clinical management
High risk	HbA1c $\geq$ 7,0% + NT-proBNP $>$ 590 pg/mL	Intensification of glycemic control. NT-proBNP monitoring every 3 months. Echocardiographic assessment every 6 months. Cardiologist consultation.
Intermediate risk	HbA1c $\geq$ 7,0% + NT-proBNP $\leq$ 590 pg/mL	Optimization of glycemic control. NT-proBNP monitoring every 6 months. Annual echocardiographic assessment.
Low risk	HbA1c $<$ 7,0% + NT-proBNP $\leq$ 590 pg/mL	Maintenance of adequate glycemic control. Annual NT-proBNP monitoring and echocardiographic assessment.

Note: The median NT-proBNP concentration in the study population was 590 pg/mL. Echocardiography was abbreviated as EchoCG.

At the first stage, HbA1c is assessed as a marker of long-term glycemic control. The second stage involves measurement of NT-proBNP, with values interpreted according to the predefined threshold of 590 pg/mL. At the third stage, the intensity of cardiovascular monitoring and follow-up is determined according to the assigned risk category.

Owing to its reliance on routinely available laboratory parameters, the proposed model may be readily applied in hemodialysis centers without the need for additional diagnostic procedures.

Discussion:

The present study demonstrated that glycated hemoglobin was independently associated with elevated NT-proBNP levels in patients with type 2 diabetes mellitus undergoing maintenance hemodialysis, with a moderate positive correlation observed specifically in the subgroup with inadequate glycemic control (HbA1c $\geq$ 7.0%). These findings support the hypothesis that chronic hyperglycemia contributes to myocardial stress in this population through mechanisms that may precede overt structural cardiac remodeling, consistent with the concept of diabetic cardiomyopathy, in which neurohormonal activation can occur independently of, or prior to, measurable changes in conventional echocardiographic indices such as ejection fraction or ventricular volumes [7].

Our results are broadly consistent with prior evidence linking HbA1c to cardiovascular risk beyond its traditional role as a glycemic marker. Jakubiak et al. demonstrated that glycated hemoglobin is associated with cardiovascular disease even in individuals without diagnosed diabetes, suggesting that HbA1c captures a dimension of vascular and myocardial risk that extends beyond glucose control per se [16]. Similarly, recent work on cardiac biomarkers in chronic kidney disease has emphasized that, despite confounding by reduced renal clearance, NT-proBNP retains meaningful prognostic value across the CKD spectrum, including in patients receiving dialysis [8]. Data from heart failure populations with impaired kidney function further support the notion that the relationship between natriuretic peptides and renal function is quantitative rather than purely confounding — natriuretic peptide concentrations remain informative once renal function is accounted for, rather than becoming uninterpretable [12]. Taken together with our findings, this suggests that the elevated NT-proBNP observed in the poorly controlled glycemic subgroup is unlikely to be fully explained by renal clearance alone.

Mechanistically, chronic hyperglycemia promotes the accumulation of advanced glycation end products (AGEs), which stiffen the myocardial extracellular matrix and impair diastolic relaxation, while simultaneously increasing oxidative stress and pro-inflammatory cytokine production (TNF-α, IL-6, IL-1β) that further compromise endothelial function [7]. In patients undergoing maintenance hemodialysis, these processes are superimposed on a milieu of chronic volume fluctuation and uremia-related endothelial injury, which may act synergistically to increase ventricular wall stress and natriuretic peptide secretion. This combination of mechanisms offers a plausible explanation for why NT-proBNP differed significantly between glycemic groups despite the absence of significant between-group differences in conventional echocardiographic parameters — the elevation in NT-proBNP may reflect an early, functional neurohormonal signal rather than established structural remodeling.

An unexpected finding was that patients with better glycemic control (HbA1c $< 7.0\%$) had significantly higher systolic and diastolic blood pressure than those with poorer control. This counterintuitive pattern should be interpreted cautiously given the cross-sectional design, which does not allow causal attribution. Several non-mutually-exclusive explanations may be considered: patients under closer clinical surveillance for hypertension may simultaneously receive more intensive glycemic management, generating a form of confounding by indication; alternatively, differences in interdialytic weight gain, residual diuresis, or antihypertensive regimens — none of which were captured in the present analysis — could independently influence both variables. Notably, systolic blood pressure retained borderline independent association with elevated NT-proBNP in the multivariable model (OR=1.03; 95% CI 1.00–1.06; $p = 0.05$), which is consistent with the well-established contribution of pressure and volume overload to natriuretic peptide release, and suggests that hemodynamic and glycemic pathways to myocardial stress in this population may be at least partially independent of one another.

Notably, glycated albumin — despite reflecting a shorter glycemic exposure window (approximately 2–3 weeks) compared with the $\sim$3-month window captured by HbA1c — showed no significant correlation with either NT-proBNP or CK-MB in either glycemic subgroup. This dissociation supports the interpretation that cumulative, longer-term hyperglycemic exposure, rather than short-term glycemic fluctuation, is more closely linked to chronic myocardial stress in this population, in line with previous observations that alternative glycemic markers do not always track cardiovascular biomarkers concordantly with HbA1c in CKD populations, where multiple non-glycemic factors (protein turnover, inflammation, AGE accumulation) can independently affect both glycated albumin and cardiovascular biomarker levels [14,15]. Given the well-documented limitations of HbA1c in advanced CKD — including its susceptibility to erythropoiesis-stimulating therapy and altered erythrocyte turnover — this finding does not eliminate the need for cautious interpretation, but it does suggest that, in this cohort, HbA1c outperformed glycated albumin as a correlate of myocardial stress.

From a clinical-utility standpoint, HbA1c demonstrated moderate discriminative performance for identifying elevated NT-proBNP (sensitivity 78.3%, specificity 61.5%, overall accuracy 68.3%; $\chi^2 = 8.42$; $p = 0.04$). While this performance is not sufficient to support HbA1c as a stand-alone diagnostic tool, it is compatible with its proposed role as a first-tier screening parameter within the

three-level cardiovascular risk stratification model presented in this study. Because both HbA1c and NT-proBNP are already routinely measured in many hemodialysis centers, the proposed model requires no additional infrastructure or cost, which may facilitate its integration into everyday clinical monitoring — particularly relevant in resource-limited dialysis settings such as those in Uzbekistan, where systematic cardiovascular risk stratification tools are not yet standardized.

Several limitations of the present study should be acknowledged. First, the cross-sectional, single-center design precludes causal inference and limits generalizability; the observed associations should be interpreted as hypothesis-generating rather than confirmatory. Second, the sample size (n=60) is relatively modest for multivariable logistic regression, and the lower bound of the confidence interval for the HbA1c–NT-proBNP association (OR=1.45; 95% CI 1.02–2.05) approached unity, indicating limited precision that should be resolved in larger cohorts. Third, multiple between-group comparisons were performed without formal correction for multiplicity; consequently, findings such as the blood pressure discrepancy between glycemic groups warrant confirmation in independent samples. Fourth, conventional echocardiographic parameters (ejection fraction, ventricular volumes) may lack sensitivity to detect early diastolic dysfunction; incorporation of tissue Doppler indices (E/e') or global longitudinal strain in future studies could clarify whether the neurohormonal signal captured by NT-proBNP precedes detectable structural or functional myocardial changes. Finally, residual confounding by dialysis-related factors — including interdialytic weight gain, timing of blood sampling relative to the dialysis session, dialysis adequacy, and erythropoiesis-stimulating agent dosage could not be fully excluded, as these variables were not systematically incorporated into the regression model.

Conclusions

Glycated hemoglobin was independently associated with elevated NT-proBNP levels in patients with type 2 diabetes mellitus undergoing maintenance hemodialysis, suggesting a relationship between long-term glycemic control and myocardial stress in this population.

The observed increase in NT-proBNP among patients with inadequate glycemic control occurred in the absence of significant differences in conventional echocardiographic parameters, indicating that neurohormonal and metabolic mechanisms may contribute to myocardial stress before the development of overt structural cardiac remodeling.

Based on the obtained findings, a model for cardiovascular risk stratification incorporating HbA1c and NT-proBNP was proposed. This approach may facilitate identification of patients at different levels of cardiovascular risk and support individualized cardiovascular monitoring strategies in the hemodialysis setting.

Further prospective studies are required to validate the proposed model and determine its clinical utility.

Authors' contribution

Conceptualization, L.D. and S.M.; methodology, L.D. and K.N.; formal analysis, K.N. and R.A.; investigation, S.M. and B.J.; resources, L.D. and S.M.; data curation, S.M. and B.J.; writing—original draft preparation, S.M. and K.N.; writing—review and editing, L.D., R.A. and S.M.; visualization, K.N. and B.J.; supervision, L.D. and R.A.; project administration, L.D. and S.M. All authors have read and agreed to the published version of the manuscript.

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Ethics approval

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. The study protocol was reviewed and approved by the local Ethics Committee (Approval No. 1/24-2437). Written informed consent was obtained from all participants prior to enrollment.

Consent for publication.

Written informed consent to participate in the study was obtained from all participants prior to enrollment. All patient data were handled confidentially and analyzed in anonymized form.

Data Availability Statement

The data supporting the findings of this study are not publicly available due to patient confidentiality and ethical considerations. De-identified data may be made available from the corresponding author upon reasonable request, subject to applicable ethical and institutional approval requirements.

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Conflict of interest

The authors declare no conflicts of interest related to this study. This research received no external funding; therefore, no external funder was involved in the study design, data collection, analysis, interpretation of results, manuscript preparation, or decision to publish.

Abbreviations

DM	Diabetes mellitus
T2DM	Type 2 diabetes mellitus
CKD	Chronic kidney disease
ESRD	End-stage renal disease
HbA1c	Glycated hemoglobin
NT-proBNP	N-terminal pro-B-type natriuretic peptide
CK-MB	Creatine kinase-MB isoenzyme
AGEs	Advanced glycation end products
LVEDV	Left ventricular end-diastolic volume
LVESV	Left ventricular end-systolic volume
SV	Stroke volume
LVEF	Left ventricular ejection fraction
OR	Odds ratio
CI	Confidence interval
IQR	Interquartile range
AUC	Area under the curve
ROC	Receiver operating characteristic
TNF-α	Tumor necrosis factor-alpha
IL-6	Interleukin-6
IL-1β	Interleukin-1 beta
EchoCG	Echocardiography

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