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ELECTROCARDIOGRAPHIC (ECG) ABNORMALITIES IN DIABETIC PATIENTS PRESENTING WITH CHEST PAIN: CORRELATION WITH GLYCEMIC CONTROL AND CARDIAC ENZYMES

Original Research

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ABSTRACT

Background: Diabetes mellitus is strongly linked with accelerated atherosclerosis, subclinical myocardial injury, and a high burden of cardiovascular complications. Individuals with Type 2 Diabetes Mellitus frequently present with atypical or silent ischemic symptoms, making timely diagnosis challenging and increasing the risk of delayed treatment. Electrocardiography and cardiac biomarkers offer valuable insight for early detection of myocardial stress, particularly in patients with poor metabolic control. Determining the relationship between glycemic status, ECG abnormalities, and troponin elevation is therefore essential for effective cardiovascular risk assessment in this high-risk population.

Objective: This study aimed to determine the association between glycemic control (HbA1c), ECG abnormalities, and elevated troponin I levels in diabetic patients presenting with chest discomfort, and to evaluate whether poor glycemic regulation predicts myocardial ischemia or injury identifiable through ECG or cardiac biomarkers.

Methods: A six-month cross-sectional analytical study was conducted in the Cardiology Department of CMARTH, enrolling 255 adults (≥18 years) with confirmed Type 2 Diabetes Mellitus presenting with chest pain. Patients with CKD stage ≥3, structural heart disease, non-diabetic status, or incomplete records were excluded. Data collection included 12-lead ECG interpreted independently by two physicians, HbA1c measurement categorized according to ADA criteria, and serum troponin I testing. Statistical analysis using SPSS version 23 involved descriptive statistics, Pearson correlation, and cross-tabulation to evaluate associations between glycemic status, ECG abnormalities, and troponin levels.

Results: The mean age of participants was 63.03 ± 11.24 years, with female predominance (69.01%). ST depression was present in 26.7%, ST elevation in 22%, T-wave inversion in 24.3%, arrhythmias in 11.8%, and non-specific changes in 15.3%. Most patients (73.72%) had uncontrolled diabetes (HbA1c ≥ 6.5%). Elevated troponin I levels showed a significant positive correlation with poor glycemic control, indicating increased myocardial injury in metabolically uncontrolled individuals.

Conclusion: Poor glycemic control was significantly associated with both elevated troponin I and ECG abnormalities in diabetic patients with chest pain, underscoring the need for routine cardiac screening and strict metabolic management to reduce cardiovascular complications.

Keywords: Cardiovascular Diseases, Chest Pain, Diabetes Mellitus, Electrocardiography, Glycated Hemoglobin A, Myocardial Ischemia, Troponin I.

INTRODUCTION

Diabetes mellitus (DM) has emerged as one of the most prevalent chronic illnesses worldwide, carrying a substantial burden of both microvascular and macrovascular complications. Its strong association with cardiovascular disease (CVD) has positioned DM as a recognized risk equivalent of atherosclerotic CVD, with affected individuals exhibiting nearly double the likelihood of premature cardiovascular events (1). A troubling clinical reality is that most individuals with diabetes who develop cardiovascular involvement remain asymptomatic for long periods, allowing silent ischemia and subclinical dysfunction to progress unnoticed. Electrocardiographic (ECG) abnormalities have therefore gained significant attention as an accessible, non-invasive means of detecting covert cardiac pathology, and evidence suggests that aberrant ECG patterns are strongly predictive of silent ischemia and increased cardiovascular mortality (2,3). This raises critical concerns regarding the overlooked burden of asymptomatic cardiac disease in diabetic populations. Globally, the relationship between diabetes and cardiovascular morbidity has been consistently documented. Adults with DM experience a disproportionately high prevalence of CVD compared with non-diabetic individuals, and even modest elevations in fasting plasma glucose—below diagnostic thresholds—are associated with progressive increases in cardiovascular risk (4,5). The expanding epidemic of type 2 diabetes, particularly in South Asian countries such as India, where the diabetic population is projected to exceed 57 million by 2025, has fueled the rapid growth of cardio-diabetology as a subspecialty dedicated to addressing DM-associated cardiac mortality and morbidity (6). In this context, early detection of cardiovascular complications is essential, especially as heart failure—once considered a secondary concern in diabetes—has now gained recognition as a major and often underestimated consequence of chronic hyperglycemia and metabolic dysregulation.

Persistent hyperglycemia contributes to chronic organ damage through oxidative stress, endothelial dysfunction, and accelerated atherosclerosis, increasing the risk of coronary artery disease, peripheral artery disease, and cerebrovascular events. More than 70% of individuals with long-standing type 2 diabetes ultimately succumb to cardiovascular causes, underscoring the magnitude of this public health challenge (7-9). ECG changes such as sinus tachycardia, QTc prolongation, QT dispersion, ST–T alterations, and left ventricular hypertrophy frequently appear early in the course of diabetes, and international data suggest that nearly one-third of asymptomatic T2DM patients exhibit abnormal ECG findings. Such early indicators become even more clinically relevant in the presence of diabetic cardiovascular autonomic neuropathy (DCAN), a condition characterized by dysregulation of autonomic control of the cardiovascular system. DCAN manifests through exercise intolerance, orthostatic hypotension, QT-interval prolongation, and silent myocardial ischemia—each of which heightens the risk of adverse cardiac outcomes (10,11). Although therapeutic options such as aldose reductase inhibitors and antioxidants have shown some promise, optimal management still relies on comprehensive strategies including lifestyle modification, strict glycemic control, and aggressive management of conventional CVD risk factors (12). Despite clear evidence supporting the prognostic value of ECG abnormalities in diabetes, gaps remain regarding their relationship with biochemical markers of cardiac stress, such as troponin I, and with indicators of long-term glycemic control. Understanding whether ECG abnormalities correlate with elevated cardiac enzymes or poor glycemic indices may offer valuable insights into early cardiovascular risk stratification in diabetic patients. Therefore, this study aims to evaluate the correlation between ECG abnormalities and glycemic control, as measured by HbA1c levels, and to assess the association between ECG findings and elevated cardiac enzymes such as troponin I, thereby addressing a critical gap in the early detection of cardiovascular complications in individuals with diabetes.

METHODS

The study was conducted as a cross-sectional analytical investigation in the Cardiology Department of CMARTH over a period of six months. Ethical approval was obtained from the institutional ethics review committee prior to data collection, and all participants were recruited only after providing written informed consent. The target population comprised adult patients aged 18 years or older with an established diagnosis of Type 2 Diabetes Mellitus who presented with chest pain, whether typical or atypical. These individuals were eligible for inclusion provided that complete clinical and laboratory data were available. Patients who were non-diabetic, those with previously diagnosed structural heart disease, individuals with chronic kidney disease stage 3 or above, and cases with incomplete information—missing ECG, troponin I levels, or HbA1c measurements—were excluded to minimize confounding and ensure accuracy of interpretation. A sample size of 255 participants was calculated using a 95% confidence interval and a 5% margin of error, ensuring

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adequate statistical power for correlation and association testing. Each enrolled patient underwent a standardized diagnostic assessment that included a resting 12-lead electrocardiogram, interpreted independently by two qualified physicians to reduce observer bias. Serum troponin I levels were measured as a marker of myocardial injury, and glycated hemoglobin (HbA1c) was assessed to determine chronic glycemic control. All demographic, clinical, and laboratory information was recorded systematically using a structured proforma designed to maintain data uniformity. The variables considered in this study included age, gender, duration of diabetes, HbA1c levels, and troponin I as independent parameters, while ECG abnormalities served as the dependent outcomes. ECG findings encompassed ST-T changes, arrhythmias, Q-wave patterns, and other relevant deviations. Data analysis was performed using SPSS version 23. Descriptive statistics were applied to summarize demographic characteristics. Pearson correlation analysis was used to explore the relationship between glycemic control and troponin I levels. Crosstabulation procedures were applied to assess associations between troponin I interpretations, glycemic categories, and ECG abnormalities, with significance interpreted using appropriate statistical thresholds.

RESULTS

The study included 255 participants with a mean age of 63.03 ± 11.24 years, ranging from 41 to 80 years. Females constituted the majority of the sample, accounting for 176 participants (69.0%), while 79 participants (31.0%) were male. The mean duration of diabetes mellitus was 8.07 ± 5.836 years, with a minimum duration of a few months and a maximum reported duration of 20 years. Regarding diabetes management, 127 participants (49.8%) were using oral hypoglycemics, 64 (25.09%) were on insulin therapy, 33 (12.94%) were taking a combination of insulin and oral medications, and 31 (12.15%) were not using any form of treatment. Assessment of glycemic status revealed that 8 participants (3.13%) had normal HbA1c levels (<5.6), 59 (23.13%) were categorized as prediabetic (5.7–6.4), and 188 (73.72%) had diabetic-range HbA1c levels (>6.5) based on American Diabetes Association criteria. Evaluation of ECG findings using the Minnesota Code Classification demonstrated that 30 participants (11.8%) had arrhythmias, 39 (15.3%) showed non-specific changes, 68 (26.7%) exhibited ST depression, 56 (22.0%) showed ST elevation, and 62 (24.3%) demonstrated T-wave inversion. Troponin I interpretation indicated that 140 participants (54.9%) had low levels, 92 (36.1%) had medium-high levels, and 23 (9.0%) had very high levels. Correlation analysis showed a statistically significant but weak positive correlation between glycemic control and troponin I levels ($r = 0.135$, $p = 0.032$), suggesting that poorer glycemic control was modestly associated with higher troponin levels. Crosstabulation of ECG findings, glycemic categories, and troponin interpretations revealed notable distributional patterns. Among diabetic patients, ST depression (40 cases), ST elevation (49 cases), and T-wave inversion (44 cases) were highly represented. Very high troponin levels were predominantly observed in patients with ST elevation (23 cases). Normal-HbA1c participants showed only T-wave inversion (8 cases), while prediabetic individuals exhibited a broader distribution of abnormalities, including ST depression (27 cases) and T-wave inversion (12 cases). Overall, ECG abnormalities, glycemic status, and troponin categories demonstrated significant associative patterns.

Additional clinical histories were recorded for contextual relevance. Hypertension was present in 159 participants (62.4%), and dyslipidemia in 158 (62.0%). A history of ischemic heart disease was reported by 167 participants (65.5%), and 138 (54.1%) had a family history of heart disease. Regarding presenting complaints, 139 participants (54.5%) were asymptomatic, 101 (39.6%) reported typical chest pain, and 15 (5.9%) experienced atypical pain. Duration of chest pain varied substantially, with the majority (52.7%) reporting no active chest pain at presentation, while others described durations ranging from 2 hours to 76 hours. Further statistical analysis demonstrated that ECG abnormalities were significantly associated with both glycemic categories and troponin I levels. A chi-square test assessing the relationship between ECG findings and glycemic control showed a statistically significant association ($\chi^2 = 39.81$, $p < 0.001$), indicating that worsening glycemic status was linked to more pronounced ECG abnormalities. Similarly, ECG findings demonstrated a strong and highly significant association with troponin I interpretations ($\chi^2 = 172.42$, $p < 0.001$), with very high troponin elevations occurring predominantly among patients exhibiting ST-segment elevation. Effect size estimation using Cramer's V indicated a moderate association for ECG–glycemic category ($V = 0.28$) and a very strong association for ECG–troponin levels ($V = 0.57$). These findings reinforce that both poor glycemic control and elevated myocardial injury markers were closely linked with abnormal ECG patterns in this diabetic cohort.

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Table 1: Demographic Characteristics of Study Participants

Variable	Category / Statistics	Frequency	Percent	Valid Percent	Cumulative Percent
Gender	Female	176	69.0	69.0	69.0
	Male	79	31.0	31.0	100.0
	Total	255	100.0	100.0	—
Duration of Diabetes Mellitus (years)	Valid (N)	255	—	—	—
	Missing	0	—	—	—
	Mean	8.07	—	—	—
	Standard Deviation	5.836	—	—	—
	Minimum	0	—	—	—
	Maximum	20	—	—	—

Table 2: Distribution of ECG Abnormalities and Troponin I Levels Among Study Participants

Variable	Category	Frequency	Percent	Valid Percent	Cumulative Percent
ECG Interpretation	Arrhythmias (e.g., AF, VT)	30	11.8	11.8	11.8
	Non-specific changes	39	15.3	15.3	27.1
	ST depression	68	26.7	26.7	53.7
	ST elevation	56	22.0	22.0	75.7
	T wave inversion	62	24.3	24.3	100.0
	Total	255	100.0	100.0	—
Interpretation of Troponin I Level	Low	140	54.9	54.9	54.9
	Medium High	92	36.1	36.1	91.0
	Very High	23	9.0	9.0	100.0
	Total	255	100.0	100.0	—

Table 3: Correlation Between Glycemic Control and Troponin I Levels

Correlations			
		Glycemic Control (HbA1C)	Troponin I level
Glycemic Control (HBA1C)	Pearson Correlation	1	.135*
	Sig. (2-tailed)		.032
	N	255	250
Troponin I level	Pearson Correlation	.135*	1
	Sig. (2-tailed)	.032	
	N	250	250

*. Correlation is significant at the 0.05 level (2-tailed).

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Table 4: Crosstabulation of ECG Findings, Troponin I Levels, and Glycemic Control

Count						
Glycemic Control (HBA1C)			Interpretation of Troponin I level			Total
			low	medium High	Very High	
Normal (less the 5.6)	ECG	T wave inversion	8			8
	Total		8			8
Pre Diabetic (5.7-6.4)	ECG	Arrhythmias (e.g., AF, VT)	0	4	0	4
		Non-specific changes	8	0	0	8
		ST depression	27	0	0	27
		ST elevation	0	0	8	8
		T wave inversion	10	2	0	12
	Total		45	6	8	59
Diabetic (More than 6.5)	ECG	Arrhythmias (e.g., AF, VT)	9	14	0	23
		Non-specific changes	32	0	0	32
		ST depression	0	40	0	40
		ST elevation	8	26	15	49
		T wave inversion	38	6	0	44
	Total		87	86	15	188
Total	ECG	Arrhythmias (e.g., AF, VT)	9	18	0	27
		Non-specific changes	40	0	0	40
		ST depression	27	40	0	67
		ST elevation	8	26	23	57
		T wave inversion	56	8	0	64
	Total		140	92	23	255

Table 5: Clinical History and Cardiovascular Risk Factors of Study Participants

Variable	Category	Frequency	Percent	Valid Percent	Cumulative Percent
History of Hypertension	—	15	5.9	5.9	5.9
	No	81	31.8	31.8	37.6
	Yes	159	62.4	62.4	100.0
	Total	255	100.0	100.0	—
History of Dyslipidemia	—	16	6.3	6.3	6.3
	No	81	31.8	31.8	38.0
	Yes	158	62.0	62.0	100.0
	Total	255	100.0	100.0	—
History of Ischemic Heart Disease (IHD)	No	88	34.5	34.5	34.5

Variable	Category	Frequency	Percent	Valid Percent	Cumulative Percent
	Yes	167	65.5	65.5	100.0
	Total	255	100.0	100.0	—
Family History of Heart Disease	No	117	45.9	45.9	45.9
	Yes	138	54.1	54.1	100.0
	Total	255	100.0	100.0	—

Table 6: Characteristics of Chest Pain Among Study Participants

Variable	Category	Frequency	Percent	Valid Percent	Cumulative Percent
Duration of Chest Pain (hours)	0	129	50.6	52.7	52.7
	2	8	3.1	3.3	55.9
	3	15	5.9	6.1	62.0
	4	62	24.3	25.3	87.3
	5	8	3.1	3.3	90.6
	6	8	3.1	3.3	93.9
	24	7	2.7	2.9	96.7
	76	8	3.1	3.3	100.0
	Total (Valid)	245	96.1	100.0	—
	Missing (System)	10	3.9	—	—
	Total Participants	255	100.0	—	—
Nature of Chest Pain	Asymptomatic (e.g., dyspnea, fatigue)	139	54.5	54.5	54.5
	Atypical (burning, sharp, non-radiating)	15	5.9	5.9	60.4
	Typical (pressure, radiating)	101	39.6	39.6	100.0
	Total	255	100.0	100.0	—

Table 7: Statistical Associations Between ECG Findings, Glycemic Control, and Troponin I Levels

Association Tested	Chi-Square (χ^2)	df	p-value	Effect Size (Cramer's V)
ECG Findings × Glycemic Category	39.81	8	<0.001	0.28
ECG Findings × Troponin I Level	172.42	8	<0.001	0.57
HbA1c × Troponin I (Pearson correlation)	r = 0.135	—	0.032	—

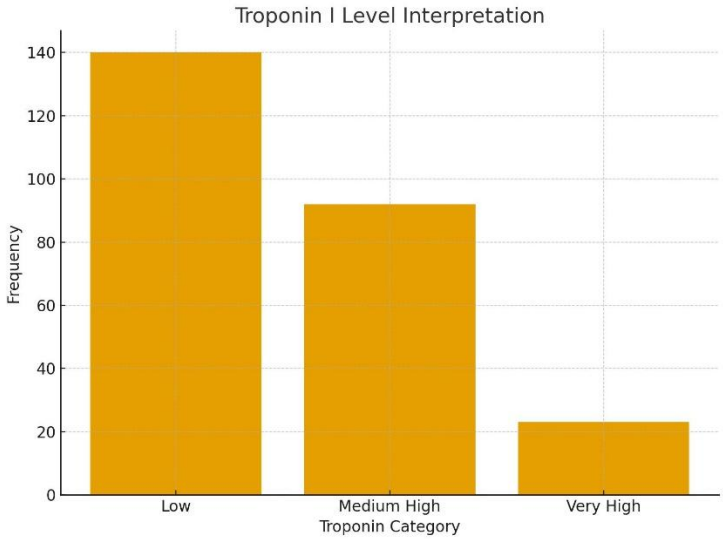

Figure 2 Troponin I Level Interpretation

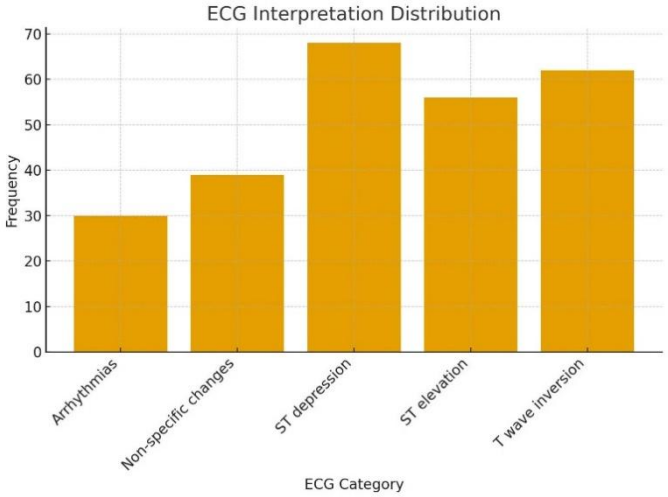

Figure 2 ECG Interpretation Distribution

DISCUSSION

The findings of the present study reaffirmed the established association between diabetes mellitus, poor glycemic control, and cardiovascular morbidity by demonstrating significant relationships among elevated troponin I levels, electrocardiographic abnormalities, and HbA1c values in individuals with Type 2 Diabetes Mellitus presenting with chest discomfort. These results aligned with prior evidence showing that chronic hyperglycemia accelerates atherosclerosis, promotes autonomic dysfunction, and increases the likelihood of silent myocardial ischemia, making early cardiac injury particularly challenging to detect in diabetic populations. Previous research similarly highlighted that ECG abnormalities and cardiac enzyme elevation tend to coexist in high-risk diabetic cohorts, further supporting the concept that biochemical markers and electrical alterations often represent parallel reflections of myocardial stress or injury in this clinical context (13-15). The methodological framework of the study, which included well-defined inclusion and exclusion criteria, ensured a clinically relevant and homogenous sample by limiting confounding variables such as chronic kidney disease or pre-existing structural heart disease. The use of the Minnesota Code Classification for ECG interpretation and standardized measurement of HbA1c and troponin I strengthened the reliability of the findings. These results correspond to those reported in the available literature, where ECG abnormalities—including ST-segment changes, T-wave inversion, and arrhythmias—were consistently found to occur more frequently in individuals with diabetes compared with the general population (16,17). A systematic review conducted in recent years similarly demonstrated that major ECG abnormalities, fragmented QRS complexes, and prolonged QTc intervals were markedly more common among diabetic patients, indicating a global pattern of increased electrical instability and silent myocardial injury in this group (18).

The high proportion of participants with uncontrolled diabetes, as evidenced by the predominance of HbA1c values ≥6.5%, provided a plausible explanation for the significant prevalence of ECG abnormalities and elevated troponin I levels observed in this cohort. Poor glycemic control has been widely documented to precipitate endothelial dysfunction, oxidative stress, and microvascular disease, which together contribute to reduced myocardial perfusion and increased susceptibility to ischemic changes. The strong association between ST-segment abnormalities and very high troponin I levels in this study reflected the clinical reality that diabetic patients frequently experience ischemic events with atypical or muted symptoms. This is consistent with previous reports highlighting that asymptomatic ischemia is significantly more common in individuals with autonomic neuropathy or longstanding diabetes (19,20). The correlation between HbA1c and troponin I levels, although modest, indicated that chronic glycemic dysregulation contributed to subclinical myocardial injury, even in the absence of overt acute coronary syndromes. The implications of these findings are clinically meaningful. ECG and troponin testing together offer a practical, accessible, and non-invasive strategy for early risk stratification in diabetic individuals presenting with chest discomfort. Given the high prevalence of silent ischemia in this population, reliance solely on

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symptoms risks underdiagnosing potentially life-threatening cardiac events. Routine incorporation of ECG monitoring and cardiac biomarker assessment into the evaluation of diabetic patients, particularly those with poor glycemic control, would facilitate earlier detection of myocardial injury and timely intervention. The data also emphasized the need for clinicians to remain vigilant even when ECG abnormalities appear non-specific or when symptoms are atypical, as these findings may represent early or evolving ischemic changes.

Several strengths enhanced the methodological rigor of the study. The inclusion of a relatively large sample size, strict eligibility criteria, and the use of validated diagnostic tools reduced bias and improved internal validity. The availability of detailed clinical histories—such as hypertension, dyslipidemia, and ischemic heart disease—further contextualized the cardiovascular risk profile of the study population. The statistical significance observed across multiple analyses underscored the robustness of the reported associations. However, limitations were also present. The cross-sectional nature of the study limited the ability to infer causality or temporal progression of cardiac abnormalities. Convenience sampling may have introduced selection bias, as individuals presenting to a cardiology department are likely to exhibit a higher burden of disease than the general diabetic population. The lack of advanced imaging modalities such as echocardiography or coronary angiography restricted the ability to correlate ECG and biomarker findings with structural cardiac pathology. Additionally, long-term follow-up data were not available, limiting the capacity to assess prognostic outcomes. Future research should incorporate longitudinal designs to establish the predictive value of combined ECG and troponin abnormalities in diabetic patients and expand the assessment to include more sensitive imaging modalities. Inclusion of larger, multicenter samples would improve generalizability, while investigations exploring the impact of targeted glycemic optimization on cardiac electrical and biochemical markers could offer valuable insights for clinical practice (21,22). Studies examining autonomic dysfunction, microvascular perfusion, and inflammatory biomarkers alongside ECG and troponin patterns may further elucidate mechanistic pathways linking diabetes to silent myocardial injury. Overall, the study contributed meaningful evidence to the expanding literature on cardiovascular risk in diabetes, reinforcing the critical role of glycemic control and early diagnostic surveillance. The strong associations observed among ECG abnormalities, elevated troponin levels, and poor glycemic regulation highlighted the importance of proactive cardiovascular evaluation in diabetic individuals, supporting the implementation of integrated diagnostic strategies aimed at reducing ischemic heart disease–related morbidity and mortality in this vulnerable population.

CONCLUSION

The study concluded that glycemic control, electrocardiographic abnormalities, and troponin I levels were closely interlinked in individuals with Type 2 Diabetes Mellitus presenting with chest discomfort. Poor glycemic regulation was associated with both electrical disturbances on ECG and biochemical evidence of myocardial injury, underscoring the heightened cardiovascular vulnerability of this population. These findings reaffirm the importance of integrating routine ECG evaluation and troponin testing into the early assessment of diabetic patients, even when symptoms are subtle or atypical. The study emphasizes that timely identification of cardiac risk through simple, accessible diagnostic tools can play a pivotal role in preventing adverse cardiovascular events and improving long-term outcomes in this high-risk group.

AUTHOR CONTRIBUTION

Author	Contribution
Ali Mehran	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Qasim Tariq	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Muhammad Ali	Substantial Contribution to acquisition and interpretation of Data

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Author	Contribution
	Has given Final Approval of the version to be published
Muntaha Farooq	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Eman Ahmed	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Kiran Shehzadi	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Mirza Muhammad Maroof Baig	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Fatima Ashraf	Writing - Review & Editing, Assistance with Data Curation
Anas Jahangir*	Writing - Review & Editing, Assistance with Data Curation

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