Zagazig University Medical Journal
www.zumj.journals.ekb.eg

https://doi.org/10.21608/zumj.2025.414710.4118 Volume 31, Issue 11 November. 2025

Manuscript:ID ZUMJ-2508-4118
DOI:10.21608/zumj.2025.414710.4118

Original Article

Frontal QRS-T Angle as a Non-invasive Predictor of the Presence of Coronary Slow Flow

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Submit date:16-8-2025
Revise date:31-8-2025
Accept date:16-9-2025

ABSTRACT

Background: Coronary slow flow (CSF) has been documented in roughly 1–7% of individuals undergoing coronary angiography and is recognized for its potential to be clinically significant. The frontal QRS–T angle [f(QRS–T)] is an easily obtainable electrocardiographic index, that provides an indicator of the heart's electrical stability. This study explored whether an abnormal f(QRS–T) angle on a standard ECG could serve as a non-invasive indicator of CSF.

Methods: In this cross-sectional investigation, 90 adult patients were scheduled for coronary angiography at Zagazig University Hospital were assessed. Comprehensive clinical, laboratory, and imaging evaluations were performed, and f(QRS–T) angle was assessed. According to corrected TIMI frame count (cTFC) values, participants were classified as having either CSF or normal coronary flow, with CSF defined by a cTFC exceeding 27 frames.

Results: Of the 90 patients studied (45 with CSF and 45 with normal flow), no significant variations emerged as regards demographic, clinical, or laboratory characteristics between both groups. Although the mean f(QRS–T) angle was revealed to be higher among the CSF group, with non-statistically significant variation (72.36° vs. 60°; $p = 0.075$). Although the QRS–T angle did not prove to be a dependable marker for predicting CSF, patients with several concurrent cardiovascular risk factors demonstrated notably greater angle values.

Conclusion: Although the f(QRS–T) angle on its own is not a consistent indicator of CSF, an increased angle seems to correlate with the accumulation of multiple cardiovascular risk factors.

Keywords: Coronary Slow Flow; Frontal QRS-T Angle;Non-invasive Predictor;Coronary Angiography.

INTRODUCTION

The entity now recognized as coronary slow flow (CSF) was initially documented in 1972, describing patients who presented with angina-like symptoms and ischemic changes on the electrocardiogram, yet demonstrated angiographically normal epicardial arteries in which contrast movement was unusually sluggish [1].CSF is identified in roughly 1–7% of those undergoing diagnostic coronary angiography [2]. Although its exact cause remains unresolved, current hypotheses implicate subtle vascular inflammation, impaired endothelial performance, and

disturbances within the coronary microcirculation [3,4].A TIMI flow grade of 2 provides a semi-quantitative means of describing the phenomenon [5,6]. For a more objective and reproducible determination, the cTFC is employed, with a value exceeding 27 frames serving as the diagnostic threshold [7,8]. Contrary to early perceptions that CSF may be harmless, research has linked it to malignant ventricular arrhythmias as well as the elevated likelihood of complications and subsequent sudden cardiac death [9].The f(QRS–T) angle, quantified by measuring the angular difference between ventricular depolarization and repolarization vectors, is readily accessible in standard 12-lead ECG reports. It serves as a simpler counterpart to the spatial QRS–T angle, that although technically more complex, provides important insights into myocardial electrical instability [10]. An angle above 90 degrees signals greater heterogeneity in electrical recovery and has been correlated with higher arrhythmic risk and poorer prognoses, particularly in individuals who have coronary artery disease [11–13].While the f(QRS–T) angle is well recognized for prognostic evaluation of various cardiac conditions, its predictive value in CSF has not been clearly defined. Few studies have addressed its relationship with CSF or the effect of multiple cardiovascular risk factors on this parameter. This gap underscores the need for targeted research combining ECG assessment with angiographic confirmation to determine its role as a simple, non-invasive marker in this setting. Although its prognostic role is established in other cardiovascular conditions, the ability of a widened f(QRS–T) angle to signal the presence of CSF remains inadequately explored.Although ECG-based markers like the frontal QRS-T angle remain underexplored in CSF research, recent studies have investigated other non-invasive biomarkers. For instance, elevated serum miRNA-22 has demonstrated promising diagnostic efficacy, with an AUC of 0.83 in differentiating CSF from normal coronary flow.Hematologic and inflammatory parameters such as red cell distribution width (RDW), mean platelet volume (MPV), platelet-to-lymphocyte ratio (PLR), and neutrophil-lymphocyte ratio (NLR) have also been identified as significant predictors of CSF [13]. The present investigation was designed to determine whether an abnormally broad f(QRS–T) angle recorded before coronary angiography could function as a non-invasive indicator for CSF.

METHODS

We performed this cross-sectional research in the Cardiology Department of Zagazig University Hospitals. The study period extended from February 2023 to February 2024, during which 90 consecutive adult patients scheduled for coronary angiography were recruited. Prior to enrollment, all participants signed informed consent forms prior to enrollment. The study protocol received approval from the Institutional Review Board of Zagazig University (ZU-IRB#9151/16-1-2022), and all procedures were carried out in accordance with the ethical guidelines of the Declaration of Helsinki [14].

Eligibility Criteria ,Participants of either gender with 18 years or older were recruited in the study if they were undergoing diagnostic coronary angiography for clinical indications. Exclusion criteria encompassed: acute

coronary syndromes; ; significant valvular heart disease; symptomatic heart failure, documented coronary artery spasm; congenital or acquired coronary anomalies such as severe stenosis or embolism; prior coronary revascularization procedures; advanced chronic kidney disease requiring renal replacement therapy; atrial arrhythmias (including atrial fibrillation, flutter, or tachycardia); cerebrovascular accidents; active systemic infections; major electrolyte disturbances; malignancy; the presence of artificial pacemakers; bundle branch block; or technically inadequate ECG tracings that compromised accurate measurement.

Electrocardiographic Analysis

All ECGs were acquired using a MAC 2000 electrocardiographic system (GE Medical Systems, Milwaukee, WI, USA). To ensure precision, recordings were digitized and magnified to 400× using specialized software. Standard calibration settings were maintained: an amplitude of 10 mm/mV, a paper speed of 25 mm/s, and a filter range of 0.15–100 Hz. Measurements were taken from lead II. The f(QRS–T) angle was automatically generated by the ECG software as the numerical difference between the QRS axis and the T-wave axis, and this was subsequently confirmed through manual calculation using the formula: $axis = tan^{-1}(\frac{Amplitude\ in\ aVF}{Amplitude\ in\ I})$ then calculating the difference. Angles >90° had considered abnormal [15].

Echocardiography

Transthoracic echocardiography was performed on all study subjects with a Vivid S5 echocardiographic system (GE Vingmed Ultrasound AS, Horten, Norway). The protocol comprised evaluation of left ventricular systolic performance (both global and regional), structural and functional assessment of the cardiac valves, and routine chamber dimension analysis. Quantification of left ventricular ejection fraction was carried out using the modified Simpson's biplane method [16].

Coronary Angiography and TIMI Frame Count

Coronary angiographic studies were carried out through either radial or femoral access, employing the Siemens Artis Zee imaging system (Siemens Medical Solutions, Erlangen, Germany). Subsequent image evaluation was carried out using a Siemens Axiom workstation. Coronary flow was assessed using the TIMI frame count technique [17], in which the number of cine frames needed for contrast dye to travel from the coronary ostium to a predefined distal reference point was determined for each principal epicardial artery. To account for its longer anatomical course, the LAD frame count was standardized by dividing by 1.7 [18]. A corrected TIMI frame count (cTFC) exceeding 27 frames was considered diagnostic of CSF [18–20].

Group Stratification: Patients were classified into two primary groups: CSF group: cTFC > 27 frames. Normal flow group: cTFC ≤ 27 frames. Each group was further subdivided based on the f(QRS–T) angle into: Normal angle: ≤ 90 degrees. Increased angle: > 90 degrees.

Statistical Analysis

All analyses were undertaken using IBM SPSS Statistics version 26 (Chicago, IL, USA). Continuous data were described as mean values with standard deviations and assessed between groups via the independent Student's t-test. Categorical variables were represented by absolute numbers and percentages, with statistical differences examined through Chi-

square analysis or Fisher's exact test when required. For non-parametric correlations, Spearman's rank correlation was applied. A p-value < 0.05 on a two-tailed test was considered to indicate statistical significance.

RESULTS

Ninety participants were included in the study, with 45 who were diagnosed with coronary slow flow and 45 demonstrating normal coronary flow, validated through cTFC assessment. As shown in Table 1 and Supplementary Figure 1, both groups exhibited similar baseline demographics—including age, gender distribution, and BMI as well as comparable prevalence of hypertension, diabetes mellitus, smoking, and dyslipidemia (all p > 0.05).Both groups didn't show statistical significant variations as regards hematological parameters—involving hemoglobin concentration, white blood cell count, and platelet count as well as in biochemical indices, including serum creatinine, uric acid, and electrolytes (sodium, potassium). Likewise, glycated hemoglobin (HbA1c), components of the lipid profile (total cholesterol, triglycerides, LDL cholesterol), and cardiac biomarkers such as CK-MB and troponin showed comparable values across both cohorts (Table 2 & Supplementary Figure2). With respect to the frontal QRS–T angle, no statistically significant variation was detected between patients in the CSF group and those with normal coronary flow (Table 3 & Supplementary Figure 3). Re-analysis with the non-parametric Mann–Whitney U test confirmed the absence of a statistically significant difference in QRS-T angle between the two groups (p = 0.081), supporting the robustness of our findings.Measurements of interventricular septal thickness (IVST), posterior wall thickness (PWT), left ventricular mass index (LVMI), left ventricular ejection fraction (LVEF), left atrial (LA) diameter, left ventricular end-diastolic diameter (LVEDD), and left ventricular end-systolic diameter (LVESD) were comparable between the two study groups, with no statistically significant variations detected (Table 4& Supplementary Figure 4).A f(QRS-T) angle exceeding 90° did not reliably predict coronary slow flow, showing 31.11% sensitivity, 93.33% specificity, a positive predictive value (PPV) of 82.4%, and a negative predictive value (NPV) of 57.5% (p = 0.091, AUC = 0.602). In the CSF group, QRS–T angle values were significantly greater in patients with three to four risk factors compared with those having none, one, or two risk factors (p = 0.04, 0.012, and 0.011, respectively). No difference was detected between the subgroups with no risk factors and those with one or two. Among individuals with normal coronary flow, QRS–T angle measurements were similar across all four subgroups (Table 5, Figure 1).

Table 1: Demographic data and Comorbidities of the groups (n=90)

		Normal coronary flow group (n=45)	Coronary Slow flow group (n=45)	P value
Age (years)	Mean ± SD	60.11 ± 7.57	58.67 ± 9.07	0.414
	Range	43 - 73	32 - 71	
Sex	Male	26 (57.78%)	22 (48.89%)	0.526
	Female	19 (42.22%)	23 (51.11%)	
Diabetes mellitus		28 (62.22%)	21 (46.67%)	0.138

Hypertension	**25 (55.56%)**	**24 (53.33%)**	**0.832**
Smoking	**28 (62.22%)**	**27 (60%)**	**0.829**

Table 2: Labo2wratory data of the groups (n=90)

		Normal coronary flow group (n=45)	Coronary Slow flow group (n=45)	P value
Hemoglobin (g/dl)	**Mean ± SD**	**13.41 ± 2.1**	**14.14 ± 1.54**	**0.061**
	Range	**10 - 16.92**	**11.38 - 16.73**	
White blood cells (10^9/ L)	**Mean ± SD**	**9.22 ± 2.66**	**10.11 ± 2.72**	**0.118**
	Range	**5.21 - 13.84**	**5.88 - 14.68**	
Platelet (10^9/ L)	**Mean ± SD**	**219.33 ± 73.6**	**241.85 ± 68.97**	**0.138**
	Range	**112.98 - 350.31**	**128.73 - 333.76**	
Creatinine (µmol/L)	**Mean ± SD**	**81.84 ± 19.41**	**89.22 ± 44.42**	**0.310**
	Range	**50.6 - 115.7**	**28.3 - 159.9**	
Uric acid (mmol/L)	**Mean ± SD**	**0.35 ± 0.13**	**0.34 ± 0.1**	**0.736**
	Range	**0.11 - 0.57**	**0.1 - 0.5**	
Sodium (mmol/L)	**Mean ± SD**	**137.44 ± 2.8**	**136.67 ± 2.78**	**0.190**
	Range	**134 - 142**	**132 - 140**	
Potassium (mmol/L)	**Mean ± SD**	**4.15 ± 0.4**	**4.28 ± 0.51**	**0.198**
	Range	**3.5 - 4.8**	**3.5 - 5**	
HBA1c (%)	**Mean ± SD**	**6.89 ± 2.26**	**6.74 ± 2.38**	**0.755**
	Range	**4.1 - 13**	**4.1 - 11.5**	
Total cholesterol (mg/dl)	**Mean ± SD**	**179.89 ± 28.73**	**178.33 ± 26.5**	**0.790**
	Range	**124 - 244**	**140 - 260**	
Triglyceride (mg/dl)	**Mean ± SD**	**130.04 ± 32.71**	**119.98 ± 24.89**	**0.104**
	Range	**70 - 190**	**86 - 177**	
LDL-cholesterol (mg/dl)	**Mean ± SD**	**99.1 ± 33**	**92.89 ± 28.76**	**0.344**
	Range	**47.4 - 173.2**	**48.8 - 180**	
CK-MB	**Mean ± SD**	**3.98 ± 0.84**	**4.16 ± 0.8**	**0.305**
	Range	**3 - 5**	**3 - 5**	
Troponin (ng/ml)	**Mean ± SD**	**0.02 ± 0.01**	**0.02 ± 0.01**	**0.141**
	Range	**0.01 - 0.03**	**0.01 - 0.04**	

Hb: Hemoglobin, W.B.C: White Blood Cells, PLT: Platelet count, Cr: Creatinine, UA: Uric Acid, Na: Sodium, K: Potassium, HbA1c: Glycated Hemoglobin, TC: Total Cholesterol, TG: Triglyceride, LDL-C: Low-Density Lipoprotein Cholesterol, CK-MB: Creatine Kinase Myocardial Band, cTn: Cardiac Troponin.

Table 3: QRS-T angle of the groups (n=90)

		Normal coronary flow group (n=45)	Coronary Slow flow group (n=45)	P value
QRS-T angle (°)	**Mean ± SD**	**72.36 ± 33.31**	**60 ± 31.69**	**0.075**
	Range	**16 - 135**	**13 – 158**	

Table 4: Echocardiographic parameters of the groups (n=90)

		Normal coronary flow group (n=45)	Coronary Slow flow group (n=45)	P value
LVEF (%)	**Mean ± SD**	**62.13 ± 4.66**	**62.67 ± 4.8**	**0.594**
	Range	**55 - 70**	**55 - 72**	

		Normal coronary flow group (n=45)	Coronary Slow flow group (n=45)	P value
LA diameter (mm)	Mean ± SD	36.4 ± 4.82	38.13 ± 4.29	0.075
	Range	29 - 43	30 - 45	
LVEDD (mm)	Mean ± SD	45.24 ± 3.55	46.33 ± 3.67	0.156
	Range	39 - 50	40 - 53	
LVESD (mm)	Mean ± SD	27.11 ± 3.87	27.64 ± 3.77	0.510
	Range	21 - 32	22 - 35	
IVST (mm)	Mean ± SD	10.64 ± 1.51	11.29 ± 2.02	0.090
	Range	8 - 13	8 - 14	
PWT (mm)	Mean ± SD	8 ± 1.4	8.4 ± 1.62	0.212
	Range	6 - 10	6 - 11	
LVMI (g/m^2)	Mean ± SD	79.4 ± 14.71	82.04 ± 14.4	0.391
	Range	54 - 106	59 – 105	

LVEF: Left ventricle ejection fraction, LA: Left atrial, LVEDD: Left ventricular end-diastolic diameter, LVESD: left ventricular end-systolic diameter, IVST: interventricular septum thickness, PWT: posterior left ventricle wall thickness, LVMI: left ventricle mass index.

Table 5: Role of frontal QRS-T angle in prediction of coronary slow flow phenomenon, QRS-T angle among both groups (n=90)

Cut-off	Sensitivity	Specificity	P.P.V	N.P.V	AUC	P value
>90	31.11%	93.33%	82.4%	57.5%	0.602	0.091

In group 1						
		No risk factor (n=3)	One risk factor (n=15)	Two risk factors (n=12)	3-4 risk factor (n=15)	P value
QRS-T angle (°)	Mean ± SD	47.3 ± 20.13	61.8 ± 33.14	64.3 ± 18.08	95.5 ± 35.75	0.008*
	Range	26 - 66	16 - 120	40 - 100	18 - 135	
P1			0.481	0.176	0.04*	
P2				0.816	0.012*	
P3					0.011*	

In GROUP 2						
		No risk factor(n=8)	One risk factor (n=12)	Two risk factors (n=12)	3-4 risk factor (n=11)	P value
QRS-T angle (°)	Mean ± SD	44.4 ± 26.61	59.7 ± 29.03	62.3 ± 23.6	76.2 ± 39.78	0.181
	Range	14 - 78	17 - 99	16 - 88	13 - 158	

P.P.V: positive predictive value, N.P.V: negative predictive value, A.U.C: area under the curve.

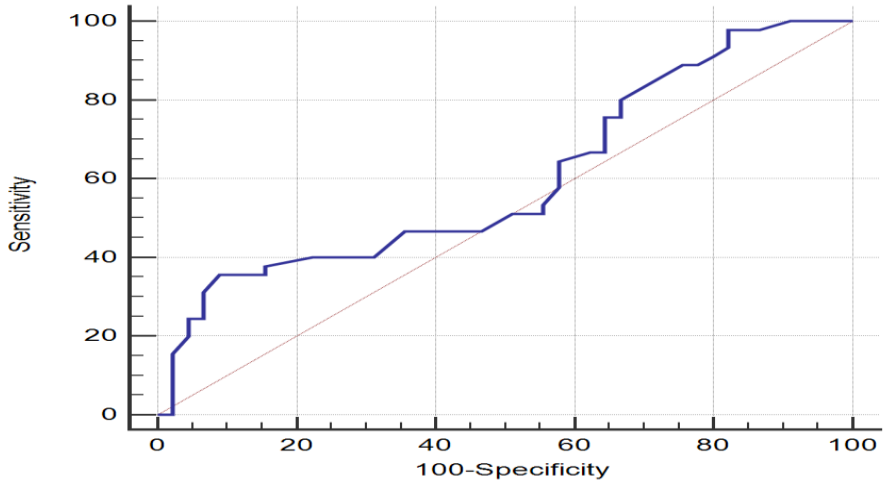

Figure 1: ROC curve of the role of frontal QRS-T angle in prediction of coronary slow flow phenomenon. the relatively gradual slope and fluctuations suggest limited sensitivity and specificity across thresholds. The test lacks strong discriminative power, warranting further optimization or combination with additional markers.

DISCUSSION

The f(QRS-T) angle reflects the disparity between ventricular activation and recovery pathways, offering a practical means to assess repolarization heterogeneity [20]. This index is conveniently accessible on standard 12-lead ECGs, as current automated systems calculate it directly by comparing the recorded QRS and T-wave axes [22].Although the difference in QRS-T angle between the groups did not reach statistical significance ($p = 0.075$), this borderline result may still suggest a potential trend worth further exploration in larger cohorts. Additionally, the modest discriminative performance (AUC = 0.602) highlights that while frontal QRS-T angle may provide supportive information, it is unlikely to serve as a stand-alone diagnostic marker. Instead, it could be considered alongside other clinical and echocardiographic parameters to improve overall risk stratification. Our study offers a novel viewpoint by combining ECG-derived frontal QRS-T angle with angiographic confirmation of CSF. Several recent studies have shown that a widened f (QRS-T) angle carries important prognostic implications in diverse cardiac patient groups [23,24]. Extensive evidence supports the role of the f(QRS-T) angle in identifying individuals at heightened risk for adverse cardiac events, including malignant arrhythmias and sudden cardiac death. Consistently, Raposeiras et al. demonstrated that a value exceeding 90° served as a powerful prognostic indicator for long-term mortality in patients with left ventricular systolic impairment following an acute myocardial infarction. [23]. However, while its role in risk stratification is well recognized in other cardiac settings, its relationship with the CSF phenomenon has not been clearly investigated. Addressing this gap, the present work sought to determine whether a markedly increased f(QRS-T) angle could act as a non-invasive indicator for the presence of CSF during coronary angiographic evaluation.In the present analysis, demographic characteristics including age and gender were comparable between the coronary slow flow group and participants with normal coronary perfusion, with no significant differences observed. This observation agrees with the findings of Askin and Tanrıverdi, who performed a case-control analysis involving 100 participants in each group CSF and control matched for

demographic variables, and similarly reported no meaningful disparity in age or gender profiles [24]. Likewise, prevalent cardiovascular risk factors, including diabetes mellitus, hypertension, and smoking, showed no significant variation between the CSF and normal flow cohorts. These results are in line with those of Elawady et al., who also documented comparable rates of diabetes, hypertension, and smoking in patients with and without the coronary slow flow phenomenon (CSFP) [25]. In further support, Özbek reported that such conventional risk factors were similarly distributed across CSFP and control groups, suggesting that they may have limited value in differentiating patients who had CSF from others with normal coronary flow [18].Both study groups those with coronary slow flow and those with normal coronary flow showed no significant differences in hemoglobin, white blood cell, or platelet levels. Likewise, serum creatinine, uric acid, sodium, and potassium values remained comparable across the two cohorts. Metabolic parameters, including HbA1c and lipid profile components (TC, TG, LDL-C, HDL-C), also showed no significant variation. Furthermore, cardiac biomarkers, namely CK-MB and troponin, did not differ appreciably, indicating that the occurrence of CSF in this cohort was not associated with measurable changes in standard hematologic, biochemical, or metabolic indices. These observations are consistent with those of Askin and Tanrıverdi, who reported no significant variation in hemoglobin, white blood cell count, platelet count, creatinine, total cholesterol, or triglyceride levels between CSF patients and controls [24]. Similarly, Elawady et al. documented no significant differences in CK-MB or troponin concentrations between individuals with and without the coronary slow flow phenomenon (CSFP) [25]. Supporting this, Kuyumcu et al. demonstrated that hemoglobin, white blood cell count, platelet count, creatinine, uric acid, and lipid profile values were broadly similar in CSF and normal coronary flow groups [26].In the present analysis, echo-derived parameters—namely LVEF, LA diameter, LVEDD, LVESD, IVST, PWT, and LVMI—were comparable between the CSF and normal flow groups, with no statistically significant differences observed. This suggests that conventional structural and functional indices may not effectively differentiate patients with CSF from those with normal epicardial flow. These observations are consistent with the work of Algamal et al., who reported similar LVEF values across CSFP and control populations [27], and with Özbek, who likewise demonstrated no significant variation in LVEF, LA diameter, LVEDD, LVESD, IVST, PWT, or LVMI between the two cohorts. The reproducibility of these findings across independent studies indicates that the pathophysiological substrate of CSF may not be adequately captured by standard echocardiographic measurements, underscoring the need for more sensitive imaging or functional assessment tools to detect subtle myocardial or microvascular alterations in this population [18].In our analysis, the QRS–T angle remained within normal limits (<90 degrees) in both groups. This observation is in line with the report by Işık et al., in which mean frontal QRS–T angles were 48 degrees in the slow flow group versus 37 degrees in the normal flow group [28], and with Özbek's findings of 51 degrees versus 27 degrees, respectively [18]. Although these values were well within the normal range, they were consistently higher among CSF patients. Furthermore, the present research demonstrated that the QRS–T angle was significantly greater in patients with three to four cardiovascular risk factors compared with those having none, one, or two risk factors (P = 0.04,

0.012, and 0.011, respectively), while no significant differences were found among the latter three subgroups. This may suggest a cumulative or synergistic impact of multiple risk factors on QRS–T angle widening, a hypothesis that warrants further confirmation through studies with larger sample sizes.This research evaluated the utility of (fQRS–T) angle in predicting CSF by integrating electrocardiographic, echocardiographic, and angiographic data. The study's strengths include the objective confirmation of CSF through cTFC and the incorporation of subgroup analyses according to cardiovascular risk profiles. Nonetheless, certain limitations should be acknowledged: its single-center nature, relatively small sample size, and cross-sectional design restrict the ability to establish causality. Additionally, potential inter-observer variability in interpreting ECG and angiographic findings may have influenced results. The absence of advanced imaging modalities or assessments of endothelial function may also have limited a more detailed exploration of underlying pathophysiological mechanisms.The relatively small sample size (90 participants) represents a limitation of our study, which may affect the generalizability of the results. Although our findings suggest a potential role of the frontal QRS-T angle as a non-invasive predictor of coronary slow flow, confirmation through larger, multicenter studies with more diverse populations is warranted to strengthen external validity. In our study, the frontal QRS-T angle did not demonstrate strong discriminative power for coronary slow flow (AUC = 0.602), and several clinical and echocardiographic parameters showed no significant differences between groups. This finding suggests that the QRS-T angle alone may not be sufficient as a predictor. However, its correlation with the number of cardiovascular risk factors indicates that it may still provide incremental value when considered as part of a broader clinical and diagnostic framework.In summary, while our findings indicate that the frontal QRS-T angle alone may not serve as a robust stand-alone predictor of coronary slow flow, its association with cardiovascular risk burden and its practicality as a simple ECG-derived marker highlight its potential as part of a multimodal diagnostic approach.

CONCLUSION

The coronary slow flow phenomena cannot be predicted by the frontal QRS-T angle. Nonetheless, accumulation of risk factors was associated with wider f(QRS-T) angles.

Conflict of Interest & Funding Statement: This work was completed independently, with no external funding, and the authors report no competing interests.

Data Availability: Requests for access to the underlying data that substantiate the results of this study may be directed to the corresponding author, who will consider providing them in accordance with institutional policies and applicable ethical guidelines.

Author Contributions: A.E.S. provided senior supervision and conceptual guidance. S.G.Z. assisted in methodological development and data interpretation. S.E.A., the corresponding author, was responsible for data collection, manuscript drafting, and coordinating revisions. M.S.G. contributed to statistical analysis and critical review of the final manuscript. All authors edited, revised, reviewed and approved the final manuscript. **Supplementary Data:**Figure S1,S2,S3and S4 .

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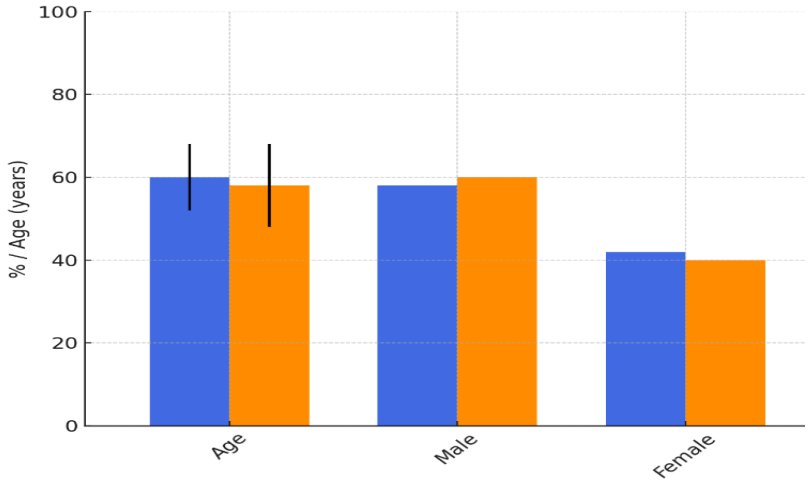

Figure S1 :Comparison between demographic data in both groups

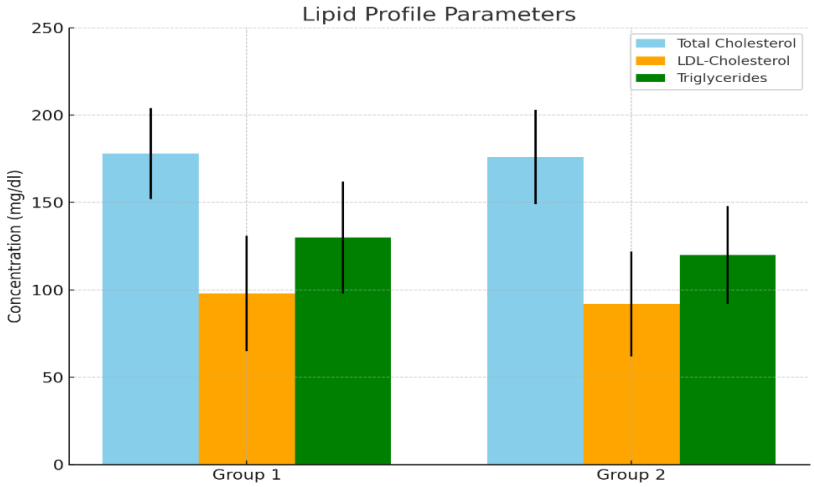

Figure S2: Comparison between lipid profile parameters in both groups

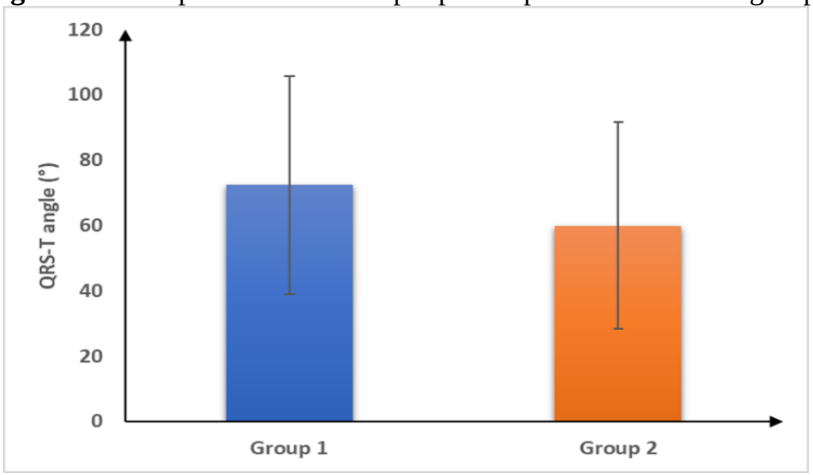

Figure S3: Mean Frontal QRS-T angle of both groups

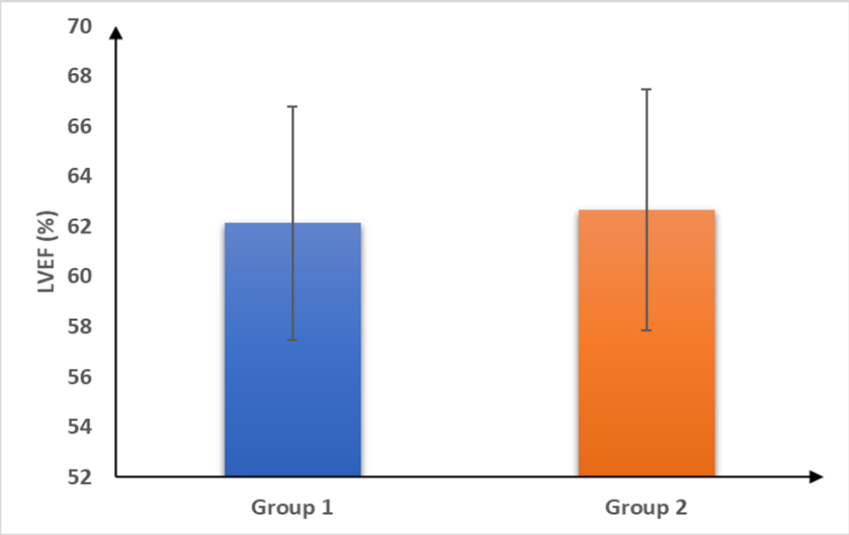

Figure S4: Comparison between LVEF in both groups

Citation

Sherif, A., Zein El Abdeen, S., Soliman, S., Ghareeb, M. Frontal QRS-T Angle as A Non-invasive Predictor of The Presence of Coronary Slow Flow. ***Zagazig University Medical Journal*****, 2025; (5420-5431): -. doi: 10.21608/zumj.2025.414710.4118**