

Original Article

Frontal T-P Angle: A Novel ECG Parameter for Predicting SYNTAX-1 and SYNTAX-2 Scores in Acute Non-ST-Segment Elevation Myocardial Infarction

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Highlights

- Novel ECG Parameter: The frontal T-P angle (fT-Pa), derived from the difference between T-wave and P-wave axes, is introduced as a new predictor for coronary artery disease (CAD) complexity in acute NSTEMI patients.
- Strong Correlation with SYNTAX-2: fT-Pa showed a significant correlation with SYNTAX-2 scores ($r = 0.229$, $*P = 0.005$), outperforming its association with SYNTAX-1, suggesting its utility in risk stratification.
- Predictive Cutoff Values: ROC analysis identified fT-Pa cutoffs of 36.5° (for SYNTAX-2 > 26.2) and 39.5° (for SYNTAX-1 > 22), offering practical thresholds for clinical use.
- Clinical Implications: fT-Pa provides a simple, reproducible tool to assess CAD severity even in patients with normal T-wave and P-wave axes, aiding early risk assessment in acute NSTEMI.

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ABSTRACT

Background: Few studies have investigated P-wave and T-wave axes, most of which focus on mortality. The frontal T-P angle (fT-Pa) is a novel ECG-derived parameter of ventricular repolarization. We aimed to evaluate the association between fT-Pa and SYNTAX-1 (SS-1) and SYNTAX-2 (SS-2) scores in patients with acute non-ST-segment elevation myocardial infarction (ANSTEMI).

Methods: This retrospective study included 158 ANSTEMI patients undergoing coronary angiography. The study population was stratified based on SS-1 (≤ 22 vs. > 22) and SS-2 (≤ 26.2 vs. > 26.2). The fT-Pa was calculated by subtracting the P-wave axis from the T-wave axis, both obtained from the ECG device's built-in software, and compared between groups.

Results: fT-Pa showed significant correlations with age ($r=0.242$, $P=0.003$), SS-2 ($r=0.229$, $P=0.005$), and T-wave axis ($r=-0.626$, $P<0.001$). Both age and fT-Pa were significantly correlated with and predictive of SS-2 ($\beta=0.679$; OR, 0.700 [95% CI, 0.584 to 0.816]; $P<0.001$ for age) and ($\beta=0.147$; OR, 0.048 [95% CI, 0.012 to 0.085; $P=0.010$ for fT-Pa). ROC curve analysis identified an fT-Pa cutoff of 36.5 (64% sensitivity, 68% specificity; AUC, 0.674; $P<0.001$) for predicting SS-2 > 26.2 , and a cutoff of 39.5 (70% sensitivity, 62% specificity; AUC, 0.692; $P=0.010$) for predicting SS-1 > 22 .

Conclusions: fT-Pa showed a stronger correlation and association with SS-2 than with SS-1. This parameter may serve as a simple, reproducible tool for predicting coronary artery disease complexity, even in patients with normal T-wave and P-wave axis ranges.

Keywords: Coronary Artery Disease; Electrophysiology; Electrocardiography; Acute Coronary Syndrome

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Introduction

Acute coronary syndrome (ACS) continues to be prevalent worldwide, with acute non-ST-segment elevation myocardial infarction (NSTEMI) constituting the majority of ACS cases. Over the years, researchers have gathered substantial evidence indicating that certain ECG parameters can predict the severity of coronary artery disease (CAD) as determined by the SYNTAX Score (SS).¹⁻³ Nonetheless, conflicting results persist regarding the association between these ECG parameters and the extent of CAD, necessitating the development of new, simpler ECG parameters to estimate SSs in acute settings. This would provide physicians with vital information on the severity of CAD.⁴

T-wave axis (TWA), a ventricular repolarization parameter, has been studied only in a limited number of studies,^{5,6} primarily focusing on mortality and incident coronary heart disease.^{7,8} Normal TWA values range from 15 to 75 degrees. The relationship between ventricular repolarization parameters and the extent of CAD has been extensively researched; nevertheless, TWA as a repolarization parameter remains under-investigated in this context. One study reported no association between TWA and the angiographic extent of CAD.⁹

P-wave axis (PWA) deviation from normal values (0–75°) has been linked to delayed atrial conduction and fibrosis.^{10,11} Abnormal PWA has been primarily associated with atrial fibrillation, cerebrovascular accidents, and cardiovascular mortality due to its arrhythmic effects.^{10,12,13} However, the relationship between PWA and the extent of CAD has not yet been investigated.

Given that both PWA and TWA are readily available automated measurements on the surface ECG and have studied effects on mortality, cardiac arrhythmia, and coronary heart diseases, we hypothesize that the angle between the T- and P-wave axes, known as the frontal T-P angle, could be more advantageous than TWA or PWA alone in predicting the extent of CAD determined by SS.

Methods

This retrospective, cross-sectional study

included 158 patients who presented to our hospital with acute NSTEMI between October 2022 and September 2023 and subsequently underwent coronary angiography. The inclusion criteria were patients aged 18 to 90 years with a diagnosis of acute NSTEMI. Patients with advanced chronic kidney disease, hyperkalemia, acute kidney failure, significant valve disease, chronic systemic inflammatory conditions, a history of cancer, atrial fibrillation, complete or incomplete bundle branch block, antiarrhythmic drug use, and a paced rhythm were excluded.

Out of the 415 acute NSTEMI patients reviewed retrospectively, 257 were excluded as they did not meet the inclusion criteria, resulting in a final sample of 158 patients. The study adhered to the guidelines outlined in the Helsinki Declaration and received approval from the Local Committee for Ethics.

The diagnosis of acute NSTEMI was established based on the following criteria: a rise and/or fall in cardiac troponin levels, along with at least one of these accompanying indicators:

1. Symptoms of cardiac ischemia
2. ST-segment depression, T-wave inversion, or transient ST-segment elevation lasting less than 20 minutes.
3. Imaging evidence of new myocardial damage or abnormal ventricular wall motion.
4. Angiographic evidence of an intracoronary thrombus¹¹.

All patients underwent a thorough evaluation comprising a detailed medical history and physical examination. Relevant data, including admission blood samples, ECGs, coronary angiography images, and echocardiograms, were collected and reviewed from patient records.

Coronary angiography was performed using standard methods, and catheterization images were analyzed to assess the complexity of CAD based on SYNTAX scores 1 and 2 (SS-1 and SS-2).³ To avoid interobserver variability, an independent cardiologist, blinded to the study data, calculated the SSs using the website <http://www.syntaxscore.com>.

Clinical data required for SS-2 calculation, including chronic obstructive pulmonary disease, gender, age, creatinine clearance, left ventricular

ejection fraction, and peripheral artery disease, were retrieved from patients' medical records.

SS-1 is categorized into three subgroups based on CAD complexity: low SS-1 (≤ 22), moderate SS-1 (23–32), and high SS-1 (≥ 33). In our study, patients were divided into two subgroups according to SS-1 and SS-2: low SS-1 ($SS1 < 22$) and moderate-to-high SS-1 ($SS-1 > 22$), as well as low SS-2 (mean $SS-2 < 26$) and high SS-2 (mean $SS-2 > 26$). No universally accepted SS-2 tertiles exist, unlike SS-1, leading to variations in subgroup categorization among studies.¹⁴

Commonly used tertiles in the literature include <17 ¹⁵; <20 , $<20-26$, and >26 ¹⁶; <26 ¹⁴; and <22 .¹⁷ Considering our study's mean SS-2 value of 26.2 and existing literature examples with a cutoff value of 26, we chose to stratify our population into two subgroups using <26 for SS-2 classification.

ECG measurements of PWA, TWA, and the frontal T-P angle were obtained from 12-lead ECG strips recorded at a paper speed of 25 mm/s and a record amplitude of 10 mm/0.1 mV. The GE Marquette (Milwaukee, Wisconsin) MAC 1200 electrocardiograph was used at a sampling rate of 500 Hz.

PWA and TWA values were derived from the ECG device's built-in software calculations. Subsequently, the frontal T-P angle was computed by subtracting the PWA from the TWA (Figure 1).

Data were analyzed using IBM SPSS Statistics for Windows, version 29.0 (IBM Corp., Armonk, NY, USA). The normality of distribution was assessed using the Kolmogorov-Smirnov test. Normally distributed continuous variables were expressed as mean \pm standard deviation, and non-normally distributed variables as median (interquartile range: 25th–75th percentiles). Categorical variables were reported as frequencies (percentages).

Intergroup comparisons (SS-1 and SS-2 subgroups) were performed using the Student t-test or the Mann-Whitney U test, as appropriate. Correlations between the frontal T-P angle and other parameters were assessed using the Spearman rank correlation. Linear regression analysis identified parameters associated with SS-2. Receiver operating characteristic (ROC) curve analysis determined optimal frontal T-P angle cutoff

values for predicting SS-1 and SS-2.

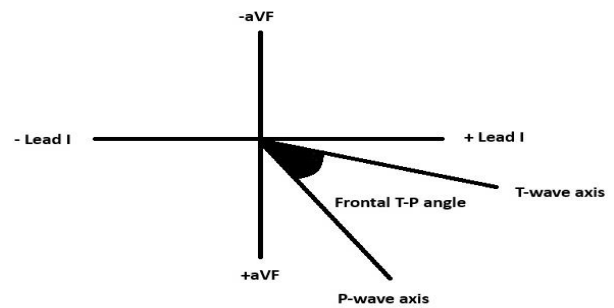


Figure 1. The image illustrates the measurement of the frontal T-P angle.

Results

Clinical, demographic, and ECG parameters are presented in (Table 1). The mean age of the study population was 63.52 ± 10.91 years, and 58.2% of participants were male.

(Table 2) compares demographic, clinical, and ECG parameters between subgroups stratified by SS-1 score (low SS-1 [≤ 22] vs. moderate-to-high SS-1 [> 22]). Demographic and clinical characteristics did not differ significantly between SS-1 subgroups (all P s > 0.05).

(Table 3) presents the same parameters stratified by SS-2 score (low SS-2 [≤ 26.2] vs. high SS-2 [> 26.2]). Similar to SS-1 subgroups, no significant differences were observed in demographic or clinical characteristics between SS-2 subgroups (all P s > 0.05).

Correlation analysis revealed significant associations between the frontal T-P angle and age ($r=0.242$, $P=0.003$), SS-2 score ($r=0.229$, $P=0.005$), and TWA ($r=-0.626$, $P<0.001$).

Multivariate linear regression analysis (Table 4) revealed that both age ($\beta = 0.679$; OR, 0.700 [95% CI, 0.584 to 0.816]; $P<0.001$) and the frontal T-P angle ($\beta=0.147$; OR, 0.048 [95% CI, 0.012 to 0.085]; $P=0.010$) maintained significant independent associations with SS-2 after covariate adjustment, whereas no significant model emerged for SS-1 prediction. ROC curve analysis demonstrated the predictive (Table 5) utility of the frontal T-P angle, identifying optimal cutoffs of 36.5 (64% sensitivity, 68% specificity; AUC, 0.674; $P<0.001$) for $SS-2 > 26.2$ and 39.5 (70% sensitivity, 62% specificity; AUC, 0.692; $P=0.010$) for $SS-1 > 22$. (Figure 2)

Table 1. Demographics, clinical, and ECG characteristics of the whole study population*

| Variable | (n=158) |
|-------------------------------|--------------------|
| SS-1 | 11 (7-18) |
| SS-2 | 25.72±11.23 |
| Age (y) | 63.52±10.91 |
| Sex | |
| Male (n, %) | 92 (58.2%) |
| Smoking (n, %) | 110 (69.6%) |
| DM (n, %) | 60 (37.9%) |
| HT (n, %) | 110 (69.6%) |
| PVD (n, %) | 11 (6.9%) |
| COPD (n, %) | 24 (15.1%) |
| LVEF (%) | 55 (50-60) |
| Troponin on admission (ng/mL) | 252.63 (118-488.2) |
| Creatinine (mg/dL) | 0.92±0.22 |
| Hg (g/dL) | 13.7±1.92 |
| K (mmol/L) | 4.21±0.22 |
| QT (ms) | 392.54±36.33 |
| QTc (ms) | 444 (431.7-461.6) |
| HR (bpm) | 79.81±13.32 |
| QRS axis (°) | 11.5 (-15.5-48.5) |
| P-wave axis (°) | 51(38.5-62.5) |
| T-wave axis (°) | 59.41±54.52 |
| Frontal QRS-T angle (°) | 47(20-95.2) |
| Frontal QRS-P angle (°) | 41.50±36.11 |
| Frontal T-P angle (°) | 33 (15-59) |

* Values are n (%), mean ± standard deviation, or median (25th and 75th percentiles). Depending on the normality of the data, either the Mann-Whitney U test or the Student t-test was used. For categorical variables, the chi-square test was applied.

DM: diabetes mellitus, HT: hypertension, PVD: peripheral vascular disease, COPD: chronic obstructive pulmonary disease, LVEF: left ventricular ejection fraction, SS-1: SYNTAX Score-1; SS-2, SYNTAX Score-2, QTc: corrected QT interval, HR: heart rate

Table 2. Subgroup comparison of the study population according to the SYNTAX-1 Score*

| Variable | SS-1 < 22 (n=30) | SS-1 > 22 (n=128) | P |
|---------------------------------|----------------------|----------------------|--------|
| SS-1 | 10 (7-15) | 26.5 (24-33.2) | <0.001 |
| SS-2 | 25.64±10.71 | 31.33±14.12 | 0.041 |
| Age (y) | 65 (56-72) | 61 (57.7-73.5) | 0.751 |
| Sex | | | 0.234 |
| Male, (n, %) | 18(60.0%) | 74 (57.8%) | |
| Smoking (n, %) | 21(70%) | 89 (69.5%) | 0.764 |
| DM (n, %) | 11 (36.6%) | 49 (38.2%) | 0.897 |
| HT (n, %) | 21(70%) | 89(69.5%) | 0.912 |
| PVD (n, %) | 2 (6.6%) | 9 (7%) | 0.356 |
| COPD (n, %) | 4 (13.3%) | 20 (15.6%) | 0.071 |
| LVEF (%) | 55(50-60) | 53(45-60) | 0.312 |
| Troponin T on admission (ng/mL) | 232.3 (124.5- 540.5) | 241.8 (123.3-530.1) | 0.210 |
| QT (ms) | 412.61±41.92 | 389.14±32.24 | 0.102 |
| QTc (ms) | 448 (425-475) | 447 (434-462) | 0.624 |
| HR (bpm) | 73.11±10.12 | 78.32±13.13 | 0.041 |
| QRS axis (°) | 13 (-16.5-46.2) | 2 (-17-56.7) | 0.943 |
| P-wave axis (°) | 51 (38-63) | 53.5 (40.2-62.7) | 0.722 |
| T-wave axis (°) | 60 (27.7-85.5) | 40 (12-89.5) | 0.298 |
| Frontal QRS-T angle (°) | 47 (20-96.5) | 41 (13.2-77.2) | 0.561 |
| Frontal QRS-P angle (°) | 37.5 (15-61.5) | 37 (10-72.5) | 0.954 |
| Frontal T-P angle (°) | 32 (13-52) | 59 (35-64) | 0.012 |

* Values are n (%), mean ± standard deviation, or median (25th and 75th percentiles). Depending on the normality of the data, either the Mann-Whitney U test or the Student t-test was used. For categorical variables, the chi-square test was applied.

DM: diabetes mellitus, HT: hypertension, PVD: peripheral vascular disease, COPD: chronic obstructive pulmonary disease, LVEF: left ventricular ejection fraction, SS-1: SYNTAX Score-1; SS-2, SYNTAX Score-2, QTc: corrected QT interval, HR: heart rate

Table 3. Subgroup comparison of the study population according to the SYNTAX-2 Score*

| Variable | SS-2<26.2 (n=85) | SS-2>26.2 (n=73) | P |
|--------------|---------------------|---------------------|--------|
| SS-1 | 11 (8-16.5) | 11 (7-18.7) | 0.841 |
| SS-2 | 18.21±5.93 | 35.61±8.21 | <0.001 |
| Age (y) | 58 (50.2-63.7) | 72 (67-76) | <0.001 |
| Sex | | | 0.567 |
| Male, (n, %) | 51(60.0%) | 42 (57.5%) | |
| Smoking | 60 (70.5%) | 50 (68.4%) | 0.479 |
| DM | 33 (38.8%) | 27 (36.9%) | 0.123 |
| HT | 58 (68.2%) | 52 (71.2%) | 0.356 |
| PVD | 6 (7.0%) | 5 (6.8%) | 0.922 |
| COPD | 13 (15.2%) | 11 (15.0%) | 0.893 |
| LVEF (%) | 52 (45-60) | 55 (50-60) | 0.643 |

| | | | |
|---------------------------------|----------------------|---------------------|--------|
| Troponin T on admission (ng/mL) | 231.2 (116.5- 538.5) | 244.2 (126.2-516.7) | 0.938 |
| QT (ms) | 410.73±40.91 | 392.32±33.71 | 0.064 |
| QTc (ms) | 444 (422-474.3) | 441 (432-460) | 0.290 |
| HR (bpm) | 73.26±10.32 | 77.32±12.63 | 0.032 |
| QRS axis (°) | 31.5 (-10.5-55) | -3.5 (-20.7-30.5) | 0.003 |
| P-wave axis (°) | 55.5 (42-63.5) | 50 (34.5-62.5) | 0.215 |
| T-wave axis (°) | 57 (27-81) | 61 (27-92.5) | 0.519 |
| Frontal QRS-T angle (°) | 41.5 (17-93.5) | 49 (25-105) | 0.265 |
| Frontal QRS-P angle (°) | 34 (14-58) | 43 (16.5-63) | 0.444 |
| Frontal T-P angle (°) | 24.5 (10-46) | 44 (23.5-68.5) | <0.001 |

* Values are n (%), mean ± standard deviation, or median (25th and 75th percentiles). Depending on the normality of the data, either the Mann-Whitney U test or the Student t-test was used. For categorical variables, the chi-square test was applied.

DM: diabetes mellitus, HT: hypertension, PVD: peripheral vascular disease, COPD: chronic obstructive pulmonary disease, LVEF: left ventricular ejection fraction, SS-1: SYNTAX Score-1; SS-2, SYNTAX Score-2, QTc: corrected QT interval, HR: heart rate

Table 4. The correlation analysis between the frontal T-P angle and other study parameters

| Variable | Spearman Rho | P |
|-----------------|--------------|--------|
| Ss1 | 0.133 | 0.109 |
| Ss2 | 0.229* | 0.005 |
| Age (y) | 0.242* | 0.003 |
| P-wave axis (°) | -0.013 | 0.871 |
| QRS-axis (°) | -0.125 | 0.123 |
| T-wave axis (°) | -0.626* | <0.001 |
| QRS-P angle (°) | 0.054 | 0.504 |
| QRS-T angle (°) | -0.112 | 0.166 |

*Correlation is significant at the 0.01 level.

Table 5. Univariate and multivariate linear regression analyses showing the variables associated with SS-2

| Variable | Univariate | | | Multivariate | | |
|-----------------|------------|---------------------------|--------|--------------|---------------------------|--------|
| | β | Unstandardized B (95% CI) | P | β | Unstandardized B (95% CI) | P |
| Age (y) | 0.72 | 0.74 (0.63-0.86) | <0.001 | 0.67 | 0.70 (0.58-0.81) | <0.001 |
| P-wave axis (°) | -0.07 | -0.03 (-0.11-0.04) | 0.359 | | | |
| QRS axis (°) | -0.17 | -0.36 (-0.06-0.003) | 0.034 | -0.05 | -0.01 (-0.03-0.01) | 0.362 |
| T-wave axis (°) | 0.12 | 0.02 (-0.01-0.05) | 0.125 | | | |
| QRS-P angle (°) | -0.01 | -0.04 (-0.05-0.04) | 0.882 | | | |
| QRS-T angle (°) | 0.06 | 0.01 (-0.01-0.04) | 0.438 | | | |
| T-P angle | 0.32 | 0.10 (0.05-0.15) | <0.001 | 0.14 | 0.04 (0.01-0.08) | 0.010 |

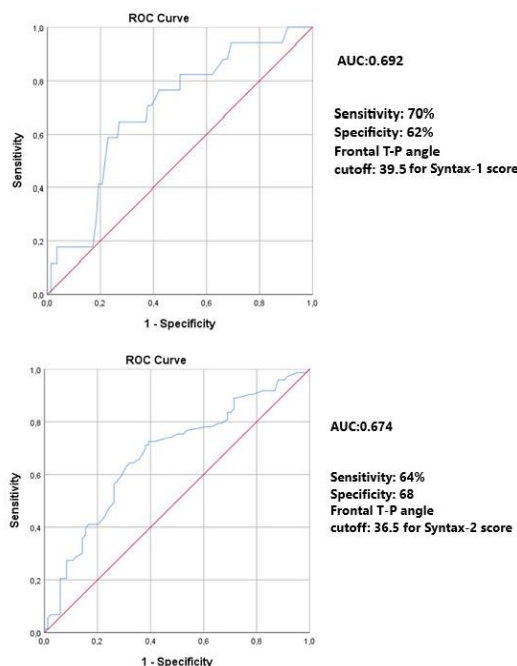


Figure 2. The images present the results of the ROC curve analysis. ROC: receiver operating characteristic

Discussion

Our study's principal findings revealed a correlation between SS-2 and TWA with the frontal T-P angle. Further, SS-2 was associated with both age and the frontal T-P angle. These results suggest that the frontal T-P angle could serve as a more robust predictor of SS-2 than SS-1 in patients with acute NSTEMI. To our knowledge, this is the first study to assess the relationship between the novel frontal T-P angle and SS.

The association between atrial depolarization, ventricular repolarization directions, and various disease conditions has garnered increasing interest in the medical field. Changes in the frontal TWA may indicate a decline in ventricular repolarization and potential ventricular microfibrosis, which, in turn, could elevate the risk of ventricular arrhythmias and sudden cardiac

death.⁹

Dilaveris et al.¹⁸ demonstrated a significant shift in TWA in hypertensive patients with left ventricular hypertrophy (LVH) than in those without LVH.

In the Rotterdam Heart study involving elderly individuals,^{7,19} a significant shift in the frontal TWA emerged as the most predictive ECG parameter among other ventricular repolarization markers for fatal or nonfatal events. Rautaharju et al. 20 demonstrated a correlation between abnormal TWA and subclinical cardiac ischemia, with an increased likelihood of coronary heart disease in elderly populations exhibiting abnormal TWA.

Assanelli et al.¹⁹ reported an association between abnormal TWA and metabolic syndrome and its components. On the other hand, Dilaveridis et al.⁸ acknowledged that although TWA and frontal planar QRS-T angle were considered parameters of global ventricular repolarization, conflicting results in the literature highlighted the need for new parameters. Still, none of these studies examined the clinical predictive value of TWA in relation to CAD complexity as measured by SS.

Notably, previous studies typically categorized their cohorts into patients with either normal or abnormal TWA and PWA, using the established normal ranges of 15 to 75 degrees and 0 to 75 degrees, respectively.^{7,10,20} Be that as it may, a substantial proportion of patients may still exhibit high SSs despite having TWA and PWA values within the normal range.

Our novel parameter, the frontal T-P angle, can address this issue by potentially distinguishing patients with moderate-to-high CAD complexity using ROC values. A frontal T-P angle of 36.5 was identified for SS-2>26.2, and a frontal T-P angle of 39.5 for SS-1>22, enabling better identification of patients with significant CAD even within normal TWA and PWA ranges.

P-wave morphology and axis are crucial in atrial proarrhythmic remodeling. Increases in atrial volume and diameter can lead to PWA shifts and result in a higher incidence of atrial arrhythmias, such as atrial fibrillation.^{21,22} Li et al.¹⁰ further demonstrated an association between abnormal PWA and increased cardiovascular and all-cause mortality rates. Despite its prognostic implications,

little is known about the diagnostic utility of PWA in assessing CAD complexity, mirroring the limited knowledge about TWA in this context.

The detrimental effects of deteriorated ventricular diastolic function in ACS settings have been well-documented.^{23,24}

Azarizman et al.²⁵ assessed diastolic dysfunction with magnetic resonance imaging in patients with chest pain and used this method as a pre-diagnostic tool for the early detection of ACS. Furthermore, it has been demonstrated that diastolic dysfunction deteriorates P-wave parameters.^{26,27} It is, therefore, reasonable to assume deviations in PWA during an ACS.

The present study has several important limitations. First, the retrospective design and relatively small sample size constrained our ability to assess prognostic implications. Second, the absence of baseline diastolic function measurements may represent a potential confounding factor in our results. Third, we did not evaluate arrhythmic complications during hospitalization, which precludes analysis of the potential relationship between the frontal T-P angle and early post-ACS arrhythmic events. These findings require validation through prospective studies with larger cohorts that incorporate comprehensive functional assessments and arrhythmia monitoring.

Conclusion

The frontal T-P angle demonstrates a stronger correlation and association with SS-2 than with SS-1. The identified cut-off values of 36.5 degrees (for SS-2>26.2) and 39.5° (for SS-1>22) may serve as practical, reproducible markers for predicting CAD complexity, even in patients with normal TWA and PWA ranges. These findings suggest that the frontal T-P angle could complement existing ECG parameters in risk stratification, although validation through larger prospective studies is warranted.

Declarations: Ethical Approval

This study was approved by the Ethics Committee of Ahi Evran University (Approval No:

2024-16/137, Date: October 8, 2024).

Funding

There is no funding for this study.

Conflict of Interest

There is no conflict of interest in this study.

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