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CARDIOVASCULAR SURGERY AND INTERVENTIONS

Predictive value of heart rate recovery and QT dispersion in determining the presence and severity of coronary artery disease

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ABSTRACT

Objectives: The aim of this study was to investigate the value of non-invasive parameters, including heart rate recovery (HRR), QT dispersion (QTd) and the Duke treadmill score (DTS), in predicting the presence and severity of coronary artery disease (CAD) as quantified by the Gensini score.

Patients and methods: In this prospective observational study, 200 patients with stable angina pectoris and positive exercise stress tests were enrolled. All participants underwent coronary angiography (CAG). Patients were categorized into three groups based on CAG results: Normal coronary arteries (27%), non-critical CAD (47%), and critical CAD (26%). Electrocardiogram markers (QTd) and exercise parameters (HRR1, HRR2, DTS, and rate-pressure product) were analyzed in relation to the Gensini score.

Results: Significant differences were observed among the three groups in QTd, HRR1, HRR2, and DTS ($p<0.001$). The critical CAD group exhibited the highest QTd and the lowest HRR and DTS values. Correlation analyses revealed that the Gensini score was strongly associated with QTd ($r=0.742$) and negatively associated with HRR1 ($r=-0.672$) and DTS ($r=-0.632$) ($p<0.001$). Receiver operating characteristic analysis identified QTd >32 ms (area under the curve [AUC]: 0.842) and HRR1 <11 bpm (AUC: 0.815) as significant predictors of critical stenosis, outperforming traditional markers. Multivariate logistic regression analysis identified QTd (odds ratio [OR]: 1.09, 95% confidence interval [CI]: 1.04-1.14, $p=0.002$), HRR1 (OR: 0.82, 95% CI: 0.75-0.90, $p=0.001$), and Duke score (OR: 0.88, 95% CI: 0.81-0.96, $p=0.015$) as independent predictors of critical CAD.

Conclusion: QTd and HRR1 are robust, non-invasive indicators of the presence and anatomical severity of CAD. Integrating these parameters into routine clinical evaluation can improve risk stratification and the early identification of patients requiring invasive intervention.

Keywords: Coronary artery disease, heart rate, arrhythmias, cardiac, exercise test, and severity of illness index.

Coronary artery disease (CAD) often remains asymptomatic until sudden cardiac death occurs, necessitating effective early diagnostic tools. While the exercise stress test (EST) is a cost-effective method, its variable sensitivity and specificity require additional parameters to enhance clinical efficacy.[1]

Heart rate recovery (HRR) is a powerful, non-invasive physiological index that reflects the dynamic interplay between the parasympathetic and sympathetic branches of the autonomic nervous system following exercise. While the overall decline in heart rate (HR) post-exertion serves as a marker of autonomic health, HRR during the first and second minutes (HRR1 and HRR2) represent distinct physiological phases. HRR1 (1-minute) is primarily driven by rapid vagal (parasympathetic) reactivation. Within the first 60 seconds of cessation, the sudden withdrawal of central command and the activation of baroreceptors lead to a surge in acetylcholine release, causing a sharp deceleration in heart rate.[2] A blunted HRR1 is widely recognized as an independent predictor of all-cause mortality and sudden cardiac death, as it signifies vagal incompetence.[3] In contrast, HRR2 reflects the subsequent

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phase of autonomic stabilization, which is more closely associated with sympathetic withdrawal and the gradual clearance of circulating catecholamines from the bloodstream. Research indicates that a delayed HRR2 is specifically linked to sustained hemodynamic recovery and advanced coronary atherosclerotic burden.[4] Furthermore, blunted HRR often serves as an early marker of autonomic dysfunction in chronic conditions such as diabetes mellitus, hypertension, and heart failure, reflecting diminished vagal tone long before clinical symptoms manifest.[5-7]

The clinical integration of these autonomic markers is particularly valuable when correlated with the anatomical severity of CAD, which is objectively quantified using the Gensini score. The Gensini score serves as a comprehensive angiographic tool that integrates the degree of luminal narrowing with the functional importance of specific vascular segments. Unlike other scoring systems, it offers a more granular assessment of the total atherosclerotic burden by incorporating lesions with as little as 25% stenosis, which are often overlooked but contribute significantly to overall disease prevalence.[8] Clinical trials have demonstrated that higher Gensini scores are strongly associated with multi-vessel disease and increased major adverse cardiovascular events.[9] By evaluating HRR and the Gensini score in tandem, clinicians can better understand how autonomic dysregulation parallels the extent of coronary artery stenosis, thereby enhancing risk stratification in patients with stable angina.

Alongside autonomic indices, QT dispersion (QTd) has gained prominence in clinical research as a crucial marker of ventricular repolarization heterogeneity.[10] QTd is defined as the difference between the maximum and minimum QT intervals measured on a standard 12-lead electrocardiogram (ECG); it reflecting the regional variations in myocardial recovery time. The clinical significance of QTd is well-documented across various cardiovascular pathologies. Seminal studies have demonstrated that elevated QTd is a potent predictor of sudden cardiac death, particularly in the post-myocardial infarction period and in patients with heart failure.[11] For instance, research has shown that patients with CAD exhibit significantly higher QTd values compared to healthy individuals, and this increase directly parallels the extent of myocardial ischemia.[12] Furthermore, contemporary studies have highlighted that QTd is not merely a static measure, but is dynamically influenced by the autonomic nervous system; a blunted vagal tone—similar to that seen in impaired HRR— often exacerbates repolarization heterogeneity. By evaluating QTd in conjunction with the Gensini score, researchers can identify a "high-risk phenotype" characterized by both advanced anatomical stenosis and heightened electrical instability.[13]

Rate pressure product (RPP), calculated as systolic blood pressure (SBP) × heart rate, reflects myocardial oxygen demand; values exceeding 10,000 are associated with increased cardiovascular risk.[14]

This study evaluates the relationship between CAD severity and the HRR, QTd, and RPP parameters. This investigation appears to be the first to analyze the combined correlation of these parameters with the Gensini score.

PATIENTS AND METHODS

Study Population

This prospective observational study enrolled consecutive patients presenting with stable angina pectoris between May 1, 2021, and December 1, 2021. Inclusion criteria were age >18 years, no prior CAD history, and stable angina pectoris. Exclusion criteria included previous coronary interventions (percutaneous coronary intervention or coronary artery bypass grafting), arrhythmias, severe valvular disease, heart failure, exhibited conduction abnormalities (such as left bundle branch block, right bundle branch block, or pacemaker rhythms), baseline ECG changes interfering with QT measurement, presence of active infections, malignancies, hematological or immunological disorders, as well as end-stage renal or hepatic diseases. A total of 1154 patients were initially screened. After applying the exclusion criteria, 200 patients were eligible (Figure 1) and successfully completed the study protocol, which included EST and invasive coronary angiography (ICA).

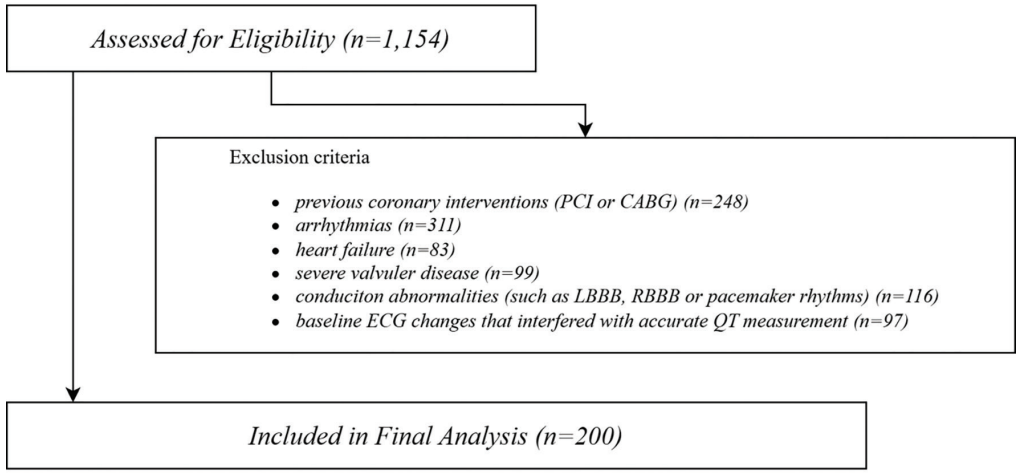

Figure 1. Flowchart of the study population recruitment and exclusion process.
CABG: Coronary artery bypass grafting; ECG: Electrocardiogram; LBBB: Left bundle branch block; PCI: Percutaneous coronary intervention; RBBB: Right bundle branch block.

Their EST parameters and coronary angiographic findings were analyzed to determine the relationship between autonomic/repolarization markers and the anatomical severity of CAD at the time of diagnosis.

Informed consent was obtained from all participants. Demographic, clinical, and laboratory data were recorded prior to coronary angiography (CAG).

QTd Measurement

QTd was calculated from a standard 12-lead resting ECG recorded at a paper speed of 25 mm/s and an amplitude of 10 mm/mV. The measurements were performed manually by two independent cardiologists who were blinded to the patients' clinical and angiographic data. To enhance precision, the ECG tracings were digitized and analyzed using high-resolution screen calipers with 3x magnification. The QT interval was measured from the onset of the QRS complex to the point where the descending limb of the T-wave met the isoelectric baseline. QTd was defined as the difference between the maximum and minimum QT intervals (QT_{max}-QT_{min}) across all measurable leads.

EST and HRR, DTS, RPP Calculation

All patients underwent a symptom-limited EST on a treadmill following the Bruce protocol. Upon reaching peak exercise, the test was not terminated abruptly; instead, an active recovery (cool-down) phase was performed, consisting of a 2-minute walk at 1.5 mph and 0% grade. HRR values were calculated as the absolute difference between the peak HR achieved during exercise and the HR recorded at specific recovery intervals:

HRR1: Calculated as (Peak HR- HR at 60 seconds of recovery).

HRR2: Calculated as (Peak HR- HR at 120 seconds of recovery).

In line with the active recovery protocol, an HRR1 value of ≤12 bpm was defined as abnormal, reflecting impaired vagal reactivation. For HRR2, values <22 bpm indicate an abnormal autonomic response.

The Duke treadmill score (DTS) was calculated using the standard formula: Exercise duration – (5×ST depression) – (4×Angina index).

Angina index:

Score 0: No angina occurs during exercise,

Score 1: Angina occurs during exercise but is not test-limiting (non-limiting angina),

Score 2: Angina is severe enough to cause termination of the test (exercise-limiting angina).

Myocardial oxygen demand was indirectly assessed by calculating the RPP. RPP was defined as the product of HR and systolic blood pressure, both measured at the peak of the EST (RPP = HR [bpm] × SBP [mmHg]).

CAG and Gensini Score

CAG was performed via the Judkins technique. Two blinded cardiologists assessed the results. CAD severity was quantified using the Gensini score, which weighs the degree of stenosis and vessel location. Critical coronary stenosis was defined as ≥50% narrowing in epicardial arteries. Patients were categorized into three groups: Normal coronary arteries, non-critical CAD (<50% stenosis or slow flow) and critical CAD.

Statistical Analysis

Analyses were performed using SPSS version 25.0. The sample size and statistical power were evaluated using G*Power software (version 3.1.9.7). A post-hoc power analysis based on the total study population of 200 participants revealed that the study had 92% statistical power to detect a medium effect size (d=0.5) with a two-tailed alpha level of 0.05. Normality was assessed using the Kolmogorov-Smirnov test. Continuous variables were expressed as mean ± SD or medians, and categorical variables as frequencies. One-Way ANOVA with Tukey's post-hoc test was used for comparisons among three groups. Pearson correlation was used to evaluate relationships between the Gensini score and EST and ECG parameters. Receiver operating characteristic (ROC) curve analysis, with Youden's index, determined optimal cut-offs, and the area under the curves (AUCs) were compared using the DeLong test. Multivariate logistic regression identified independent predictors of critical CAD. Statistical significance was set at $p<0.05$.

Ethics Committee Approval

The study was conducted in accordance with the Declaration of Helsinki, and approval was obtained from the Local Ethics Committee (University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital Ethics Committee, approval date: 19.04.2021, decision no: 2021-08-11).

RESULTS

Patient Characteristics

The study included 200 patients (57% male; mean age 56.21±9.35 years). Angiographic results identified normal coronary arteries in 27% of the population, non-critical CAD in 47%, and critical CAD in 26%. Demographic and laboratory findings were statistically similar across all groups ($p>0.05$) (Table 1).

Exercise and ECG Parameters

Significant variations were observed in exercise and ECG parameters across the study groups (Table 2). QTd values increased significantly with CAD severity and peaked in the critical CAD group ($p<0.001$). Conversely, HRR1, HRR2, DTSs, and RPP values significantly declined as disease severity progressed ($p<0.001$ for all measures except RPP, $p=0.003$). Post-hoc analyses confirmed that QTd and HRR parameters were statistically distinct for each group, indicating their discriminative potential.

Correlation and ROC Analysis

The mean Gensini score was 21.35±28.19 (range 0-166). Correlation analyses revealed that the Gensini score was strongly associated with QTd ($r=0.742$) and negatively associated with HRR1 ($r=-0.672$) and Duke scores ($r=-0.632$) ($p<0.001$ for all) (Figure 2a-c). ROC analysis identified QTd >32 ms (AUC: 0.842, 82% sensitivity, 76% specificity) and HRR1 <11 bpm (AUC: 0.815, 78% sensitivity, 80% specificity) as significant predictors of critical stenosis (Figure 3). According to the DeLong test, the AUCs for QTd and HRR1 were significantly higher than those of the Duke score and RPP ($p<0.05$), confirming their superior diagnostic performance.

Table 1. Baseline demographic, clinical, and laboratory characteristics of the study groups

Variables	Normal (n=54)	Non-critical CAD (n=94)	Critical CAD (n=52)	p-value
Age (years)	55.4±8.2	56.7±7.9	56.1±8.5	0.643[a]
Male gender, n (%)	30 (55.5%)	54 (57.4%)	30 (57.7%)	0.969[b]
Hypertension, n (%)	24 (44.4%)	43 (45.7%)	23 (44.2%)	0.983[b]
Diabetes mellitus, n (%)	15 (27.7%)	26 (27.6%)	14 (26.9%)	0.993[b]
Smoking, n (%)	19 (35.2%)	34 (36.2%)	18 (34.6%)	0.981[b]
Medications, n (%)				
ACE inhibitors/ARBs	24 (44.4%)	41 (43.6%)	23 (44.2%)	0.996[b]
Beta-blockers	11 (20.4%)	19 (20.2%)	10 (19.2%)	0.985[b]
Statins	17 (31.5%)	31 (33.0%)	17 (32.7%)	0.982[b]
Antiplatelet therapy	2 (3.7%)	4 (4.3%)	2 (3.8%)	0.986[b]
Laboratory parameters				
Glucose (mg/dL)	104.2±22.5	106.8±24.1	108.4±21.7	0.612[a]
Creatinine (mg/dL)	0.88 (0.80-0.96)	0.91 (0.82-1.02)	0.89 (0.81-0.98)	0.584[c]
Total cholesterol (mg/dL)	192.5±35.2	198.4±38.6	201.2±40.1	0.425[a]
LDL-cholesterol (mg/dL)	124.6±28.4	128.9±30.2	132.5±32.7	0.358[a]
HDL-cholesterol (mg/dL)	44 (38-51)	42 (36-49)	41 (39-42)	0.410[c]
Triglycerides (mg/dL)	154.3 (112.4-196.2)	162.7 (116.6-208.8)	165.4 (118.1-212.7)	0.635[c]
Hemoglobin (g/dL)	13.8±1.4	13.6±1.2	13.5±1.5	0.492[a]
WBC count (10^3/µL)	7.8±1.9	8.1±2.2	8.3±2.1	0.448[a]

Data are presented as mean ± SD, median (interquartile range), or n (%).
ACE: Angiotensin-converting enzyme; ARB: Angiotensin receptor blocker; CAD: Coronary artery disease; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; WBC: White blood cell; SD: Standard deviation; [a]: One-Way ANOVA; [b]: Chi-square or Fisher's exact test; [c]: Kruskal-Wallis test.

Table 2. Comparison of exercise stress test and ECG parameters among groups

Variables	Normal (n=54)	Non-critical CAD (n=94)	Critical CAD (n=52)	p-value
QTd (ms)	13.63±12.78[a]	22.62±8.79[b]	41.21±11.96[c]	<0.001
HRR1 (bpm)	33.33±9.94[a]	26.32±11.30[b]	9.79±2.15[c]	<0.001
HRR2 (bpm)	49.87±11.51[a]	43.63±13.18[b]	22.52±3.84[c]	<0.001
Duke score	3.20±1.69[a]	-1.46±5.24[b]	-6.29±3.66[c]	<0.001
RPP (bpm×mmHg)	26,850±4200[a]	25,120±5100[b]	22,450±4850[b]	0.003

Data are presented as mean ± standard deviation. P<0.05 was considered statistically significant.
CAD: Coronary artery disease; HRR1: Heart rate recovery at 1 minute; HRR2: Heart rate recovery at 2 minutes; QTd: QT dispersion; RPP: Rate-pressure product; [a]: Statistical significance was determined by One-Way ANOVA; [b]: Tukey's post-hoc test was used for multiple comparisons. Different superscript letters ([a, b, c]) in the same row indicate significant differences between groups (p<0.05).

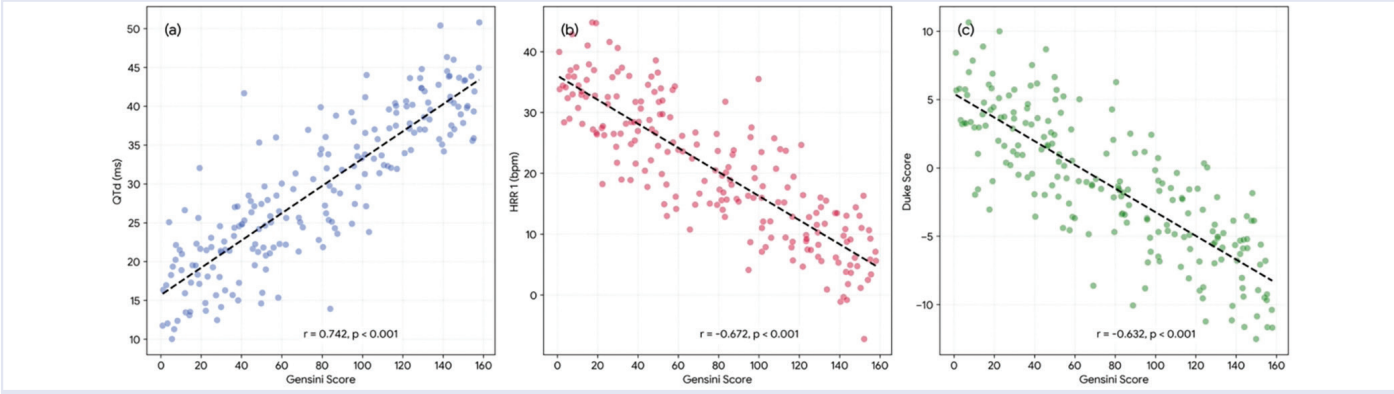

Figure 2. Scatter plots showing the Pearson's correlation coefficients between the Gensini score and study parameters: (a) strong positive correlation with QTd (r=0.742, p<0.001); (b) significant negative correlation with HRR1 (r=-0.672, p<0.001); (c) significant negative correlation with Duke treadmill score (r=-0.632, p<0.001).
QTd: QT dispersion; HRR1: Heart rate recovery at 1 minute.

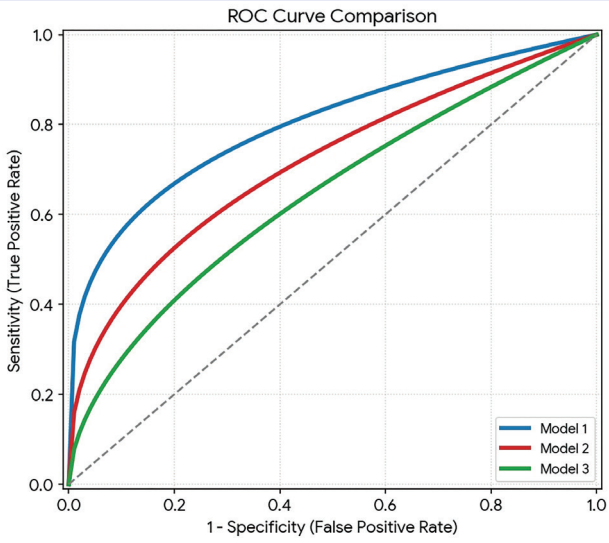

Figure 3. Comparison of receiver operating characteristic (ROC) curves of QTd, HRR1, and Duke score for the prediction of critical coronary artery disease. The AUCs were 0.842 for QTd, 0.815 for HRR1, and 0.760 for the Duke score.

QTd: QT dispersion; HRR1: Heart rate recovery at 1 minute; AUC: Area under the curve.

Logistic Regression Analysis

Univariate analysis showed that QTd, HRR1, HRR2, Duke score, and RPP were significantly associated with critical CAD. After adjusting for multicollinearity (excluding HRR2 due to VIF >10), multivariate logistic regression identified QTd (odds ratio [OR]: 1.09, 95% confidence interval [CI]: 1.04-1.14, p=0.002), HRR1 (OR: 0.82, 95% CI: 0.75-0.90, p=0.001), and Duke score (OR: 0.88, 95% CI: 0.81-0.96, p=0.015) as independent predictors of critical CAD [Table 3].

DISCUSSION

Our study demonstrates a strong negative correlation between HRR1 and the Gensini score, a finding that aligns with the results of Grad and Zdrenghea,[15] who established blunted HRR as a hallmark of autonomic dysfunction in patients with myocardial ischemia. Similar to our findings, Lachman et al.[16] reported that HRR values significantly decrease as the number of stenotic vessels increases.

However, some differences were observed when comparing our data to earlier studies. For instance, while some researchers reported a lower mean HRR1 value in CAD groups (e.g., <12 bpm), our value was 11 bpm.[16] This slight discrepancy may be attributed to our use of an active recovery protocol, which is known to produce higher HRR values than the passive supine recovery used in other cohorts. A notable difference in our study was the significant correlation of HRR2 with multi-vessel disease, which was more pronounced than in some previous reports.[17] We hypothesize that this reflects either more severe sympathetic overactivity or delayed catecholamine clearance in our specific patient population, highlighting the added value of measuring late-phase recovery in chronic coronary syndromes.

Regarding ventricular repolarization, we found a strong positive correlation between QTd and the Gensini score (r=0.742). This aligns with Bergfeldt et al.[18] and Dahrab et al.,[19] who proposed that QTd provides a non-invasive measure of repolarization heterogeneity related to the extent of coronary narrowing. With an AUC of 0.842, our findings confirm that a resting QTd >32 ms is a robust predictor of critical stenosis.

A noteworthy aspect of our study design is the use of the EST as a gateway to ICA. We acknowledge that the 2019 and 2024 European Society of Cardiology (ESC) Guidelines for chronic coronary syndromes have shifted the diagnostic paradigm away from EST toward non-invasive anatomical or functional imaging, such as coronary computed tomography angiography or stress echocardiography.[20] However, we hypothesized that the diagnostic sensitivity and specificity of EST for detecting CAD could be significantly enhanced by integrating it with HRR and QTd. By identifying patients with a high "anatomical burden" (as shown by higher Gensini scores) using these autonomic and electrical markers, our study provides evidence that EST can offer vital insights into coronary plaque load and myocardial vulnerability, particularly in settings where advanced imaging is not immediately available.

Recent large-scale meta-analyses have reaffirmed the prognostic power of HR dynamics in cardiovascular health. For instance, Qiu et al.[21] demonstrated that each 10-bpm decrease in HRR was associated with a 13% increase in the risk of cardiovascular events, emphasizing HRR as a fundamental marker of autonomic stability. Furthermore, contemporary research by Giga et al.[22] has highlighted that impaired HRR is not only a marker of mortality but also a significant predictor of the complexity and anatomical extent of coronary lesions. In alignment with these findings, our study shows that lower HRR1 and

Table 3. Univariate and multivariate logistic regression analysis of independent predictors for critical coronary artery disease

Variables	Univariate OR (95% CI)	p-value	Multivariate OR (95% CI)	p-value
Age	1.02 (0.98-1.06)	0.342	-	NS
Hypertension	1.15 (0.85-1.55)	0.412	-	NS
Diabetes mellitus	1.20 (0.88-1.64)	0.285	-	NS
QTd	1.12 (1.08-1.16)	<0.001	1.09 (1.04-1.14)	0.002
HRR1	0.78 (0.72-0.85)	<0.001	0.82 (0.75-0.90)	0.001
Duke score	0.82 (0.76-0.88)	<0.001	0.88 (0.81-0.96)	0.015
HRR2	0.85 (0.80-0.91)	<0.001	-	NS†
RPP	0.98 (0.96-1.01)	0.078	-	NS

CI: Confidence interval; HRR1: Heart rate recovery at 1 minute; HRR2: Heart rate recovery at 2 minutes; NS: Non-significant; OR: Odds ratio; QTd: QT dispersion; RPP: Rate-pressure product; p<0.05 was considered statistically significant; †: HRR2 was excluded from the multivariate model due to high multicollinearity with HRR1 (variance inflation factor >10).

HRR2 values directly correlate with higher Gensini scores, suggesting that autonomic dysfunction parallels the atherosclerotic burden. Similarly, recent studies on repolarization indices have underscored the value of QTd in identifying high-risk subsets among stable patients.[23] By integrating these modern insights with our findings, we suggest that the synergistic use of HRR and QTd enhances the diagnostic yield of EST beyond traditional ST-segment analysis, consistent with the evolving recommendations of the 2024 ESC Guidelines, which prioritize comprehensive risk stratification.

In our multivariate analysis, RPP did not emerge as an independent predictor of critical CAD. While the confounding influence of beta-blocker therapy on HR and blood pressure dynamics partially explains this finding, the physiological limitations of RPP itself must be considered. Although RPP is commonly used as a surrogate for myocardial oxygen consumption (MVO_2), it primarily reflects the heart's external work. As demonstrated in the seminal study by Kal et al.,[24] RPP may not fully account for all determinants of MVO_2, such as ventricular wall tension and contractility, particularly in the presence of obstructive CAD. Our findings suggest that markers of autonomic reactivation and repolarization heterogeneity provide more granular insights into the anatomical severity of CAD than traditional hemodynamic indices like RPP.

The present study demonstrates that impaired HRR1 and HRR2, increased QTd, and lower DTSs are significantly associated with the anatomical severity of CAD. Multivariate analysis identified QTd, HRR1, and DTS as independent predictors of critical CAD, suggesting that these non-invasive markers provide valuable diagnostic insights regarding the atherosclerotic burden prior to angiography.

To our knowledge, this is the first study to specifically investigate the correlation between HRR1 and the Gensini score. Although QTd and DTS require detailed calculations, HRR1 stands out as a more practical and easily measurable parameter in daily clinical practice. Our results demonstrate that this simple index not only reflects autonomic dysfunction but also serves as a robust indicator of the presence and severity of coronary atherosclerosis.

Our study has several limitations. First, it was a single-center study with a relatively small sample size, which may limit the generalizability of our findings. Second, the use of medications such as beta-blockers and calcium channel blockers, although similar across groups, might have influenced the HR and blood pressure responses during exercise. Finally, CAG is a 2D lumen-based assessment; hence, more advanced imaging such as intravascular ultrasound could provide a more detailed analysis of the atherosclerotic plaque burden.

In conclusion, QTd and HRR1 are powerful, non-invasive, and easily obtainable parameters that correlate strongly with the anatomical severity of CAD as measured by the Gensini score. A resting QTd >32 ms and an HRR1 <11 bpm are independent predictors of critical coronary stenosis. Incorporating these markers into routine EST evaluations can significantly enhance risk stratification and assist clinicians in the early identification of patients requiring invasive intervention.

Ethics

Ethics Committee Approval: The study was conducted in accordance with the Declaration of Helsinki, and approval was obtained from the Local Ethics Committee (University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital Ethics Committee, approval date: 19.04.2021, decision no: 2021-08-11).

Informed Consent: Informed consent was obtained from all participants.

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For transparency, the authors note that an artificial intelligence-assisted language model (ChatGPT, OpenAI) was utilized to support language correction. This assistance was limited to linguistic refinement; all scientific content, critical analysis, and final editorial decisions were made exclusively by the authors.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ö.K., R.A., F.N.T.Ç.; Concept: Ö.K., R.A., F.N.T.Ç.; Design: Ö.K., R.A., F.N.T.Ç.; Data Collection or Processing: Ö.K., R.A., F.N.T.Ç.; Analysis or Interpretation: Ö.K., R.A., F.N.T.Ç.; Literature Search: Ö.K., R.A., F.N.T.Ç.; Writing: Ö.K., R.A., F.N.T.Ç.

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