ORIGINAL RESEARCH

Athens QRS Score as a Predictor of Coronary Artery Disease in Patients With Chest Pain and Normal Exercise Stress Test

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***Background*—**The diagnostic value of the Athens QRS score to detect obstructive coronary artery disease CAD in patients with otherwise normal exercise stress test remains unclear.

***Methods and Results*—**We analyzed 458 patients who underwent exercise stress test with or without myocardial perfusion imaging within 2 months of coronary angiography from 2008 to 2011. Patients (n=173) with abnormal stress test based on ST segment criteria were excluded. The Athens QRS score ≤5 was defined as abnormal. In our study cohort, 285 patients met the inclusion criteria and were divided into 2 groups: low Athens QRS score (LQRS, n=56), with QRS score ≤5 and normal Athens QRS score normal Athens QRS score, n=229), with QRS score >5. The presence of single-vessel and multivessel obstructive CAD was higher in LQRS than in normal Athens QRS score patients (47% versus 7.5% and 30% versus 3.8%, respectively, all $P<0.001$). Logistic regression analysis showed that the likelihood of CAD was strongly and independently associated with LQRS (odds ratio=36.81, 95% CI: 10.77–120.47), diabetes (odds ratio=6.49, 95% CI: 2.41–17.49), lower maximum heart rate (odds ratio=0.92, 95% CI: 0.88–0.95, all $P<0.001$), and older age (odds ratio=1.93, CI: 1.88–1.97, $P=0.002$).

***Conclusions*—**In a clinical cohort of patients with chest pain and normal exercise stress test, LQRS score is a strong independent predictor of presence of CAD. LQRS patients have a 6-fold higher prevalence of CAD and may warrant further evaluation even with reassuring exercise stress test. **(*J Am Heart Assoc*. 2016;5:e002832 doi: 10.1161/JAHA.115.002832)**

Key Words: coronary artery disease • exercise stress test • QRS score

Exercise stress test (EST) is one of the most common noninvasive modalities used for coronary artery disease (CAD) detection, in spite of the limitations including low sensitivity and specificity. The sensitivity and specificity of the conventional ST-segment depression criteria during EST has been reported between 47% and 91% and 69% and 97%, respectively.[1–6] In a meta-analysis involving 24 074 patients, EST showed a mean sensitivity and specificity of 68% and 77%, respectively.[7] Other electrographic and nonelectrocardiographic criteria have been proposed to improve the diagnostic efficacy of EST for CAD detection and quantification. These parameters include exercise-induced ST-segment depression and heart rate (HR)–adjusted ST depression (ST/HR index), ST-segment shift relative to exercise-induced increments in HR (ST/HR slope), and Q-, R-, and S-wave amplitude changes observed on ECGs during exercise stress testing and nonelectrocardiographic-based parameters including HR, sex, chest pain type, and systolic blood pressure.[8–17] Among these parameters, the Athens QRS score, which is based on immediate postexercise changes in the Q-, R-, and S-wave amplitudes, proposed by Michaelides et al,[18] has been demonstrated to be highly predictive of CAD. Previous studies have revealed the value of the Athens QRS score in conjunction with ST depression in detecting CAD.[18–24] However, its significance in patients with suspected CAD and normal EST, without exercise-induced ST depression was not studied. Accordingly, we sought to determine the predictive and diagnostic value of the Athens QRS score in patients with otherwise normal EST based on the conventional ST-segment depression criteria.[19]

Methods

After obtaining approval from the Institutional Board Review, we retrospectively analyzed 458 patients who underwent EST with or without technetium-99 scintigraphy within 2 months of coronary angiography at Bronx Lebanon Hospital Center.

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Requirement for informed consent was waived. The indications for cardiac catheterization were as follows: recurrent admissions secondary to ongoing chest pain, positive imaging portion of EST, or high clinical suspicion for CAD (based on American College of Cardiology Foundation/American Heart Association 2011 guidelines).[24] The cohort predominantly comprised Hispanic and African American patients, groups that have been grossly unrepresented in clinical studies. Patients with abnormal EST results, based on ST-segment criteria, as well as patients with a bundle branch block, left ventricular hypertrophy (based on ECG and echocardiography criteria) and the ones on digitalis at rest, were excluded from the study (n=173). The Athens QRS score ≤5 was defined as abnormal.[18,19,23] Two hundred eighty-five patients met the inclusion criteria and were divided into 2 groups: low Athens QRS score (LQRS, n=56 patients) and normal Athens QRS score (NQRS, n=229 patients).

Diabetes mellitus was defined as the presence of a fasting blood sugar ≥126 mg/dL, self-reported diabetes, or use of an antihyperglycemic drug.[25] Hypertension was defined as having as systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg or use of antihypertensive medications. Smoking was determined by patient history and was categorized as ever versus never. Dyslipidemia was defined as fasting serum total cholesterol concentration of >220 mg/dL, low-density lipoprotein cholesterol concentration >140 mg/dL, high-density lipoprotein cholesterol concentration <40 mg/dL, or triglyceride concentration >150 mg/dL or as taking lipid-lowering medications. Obesity was defined as a body mass index ≥30. Patients with a history of myocardial infarction on chart review, pathological Q waves on the ECG, or history of prior percutaneous coronary intervention, coronary artery bypass graft, any revascularization, or symptomatic heart failure were excluded from the analysis.

All subjects underwent the Bruce Stress protocol as a part of EST. The protocol consisted of 3-minute stages with increasing speed and incline (incremental increase of workload). Patients were monitored using a 12-lead ECG. Printouts of the ECG tracings on paper and digital formats were obtained at rest and at 3-minute intervals. Upon completion of the exercise protocol, the ECG was also recorded. The patient's condition was monitored, including blood pressure, which was measured at the beginning of the test and at the end of each stage. All of the tests were performed using a Quinton Q-Stress TM55 Treadmill and a Q-Stress V3.5 Exercise Test Monitor (Cardiac Science, Waukesha, WI). The PDF files of the ECGs were extracted and analyzed using Adobe Acrobat Professional (Adobe Systems, San Jose, CA).The Distance Tool was used to measure amplitudes of Q-, R-, and S-waves.[26,27]

The ST-segment criteria that were used to exclude patients from this study were the following: A horizontal ST-segment depression of ≥1 mm at 60 ms beyond the J point, a downsloping ST-segment depression of ≥1 mm at the J point, and an upsloping ST-segment depression of ≥2, 80 ms beyond the J point.[18,19]

In order to avoid measurement and expectation bias, the EST ECGs were evaluated by 2 physicians, including an electrophysiologist (E.S.), who were blind to the medical history and coronary angiography results. The QRS scores were calculated using the algorithm described earlier by Michaelides et al.[18] The ECGs from the patients at rest and immediately after exercise (at t=0 s) were evaluated. The amplitudes of Q-, R-, and S-waves were measured in lead aVF and V5 (Figures 1 and 2). These amplitudes were measured from the iso-electric line to the peak of the R-wave and to the nadir of the Q- and S-waves. The score was then calculated by subtracting postexercise changes in the Q-, R-, and S-wave amplitudes in leads aVF and V5 compared to rest. The resulting values from these 2 leads were then added to generate the QRS score for that particular individual as shown below.

$$\text{QRS score} = (\Delta R - \Delta Q - \Delta S)_{aVF} + (\Delta R - \Delta Q - \Delta S)_{V5}$$

where ΔR=amplitude of R-wave at rest−amplitude of R-wave at immediately after exercise; ΔQ=amplitude of Q-wave at rest−amplitude of Q-wave at immediately after exercise; ΔS=amplitude of S-wave at rest−amplitude of S-wave at immediately after exercise.

Significant CAD was defined as angiographic evidence of ≥70% stenosis in any major vessel or ≥50% in left main coronary artery.[28,29]

A statistical analysis was performed using SPSS software version 23.0. Normally distributed, continuous variables were expressed as mean±SD and were compared by using 2-tailed unpaired *t* test. Chi-square and Fisher exact tests were used

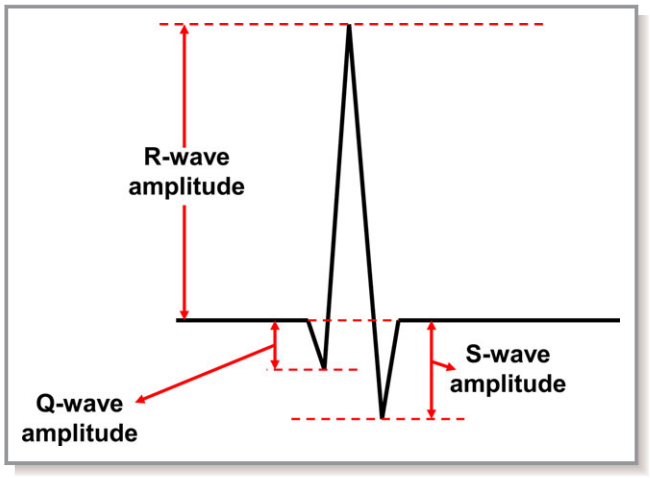

Figure 1. This figure illustrates the measurement of the Q-, R-, and S-waves. These amplitudes were measured from the iso-electric line to the peak of the R-wave and to the nadir of Q- and S-waves.

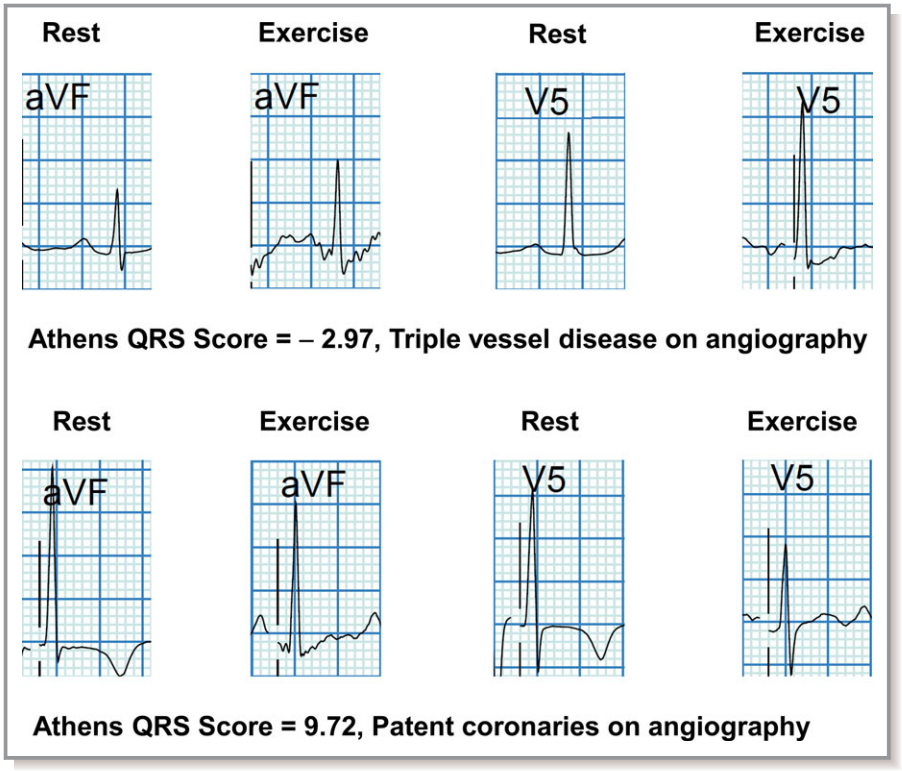

Figure 2. This figure demonstrates a comparison of exercise-induced changes in the Q-, R-, and S-waves in lead aVF and V_5 between a patient with triple vessel disease and a patient with patent coronaries evident on coronary angiography. The Athens QRS score is very low in a patient with triple vessel disease (−2.97) as compared to a patient with patent coronaries (+9.72).

for categorical variables. Cross-tabulation was used to check the independence among mutually exclusive variables. A *P*-value ≤0.05 was considered statistically significant. In this study, we used binary logistic regression analysis applying the backwards stepwise method with CAD as a dependent variable and demographical risk factors and stress parameters as independent variables to see the independent predictors of CAD. The variables that were included in the first model were QRS score, hypertension, diabetes, dyslipidemia, obesity, history of myocardial infarction, history of smoking, sex, age, resting HR, systolic and diastolic blood pressures, maximum HR, systolic and diastolic blood pressures, Metabolic equivalent of task (METs) and left ventricular ejection fraction. In subsequent steps, variables were removed 1 by 1. In the final model (step number 13) the variables included were QRS score, diabetes, age, maximum HR, and METs. The statistical tests were adjusted for small sample sizes or any bias by using the bootstrapping analysis. Sensitivity and specificity were calculated by standard methods.

Results

As shown in Tables 1 and 2, LQRS and NQRS patients were similar in their clinical and demographic variables. There were 3 patients in each group with left ventricular ejection fraction <50%. These patients did not have symptomatic congestive heart failure by history (Table 1). Among EST parameters, the maximum HR and mean maximum systolic blood pressure were lower and maximum diastolic blood pressure was higher in patients with LQRS. The number of patients who reached 85% of the predicted maximum HR was also significantly lower in patients with LQRS. Among demographic characteristics and EST parameters grouped based on presence or absence of CAD, diabetes, lower maximum HR, and lower QRS score were found to be significantly correlated with presence of CAD as seen in Table 3.

Single-vessel CAD, as well as multivessel CAD, were more prevalent among patients with LQRS compared to NQRS patients as seen in Figure 3 (47% versus 7.5% and 30% versus 3.8%, respectively, with $P<0.001$). The overall prevalence of any type of obstructive (single- or multivessel) CAD was also higher in LQRS patients compared to NQRS patients (77% versus 11.3%, $P<0.001$). In bivariate analysis, LQRS, lower maximum HR and maximum systolic blood pressure, and diabetes mellitus correlated significantly with prevalence of CAD (all $P<0.05$). The sensitivity and specificity of LQRS for detection of CAD were 62% and 94%, respectively.

Table 1. Clinical and Demographic Characteristics of Patients Grouped Based on Athens Score

Demographics	LQRS, n=56	NQRS, n=229	*P* Values
Mean age, y	55.0±9.1	54.6±9.8	0.814*
Males	30 (54%)	115 (50%)	0.653†
Hispanics	36 (64%)	118 (52%)	0.223†
African Americans	16 (29%)	86 (37%)	
Others	4 (7%)	25 (11%)	
Diabetes	32 (57%)	120 (52%)	0.553†
Hypertension	46 (82%)	190 (83%)	0.883†
Dyslipidemia	32 (57%)	121 (53%)	0.563†
Obese	32 (57%)	123 (54%)	0.644†
Mean BMI, kg/m^2	30.6±4.8	30.3±5.4	0.625*
History of MI	1 (1.8%)	0 (0%)	0.196‡
History of smoking	10 (18%)	38 (17%)	0.821†
EF <50%	3 (5.3%)	3 (1.3%)	0.204‡

BMI indicates body mass index; EF, ejection fraction; LQRS, low Athens QRS score; MI, myocardial infarction; NQRS, normal Athens QRS score.
*Two-tailed *t* test.
†Chi square/cross-tab test.
‡Fisher exact test.

In binary logistic regression analysis with CAD as a dependent variable and stress parameters as independent variables, LQRS, diabetes mellitus, lower maximum HR, and older age were strongly and independently associated with presence of CAD (P<0.05) (Table 4). As resting HR and maximum HR, resting systolic blood pressure and maximum systolic blood pressure, and resting diastolic blood pressure and maximum diastolic blood pressure are collinear; therefore, a series of multivariate models were performed substituting 1 collinear variable for the other. All these models confirmed the strong independent association of LQRS (odds ratio range from 31 to 67) and diabetes mellitus (odds ratio range from 6.4 to 7.5) to CAD (data not shown). There were

Table 2. Stress Test Parameter Characteristics of Patients Grouped Based on Athens Score

	LQRS	NQRS	*P* Values
Exercise stress test	15 (26%)	69 (30%)	0.631*
NST Bruce protocol	41 (74%)	160 (70%)	0.631*
Positive MPI among patients with NST Bruce protocol	15 (27%)	50 (21%)	0.576*
Resting heart rate, bpm	81±9.8	78±11.0	0.065†
Resting systolic blood pressure, mm Hg	127±9.9	126±9.4	0.481†
Resting diastolic blood pressure, mm Hg	80±8.9	79±7.3	0.381†
Maximum heart rate, bpm	133±14.5	149±13.2	<0.001†
Maximum systolic blood pressure, mm Hg	162±12.2	169±2.1	<0.001†
Maximum diastolic blood pressure, mm Hg	101±3.5	95±8.1	<0.001†
METs	9.65±1.89	10.1±2.15	0.085†

bpm indicates beats per minute; LQRS, low Athens QRS score; METS, metabolic equivalent of task; MPI, myocardial perfusion imaging; NQRS, normal Athens QRS score; NST, nuclear stress test.
*Chi square/cross-tab test.
†Two-tailed *t* test.

Table 3. Clinical, Demographic, and Stress Test Characteristics of Patients Grouped Based on Coronary Artery Disease (CAD)

Demographics	CAD Present, N=69	CAD Absent, N=216	*P* Value
Mean age, y	54.4±10.2	54.8±9.5	0.759*
Males	32 (46%)	113 (52%)	0.391†
Hispanics	42 (61%)	114 (52%)	
African Americans	21 (30%)	81 (37%)	0.493†
Others	6 (8.7%)	21 (9.7%)	
Diabetes	51 (74%)	101 (47%)	<0.001†
Hypertension	57 (83%)	179 (82%)	0.974†
Dyslipidemia	41 (59%)	112 (52%)	0.273†
Obese	36 (52%)	119 (55%)	0.675†
BMI, kg/m^2	30.2±5.1	30.3±5.3	0.806*
History of MI	0 (0%)	1 (0.5%)	1.000‡
History of smoking	11 (16%)	37 (17%)	0.821†
Stress test parameters			
Resting heart rate	79.1±9.6	78.3±11.2	0.588*
Resting systolic blood pressure, mm Hg	127.1±9.7	127.0±9.5	0.956*
Resting diastolic blood pressure, mm Hg	78.8±8.5	79.4±7.3	0.579*
Maximum heart rate	134.1±14.2	149.4±13.3	<0.001*
Maximum systolic blood pressure, mm Hg	164.9±12.5	168.57±12.2	0.036*
Maximum diastolic blood pressure, mm Hg	98.0±14.1	95.9±8.2	0.258*
METs	10.1±1.9	10.0±2.1	0.868*
LVEF	57.9±8.5	59.2±6.4	0.273*
QRS score	3.9±4.5	10.5±3.2	<0.001*

BMI indicates body mass index; LVEF, left ventricular ejection fraction; METs, metabolic equivalent of task; MI, myocardial infarction.
*Two-tailed *t* test.
†Chi square/cross-tab test.
‡Fisher exact test.

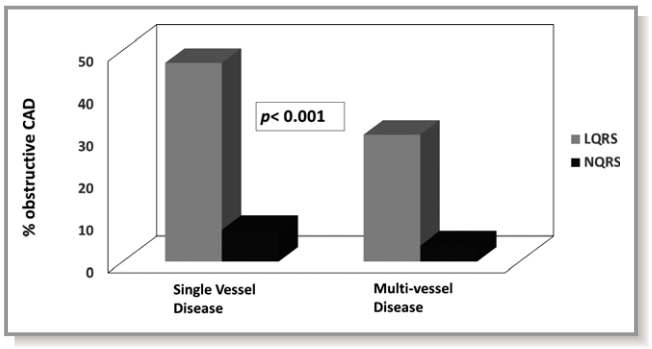

Figure 3. CAD in LQRS patients and NQRS patients. LQRS indicates patients with abnormal QRS score and no ST depression; NQRS, patients with normal QRS score and no ST depression.

no interactions between independent variables in the multivariate model.

Discussion

In this study, we evaluated the predictive and diagnostic value of LQRS score in patients with otherwise normal EST, without exercise-induced ST depression, and found the LQRS to be a strong independent predictor of presence of CAD. LQRSs were found to have 6-fold higher prevalent (77 versus 11.3%) CAD even with reassuring exercise stress test and no ST-segment depression compared to NQRS patients. Other independent predictors of CAD were diabetes mellitus, older age, and lower maximum HR achieved during stress test similar to previous reports.[17–19,30] Diabetes mellitus is a well-established risk factor for CAD. Krul et al, in their study, demonstrated diabetes to be an independent predictor of more extensive CAD as well as more obstructive CAD, particularly in women.[31] Our study showed that maximum HR achieved during stress test is also independently associated with CAD. The group with higher prevalence of CAD had a lower mean maximum HR. This is most likely due to the fact that patients with significant CAD do not achieve the maximum predicted HR during stress test, with chest pain limiting exercise duration. The sensitivity and specificity were also in concordance with those of previous literature.[18–23]

Table 4. Logistic Regression Analysis With Coronary Artery Disease as Dependent Variable*

Variable	Odds Ratio	95% CI		*P* Values
		Lower	Upper	
Athens QRS score	36.988	11.022	124.124	<0.001
Diabetes mellitus	6.052	2.247	16.298	<0.001
Maximum heart rate	0.915	0.880	0.952	<0.001
Age	0.928	0.882	0.973	0.003
Male	0.461	0.192	1.106	0.083
METs	1.192	0.970	1.464	0.094
Obesity	0.514	0.222	1.191	0.121
Dyslipidemia	1.865	0.791	4.399	0.155
Resting heart rate	0.978	0.941	1.018	0.280
Resting diastolic BP	0.972	0.909	1.040	0.414
Maximum diastolic BP	0.985	0.938	1.034	0.549
Hypertension	1.103	0.354	3.434	0.866
Resting systolic BP	1.005	0.960	1.052	0.819
Maximum systolic BP	0.993	0.957	1.030	0.694
LVEF	0.979	0.920	1.041	0.491

BP indicates blood pressure; LVEF, left ventricular ejection fraction; METs, metabolic equivalent of task.
*Binary stepwise logistic regression analysis.

EST is the most frequent, noninvasive modality used as the initial evaluation for CAD detection in patients presenting with chest pain. Electrocardiographic criteria for CAD detection using EST are based on standardized ST-segment displacement. A complete dependence on the conventional ST-segment depression for detection of CAD or stress-induced myocardial ischemia decreases the sensitivity of the EST and could lead to an increased number of false-negative tests.[1–23,30–34] A standardized EST detects more severe CAD such as obstructive multivessel disease or left main, but may miss those with less severe disease, resulting in false-negative results.[34] Great effort has been made to improve the efficacy of EST by combining more ECG and non-ECG parameters for quantifying and detecting CAD.[8–17] Immediate postexercise changes in Q-, R-, and S-waves have been studied in patients with CAD. Michaelides et al[18] published a new diagnostic criterion and created an algorithm called Athens QRS score, which incorporates postexercise amplitude changes in Q-, R-, and S-waves into 1 index. This index inversely correlated with the extent of myocardial ischemia and with a number of obstructive coronary arteries. Following Michaelides et al, several other studies have been done to evaluate the value of Athens QRS scores in conjunction with the traditional ST-segment depression criteria.[18–23]

The Q-wave amplitude typically increases in individuals with normal coronaries and decreases or fails to increase in patients with CAD. The mechanism of this discrepancy may be associated with the loss of contraction in the septal region as a result of abnormal activation of the septum caused by left anterior descending artery stenosis.[12,15,18]

The postexercise R-wave amplitude typically decreases in normal individuals and generally increases in patients with CAD.[8–11,13,14] This was initially explained by the phenomenon called the "Brody Effect," in which the larger R-wave amplitude is caused by the increase in myocardial mass as a consequence of an increase in left ventricular volume in

patients with reduced myocardial contractility.[35] Later on, the more accepted explanation of R-wave amplitude change was based on animal experiments, which showed that the myocardial conduction velocity increases for a very short period of time (≈30 s) during ischemia and decreases afterwards. This change is thought to be associated with a similar biphasic change in the R-wave amplitude during ischemia (ie, a short duration decrease followed by an increase).[8,10] This conduction delay, which is caused by a delayed depolarization in parts of the left ventricle, can cause an increase in the size of QRS deflection.[8,10] On a molecular level, this theory can be explained by a progressive elevation in extracellular potassium concentration due to leakage from the ischemic myocardial cells that boosts the conductivity in the beginning and then reduces it afterwards, thus producing the R-wave amplitude changes.[10] The mechanism of postexercise S-wave amplitude changes is not very clear.[16]

We believe that patients who develop significant ischemia during EST have decreased myocardial contractility with a relative increased intraventricular cavity size, similar to transient ischemic dilation.[36] This is expressed as an increase of amplitude of R-wave as predominant representation of ventricular depolarization on the ECG with the caveat that R-wave change in 1 lead could be represented as S-wave or Q-wave change in other leads at the same time. We acknowledge that this is an inherent limitation of the Athens QRS score and other methods that utilize ECG criteria for detecting CAD.

Michaelides et al[18] conducted a study involving 274 patients. Their results revealed an inverse correlation of QRS score with CAD extensiveness, exercise-induced myocardial perfusion defects detected by stress myocardial perfusion (single photon emission computed tomography), and segmental wall motion defects evident by radionuclide ventriculography. In another prospective study, they found LQRS score having sensitivity of 86% and specificity of 79%. These sensitivity and specificity values were higher than the individual Q-wave (75% and 50%), R-wave (65% and 55%), and S-wave (70% and 10%) as well as ST depression (62% and 70%) values found in their study. In 1996, Van Campen et al[19] demonstrated a significant inverse correlation of QRS score with the extent of ischemia detected by single photon emission computed tomography with sensitivity and specificity of 88% and 85%, respectively. As indicated in previous studies, exercise-induced ST-segment depression had a lower sensitivity (53%) for ischemic heart disease detection. They also found the predictive accuracy of QRS score to be higher than any other Q-, R-, or S-wave amplitude changes.

Toth et al,[20] in their study, correlated the QRS scores and cumulative ST depression to the findings of single photon emission computed tomography or coronary angiography. They found significant inverse correlation between QRS score and severity of coronary artery disease visualized on angiography and perfusion defects detected by myocardial perfusion imaging. However, in this study, the cumulative ST-segment depression did not correlate significantly with QRS scores and the QRS scores were unrelated to the exercise-induced ST depression. In a study cohort consisting of 135 patients, Furuse et al found QRS score sensitivity and specificity to be 70% and 61%, respectively.[21] In a study by Cin et al[22] using a QRS score cut-off point of <5 mm for mild-to-moderate and <3 mm for high-risk patients, they showed that a QRS score <5 mm was correlated with significant and extensive CAD. Rajput et al,[23] in a prospective study involving 40 patients, revealed a significant inverse correlation of QRS score to the CAD severity with an overall sensitivity and specificity 92% and 80% using a cut-off of ≤5 mm compared to the ST-segment displacement with sensitivity and specificity of 72% and 67%, respectively.

In our study, the sensitivity and specificity were found to be 62% and 94%, for CAD detection, respectively. A high specificity can be explained with the fact that we excluded all patients with ST depression during EST. In absence of ST depression, a QRS score of >5 gives a higher specificity to the test (ie, there is less of a chance for a subject to have an obstructive CAD). In all the investigations mentioned above, Athens QRS scores were utilized to evaluate and confirm the usefulness and sensitivity, as well as specificity, of QRS scores either in comparison with the ST-segment depression criteria or in conjunction with it. Our study extends the utility of the Athens QRS score in CAD detection in the group of patients who have otherwise normal EST, and may provide an additional noninvasive method for identifying patients with CAD.

Our sample size (n=56) in the study group may be considered small; however, our criteria used to determine the study group (ie, group of patients with abnormal QRS score and otherwise normal ST-segment responses during EST who underwent coronary angiography within 2 months) are rigorously stringent (ongoing chest pain, high clinical suspicion for CAD or positive imaging portion of EST). This is because most patients who have normal exercise stress test with no ST depression do not undergo coronary angiography. However, as per American College of Cardiology/American Heart Association guidelines, patients with recurrent chest pain are class II indication for coronary angiography.[24] Our study was done predominantly on Hispanic and African American patients and therefore may not be applicable in patients of other ethnicities. However, we consider this a strength of the study, as these groups are grossly underrepresented in clinical studies. Our study was the first to evaluate the utility of the Athens score in patients with normal EST; therefore, prospective studies are needed in large, diverse populations.

In a clinical cohort of patients with chest pain and normal EST, LQRS score is a strong independent predictor of presence of CAD. LQRS patients have a 6-fold higher risk of

having CAD and may warrant further evaluation with another imaging modality, such as nuclear EST, stress echocardiography, or cardiac catheterization. Patients with LQRS should be evaluated further for presence of CAD even without any exercise-induced ischemic changes based on the conventional ST-segment criteria. Furthermore, patients with recurrent or persistent chest pain, but normal QRS score, and absence of ST depression, may not require further invasive testing. This score provides not only a useful index for the diagnosis but also for the assessment of CAD severity.[18–23] Calculation of Athens score can be incorporated in the software in currently available exercise stress testing equipment with minimal cost and difficulty. The QRS score can be calculated routinely with additional predictive value for CAD and its severity.

Disclosures

None.

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