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Electrocardiographic QRS-T angle and the risk of incident silent myocardial infarction in the Atherosclerosis Risk in Communities study

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Abstract

Background—Silent myocardial infarction (SMI) accounts for about half of the total number of MIs, and is associated with poor prognosis as is clinically documented MI (CMI). The electrocardiographic (ECG) spatial QRS/T angle has been a strong predictor of cardiovascular outcomes. Whether spatial QRS/T angle also is predictive of SMI, and the easy-to-obtain frontal QRS/T angle will show similar association are currently unknown.

Methods—We examined the association between the spatial and frontal QRS/T angles, separately, with incident SMI among 9498 participants (mean age 54 years, 57% women, and 20% African-American), who were free of cardiovascular disease at baseline (visit 1, 1987–1989) from the Atherosclerosis Risk in Communities (ARIC) study. Incident SMI was defined as MI occurring after the baseline until visit 4 (1996–1998) without CMI. The frontal plane QRS/T angle was defined as the absolute difference between QRS axis and T axis. Values greater than the sex-specific 95th percentiles of the QRS/T angles were considered wide (abnormal).

Results—A total of 317 (3.3%) incident SMIs occurred during a 9-year median follow-up. In a model adjusted for demographics, cardiovascular risk factors and potential confounders, both abnormal frontal (HR 2.28, 95% CI 1.58–3.29) and spatial (HR 2.10, 95% CI 1.44–3.06) QRS/T angles were associated with an over 2-fold increased risk of incident SMI. Similar patterns of associations were observed when the results were stratified by sex.

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Disclosures

The authors have no conflicts of interests to disclose.

Conclusions—Both frontal and spatial QRS/T angles are predictive of SMI suggesting a potential use for these markers in identifying individuals at risk.

Keywords

Electrocardiography; QRS/T angle; Silent myocardial infarction

Introduction

Heart disease still is the leading cause of death globally. In 2013, about 14% of total deaths in the United States were due to coronary heart disease (CHD), with over 660,000 new myocardial infarctions (MI) with clinical manifestations (CMI) as well as 160,000 accidentally discovered asymptomatic silent MIs (SMI) [1–3]. Reported SMIs thus accounted for over 20% of all new MIs [3–9]. In a recent analysis from the Atherosclerosis Risk in Communities (ARIC) study [10], we showed that asymptomatic or SMI, accounted for about 45% of the total number of MIs in the study. These SMIs were associated with an increased risk of death comparable to that of CMI.

Abnormally wide spatial QRS/T angle has been repeatedly shown to be among the strongest ECG markers of abnormal repolarization predictive of cardiovascular disease (CVD) events and mortality [11–20]. Nevertheless, computation of the spatial QRS/T angle from the standard 12-lead ECG requires several steps which is a challenge in its wide utilization. On the other hand, the frontal plane QRS/T angle could be easily calculated as the absolute value of the difference between QRS axis and T axis. Whether spatial QRS/T angle is predictive of SMI and whether the easy-to-obtain frontal QRS/T angle will show similar association with SMI are currently unknown. Therefore, the primary objective of the present study was to perform a comparative evaluation of the utility of the frontal plane and spatial QRS/T angle as predictors of incident SMI.

Methods

The Atherosclerosis Risk in Communities (ARIC) study prospectively enrolled 15,792 men and women between 45 and 64 years of age, which is a population-based multicenter study designed to investigate the natural history and cause of atherosclerotic and CVD from 4 US communities: Forsyth County, North Carolina; Jackson, Mississippi; suburbs of Minneapolis, Minnesota; and Washington County, Maryland. Eligible participants were interviewed at home and then invited to a baseline clinical examination between 1987 and 1989. They attended 3 additional clinical examinations at every 3 yearly and a recent 5th examination completed in 2013 for which data are not included here. Participants were interviewed by phone annually. Details of the ARIC Study design, protocol sampling procedures, and selection and exclusion criteria were published previously [21]. The study was approved by each study site's institutional review board. All participants provided written informed consent.

For the purpose of this analysis, we included all ARIC participants with good quality and complete ECG data at visits 1 to 4 as well as outcome events after visit 4. We excluded the following participants: 47 with reported race other than African-American or white, 136 with

poor quality ECG, 3775 with missing ECG in any of the ARIC first 4 visits including those who died during this period, 429 with ECG diagnosis of bundle branch block, external pacemaker or Wolff–Parkinson–White pattern, and 201 with missing one or more of baseline CVD risk factors. We also excluded 1706 participants with history of CVD at baseline which was defined as the presence of ECG evidence of MI, or a self-reported history of physician-diagnosed MI, coronary artery bypass surgery, coronary angioplasty, heart failure, or stroke. After all exclusions ($n = 6294$), a total of 9498 remained and were included in the analysis.

Identical electrocardiographs (MAC PC, Marquette Electronics Inc., Milwaukee, WI) were used at all clinic sites, and resting, 10 seconds standard simultaneous 12-lead ECGs were recorded in all participants using strictly standardized procedures. All ECGs were processed in a central ECG laboratory (initially at Dalhousie University, Halifax, Nova Scotia, Canada and later at the EPICARE Center, Wake Forest School of Medicine, Winston-Salem, NC, USA), where all ECGs were visually inspected for technical errors and inadequate quality.

Spatial QRS/T angle was computed from the mean QRS and T vectors of the quasi-orthogonal XYZ leads, and in turn computed using the Kors transform [11]. Frontal QRS/T angle was defined as the absolute value of the difference between the frontal plane QRS axis and T axis adjusted to the minimal angle using $(360^\circ - \text{angle})$ for angles $>180^\circ$ [12] (Supplementary Figures 1 and 2). Values greater than the sex-specific 95th percentile of the frontal and spatial QRS/T angles were considered abnormal while values between 95th and 75th were considered borderline. Left ventricular hypertrophy (LVH) was defined by Cornell voltage criteria (RaVL amplitude + SV3 amplitude) using the following sex-specific cut-off points: 2200 microvolt (μV) in women and 2800 μV in men.

Incident SMI was defined as ECG evidence of new MI without clinically documented MI (CMI) after the baseline until ARIC visit 4 (1996–1998). ECG evidence of MI was defined by new appearance of a major Q wave or a minor Q wave with major ischemic ST-T changes according to the Minnesota ECG Classification [10,21–23].

Frequency distributions of the variables used in analyses were first inspected to rule out anomalies and outliers. Descriptive statistics were used to determine mean values, standard deviations, and percentile distributions for continuous variables, and frequencies and percentages for categorical variables. Cox's proportional hazards analysis was used to examine the association between abnormal, borderline and normal (reference) frontal and spatial QRST angle (separately), with SMI and CMI (vs. no MI) occurring from visit 1 to visit 4. Models were adjusted as follows: Model-1 adjusted for demographics (age, sex and race), and Model-2 adjusted for demographics plus study center, body mass index, systolic blood pressure, smoking status, education, hypertension, diabetes mellitus, total ratio of total cholesterol/high-density lipoprotein cholesterol, use of cholesterol lowering medications, use of aspirin, family history of CHD, ECG-LVH by Cornell voltage, and serum creatinine at baseline. Given the known sex difference in QRS/T angle, subgroup analyses by sex using similar models were also conducted. All analyses were performed with SAS version 9.4 (SAS Institute Inc., Cary, NC).

Results

A total of 9498 participants (age 54.0 ± 5.7 years, 56.9% women, 20.3% African American) were included in the analysis. Table 1 shows the demographic, clinical, and ECG characteristics of the study participants stratified by spatial QRS/T angles categories at baseline. Compared to those with normal QRS/T angle, participants with wide QRS/T angle were more likely to be older, to be African-American, and to have diabetes and hypertension. They also were more likely to have lower education level, and higher values for body mass index and systolic blood pressure. Table 1 also shows that the participants with wide QRS/T angle had higher rates of incident MI (SMI and CMI).

During a median of 8.9 years of follow-up from baseline through the 4th ARIC visit, 317 participants developed SMI while 386 developed CMI. Using a sex-specific cut point at 95th percentile for abnormal QRS/T angle and at 75th percentile cutpoint for borderline angle at baseline (Table 2), participants with wide spatial QRS/T angle as well as frontal plane QRS/T angle had significantly higher rates of incident SMI than participants with normal QRS/T angles (8.5 vs. 3.2, and 9.4 vs. 3.3 per 1000 person-years, respectively). In multivariable adjusted models, wide spatial and frontal QRS/T angles exceeding 95th percentiles were both associated with an over 2-fold increased risk of incident SMI, and near 2-fold increase risk for all incident all MIs (including both of SMI and CMI) (Table 3 and Fig. 1).

In addition to the new Q wave development, about 18.3% of the participants in the SMI group had other major ECG abnormality during follow up (28 new complete bundle branch blocks, 28 ECG-LVH by Cornell voltage criteria, and 6 new atrial fibrillations). Among those, 29% were found on the ECG after SMI event, and 52% were shown on the same ECG with Q wave SMI event.

In multivariable adjusted models, common ECG markers of ventricular depolarization or repolarization were not associated with increased risk of SMI in our study. The association of SMI with prolonged QRS duration and prolonged QT interval had hazard ratios of 1.20 (95% confidence Intervals 0.79–1.83) and 1.35 (0.89–2.03), respectively.

Discussion

The purpose of this investigation was to examine and compare the frontal plane and spatial QRS/T angle as predictors of incident SMI. The key finding from our study is that both wide spatial and frontal QRS/T angles are equally predictive of incident SMI.

The QRS/T angle is a sensitive and reproducible marker of abnormal ventricular depolarization and repolarization which is able to detect depolarization and repolarization abnormalities before other common markers are apparent as QRS duration or QT interval [13–16]. Abnormal ventricular repolarization is an established mechanism for arrhythmogenesis. Wide QRS/T angles might be associated with myocardial structure and functional change. Over the years, a wide spatial QRS/T angle has been repeatedly shown to be associated with increased risk of all-cause mortality, cardiac mortality, and sudden

cardiac death in several cohort studies [11–20]. To our knowledge this is the first report documenting that spatial QRS/T angles also is associated with risk of incident SMI.

Despite the interest in spatial QRS/T angle and the repeated evidence of its usefulness as a predictor of CVD outcomes, its wide application has been challenged by being not readily available as an output from ECG machine currently in use, and being not familiar to most physicians. Conversely, the frontal QRS/T angle could be readily available from routine 12-lead ECGs, and simpler to calculate. Similar to spatial QRS/T angle, frontal QRS/T angle also reflects both ventricular depolarization and repolarization. A decade ago, we first reported that the frontal plane QRS/T angle was a 'suitable clinical substitute' for spatial QRS/T angle with respect to CHD risk prediction in the ARIC study [12]. Since then, the frontal QRS/T angle has been intensively studied in various populations, as a predictor of different outcomes including heart failure, atrial fibrillation, sudden cardiac death, and all-cause mortality [16–18,24–26]. Our study adds SMI to this list of outcomes, for the first time.

Our results should be read in the context of certain limitations. The population studied was restricted to those with white and African-American ethnicity, which impact the generalizability if the results to other races/ethnicities. Also, analyses of subgroups by sex might have been limited by the small numbers in the subgroups, and hence the interaction results should read with caution. Finally and similar to other studies with similar design, residual confounding is always a possibility. SMI in the present study is MI with ECG evidence of a major Q wave or a minor Q wave with major ischemic ST-T changes, and CMI is clinically documented MI with or without Q wave. It is not possible to detect NQWMIs with the ECGs alone. Our study has several strengths. The ARIC study is a large, well-designed prospective cohort study with long term follow-up. The study included highly standardized ECG procedures and carefully documented events ascertained by an independent adjudication committee.

Conclusions

Wide spatial and frontal plane QRS/T angles are both significant predictors of incident SMI suggesting a potential use for these markers in identifying individuals at risk. Hence, the frontal plane QRS/T angle could be used as a practical substitute for the more complex spatial QRS/T angle for early identification of individuals at risk for silent MI.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Fig. 1.

Risk of incident myocardial infarction by QRS/T angle category and gender. CI = confidence interval. Cutpoints listed in Table 2: normal, QRS/T angle $\leq 75^{\text{th}}$ percentile; borderline, $>75^{\text{th}}$ percentile and $\leq 95^{\text{th}}$ percentile; wide, $>95^{\text{th}}$ percentile. Model adjusted for age, sex, race, study site, body mass index, education, smoking status, systolic blood pressure, blood pressure lowering medications, diabetes mellitus, ratio of total cholesterol to high density lipoprotein, use of cholesterol-lowering medications, use of aspirin, family history of coronary heart disease, ECG-LVH by Cornell voltage, and serum creatinine at baseline.

Table 1

Baseline (1987–1989) participants characteristic stratified by spatial QRS/T angle groups and outcomes during follow-up (1996–1998).

<i>N</i> = 9498	Mean ± SD or <i>n</i> (%),			<i>p</i> -Value ^c	<i>p</i> -Value ^d
	Normal QRS/T (<i>n</i> = 7167)	Borderline QRS/T (<i>n</i> = 1851)	Wide QRS/T (<i>n</i> = 480)		
Age (years)	54 ± 5.7	54 ± 5.6	55 ± 5.7	<.0001	<.0001
Women	4085 (57)	1038 (56)	277 (58)	.3789	.4835
African-American	1424 (19.9)	356 (19.2)	150 (31.3)	<.0001	<.0001
Education high school	3644 (50.8)	961 (51.9)	266 (55.4)	.0180	.2526
Current smoker	1407 (19.7)	490 (26.5)	117 (24.4)	<.0001	.9251
Body mass index (kg/m ²)	27 ± 4.9	27 ± 5.2	28 ± 5.8	<.0001	<.0001
Systolic blood pressure (mm Hg)	118 ± 16	120 ± 18	128 ± 23	<.0001	<.0001
Hypertension	1875 (26.3)	518 (28.1)	234 (49.0)	<.0001	<.0001
Antihypertensive medication	1607 (22.4)	408 (22.1)	176 (36.7)	<.0001	<.0001
Diabetes	528 (7.4)	160 (8.7)	73 (15.3)	<.0001	<.0001
Ratio of total/HDL cholesterol	4.5 ± 1.6	4.5 ± 1.9	4.7 ± 1.7	.0020	<.0001
Cholesterol lowering medication	168 (2.4)	43 (2.3)	10 (2.1)	.5370	.4862
Aspirin use	3218 (45.3)	886 (48.3)	222 (46.7)	.0100	.5270
Family history of coronary heart disease	2861 (39.9)	751 (40.6)	187 (39.0)	.4380	.2177
Serum creatinine (mg/dL)	1.1 ± 0.3	1.1 ± 0.2	1.1 ± 0.2	<.0001	.0002
Left ventricular hypertrophy ^a	90 (1.0)	9 (2.8)	7 (1.8)	<.0001	<.0001
QRS/T angle—spatial (°)	55 ± 18	94 ± 11	127 ± 15	<.0001	<.0001
QRS/T angle—frontal (°)	19 ± 16	29 ± 24	56 ± 42	<.0001	<.0001
Heart rate (beats/min)	65 ± 9.5	67 ± 10	68 ± 11	<.0001	.0046
QRS duration (ms)	90 ± 9.1	93 ± 9.6	94 ± 10	<.0001	.0026
QTrr interval (ms) ^b	414 ± 14	416 ± 15	418 ± 16	<.0001	.0137
<i>Outcomes</i>					
Silent myocardial infarction	198 (2.8)	85 (4.6)	34 (7.1)	<.0001	<.0001
Clinical myocardial infarction	274 (3.8)	73 (3.9)	39 (8.1)	<.0001	<.0001

^aLeft ventricular hypertrophy was defined by Cornell voltage criteria.

^bQTrr interval is the rate-adjusted QT interval as a linear function of the RR interval.

Value comparing three groups (Wide QRS/T angle for >95th percentile, Borderline QRS/T angle for >75th and 95th percentile, and Normal QRS/T angle for 75th percentile) with ternary level of QRS/T angle including 75th percentile using analysis of variance and Chi-squared for continuous and categorical variables, respectively.

Value comparing groups with wide QRS/T angle and borderline QRS/T angle using unpaired Student T test and chi-squared for continuous and categorical variables, respectively.

Table 2

The cutpoints for borderline and wide QRS/T angles by gender and race.

	<i>N</i>	<u>Spatial QRS/T angle</u>			<u>Frontal QRS/T^a angle</u>		
		Mean	BL ^b	Wide ^c	Mean	BL ^b	Wide ^c
All participants	9498	65.9	83	114	22.7	31	63
Men	4098	73.6	91	120	23.4	31	66
Women	5400	60.2	76	104	22.1	30	61

^aFrontal plane QRS/T angle calculated as the absolute value of (QRS axis – T axis). If absolute value >180°, then subtract it from 360°.

^bBL (borderline) is for QRS/T angles >75th percentile and 95th percentile.

^cWide for QRS/T angles >95th percentile.

Table 3

Hazard ratios with 95% confidence intervals for risk of incident myocardial infarction by QRS/T angle category.

	Events/1000 person years	Model-1 ^a , HR (95% CI)	p Value	Model-2 ^b , HR (95% CI)	p Value
<i>Risk of incident silent myocardial infarction (N = 9112)</i>					
Spatial QRS/T angle					
Normal	3.2	1 (ref.)		1 (ref.)	
Borderline	5.3	1.66 (1.29–2.15)	<.0001	1.51 (1.16–1.96)	.0023
Wide	8.5	2.59 (1.79–3.73)	<.0001	2.00 (1.35–2.96)	.0005
Frontal QRS/T angle					
Normal	3.3	1 (ref.)		1 (ref.)	
Borderline	4.7	1.31 (1.01–1.71)	.0437	1.23 (0.94–1.61)	.1372
Wide	9.4	2.72 (1.92–3.84)	<.0001	2.28 (1.59–3.27)	<.0001
<i>Risk of incident myocardial infarction (both of silent and clinical, N = 9498)</i>					
Spatial QRS/T angle					
Normal	7.6	1 (ref.)		1 (ref.)	
Borderline	9.8	1.30 (1.09–1.56)	.0039	1.17 (0.97–1.41)	.1018
Wide	18.2	2.39 (1.87–3.07)	<.0001	1.87 (1.43–2.44)	<.0001
Frontal QRS/T angle					
Normal	7.4	1 (ref.)		1 (ref.)	
Borderline	10.4	1.35 (1.13–1.60)	.0009	1.25 (1.05–1.49)	.0147
Wide	17.9	2.25 (1.75–2.89)	<.0001	1.86 (1.43–2.41)	<.0001

Cutpoints listed in Table 2: normal, QRS/T angle 75th percentile; borderline, >75th percentile and 95th percentile; wide, >95th percentile.

^aModel 1: adjusted for age, sex, and race.

^bModel 2: adjusted for the variables in Model-1 plus study site, body mass index, education, smoking status, systolic blood pressure, blood pressure-lowering medications, diabetes mellitus, ratio of total cholesterol to high-density lipoprotein, use of cholesterol-lowering medications, use aspirin, family history of coronary heart disease, ECG-LVH by Cornell voltage, and serum creatinine at baseline.