Original Article

Prediction of nonviable myocardium by ECG Q-Wave parameters: A 3.0 T cardiovascular magnetic resonance study

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ABSTRACT

Introduction: The presence of a Q-wave on a 12-lead electrocardiogram (ECG) has been considered a marker of a large myocardial infarction (MI). However, the correlation between the presence of Q-waves and nonviable myocardium is still controversial. The aims of this study were to 1) test QWA, a novel ECG approach, to predict transmural extent and scar volume using a 3.0 Tesla scanner, and 2) assess the accuracy of QWA and transmural extent.
Methods: Consecutive patients with a history of coronary artery disease who came for myocardial viability assessment by CMR were retrospectively enrolled. Q-wave measurements parameters including duration and maximal amplitude were performed from each surface lead. A 3.0 Tesla CMR was performed to assess LGE and viability.
Results: Total of 248 patients were enrolled in the study (with presence (n = 76) and absence of pathologic Q-wave (n = 172)). Overall prevalence of pathologic Q-waves was 27.2% (for LAD infarction patients), 20.0 % (for LCX infarction patients), and 16.8% (for RCA infarction patients). Q-wave area demonstrated high performance for predicting the presence of a nonviable segment in LAD territory (AUC 0.85, 0.77—0.92) and a lower, but still significant performance in LCX (0.63, 0.51—0.74) and RCA territory (0.66, 0.55—0.77). Q-wave area greater than 6 ms mV demonstrated high performance in predicting the presence of myocardium scar larger than 10% (AUC 0.82, 0.76—0.89).
Conclusion: Q-wave area, a novel Q-wave parameter, can predict non-viable myocardial territories and the presence of a significant myocardial scar extension.

1. Introduction

The presence of a Q-wave on a 12-lead electrocardiogram (ECG) has been considered a marker of a large myocardial infarction (MI).

Late gadolinium enhancement (LGE) cardiovascular magnetic resonance (CMR) has become increasingly recognized as the gold standard for assessment of myocardial viability. Despite abundant literature, the correlation between the presence of Q-waves and nonviable myocardium is still controversial. Some studies have shown that the total size of the MI, rather than the transmural

extent, is the primary determinant of the presence of a Q-wave [The pathologic basis of Q-wave].

In myocardial infarction, necrosis of sufficient myocardial tissue may lead to decreased R-wave amplitude or the presence of abnormal Q-waves. Abnormal Q-waves are considered markers of transmural myocardial infarction.[1] However, highly detailed correlative studies on ECG-pathology relationship have indicated that transmural infarcts may occur without Q-waves and that subendocardial infarcts may sometimes be associated with Q-waves.[2] This reduces the efficacy of Q-waves as a diagnostic tool for transmural infarction.

Myocardium viability assessment using CMR is based on LGE imaging, with an enhancement area representing gadolinium retention in fibrotic tissue or scar. It is used to assess myocardial viability and benefits from coronary revascularization. Kim et al demonstrated that the transmural extent of a myocardial scar detected by LGE imaging was shown to accurately predict a progressive decrease in functional recovery despite successful coronary revascularization.[3] LGE imaging is easy to perform and interpret. A 50% transmural cut-off point is sensitive in predicting segmental contractile recovery.[3] Q-wave area (QWA) is a novel ECG parameter which shows robust correlation with scar volume over time independent of infarct shrinkage. Nevertheless, itscorrelation with myocardial viability is unknown.

Most previous reports were completed using 1.5 Tesla CMR scanners, while a 3 Tesla scanner can provide a higher signal-to-noise ratio (SNR) than a 1.5 Tesla scanner and results in better image quality. The aims of this study were two-fold 1) testing QWA, a novel ECG approach, to predict transmural extent and scar volume using a 3.0 Tesla scanner, and 2) the accuracy of QWA and transmural extent.

2. Method

2.1. Patient population

Consecutive patients with a history of coronary artery disease who presented for myocardial viability assessment by CMR between October 2015 and November 2016 were retrospectively enrolled. Patients were excluded for recent (within 30 days) myocardial infarction, hypertrophic cardiomyopathy, myocarditis, and conduction abnormalities (bundle branch block and pre-excitation syndrome). The study protocol and ethical consideration were approved by the institutional review board on human research.

2.2. Electrocardiographic (EKG) assessment

The digital 12-lead EKG was acquired using a PageWriter Trim-III machine (Philips, Netherlands). The standard default settings were a speed of 25 mm/s with a voltage of 10 mm/mV[4] Q-wave measurements from multiple perspectives including duration and maximal amplitude were performed from each surface lead.

The established American College of Cardiology/European Society of Cardiology consensus criteria (Third Universal Definition of Myocardial Infarction in 2012)were used to define pathological Q-waves including: Q-waves in leads V2—V3 $\geq$ 20 ms; QS complex in leads V2 and V3; Q-wave $\geq$ 30 ms and $\geq$0.1 mV deep; QS complex in leads I, II, aVL, aVF or V4—V6 in any 2 leads of consecutive lead grouping (I, aVL, V6; V4—V6; II, III and aVF); or an R-wave $\geq$ 40 ms in V1—V2 and R/S $\geq$ 1 with a concordant positive T-wave.[5] The Q-wave parameters including Q-wave duration (ms), depth (mV) and QWA (ms.mV) were manually measured on an IntelliSpace ECG orTraceMasterVue ECG management system (Philips, Netherlands) utilizing the digital zoom-in feature to determine the precise

valueQWA was calculated by its triangle geometric shape. The Q-wave on 12-lead ECG was then colocalized with the vascular territory. Q-waves in V1—V4 were compatible with LAD territory. Q-wave in II, III or aVF were compatible with RCA territory and LCX territory accounts for Q-wave in I, aVL, V5 or V6. Summation of QWA and Q wave depth were analyzed in accordance with each vascular territory.

The ECG analysis were completed by two experienced cardiologists, unaware of the CMR data. In cases where there was conflicting data, a third physician who was blinded to CMR findings was brought in to adjudicate.

2.3. Cardiac magnetic resonance imaging (CMR) protocol

The CMR imaging was performed on a 3.0-T scanner (Skyra, Siemens, Erlangen, Germany). Breath-hold retrospectively ECG-gated cine steady state free precession (SSFP) images were acquired in the 2-chamber, 3-chamber, and 4-chamber views. A short axis stack covering the entire left ventricle (8-mm slices without gap) was used to calculate left ventricular (LV) ejection fraction. Sequence parameters were repetition time 2.5 ms, echo time 1.26 ms, flip angle 60°, field-of-view 320 mm, matrix 256 × 192, and temporal resolution 35—40 ms. For the scar image, standard delayed-enhancement imaging was performed in the same slice positions as the cine imaging using a segmented inversion-recovery gradient-echo sequence (6-mm slices with 4-mm gaps) approximately 10 min after administration of intravenous contrast (gadobenatedimeglumine, 0.1 mmol/kg).[6,7]

2.4. CMR analysis

The post-processing analyses were performed using Synapse PACS (Fujifilm medical system U.S.A., Inc., Massachusetts, USA). The LV chamber and function quantification were automatically measured by the software. The LV myocardial scar location and volume were visually estimated by grading the transmurality of the hyperenhancement area in each myocardial segment and summarizing the total scar volume.[8] In addition, the myocardial viability was defined further by the coronary territory.

2.5. AHA 17-segment model and their supplied coronary artery

The left ventricular (LV) myocardium was divided into 3 levels (base, mid, apex). The basal level was comprised of a basal anterior wall (segment 1, S1), basal anteroseptal wall (S2), basal inferoseptal wall (S3), basal inferior wall (S4), basal inferolateral/posterior wall (S5) and basal anterolateral/lateral wall (S6). The mid-level was comprised of the mid anterior wall (S7), mid anteroseptal wall (S8), mid inferoseptal wall (S9), mid inferior wall (S10), mid infero-lateral/posterior wall (S11) and mid anterolateral/lateral wall (S12). The apical level was comprised of the apical anterior wall (S13), apical septal wall (S14), apical inferior wall (S15), apical lateral wall (S16) and apical cap (S17).[9] The LV myocardium segments are supplied by different coronary arteries. The left anterior descending (LAD) artery supplies a total of 7 segments including S1, S2, S7, S8, S13, S14 and S17. The left circumflex (LCX) artery supplies a total of 5 segments including S5, S6, S11, S12 and S16. The right coronary artery (RCA) supplies a total of 5 segments including S3, S4, S9, S10 and S15.

2.6. Myocardial scar analysis

The sum of the hyperenhancement area was determined by software-assisted visual analysis[1,8] and expressed as a percentage of the total scar volume over the entire LV myocardium volume. The

Table 1
Baseline characteristics.

	Overall (N = 248)	No Q-wave (N = 172)	Q-wave (N = 76)	P-Values
Sex: male, n (%)[a]	142 (57.3%)	81 (47.1%)	61 (80.3%)	<0.001
Age year, mean (SD)[b]	66.0 (11.4)	66.5 (11.1)	65.0 (11.9)	0.336
DM, n (%)[a]	105 (42.3%)	70 (40.7%)	35 (46.1%)	0.517
HT, n (%)[a]	156 (62.9%)	104 (60.5%)	52 (68.4%)	0.292
DLP, n (%)[a]	124 (50.0%)	77 (44.8%)	47 (61.8%)	0.019
PCI, n (%)[a]	45 (18.1%)	22 (12.8%)	23 (30.3%)	0.002
CABG, n (%)[a]	10 (4.0%)	5 (2.9%)	5 (6.6%)	0.315
Diagnosis[a]				
SCAD, n (%)	233 (94.0%)	170 (98.8%)	63 (82.9%)	<0.001
UA, n (%)	1 (0.4%)	0 (0%)	1 (1.3%)	
NSTEMI, n (%)	5 (2.0%)	2 (1.2%)	3 (3.9%)	
STEMI, n (%)	9 (3.6%)	0 (0%)	9 (11.8%)	
MI duration, month (median)[b]	8.5	9.5	8.0	0.070
CMR parameter				
Nonviable segment percent of each territory, mean (SD)[b]				
LAD	11.4 (27.2)	2.16 (11.7)	32.3 (38.5)	<0.001
RCA	5.32 (16.8)	1.98 (9.59)	12.9 (25.2)	<0.001
LCX	6.29 (20.0)	2.56 (12.3)	14.7 (29.5)	<0.001
Overall	8.11 (15.7)	2.22 (8.15)	21.4 (20.0)	<0.001
Scar volume %, mean (SD)[b]	8.01 (12.8)	2.79 (7.07)	19.8 (14.9)	<0.001
LVEF, mean (SD)[b]	51.3 (17.6)	56.2 (15.8)	40.1 (16.6)	<0.001

CABG, coronary artery bypass graft; DLP, dyslipidemia; DM, diabetes mellitus; HT, hypertension; LAD, left anterior descending artery; LCX, left circumflex artery LVEF, left ventricular ejection fraction; NSTEMI, non-ST segment elevation myocardial infarction; PCI, percutaneous coronary intervention; RCA, right coronary artery; SCAD, stable coronary artery disease; STEMI, ST segment elevation myocardial infarction; UA, unstable angina.

[a] chi square test.
[b] t-test.

myocardial scar was visually classified further by its transmural extent (percent of LV wall thickness) in each myocardial segment using a 5-level scoring system: 0 = no scar; 1 = scar extended 1–25% transmurality; 2 = scar extended 26–50% transmurality; 3 = scar extended 51–75% transmurality; and 4 = scar extended >75% transmurality. Nonviable segments were defined as a segment with a transmural extent of scar >50% of the wall thickness.[1,3]

2.7. Non-viable myocardium defined by coronary territory

The number of non-viable segments determined the viability of myocardium supplied by the coronary artery. When the number of non-viable segments was greater than 50% of the total segments supplied by the coronary artery, we defined that territory as non-viable.

2.8. Statistical analysis

Quantitative data were expressed as mean ± SD and median (25th to 75th percentile) for continuous variables and as frequencies with percentages for categorical variables. Unpaired Student's t test and a Chi-square test were used to compare differences between groups of continuous and categorical variables. Receiver-operating characteristic (ROC) curves were constructed to assess association of QWA and transmural extent. In addition, ROC curves were used to evaluate the diagnostic performance of QWA. Area under the curve (AUC) comparisons were performed based on Z-transformation following established methods.[9] Two-sided p<0.05 was considered indicative of statistical significance. Calculations were performed using R software (R Foundation for Statistical Computing, Vienna, Austria).

3. Result

A total of 286 subjects were enrolled. After the exclusion of 38 patients who did not meet inclusion criteria based on comorbidities or poor-qualityimaging,a total of 248 patients were enrolled in the study. Table 1 presents baseline characteristics of the study population stratified based on presence (n = 76) and absence of pathologic Q-wave (n = 172). With respect to infarct distribution, pathologic Q-waves were more common with every infarction territory. Overall prevalence of pathologic Q-waves was 27.2% (for LAD infarction patients), 20.0 (for LCX infarction patients), and 16.8% (for RCA infarction patients).

3.1. Q-wave area and viable myocardium of respective territory

Different vascular territories provide different c-statistics for QWA in predicting non-viable myocardium in that respective territory. Q-wave area demonstrated high performance for predicting the presence of a nonviable segment in LAD territory (AUC 0.85, 0.77–0.92, Fig. 1a) and a lower, but still significant performance in LCX (0.63, 0.51–0.74) and RCA territory (0.66, 0.55–0.77). Selected thresholds for QWA provided a high specificity (95% in LAD, 95% in LCX, and 92% in RCA) (Table 2). Different territories are supplied with different myocardial area, making their unique cut off number of the threshold to determine respective nonviable vascular territories.

3.2. Q-wave area and scar volume

Q-wave area greater than 6 ms mV demonstrated high performance in predicting the presence of myocardium scar larger than 10% (AUC 0.82, 0.76–0.89). The selected threshold provided 73% sensitivity and 87% specificity yielding positive likelihood ratio of 5.78 and negative likelihood ratio of 0.31 (Fig. 1b).

4. Discussion

Our study demonstrated the relationship between QWA and the presence of non-viable myocardium in the respective territories. This study provides one of the first analyses using a 3 Tesla CMR and a simplified quantification method for QWA compared to the previously more complicated method for QWA quantification.[10–13] Our enrolled subjects had stable infarct size due to timing of CMR

(a)

Q wave area vs presence of nonviable segment in LAD territory

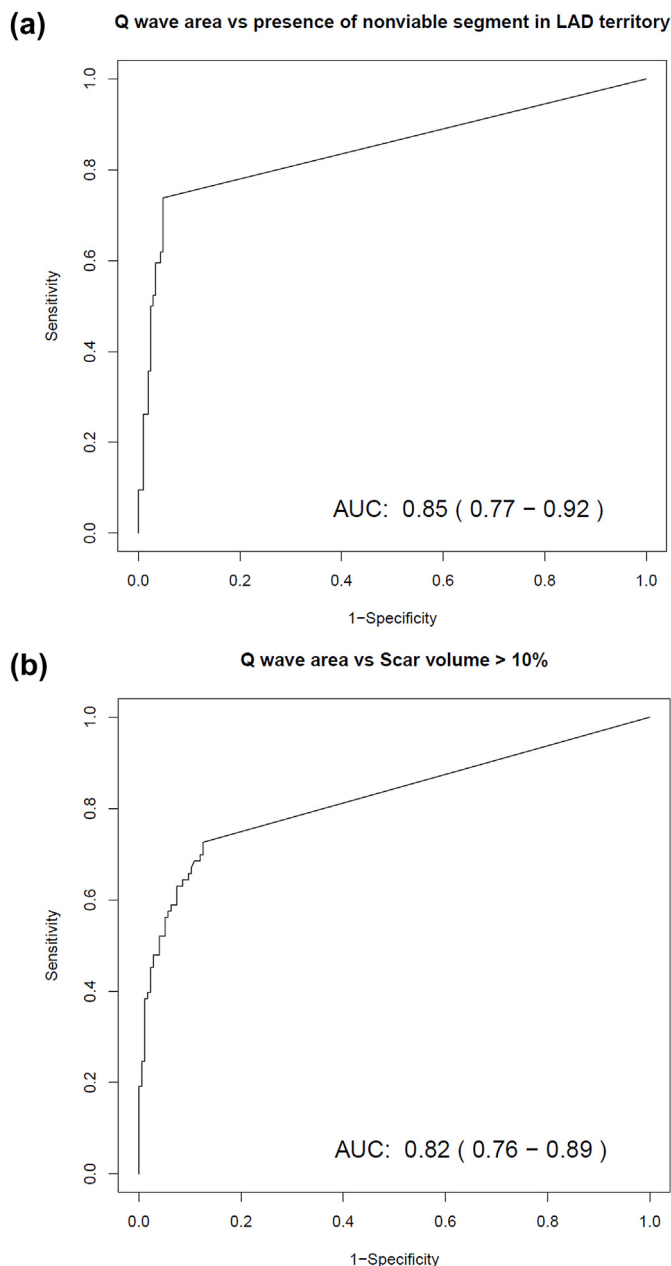**(b)**

Q wave area vs Scar volume > 10%

Fig. 1. 1a Diagnostic performance of QWA for LAD territory (AUC, Area Under the Curve). 1b Diagnostic performance of QWA on prediction of significant LV scar volume (AUC, Area Under the Curve).

assessment in contrast to previous studies on acute myocardial infarctions.[14] Our results support the same direction found in prior studies revealing the correlation between global QWA and total scar volume.

73% of enrolled patients have non-Q wave MI which is consistent with the significant findings from prior studies demonstrating a higher prevalence of non-Q wave MI in stable coronary artery disease patients due to unrecognized myocardial infarction.[15,16]

4.1. Q-wave area and viable myocardium of respective territory

Unique findings from our study also include using QWA to assess viability in respective territories and QWA on LAD has the highest performance on predicting non-viable. Furthermore, all territories showed a very high specificity (92—95%) for non-viable diagnosis from QWA. Possible explanations for a very high predictive value in LAD territory include proximity of anterior chest wall electrode placement to the anterior wall myocardium, greater number of leads on the anterior wall, and less intervening structure between the lead position and myocardial wall compared to areas along the LCX and RCA territories. Also, seven segments in LAD territory are likely exclusively supplied by LAD. Since some segments in LCX and RCA territories may be supplied by other coronary arteries, this may have caused the lower accuracy of QWA in LCX and RCA territories than QWA in LAD territory.

4.2. Q-wave area and scar volume

Infarct size expansion was accompanied by an increment of QWA. QWA at a threshold of 6 ms. mVis an indicator to determine the significant scar volume (10%). Consistent with our findings, prior data have also demonstrated that larger QWA size is correlated with larger infarct size. A threshold of 10% was selected based on the study that the given value is related to an increase in mortality in patients with MI.[14]

Compared to the 1.5 tesla scanner used in prior studies, our study demonstrated similar performance in identifying total scar volume >10% in LV with the 3.0 tesla scanner.[14] We must also consider that the temporal relationship between performing LGE imaging and timing of MI impacted our results. Kochav et al Conducted a study with patients enrolled in two separate periods, early (within 3—5 days) and late (2—6 weeks).[14] This timing selection is different from our study which excluded persons with a recent MI (within 4 weeks after onset of MI). Given the wider range of post—MI timing in Kochav et al, LGE might not truly reflect a stable infarct size for some of the early enrollees in their study. In our study, examining scars 8.5 months after MI can preclude any confounding from an evolving scar due to a recent MI. Also, the extent

Table 2
Diagnostic performance of QWA for the nonviable vascular territories.

	LAD	LCX	RCA
Threshold for QWA (ms.mV)	22.5	5.1	15.4
AUC (95%CI)	0.85 (0.77—0.92)	0.63 (0.51—0.74)	0.66 (0.55—0.77)
Sensitivity (%)	74	30	42
Specificity (%)	95	95	92
Positive likelihood ratio	15.2	5.95	5.03
Negative likelihood ratio	0.28	0.74	0.64

AUC, Area Under the Curve; LAD, left anterior descending artery; LCX, left circumflex artery; QWA, Q wave area; ms.mV, millisecond-millivolt; RCA, right coronary artery.

of the scar can determine the patients with the propensity to myocardial function recovery following revascularization.[17,18]

Concerning ECG manifestation of scar size, our findings emphasize a regional difference in diagnostic accuracy. Although the exact mechanism to explain the discrepancy across territories remains unidentified, many possible factors can be considered including left ventricular function, amount of surrounding healthy tissues, body contour, and other concomitant subclinical myocardial disease.

In terms of clinical application, the use of QWA can be applied when screening patients at high risk of significant infarction and triaging investigation in resource-limited settings. This can help achieve the most efficient use of expensive and limited available equipment such as the high tesla cardiac MRI. Another important outcome of our study is that we validated the method of measurement of QWA using simple techniques that have been validated with other software based QWA measurement.[19]

Our study limitations are noteworthy. Firstly, the relationship between Q-wave and MI reported in this study cannot be generalized to patients with conditions that can mask the Q-wave such as left bundle branch block and fascicular block or mask a condition with Q-wave other than myocardial infarction such as hypertrophic cardiomyopathy or Wolff-Parkinson-White syndrome. In addition, the culprit infarct-related artery was not identified and compared with Q-wave parameters or scar location in this study. However, it is widely known that there is extreme variability in the coronary blood supply to a specific myocardial territory especially at the lateral wall area. As such, we colocalized the Q-wave on the surface ECG with the scar location on the myocardium instead of at the designated vascular supply.

This study did not include a coronary angiogram in the analysis due to its imperfect ability to determine the presence of myocardial ischemia. A coronary angiogram without a functional test might not provide an actual ischemic state of supplied myocardium despite the fact that an interventional cardiologist ascertained visualized obstruction. Furthermore, the increasing role of ischemia assessment tools has shifted toward CMR before the final decision on revascularization on that respective coronary artery lesions. Hence, we did not include a coronary angiogram into our part of the study as it does not provide superior detail of scar over CMR.[20]

In conclusion, Q-wave area, a novel Q-wave parameter, can predict non-viable myocardial territories and the presence of a significant myocardial scar extension (more than 10% scar volume).

Authors' contributions

YV, MT, and PC carried out image acquisition. PK, AP, and NT carried out data collection and analysis. PC, PM, RC, and SB conceived the study. PC is essentially an intellectual contributor. All authors have contributed to the final manuscript and reviewed it.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ihj.2022.02.001.

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