Both baseline Selvester QRS score and change in QRS score predict prognosis in patients with acute ST-segment elevation myocardial infarction after percutaneous coronary intervention

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Background: We aimed to demonstrate the prognostic value of Selvester QRS scores in patients with acute ST-segment elevation myocardial infarction (STEMI).

Methods: In this prospective, observational study, we screened 289 patients with acute STEMI who underwent percutaneous coronary intervention (PCI) from 1 January 2014 to 1 June 2015 at the Second Hospital of Dalian Medical University. Selvester QRS scores were calculated at the time of hospital admission and within 24 h after treatment for PCI. The primary endpoint was the 2-year mortality rate, and the secondary endpoint was any nonfatal major adverse cardiovascular event (MACE).

Results: Of the 289 patients, the QRS score increased in 115 (39.8%), and the 2-year mortality and MACE rates were significantly higher in these patients than in those in whom the QRS score decreased or remained unchanged after the treatment of PCI. Multivariable Cox regression analysis revealed that both baseline QRS scores and changes in QRS scores were independently associated with the 2-year mortality rate [hazard ratio (HR) 1.462, 95% confidence interval (95% CI) 1.279–1.671 and HR

5.122, 95% CI 2.128–12.328, respectively), MACE rate (HR 1.119, 95% CI 1.019–1.229 and HR 2.585, 95% CI 1.260–5.303, respectively) and composite endpoint (HR 1.137, 95% CI 1.047–1.236 and HR 3.152, 95% CI 1.704–5.829, respectively) after adjusting for other risk factors.

Conclusion: In conclusion, both baseline Selvester QRS scores and changes in QRS scores independently predicted poor outcomes in patients with acute STEMI who underwent PCI. *Coron Artery Dis* 31: 403–410 Copyright © 2020 The Author(s). Published by Wolters Kluwer Health, Inc.

Introduction

Although the treatments for primary percutaneous coronary intervention (PCI) and other reperfusion therapies are widely performed, acute ST-segment elevation myocardial infarction (STEMI) remains a devastating disease that is associated with significantly increased morbidity and mortality [1–3]. Clinical trials have identified multiple prognostic predictors in patients with acute STEMI who have undergone PCI; these include cardiac troponin T, hemoglobin A1C, lipoprotein-associated phospholipase A2, and so on [4–8]. The Selvester QRS score contains 31 total possible points, with each point reflecting myocardial infarction involving 3% of the left ventricle; multiple versions of this test have been developed for widespread use in past decades [9,10].

Recently, the Selvester QRS score was demonstrated to be a strong predictor of infarct size and poor outcomes in patients with STEMI [11–15]. Previous studies of the relationship between Selvester QRS scores and clinical outcomes in STEMI patients have usually used a single measurement of the QRS score. However, the Selvester QRS score may be a dynamic and variable, exhibiting rapid changes in patients receiving PCI. In the present study, we hypothesized that exploring the changes that occur in the Selvester QRS score in patients with acute STEMI after PCI may provide more prognostic information beyond a single measurement.

Methods
Study design
We performed a prospective observational study involving 426 consecutive adult patients (27–90 years old) who were admitted to the Second Hospital of Dalian Medical University from 1 January 2014 to 1 June 2015. The following participants were eligible for the study: (1)

patients with acute STEMI according to the 2013 ACCF/AHA Guidelines for the management of STEMI [16] and (2) patients with successful primary PCI (stable thrombolysis in myocardial infarction III blood flow and <30% residual stenosis in the target vessel). The exclusion criteria included a history of myocardial infarction (n = 85), non-ST segment elevation acute coronary syndrome (n = 42) and refusal to participate in this study (n = 10). A total of 289 adult patients, including 220 (76.1%) male and 69 (23.9%) female patients, were ultimately enrolled in our study. The protocol met the Strengthening the Reporting of Observational Studies in Epidemiology [17] and Standards for Reporting Diagnostic Accuracy criteria [18]. This study was performed in accordance with approved guidelines, protocols, and regulations and approved by the Ethics Committee of the Second Hospital of Dalian Medical University. Written informed consent was obtained from each patient.

Demographic, clinical and biochemical data were collected for all the patients enrolled in the study immediately after they were admitted to the hospital and before any in-hospital treatments. All of the patients were imaged during their hospital stay in the supine position using an ultrasound system (iE33 xMATRIX Echocardiography System; Philips Healthcare, Best, The Netherlands) to evaluate cardiac structure and function.

The primary endpoint was all-cause mortality within 2 years after their hospital discharge (each patient's death status and date of death were determined through reviews of hospital records and telephone calls to their home), and the secondary endpoint was any nonfatal major adverse cardiovascular event (MACE), which was defined as a composite of nonfatal MI [international classification of diseases, 10th revision (ICD-10) codes I21, I22], nonfatal stroke (ICD-10 codes I60–69) and readmission for unstable angina (ICD-10 code I20) or heart failure (ICD-10 code I50), during the 2-year follow-up period. Moreover, the composite endpoint consisted of the 2-year mortality and nonfatal MACE rates. We considered an event nonfatal only if the patient survived to the end of the scheduled follow-up period.

Selvester QRS score
A 12-lead electrocardiogram (ECG) was recorded at the time of hospital admission before any in-hospital treatment and within 24 hours after treatment for PCI by electrocardiograph (FCP-7541; Fukuda Denshi Co. Ltd, Tokyo, Japan). The QRS score was calculated according to a 50-criteria 31-point Selvester QRS scoring system [19] first at the time of hospital admission (baseline Selvester QRS score) and then within 24 hours after treatment for PCI. The QRS score was manually calculated by two expert cardiologists according to an algorithm, as previously reported. The cardiologists were blinded to the patient outcomes.

Treatments
All STEMI patients were treated according to standard clinical practice. The artery puncture site was left to the discretion of the operators, although the radial approach was strongly recommended to avoid bleeding complications at the puncture site. In accordance with revascularization guidelines, dual anti-platelet therapy consisting of aspirin (300 mg per os followed by 100 mg daily) and clopidogrel (300 mg loading dose followed by 75 mg daily) was initiated and continued for at least 1 year. Enoxaparin, 6000 iu subcutaneously twice a day, was prescribed after PCI and lasted at least 7 days. Moreover, statins, angiotensin-converting enzyme inhibitors, β-blockers and nitrates were used if there were no contraindications.

Echocardiography measurements
All of the echocardiography measurements were performed within 7 days after PCI. Two experienced operators in our hospital performed and analyzed the echocardiographic data according to the guidelines of the American Society of Echocardiography (ASE). Measurements were acquired from standard parasternal and apical views using a Sequoia C512 Ultrasound Unit (Acuson, Thousand Oaks, California, USA) with a linear probe (model 3V2c; 2–3 MHz). The operators remained blinded to patient outcomes. The left ventricular mass index (LVMI) and left atrial volume index (LAVI) were determined using the formula suggested by the ASE guidelines [20].

Statistical analyses
SPSS 22.0 software was used for all analyses. Continuous variables are reported as the mean ± SD or as medians [interquartile ranges (IQRs)], and categorical variables are described as proportions. We divided the included patients into two groups according to the changes in Selvester QRS scores. We used two-sample t-tests or the Mann–Whitney U test to compare continuous variables between groups and Pearson's chi-squared test (χ^2) to compare categorical variables between groups. Spearman correlation coefficients were calculated between QRS scores and echocardiographic parameters. Prognostic indicators of mortality and nonfatal MACE were determined by a univariable Cox proportional hazards model, and variables with P values <0.1 were included in the multivariable Cox proportional hazards model. Cumulative survival curves were generated as a function of time by Kaplan–Meier analysis and compared by log-rank tests. All statistical tests were two-tailed, and $P < 0.05$ was considered statistically significant.

Results
Subject characteristics
All patients in this study were divided into two groups according to the observed changes in their QRS score (ΔQRS score) between before and after treatment with

PCI (Table 1); these changes ranged from –7 to +10 (0.2 ± 1.3). The ΔQRS score >0 group included patients who showed any increase in Selvester QRS score at 24 hours post-PCI, while the ΔQRS score ≤0 group included those patients with a negative or no changes in QRS score after PCI. We compared patients with and without an elevated QRS score. Patients with a positive change in QRS score had lower DBP levels, higher Killip class and higher QRS score after PCI than were found in patients with a negative or no change in QRS score. However, there was no difference in baseline QRS score, comorbidities, treatments or echocardiographic parameters between the two groups.

The mean QRS score measured on admission was 5.2 ± 1.0, while the mean QRS score measured after treatment with PCI was 5.3 ± 1.4. The mean ΔQRS score was 0.2 ± 1.3. A total of 115 (39.8%) patients showed a positive change in the QRS score, while 174 (60.2%) patients showed a decrease in the QRS score after PCI (Table 2).

Correlations between QRS scores and echocardiographic parameters and cardiac enzyme levels

Both the baseline QRS score and ΔQRS score were positively correlated with LVMI (r = 0.148 and 0.158, respectively), LAVI (r = 0.218 and 0.152, respectively) and left ventricular ejection fraction (LVEF) (r = 0.225 and 0.275, respectively). Moreover, there was also a significant negative correlation between both the baseline QRS score and the ΔQRS score and LVEF (r = –0.263 and –0.236, respectively). Nevertheless, the baseline QRS score but not the ΔQRS score was positively correlated with troponin I (TnI) levels (r = 0.206), peak creatine kinase-MB (CKMB) levels (r = 0.176) and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels (r = 0.212) (Table 3).

Associations between QRS scores and clinical outcomes

After hospital discharge, the survivors were followed up for a median of 19.8 months (IQR 17.0–22.7 months). Both the incidence of any MACE (44.3% versus 8.6%, *P* < 0.001) and the 2-year mortality rate (15.7% versus 4.0%, *P* = 0.001) were significantly higher in the ΔQRS score >0 group than in the ΔQRS score ≤0 group (Table 1).

As shown in Table 4, the univariable Cox regression analysis revealed that the risk of 2-year mortality was related to a ΔQRS score >0, the baseline QRS score, leucocyte levels, Killip class, increased TnI levels, increased CKMB levels, increased NT-proBNP levels, serum creatinine levels and low-density lipoprotein (LDL) levels, while any MACE was related to a ΔQRS score >0, the baseline QRS score, increased TnI levels, increased CKMB levels, increased NT-proBNP levels, serum creatinine levels and Killip class. Based on multivariable Cox regression analysis, both the baseline QRS score [hazard ratio (HR) 1.279, 95% CI 1.279–1.671, *P* < 0.001) and a ΔQRS score >0 (HR 5.122, 95% CI 2.218–13.328, *P* = 0.02) were significant predictors of mortality after adjustment for age, sex, BMI, leucocyte counts, Killip class, DBP, and TnI, CKMB, NT-proBNP, serum creatinine and LDL levels. There were 66 (28.9%) patients who had a MACE. The baseline QRS score (HR 1.119, 95% CI 1.019–1.229, *P* = 0.019) and ΔQRS score >0 (HR 2.585, 95% CI 1.260–5.303, *P* = 0.010) remained crucial predictors of a MACE even after adjustment for age, sex, BMI, Killip class, and TnI, CKMB, NT-proBNP and serum creatinine levels (Table 4). Moreover, both the baseline QRS score (HR 1.1137, 95% CI 1.047–1.236, *P* = 0.002) and a ΔQRS score >0 (HR 3.152, 95% CI 1.704–5.829, *P* < 0.001) exerted additive effects on the risk of the composite endpoint after adjusting for other risk factors (Table 5).

Table 1 Characteristics of all patients on admission and outcome

Characteristic	ΔQRS > 0 (n = 115)	ΔQRS ≤ 0 (n = 174)	*P* value
Age (year)	63.6 ± 11.6	61.6 ± 11.4	0.320
Sex, male, n (%)	84 (73.0)	136 (78.2)	0.155
BMI (kg/m²)	24.8 ± 3.4	25.2 ± 2.9	0.318
Preexisting clinical conditions			
CKD, n (%)	13 (11.3)	12 (6.9)	0.214
Hypertension, n (%)	64 (55.7)	88 (50.6)	0.399
Diabetes, n (%)	40 (34.8)	50 (28.7)	0.284
Dyslipidemia, n (%)	13 (11.3)	24 (13.8)	0.537
Medication after discharge			
Statins, n (%)	78 (67.8)	104 (59.8)	0.111
β-blockers, n (%)	71 (61.7)	102 (58.6)	0.598
ACEI/ARB, n (%)	68 (59.1)	93 (53.1)	0.343
Characteristics on admission			
DBP (mmHg)	78.1 ± 13.9	67.3 ± 11.0	0.008
Leucocyte (×10⁹/L)	9.3 ± 1.9	8.5 ± 1.3	0.154
Hemoglobin (g/L)	139.9 ± 17.3	140.0 ± 17.4	0.957
TnI (μg/L)	33.7 ± 7.5	21.9 ± 7.0	0.194
Baseline QRS scores, points	4.7 ± 1.0	5.6 ± 1.3	0.062
QRS scores after PCI, points	6.4 ± 1.3	4.6 ± 1.1	<0.001
Peak CKMB (U/L)	69.7 ± 15.7	61.1 ± 17.7	0.542
NT-proBNP (pg/ml)	532.8 ± 34.4	322.0 ± 51.0	0.141
Serum creatinine (μmol/L)	77.1 ± 18.0	72.9 ± 22.5	0.154
Total cholesterol (mmol/L)	4.9 ± 1.2	5.0 ± 1.4	0.859
Serum triglyceride (mmol/L)	1.6 ± 0.8	1.7 ± 0.7	0.437
High-density lipoprotein (mmol/L)	1.1 ± 0.2	2.2 ± 0.6	0.448
LVMI (g/m²)	103.2 ± 28.5	100.7 ± 26.9	0.472
LAVI (ml/m²)	27.8 ± 2.9	27.9 ± 2.8	0.770
E/e′	1.0 ± 0.4	1.0 ± 0.3	0.477
Killip			0.018
Class I	77 (67.0)	129 (74.1)	
Class II	22 (19.1)	36 (20.7)	
Class III	3 (2.6)	5 (2.9)	
Class IV	13 (11.3)	4 (2.3)	
Outcomes			
2-year mortality	18 (15.7)	7 (4.0)	0.001
MACE	51 (44.3)	15 (8.6)	<0.001
Readmission[a]	29 (25.2)	9 (5.2)	<0.001
Non-fatal MI	20 (17.4)	5 (2.9)	<0.001
Non-fatal stroke	2 (1.7)	1 (0.6)	0.341

ACEI/ARB, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers; CKD, chronic kidney disease; CKMB, creatine kinase-MB; LAVI, left atrial volume index; LVMI, left ventricular mass index; MACE, major adverse cardiovascular event; NT-proBNP, N-terminal pro-brain natriuretic peptide; PCI, percutaneous coronary intervention; TnI, troponin I.

[a]Readmission means readmission for unstable angina or heart failure.

Table 2   Change in QRS score during the treatment of percutaneous coronary intervention

Variables	Points
QRS score at admission, mean ± SD	5.2 ± 1.0
QRS score at admission, median (IQR)	5.0 (3.0–7.0)
QRS score after PCI, mean ± SD	5.3 ± 1.4
QRS score after PCI, median (IQR)	5.0 (3.0–8.0)
ΔQRS score, mean ± SD	0.2 ± 1.3
Positive change of QRS score, n (%)	115 (39.8)
ΔQRS score, mean ± SD	2.5 ± 1.9
Negative or static change of QRS score, n (%)	174 (60.2)
ΔQRS score, mean ± SD	−1.2 ± 1.3

IQR, interquartile range; PCI, percutaneous coronary intervention.

Table 3   Correlation between baseline Selvester QRS scores and ΔQRS score and echocardiographic parameters and cardiac enzyme levels

Variables	Baseline QRS score		ΔQRS score	
	r	P value	r	P value
LVMI (g/m^2)	0.148	0.034	0.158	0.010
LAVI (ml/m^2)	0.218	0.002	0.152	0.013
E/e′	0.225	<0.001	0.275	<0.001
LVEF	−0.263	<0.001	−0.236	<0.001
TnI	0.206	0.002	0.065	0.270
Peak CKMB	0.176	0.009	0.074	0.210
NT-proBNP	0.212	0.001	0.099	0.091

CKMB, creatine kinase-MB; E/e′, diastolic left ventricular function; LAVI, left atrial volume index; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; NT-proBNP, N-terminal pro-brain natriuretic peptide.

Figure 1 shows that patients who showed an increase in the ΔQRS score and had a baseline QRS score > the median had the highest overall mortality rate, while patients with a ΔQRS score >0 alone or a baseline QRS score > the median value had a better cumulative survival rate.

Discussion

This study demonstrates that both a high baseline QRS score at the time of hospitalization and a high ΔQRS score after PCI were associated with higher 2-year mortality and a greater risk of a MACE in acute STEMI patients who underwent PCI. Moreover, when the patients were divided into two groups based on changes in the QRS score between before and after PCI, the risk of a MACE and the 2-year mortality rate increased by 1.6-fold and 4.1-fold, respectively, in the group with positive changes in the QRS score after adjustment for a variety of other clinical and laboratory variables.

The association between the QRS score and cardiac function has been demonstrated in previous studies. Palmeri et al. [21] found that there was a good correlation between QRS scores and LVEF in a study of 55 patients who did not have left ventricular hypertrophy or conduction abnormalities. Similarly, in the present study, based on Spearman's correlation coefficient, there was a negative correlation between the baseline QRS score and LVEF. In addition to its link to cardiac function, baseline QRS

scores have also been associated with infarct size and impaired myocardial reperfusion in patients with acute myocardial infarction [13,22]. Moreover, a recent prospective cohort study that investigated the prognostic value of baseline QRS scores on MACE or mortality, Uyarel et al. [23] included 112 acute STEMI patients who underwent successful primary PCI and demonstrated that a high QRS score (≥4) was a strong and independent predictor of incomplete ST-segment recovery and the 30-day risk of MACE [odds ratio (OR) 4.1, 95% CI 1.5–10.7 and OR 1.8, 95% CI 1.1–2.9, respectively]. Similar to these results, both the 2-year mortality and MACE rates were strongly associated with baseline QRS scores, and the association between baseline QRS scores and poor outcomes was not weakened even after adjusting for other variables. Moreover, a higher QRS score at discharge has also been verified to be associated with poor outcomes in previous clinical trials. Tjandrawidjaja et al. [14] conducted a prospective study of 5745 patients with PCI-treated STEMI and found that a higher QRS score at hospital discharge was an independent and prognostically relevant metric. Using the data obtained from the GUSTO-I trial, Barbagelata et al. [24] found that the risk of death was higher in patients with higher QRS scores (≥10) than in those with a QRS score <10 (30-day, 8.9% versus 2.9%, 1-year, 12.6% versus 5.4%). Nevertheless, to the best of our knowledge, this is the first study to investigate the relationship between ΔQRS scores and the 2-year prognosis in acute STEMI patients who underwent PCI, and we found that the risk of experiencing a MACE and the 2-year mortality rate were 1.6-fold and 4.1-fold higher, respectively, in the group that showed positive changes in the QRS score.

What the QRS score means with regard for pathophysiology remains unclear, although several findings from previous studies may feasibly support the notion that this phenomenon is dynamic. In patients with STEMI, QRS prolongation was considered a mainly dynamic phenomenon that was induced by ischemia and likely relieved by successful reperfusion [25]. Kacmaz et al. [26] conducted a study of 165 acute myocardial infarction patients who were administered fibrinolytic therapy for reperfusion and found that there was a more significant narrowing in QRS duration (calculated by subtracting the post fibrinolysis QRS duration from the admission QRS duration), indicating that the QRS score increased more after reperfusion therapy in the reperfusion group than in the impaired reperfusion group. We hypothesized that in patients with larger ischemic areas, who have higher baseline QRS scores, positive or no change in the QRS score may reflect impaired reperfusion.

This study has several major clinical significances. First, to our knowledge, this is the first study to investigate the relationship between ΔQRS scores and 2-year prognoses. Second, we employed a prospective observational

Table 4 Cox proportional hazards analysis for 2-year mortality and major adverse cardiovascular event (n = 289)

|	Univariate		Multivariable	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
For 2-year mortality				
QRS score at admission	3.185 (1.856–5.464)	<0.001	1.462 (1.279–1.671)	<0.001
ΔQRS >0	7.144 (1.222–14.767)	<0.001	5.122 (2.128–12.328)	0.020
Age	0.927 (0.864–1.095)	0.136	1.018 (0.983–1.054)	0.309
Male (versus female)	0.639 (0.275–1.482)	0.297	0.498 (0.070–3.565)	0.488
BMI	1.078 (0.882–1.271)	0.236	0.152 (0.949–1.398)	0.152
Leucocyte	1.151 (1.064–1.244)	<0.001	1.042 (0.906–1.199)	0.565
DBP	0.969 (0.940–0.999)	0.043	0.860 (0.783–0.945)	0.002
Preexisting CKD	1.002 (0.235–4.263)	0.998		
Hypertension	1.665 (0.748–3.708)	0.212		
Diabetes	1.365 (0.544–3.425)	0.507		
Dyslipidemia	1.865 (0.440–7.915)	0.398		
Troponin I, per 10 ug/L increase	1.028 (1.009–1.057)	0.038	1.124 (1.001–1.263)	0.048
CKMB, per 10 IU/L increase	1.031 (1.007–1.054)	0.010	1.020 (0.956–1.190)	0.080
NT-proBNP, per 10 pg/mL increase	1.012 (1.006–1.012)	<0.001	1.021 (1.001–1.107)	0.040
Killip class	2.209 (1.616–3.019)	<0.001	2.872 (1.264–6.527)	0.012
Serum creatinine	1.013 (1.002–1.025)	0.027	1.036 (0.893–1.631)	0.217
Total cholesterol	0.991 (0.866–1.135)	0.900		
Serum triglyceride	0.725 (0.419–1.255)	0.250		
Low-density lipoprotein	1.222 (1.073–1.393)	0.003	1.065 (1.025–1.107)	0.001
For MACE				
QRS score at admission	1.089 (1.038–1.143)	<0.001	1.119 (1.019–1.229)	0.019
ΔQRS>0	5.350 (3.006–9.524)	<0.001	2.585 (1.260–5.303)	0.010
Age, year	1.001 (0.980–1.022)	0.921	0.969 (0.934–1.005)	0.094
Male (versus female)	0.863 (0.501–1.487)	0.596	1.322 (0.555–3.146)	0.529
BMI	0.981 (0.915–1.051)	0.583	0.989 (0.888–1.103)	0.848
Leucocyte	1.006 (0.988–1.024)	0.508		
DBP	0.990 (0.972–1.008)	0.281		
Preexisting CKD	1.661 (0.670–4.118)	0.273		
Hypertension	1.467 (0.889–2.419)	0.134		
Diabetes	1.126 (0.636–1.995)	0.684		
Dyslipidemia	1.056 (0.522–2.136)	0.880		
Troponin I, per 10 ug/L increase	1.089 (1.038–1.143)	<0.001	1.058 (1.034–1.082)	0.001
CKMB, per 10 IU/L increase	1.029 (1.009–1.048)	0.003	1.032 (1.009–1.054)	0.005
NT-proBNP, per 10 pg/mL increase	1.014 (1.006–1.022)	0.001	1.005 (1.002–1.012)	0.002
Killip class	1.984 (1.453–2.707)	<0.001	1.796 (1.438–2.243)	<0.001
Serum creatinine	1.012 (1.004–1.020)	0.002	1.006 (0.994–1.018)	0.308
Total cholesterol	0.974 (0.857–1.107)	0.685		
Serum triglyceride	0.991 (0.793–1.238)	0.935		
Low-density lipoprotein	1.029 (0.777–1.364)	0.840		

95% CI, 95% confidence interval; CKD, chronic kidney disease; CKMB, creatine kinase-MB; HR, hazard ratio; MACE, major adverse cardiovascular event; NT-proBNP, N-terminal pro-brain natriuretic peptide.

Table 5 Cox proportional hazards analysis for composite endpoint (n = 289)

	Univariate		Multivariable	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
QRS score at admission	1.123 (1.044–1.207)	0.002	1.137 (1.047–1.236)	0.002
ΔQRS >0	4..964 (3.002–8.209)	<0.001	3.152 (1.704–5.829)	<0.001
Age	1.005 (0.986–1.025)	0.613	0.989 (0.961–1.018)	0.453
Male (versus female)	0.779 (0.472–1.285)	0.328	0.836 (0.450–1.750)	0.636
BMI	0.941 (0.891–0.995)	0.031	0.992 (0.931–1.056)	0.792
Leucocyte	1.108 (1.058–1.161)	<0.001	1.042 (0.963–1.127)	0.307
DBP	0.984 (0.967–1.000)	0.057	0.860 (0.783–0.945)	0.002
Preexisting CKD	1.799 (0.949–3.411)	0.072	1.629 (0.513–5.173)	0.408
Hypertension	1.078 (0.690–1.684)	0.742		
Diabetes	1.457 (0.927–2.290)	0.103		
Dyslipidemia	0.870 (0.448–1.691)	0.682		
Troponin I, per 10 µg/L increase	1.022 (1.005–1.040)	0.013	0.986 (0.944–1.029)	0.505
CKMB, per 10 IU/L increase	1.028 (1.012–1.043)	<0.001	1.019 (0.988–1.050)	0.232
NT-proBNP, per 10 pg/ml increase	1.015 (1.008–1.022)	<0.001	1.006 (0.998–1.015)	0.149
Killip class	1.808 (1.466–2.230)	<0.001	1.653 (1.191–2.294)	0.003
Serum creatinine	1.008 (1.001–1.015)	0.029	1.006 (0.994–1.017)	0.352
Total cholesterol	0.983 (0.896–1.077)	0.900		
Serum triglyceride	0.921 (0.746–1.137)	0.445		
Low-density lipoprotein	1.092 (0.852–1.399)	0.487		

95% CI, 95% confidence interval; CKD, chronic kidney disease; CKMB, creatine kinase-MB; HR, hazard ratio; NT-proBNP, N-terminal pro-brain natriuretic peptide.

Fig. 1
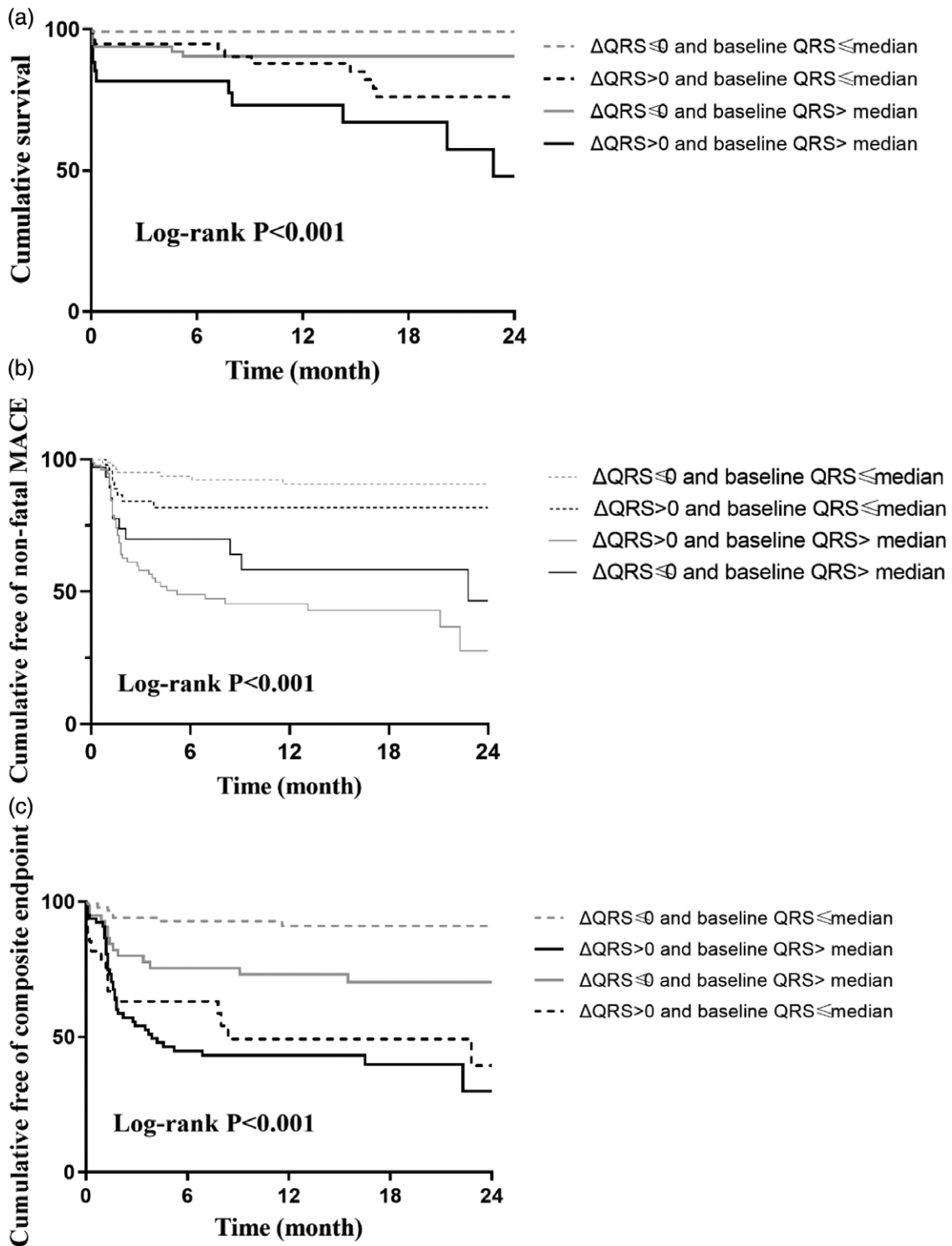
Kaplan–Meier curves for 2-year survival (a), free of non-fetal MACE (b) and free of composite endpoint (c) according to the level of baseline QRS score and ΔQRS score. MACE, major adverse cardiovascular event.

design and a rigorous protocol for patient screening and performed the QRS measurements in a blinded manner. Third, in all of the patients in this study, echocardiography was performed during their hospital stay and the correlations between QRS scores and both echocardiographic parameters and cardiac enzyme levels calculated. Finally,

the use of baseline QRS scores and the ΔQRS score as predictors for short-term prognosis was assessed during the 2-year follow-up period in these acute STEMI patients, further broadening the clinical implications of the QRS score in disease prognosis.

This study also has some limitations. First, it was a single-center study of 289 patients. Second, we measured the QRS score at admission and calculated the change in the QRS score between before and after treatment with PCI, but we did not calculate the QRS score at discharge or during the 2-year follow-up, although these parameters may also have been predictors, as shown in previous studies. Finally, only 15 patients died during the 2-year follow-up period, and further large longitudinal studies are therefore needed to verify our findings.

Conclusion

In summary, the current study shows that both the baseline QRS score and the ΔQRS score could serve as early predictors for the risk of MACE and mortality in acute STEMI patients who undergo PCI treatment. If further confirmed, given that the QRS score is a readily available parameter that does not require additional costs, we propose that it could be used as a useful index for stratifying the risk of experiencing a MACE and mortality. Moreover, patients who experienced an increase in the QRS score during treatment with PCI should be carefully followed up. Continuous follow-up of the QRS score could provide information useful for the management of patients with acute STEMI. Some problems remain to be further investigated; these include how often high-risk patients should be followed-up, how the health care costs incurred by patients should be covered, and so on.

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