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
Qian Liu, Yong Zhang, Pengqiang Zhang, Junbo Zhang, Xiaojiao Cao, Shanshan He and Donghui Yang

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.

Keywords: acute ST-segment elevation myocardial infarction, percutaneous coronary intervention, prognosis, Selvester QRS score

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; 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 thrombolyisis in myocardial infarction III blood flow and <30%
residual stenosis in the target vessel). The exclusion cri-
teria 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 col-
clected for all the patients enrolled in the study imme-
diately 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 clas-
sification of diseases, 10th revision (ICD-10) codes I21,
I22], nonfatal stroke (ICD-10 codes I60–69) and read-
mission 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 con-
sidered 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 treat-
ment and within 24 hours after treatment for PCI by
electrocardiograph (FCP-7541; Fukuda Denshi Co. Ltd,
Tokyo, Japan). The QRS score was calculated accord-
ing to a 50-criteria 31-point Selvester QRS scoring sys-
tem [19] first at the time of hospital admission (baseline
Selvester QRS score) and then within 24 hours after treat-
ment for PCI. The QRS score was manually calculated by
two expert cardiologists according to an algorithm, as pre-
viously 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 compli-
cations at the puncture site. In accordance with revasculari-
zation guidelines, dual anti-platelet therapy consisting of
aspirin (300 mg per os followed by 100 mg daily) and clopi-
dogrel (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, angioten-
sin-converting enzyme inhibitors, β-blockers and
nitrates were used if there were no contraindications.

Echocardiography measurements
All of the echocardiography measurements were per-
formed 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 (χ²) to
compare categorical variables between groups. Spearman
correlation coefficients were calculated between QRS
scores and echocardiographic parameters. Prognostic indi-
cators 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 mul-
tivariable 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 con-
sidered 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 change 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, 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 ΔQRS score. Among them, 73 (25.6%) patients showed any increase in Selvester QRS score at 24 hours post-PCI, while the ΔQRS score >0 group included patients who had a MACE. The baseline QRS score, leucocyte count, 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 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 ΔQRS score >0, the baseline QRS score, increased TnI levels, increased CKMB levels, increased NT-proBNP levels, 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 a ΔQRS score >0 (HR 3.152, 95% CI 1.260–5.303, P = 0.02) were significant predictors of mortality after adjustment for age, sex, BMI, leucocyte counts, Killip class, DBP, 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 a ΔQRS score >0 (HR 3.152, 95% CI 1.260–5.303, P = 0.010) remained crucial predictors of a MACE even after adjustment for age, sex, BMI, leucocyte counts, Killip class, DBP, TnI, CKMB, NT-proBNP, serum creatinine and LDL 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   |
| 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 (>10⁷/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 |         |                  |         |
| 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    | 320.2 ± 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)    | 278.2 ± 29.8    | 278.2 ± 29.8     | 0.770   |
| Killip           |                  |                  |         |
| Class I         | 77 (67.0)       | 129 (74.1)       | 0.018   |
| Class II        | 22 (19.1)       | 36 (20.7)        |         |
| Class III       | 3 (2.6)         | 5 (2.9)          | 0.0001  |
| 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*    | 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.

*Readmission means readmission for unstable angina or heart failure.

**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).
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) |

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/m²)   | 0.148  | 0.034  | 0.158  | 0.010  |
| LAVI (m²/m²)  | 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 |
| Tnl           | 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.

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]. Kaemaz 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 prognosis. Second, we employed a prospective observational 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 (≥10) 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.

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 our knowledge, this is the first study to investigate the relationship between ΔQRS scores and 2-year prognosis. Second, we employed a prospective observational
QRS score predicts prognosis in patients with acute STEMI
Liu et al.

### 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 (1.856–5.644) | <0.001 | 1.462 (1.279–1.671) | <0.001 |
| ∆QRS >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 | 0.915 (0.949–1.398) | 0.152 |
| Leucocyte           | 1.151 (1.064–1.244) | <0.001 | 1.042 (0.906–1.199) | 0.565 |
| DBP                 | 0.989 (0.940–0.999) | 0.043 | 0.860 (0.783–0.945) | 0.002 |
| Preexisting CKD     | 1.002 (0.233–4.263) | 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 l, 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 | 0.320 (1.001–1.137) | 0.400 |
| Killip class        | 2.209 (1.618–3.019) | <0.001 | 2.872 (1.264–6.527) | 0.012 |
| Serum creatinine    | 1.013 (1.002–1.025) | 0.027 | 1.036 (0.893–1.361) | 0.217 |
| Total cholesterol   | 0.991 (0.886–1.135) | 0.800 |               |        |
| Serum triglyceride  | 0.725 (0.419–1.253) | 0.250 |               |        |
| Low-density lipoprotein | 1.222 (1.073–1.393) | 0.003 | 1.065 (1.025–1.107) | 0.001 |

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 |
| For MACE            |            |               |            |        |
| QRS score at admission| 1.089 (1.038–1.143) | <0.001 | 1.119 (1.019–1.229) | 0.019 |
| ∆QRS >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) | 0.586 | 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        | 1.487 (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 l, per 10 ug/L increase | 1.088 (1.038–1.143) | <0.001 | 1.058 (1.034–1.082) | 0.001 |
| CKMB, per 10 IU/L increase | 1.023 (1.009–1.048) | 0.003 | 1.032 (1.009–1.054) | 0.005 |
| NT-proBNP, per 10 pg/mL increase | 1.012 (1.006–1.012) | <0.001 | 0.320 (1.001–1.137) | 0.400 |
| Killip class        | 1.984 (1.453–2.707) | <0.001 | 1.766 (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 | 1.023 (0.777–1.384) | 0.840 |               |        |

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

**Acknowledgements**

Conflicts of interest

There are no conflicts of interest.

**References**

1. Kedev S, Sukmawan R, Kalpak O, Dharma S, Antov S, Kostov J, et al. Transradial versus transfemoral access for female patients who underwent primary PCI in STEMI: two years follow-up data from acute STEMI interventional registry. *Int J Cardiol* 2016; 217 (Suppl):S16–S20.

2. Choudry FA, Weerackody RP, Timmis AD, Wragg A, Mathur A, Sporton S, et al. Importance of primary percutaneous coronary intervention for reducing mortality in ST-elevation myocardial infarction complicated by out of hospital cardiac arrest. *Eur Heart J Acute Cardiovasc Care* 2015; 4:378–385.

3. Jernberg T, Johanson P, Held C, Svennblad B, Lindback J, Wallentin L. Association between adoption of evidence-based treatment and survival for patients with ST-elevation myocardial infarction. *JAMA* 2011; 305:1677–1684.

4. Wu X, Zhang Y, Wu Z, You W, Liang F, Ye F, et al. Plasma lipoprotein-associated phospholipase A2 level is an independent predictor of high thrombus burden in patients with acute ST-segment elevation myocardial infarction. *Int Heart J* 2016; 57:689–696.

5. Cicek G, Korkmaz A. Two-year prognosis of admission hemoglobin A1c following a primary percutaneous coronary intervention. *Coron Artery Dis* 2016; 27:673–681.

6. Kacprzak M, Zielińska M. Prognostic value of myeloperoxidase concentration in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. *Int J Cardiol* 2016; 223:452–457.

7. Balta S, Cökt T, Ozturk C, Kaya MG, Aparci M, Yildirim AO, et al. The relation between monocyte to HDL ratio and no-reflow phenomenon in the patients with acute ST-segment elevation myocardial infarction. *Am J Emerg Med* 2016; 34:1542–1547.

8. Leibundgut G, Gick M, Morel O, Ferenc M, Werner KD, Comberg T, et al. Discordant cardiac biomarker levels independently predict outcome in ST-segment elevation myocardial infarction. *Clin Res Cardiol* 2016; 105:432–440.

9. Strauss DG, Selvester RH. The QRS complex—a biomarker that “images” the heart: QRS scores to quantify myocardial scar in the presence of normal and abnormal ventricular conduction. *J Electrocardiol* 2009; 42:85–96.

10. Ling Z, Chelliah S, Selvester RH, Wagner G, Strauss DG. A detailed guide for quantification of myocardial scar with the Selvester QRS score in the presence of electrocardiogram confounders. *J Electrocardiol* 2011; 44:544–554.

11. Watanabe N, Isobe S, Okumura T, Mori H, Yamada T, Nishimura K, et al. Relationship between QRS score and microvascular obstruction after acute anterior myocardial infarction. *J Cardio* 2016; 67:321–326.

12. Carlisen EA, Bang LE, Ahtarosvski KA, Engstrom T, Keber L, Kelbaek H, et al. Comparison of Selvester QRS score with magnetic resonance imaging measured infarct size in patients with ST elevation myocardial infarction. *J Electrocardiol* 2012; 45:414–419.

13. Kosuge M, Ebina T, Hita K, Iwashahi N, Teukahara K, Endo M, et al. High QRS score on admission strongly predicts impaired myocardial reperfusion in patients with a first anterior acute myocardial infarction. *Circ J* 2011; 75:626–632.

14. Tjandrawidjaja MC, Fu Y, Westerhout CM, Wagner GS, Granger CB, Armstrong PW; APEX-AMI Investigators. Usefulness of the QRS score as a strong prognostic marker in patients discharged after undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. *Am J Cardiol* 2010; 106:630–634.

15. Abdel-Salam Z, Wafa S, Kamel S, Nammas W. The modified Selvester QRS score: can we predict successful ST segment resolution in patients with myocardial infarction receiving fibrinolytic therapy? *Cardiol J* 2010; 17:367–373.

16. O’Gara PT, Kushner FG, Ascheim DD, Casey DE Jr, Chung MK, de Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. *J Am Coll Cardiol* 2013; 61:e78–e140.

17. Illing E, Altman DG, Egger M, Poosock SJ, Gatsche PC, Vandebroucke JP, STROBE Initiative. The strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *Ann Intern Med* 2007; 147:573–577.

18. Bossuyt PM, Reitsma JB, Bruin DE, Gatsonis CA, Glasziou PP, Irwig LM, et al.; STARD Group. Toward complete and accurate reporting of studies of diagnostic accuracy: the STARD initiative. *Acad Radiol* 2003;10:644–649.

19. Horacek BM, Warren JW, Albano A, Palmeri MA, Rembert JC, Greenfield JC Jr, Wagner GS. Development of an automated Selvester scoring system for estimating the size of myocardial infarction from the electrocardiogram. *J Electrocardiol* 2006; 39:162–168.

20. Lang RM, Biering M, Devereux RB, Flachkampf FA, Foster E, Pellikka PA, et al.; Chamber Quantification Writing Group; American Society of Echocardiography’s Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society Of Echocardiography’s guidelines and standards committee and the chamber quantification writing group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr* 2005; 18:1440–1463.

21. Palmeri ST, Harrison DG, Cobb FR, Morris KG, Harrell FE, Iedeker RE, et al. A QRS scoring system for assessing left ventricular function after myocardial infarction. *N Engl J Med* 1982; 306:4–9.

22. Shimoni H, Kosuge M, Morimoto T, Watanabe H, Taniguchi T, Nakatsuka K, et al.; CREDO-Kyoto AMI Investigators. QRS score at presentation electrocardiogram is correlated with infarct size and mortality in ST-segment elevation myocardial infarction patients undergoing primary percutaneous coronary intervention. *Circ J* 2017; 81:1129–1136.

23. Uyarel H, Cam N, Okmen E, Kasikcioglu H, Tartan Z, Akgul O, et al. Level of Selvester QRS score is predictive of ST-segment resolution and 30-day outcomes in patients with acute myocardial infarction undergoing primary coronary intervention. *Am Heart J* 2006; 151:1231–1239.
24 Barbagelata A, Califf RM, Sgarbossa EB, Knight D, Mark DB, Granger CB, et al. Prognostic value of predischarge electrocardiographic measurement of infarct size after thrombolysis: insights from GUSTO I Economics and Quality of Life substudy. Am Heart J 2004; 148: 795–802.

25 Tsukahara K, Kimura K, Kosuge M, Shimizu T, Sugano T, Hibi K, et al. Clinical implications of intermediate QRS prolongation in the absence of bundle-branch block in patients with ST-segment-elevation acute myocardial infarction. Circ J 2005; 69: 29–34.

26 Kacmaz F, Maden O, Aksuyek S, Ureyen C, Alyan O, Erbay AR, et al. Relationship of admission QRS duration and changes in QRS duration with myocardial reperfusion in patients with acute ST segment elevation myocardial infarction (STEMI) treated with fibrinolytic therapy. Circ J 2008; 72: 873–879.