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Myocardium at risk assessed by electrocardiographic scores and cardiovascular magnetic resonance - a MITOCARE substudy

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Abstract

Introduction: The myocardium at risk (MaR) represents the quantitative ischemic area destined to myocardial infarction (MI) if no reperfusion therapy is initiated. Different ECG scores for MaR have been developed, but there is no consensus as to which should be preferred.
Objective: Comparisons of ECG scores and Cardiac Magnetic Resonance (CMR) for determining MaR.
Methods: MaR was determined by 3 different ECG scores, and by CMR in ST-segment elevation MI (STEMI) patients from the MITOCARE cardioprotection trial. The Aldrich score (AL) is based on the number of leads with ST-elevation for anterior MI and the sum of ST-segment elevation for inferior MI on the admission ECG. The van Hellemond score (VH) considers both the ischemic and infarcted component of the MaR by adding the AL and the QRS score, which is an estimate of final infarct size. The Hasche score is based on the maximal possible infarct size determined from the QRS score on the baseline ECG.
Results: Ninety-eight patients (85% male, mean age 61 years) met STEMI criteria on their admission ECG and underwent CMR within 3–5 days after STEMI. Mean MaR by CMR was 41.2 ± 10.2 and 30.3 ± 7.2 for anterior and inferior infarcts, respectively. For both anterior and inferior infarcts the Aldrich (18.2 ± 5.1 and 18.6 ± 6.0) and Hasche (25.3 ± 9.8 and 26.4 ± 8.8) scores significantly underestimated MaR compared to MaR measured by CMR. In contrast, MaR by the van Hellemond score (37.0 ± 14.2 and 31.7 ± 12.8) was comparable to CMR.
Conclusion: We tested the performance of the electrocardiographic estimation of myocardium area at risk by Aldrich, Hasche and van Hellemond ECG scores in comparison to MaR measured by CMR in STEMI patients. MaR by the van Hellemond score and CMR were comparable, while Aldrich and Hasche underestimated MaR.

Keywords: Myocardium at risk; Electrocardiogram; Cardiovascular magnetic resonance

Introduction

The ECG is ideally recorded at the time of first medical contact in the prehospital setting for early triage and initiation of treatment in patients with chest pain. The myocardium at risk (MaR) represents the quantitative ischemic area destined to myocardial infarction (MI) if no reperfusion therapy is initiated, and is thus a measure of what can be saved by rapid reperfusion. Different ECG scores for MaR have been developed, but there is no consensus as to which should be the preferred method. Cardiovascular magnetic resonance (CMR) is considered a reference standard for determining MaR [1]. However, comparisons to the various ECG methods remain scarce. The purpose of this study was to test the performance of various

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ECG scores in estimating MaR from the admission 12-lead ECG in comparison to CMR measured MaR.

Methods

Patient population

The MITOCARE study was a phase II, multicenter, randomized, double-blind, placebo controlled study assessing the safety and efficacy of TRO40303 for reduction of reperfusion injury in patients undergoing primary percutaneous coronary intervention (pPCI) for acute MI [2]. However, the main results showed no effect of TRO40303 in limiting reperfusion injury of the ischemic myocardium [3]. The MITOCARE inclusion criteria were: First AMI; Occlusion affecting one of the main coronary arteries (left anterior descending artery (LAD), dominant or balanced right coronary artery (RCA), or dominant or balanced left circumflex artery (LCX)); Nitrate resistant chest pain for at least 30 min; Presentation within 6 h of onset of chest pain; New ST-segment elevation in two contiguous leads (cut-off points: ≥ 0.2 mV in men or ≥ 0.15 mV in women in leads V2–V3, and/or ≥ 0.1 mV in other leads); and Thrombolysis in myocardial infarction (TIMI) flow grade 0–1 before pPCI [2]. The 165 patients enrolled in the MITOCARE study were eligible for the present study. Patients were excluded if they did not undergo CMR imaging, did not meet the ST-segment elevation criteria listed above, had incomplete or missing ECG, or had ECG confounders e.g. bundle branch block.

Electrocardiographic analysis

Standard 12-lead electrocardiograms were recorded at hospital admission, at 72 h, and 30 days following pPCI. The electrocardiographic analyses were performed in the ECG Core Laboratory at Rigshospitalet (Copenhagen, Denmark). ST-segment deviation was measured manually to the nearest 0.5 mm at the J-point in all 12 leads using the TP-segment as the isoelectric line. Alternatively, the PR-segment was used if the TP-segment was not distinct. The ECG investigators were blinded to all patient data.

ECG measures of myocardium at risk

Aldrich score

The Aldrich score [4] estimates the extent of acute ischemia by considering quantitative changes in leads with at least 0.1 mV ST-segment elevation on the presenting ECG. The score was developed in a population not receiving reperfusion therapy, and thus the method predicts the area at risk of infarction in case of no reperfusion treatment as the percentage of the left ventricle. The MaR was calculated for anterior and inferior infarction as:

$$\text{Anterior infarction : Myocardium at risk\%} = 3\,[1.5\,(\text{no.of leads with ST}\uparrow) - 0.4]$$

$$\text{Inferior infarction : Myocardium at risk\%} = 3\left[0.6\left(\sum \text{ST}\uparrow \text{II, III, aVF}\right) + 2.0\right]$$

QRS score

The Selvester QRS score estimates the final MI size [5,6]. The score was initially developed to assess the percentage of infarcted left ventricle mass in the chronic phase in patients who had not received reperfusion therapy using anatomical post-mortem histopathology studies of anterior and inferior infarcts [7–9]. The score has since been validated using technetium Tc 99 m pyrophosphate single photon emission computed tomography (SPECT) and delayed enhancement magnetic resonance imaging [10,11]. The QRS score system contains 53 criteria considering Q and R wave duration and relative Q, R and S wave amplitudes awarding a maximum of 31 points, each representing approximately 3% infarction of the left ventricle. The QRS score was determined at the admission, 72-h, and 30-day ECG ($QRS_{baseline}$, QRS_{72h}, QRS_{30D}).

van Hellemond score

The van Hellemond score [12,13] was developed based on the assumption that the Aldrich score only estimates the ischemic component of the MaR and not considers the presence of any infarcted component. Thus, the sum of the Aldrich score and the QRS score obtained on the admission ECG ($QRS_{baseline}$) were added in the van Hellemond score to estimate the MaR by considering both the ischemic and the infarcted component.

$$\text{Van Hellemond score} = \text{Aldrich score} + QRS_{baseline}$$

Hasche score

The QRS score performed in the chronic phase estimates final infarct size. Hasche et al. [14] identified MaR on the baseline ECG by assigning the maximum potential QRS score for all leads exhibiting ≥ 1 mV ST-segment elevation. For patients with inferior infarcts, leads V_1 and V_2 were included as a posterior extension of the risk region if there was ≥ 1 mV ST-segment depression in these leads. The sum of the initial QRS scores (QRS_{max}) was considered to represent the potential maximum infarct size analogous to the extent of the MaR.

$$\text{Hasche score} = QRS_{Max}$$

Cardiac biomarkers and echocardiography

Creatine kinase myocardial band (CKMB) and troponin I were obtained at admission and repeatedly over the next 3 days. Global left ventricular function was assessed by echocardiography performed between day 3 and day 5 post pPCI and at 1 month.

Cardiovascular magnetic resonance

CMR images were acquired 3 to 5 days after the acute infarction on whole-body MRI scanners. MaR was assessed by gadolinium-based contrast enhanced steady state free precession cine imaging [15]. Final infarct size was determined by late gadolinium enhanced (LGE) images using a previously described automatic infarct quantification method [16].