Relationship between ST-segment resolution and anterior infarct size after primary percutaneous coronary intervention: analysis from the INFUSE-AMI trial

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Abstract

Aims: ST-segment resolution (STR) after reperfusion therapy has been shown to correlate with prognosis in patients with ST-segment elevation myocardial infarction (STEMI). We investigated whether acute ECG measurements also correlate with ultimate infarct size.

Methods and Results: The INFUSE-AMI trial randomized 452 patients with anterior STEMI to intracoronary bolus abciximab vs. no abciximab, and to thrombus aspiration vs. no aspiration. Infarct size as percentage of total LV mass was calculated by cardiac magnetic resonance imaging (MRI) 30 days post intervention. Five ECG methods were analysed for their ability to predict MRI infarct mass: (1) summed STR across all infarct-related ECG leads (Σ STR); (2) STR in the single lead with maximum baseline ST-segment elevation (maxSTR); (3) summed residual ST-segment elevation across all infarct-related leads at 60 min post intervention (Σ ST residual); (4) maximum residual ST-segment elevation in the worst single lead at 60 min post intervention (maxST residual); (5) number of new significant Q-waves (Qwave) at 60 min. All ECG methods strongly correlated with 30-day MRI infarct mass (all $p < 0.003$). Simpler ECG measurements such as maxST residual and Qwave were as predictive as more complex measurements. A subset analysis of 158 patients who had microvascular obstruction (MVO) determined by MRI 5 days post intervention also showed strong correlations of MVO with the ECG measures.

Conclusions: ST-segment and Q-wave changes after primary PCI in anterior STEMI strongly correlated with 30-day infarct size by MRI. In particular, maxST residual and Qwave at 60 min are simple ECG parameters that offer rapid analysis for prognostication.

Keywords

Cardiac magnetic resonance imaging, electrocardiogram, ST elevation myocardial infarction, ST-segment resolution

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Introduction

Salvaging myocardium is the primary goal of reperfusion therapy, since infarct size correlates strongly with mortality after acute ST-segment elevation myocardial infarction (STEMI).¹ Estimating infarct size after reperfusion therapy thus offers important prognostic utility. Laboratory measures of infarct size include biomarkers such as creatine phosphokinase, lactate dehydrogenase, and troponin levels.

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While these tests are readily available and can be obtained in the acute setting, they are imprecise in comparison to infarct size assessed by cardiac imaging.^{2–5} Cardiac magnetic resonance imaging (MRI) has emerged as the technique of choice to measure infarct size and has been shown to be able to distinguish between reversible and irreversible ischaemic injury.⁶

ST-segment changes on the electrocardiogram (ECG) are one of the hallmarks of acute MI. The evolution of ST-segment changes has been shown to correlate with reperfusion success and prognosis, both with mechanical and pharmacological therapy.^{7–12} Multiple ST-segment parameters have been analysed during acute MI. Both simple and more complex measurements of ST-segment resolution (STR) have been shown to correlate with clinical events and peak serum biomarker levels.^{13,14} However, the correlation between biomarkers and infarct size is imperfect, and only a few small studies have examined the correlation between STR and infarct size determined by cardiac MRI.^{15–17}

In the prospective, randomized Intracoronary Abciximab and Aspiration Thrombectomy in Patients With Large Anterior Myocardial Infarction (INFUSE-AMI) trial, cardiac MRI was used to assess infarct size in patients undergoing primary percutaneous coronary intervention (PCI) for STEMI. Using the INFUSE-AMI trial database, we therefore sought to determine the correlation between various acute post-PCI ECG measurements and infarct size as determined by MRI performed 30 days after reperfusion therapy. We hypothesized that a correlation exists, which may lend useful clinical utility.

Methods

Study design

The INFUSE-AMI protocol and principal results have been recently published.^{18,19} This was a multicentre study that randomized patients with proximal or mid-left anterior descending artery occlusion in a 2×2 factorial design to bolus intracoronary abciximab vs. no abciximab and to manual aspiration thrombectomy vs. no thrombectomy. All patients received aspirin, clopidogrel, or prasugrel, and bivalirudin during the procedure. The primary endpoint was MRI infarct size at 30 days post intervention, expressed as percentage of total left ventricular mass. A subset of patients also had MRI performed 5 days post intervention in order to determine the degree of microvascular obstruction (MVO). ECG criteria for enrolment included ≥1-mm ST-segment elevation in the anterior precordial leads or new left-bundle branch block. Quantitative ST-segment and Q-wave analyses were performed from ECGs collected at baseline and 60 min post intervention. For the purposes of the current analysis, only paired, evaluable ECGs were utilized, and thus left-bundle branch block, paced, or

ventricular rhythms were excluded. ST-segment elevation on each lead at 40 ms after the end of the QRS was hand measured by core laboratory cardiologists to the nearest 0.1 mm using digital callipers, using the TP segment as baseline. Significant new Q-waves were defined as those not present on the baseline ECG and meeting established ACC/AHA criteria, namely Q-wave wider than 20 ms or QS complex in leads V2 or V3, or Q-wave wider than 30 ms and deeper than 1 mm or QS complex in the other leads.²⁰ ECG measurements were confirmed by a second ECG reader. ECGs and MRIs were analysed at independent core laboratories as previously described,^{18,19} with the readers blinded to treatment assignment and clinical outcomes.

ECG parameters

ECG measurements were defined as follows:

- summed percentage STR from the pre- to the post-intervention ECGs across all infarct-related leads (Σ STR), stratified as <30%, 30–70%, or >70%;
- percentage STR in the single lead with maximum baseline ST elevation (maxSTR), also stratified as <30%, 30–70%, or >70%;
- summed residual ST-segment elevation across all infarct-related leads on the 60-min post intervention ECG (Σ ST residual), stratified as <2, 2–4, or >4 mm;
- maximum residual ST-segment elevation in the worst single lead on the 60-min post-intervention ECG (maxST residual), stratified as <1, 1–3, or >3 mm;
- number of new significant Q-waves (Qwave) at 60 min post intervention, stratified as 0, 1–3, or >3.

Statistical analysis

Continuous variables are reported as mean and standard deviation, and categorical variables are reported as percentages. Spearman rank correlation coefficients were calculated for the various ECG measurements against MRI infarct mass. A two-tailed Student's t-test was also used to compare MRI infarct mass based on the three-step stratification schemes of the ECG measurements.¹⁴ A *p*-value <0.05 was considered statistically significant. All statistical analyses were performed using SAS version 9.2 (Cary, NC, USA).

Results

Baseline features

Between 28 November 2009 and 2 December 2011, 452 patients were enrolled into the INFUSE-AMI trial. The pooled baseline patient characteristics are shown in Table 1. There was a low incidence of prior MI and prior coronary intervention. Two-thirds of the patients had a

Table 1. Baseline patient characteristics.

Characteristic	Study population (n=452)
Age (years)	60.4±11.9
Male sex	331 (73.2)
Body mass index (kg/m ²)	27.3±4.6
Killip class	
I	372 (82.2)
II	40 (8.8)
III	5 (1.2)
Hypertension	142 (31.4)
Diabetes	49 (10.8)
Hyperlipidaemia	69 (15.2)
Prior myocardial infarction	3 (0.7)
Prior PCI	10 (2.2)
Cigarette smoking	278 (61.6)
Infarct lesion location	
Proximal LAD	296 (65.5)
Mid LAD	185 (40.9)
Left ventricular ejection fraction (%)	43.1±12.2
Symptom to balloon (min)	170±63

PCI= percutaneous coronary intervention, LAD = left anterior descending coronary artery.

proximal left anterior descending artery (LAD) infarct, and PCI was performed in most within 3 h of symptom onset. Thrombolysis in Myocardial Infarction (TIMI) 3 flow was restored in 413 (91.4%) patients.

MRI and ECG acquisition

There were 439 patients alive at 30 days, in whom a MRI was performed in 382 (the remainder not completing the MRI primarily due to refusal, withdrawal, or inability to complete the scan). Of those, 353 MRIs were deemed analysable for infarct size, with the remainder inadequate due to poor image acquisition. Mean infarct size at 30 days was 22.4±15.2 g (16.5±10.2% of total LV mass). Amongst the 353 patients with analysable MRIs, 320–328 pairs of evaluable ECGs were available for the various ECG measurements. There were 168 patients who also had MRI performed at 5 days post intervention in order to measure MVO. The mean value of MVO was 3.6±6.1 g. Of the 168 patients with an early MRI, 158 had paired, evaluable ECGs. An analysis of a random sampling of 50 ECGs showed that the intra- and interobserver variability in ST measurements was less than 0.2 mm. Figure 1 shows the distribution of ST-segment and Q-wave measurements based on the stratification schemes.

Relationship between ECG parameters and infarct size

Table 2 shows the correlations between the various ST-segment and Q-wave measurements and infarct size at 30 days, as well as the correlations with MVO at 5 days post intervention. All ECG measurements performed well

Figure 1. Distribution of ST-segment and Q-wave measurements by ECG measurements and frequencies based on tertiary stratification schemes.

See Methods for definition of individual ECG measurements.

Table 2. Correlations between electrocardiogram measurements, magnetic resonance imaging infarct mass, and microvascular obstruction at 5 days post intervention.

Variable	A	B	C	p-values			
				Spearman rank	A vs. B	A vs. C	B vs. C
ΣSTR (%)	<30%	30–70%	>70%				
%infarct mass	17.6±9.3 (n=61)	18.1±9.3 (n=98)	14.7±10.9 (n=163)	0.0007	0.75	0.06	0.009
MVO% mass	1.9±2.1 (n=22)	2.7±3.4 (n=47)	1.8±3.5 (n=89)	<0.0001	0.37	0.92	0.16
MaxSTR	<30%	30–70%	>70%				
%infarct mass	18.1±10.1 (n=39)	17.5±9.0 (n=103)	15.2±10.8 (n=178)	0.003	0.76	0.10	0.06
MVO% mass	2.4±2.9 (n=10)	2.3±2.9 (n=53)	1.9±3.6 (n=94)	<0.0001	0.91	0.69	0.59
ΣST residual	<2 mm	2–4 mm	>4 mm				
%infarct mass	12.0±10.5 (n=89)	13.6±9.7 (n=87)	20.2±8.8 (n=152)	<0.0001	0.28	<0.0001	<0.0001
MVO% mass	0.6±1.5 (n=41)	1.3±2.7 (n=45)	3.4±3.8 (n=72)	<0.0001	0.29	<0.0001	0.0004
MaxST residual	<1 mm	1–2 mm	>2 mm				
%infarct mass	12.2±11.0 (n=90)	15.9±9.8 (n=146)	20.7±8.2 (n=92)	<0.0001	0.005	<0.0001	0.0003
MVO% mass	0.5±1.0 (n=44)	2.2±3.7 (n=70)	3.6±3.4 (n=44)	<0.0001	0.006	<0.0001	0.02
Qwave	0	1–3	>3				
%infarct mass	11.3±10.1 (n=106)	18.7±9.2 (n=206)	24.1±7.5 (n=12)	<0.0001	<0.0001	<0.0001	0.06
MVO% mass	0.4±0.9 (n=46)	2.7±3.6 (n=103)	3.7±5.0 (n=9)	<0.0001	<0.0001	0.005	0.35

ΣSTR, summed STR across all infarct-related ECG leads; maxSTR, STR in the single lead with maximum baseline ST-segment elevation; ΣST residual, summed residual ST-segment elevation across all infarct-related leads at 60 min post intervention; maxST residual, maximum residual ST-segment elevation in the worst single lead at 60 min post intervention; Qwave, number of new significant Q-waves at 60 min.

in terms of overall correlation with MRI infarct size, and simple measurements such as maxST residual or Qwave were equivalent to the more complex STR measurements. Residual ST-segment and Q-wave measurements were superior to the STR methods in terms of the comparisons based on the three-step stratification schemes. Likewise, the ECG measures correlated well with MVO, with the residual ST-segment and Q-wave measurements also performing well in terms of the three-step stratification scheme. Table 3 shows the correlation between the residual ST-segment and Q-wave measurements and 30-day outcomes. Only new Q-waves had positive predictive value for mortality at 30 days.

Discussion

This study demonstrates that among patients undergoing primary PCI, ECG measurements in the acute setting of MI correlate with 30-day infarct size as determined by cardiac MRI. Simple measurements such as maxST residual and Qwave correlated as well with infarct size as did more complex measurements that involved STR. In addition, the ECG measures correlated well with MVO as determined by MRI at 5 days post intervention. To our knowledge, this is the largest study to correlate infarct size as determined by MRI with a variety of ECG measurements in patients with acute STEMI. Previous studies were small in size, and most analysed only one or two ECG parameters.^{15–17,21}

Prior studies have also demonstrated that STR measurements after primary PCI have prognostic value for predicting clinical outcomes.^{7–12} Less complete ST-segment

resolution, or more residual ST-segment elevation may be thought of as representing unstable substrate vulnerable to further ischaemic events. The ECG measures correlated well with MVO as determined by early MRI, consistent with this concept. The current analysis also suggests that ECG measurements in the acute setting correspond to the ultimate amount of myocardial damage.

Our data parallels that reported from the CADILLAC trial¹³ and the study by Buller et al.¹⁴ in that simple ST-segment and Q-wave parameters performed as well as more complex measurements (for the prediction of clinical outcomes and biomarker-estimated infarct size, respectively). The CADILLAC report represented a subset of a larger group of patients and included a wide (4 h) window for post-intervention ECGs.¹³ Buller et al.¹⁴ made use of biomarkers and showed a directional correlation with peak levels but did not include cardiac imaging for infarct size. The INFUSE-AMI trial population, in contrast, was a very homogeneous group of patients that used strict ECG recording times and independent, blinded core laboratories for ECG and MRI analysis. Patients in INFUSE-AMI had a very low incidence of prior MI or PCI and thus represented a group with large *de novo* anterior infarcts ideal for the current analysis. Similar to Buller et al.,¹⁴ we found utility in a three-step rather than binary cut point for ST-segment (especially maxST residual) and Q-wave measurements, in that a gradient in infarct size could be further differentiated.

Early post-reperfusion ST-segment measurements as a surrogate indicator of infarct size and clinical prognosis are practical given their low cost and ready availability in the

Table 3. ECG measurements and 30-day outcomes.

ΣSTR	<30%	30–70%	>70%	p-value
Death	0.0% (n=0)	3.2% (n=4)	3.3% (n=7)	0.30
Reinfarction	1.4% (n=1)	0.0% (n=0)	1.0% (n=2)	0.49
Death/reinfarction	1.4% (n=1)	3.2% (n=4)	3.8% (n=8)	0.62
MaxSTR	<30%	30–70%	>70%	
Death	0.0% (n=0)	1.6% (n=2)	3.9% (n=9)	0.20
Reinfarction	2.0% (n=1)	0.0% (n=0)	0.9% (n=2)	0.33
Death/reinfarction	2.0% (n=1)	1.6% (n=2)	4.4% (n=10)	0.32
ΣST residual	<2 mm	2–4 mm	>4 mm	
Death	5.7% (n=7)	0.9% (n=1)	1.7% (n=3)	0.04
Reinfarction	1.8% (n=2)	0.0% (n=0)	0.6% (n=1)	0.31
Death/reinfarction	6.5% (n=8)	0.9% (n=1)	2.3% (n=4)	0.03
MaxST residual	<1 mm	1–2 mm	>2 mm	
Death	4.0% (n=5)	3.4% (n=6)	0 (n=0)	0.12
Reinfarction	0.9% (n=1)	0.6% (n=1)	0.9% (n=1)	0.93
Death/reinfarction	4.9% (n=6)	4.0% (n=6)	0.9% (n=1)	0.22
Qwave	0	1–3	>3	
Death	3.8% (n=5)	1.2% (n=3)	15.0% (n=3)	0.0006
Reinfarction	1.5% (n=2)	0.4% (n=1)	0% (n=0)	0.44
Death/reinfarction	4.3% (n=6)	1.6% (n=4)	15.0% (n=3)	0.002

ΣSTR, summed STR across all infarct-related ECG leads; maxSTR, STR in the single lead with maximum baseline ST-segment elevation; ΣST residual, summed residual ST-segment elevation across all infarct-related leads at 60 min post intervention; maxST residual, maximum residual ST-segment elevation in the worst single lead at 60 min post intervention; Qwave, number of new significant Q-waves at 60 min.

post-PCIs setting. The early recognition of poor STR may imply the need for further interventions to enhance myocardial reperfusion. In this regard and as previously reported,^{13,14,22} a simple measure such as maxST residual at 60–90 min post PCI is at least equivalent to more complex measurements, is readily obtainable, and does not require comparison to the pre-intervention ECG.

There are several limitations of our study. INFUSE-AMI was restricted to patients with anterior infarcts due to proximal or mid LAD occlusion, and our conclusions may not apply to patients with non-anterior or prior MI. In addition, we analysed infarct size at 30 days and infarct maturation may be complete months later. Other than new Q-waves, the ECG parameters did not positively predict outcomes or mortality at 1 month, although the power of our study was limited in this regard given the low rate of death at this time point. However, other analyses from INFUSE-AMI examining microcirculatory reperfusion were able to show lower 30-day mortality in patients with myocardial blush grade 2/3 versus 0/1.²³

In conclusion, in patients with anterior STEMI undergoing primary PCI, 60-min post-reperfusion ECG measurements of infarct evolution correlated well with MRI measured infarct size at 30 days in the INFUSE-AMI study. Simple measurements such as the residual extent of ST-segment elevation and the development of new Q-waves performed as well as more complex measurements involving STR. Because of its low cost, ready availability, and demonstrated clinical prognostic utility, the ECG remains

an essential tool in the diagnosis and management of patients with acute MI.

Conflict of interest

GWS is a consultant to the Atrium Medical Corporation. CMG has received research grant support from Atrium Medical Corporation. The other authors declare no conflict of interest.

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