Potential significance of spontaneous and interventional ST-changes in patients transferred for primary percutaneous coronary intervention: observations from the ST-MONitoring in Acute Myocardial Infarction study (The MONAMI study)

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Aims In patients with ST-elevation myocardial infarction (STEMI) scheduled for primary percutaneous coronary intervention (primary PCI), acute risk-assessment may be valuable for tailoring of adjunctive therapy at the time of coronary intervention. The present study was designed to quantify pre-, per-, and post-interventional ST-changes, to evaluate whether a pre-specified continuous ST-monitoring classification provides potential prognostic information in the pre- and per-interventional phase, and to compare post-interventional ST-resolution parameters derived from continuous ST-monitoring and snapshot ECGs, respectively.

Methods and results In 92 STEMI patients, continuous ST-monitoring was initiated in the pre-hospital phase and continued during and 90 min following PCI. Patients were divided into three groups: (A) patients achieving spontaneous ST-resolution before PCI; (B) patients with preserved ST-elevation immediately before PCI and with no increase in ST-elevation during PCI; and (C) patients with preserved ST-elevation immediately before PCI and with increase in ST-elevation during PCI. Groups A (n = 22), B (n = 43), and C (n = 27) differed in peak level of troponin-T (1.4, 4.7, and 7.2 μg/L, P < 0.001), creatinine kinase MB isoenzyme (35, 150, and 325 μg/L, P < 0.001), and N-terminal pro-brain natriuretic peptide (NT-pro-BNP) (183, 175, and 269 pmol/L, P = 0.084) during admission, and left ventricular ejection fraction evaluated within 2 h of PCI (0.53, 0.48, and 0.45, P = 0.047) and after 3 months (0.58, 0.54, and 0.45, P < 0.001). Groups B and C also differed in time from first balloon inflation to ≥70% resolution of ST-elevation (14 vs. 42 min, P = 0.002), whereas no differences were observed in traditional 90 min ST-resolution analysis or angiographically assessed parameters.

Conclusion STEMI patients transferred for primary PCI are heterogeneous with respect to pre- and per-interventional ST-changes, and a pre-specified ST-monitoring classification seems useful for stratification of patients at time of PCI into groups with low, intermediate, and high risk profile. Furthermore, post-interventional ST-monitoring indicates that traditional 90 min ST-resolution analysis may have limited value in the era of primary PCI.

Introduction

In patients with ST-elevation myocardial infarction (STEMI) scheduled for primary percutaneous coronary intervention (primary PCI), a successful restoration of coronary epicardial blood flow may be achieved spontaneously1 or by coronary intervention.2-4 However, even among patients achieving thrombolysis in myocardial infarction (TIMI) flow grade 3, more than one-third have impaired myocardial tissue perfusion and an unfavourable outcome.5-10 Therefore, angiographically assessed parameters may not be the ideal risk-assessment parameters for tailoring of adjunctive pharmacological11-13 and mechanical treatment14 at the time of PCI. Other methods have been proposed for acute risk-assessment: rise of markers indicative of myocardial damage or increased wall tension,15-17 evaluation of myocardial tissue perfusion by myocardial contrast...
echocardiography or single-photon emission computerized tomography (SPECT), or traditional ST-resolution analysis ≥ 90 min following balloon inflation. The clinical value of the latter methods may be questioned, because prognostic information is not available until at a time when irreversible myocardial damage may have occurred or because the methods are not generally applicable on a 24 h basis. In clinical practice, a non-interventional and easy applicable method would be ideal for acute risk-assessment at the time of PCI. There is limited evidence concerning the potential prognostic significance of spontaneous and interventional ST-changes occurring in STEMI patients transferred for primary PCI. We hypothesized that continuous ST-monitoring before and during coronary intervention may candidiate as a valuable non-interventional method for acute risk-assessment. Accordingly, the prospective ST-MONitoring in Acute Myocardial Infarction study (The MONAMI study) was conducted with a three-fold purpose: to quantify pre-, per-, and post-interventional ST-changes in STEMI patients transferred to an interventional hospital for primary PCI, to evaluate whether a pre-specified continuous ST-monitoring classification provides potential prognostic information in the pre- and per-interventional phase, and to compare post-interventional ST-resolution parameters derived from the analysis of continuous ST-monitoring data and snapshot ECGs, respectively.

Methods

Study population

This study was performed between 1 November 2002 and 31 January 2004. The study region consisted of 250 000 inhabitants and were serviced by two local hospitals without primary PCI facilities (Randers County Hospital and Silkeborg County Hospital, Denmark). Consecutive patients living in the study region and suspected of STEMI were eligible for inclusion in the study if fulfilling the inclusion criteria: (i) a tentative diagnosis of STEMI; (ii) if no significant ST-elevation (ST-elevation in two related leads; ≥0.1 mV in leads V1-V3, aVL, and V4-V6 and ≥0.2 mV in leads V1-V3) was present from the analysis of continuous ST-monitoring data; (iii) if no PCI was performed; or (iv) if ST-monitoring data were incomplete (ST-monitoring analysis not possible).

Baseline characteristics, pre-hospital data, in-hospital data, and outcome data

The following data were registered prospectively from ambulance records and patient records: age, sex, history of hypertension, diabetes, angina pectoris (AP), previous myocardial infarction (MI), previous medical treatment, systolic and diastolic blood pressure, time of symptom onset, arrival time at the local hospital, and at the interventional hospital, respectively, acute medical treatment initiated before arrival at the interventional hospital and during coronary intervention, and infarct location according to the index ECG (anterior if significant ST-elevation in at least two of the leads I, aVL, and V1-V6 and non-anterior if significant ST-elevation in at least two of the leads II, III, aVF, and V5-V6). Time of first balloon inflation was registered prospectively at the catheterization laboratory.

Acquisition of 12-lead ECGs and sampling of continuous ST-monitoring data

A commercial monitor–defibrillator (LIFEPAK 12, Medtronic Emergency Response Systems, USA) was used for 12-lead ECG acquisition and sampling of continuous ST-monitoring data. The analogue ECG signals were digitized at a sample rate of 500 Hz for processing by the GE/Marquette Medical Systems 12SL ECG interpretive algorithm. Sampling of continuous ST-monitoring data was initiated automatically when the first 12-lead ECG was acquired at the discretion of the ambulance staff, either in the pre-hospital phase or at the local hospital (Figure 1). At 30 s interval, the ST-monitoring programme generated a median QRST-complex for all 12 leads on the basis of a 10 s epoch of ECG data. From each of these median QRST-complexes, the programme estimated the ST-deviation at the STM point, halfway between the J-point of the QRST-complex and the start of the T-wave. If a ≥ 0.1 mV change in ST-deviation lasted for ≥ 2.5 min, then the software automatically acquired and stored a 10 s 12-lead ECG waveform. Before or during transportation to the interventional hospital, ambulance staff replaced the traditional electrodes with radiolucent carbon fibre lead wire electrodes (Ambu Blue Sensor QR electrodes, Ambu A/S, Denmark), enabling ST-monitoring to be continued during PCI. ST-monitoring was terminated 90 min following first balloon inflation (Figure 1). All 12-lead ECG waveforms and continuous ST-monitoring data were transferred to a computer and stored by a random key for subsequent blinded analysis.

Figure 1 Study setup. *Plasma sampling for subsequent analysis of biochemical markers; † echocardiography; ‡ local hospital bypassed in patients transferred directly to the interventional hospital.
Analysis of continuous ST-monitoring data

Commercial software (CodeStat Suite, Medtronic Emergency Response Systems) was used for the analysis of continuous ST-monitoring data. If transient abnormal electrical conduction (bundle branch block or intraventricular block) was present in the 12-lead ECG waveforms, then associated ST-monitoring data were excluded from the analysis.

Pre-interventional ST-monitoring data, i.e. data acquired before any coronary intervention (wire, distal protection device, suction device, balloon inflation, stent or intravascular ultrasound), were analysed to detect spontaneous resolution of ST-elevation before coronary intervention (resolution of ST-elevation to a level <0.1 mV in leads I, II, III, aVF, aVL, and V4-V6 and <0.2 mV in leads V1–V3). The cumulated level of ST-elevation before primary PCI was estimated by summatng the maximal pre-interventional level of ST-elevation in each anterior leads (>0.1 mV ST-elevation required in I, aVL, and V4–V6 and ≥0.2 mV required in V1–V3) and non-anterior leads (>0.1 mV ST-elevation required in II, III, aVF, and V5–V6), respectively.

Per-interventional ST-monitoring data, i.e. data acquired during coronary intervention, were analysed for the presence of procedural increase in ST-elevation in any lead (apart form aVR). Procedural increase in ST-elevation was defined as a >0.1 mV increase in ST-elevation during coronary intervention (compared with the level of ST-elevation in the same lead immediately before coronary intervention) and lasting ≥2.5 min. This classification was determined by the ST-change required for automatic acquisition of ECGs by the LIFEPAK-12, and accordingly, any significant increase in ST-elevation was documented by both continuous ST-monitoring data and a snapshot 12-lead ECG waveform. A similar approach is implemented by other investigators evaluating the benefit of continuous ST-monitoring in the pre-hospital phase.25

Post-interventional ST-monitoring data and snapshot ECGs were evaluated to estimate the time from first balloon inflation to achievement of ≥70% ST-resolution (minutes) and achievement of ≥70% ST-resolution (yes/no) 30, 60, and 90 min following first balloon inflation.

Continuous ST-monitoring classification

Patients were stratified into three groups according to the analysis of pre- and per-interventional ST-monitoring data: (A) patients achieving spontaneous ST-resolution before PCI; (B) patients with preserved ST-elevation immediately before PCI and with no increase in ST-elevation during PCI; and (C) patients with preserved ST-elevation immediately before PCI and with increase in ST-elevation during PCI (Figure 2).

Plasma sampling and analysis of biochemical markers

An initial blood sample was drawn either on arrival at the catheterization laboratory or immediately following PCI. Additional blood samples were drawn ~6, 12, and 24 h and 2, 3, and 4 days following primary PCI and at a 3-month follow-up visit (Figure 1). Samples were centrifuged immediately at 2000 g for 10 min. Plasma was separated and stored at ~80 °C for subsequent analysis of biochemical markers. An Elecsys 1010 with reagents from the same supplier (Roche A/S, Copenhagen, Denmark) was used for the analysis of troponin-T (TnT) (reference level <0.10 μg/L) and Nt-pro-BNP level (reference level <15 pmol/L).26 The level of high-sensitive (hs) C-reactive protein was measured using a BN Prospect with reagents from the same supplier (Dade Behring, Stockholm, Sweden) (reference level <3.0 mg/L). All analyses were done at the Department of Clinical Biochemistry, Skejby Sygehus, Aarhus University Hospital, Denmark. Among samples drawn during the initial admission, peak level of the aforementioned biochemical markers was determined and implemented in further analysis. However, the peak level of creatinine kinase MB isoenzyme (CKMB) (normal level <10.0 μg/L) was determined from the routine blood samples taken during admission.

Echocardiography

A three-dimensional echocardiography examination was performed within 2 h of the first balloon inflation and after 3 months (Figure 1). A total of six scan plans of the left ventricle were achieved by apical rotation of the transducer with a hand-held rotation device triggered by the R-wave of the electrocardiogram (GE Vingmed, Ultrasound, Horton, Norway). Data were digitized and stored by a random key for subsequent blinded analysis. Left ventricular ejection fraction (LVEF) was evaluated on the basis of the digitized two-dimensional images with the use of commercially available software programme for three-dimensional volume reconstruction (Echopac-3D, GE Vingmed Ultrasound).27,28

Quantitative analysis of coronary angiographies

Experienced primary PCI operators (L.T., J.F.L., and H.R.A.) assessed the coronary angiographies blinded to patient data and classified as follows: number of diseased vessels, TIMI flow grade in the infarct-related artery (IRA) at first contrast injection,29 spontaneous visible coronary collaterals (Rentrop grade 1, 2, or 3),30 maximal thrombus grade in the IRA (<5 or ≥5 mm in diameter), plaque burden (lesion <5 or ≥5 mm in length), lesion calcification (yes/no), procedural TIMI flow grade reduction (yes/no), and sign of distal embolization (yes/no). From the final contrast injection in the IRA, the following parameters were evaluated: TIMI flow grade, corrected TIMI Frame Count (CTFC) at 30 frames/s,27 and myocardial blush grade according to the classification by the Zwolle Myocardial Infarction Study Group.32

Statistics

With β = 0.20 and α = 0.05, it was estimated that 48 patients (3n) were needed in the study to detect a factor 2 difference between two groups in the level of biochemical markers, provided that the standard deviation for biochemical markers was approximately two-thirds of the mean level of biochemical markers. Similarly, it was estimated that 93 patients (3n) were needed to detect a 0.05 difference in LVEF, if the standard deviation of LVEF was 0.07. Accordingly, the study was planned for inclusion of 100–125 patients. Dichotomous data are presented as number (%) or number/valid cases (%). Continuous variables are presented as medians (25–75th percentiles). Fisher’s exact test, Mann-Whitney test, and Kruskall-Wallis test were used as appropriate for comparison of the groups. The statistical significance level was P ≤ 0.05 (two-sided test). The software package SPSS 10.0 was used for statistical analyses.

Ethical considerations

The local Ethics Committee approved the study. Blood sampling and echocardiography examinations required written informed consent from the patient.

Results

During the study period, 199 patients were transferred from the study region to the interventional hospital for primary PCI because of suspected STEMI and 139 were transported in ambulances with equipment for acquisition of continuous ST-monitoring data. ST-monitoring was initiated in 122 patients who were initially enrolled in the present study. However, a diagnosis of STEMI was not confirmed in 13 patients: in one patient no significant ST-elevation was detectable from the analysis of continuous ST-monitoring data, in seven patients PCI was not performed, and in nine...
Patients per-interventional ST-monitoring data were incomplete. The remaining 92 patients were stratified according to the proposed ST-monitoring classification into group A ($n = 22$), group B ($n = 43$), and group C ($n = 27$). No patients in group A presented increase in ST-elevation during coronary intervention. All patients had aspirin treatment initiated in the ambulance or at the local hospital. Clopidogrel and heparin treatment was initiated at the local hospital ($n = 85$) or on arrival at the catheterization laboratory (seven group B patients transferred directly to the interventional hospital). Glycoprotein IIb/IIIa inhibitor treatment was initiated during coronary intervention in 10 (46%), 27 (63%), and 23 (85%) patients in groups A, B, and C ($P = 0.014$). Stents were routinely used in all patients. Group C patients presented the most pronounced cumulated ST-elevation before PCI, whereas no differences were found between groups in other baseline characteristics including the duration of ST-monitoring before coronary intervention (Table 1). In group C, the median increase in ST-elevation during coronary intervention was 0.3 (0.1–0.5) mV in the lead with the maximum pre-interventional ST-elevation and lasted 7 (5–22) min. TIMI flow grade 3 in the IRA at first contrast injection was more frequently observed in group A (Table 2). Group A patients also presented the lowest CTFC at the end of the procedure, whereas no differences were observed between groups concerning other angiographic parameters (Table 2). When only comparing groups B and C, no differences were found in any of the angiographically assessed parameters. Groups A, B, and C differed in peak level of TnT (1.4, 4.7, and 7.2 μg/L, $P < 0.001$), CKMB (35, 150, and 325 μg/L, $P < 0.001$), and Nt-pro-BNP (183, 175, and 269 pmol/L, $P = 0.084$) as estimated from samples drawn during admission and in LVEF evaluated acutely (0.53, 0.48, and 0.45, $P = 0.047$).

Figure 2  ST-monitoring data and ECG complexes acquired before, during, and following coronary intervention. Group A, patients achieving spontaneous ST-resolution before coronary intervention; group B, patients with preserved ST-elevation immediately before coronary intervention and with no increase in ST-elevation during coronary intervention; and group C, patients with preserved ST-elevation immediately before coronary intervention and with increase in ST-elevation during coronary intervention. Increase in ST-elevation: ≥0.1 mV increase in ST-elevation and of ≥2.5 min duration during coronary intervention (compared with pre-interventional level of ST-elevation).
(Table 3). Groups B and C also differed in time from first balloon inflation to ≥70% ST-resolution (14 vs. 42 min, \( P < 0.002 \)) and in the proportion achieving ≥70% ST-resolution 30 min following balloon inflation (61 vs. 19%, \( P = 0.001 \) (Table 3). However, no differences were found in the proportion achieving ≥70% ST-resolution 60 and 90 min following first balloon inflation (Table 3). In 87 patients, 3-month follow-up data were available (four patients died prior to follow-up and one patient did not wish to participate), and significant differences were observed in Nt-pro-BNP (26, 47, and 74 pmol/L, \( P = 0.008 \)) and LVEF (0.58, 0.54, and 0.45, \( P < 0.001 \) (Table 3).

### Discussion

The present study is the first study combining pre-hospital and in-hospital continuous ST-monitoring to achieve a comprehensive evaluation of ST-changes occurring in the pre-, per-, and post-interventional phase. The analysis of pre-interventional ST-monitoring data showed that one-fourth of STEMI patients transferred to an interventional hospital for primary PCI achieved spontaneous ST-resolution before coronary intervention. This number is considerably higher than expected, despite the use of very conservative ECG criteria to accept a diagnosis of spontaneous ST-resolution (absolute resolution of ST-elevation required in the present study compared with ≥50% relative resolution of ST-elevation required in a previous study).

All patients were treated with aspirin and most patients with heparin and clopidogrel before transfer to the interventional hospital. It may be speculated whether the latter medications contributed to the high incidence of spontaneous ST-resolution observed. This is partly supported by previous findings, and patients achieving spontaneous ST-resolution may be considered ‘pharmacological responders’. Spontaneous ST-resolution was associated with less myocardial damage as indicated by a low level of the biochemical markers TnT and CKMB and delayed resolution of ST-elevation, thus supporting that these patients are low-risk patients.

An appropriate future strategy in patients achieving spontaneous ST-resolution during transfer to an interventional hospital may be acute PCI followed by prompt referral back to the local hospitals. The safety of such ‘drive-in primary PCI’ needs further delineation.

From the analysis of per-interventional ST-monitoring data, the present study indicates that continuous ST-monitoring may provide important prognostic information also among patients having preserved ST-elevation immediately before coronary intervention. An increase in ST-elevation during coronary intervention was found to be associated with a higher level of the biochemical markers TnT, CKMB, and Nt-pro-BNP and delayed resolution of ST-elevation, hence indicating a greater extent of myocardial damage and a compromised microvascular perfusion. Furthermore, no improvement in LVEF was observed in group C patients during follow-up, indicative of irreversible myocardial damage in the latter patients. In previous studies, the analysis of ST-changes in relation to PCI has been based on the acquisition of snapshot ECGs acquired at various fixed intervals following balloon inflation.
Any increase in ST-elevation at the time of PCI may, therefore, have been underestimated or even missed. No previous studies have implemented continuous ST-monitoring for the evaluation of per-interventional ST-changes. Moreover, previous studies have been small in sample size and the cohorts highly selected, i.e., primarily including patients with anterior MI, presenting with TIMI flow 0 or 1, and achieving TIMI flow 3 during coronary intervention.37,38,40 Thus, the present study implementing continuous ST-monitoring and consisting of a rather unselected cohort of STEMI patients documents that increase in ST-elevation during coronary intervention is common even in the general population of STEMI patients. The ST-changes observed are considered indicative of damaged microvascular integrity, the cause of which may be multifactorial.41,42 From the analysis of single ECGs, Takana et al.8 have reported that increase in ST-elevation was observed immediately after angioplasty in most of no-reflow cases and considered this phenomenon to be caused by intervention-induced distal embolization of lipid pool-like plaque content rather than reperfusion.

### Table 2: Coronary angiography data according to study group

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 22)</th>
<th>Group B (n = 43)</th>
<th>Group C (n = 27)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of diseased vessels</td>
<td>2 (1–3)</td>
<td>1 (1–3)</td>
<td>2 (1–3)</td>
<td>0.93</td>
</tr>
<tr>
<td>IRA findings at first contrast injection</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIMI flow grade 3</td>
<td>13/22 (59%)</td>
<td>6/43 (14%)</td>
<td>2/27 (7%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Instantly visible collateralsa</td>
<td>7/21 (33%)</td>
<td>18/43 (42%)</td>
<td>14/26 (54%)</td>
<td>0.36</td>
</tr>
<tr>
<td>IRA findings during the procedure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombus length ≥5 mm</td>
<td>10/22 (46%)</td>
<td>24/43 (56%)</td>
<td>15/27 (56%)</td>
<td>0.70</td>
</tr>
<tr>
<td>Plaque length ≥5 mm</td>
<td>16/22 (73%)</td>
<td>38/43 (88%)</td>
<td>23/27 (85%)</td>
<td>0.26</td>
</tr>
<tr>
<td>Plaque calcification</td>
<td>10/22 (46%)</td>
<td>14/43 (33%)</td>
<td>9/27 (33%)</td>
<td>0.56</td>
</tr>
<tr>
<td>TIMI flow reduction during coronary intervention</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distal embolization</td>
<td>1/22 (5%)</td>
<td>6/43 (14%)</td>
<td>4/25 (16%)</td>
<td>0.44</td>
</tr>
<tr>
<td>IRA findings according to final contrast injection</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIMI flow grade 3</td>
<td>21/22 (96%)</td>
<td>31/43 (72%)</td>
<td>21/27 (78%)</td>
<td>0.09</td>
</tr>
<tr>
<td>Corrected TIMI frame count</td>
<td>21 (19–28)</td>
<td>29 (24–43)</td>
<td>37 (22–52)</td>
<td>0.007</td>
</tr>
<tr>
<td>Myocardial blush grade 3</td>
<td>15/20 (75%)</td>
<td>24/39 (62%)</td>
<td>12/24 (50%)</td>
<td>0.24</td>
</tr>
</tbody>
</table>

Dichotomous data presented as number/valid cases (%) and continuous data as median values (25–75th percentiles).

aRentrop grade 1, 2, or 3 collaterals from contralateral or ipsilateral vessels towards IRA.

### Table 3: Paraclinical data according to study group

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 22)</th>
<th>Group B (n = 43)</th>
<th>Group C (n = 27)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biomarkers ≤4 days of primary PCI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak troponin-T (µg/L)</td>
<td>1.4 (0.4–2.8)</td>
<td>4.7 (3.2–9.6)</td>
<td>7.2 (4.5–13.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak CKMB (µg/L)</td>
<td>35 (18–100)</td>
<td>150 (62–303)</td>
<td>325 (156–424)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak NT-pro-BNP (pmol/L)</td>
<td>183 (119–296)</td>
<td>175 (109–331)</td>
<td>269 (189–640)</td>
<td>0.084</td>
</tr>
<tr>
<td>Peak hs C-reactive protein (mg/L)</td>
<td>20 (16–42)</td>
<td>30 (11–94)</td>
<td>35 (19–81)</td>
<td>0.28</td>
</tr>
<tr>
<td>NT-pro-BNP (pmol/L) at 3 months</td>
<td>26 (15–94)</td>
<td>47 (25–96)</td>
<td>74 (33–230)</td>
<td>0.008</td>
</tr>
<tr>
<td>Three-dimensional echo</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF ≤2 h after first balloon inflation</td>
<td>0.53 (0.50–0.57)</td>
<td>0.48 (0.41–0.55)</td>
<td>0.45 (0.37–0.53)</td>
<td>0.047</td>
</tr>
<tr>
<td>LVEF at 3-month follow-up visit</td>
<td>0.58 (0.54–0.63)</td>
<td>0.54 (0.46–0.57)</td>
<td>0.45 (0.38–0.53)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Continuous ST-monitoring analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time to ≥70% ST-resolution (min)</td>
<td>—</td>
<td>14 (4–37)</td>
<td>42 (25–79)</td>
<td>0.002</td>
</tr>
<tr>
<td>ECG analysis at fixed intervals</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥70% ST-resolution 30 min after primary PCI</td>
<td>—</td>
<td>26 (61%)</td>
<td>5 (19%)</td>
<td>0.001</td>
</tr>
<tr>
<td>≥70% ST-resolution 60 min after primary PCI</td>
<td>—</td>
<td>31 (72%)</td>
<td>14 (62%)</td>
<td>0.12</td>
</tr>
<tr>
<td>≥70% ST-resolution 90 min after primary PCI</td>
<td>—</td>
<td>35 (81%)</td>
<td>17 (63%)</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Dichotomous data are presented as number (%) and continuous data as median values (25–75th percentiles).
injury. The present study challenges this ‘distal embolization theory’ as the major reason for an increase in ST-elevation during PCI, because the incidence of procedural TIMI flow reduction or angiographically detectable distal embolization was comparable between groups, as also supported by previous findings. Absence of collaterals has also been considered to contribute to the increase in ST-elevation during PCI. The latter explanation is also questioned by the present and previous studies because of the comparable angiographic findings observed in patients with and without per- and post-interventional increase in ST-elevation. Further studies are needed not only to clarify why some patients suffer from impaired myocardial tissue perfusion following PCI despite successful restoration of epicardial coronary blood flow, but also to evaluate if the proposed ST-monitoring classification yields independent prognostic information in the acute phase, and to evaluate whether myocardial tissue perfusion can be improved by adjunctive pharmacological or mechanical treatment at the time of PCI.

Finally, the analysis of post-interventional ST-monitoring data indicates that the value of a traditional 90 min ST-resolution parameter may be questionable in patients treated with primary PCI. Thus, no differences were observed between groups B and C in the latter parameter, despite the fact that significant differences were observed in time to the achievement of complete ST-resolution (derived from the analysis of continuous ST-monitoring data), in 30 min ST-resolution parameters (derived from single ECGs), and in the majority of surrogate parameters of mortality assessed. These findings are partly supported by a previous study, showing that patients with normalized vs. impaired myocardial tissue perfusion following PCI differ in 30 min ST-resolution parameters but not in 60 or 90 min ST-resolution parameters. This calls for a reconsideration of the prognostic value of ST-resolution analysis in STEMI patients treated with primary PCI. The 90 min ST-resolution parameter may be a relic from the fibrinolytic theory as the major reason for an increase in ST-elevation during coronary intervention. Therefore, it may be more appropriate in future studies to implement a four-group ST-monitoring classification, even though no group A patients presented increase in ST-elevation during coronary intervention in the present study. Finally, Bonferroni corrections should have been considered because of the experiment-wise type I error, and a reduction in α-level to 0.017 (0.05/3) should have been considered because three groups were compared. As the latter corrections were not implemented, one in seven comparisons might be significant by chance.

Conclusions

STEMI patients transferred for primary PCI are heterogeneous with respect to pre- and per-interventional ST-changes, and a pre-specified ST-monitoring classification seems useful for stratification of patients at time of PCI into groups with low, intermediate, and high risk profile. Furthermore, post-interventional ST-monitoring indicates that traditional 90 min ST-resolution analysis may have limited value in the era of primary PCI.

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References


Three-dimensional visualization of severe pericardial calcification in constrictive pericarditis using multidetector-row computed tomography

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A 73-year-old male patient was admitted due to progressive dyspnoea on exertion during the last 6 months. Chest pain or syncope was not reported.

On admission, blood pressure was 100/60 mmHg. Heart sounds were low without pericardial friction rub. Pulmonary rales were absent, but there were pronounced pre-tibial oedema. The patient had jugular venous distention. The ECG showed low voltage and atrial fibrillation with a heart rate of about 130 b.p.m. Bicycle stress ECG showed reduced exercise tolerance (terminated at the change from 50 to 75 W due to dyspnoea). Echocardiography showed preserved left ventricular function (ejection fraction 65%), both atria were dilatated in the longitudinal axis and echogenic pericardial structures were seen. Transmitral flow pattern showed exaggerated respiratory variation in inflow velocities (>-25%), indicating constriction (Panel A).

Chest radiograph showed no signs of pulmonary oedema but pericardial calcifications. Because of suspected constrictive pericarditis, multislice computed tomography (CT) of the heart was performed (16 × 0.75 mm collimation, 370 ms rotation, 6 mm table feed, 120 kV, 150 mA). Transaxial images as well as three-dimensional reconstructions were rendered. Extensive pericardial calcifications were documented (Panels B, C, and D; movie: see Supplementary material available at European Heart Journal online) leading to the diagnosis of calcified constrictive pericarditis.

The use of CT images for visualization of pericardial calcium in constrictive pericarditis has been previously reported. However, it had so far been limited to two-dimensional imaging.

Sixteen-slice CT permits acquisition of a cardiac high-resolution data set in ~10 s with a temporal resolution of 185 ms. It thus provides for detailed, high resolution three-dimensional visualization of pericardial calcifications that may facilitate surgical therapy.

Panel A. Echocardiographic transmitral flow pattern with exaggerated respiratory variation in inflow velocities (-25%).

Panel B. Transaxial CT image (slice thickness 3.0 mm) of the heart at mid-ventricular level, demonstrating severe calcification of the pericardium (arrows). In addition, bilateral pleural effusion is seen. AO, descending aorta; PE, pleural effusion.

Panel C. Three-dimensional volume rendering technique reconstruction of the whole chest. Nearly circumferential pericardial calcification is seen (structures of high CT density, such as bone and calcification, are rendered in white colour).

Panel D. Three-dimensional volume rendering technique reconstruction of the heart. To exclude the non-calcified part of the heart, a threshold of 130 Hounsfield units (common threshold for coronary calcification in CT imaging) was chosen.