JACC: CARDIOVASCULAR INTERVENTIONS VOL. 2, NO. 2, 2009

ISSN 1936-8798/09/$36.00
PUBLISHED BY ELSEVIER INC. DOI: 10.1016/j.jcin.2008.07.010

Intracoronary Electrocardiogram Recording With a Bare-Wire System

Perioperative ST-Segment Elevation in the Intracoronary Electrocardiogram Is Associated With Myocardial Injury After Elective Coronary Stent Implantation

Tadayuki Uetani, MD, PHD,* Tetsuya Amano, MD, PHD,* Soichiro Kumagai, MD,* Hirohiko Ando, MD,* Kiminobu Yokoi, MD,* Tomohiro Yoshida, MD,* Bunichi Kato, MD, PHD,* Masataka Kato, MD,* Nobuyuki Marui, MD, PHD,* Michio Nanki, MD, PHD,* Tatsuaki Matsubara MD, PHD,† Hideki Ishii, MD, PHD,‡ Hideo Izawa, MD, PHD,‡ Toyoaki Murohara, MD, PHD‡

Nagoya, Japan

Objectives With an intracoronary electrocardiogram (IcECG) recording with insulated polymer-coated guidewire without balloon catheter, we sought to examine the association between ST-segment elevation in the IcECG after elective stenting and myocardial injury.

Background An IcECG is a sensitive method to detect local myocardial ischemia. Occasionally, persistent ST-segment elevation in the IcECG was recorded after successful coronary intervention. Conventionally IcECG was recorded with a guidewire and over-the-wire system.

Methods Patients who underwent elective stenting were enrolled (n = 339). The IcECG both at baseline and after procedure were obtained with a guidewire with an insulating coated shaft suitable for IcECG recording. The presence of chest pain after percutaneous coronary intervention was recorded. Cardiac biomarkers were examined 18 h after the procedure.

Results The ST-segment elevation in the IcECG after procedure was recorded in 65 patients, and no change was recorded in 274 patients. Troponin-T, creatine phosphokinase, and creatine kinase MB isoform after the procedure were significantly higher in patients with post-procedural ST-segment elevation in the IcECG than patients without ST-segment elevation. Multivariate analysis demonstrated that ST-segment elevation in the IcECG is an independent predictor of post-procedural myocardial injury. The incidence of ST-segment elevation in the IcECG was significantly higher in patients with post-procedural chest pain than patients without chest pain ($p < 0.001$).

Conclusions We demonstrated a facile method to record IcECG with a guidewire with a polymer-coated shaft. The IcECG is a useful method for predicting post-procedural myocardial injuries. (J Am Coll Cardiol Intv 2009;2:127–35) © 2009 by the American College of Cardiology Foundation

From the *Department of Cardiology, Chubu Rosai Hospital, Nagoya, Japan; †Department of Internal Medicine, Aichi-Gakuin School of Dentistry, Nagoya, Japan; and the ‡Department of Cardiology, Nagoya University Graduate School of Medicine, Nagoya, Japan.

Manuscript received April 15, 2008; revised manuscript received July 16, 2008, accepted July 30, 2008.

Post-procedural myocardial injury after apparent uncomplicated percutaneous coronary intervention (PCI) is not uncommon (1–3). Previously subclinical creatine kinase-MB (CK-MB) elevation in 5% to 30% of patients after successful PCI was reported. Some biochemical markers are more sensitive and specific in detecting myocardial damages.

Formerly, modest elevations of cardiac enzymes after successful PCI were considered relatively benign, and clinical implications of this phenomenon were not fully investigated. Recently, some trials demonstrated that elevated cardiac biomarkers after apparently successful PCI were associated with increased risks of adverse cardiac events (4–6).

Intracoronary electrocardiogram (IcECG) with a guidewire as a unipolar electrode represents local epicardial ECG. Unipolar IcECG seemed to be more sensitive than surface ECG for detecting local ischemia during coronary interventions (7,8). Some studies demonstrated IcECG can be used to assess myocardial viability in stable angina and acute myocardial infarctions (9,10).

Previously, an over-the-wire or monorail catheter had to be crossed at the target lesion to provide insulation of the guidewire in the part proximal to the stenosis during IcECG recording (9). However, in a case of severe stenosis or coronary occlusion, severe ischemic changes of IcECG might occur if a balloon catheter is wedged into the stenosis. Therefore, we recorded the IcECG with a guidewire with an insulated polymer cover to overcome these disadvantages caused by usage of the over-the-wire catheters.

Abbreviations and Acronyms

CK-MB = creatine kinase-MB
IcECG = intracoronary electrocardiogram
PCI = percutaneous coronary intervention
TIMI = Thrombolysis In Myocardial Infarction
TnT = troponin-T

In most cases, ST-segment elevation appeared during balloon inflation and resolved promptly after deflation of the balloons. Previous studies have reported that persistent ST-segment elevation after the balloon deflation was observed in some cases (9). In our own experience, persistent ST-segment elevation in the IcECG compared with the baseline IcECG was recorded occasionally after successful PCI without surface ECG abnormality. Thus, we postulate persistent ST-segment elevation in the IcECG reflected local myocardial injury after the PCI. Therefore, we designed this study to evaluate the relation between post-procedural ST-segment elevation in the IcECG and the elevation of biochemical markers of myocardial injury after elective PCI compared with surface ECG.

Methods

Study population. From September 2004 to December 2006, 339 consecutive patients who underwent apparently successful elective coronary stent implantations in Chubu Rosai Hospital, Nagoya, Japan, were enrolled in this study, which was approved by the ethics committee of the hospital. All had angina, documented myocardial ischemia, or both and signed informed consent for the study. The exclusion criteria of this study were patients with: 1) emergency coronary angioplasty within 24 h of onset; 2) elevated pre-procedural cardiac biomarker; 3) active congestive heart failure; 4) severe lesion characteristics not suitable for soft-tip guidewire; 5) angioplasty with debulking device (directional coronary atherectomy or rotational atherectomy); 6) Thrombolysis In Myocardial Infarction (TIMI) flow grade 1 to 2 of target vessel at the end of procedure; and 7) multivessel stenting in a single procedure. This study was approved by the ethics committee of our hospital.

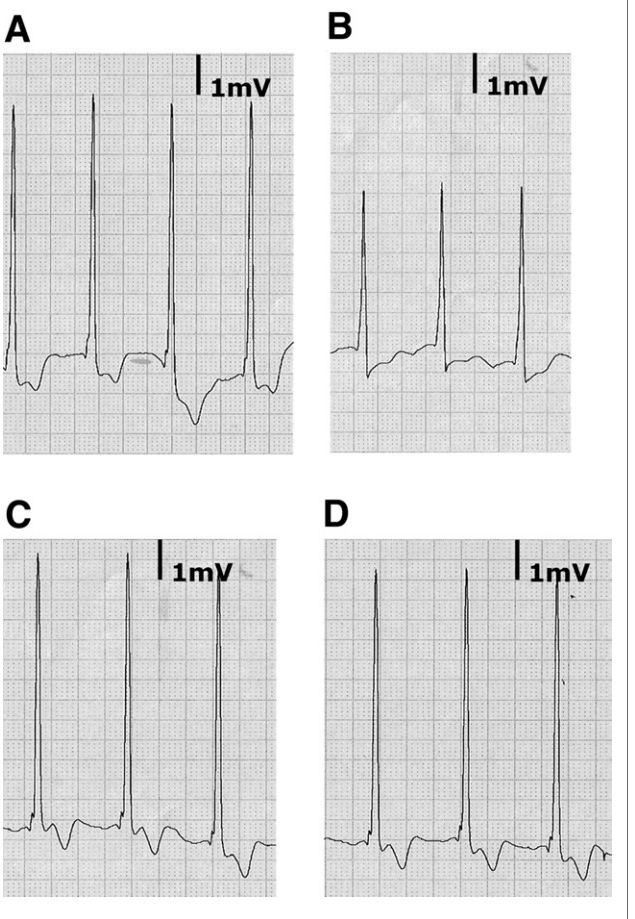

Figure 1. IcECG

Representative intracoronary electrocardiogram (IcECG) waves of the left anterior descending artery with a conventional uninsulated guidewire with over-the-wire system **(A)** and without an over-the-wire systems **(B)**, with a polymer-covered guidewire with an over-the-wire system **(C)** and without over-the-wire systems **(D)**. All IcECGs were recorded with a paper speed of 50 mm/s and a calibration of 10 mm = 1 mV.

Study protocol. The study protocol was as follows. First, we recorded the baseline surface ECG and IcECG after positioning the guidewire in the distal part of a target vessel. Then coronary stent implantation was performed after the pre-procedural coronary angiography and intravascular ultrasound (IVUS) recording. We also recorded post-procedural coronary angiography and IVUS. After confirmation of an uncomplicated PCI, we recorded the final surface ECG and IcECG. Finally, we obtained blood samples at approximately 18 h after the PCI to evaluate cardiac biomarkers.

Angiography and IVUS were evaluated by an independent investigator not involved in the procedures who is unaware of the final outcomes. A computerized quantitative analysis system (QCA-CMS system version 6.0.39.0, MEDIS, Leiden, the Netherlands) was employed with the guiding catheter for calibration. The IVUS studies were performed with a mechanical sector scanner (Atlantis SR Pro, Boston Scientific Corp., Natick, Massachusetts) and motorized transducer pullback system (0.5 mm/s).

PCI procedure. All patients underwent elective coronary stent implantation with or without balloon pre-dilation. All patients received antiplatelet agents, for at least 24 h before the procedure. Furthermore, 10,000 IU of heparin was administered before the procedure, and an additional bolus of 1,000 to 2,000 IU was given every hour if the procedure lasted for more than 1 h. Patients without contraindication received ticlopidine (200 mg b.i.d.). No patient received glycoprotein IIb/IIIa receptor inhibitor, which is not approved in Japan. All procedures were performed with 6- to 8-F guiding catheters by either a transradial or transfemoral approach. Successful PCI defined as <50% residual stenosis with final TIMI flow grade 3 was performed in all the enrolled patients. Staff nurses in the catheterization laboratory who were independent of this study routinely recorded the presence or absence of chest pain after the PCI in all patients. Post-procedural chest pain was defined as varying degrees of typical or atypical chest pain at the end of PCI procedures.

IcECG recording. A 0.014-inch diameter guidewire (Hi-Torque Balance Middle Weight-Universal, Abbott Vascu-

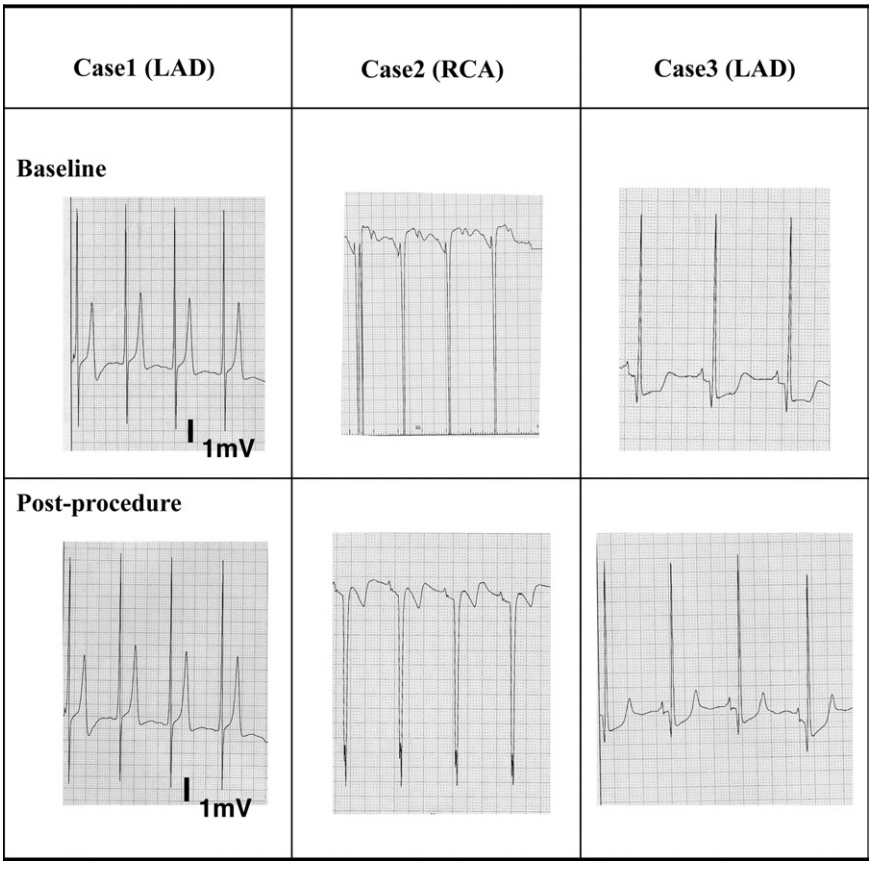

Figure 2. Representative IcECGs of Patient Without ST-Segment Elevation

There is no ST-segment change between baseline and post-procedural waves. All intracoronary electrocardiograms (IcECGs) were recorded with a paper speed of 50 mm/s and a calibration of 10 mm = 1 mV. LAD = left anterior descending coronary artery; RCA = right coronary artery.

lar; Santa Clara, California) was used. The proximal shaft of this guidewire was coated with an insulated polymer cover and a distal uninsulated component was used as a unipolar electrode. The uninsulated tip of the guidewire was placed at the distal epicardial position of the target lesion. The uninsulated proximal end was connected to the chest lead terminal (V_1 lead) of RMC-3100 (Nihon Kohden, Tokyo, Japan). During IcECG recording, limb leads and chest leads (V_2 to V_6) of surface ECG were recorded simultaneously.

Intracoronary and surface ECG were calibrated (10 mm = 1 mV) and recorded simultaneously. Baseline IcECG was recorded before usage of IVUS catheters, balloon catheters, and coronary stents crossing of target lesion. At the end of PCI, the guidewire was placed in the same position as the baseline IcECG, and the final IcECG was recorded.

The IcECG waveforms of the left anterior descending coronary artery with conventional uninsulated guidewire with and without over-the-wire catheter (bare wire) are illustrated in Figures 1A and 1B. These waveforms changed, depending on the tip position of the balloon catheter. On the contrary, the waveform of IcECG with the guidewire with a polymer cover was not influenced by use or disuse of over-the-wire catheter or by tip position of the balloon catheter (Figs. 1C and 1D). No adjunctive procedure or intracoronary drug administration was done after recording the final IcECG.

Evaluation of cardiac biomarkers. Blood was sampled 18 h after the procedure. Serum troponin-T (TnT) was measured with an enzyme immunoassay kit (Roche Diagnostics, Tokyo, Japan). The detection limit of this TnT assay system is 0.03 ng/ml, and linearity is achieved from 0.1 to 2 ng/ml. A TnT level lower than 0.03 ng/ml was considered as 0 ng/ml and 0.03 to approximately 0.1 ng/ml was considered as 0.1 ng/ml. A TnT level higher than 2 ng/ml is considered as 2 ng/ml. We defined a post-procedural TnT level higher than 0.1 ng/ml, which was manufacturers' clinical cutoff value, as a post-procedural myocardial injury. The CK-MB activity was measured with an immunoinhibition assay kit (Sysmex, Kobe, Japan). Creatine phosphokinase level was assessed with the UV-rate method (Daiichi Pure Chemicals, Tokyo, Japan).

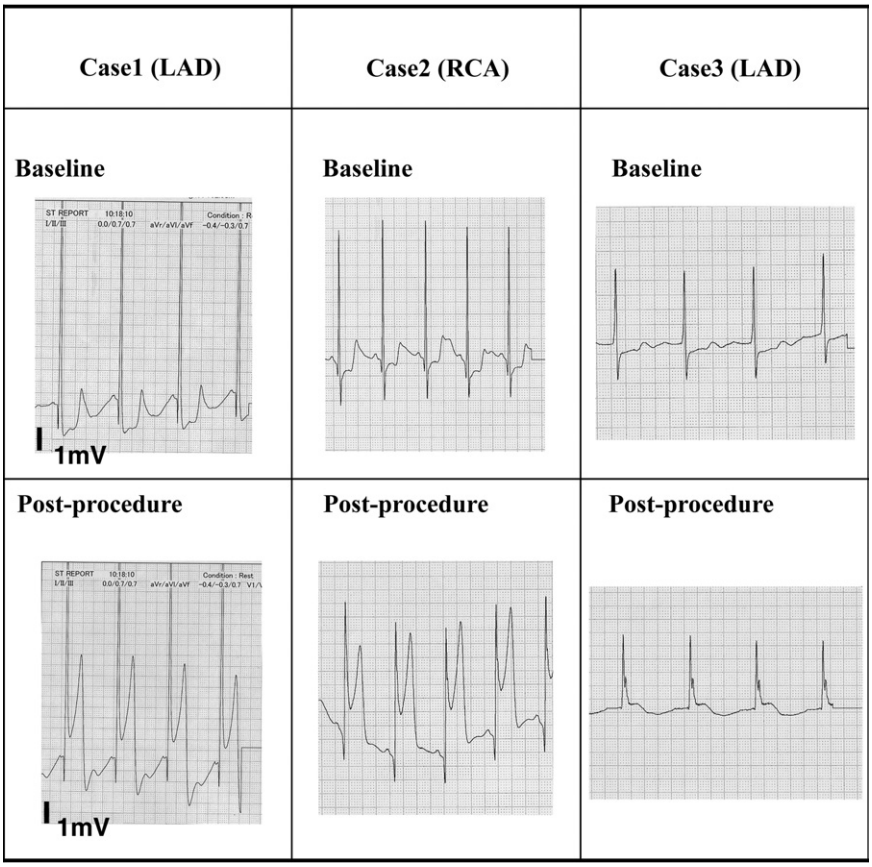

Figure 3. Representative IcECGs of Patient With ST-Segment Elevation

Post-procedural IcECG shows ST-segment elevation compared with baseline electrocardiogram (ECG). All IcECG were recorded with a paper speed of 50 mm/s and a calibration of 10 mm = 1 mV. Abbreviations as in Figure 2.

Table 1. Clinical Characteristics of Patients With ST-Segment Elevation in the IcECG and Without ST-Segment Elevation

	ST-Segment Elevation (−)	ST-Segment Elevation (+)	p Value
Number of patients	274	65	
Male	182 (66.4%)	43 (66.2%)	>0.99
Age, yrs	69.6 ± 8.5	69.9 ± 9.2	0.83
Diabetes mellitus	155 (56.9%)	38 (58.5%)	0.89
Hypertension	202 (73.7%)	51 (78.5%)	0.53
Hyperlipidemia	186 (67.9%)	49 (75.4%)	0.30
Obesity	98 (35.8%)	17 (26.2%)	0.18
Smoking	75 (27.4%)	17 (26.2%)	0.97
Previous infarction	79 (28.8%)	12 (18.5%)	0.12
Hemodialysis	5 (1.8%)	3 (4.6%)	0.38
History of ACS	73 (26.6%)	20 (30.8%)	0.61
LVEF <50%	30 (10.9%)	6 (9.2%)	0.86
Aspirin	249 (90.9%)	59 (90.8%)	>0.99
Calcium antagonists	105 (38.3%)	27 (41.5%)	0.74
Beta-blocker drugs	113 (41.2%)	27 (41.5%)	>0.99
Nitrates	110 (40.1%)	26 (40.0%)	>0.99
Statins	138 (50.4%)	28 (43.1%)	0.36
ARB	94 (34.3%)	21 (32.3%)	0.87
ACEi	44 (16.1%)	10 (15.4%)	>0.99
Insulin	39 (14.2%)	14 (21.5%)	0.20
Systolic blood pressure (mm Hg)	138.0 ± 28.8	140.8 ± 29.4	0.56
GFR (ml/min/1.73 m^2 BSA)	72.4 ± 24.1	65.6 ± 22.2	0.04
Total cholesterol (mg/dl)	193.0 ± 37.8	201.9 ± 44.0	0.33
HDL cholesterol (mg/dl)	45.1 ± 15.8	45.8 ± 17.3	0.44
Triglyceride (mg/dl)	161.8 ± 110.4	167.5 ± 82.3	0.66
LDL cholesterol (mg/dl)	107.4 ± 29.4	119.5 ± 28.8	0.29
HbA1c (%)	6.5 ± 1.5	6.5 ± 1.3	0.57

Values are mean ± SD or n (%) of patients.

ACEi = angiotensin converting enzyme inhibitor; ACS = acute coronary syndrome; ARB = angiotensin-II receptor blocker; BSA = body surface area; GFR = glomerular filtration rate; HbA1c = hemoglobin A1c; HDL = high-density lipoprotein; IcECG = intracoronary electrocardiogram; LDL = low-density lipoprotein; LVEF = left ventricular ejection fraction.

Statistics. All data are indicated in mean ± SD values. Statistical analysis was conducted with Stat-View 5.0 (SAS Institute, Cary, North Carolina). A comparison of continuous variables was achieved with the unpaired Student *t* test or a Mann-Whitney *U* test. Chi-square analysis or the Fisher exact probability test was used for group comparison of categorical variables. Univariate and multivariate logistic regression analysis were constructed to evaluate the predictor of post-procedural positive TnT. Multivariate logistic regression analysis was conducted to assess the clinical, lesion, and procedural factors associated with post-procedural TnT. Variables with a significance level of <0.2 in the univariate analysis and possible confounding factors (age, coronary risk factor, history of myocardial infarction, and de novo lesion) were considered to be candidate variables for inclusion in the multivariable analysis. Differences were considered significant at $p < 0.05$.

Results

Post-procedural ST-segment elevation in the IcECG. Post-procedural ST-segment elevation in the IcECG was recorded in 65 (19.2%) patients. In 59 (91%) of these patients, no surface ECG change was observed. In 6 patients, ST-segment elevation was recorded in surface and IcECG. Representative IcECG of patients with ST-segment elevation and without ST-segment elevation were shown in Figures 2 and 3.

Clinical, lesion, and procedural characteristics. Demographic data of the study population are presented in Table 1. No statistically significant differences existed between the 2 groups, except that the glomerular filtration rate level of patients with ST-segment elevation in the IcECG was lower than in patients without ST-segment elevation.

Detailed lesion and procedural characteristics are presented in Table 2. Lesion lengths of patients with ST-segment elevation in the IcECG were significantly longer than patients without changes.

Post-procedural cardiac biomarkers. Cardiac biomarkers of each group were demonstrated in Figure 4. Post-procedural TnT, CK-MB, and creatine phosphokinase were significantly higher in patients with ST-segment elevation in the IcECG (0.34 ± 0.47 vs. 0.06 ± 0.09, $p < 0.001$, 27.7 ±

Table 2. Lesion and Procedural Characteristics of Patients With ST-Segment Elevation in the IcECG and Without ST-Segment Elevation

	ST-Segment Elevation (−)	ST-Segment Elevation (+)	p Value
Number of lesions	274	65	
Target: LAD	126 (46.0%)	27 (41.5%)	0.39
Diagonal branch	2 (0.8%)	1 (1.7%)	0.46
Target: RCA	104 (38.0%)	25 (38.5%)	0.61
Target: LCX	41 (15.0%)	13 (20.0%)	0.57
Restenotic	39 (14.2%)	8 (12.3%)	0.84
Type B2/C	98 (35.8%)	31 (47.7%)	0.09
QCA: RD (mm)	2.53 ± 0.68	2.39 ± 0.65	0.29
QCA: MLD (mm)	1.36 ± 0.31	0.81 ± 0.42	0.28
QCA: %DS (%)	58.5 ± 22.3	63.7 ± 18.3	0.14
IVUS: lesion length (mm)	17.0 ± 9.5	21.4 ± 12.4	0.02
IVUS : lesion EEM (mm^2)	9.77 ± 4.36	9.73 ± 3.71	0.94
Direct stenting	57 (20.8%)	15 (23.1%)	0.81
Number of stents (n)	1.34 ± 0.66	1.48 ± 0.69	0.13
Max ballooning pressure (atm)	14.6 ± 7.0	14.3 ± 7.8	0.79
Total ballooning time (s)	143.3 ± 103.3	161.9 ± 114.0	0.21
Stent length (mm)	23.6 ± 13.0	27.0 ± 14.0	0.06

Values are mean ± SD or n (%) of patients.

%DS = percent diameter stenosis; EEM = external elastic membrane-cross-sectional area; IcECG = intracoronary electrocardiogram; IVUS = intravascular ultrasound; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; MLD = minimum lumen diameter; Max = maximum; QCA = quantitative coronary angiography; RCA = right coronary artery; RD = reference diameter; Type B2/C = American Heart Association/American College of Cardiology classification type B2 or type C.

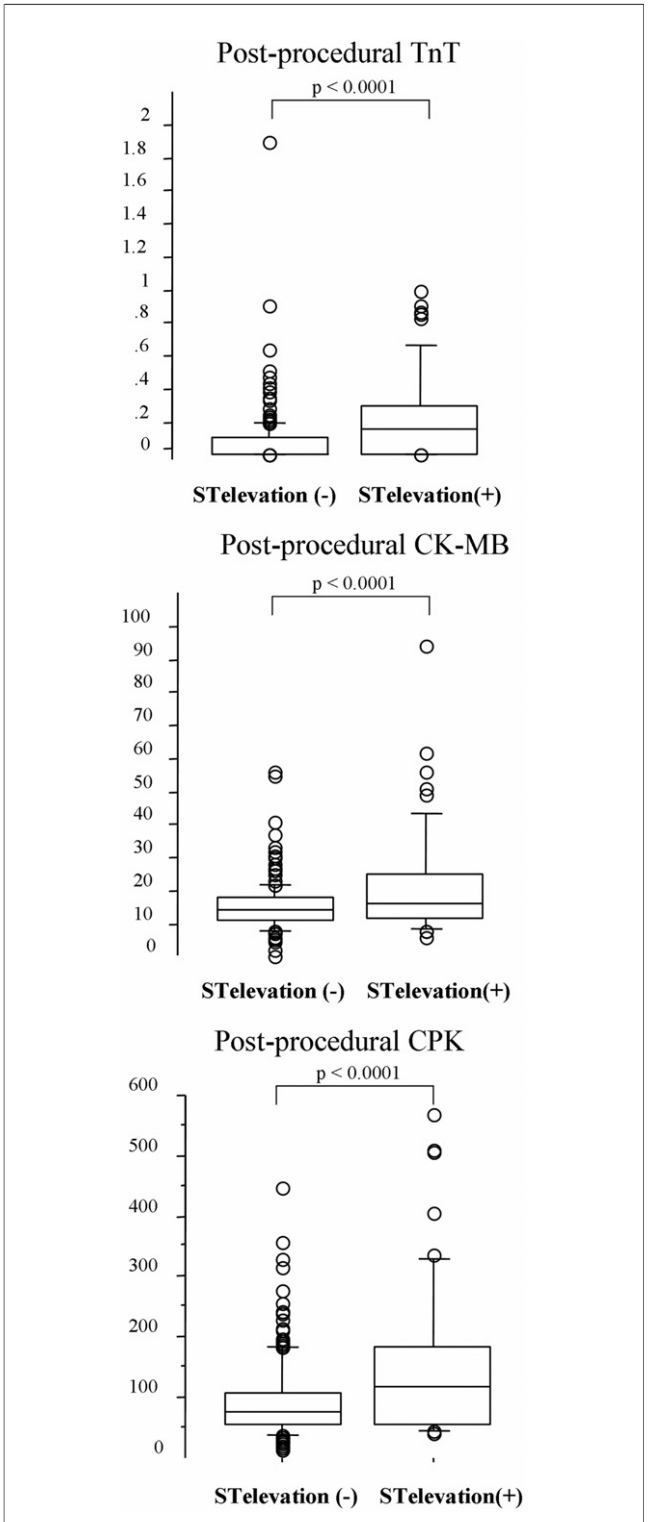

Figure 4. Cardiac Biomarkers of Patients With ST-Segment Elevation in the IcECG and Without ST-Segment Elevation

Post-procedural troponin-T (TnT), creatine kinase-MB isozyme (CK-MB) and creatine phosphokinase (CPK) were significantly higher in patients with ST-segment elevation in the intracoronary electrocardiogram (IcECG).

27.5 vs. 15.7 ± 13.7, $p < 0.001$, and 196.8 ± 240.2 vs. 90.0 ± 60.2, $p < 0.001$, respectively).

Association between post-procedural chest pain and ST-segment elevation in IcECG. The ST-segment elevation in the IcECG was recorded in 25 (54.3%) patients with post-procedural chest pain and in 40 (13.7%) patients without chest pain ($p < 0.001$). Surface ECG change was recorded in 5 (10.9%) patients with post-procedural chest pain.

Predictor of post-procedural myocardial injury. The independent predictors of positive TnT were glomerular filtration rate, lesion length, and ST-segment elevation in the IcECG in multiple logistic regression analysis (Table 3). Sensitivity, specificity, positive predictive value, and negative predictive value of ST-segment elevation in the IcECG, post-procedural chest pain, and surface ECG change for predicting post-procedural troponin elevation are shown in Table 4. Sensitivity and specificity of ST-segment elevation of IcECG were 54.9% and 92.2%, respectively. As compared with surface ECG, IcECG shows significantly higher sensitivity for positive TnT.

Angiographic and clinical outcomes. The incidence of angiographic complication, post-procedural, and the incidence of in-hospital major adverse cardiac events (defined as myocardial infarction and cardiac death) are shown in Table 5. A higher incidence of angiographic and clinical complications in patients with ST-segment elevation of IcECG was demonstrated.

Discussion

Major findings. In this study, we demonstrated that incidence of ST-segment elevation in the IcECG after successful elective PCI amounted to approximately 19%. We also showed the association between ST-segment elevation in the IcECG and the higher level of post-procedural cardiac biomarkers.

IcECG recording. A previous study demonstrated that IcECG is more sensitive compared with surface ECG for detecting myocardial ischemia during the PCI procedure (7,8). The ST-segment elevation in the IcECG after balloon deflation was documented, but the actual incidences and clinical significance of this phenomenon were not well-described. In this study, we recorded post-procedural ST-segment elevation in the IcECG without surface ECG change in 17% of the study population. Conversely, change of surface ECG without ST-segment elevation in the IcECG was recorded in 3 patients (1%). The IcECG is more sensitive and specific for detection of post-procedural myocardial injury of the treated area than surface ECG.

Post-procedural myocardial injury and IcECG. Significant post-procedural myocardial injury with cardiac biomarker elevation is not uncommon after apparently successful PCI (2,11). Contrast-enhanced MRI revealed myocardial

Table 3. Univariate and Multivariate Predictors of Post-Procedural Troponin-T Elevation After Coronary Stenting

	OR (95% CI)	p Value
Variables (univariate)		
ST-segment elevation of IcECG	14.4 (7.67–14.4)	<0.001
Lesion length (IVUS, mm)	1.04 (1.01–1.06)	0.001
Chest pain	3.56 (1.87–6.78)	0.001
GFR (ml/min/1.73 m^2 BSA)	0.98 (0.97–0.99)	0.002
Statins	0.67 (0.41–1.11)	0.12
Hypertension	1.54 (0.84–2.85)	0.16
ST-segment elevation of surface ECG	2.02 (0.64–6.36)	0.23
Previous myocardial infarction	1.37 (0.80–2.36)	0.25
Age	1.02 (0.99–1.05)	0.31
Smoking	1.30 (0.75–2.27)	0.35
Obesity	1.13 (0.66–1.93)	0.66
Restenotic lesion	0.95 (0.46–1.97)	0.89
Hyperlipidemia	1.01 (0.59–1.74)	0.96
Variables (multivariate)		
ST-segment elevation of IcECG	12.8 (6.14–26.64)	<0.001
GFR (ml/min/1.73 m^2 BSA)	0.98 (0.96–0.99)	0.01
Lesion length (IVUS, mm)	1.03 (1.00–1.05)	0.07
Hyperlipidemia	0.63 (0.30–1.33)	0.23
Previous myocardial infarction	1.54 (0.70–3.40)	0.28
Chest pain	1.54 (0.62–3.82)	0.35
Statins	0.71 (0.370–1.36)	0.31
Restenotic lesion	0.68 (0.25–1.87)	0.46
Age	0.98 (0.94–1.03)	0.48
Diabetes	1.19 (0.56–2.52)	0.65
Smoking	1.21 (0.44–3.29)	0.72
Obesity	1.13 (0.56–2.28)	0.72
Hypertension	0.98 (0.43–2.23)	0.97

CI = confidence interval; IVUS = intravascular ultrasound; OR = odds ratio; other abbreviations as in Table 1.

necrosis in patients with post-procedural CK-MB elevation (12). It is well-known that surface 12-lead ECG is rather insensitive for detecting minor myocardial injury. Left ventricular electromechanical mapping demonstrated evidences of electromechanical changes after successful PCI (13). We demonstrated that monitoring ST-segment changes of IcECG is a speedy and economical method for identifying risks of cardiac injuries after the PCI procedures in the catheterization laboratory.

The mechanism of myocardial injury after a successful PCI is debatable. Several factors that are related to post-procedural cardiac biomarker elevation were indicated, such as plaque burden of the target lesion, lesion length, side branch occlusion, platelet-monocyte aggregate, age, duration of balloon inflation, number of stents deployed, stent expansion adjunctive to debulking devices, and statin administration (3,14–18). These findings suggested procedure-related microembolization is an important mechanism of biomarker elevation.

Table 5. Angiographic and In-Hospital Outcomes

	ST-Segment Elevation (−)	ST-Segment Elevation (+)	p Value
Large side branch occlusion	1 (0.4%)	4 (6.2%)	<0.01
Distal embolism	1 (0.4%)	3 (4.6%)	<0.01
Transient slow flow during procedure	15 (5.5%)	17 (26.2%)	<0.01
In-hospital MACE	1 (0.4%)	5 (7.7%)	<0.01

MACE = major adverse cardiac event.

Recently, remarkably low restenosis rates of drug-eluting stents were demonstrated, and usage of coronary stents rapidly increased. Coronary stents might increase risks of post-procedural myocardial injury due to side branch occlusion and distal embolism. Larger stent expansion associated with a higher level of post-procedural CK-MB suggests a trade-off between optimal stent implantation and post-procedural myocardial injury (19). Therefore distal protection devices and an aspiration system were introduced to prevent embolization during PCI for acute myocardial infarction patients or saphenous vein graft interventions (20,21). Some studies demonstrated that intravenous nicorandil administration reduced cardiac biomarker elevation after coronary stenting procedures or primary PCI (22,23). Evaluation of IcECG might provide useful information on indicating and monitoring of these interventions.

Recently Balian et al. (24) demonstrated that intracoronary ST-segment shift after a successful PCI was associated with post-procedural myocardial injury and worse clinical outcome. Our study showed results similar to this report surveyed around the same period. Additionally we also demonstrated usefulness of a polymer-covered uninsulated guidewire and association with ST-segment elevation in IcECG and persistent chest pain after the procedure.

Table 4. Prediction of Post-Procedural Troponin-T Elevation After Stenting by IcECG, Surface ECG, and Post-Procedural Persistent Chest Pain

	Sensitivity	Specificity	PPV	NPV	p Value
ST-segment elevation of IcECG	54.9%	92.2%	69.2%	86.5%	<0.001
Persistent chest pain	26.8%	90.7%	47.8%	79.5%	<0.001
Surface ECG change	6.1%	96.9%	38.5%	76.4%	0.31

IcECG = intracoronary electrocardiogram; NPV = negative predictive value; PPV = positive predictive value.

Chest pain after successful PCI and IcECG findings. Persistent chest pain after a successful PCI was not uncommon in patients without angiographic complications. Recently, an association between post-procedural chest pain and myocardial injury with enzyme elevation after the PCI procedure was demonstrated (25). In this study, a higher incidence of ST-segment elevation in IcECG was demonstrated in patients with chest pain after PCI than in those without chest pain. An association between post-procedural chest pain and TnT elevation was also demonstrated in this study. Therefore, this finding indicated that minor myocardial injury might play an important role in the development of post-procedural chest pain.

Study limitations. A major limitation of this study is the fact that the definition of ST-segment elevation in the IcECG that reflects local myocardial ischemia has not been established. The shift of distal-tip position can induce an apparent change of IcECG waveform such as T-wave inversion or change of amplitude of QRS complex. Thus, we defined persistent ST-segment elevation in the IcECG as an ischemic change. However, ST-segment shifts defined as an elevation or depression of ST-segment ≥1 mm compared with the baseline allowed more sensitive identification (74% sensitivity) of post-procedural myocardial injury (24).

Glycoprotein IIb/IIIa receptor inhibitors have shown to reduce the morbidity and mortality of patients undergoing high-risk PCI (26,27). A previous study demonstrated glycoprotein IIb/IIIa receptor inhibitor administration reduced the incidence of post-procedural cardiac biomarker elevation (28). Thus, we speculated that administration of these drugs have an influence on the appearance of ST-segment elevation in the IcECG. Although many evidences support the benefit of these drugs for patients with PCI, none of the patients in this study population had been administered these drugs, because they are yet to be approved in our country.

Conclusions

Intracoronary ECG provides useful information predicting post-procedural myocardial injury easily and inexpensively in patients who have undergone apparently successful PCI procedures. An association between ST-segment elevation of IcECG and persistent chest pain after procedures suggested that a minor ischemic event might be a potential mechanism of persistent chest pain.

Acknowledgments

The authors would like to thank Mr. Takahiko Matsumoto, Mr. Hiroyuki Furuta, and Mr. Shinji Watanabe for their technical support of the IcECG recording and Mr. Katsu Endo (Abbott Vascular) for supplying technical information about the guidewire.

Reprint requests and correspondence: Dr. Tadayuki Uetani, Department of Cardiology, Chubu Rosai Hospital, 1-10-6, Komei, Minato-ku, Nagoya, Japan. E-mail: uetani@med.nagoya-u.ac.jp.

REFERENCES

1. Klein LW, Kramer BL, Howard E, et al. Incidence and clinical significance of transient creatine kinase elevations and the diagnosis of non non-Q wave myocardial infarction associated with coronary angioplasty. J Am Coll Cardiol 1991;17:621–6.
2. Saadeddin SM, Habbab MA, Sobki SH, et al. Minor myocardial injury after elective uncomplicated successful PTCA with or without stenting: detection by cardiac troponins. Catheter Cardiovasc Interv 2001;53:188–92.
3. Abdelmeguid AE, Topol EJ, Whitlow PL, et al. Significance of mild transient release of creatine kinase-MB fraction after percutaneous coronary interventions. Circulation 1996;94:1528–36.
4. Simoons ML, van den Brand M, Lincoff M, et al. Minimal myocardial damage during coronary intervention is associated with impaired outcome. Eur Heart J 1999;20:1112–9.
5. Gruberg L, Fuchs S, Waksman R, et al. Prognostic value of cardiac troponin I elevation after percutaneous coronary intervention in patients with chronic renal insufficiency: a 12-month outcome analysis. Catheter Cardiovasc Interv 2002;55:180–1.
6. Garbarz E, Inug B, Lefevre G, et al. Frequency and clinical implications of discordant creatine kinase-MB and troponin measurements in acute coronary syndromes. Am J Cardiol 1999;84:515–8.
7. Meier B, Rutishauser W. Coronary pacing during percutaneous transluminal coronary angioplasty. Circulation 1985;71:557–61.
8. Friedman PL, Shook TH, Kirshenbaum JM, et al. Value of the intracoronary electrocardiogram to monitor myocardial ischemia during percutaneous transluminal coronary angioplasty. Circulation 1986;74:330–9.
9. Abaci A, Oguzhan A, Topsakal R, et al. Intracoronary electrocardiogram and angina pectoris during percutaneous coronary interventions as an assessment of myocardial viability: comparison with low low-dose dobutamine echocardiography. Catheter Cardiovasc Interv 2003;60:469–76.
10. Yajima J, Saito S, Honye J, et al. Intracoronary electrocardiogram for early detection of myocardial viability during coronary angioplasty in acute myocardial infarction. Int J Cardiol 2001;79:293–9.
11. Fuchs S, Gruberg L, Singh S, et al. Prognostic value of cardiac troponin I re-elevation following percutaneous coronary intervention in high-risk patients with acute coronary syndromes. Am J Cardiol 2001;88:129–33.
12. Riccardi MJ, Uw E, Davidson CJ, et al. Visualization of discrete microinfarction after percutaneous coronary intervention associated with mild creatine kinase-MB elevation. Circulation 2001;103:2780–3.
13. Perin EC, Silva GV, Sarmento-Leite R, et al. Left ventricular electromechanical mapping: preliminary evidence of electromechanical changes after successful coronary intervention. Am Heart J 2002;144:693–701.
14. Johansen O, Brekke M, Strømme JH, et al. Myocardial damage during percutaneous transluminal coronary angioplasty as evidenced by troponin T measurements. Eur Heart J 1998;19:112–7.
15. Mandadi VR, DeVoe MC, Ambrose JA, et al. Predictors of troponin elevation after percutaneous coronary intervention. Am J Cardiol 2004;93:747–50.
16. Briguoria C, Colombo A, Airoldi F, et al. Statin administration before percutaneous coronary intervention: impact on periprocedural myocardial infarction. Eur Heart J 2004;25:1822–8.
17. Mehran R, Dangas G, Mintz GS, et al. Atherosclerotic plaque burden and CK-MB enzyme elevation after coronary interventions intravascular ultrasound study of 2256 patients. Circulation 2000;101:604–40.
18. Natarajan MK, Kreatsoulas C, Velianou JL, et al. Incidence, predictors, and clinical significance of troponin-I elevation without creatine kinase elevation following percutaneous coronary interventions. Am J Cardiol 2004;93:750–3.

19. Iakovou I, Mintz GS, Dangas G, et al. Increased CK-MB release is a "trade-off" for optimal stent implantation: an intravascular ultrasound study. J Am Coll Cardiol 2003;42:1900–5.
20. Umeda H, Katoh T, Iwase M, et al. The distal protection during primary percutaneous coronary intervention alleviates the adverse effects of large thrombus burden on myocardial reperfusion. Circ J 2006;70:232–8.
21. Hofmann M, Storger H, Schwarz F, et al. Percutaneous saphenous vein graft interventions with and without distal filter wire protection. J Interv Cardiol 2005;18:475–9.
22. Murakami M, Iwasaki K, Kusachi S, et al. Nicorandil reduces the incidence of minor cardiac marker elevation after coronary stenting. Int J Cardiol 2006;107:48–53.
23. Ishii H, Ichimiya S, Kanashiro M, et al. Impact of a single intravenous administration of nicorandil before reperfusion in patients with ST-segment-elevation myocardial infarction. Circulation 2005;112:1284–8.
24. Balian V, Galli M, Marcassa C, et al. Intracoronary ST-segment shift soon after elective percutaneous coronary intervention accurately predicts periprocedural myocardial injury. Circulation 2006;114:1948–54.
25. Kini AS, Lee P, Mitre CA, et al. Postprocedure chest pain after coronary stenting: implications on clinical restenosis. J Am Coll Cardiol 2003;41:33–8.
26. The CAPTURE Investigators. Randomized placebo-controlled trial of abciximab before and during coronary intervention in refractory unstable angina: the CAPTURE Study. Lancet 1997;349:1429–35.
27. The EPILOG Investigators. Platelet glycoprotein IIb/IIIa receptor blockade and low-dose heparin during percutaneous coronary revascularization. N Engl J Med 1997;336:1689–96.
28. Bonz AW, Lengenfelder B, Strotmann J, et al. Effect of additional temporary glycoprotein IIb/IIIa receptor inhibition on troponin release in elective percutaneous coronary interventions after pretreatment with aspirin and clopidogrel (TOPSTAR trial) J Am Coll Cardiol 2002;40:662–8.

Key Words: coronary intervention ■ intracoronary electrocardiogram ■ myocardial injury.