Intracoronary ST-Segment Shift Soon After Elective Percutaneous Coronary Intervention Accurately Predicts Periprocedural Myocardial Injury

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Background—Elevation of cardiac biomarkers after coronary angioplasty (percutaneous coronary intervention [PCI]) reflects periprocedural myocardial damage and is associated with adverse cardiac events. We assessed whether periprocedural myocardial damage that occurs despite successful PCI could be rapidly and easily identified by intracoronary ST-segment recording with the use of a catheter guidewire.

Methods and Results—In 108 consecutive stable patients undergoing elective single-vessel PCI, we recorded unipolar ECG from the intracoronary guidewire in the distal coronary before PCI and 2 minutes after the last balloon inflation. After PCI, intracoronary ST-segment shift ≥1 mm from baseline was considered significant. Troponin I levels were measured at baseline and at 8 and 24 hours after intervention, and myocardial damage was defined as troponin I increase above the upper normal value after intervention. All patients had normal cardiac marker values before PCI, and PCI was successful in all (residual stenosis <20%, Thrombolysis in Myocardial Infarction grade 3 flow). After PCI, long-term follow-up data were collected; myocardial damage was detected in 50 patients (46%), although abnormal creatine kinase-MB values were documented in only 11 (10%). Significant intracoronary ST-segment shift after PCI was present in 40 patients (37%; group A) and absent in the remaining 68 (63%; group B). Procedural myocardial damage was documented in 37 group A patients (93%) and in 13 group B patients (19%; $P<0.001$); significant ECG changes were found on standard ECG after intervention in only 5 patients (13%) and 1 patient (1%) ($P<0.05$). Sensitivity of intracoronary ST-segment shift for predicting myocardial damage was 74%, and specificity was 95%, with positive and negative predictive values of 93% and 81%, respectively. On multivariate analysis, intracoronary ST-segment shift was the sole independent predictor of myocardial damage (odds ratio, 54.1; 95% confidence interval, 12.1 to 240; $P<0.0001$). At a median follow-up of 12±5 months, major coronary event–free survival was significantly worse in group A patients (log-rank test $\chi^2=4.0$; $P<0.05$).

Conclusions—After successful single-vessel PCI, intracoronary ST-segment shift allows the prompt and inexpensive identification of patients developing myocardial injury, who may require adjunctive therapy and longer in-hospital stay. **(*Circulation*. 2006;114:1948-1954.)**

Key Words: angioplasty ■ electrocardiography ■ infarction ■ revascularization

Elevations of creatine kinase myocardial isoform (CK-MB) and cardiac troponin are relatively common after successful percutaneous coronary intervention (PCI).[1] Contrast-enhanced magnetic resonance imaging (MRI) studies have shown that even mild cardiac troponin elevations after PCI are associated with discrete microinfarction findings.[2,3]

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Myocardial damage in patients who have undergone successful PCI is mainly related to distal vessel thrombosis, embolization of plaque debris and platelet aggregates, and side-branch occlusion.[4,5] Inhibition of platelet aggregation by glycoprotein (GP) IIb/IIIa receptor antagonists or thienopyridines significantly reduces periprocedural myocardial injury and cardiac events.[6–8] A diagnostic tool able to detect early periprocedural myocardial damage could be of value in guiding management decisions and improving outcomes.

Unipolar intracoronary ECG recording from the angioplasty catheter guidewire has been shown to be more sensitive and reliable in detecting regional myocardial ischemia during balloon inflation than standard ECG.[9] We speculated that periprocedural intracoro-

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nary ST recording could provide simple and reliable information concerning the occurrence of periprocedural ischemia leading to myocardial damage. With this aim, we recorded intracoronary ECGs in patients undergoing elective PCI and correlated the ECG findings with the occurrence of myocardial damage.

Methods

Population

Between April 2003 and December 2004, 114 consecutive patients referred to our catheterization laboratory for elective PCI of a single lesion in a major native coronary artery were considered for this study. Men and women who were at least 18 years old were eligible for the study if they had normal CK-MB and cardiac troponin I (cTnI) values before the procedure and were in stable condition, without anginal pain in the previous 48 hours. Further criteria for inclusion were that the PCI procedure was successful and an optimal final result was obtained, ie, a Thrombolysis in Myocardial Infarction (TIMI) flow grade 3 in the treated vessel with a residual stenosis ≤20%, in the absence of major (>1.5 mm) side-branch occlusion or evident distal embolization. Unstable patients, patients with ventricular conduction disturbances on standard ECG or ventricular pacing, and those who had procedural complications were excluded. The study protocol was approved by the local Ethics Committee for Human Research. Informed consent was obtained from all patients.

Blood samples for serum cTnI and CK-MB were drawn at baseline and 8 and 24 hours after the procedure. cTnI assay was performed by a radioimmunoassay analyzer (Stratus CS- STAT Fluorometric Analyzer; Dade Behring, Inc, Deerfield, Ill); the upper normal limit (UNL) for cTnI was 0.1 ng/mL (the 99th percentile of the distribution of a reference control group with an analytical imprecision ≤10%). After PCI, a cTnI increase above the UNL in at least 1 of the 2 postprocedural samples was considered a marker of myocardial damage. CK-MB assay was performed with the use of CK-MB Immuno (Sentinel Diagnostics, Milan, Italy). After PCI, a CK-MB elevation was defined as a level above the UNL (10 U/L) in at least 1 of the 2 postprocedural samples. The degree of CK-MB or cTnI postprocedural increase was expressed as the CK-MB or cTnI peak ratio, ie, the maximum marker value divided by its UNL.

Percutaneous Coronary Intervention

Balloon angioplasty and stent implantation were performed according to standard clinical practice by the femoral approach. A total of 100 U/kg of heparin was administered intravenously at the start of the procedure (70 U/kg in patients also treated with GPIIb/IIIa inhibitors), followed by additional boluses as needed to maintain an activated clotting time >300 seconds. Ticlopidine 250 mg twice daily or clopidogrel 75 mg/d (after a preprocedure loading dose of 500 and 300 mg, respectively) was administered unless already started in the days preceding PCI. Use of GPIIb/IIIa inhibitors during the procedure was allowed at the operator's discretion. Statin therapy before the index PCI was considered if treatment had started at least 5 days before PCI.

At the end of PCI, anterograde coronary flow in the target vessel was assessed according to TIMI classification.[10] Angiographic parameters were assessed offline by personnel unaware of study allocation. Quantitative assessment was performed with the use of an automated edge detection system (Quantification Software Package Philips Integris H5000 C; Philips Medical Systems, Amsterdam, the Netherlands). Minor side-branch closure was defined as a TIMI flow grade <3 in a side branch of ≤1.5-mm diameter with normal pre-PCI flow. The number and duration of balloon inflations were recorded; the cumulative inflation time was computed by adding the time of each inflation. Total stent length in case of multiple stent implantations was also calculated by adding each stent length.

Intracoronary and Standard ECG Recording and Analysis

Intracoronary unipolar ECG was obtained and recorded by connecting the proximal tip of the catheter guidewire (Hi-Torque Balance

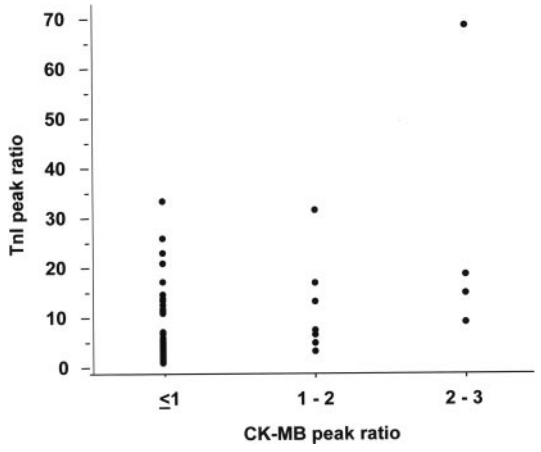

Figure 1. Distribution of cTnI (TnI) increase according to the corresponding CK-MB peak ratio.

TABLE 1. Demographic, Clinical, and Angiographic Data According to the Occurrence of Procedural Myocardial Damage

	Present (n=50)	Absent (n=58)	*P*
Male sex, n (%)	46 (92)	48 (83)	NS
Age, mean±SD, y	63±9	61±11	NS
Current smoker, n (%)	16 (32)	18 (31)	NS
Hypertension, n (%)	27 (54)	34 (59)	NS
Dyslipidemia, n (%)	16 (32)	22 (38)	NS
Diabetes, n (%)	11 (22)	14 (24)	NS
Angina, n (%)			
Stable	22 (44)	34 (59)	NS
Unstable IB*	2 (4)	5 (9)	NS
Unstable IIB*	26 (52)	19 (33)	NS
Single-vessel disease, n (%)	23 (46)	37 (63)	NS
Multiple-vessel disease, n (%)	27 (54)	21 (37)	NS
Vessel treated, n (%)			
Left anterior descending	15 (30)	21 (36)‡	NS
Left circumflex	16 (32)	19 (33)‡	NS
Right coronary artery	17 (34)	17 (29)‡	NS
Diagonal/intermediate branch	1 (2)	1 (1)‡	NS
Saphenous vein graft	1 (2)	1 (1)‡	NS
Lesion type B2/C†, n (%)	44 (88)	29 (50)	0.001
Balloon inflation time, s	87±61	49±37	<0.0001
Stent length, mm	26±14	17±8	<0.0001
No. of stents per patient	1.5±0.7	1.1±0.4	<0.0005
Stent overdilation, n (%)	22 (44)	9 (16)	0.001
Use of GPIIb/IIIa inhibitors, n (%)	14 (28)	9 (16)	NS
Use of drug-eluting stents, n (%)	14 (28)	15 (26)	NS
Abnormal baseline CRP levels, n (%)	17 (34)	15 (26)	NS
Statin therapy before PCI	25 (50)	28 (48)	NS

CRP indicates C-reactive protein.

*According to the Braunwald classification.[25]

†American Heart Association/American College of Cardiology type of lesion classification.[26]

‡Numbers do not add up to 100% because of overlap between groups.

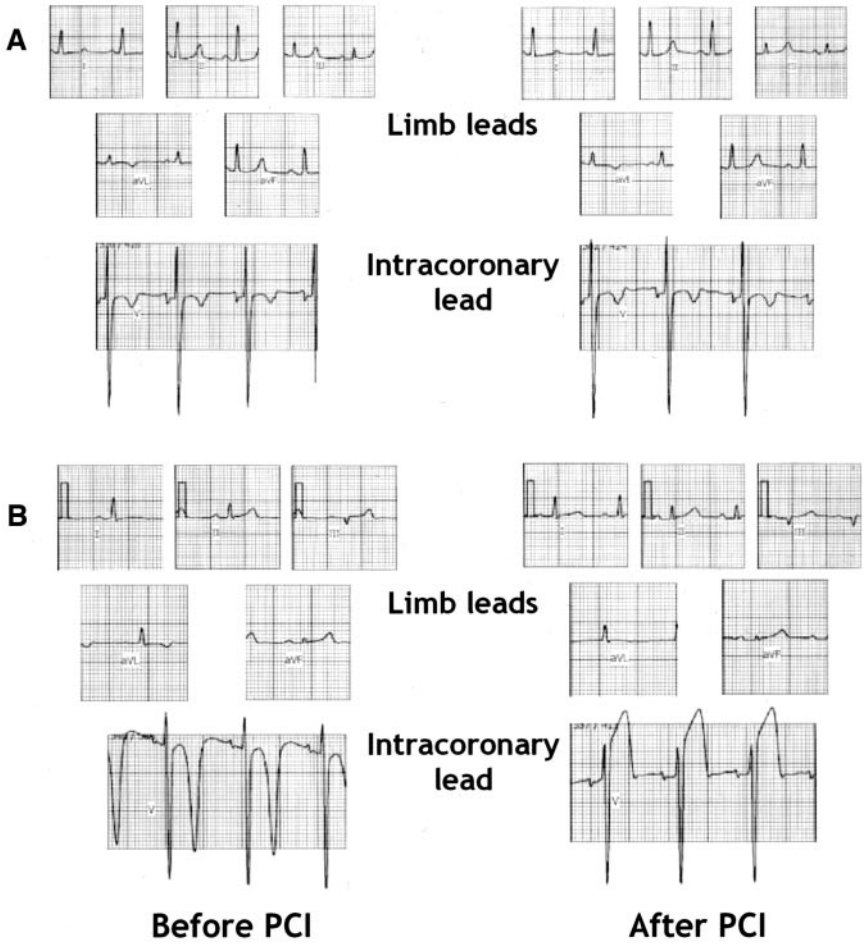

Figure 2. Examples of ECG recording before and after PCI in a patient without any changes (A) and in another patient with ST segment shift after the procedure (B). Standard ECG (limb leads) and intracoronary ECG are shown.

MiddleWeight, Guidant, Indianapolis, Ind; ATW, Cordis, a Johnson & Johnson Company, Miami Lakes, Fla) to a multichannel ECG recorder (MacLab 2000, Marquette Medical Systems, Inc, Milwaukee, Wis) with a paper speed of 25 mm/s and 10 mm/mV of signal amplitude, as previously described.[11] After the guidewire passed the stenosis and was positioned distally in the culprit vessel, intracoronary ECG was recorded (baseline intracoronary ECG recording) and then repeated 2 minutes after the last balloon inflation (post-PCI recording). Both baseline and post-PCI intracoronary ECG recordings were preceded by the intracoronary injection of 200 μg nitroglycerin. If the target lesion was located before a major vessel bifurcation, the guidewire tip was positioned in the major distal vessel. Intracoronary ST-segment changes were measured 20 ms after the end of the QRS or QS complexes by a lens-intensified hand-held caliper, approximated to the nearest 0.5 mm. The isoelectric line was considered the T-P segment preceding the QRS (or QS) complex. Three consecutive QRS complexes were analyzed, and mean ST-shift values were calculated. After PCI, intracoronary ST shift (elevation or depression) was considered significant if $\geq$1 mm compared with the corresponding baseline value.

Standard 12-lead ECGs were also recorded before and at the end of PCI and then 8 and 24 hours later. After PCI, the appearance of a new Q wave, T-wave inversion, or ST-segment shifts from baseline ECG were considered. Both intracoronary and standard ECG were analyzed by 2 observers unaware of other clinical and laboratory data.

Follow-Up

Follow-up was performed at regular intervals in the outpatient clinic or through telephone interviews by trained personnel, personal communication with the patient's physician, and, in the case of rehospitalization, by reviewing the patient's hospital records. Patients who underwent further coronary revascularization were censored at the time of the new procedure. Adverse events included death, nonfatal myocardial infarction (MI), or a new coronary revascularization procedure (coronary bypass surgery, repeat target lesion PCI, or PCI for a new lesion); major coronary events included death or nonfatal MI.

Statistical Analysis

Statistical analysis was performed with the use of StatView, version 5.0 (SAS Institute, Cary, NC). Normality of the data was verified by the Kolmogorov-Smirnov test. Continuous data are reported as mean$\pm$SD. Student t test for unpaired data was used to test differences between groups, and differences in rates of occurrence of categorical variables were compared by the χ^2 test with Yates correction or Fisher exact test, when appropriate. A multiple logistic regression analysis was used to identify the variables that were independently correlated with procedural myocardial damage, among those significantly associated on univariate analysis, with forward stepwise selection. Variables with a probability value <0.05 were entered in the analysis, and variables with a probability value >0.10 were removed.

At follow-up data analysis, differences in major coronary event rates between patients with or without intracoronary ST-segment shift were analyzed with Kaplan-Meier and compared by means of the log-rank test. Odds ratios with 95% confidence intervals were calculated. All tests of significance were 2-tailed, and a probability value <0.05 was considered significant.

The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results

Among the 114 patients originally considered for this study, 2 patients had a noninterpretable intracoronary ECG, and 4 pa-

TABLE 2. Demographic, Clinical, and Angiographic Data According to Presence (Group A) or Absence (Group B) of Significant Intracoronary ST-Segment Shift After PCI

	Group A (n=40)	Group B (n=68)	*P*
Male sex, n (%)	37 (93%)	57 (84)	NS
Age, mean±SD, y	63±10	61±10	NS
Current smoker, n (%)	15 (38)	19 (28)	NS
Hypertension, n (%)	25 (63)	36 (53)	NS
Dyslipidemia, n (%)	13 (33)	25 (37)	NS
Diabetes, n (%)	10 (25)	15 (22)	NS
Angina, n (%)			
Stable	15 (38)	41 (60)	NS
Unstable IB*	1 (3)	6 (9)	NS
Unstable IIB*	24 (60)	21 (31)	NS
Single-vessel disease, n (%)	20 (40)	40 (59)	NS
Multiple-vessel disease, n (%)	20 (40)	28 (41)	NS
Vessel treated, n (%)			
Left anterior descending	14 (35)‡	22 (32)‡	NS
Left circumflex	14 (35)‡	21 (31)‡	NS
Right coronary artery	10 (25)‡	23 (34)‡	NS
Diagonal/intermediate branch	1 (3)‡	1 (1)‡	NS
Saphenous vein graft	1 (3)‡	1 (1)‡	NS
Lesion type B2/C†, n (%)	35 (88)	38 (56)	<0.01
Balloon inflation time, s	86±65	55±41	<0.01
Stent length, mm	27±14	18±9	<0.001
No. of stents per patient	1.6±0.7	1.1±0.4	<0.001
Stent overdilation, n (%)	15 (38)	16 (24)	NS
Use of GPIIb/IIIa inhibitors, n (%)	13 (33)	10 (15)	0.05
Use of drug-eluting stents, n (%)	11 (28)	14 (21)	NS
Abnormal baseline CRP levels, n (%)	11 (28)	21 (31)	NS
Statin therapy before PCI	20 (50)	32 (47)	NS

CRP indicates C-reactive protein.

*According to the Braunwald classification.[25]

†American Heart Association/American College of Cardiology type of lesion classification.[26]

‡Numbers do not add up to 100% because of overlap between groups.

tients had incomplete cTnI and CK-MB data. The remaining 108 patients represent the study population. All patients had baseline cTnI and CK-MB values within normal limits before PCI.

According to cTnI rise after PCI, periprocedural myocardial damage was detected in 50 patients (46%). Most abnormal cTnI values were >5 times the UNL (23 of 50 patients; 46%); in 15 other patients (30%), cTnI values were between 3 and 5 times the UNL. After PCI, abnormal CK-MB values were observed in only 11 patients (10.2%), all associated with cTnI values >2 times the UNL. The magnitude of cTnI and CK-MB elevations after the procedure is shown in Figure 1. Demographic, clinical, and major angiographic findings of the study cohort according to the occurrence of periprocedural myocardial damage are reported in Table 1. Patients were comparable for coronary risk factors and clinical presentation (ie, stable/unstable status). In patients with periprocedural myocardial damage, lesions treated were more complicated; they required longer balloon inflations, more stents of greater length, and more stent overdilation (Table 1).

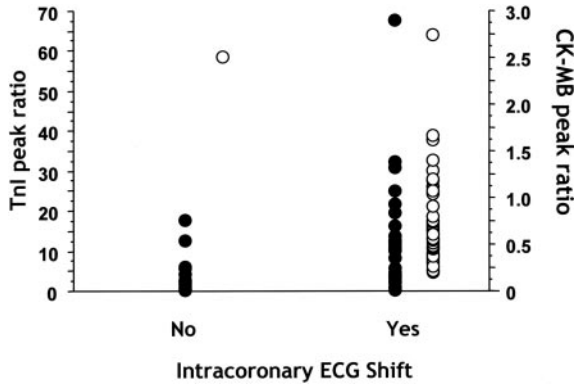

Figure 3. Scatterplot showing the distribution of cTnI (TnI) (black dots) and CK-MB (open dots) peak ratio after PCI, according to presence or absence of significant intracoronary ST-segment shift after PCI.

Standard and Intracoronary ECG Findings

On standard ECG recorded after PCI, no patient developed new Q waves. Other ECG changes (ST segment downsloping with T-wave inversion) were documented in only 6 patients (6%) (5 patients with and 1 without periprocedural myocardial damage; $P=0.09$).

On baseline intracoronary ECG recording, no patient showed ST-segment elevation >1 mm from the isoelectric line. On post-PCI intracoronary ECG recording, a significant ST-segment shift was present in 40 patients (37%; group A; ST-segment elevation in 31 patients and ST depression in 9 other patients) and absent in the remaining 68 (63%; group B). Examples of intracoronary ECG recordings are shown in Figure 2.

Group A and B patients were comparable for coronary risk factors and clinical presentation (ie, stable/unstable status). Major clinical and instrumental findings in groups A and B are reported in Table 2. Procedural myocardial damage was documented in 37 group A patients (93%) and in 13 group B patients (19%; $P<0.001$). Magnitude of increase of cTnI and CK-MB after the procedure in group A and B patients is shown in Figure 3. Mean ST-segment shift was 3.4±2.8 mm in patients with myocardial damage versus 0.2±0.8 mm in those without myocardial damage ($P<0.001$).

Sensitivity of intracoronary ST-segment shift after PCI for predicting procedural myocardial damage was 74%, specificity was 95%, and the predictive positive and negative accuracy values were 93% and 81%, respectively; overall accuracy was 85%. Sensitivity of intracoronary ST-segment shift for predicting CK-MB values above the UNL after the procedure was 91%, and specificity was 59%; predictive positive and negative accuracy values were 20% and 98%, respectively, and overall accuracy was 71%.

Prediction of Procedural Myocardial Damage

When the variables significantly related to procedural myocardial damage (Table 1) were analyzed by logistic regression, a significant intracoronary ST-segment shift after PCI was the sole independent predictor of procedural myocardial damage (odds ratio, 54.1; $P<0.0001$) (Table 3).

TABLE 3. Logistic Model Coefficients for cTnI Increase

				95% Confidence Limits	
Variable	χ^2	*P*	Odds Ratio	Lower	Upper
ST-segment shift after PCI	27.42	<0.0001	54.1	12.1	240.7
Stent overdilation	5.78	0.16	1.95	0.85	16.74
Inflation time	2.22	0.13	0.99	0.97	1.01
Stent length	0.18	0.67	0.98	0.91	1.06

Follow-Up Data

Complete follow-up was obtained in 107 of 108 patients (99.1%). During a median follow-up period of 12±5 months, 1 patient died of fatal MI; nonfatal MI occurred in 4 patients (4%), and 17 other patients (16%) underwent coronary revascularization (surgical revascularization in 5, repeat target lesion PCI in another 8, and PCI of a new lesion in 4 patients). When clinical and instrumental findings in patients with and without major coronary events at follow-up were compared, no significant differences were observed regarding risk factors, clinical presentation, coronary anatomy, medical treatment before and during PCI, procedural issues (balloon inflation time, stent length, and overdilation), and biomarker changes after PCI. Intracoronary ST-segment shift after PCI was the sole variable showing a trend toward a higher crude event rate: It was found in 4 of 5 patients (80%) who suffered nonfatal MI or death at follow-up, and in 36 of 102 (35%) who did not ($P=0.06$).

Follow-up data analyzed according to the intracoronary ST segment shift after PCI are reported in Table 4. Major coronary event–free survival was significantly worse in group A patients (log-rank test $\chi^2=4.0$; $P<0.05$) (Figure 4).

Discussion

The main finding of the present study is that intracoronary recording of ST segment after uncomplicated PCI allows the early identification of low-risk patients developing periprocedural myocardial damage. Earlier detection of myocardial damage could offer potential advantages in terms of enabling adjustment of therapeutic strategy and improvement of outcomes.

Myocardial Damage After Uncomplicated PCI

Although occlusion of a large side branch, flow-limiting dissection, and distal embolization of a large thrombus during PCI are well-known causes of large periprocedural MI, elevation of cardiac markers is relatively common even after uncomplicated PCI in otherwise asymptomatic patients; myocardial necrosis in this setting could result from embolization of plaque microparticle debris, intravascular friable material, clots, or cholesterol crystals. Final minimal luminal diameter independently correlates with CK-MB elevation, suggesting that strategies that maximize lumen dimensions to reduce restenosis may cause deeper injury of the vessel wall.[12] Abnormal postprocedural troponin elevations have been documented in at least 50% of patients undergoing PCI.[5,13] In a recent prospective multicenter cohort study that included 3494 consecutive patients undergoing PCI, abnormal postprocedural CK-MB and cTnI values were found in 16% and 44% of patients, respectively.[14] In our population of stable patients undergoing elective single-vessel PCI, the rates of abnormal CK-MB and cTnI elevation after intervention (10% and 46% of cases, respectively) were similar to those reported by others.[13,14]

A recent meta-analysis of 23 230 patients undergoing PCI in 7 large prospective trials showed that long-term mortality risk increases at any level above normal postprocedural CK-MB,[15] although in most studies postprocedural elevation of CK-MB, but not of cTnI, has been found to influence long-term mortality.[14,16] However, even mild cTnI elevation after PCI has been shown to be associated with discrete microinfarction findings on contrast-enhanced MRI studies.[2,3]

Several periprocedural therapies have been proposed to reduce the extent of post-PCI CK-MB elevations, including intracoronary administration of adenosine, β-blockers, verapamil,[17–19] and the prophylactic administration of GPIIb/IIIa receptor antagonists and thienopyridines to suppress platelet aggregation.[6–8] A diagnostic tool capable of early prediction of procedure-related myocardial damage could be of clinical value because the results of cardiac

TABLE 4. Major Adverse Events During Follow-Up According to Presence (Group A) or Absence (Group B) of Significant Intracoronary ST-Segment Shift After PCI

	Group A (n=40)	Group B (n=67)	Odds Ratio (95% CI)	*P*
Death, n (%)	1 (2.5)	0	...	...
Nonfatal MI, n (%)	3 (7.5)	1 (1.5)	5.4 (0.5-54.1)	NS
Cardiac death or MI, n (%)	4 (10)	1 (1.5)	7.4 (0.80-69.1)	0.06
Repeat PCI, n (%)	5 (12.5)	7 (10.4)	1.2 (0.36-4.22)	NS
Target lesion, n	3	5	...	...
New lesions, n	2	2	...	...
CABG, n (%)	4 (10)	1 (1.5)	7.4 (0.8-69.1)	0.06
Cumulative events, n (%)	10 (25.0)	7 (10.4)	2.8 (0.8-8.2)	0.06

CI indicates confidence interval; CABG, coronary artery bypass grafting. Ellipses indicate analysis not done because of too few events.

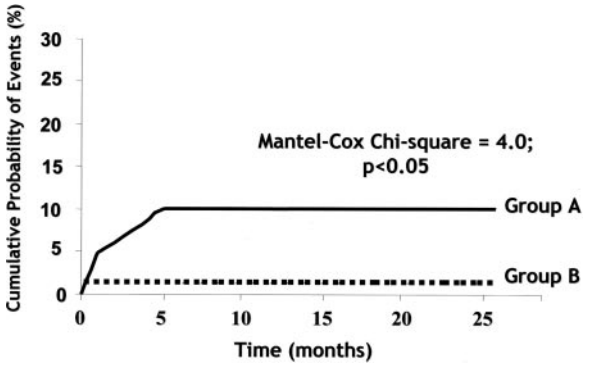

Figure 4. Cumulative frequency of major coronary events (death or nonfatal MI) in patients with (group A) or without (group B) significant intracoronary ST-segment shift after PCI.

markers after PCI influence early management strategies as well as discharge strategies. According to current guidelines, patients with a CK-MB index increase >3 times the UNL should be treated as having an MI and recommended for further observation and management per standard practice for MI.[20–22] Unfortunately, standard ECG changes and chest pain after PCI are inaccurate in the assessment of periprocedural damage.

Intracoronary ST-Segment Shift

At ECG recording during acute myocardial ischemia, the magnitude of the current of injury is influenced by the distance of the recording electrode from the region of ischemia, and an electrode placed on the surface of the heart is more accurate than surface ECG leads in detecting ischemic ST-segment changes.[9] We recently documented that intracoronary ECG recording during primary PCI is accurate in detecting ECG changes from the infarct area and predicts late infarct zone recovery.[11] In the present study, a significant intracoronary ST-segment shift accurately predicted even mild post-PCI myocardial damage (global accuracy of 85% for any abnormal cTnI value after PCI). It was also very sensitive in forecasting a more severe cardiac marker release (sensitivity and negative predictive value of 91% and 98%, respectively, for any abnormal CK-MB increase). Of note, in our study a significant intracoronary ST-segment shift after PCI was the sole independent predictor of procedural myocardial damage at multivariable analysis.

Several patients showed abnormal ST-segment shift without subsequent myocardial damage; possibly in these patients an otherwise "innocent" embolization occurred (ie, without distal myocardial damage despite the transient ischemia that was correctly recorded by the guidewire). Alternatively, myocardial damage was eventually prevented by the aggressive antithrombotic treatment.

In contrast, abnormal cTnI values after PCI in the absence of ST-segment shift were documented in only 8 patients; a small side-branch occlusion in the stented segment was found in 3 of them. Two different patterns of myocardial damage after PCI have recently been reported with the use of contrast-enhanced MRI[2,3]: In the first pattern, necrotic tissue was immediately adjacent to the implanted stent; in the second pattern, the necrotic tissue was located more distally in the myocardium subtended by the stented artery.

Intracoronary ST-Segment Shift and Outcome

After an otherwise successful procedure, the issue of whether any elevation of CK-MB has an independent association with subsequent mortality is widely debated.[4,5,14,15,23] The incremental clinical utility and cost-effectiveness of preventing small degrees of periprocedural embolization in patients with perceived low risk may not be attractive. Although periprocedural non–Q-wave MIs, the predominant events, were significantly prevented by administration of GPIIb/IIIa receptor inhibitors in most trials,[6,7] GPIIb/IIIa inhibition seems to be unnecessary in low-risk patients undergoing elective PCI, if they are adequately pretreated.[24] Unfortunately, even careful angiographic assessment cannot fully predict which patients will ultimately develop myonecrosis from lower levels of periprocedural embolization. A simple finding of such intracoronary ST-segment shift after PCI could be used as an indicator for safe early discharge or for the need for troponin or CK-MB assessment in centers where this is not routine. An early "bail-out" use of more potent antithrombotic therapies based on guidewire electrogram abnormalities could attenuate the sequelae of distal debris embolization and platelet aggregation, reduce the ongoing myocardial injury, and improve clinical outcome.

In the present study, a significant relationship was found between major adverse events during the follow-up and intracoronary ST-segment shift, the latter proving to be the sole variable with a trend toward more major coronary events of borderline significance. However, our study was neither aimed nor powered to assess the relationship between intracoronary ST-segment shift and long-term outcome; moreover, only low-risk patients in stable conditions and with normal cardiac marker values were considered, and very few (n=5) major coronary events occurred at follow-up. Intracoronary ST-segment shift after PCI could result in improved diagnosis of periprocedural MI and allow consideration for institution of adjunctive therapies aimed at reducing MI and improving other late outcomes. Further study is required to determine whether such a strategy can provide these benefits.

Conclusions

Intracoronary ST-segment shift recording after uncomplicated PCI promptly and inexpensively allows the early identification of those patients developing periprocedural myocardial damage, who may require adjunctive therapeutic interventions and longer in-hospital stay despite "successful" PCI.

Disclosures

None.

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CLINICAL PERSPECTIVE

Elevations of creatine kinase myocardial isoform and troponin are relatively common after successful percutaneous coronary intervention (PCI), and even mild troponin elevations after PCI are associated with discrete microinfarction findings. A diagnostic tool able to detect early periprocedural myocardial damage could be of value in guiding management decisions and improving outcomes. From the intracoronary guidewire in the distal coronary, before PCI and after the last balloon inflation, we recorded unipolar ECG in 108 consecutive stable patients undergoing elective single-vessel coronary angioplasty and correlated the intracoronary ECG findings with the occurrence of myocardial damage, defined as troponin I increase above the upper normal value after intervention. All patients had normal baseline cardiac markers and a successful coronary intervention; after coronary angioplasty, myocardial damage was detected in 46% of patients. Sensitivity of intracoronary ST-segment shift for predicting myocardial damage was 74%, and specificity was 95%, with positive and negative predictive values of 93% and 81%, respectively. On multivariate analysis, intracoronary ST-segment shift was the sole independent predictor of myocardial damage; major coronary events at a follow-up of 12±5 months were more common in patients showing significant intracoronary ST-segment shift after PCI. Intracoronary recording of ST segment after uncomplicated PCI could promptly and inexpensively improve the diagnosis of periprocedural infarction and allow consideration for institution of adjunctive therapies aimed at reducing myocardial infarction and improving late outcome. Further study is required to determine whether such a strategy can provide these benefits.

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Intracoronary ST-Segment Shift Soon After Elective Percutaneous Coronary Intervention Accurately Predicts Periprocedural Myocardial Injury

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