The outcome of primary percutaneous coronary intervention for stent thrombosis causing ST-elevation myocardial infarction

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Background There are very few scientific data about the effectiveness of primary percutaneous coronary intervention (PCI) in patients with ST-elevation myocardial infarction (STEMI) due to stent thrombosis (ST). The purpose of the present study is to investigate the efficacy and outcome of primary PCI for STEMI due to ST in the largest consecutive patient population with ST reported to date.

Methods A total of 2,644 consecutive STEMI patients undergoing primary PCI were retrospectively enrolled into the present study. The primary end point of this study was *successful angiographic reperfusion* defined as postprocedural Thrombolysis In Myocardial Infarction grade III flow. The secondary end points were cardiovascular death and reinfarction.

Results Stent thrombosis was the cause of STEMI in 118 patients (4.4%). In patients with ST, angiographic success (postprocedural Thrombolysis In Myocardial Infarction grade III flow) was worse than in patients with de novo STEMI (76.3% vs 84.8%, $P = .01$). Patients with ST had significantly higher incidence of in-hospital cardiovascular mortality than patients with de novo STEMI (10.2% vs 5.3%, $P = .02$). In-hospital reinfarction rate was similar in both groups. In addition, long-term (mean 22 months) cardiovascular mortality and reinfarction rates were significantly higher in patients with ST compared with those without (17.4% vs 10.5%, $P = .02$ and 15.6% vs 9.5%, $P = .03$, respectively).

Conclusions Primary PCI for treatment of ST is less effective, and these patients are at increased risk for in-hospital and long-term mortality compared with patients undergoing primary PCI due to de novo STEMI. (Am Heart J 2010;159:672-6.)

The clinical presentation of a coronary stent thrombosis (ST) is very often an ST-elevation myocardial infarction (STEMI)[1,2]; and according to the current guidelines, the preferred treatment should be primary percutaneous coronary intervention (PCI).[3] However, there are few reports concerning the outcome of PCI for ST treatment.[4,5] Furthermore, these studies included also patients with acute coronary syndromes, which means a heterogeneous patient population in terms of clinical presentation.[4,5] The short- and long-term outcome of patients undergoing primary PCI for de novo STEMI has been recently indicated.[6] Nevertheless, very few scientific data are available concerning the effectiveness of primary PCI in patients with STEMI due to ST.[7,8] Therefore, the purpose of the present study is to investigate the efficacy and outcome of primary PCI for STEMI due to ST in the largest consecutive patient population with ST reported to date.

Methods

Patient populations

In a retrospective study, 2,644 consecutive patients with STEMI presenting at the institution of Siyami Ersek Thoracic and Cardiovascular Surgery Center, Training and Research Hospital, between October 2003 and March 2008 were included. The study inclusion criteria were as follows: electrocardiogram (ECG) revealing *STEMI*, defined as >30 minutes of continuous typical chest pain and ST-segment elevation ≥2 mm in 2 contiguous electrocardiography leads within 12 hours of symptom onset or up to 18 hours if there was evidence of continuing ischemia or hemodynamic instability. Exclusion criterion for the study entry was the development of ST during the index procedure or within 24 hours of stent implantation (acute ST). Acute, subacute, late, and very late stent thromboses

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were defined according to the Academic Research Consortium.[9] Primary PCI for definite ST was considered a PCI due to angiographically confirmed thrombus that originated in the stent or in the segment 5 mm proximal or distal to the stent.[9] Accordingly, patients with primary PCI were divided in an ST group and a no-ST group. The study protocol was approved by the Ethics Committee of Siyami Ersek Thoracic and Cardiovascular Surgery Center, Training and Research Hospital.

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Analysis of patient data

The patients' demographic information, cardiovascular history, and risk factors (smoking, hypercholesterolemia, hypertension, and diabetes mellitus [DM]) were obtained from medical records. Blood values that were determined at hospital admission and on a daily basis during patient stay in hospital were recorded from medical reports. A 12-lead ECG was recorded in each patient just after hospital admission; and also, MI type was recorded from ECGs.

Coronary angiography, primary angioplasty, and stenting

All patients received chewable 300 mg aspirin and clopidogrel (300-mg loading dose) before coronary angiography. Angiographic data of the patients were evaluated from catheter laboratory records. Emergency coronary angiography and angioplasty were performed by the percutaneous femoral approach. Heparin (10,000 IU) was administered when arterial access was secured. After visualizing the left and right coronary arteries, 2.5 mg of nitrate was selectively injected into the infarct-related artery (IRA) to rule out possible coronary spasm. Angiographic assessments were made at the treating hospital by visual assessment. Primary angioplasty including balloon angioplasty and/or stent implantation was performed only for IRA according to lesion type. *Optimal angiographic success* was defined as final Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 and <20% in the IRA at the end of the procedure.[10] After angioplasty, all patients were admitted to the coronary care unit. Five hundred units per hour of intravenous heparin or 1 mg/(kg d) of subcutaneous low–molecular-weight heparin was given to patients who are at high risk for systemic emboli such as atrial fibrillation, previous emboli history, known left ventricular thrombus, or cardiogenic shock. One hundred milligrams aspirin and 75 mg clopidogrel were continued in all patients. The use of glycoprotein IIb/IIIa inhibitors was left to the discretion of the operator. Concomitant medical treatment with β-blockers, angiotensin-converting enzyme inhibitors, and statins was prescribed according to American College of Cardiology/American Heart Association guidelines.

After hospital discharge, dual antiplatelet therapy was recommended for ≥1 month in patients who received a bare metal stent and for ≥6 months in patients who received a drug-eluting stent.

Definition

Time to reperfusion was measured as the time from symptom onset to the coronary reperfusion obtained with balloon inflation. *Door-to balloon time* was defined as the time between hospital admission and balloon inflation. *Cardiogenic shock* was defined as prolonged hypotension (systolic blood pressure <85 mm Hg), with evidence of decreased organ perfusion caused by severe left ventricular dysfunction, right ventricular infarction, or mechanical complications of infarction. *Renal failure* was defined as a glomerular filtration rate (GFR) <60 mL/(min 1.73 m^2), which was estimated by the simplified Modification of Diet in Renal Disease equation.[11] *Patient with diabetes mellitus* (DM) was defined as the patient with documented DM using either oral hypoglycemic agents or insulin treatment at admission. *Hypercholesterolemia* was defined as total cholesterol ≥200 mg/dL or use of cholesterol-lowering agents. *Cardiovascular mortality* was defined as unexplained sudden death, death due to acute MI, heart failure, and arrhythmia. *Reinfarction* was defined as an increase in creatine kinase (CK) of more than twice the last value associated with CK-MB ≥10% of the total CK and ST-segment reelevations. Follow-up data were obtained from hospital records or by interviewing with (directly or by telephone) patients, their families, or their personal physician. The primary end point of this study was *successful angiographic reperfusion* defined as postprocedural TIMI grade III flow. The secondary end points were in-hospital and long-term (mean 22 months) cardiovascular death and reinfarction.

Statistical analysis

Quantitative variables were expressed as mean value ± SD, and qualitative variables were expressed as percentage. Comparison of parametric values between 2 groups was performed by means of 2-tailed Student *t* test. Categorical variables were compared by the likelihood-ratio χ^2 test or Fisher exact test. Backward stepwise multivariate Cox regression analysis was performed to identify independent predictors of long-term cardiovascular mortality and reinfarction. Unsuccessful procedure, DM, age >70 years, female gender, hypertension, current smoker, admission cardiogenic shock, ST at admission, renal failure, anterior MI, and PCI history were entered into the model. Reinfarction was also entered in this model for cardiovascular mortality. The cumulative survival curves for long-term cardiovascular mortality were constructed with the use of the Kaplan-Meier method with differences assessed with the log-rank test. A P value < .05 was considered statistically significant. All statistical studies were carried out with SPSS program (version 15.0; SPSS, Chicago, IL).

Results

Patient characteristics

The mean follow-up time of the patients was 22 months. The clinical characteristics and risk factors in the 2 groups are summarized in Table I. In 2,644 consecutive patients with STEMI treated by primary PCI, ST was the cause of STEMI in 118 patients (4.4%), of which 34.7% (n = 41) were subacute ST, 30.6% (n = 36) were late ST, and 34.7% (n = 41) were very late ST, at the baseline. Patients with ST had similar risk profile to patients with de novo STEMI but a higher rate of PCI, MI, and bypass history. In addition, renal failure and

Table I. Baseline characteristics of study patients

	ST		
	Yes (n = 118)	**No (n = 2526)**	***P* value**
Age, y (SD)	57.4 (11.4)	56.6 (11.9)	.48
Female gender, n (%)	17 (14.4)	439 (17.3)	.42
DM, n (%)	30 (25.4)	638 (25.2)	.96
Hypertension, n (%)	58 (49.1)	1038 (41.1)	.12
Current smoker, n (%)	81 (68.8)	1557 (61.6)	.13
Hypercholesterolemia, n (%)	40 (33.6)	926 (36.6)	.52
Family history, n (%)	24 (20.5)	432 (17.1)	.35
PCI history, n (%)	118 (100)	93 (3.7)	<.001
Bypass history, n (%)	9 (7.6)	69 (2.7)	.002
MI history, n (%)	54 (45.7)	216 (8.6)	<.001
Anterior MI, n (%)	66 (55.9)	1230 (48.6)	.12
Admission cardiogenic shock, n (%)	9 (7.6)	100 (3.9)	.05
Creatinine at admission, mg/dL (SD)	1.11 (0.72)	0.98 (0.37)	.001
Creatinine at discharge, mg/dL (SD)	1.23 (0.94)	1.09 (0.55)	.01
Renal failure, n (%)	22 (18.5)	305 (12.1)	.04

Table II. Angiographic and procedural characteristics of patients

	ST		
	Yes (n = 118)	**No (n = 2526)**	***P* value**
Culprit lesion			
LMCA, n (%)	1 (0.8)	5 (0.19)	.14
LAD, n (%)	65 (55)	1236 (48.9)	.17
CX, n (%)	13 (11)	338 (13.3)	.34
RCA, n (%)	39 (33.1)	19 (36.7)	.42
Bypass graft, n (%)	0 (0)	0 (0.7)	.27
3-Vessel disease	27 (22.8)	660 (26.1)	.43
Proximal location of the lesion, n (%)	72 (61.5)	1351 (53.5)	.08
Stent use, n (%)	50 (42.3)	2100 (83.1)	<.001
Drug-eluting stent implantation	3 (2.5)	63 (2.4)	.21
Tirofiban use, n (%)	67 (56.7)	1182 (46.7)	.03
Angiographic success, n (%) (postprocedural TIMI grade III flow)	90 (76.3)	2143 (84.8)	.01
Volume of contrast medium, mL (SD)	239.9 (109.1)	236.6 (93.8)	.71
Reperfusion time, h (SD)	2.4 (1.9)	3.3 (2.4)	.004
Door-to-balloon time, min (SD)	32 (21)	34 (23)	.47

LMCA, Left main coronary artery; *LAD*, Left anterior descending coronary artery; *CX*, circumflex coronary artery; *RCA*, right coronary artery.

cardiogenic shock at admission were more prevalent in the patients with ST.

Angiographic and procedural characteristics

Angiographic and procedural characteristics are depicted in Table II. Culprit lesions were similar in the 2 groups. The patients with ST had significantly lower incidence of stent use (42.3% vs 83.1%, $P < .001$) and higher incidence of tirofiban use (56.7% vs 46.7%, $P = .03$). In patients with ST, angiographic success (post-procedural TIMI grade III flow) was worse than in patients with de novo STEMI (76.3% vs 84.8%, $P = .01$). Door-to-balloon time was similar in both groups, but reperfusion time was shorter in patients with ST.

Table III. In-hospital and long-term (mean 22 months) cardiac events of all study patients

	ST		
	Yes (n = 118)	**No (n = 2526)**	***P* value**
In-hospital cardiac events			
In-hospital mortality, n (%)	12 (10.2)	136 (5.3)	.02
Reinfarction, n (%)	5 (4.2)	55 (2.17)	.14
In-hospital mortality or reinfarction, n (%)	16 (13.5)	185 (7.3)	.12
Long-term cardiac events (including in-hospital events)	(n* = 115)	(n† = 2483)	
Mortality, n (%)	20 (17.4)	261 (10.5)	.02
Reinfarction, n (%)	18 (15.6)	238 (9.5)	.03
Mortality or reinfarction, n (%)	36 (31.3)	460 (18.5)	.001

* n = 115 for ST group (there was no follow-up for 3 patients).
† n = 2483 for no-ST group (there was no follow-up for 43 patients).

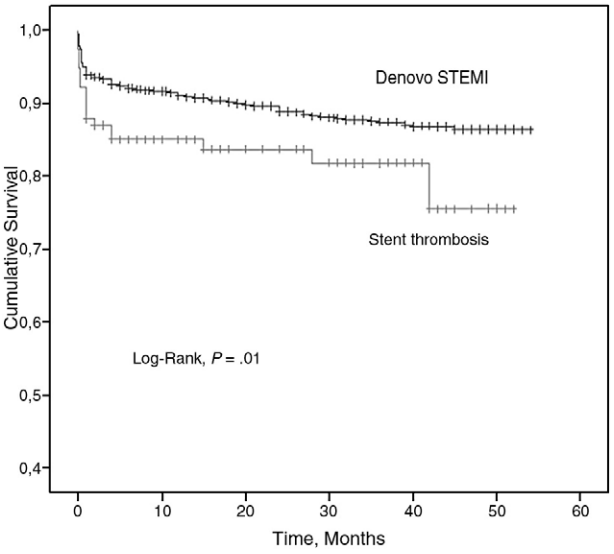
In-hospital and long-term outcomes

Table III presents the in-hospital and long-term outcomes after primary PCI. Patients with ST had significantly higher incidence of in-hospital cardiovascular mortality than patients with de novo STEMI (10.2% vs 5.3%, respectively; $P = .02$). In-hospital reinfarction rate was similar in both groups. Follow-up data after discharge were not obtained for 3 (2.5%) patients with ST and 43 (1.7%) patients with de novo STEMI. Long-term cardiovascular mortality and reinfarction rates were significantly higher in patients with ST compared with those without. (17.4% vs 10.5%, $P = .02$; 15.6% vs 9.5%, $P = .03$, respectively). In the long-term follow-up, the ST-driven reinfarction in ST group was more frequent compared with the patients with de novo STEMI (73% vs 55%, $P = .02$). The Kaplan-Meier survival plot for cardiovascular mortality in both groups is presented in Figure 1.

Independent predictors of long-term cardiovascular mortality and reinfarction

Independent predictors of long-term cardiovascular mortality were determined by multivariate Cox regression analysis. These predictors of long-term cardiovascular mortality are depicted in Table IV. Cardiogenic shock, unsuccessful procedure, age >70 years, renal failure, DM, reinfarction, and anterior MI were found to be independent predictors of long-term cardiovascular mortality.

Figure 1



Kaplan-Meier curves for long-term cardiovascular mortality for patients with ST and those with de novo STEMI.

Independent predictors of long-term reinfarction were also determined by multivariate Cox regression analysis (Table IV). The following were considered independent variables of long-term reinfarction: ST at admission (odds ratio [OR] 1.95, 95% CI 1.13-3.37, $P = .01$), current smoker (OR 1.49, 95% CI 1.09-2.03, $P = .01$), and DM (OR 1.43, 95% CI 1.05-1.96, $P = .02$).

Discussion

The present study, the largest to date examining the efficacy of primary angioplasty in ST, produced several clinical implications. Firstly, ST is a relatively rare cause of STEMI. Secondly, primary PCI is less effective in the recanalization of the IRA of patients with STEMI due to ST. Thirdly, patients with ST have a poor long-term outcome compared with patients with de novo STEMI. Finally, patients with ST are more likely to die or develop reinfarction at follow-up.

Stent thrombosis accounted for 4.4% of the primary PCI for STEMI during the 5 years of study period. Stent thrombosis occurred at different intervals from stent implantation. The differences in baseline clinical characteristics between the 2 groups were the higher prevalence of previous MI, previous PCI, and GFR in the ST group. It is not surprising, as we already know, that acute coronary syndrome and renal failure are risk factors for ST.[12]

Compared with patients with de novo STEMI, primary PCI was less effective in the recanalization of the IRA of patients with STEMI due to ST. An optimal result was obtained only in 76% of cases with ST, compared with 84% in patients with de novo STEMI ($P = .01$). In accordance with our results, Chechi et al[8] showed a lower successful reperfusion rate (80%) in patients with ST. Previous studies investigating reperfusion methods for ST showed an unacceptably high failure rate because of residual thrombus after balloon angioplasty alone.[13] Even supplementary therapy with intracoronary fibrinolysis showed no clinical benefit.[13] In contrast, a small study suggested a clinical benefit with the use of GP IIb/IIIa inhibitors during primary PCI for ST.[14] In our series, the patients with ST had significantly higher incidence of tirofiban use (56.7% vs 46.7%, $P = .03$); but postprocedural TIMI grade III flow remained worse compared with patients with de novo STEMI. Parodi et al[7] reported a 96% success rate in patients with STEMI due to ST treated with primary PCI and concluded that primary PCI for treatment of ST mirrors the results of primary PCI in the setting of a primary coronary event. Compared with our results (76%), this is a very high success rate. Wenawesser et al[4] reported that the use of GP IIb/IIIa is associated with increased reperfusion success in patients undergoing PCI for ST. The low incidence of GP IIb/IIIa usage may play an important role in our low reperfusion rate of ST. In addition to the high incidence of GP IIb/IIIa use (75%), the stenting rate of patients with ST is also very high (83%) in the series of Parodi et al.[7] Regarding that balloon angioplasty alone has a poor reperfusion rate,[13] the low incidence of stent usage rate (42%) in our study may also be considered to be another reason for our low success rate. In the series of Parodi et al,[7] rheolytic thrombectomy was also a tool in dealing with ST. Our poor success rate may also be related partly to not using thrombectomy, a fairly accepted intervention to manage fresh thrombus burden in patients with ST.[15,16]

Clinical consequences of ST are generally severe, including short-term mortality rates up to 20% to 25% and MI in 60% to 70% of cases.[17] In our study, patients with ST showed higher in-hospital and long-term mortality rate than the patients with de novo STEMI ($P = .02$). The worse clinical characteristics of the patients with ST may partly explain the adverse outcome. For instance, the GFR was low at the baseline in patients

Table IV. Independent predictors of long-term (mean 22 months) cardiovascular mortality and reinfarction in all study patients

	OR	95% CI	*P* value
Cardiovascular mortality			
Cardiogenic shock	9.16	5.0-16.73	<.001
Unsuccessful procedure	4.76	3.35-6.77	<.001
Age >70 y	3.2	2.19-4.67	<.001
Renal failure	3	2.03-4.42	<.001
DM	2.94	2.1-4.12	<.001
Reinfarction	2.85	1.85-4.41	<.001
Anterior MI	1.45	1.03-2.05	.03
Reinfarction			
ST at admission	1.95	1.13-3.37	.01
Current smoker	1,49	1.09-2.03	.01
DM	1.43	1.05-1.96	.02

with ST. In a study conducted by Sadeghi et al,[18] it has been shown that reduced GFR was a powerful independent predictor of 30-day mortality and remained associated with reduced survival at 1 year in patients undergoing primary PCI. Likewise, we found renal failure to be an independent mortality predictor for long-term mortality in the study population.

In particular, we found that patients with ST had a higher rate of cardiogenic shock at presentation despite it being statistically nonsignificant. This finding could in part explain the higher in-hospital mortality because the development of cardiogenic shock during STEMI is characterized by poor prognosis, with an early mortality of up to 50% despite reperfusion therapy.[19] Cardiogenic shock was also the strongest independent predictor of long-term mortality in our study population.

Patients with ST were more likely to die or develop reinfarction at follow-up. Stent thrombosis resulted as an independent strong predictor of reinfarction. Reinfarction, mainly driven by ST recurrence, was frequent despite dual antiplatelet therapy recommendation according to current guidelines. Therefore, more aggressive management seems justified pending proof of efficacy of specific strategies, including extensive use of stents with a meticulous implantation technique and an intensive, prolonged antiplatelet therapy[20] for the primary and secondary prevention of ST.

In conclusion, primary PCI for treatment of ST is less effective; and these patients are at increased risk for in-hospital and long-term mortality compared with patients undergoing primary PCI due to de novo STEMI.

Study limitations

Several limitations need to be kept in mind when interpreting the results of this study. Firstly, this study carries the well-known limitation of the retrospective design. Secondly, microvascular perfusion, the main target of a myocardial reperfusion, was not assessed in this study. If it had been assessed, it might have provided additional information.

Disclosures

All authors explicitly declare that this study has no conflict of interest.

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