

Stent thrombosis in acute coronary syndromes: Patient-related factors and operator-related factors

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ABSTRACT

Objective: Stent thrombosis (ST) is a common phenomenon in acute coronary syndromes (ACS) when compared to stable coronary artery disease. This study analyzed the patient- and operator-related risk factors of ST in ACS.

Methods: Coronary angiograms of 1738 consecutive ACS patients admitted in a large tertiary center between year 2014 and 2016 were analyzed retrospectively for the presence of ST. The paired angiograms [ST in ACS during and after percutaneous coronary intervention (PCI)] of the patients were analyzed by two independent observers, with focus on lesion characteristics and procedure techniques. Clinical and laboratory data were collected.

Results: Stent thrombosis was found in 29 (1.6%) ACS patients, with a combination of at least one clinical/laboratory risk factor and one lesion/operator risk factor identified in 28 (96%) out of the 29 ACS patients with ST. The following risk factors for ST were found: Renal insufficiency (OR=4.14, $p<0.001$, 95% CI=1.73-9.88), type 2 diabetes (OR=2.21, $p=0.034$, 95% CI=1.06-4.61), excessive alcohol consumption (OR=3.12, $p=0.023$, 95% CI=1.17-8.33), stent implantation for ST-elevation myocardial infarction (STEMI) (OR=2.28, $p=0.029$, 95% CI=1.08-4.81), left main (LM) or left anterior descending artery (LAD) as culprit lesion (OR=2.80, $p=0.010$, CI 95%=1.27-5.95), and absence of antiplatelet therapy prior to ST (OR=3.58, $p=0.002$, 95% CI=1.60-7.96). The following lesion/operator possible risk factors were identified: Bifurcation lesion ($n=7$; 24%), heavy coronary calcifications ($n=13$; 44%), in-stent restenosis with secondary plate rupture ($n=6$, 20%), inappropriate stent size selection ($n=6$, 20%), and errors in periprocedural drug administration ($n=4$, 14%).

Conclusion: ST occurred in 1/62 ACS patients after PCI. A combination of clinical/laboratory and lesion/operator risk factors were present in almost all ACS patients with ST. This finding may support the search for strictly individualized strategies for the treatment of ACS patients with ST after PCI. (*Anatol J Cardiol* 2020; 24: 274-9)

Keywords: stent thrombosis, risk factors, acute coronary syndrome, antithrombotic treatment

Introduction

Stent thrombosis (ST) is the most dreadful complication of percutaneous coronary intervention (PCI) (1-3). Over the last two decades, coronary angioplasty with stent implantation has gained a firm position in treating patients with coronary vascular disease (CVD). PCI is used alongside with cardiovascular surgery, especially in acute coronary syndrome. Although the incidence of ischemic heart disease is on a decline, the absolute number of coronary atherosclerotic patients treated by implantation of stent is on the increase (4). The benefits of the stent implantation used for acute and elective cases of CVD are indisputable (5). Every medical procedure which leaves an artificial device (such as stent implantation) in an organism carries with certain risks (which in this case is stent thrombosis).

Stent thrombosis is the complete occlusion of a coronary vessel in place of previously implanted stent. This situation usually results in ST-elevation myocardial infarction (STEMI) (6, 7) with very poor prognosis. Mortality rate of this event was reported as high as 40% (8). The prevalence of ST in the modern era of drug eluting stent is between 1.1 and 1.9% (9-12). The clinical risk factors for the development of stent thrombosis are well known.

The previous works on ST suggest acute coronary syndrome as a reason for the stent implantation. However, history of some chronic conditions, vascular dysfunction, and progression of the neoatherosclerosis are the most common risk factors for the development of ST (13-20). In this study we analyzed the clinical, laboratory and operator/lesion characteristics of ACS patients with ST in order to determine the possible risk factors for developing ST.

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Methods

Coronary angiograms of 1738 consecutive patients (admitted in large tertiary center between year 2014 and 2016) with acute coronary syndrome (ACS) including ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (non-STEMI) and unstable angina pectoris were analyzed retrospectively for the presence of ST. There were no exclusion criteria. Paired angiograms (First PCI from the time of stent implantation and second PCI at the time of ST) from 29 ACS patients with ST were analyzed by two independent observers, with focus on lesion characteristics and procedure techniques. The stent type, as well as the post-dilatation procedure followed, was based on the discretion of the operator. Clinical and laboratory data collated from a review of patients' charts were included in the analysis. The analyzed patient characteristics were sex, age, presence of diabetes mellitus 2 type, renal insufficiency, arterial hypertension, dyslipidemia, current smoking status, and regular and excessive consumption of alcoholic beverages (defined as more than 1 standard drink per day). Renal insufficiency was defined as the history of chronic kidney disease stage 3. Patients with type 2 diabetes were included, provided that they were on diet or treated orally with anti-diabetic medications or insulin. The analyzed laboratory characteristic (at time the of stent implantation) were hemoglobin level, creatinine level, troponin level and platelet count. Other characteristics studied were reason for stent implantation (initial diagnosis of STEMI), culprit lesion treated with stent in left main (LM) or left anterior descending artery (LAD), and non-usage of anti-aggregation medication before ST. In our study, only ACS patients with angiographic or post-mortem verified occlusions in stented area were included. The acute and late ST were included according to ARC criteria (21). The follow-up mortality data was obtained from the national health insurance registry. The determination of the possible lesion/operator risk factors for ST was done after a detailed review of literature on the topic, and was decided upon by the investigators.

Statistical analysis

The test for normal distribution of data was by Kolmogorov-Smirnov normality test. Data was expressed as the mean±standard deviations. Student's t-test was done to check for differences between two groups of quantitative data. Chi-square or Fisher's exact test was used (where applicable) to check for the differences between groups of categorical data which were expressed as counts and percentages. The importance of risk factors were subjected to univariate logistic regression. P values less than 0.05 were adopted as significant. All statistical analysis was done using IBM SPSS version 23.

Results

ST was found in 29 (1.6%) of 1738 ACS patients screened. The mortality after two years follow-up was 30%. Two thirds of the

ACS patients with ST were men but were non-significantly different from the women. More than half of the analyzed patients were older than 65 years. The other characteristics which did not differ statistically ($p>0.05$) were history of arterial hypertension, dyslipidemia and smoking. Statistical significant differences were found for renal insufficiency (24% vs. 7%, $p<0.001$), type 2 diabetes (48% vs. 29%, $p=0.034$), alcohol consumption (17% vs. 6%, $p=0.023$), STEMI as reason for stent implantation (58% vs. 36%, $p=0.029$), LM or LAD as a culprit lesion (65% vs. 40%, $p=0.010$) and non-usage of anti-aggregation medications before ST (31% vs. 8%, $p=0.002$). There was a statistical significant difference in the absolute platelet count (243 ± 70 vs. 273 ± 75 , $p=0.005$). The laboratory levels of creatinine, troponin and hemoglobin between the two groups were non-significant ($p>0.05$). The summary of these characteristics are shown in Table 1 and 2. The following risk factors for ST were found: renal insufficiency (OR=4.14, $p<0.001$, 95% CI=1.73-9.88), type 2 diabetes (OR=2.21, $p=0.034$, 95% CI=1.06-4.61), excessive alcohol consumption (OR=3.12, $p=0.023$, 95% CI=1.17-8.33), stent implantation for STEMI (OR=2.28, $p=0.029$, 95% CI=1.08-4.81), LM or LAD as culprit lesion (OR=2.80, $p=0.010$, 95% CI=1.27-5.95), and non-usage of anti-aggregation medications before ST (OR=3.58, $p=0.002$, 95% CI=1.60-7.96). Overview of the risk factors is in Table 3. The average length of hospital stay for the group with ST (6.9 ± 2.7 days) was non-significantly ($p=0.20$) different from that of the control group (5.14 ± 3.43 days).

The stents in the ST were 19.07 ± 6.02 mm in length, with the nominal diameter of 3.32 ± 0.44 mm. There was no significant ($p>0.05$) difference between the nominal diameter and the real diameter after stent implantation. The most frequent stent types used were metallic stents (BMS, 27%) and drug-eluting stents of second generation with everolimus (EES, 24%). The various stent types are shown in Table 4. There were no first generation DES or bioresorbable stents with ST in our study. Post-dilatation was used only in two patients (6%) in the ST group. An average of 1.62 ± 0.55 stent was implanted per patient in the ST group, and this was significantly different ($p<0.001$) from that of the control group (1.15 ± 0.11). A suboptimal result of stenting procedure was more often documented for patients in the ST group [3 (10%) vs. 54 (3%), $p=0.02$].

The possible lesion/operator risk factors of ST can be divided into several parts. The lesion related factors were bifurcation lesion ($n=7$; 24%), heavy coronary calcifications ($n=13$; 44%), in-stent restenosis with secondary plate rupture and ST ($n=6$, 20%). Operator related factors were wrong stent size selection (diameter under-sizing or incomplete lesion coverage) ($n=6$, 20%), and errors in periprocedural drug administration ($n=4$, 14%). The patient-related factor errors in the use of anti-aggregation medications was identified as responsible for ST in 3 patients while the other factors were identified in 4 patients. An overview of the lesion/operator risk factors is shown in Figure 1. A combination of at least one clinical/laboratory risk factor and one lesion/operator risk factor was identified in 28 (96%) out of the 29 ACS patients with ST.

Table 1. Basic clinical characteristics

	Without stent thrombosis, n (%)	With stent thrombosis, n (%)	P-value
Male sex	1205 (70)	24 (82)	0.157
Age above 65 years	1013 (59)	17 (58)	0.619
Type 2 diabetes	507 (30)	14 (48)	0.034
Chronic kidney disease	122 (7)	7 (24)	<0.001
Arterial hypertension	1096 (64)	17 (59)	0.540
Dyslipidemia	567 (33)	13 (45)	0.191
Active smoking	878 (51)	13 (45)	0.485
Alcohol abuse	107 (6)	5 (17)	0.023
STEMI as an indication for the initial PCI	654 (38)	17 (58)	0.029
LM or LAD as culprit vessel	698 (41)	19 (65)	0.010
Absence of antiplatelet therapy prior to ST	191 (11)	9 (31)	0.002
Total	1709 (100)	29 (100)	

LM - left main; LAD - left anterior descending artery

Table 2. Basic laboratory characteristics

	Without stent thrombosis (mean±SD)	With stent thrombosis (mean±SD)	P-value
Creatinine on admission (ug/L)	102±73	98±32	0.297
Troponin on admission (mg/L)	764±1501	1080±2232	0.230
Hemoglobin on admission (g/L)	137±18	132±18	0.083
Platelet count on admission (10⁹/L)	243±70	273±75	0.005

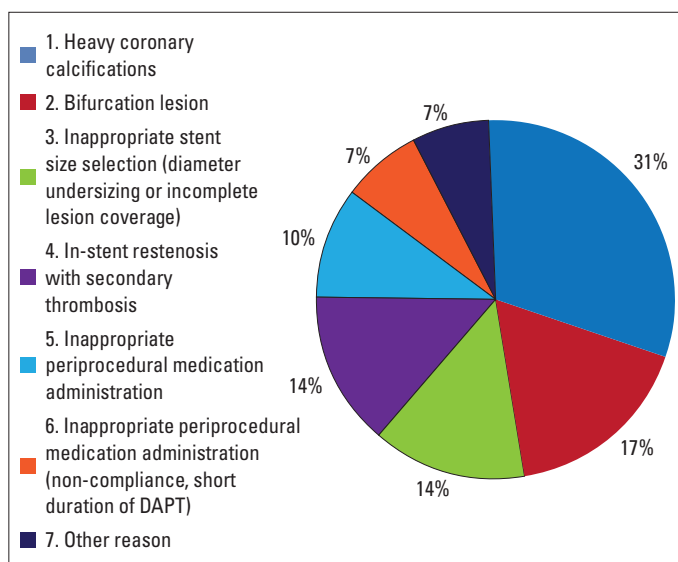


Figure 1. The possible lesion/operator risk factors of ST

Discussion

The main findings of this study is as follows: (1) the mortality rate of ST in our study was in consonant with that of other stud-

ies (22-24); (2) We identified possible lesion/operator risk factors, which are imminent threat of ST, in all patients with ST; (3) the risk of ST was much higher when several risk factors were combined. Despite the technology advancements in stent design and improved antiplatelet therapy, there is an increase in mortality rate of ST patients. As the number of patients treated by PCI increases, there is corresponding increase in the absolute count of the patients who are endangered. The risk factors can be divided into several categories which are characteristics of the patient, lesion characteristics and procedure outcome (25). Our results, considering the patient related risk factors, revealed statistical significant occurrence of stent thrombosis among patients with diabetes mellitus and renal impairment. Similar results have been demonstrated in a study conducted by Tada et al. (26) and Iakovou et al. (27). Excessive alcohol consumption is not frequently described as a risk factor in literature. In 2004, a study on alcoholic consumption and restenosis revealed a lower restenosis rate in patients consuming higher amounts of alcohol (28). The difference in the pathophysiology between restenosis and ST substantiates the findings in our study. The lesion characteristics, significant risk for ACS STEMI as a reason for stenting, and culprit lesion in LM or LAD (29) are not less important. The proposed mechanisms for this finding are malapposition,

Table 3. Risk factors for stent thrombosis

Risk factor	Odds ratio	P-value	95% Confidence interval
Male sex	2.75	0.157	0.76-5.29
Age above 65 years	0.83	0.619	0.41-1.70
Type 2 diabetes	2.21	0.034	1.06-4.61
Chronic kidney disease	4.14	0.001	1.73-9.88
Arterial hypertension	0.79	0.540	0.37-1.67
Dyslipidemia	1.64	0.191	0.78-3.43
Active smoking	0.76	0.485	0.37-1.61
Alcohol abuse	3.12	0.023	1.17-8.33
STEMI as an indication for the initial PCI	2.28	0.029	1.08-4.81
Left main or LAD as culprit vessel	2.75	0.01	1.27-5.95
Absence of antiplatelet therapy prior to ST	3.58	0.002	1.60-7.96

LAD - left anterior descending artery; PCI - percutaneous coronary intervention

Table 4. Stent types with ST

Stent type	n (%)
Everolimus eluting stent	7 (24)
Zotarolimus eluting stent	5 (17)
Biolimus eluting stent	5 (17)
Sirolimus eluting stent	3 (10)
Other type drug eluting stent	1 (3)
Bare metal stent	8 (27)

and stent struts crossing an ostial side branch (30). In addition, there were significantly more suboptimal results from index stent implantation, as well as more stents implanted per patient. This suggest that the complexity of the procedure itself plays a crucial role in ST. Similar findings was reported by Lüscher et al. (31). The percentage of patients who were subjected to post-dilatation was only 6% in ST group. The need for post-dilatation to achieve optimal stent diameter was demonstrated in POSTIT trial. Its positive effect on lowering the rate of ST has also been demonstrated. The heavy calcification and bifurcation lesion as a lesion related risk factors have been well documented (32, 33). The German authors have demonstrated that a decreased stent symmetry in calcified lesions predisposes to malposition of the stent (34). This pathologic findings have suggested that the arterial branch points are predisposed to the development of atherosclerotic plaque and thrombus because they are foci with shear stress (35). The next objects of interest are the stents themselves. The diameter of struts, biocompatibility of polymer containing drug which delays the endothelization, thus preventing restenosis, are important technical characteristics which could influence the development of ST. The dynamics of release of this drug is an important variable that affects the outcome of

the procedure significantly (36). In this study, there were only second generation DES and BMS in the non-randomized setting. We didn't analyze the rate of stent thrombosis between the stent types because DES and BMS were used in the ratio of 2:1. The last set of factors which can predispose to ST is the periprocedural technique of implantation, and the medication given to the patient during and after coronary intervention. Inappropriate stent size selection (diameter under-sizing or incomplete lesion coverage) was identified as the third most frequent predisposing factor for ST. Poorly conducted peri- and post procedural medication (mainly wrong dosage of heparin or non-usage of antiplatelet therapy during or few hours after stent implantation) was identified in 14% of patients with ST. Noncompliance in the usage of dual antiplatelet therapy is a well-known risk factor for ST. The absence of any antiplatelet therapy (ASA or P2Y12 inhibitors) predisposes to statistically higher rate of ST (37). In this study, the risk of ST after discontinuing the antiplatelet therapy was almost four times higher. The importance of patient compliance is therefore imminent. Finally, the combination of risk factors rather than only one, may probably be responsible for the complex events seen in the ST patients in our study. A combination of at least one clinical/laboratory and one lesion/operator risk factor was identified in 28 (96%) out of the 29 patients with ST. This finding suggests that the development of ST is influenced by multiple factors on the side of the patient as well on the side of the operator.

Study limitations

The observational study is by nature selectively bias. The stent type and length were in a non-randomized setting, and was solely the choice of the interventional cardiologist. We did not conduct any advanced visualization for the lesions. On the other hand, the visualization of every implanted stent by IVUS or OCT is inapplicable to conventional or real-life clinical practice, and not

even for patients who are unstable during procedure. Therefore, the review of angiograms was often only guided during by the interventional cardiologist.

Conclusion

Stent thrombosis occurred in 1/62 ACS patients after PCI. A combination of clinical/laboratory and lesion/operator risk factors were present in almost all ACS patients with ST. This finding may support the search for strictly individualized strategies for the treatment of ACS patients with ST after PCI.

Conflict of interest: None declared.

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