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Clinical

Prevalence and outcomes of non-ST-segment elevation myocardial infarction resulting from stent thrombosis☆

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ABSTRACT

Background/Purpose: Stent thrombosis is an infrequent yet one of the most feared complications after stent implantation. Stent thrombosis most commonly manifests as ST-segment elevation myocardial infarction, thus the data regarding non-ST-segment elevation myocardial infarction (NSTEMI) resulting from stent thrombosis are still sparse. The aim of the study is to evaluate the prevalence and outcomes of NSTEMI resulting from stent thrombosis.

Methods/Materials: We performed a retrospective analysis of 378 consecutive NSTEMI patients who underwent coronary angiography. Patients were divided into those with and without stent thrombosis. The primary outcome was in-hospital mortality. Secondary outcome was the incidence of large myocardial infarction defined as a peak troponin I value greater than 90th percentile of the entire study population (26.5 μg/L).

Results: Among 378 patients with NSTEMI, 12 (3.2%) patients had angiographically confirmed definite stent thrombosis. With respect to the timing of stent thrombosis, 2 patients had early, 3 had late and 7 had very-late stent thrombosis. Patients with stent thrombosis had a higher incidence of large myocardial infarction (33% vs. 9%, $p = 0.02$) and a higher albeit statistically insignificant peak troponin value (interquartile, 4.62 [0.19–64.0] μg/L vs. 1.21 [0.14–7.12] μg/L, $p = 0.25$) compared to those without stent thrombosis. There was no significant difference in in-hospital mortality between the two groups (8% vs. 2%, $p = 0.2$).

Conclusions: Stent thrombosis accounted for 3.2% cases of NSTEMI in our cohort of patients and patients with NSTEMI resulting from stent thrombosis had a higher incidence of large myocardial infarction.

1. Introduction

Stent thrombosis is an infrequent yet one of the most feared complications after stent implantation. Stent thrombosis most commonly manifests as ST-segment elevation myocardial infarction (STEMI) [1,2], and has been reported to account for about 3% to 9% cases of STEMI in contemporary practice [3–5]. Stent thrombosis has been shown to be associated with a lower rate of successful perfusion after coronary intervention and worse clinical outcomes in STEMI population [1,4]. Although STEMI is the most common presentation of stent thrombosis, 23% of the patients with stent thrombosis presented as non-ST-segment elevation myocardial infarction (NSTEMI) according to the largest registry data on stent thrombosis [2]. In contrast to data in the STEMI population, the data regarding NSTEMI due to stent thrombosis are still sparse. In this context, we aimed to evaluate the prevalence and outcomes of NSTEMI resulting from stent thrombosis.

2. Material and methods

A retrospective analysis was performed on all patients who underwent coronary angiography from January 2013 to June 2014 at Mount Sinai Beth Israel Hospital. Two researchers independently reviewed the emergency department records, in-hospital admission records and cardiac catheterization procedure records to identify patients with NSTEMI. Myocardial infarction (MI) was diagnosed according to the European Society of Cardiology and American College of Cardiology criteria [6].

Inclusion criteria were 1) troponin value greater than the 99th percentile reference value before cardiac catheterization; 2) chest pain (or anginal equivalent) or ischemic change on the electrocardiogram including horizontal or down-sloping ST-segment depressions (≥0.05 mV) or T-wave inversion (≥0.1 mV) in two or more contiguous leads; and 3) absence of ST elevation and new left bundle branch block on the electrocardiogram. Exclusion criteria were; 1) cardiac catheterization more than 5 days after presentation ($n = 143$); 2) nonobstructive coronary artery disease (CAD) ($n = 103$); 3) severe aortic stenosis, hypertrophic cardiomyopathy, self-reported cocaine use within 5 days,

Abbreviations: NSTEMI, non-ST-segment elevation myocardial infarction; STEMIST, segment elevation myocardial infarction; MI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; CAD, coronary artery disease; TIMI, Thrombolysis In Myocardial Infarction.

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cardiac arrest, ventricular tachycardia, supraventricular tachycardia with heart rate greater than 150 beats per min, implantable cardioverter defibrillator shock, and blood pressure on presentation >230/130 mmHg (n = 49); 4) subsequent documented diagnosis of Takotsubo cardiomyopathy, myocarditis, and pulmonary embolism (n = 15); and; 5) insufficient data for analysis (n = 40).

The present study complied with the Declaration of Helsinki and was approved by the institutional review board of our institution. Patients' demographic data and risk factors along with admission characteristics including hemodynamic parameters were obtained. The data regarding the treatment administered during hospitalization were also obtained. Baseline laboratory data including creatinine and cardiac troponin I were recorded. Cardiac troponin I levels were measured using the second-generation VITROS® (Ortho-Clinical Diagnostics Inc., NJ, USA). The upper limit of normal for cardiac troponin I was 0.034 μg/L, which represented the 99th percentile reference value. Troponin I was measured serially at approximately 6-h intervals before and after catheterization, as clinically indicated, with the highest level designated as the peak troponin. Estimated glomerular filtration rate was calculated using Modification of Diet in Renal Disease Study formula [7]. Electrocardiograms obtained on presentation were reviewed by two independent reviewers in a blinded fashion. ST-segment depressions ≥0.05 mV in more than two contiguous leads were recorded. Transthoracic echocardiography was performed during hospitalization, and left ventricular ejection fraction was calculated using either the Teichholz or biplane Simpson's method.

All patients underwent cardiac catheterization within 5 days after presentation. We used the cutoff of 5 days since angiography performed at a later time is less likely to represent the actual coronary flow at the time of the event. Revascularization procedures including percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG) were performed at the discretion of the treating physician. An independent cardiologist blinded to the clinical data interpreted all coronary angiography visually, and the assessment was compared with the primary assessment by the treating cardiologist. In case of discrepancy in the assessments, a third investigator made the final interpretation. In line with the standard definition of flow-limiting stenosis [8,9], obstructive CAD was defined as stenosis greater than or equal to 70% (50% for the left main coronary artery). Angiographic findings including the number of diseased vessels, ante-grade coronary flow according to the standard Thrombolysis In Myocardial Infarction (TIMI) criteria [10], preprocedural thrombus grade according to TIMI study group [11] and the nature of revascularization procedures were recorded. Stent thrombosis was defined according to the Academic Research Consortium criteria [12]. Angiographic successful PCI was defined as achieving a residual stenosis in the coronary artery of <50% with TIMI 3 flow or improved TIMI flow (from grade 0 or 1 before coronary intervention to grade 2 after coronary intervention). Three-vessel disease was present if there were obstructive CAD in all 3 major epicardial coronary arteries (left anterior descending, left circumflex and right coronary arteries). The infarct-related artery was determined based on the electrocardiogram, echocardiogram, and coronary angiogram findings. Multi territory ischemia was diagnosed in the presence of a stenosis ≥90% in more than one of the three major coronary arteries without a single identifiable culprit lesion.

The primary outcome was in-hospital mortality. Secondary outcome was the incidence of large MI defined as a peak troponin I greater than 90th percentile of the entire study population (26.5 μg/L). The cutoff value was determined based on a previous study demonstrating that the peak troponin level correlated with in-hospital mortality in a NSTEMI population, and that patients with a peak troponin level greater than 90th percentile of the study population had the worst prognosis [13]. In addition, in-hospital cardiogenic shock and heart failure were recorded.

Patients were categorized into those with and without stent thrombosis. Data are expressed as number (percentage) or median (interquartile range). Dichotomous variables were compared using the chi-squared test or Fisher's exact test. For continuous variables, the Shapiro–Wilk test was used to check the normality of the distribution. Continuous variables were compared using either the Student's t-test or Wilcoxon rank sum test, as deemed appropriate. A 2-sided p value < 0.05 was considered statistically significant. All statistical analyses were performed with R software, version 3.0.1 (The R Foundation for Statistical Computing, Vienna, Austria).

3. Results

After exclusion, 378 patients who underwent coronary angiography within 5 days after presentation with the diagnosis of NSTEMI were included in the final analysis. Among 378 patients, 12 (3.2%) patients had angiographically confirmed definite stent thrombosis. Baseline characteristics are summarized in Table 1. There was no significant difference in baseline characteristics except for a higher rate of current smoking and previous PCI in patients with stent thrombosis. At the time of the events, 6 out of 12 patients with stent thrombosis were taking dual antiplatelet therapy. With respect to the timing of stent thrombosis, 2 patients had early, 3 had late and 7 had very-late stent thrombosis. The types of previous stents were bare metal stent in 1 patient, drug-eluting stents in 9 patients and unknown in 2 patients. Patients with stent thrombosis had a higher albeit statistically insignificant peak troponin value compared with those without stent thrombosis.

Angiographic characteristics are summarized in Table 2. The median interval from the presentation to cardiac catheterization was 1.0 (0.6–2.1) days in the entire study population and 179 patients (47%) underwent cardiac catheterization within 24 h after presentation.

Table 1
Patients characteristics, hemodynamic, laboratory and echocardiographic data.

	Stent thrombosis (n = 12)	No stent thrombosis (n = 366)	p value
Age (years)	64 [54–70]	67 [58–78]	0.28
Men	10 (83)	234 (64)	0.23
Hypertension	11 (92)	282 (77)	0.31
Diabetes	2 (17)	152 (42)	0.13
Hyperlipidemia	9 (75)	214 (58)	0.37
Current smoking	6 (50)	84 (23)	0.04
Family history of CAD	2 (17)	80 (22)	1
Previous myocardial infarction	2 (17)	68 (19)	1
Previous PCI	12 (100)	121 (33)	<0.001
Previous CABG	1 (8)	57 (16)	0.7
Hemodynamic and laboratory data			
Systolic blood pressure (mmHg)	130 [115–136]	145 [125–161]	0.02
Heart rate (beats/min)	80 [73–84]	79 [70–94]	0.85
Killip class >1 on admission	1 (8)	57 (16)	0.7
eGFR (mL/min/1.73 m^2)	87 [64–95]	72 [52–89]	0.17
Troponin I on presentation (μg/L)	0.14 [0.03–1.56]	0.13 [0.05–1.04]	0.67
Peak troponin I (μg/L)	4.62 [0.19–64.0]	1.21 [0.14–7.12]	0.25
Electrocardiographic and echocardiographic data			
ST-segment depression	4 (33)	133 (36)	1
Left ventricular ejection fraction (%)	45 [42–56]	60 [43–61]	0.2
In-hospital treatment			
Aspirin	12 (100)	361 (99)	1
Thienopyridine	12 (100)	294 (80)	0.13
ACE inhibitor	10 (83)	200 (55)	0.07
Angiotensin receptor blocker	0 (0)	49 (13)	0.38
β blocker	11 (92)	333 (91)	1
Statin	12 (100)	359 (98)	1
Heparin infusion	6 (50)	269 (73)	0.1
Enoxaparin	1 (8)	24 (7)	0.57

Data are presented as n (%) or median [interquartile range].
CAD: coronary artery disease, PCI: percutaneous coronary intervention, CABG: coronary artery bypass grafting, TIMI: Thrombolysis In Myocardial Infarction, eGFR: estimated glomerular filtration rate, ACE: angiotensin converting enzyme.