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Original Article

The outcome of primary percutaneous coronary intervention in patients with stent thrombosis

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ABSTRACT

Background: Primary percutaneous coronary intervention (PCI) is a recommended management strategy for patients with de novo ST-segment elevation myocardial infarction (STEMI). Still, the efficacy of primary PCI in-stent thrombosis (ST) induced STEMI is unclear. The aim was to assess the clinical characteristics and the in-hospital outcomes of patients undergoing primary PCI for STEMI caused by acute, sub-acute, or late ST.

Methods: A sample of hundred consecutive patients who presented with STEMI due to ST were included in this study. The angiographic evidence of a flow-limiting thrombus or total vessel occlusion (thrombolysis in myocardial infarction (TIMI) flow grade 0 to II) at the site of the previous stent implant was taken as ST. Primary PCI was performed, and all enrolled patients and in-hospital mortality were observed.

Results: Male patients were 69, and the mean age was 58.9 ± 7.78 years. ST was categorized as acute in 40 patients, sub-acute in 53, and late in the remaining seven patients. Killip class III/IV was observed in 45 patients. Dissection was observed in 25, under deployment in 74, and/or malposition in 24 patients. Thrombus aspiration was performed in 97, plain old balloon angioplasty in 76, and stenting in 22 patients. Final TIMI III flow was achieved in 32 patients. During a mean hospital stay of 4.93 ± 2.46 days, the mortality rate was 27%.

Conclusion: In-hospital mortality after primary PCI was observed in more than 1/4th of the patients with STEMI due to ST undergoing primary PCI.

Disclaimer

None to declare.

1. Introduction

Emergency percutaneous coronary revascularization, with or without stenting, is the preferred and recommended mode of management for patients with ST-segment elevation myocardial infarction (STEMI). Worldwide adoption of primary percutaneous coronary intervention (PCI) has significantly improved the survival and outcomes of these patients.[1] Owing to the advancements in the mechanical and pharmacological aspects of the percutaneous interventions, outcomes have been and enhanced for most STEMI patients. Still, stent thrombosis (ST) remained a relatively rare but dreaded complication of PCI.[2] The introduction of drug-eluting stents (DES) was undoubtedly a milestone however, ST was one of the main safety concerns with 1st generation DES this has been addressed in the 2nd generation DES with biodegradable or biocompatible durable polymer and stent platform with thinner strut.[3,4]

The pathophysiological mechanism behind the development of ST is not very clear, and multiple triggers have been postulated; lesion morphology and characteristics, adherence with dual antiplatelet therapy (DAPT), presence of diabetes, and stent type and design are the commonly reported factors.[5,6] The histopathological examinations of thrombus samples revealed heterogeneity in its composition, erythrocytes, fibrin/fibrinogen fragments, and platelet-rich thrombus were widely observed in histopathological

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findings.[7] While stent under-expansion, struts malapposition, isolated uncovered struts, coronary evagination, neoatherosclerotic lesions, and neointimal hyperplasia are reported to be the commonly observed mechanisms of ST as reported by an optical coherence tomographic evaluation of confirmed ST in a multicenter registry.[8] Another registry-based study reported high platelet reactivity in more than 2/3rd of the patients with STEMI due to ST.[9]

ST can be categorized as acute, sub-acute, or late ST based on the timing of incidence after stent deployment and as definite, probable, or possible based on certainty and evidence.[10] A typical clinical manifestation of ST is STEMI however, due to differences in its pathophysiology, compared to de novo STEMI, it carries a significantly higher mortality rate ranging from 20% to 40%.[11,12] STEMI due to ST is reported to be associated with an increased risk of microvascular obstruction and distal embolization due to ruptured atheromatic plaque in patients with neoatherosclerosis, while, thrombogenicity of uncovered stent struts, disturbance of blood flow, and polymer materials can be possible underlying factors for patients without underlying plaque pathology.[13] Despite the pathophysiological, histopathological, and clinical distinction of STEMI due to ST, guidelines are not very clear about the optimal management strategy for this high-risk group of patients.[14] Thrombus aspiration, balloon angioplasty and stenting in some cases have been adopted for the management of STEMI due to ST.[8,14]

Therefore, in this study, we aimed to assess the clinical and procedural characteristics and the in-hospital outcomes of patients undergoing primary PCI for STEMI caused by acute, sub-acute, or late ST.

2. Methods

A sample of hundred consecutive patients who presented with STEMI due to ST at tertiary care hospital were included in this study. This study was conducted from April 2021 to March 2022 after approval from the institution's ethical review committee (ERC-60/2020). Study inclusion criteria were; both male and female adult patients (≥18 years), diagnosed with STEMI due to acute, sub-acute, or late stent thrombosis and undergoing primary PCI. Patients who refused to participate or did not undergo primary PCI for any reason were excluded from the study.

The angiographic evidence of a presence of flow-limiting thrombus or total vessel occlusion (thrombolysis in myocardial infarction (TIMI) flow grade 0 to II) at the site of the previous stent implant was taken as ST. ST was further categorized as acute, sub-acute, or late ST based on the time of onset after the stent deployment i.e. within 24 h, up to 30 days, or beyond 30 days, respectively.[10] The drug eluting stent (resolute integrity, paclitaxel or zotarolimus) are implanted in most patients. Diagnosis of STEMI was made based on the history of chest pain at presentation (>20 min) and electrocardiographic (ECG) changes. All patients diagnosed with STEMI presented within 12 h of symptoms were immediately taken to the catheterization laboratory. ST was confirmed based on angiography, and primary PCI procedures were performed by on call interventional cardiology team. During most (>90%) of the procedures, infusion of Glycoprotein IIb/IIIa (GP IIb/IIIa) inhibitors as administered. Thrombus aspiration (export), plain balloon angioplasty (POBA), and/or stenting were performed at the operator's discretion. However, pre-, peri-, and post-procedure management of all the patients was according to standard care with the administration of the recommended dose of unfractionated heparin, DAPT, and/or glycoprotein inhibitors (IIb/IIIa). Post-procedure all patients were prescribed with Asprin (100 mg) and Ticagrelor (90 mg BD) in 50 patients, and the remaining were prescribed with Clopidogrel. All patients were prescribed Statin (40 mg single dose of Atorvastatin).

Enrolled patients were observed for in-hospital mortality. Patients' clinical and procedure-related details were collected on a structured proforma. Details of previously deployed stents were obtained from patients' files and hospital records, this included type of stent and length and diameter of the stent. Mechanisms of ST, such as dissection, under deployment, or malposition, were reported by the primary operator on conventional angiogram. Patients' adherence with DAPT (aspirin, clopidogrel, or ticagrelor) and stains was taken. Analysis of data was carried out with the help of IBM SPSS version 21. Variables of continuous nature were summarized as either mean ± standard deviation (SD) or median interquartile range (IQR) and compared by in-hospital mortality status with the help of an appropriate independent sample t-test or Mann–Whitney U test. Similarly, variables of categorical nature were summarized as frequency (%) and compared by in-hospital mortality status with the help of the Chi-square test/Likelihood ratio/Fisher's Exact test with a 5% level of significance.

3. Results

A total of 100 patients were included in this study, out of which male patients were 69 and mean age was 58.9 ± 7.78 years. ST was categorized as acute in 40 patients, sub-acute in 53, and late in the remaining 7 patients. Killip class III/IV was observed in 45 patients. Dissection was observed in 25, under deployment in 74, and/or malaposition in 24 patients. Thrombus aspiration was performed in 97, plain old balloon angioplasty in 76, and stenting in 22 patients. The intra-aortic balloon pump (IABP) was placed in 21 (21%) patients. Final TIMI III flow was achieved in only 32 patients. During mean hospital stay of 4.93 ± 2.46 days, mortality rate was 27% (27). Mortality was due to arrhythmias in 15 patients, heart/pump failure in 10 patients, mechanical complications in one patient, and one patient died due to multi-organ failure. Distribution of and comparison of ' clinical and procedure-related details by survival status after primary percutaneous coronary intervention for stent thrombosis-induced ST-segment elevation myocardial infarction are presented in Table 1.

4. Discussion

The primary PCI is a recommended management strategy for patients with de novo STEMI, but the efficacy of primary PCI in ST-induced STEMI is unclear. Minimal data are available in this regard, especially from the developing world. Therefore, this study evaluated the in-hospital mortality rate and clinical profile of patients undergoing primary PCI for STEMI due to ST. The in-hospital mortality rate after primary PCI was observed to be 27%. The clinical outlook of the ST-induced STEMI was predominantly male, killip III/IV at presentation.

Mostly hypertensive, nearly 2/3rd diabetics, nearly half smokers, mostly with high thrombus burden (IV/V), multi-vessel disease, and mostly left main or anterior territory involvement.

Although, local evidence on the incidence of ST after primary PCI is very limited. Some of the recent studies from our population reported the incidence of acute or sub-acute ST as 4.9% and 5.8%.[15,16] However, the incidence of ST reported from various other regions is ranging from 2% to 3.7%,[17–20] which is much lower than that reported for our population. In this study 93% of the cases were found to have acute or sub-acute stent thrombosis, similar to our observations Singh K et al[21] also reported 79% of ST cases within first 24 h of the stent deployment.

Contrary to our findings, very late ST was observed in 85% of the patients in a study of 128 consecutive patients with ST by

Table 1
Distribution of and comparison of patients' clinical and procedure-related details by survival status after primary percutaneous coronary intervention for stent thrombosis induced ST-segment elevation myocardial infarction.

Characteristics	Total	In-hospital Outcome		*P*-value
		Survived	Mortality	
Total (N)	**100**	**73 (73%)**	**27 (27%)**	**-**
Gender				
Male	69% (69)	68.5% (50)	70.4% (19)	0.857a
Female	31% (31)	31.5% (23)	29.6% (8)	
Age (years)	58.9 ± 7.78	58.71 ± 6.58	59.41 ± 10.5	0.750b
Chest pain to ER time (h)	2 [1.65–3]	2 [1.3–3]	3 [2–5]	0.107c
ER To Lab time (minutes)	60 [30–72.5]	60 [30–68]	60 [30–120]	0.614c
Killip Class				
I	14% (14)	12.3% (9)	18.5% (5)	<0.001a
II	41% (41)	53.4% (39)	7.4% (2)	
III	32% (32)	24.7% (18)	51.9% (14)	
IV	13% (13)	9.6% (7)	22.2% (6)	
Co-morbid conditions				
Hypertension	80% (80)	83.6% (61)	70.4% (19)	0.143a
Diabetes Mellitus	72% (72)	69.9% (51)	77.8% (21)	0.434a
Smoking	47% (47)	47.9% (35)	44.4% (12)	0.755a
Family history of CAD	5% (5)	4.1% (3)	7.4% (2)	0.610d
Stent Thrombosis				
Acute	40% (40)	38.4% (28)	44.4% (12)	0.099e
Sub-acute	53% (53)	52.1% (38)	55.6% (15)	
Late	7% (7)	9.6% (7)	0% (0)	
Thrombus grade				
I	1% (1)	0% (0)	3.7% (1)	0.396e
II	3% (3)	2.7% (2)	3.7% (1)	
III	17% (17)	16.4% (12)	18.5% (5)	
IV	35% (35)	32.9% (24)	40.7% (11)	
V	44% (44)	47.9% (35)	33.3% (9)	
Stent type				
Drug-eluting stent	99% (99)	98.6% (72)	100% (27)	>0.999d
Bare metal stents	1% (1)	1.4% (1)	0% (0)	
Total length of stent	27.64 ± 7.12	27.96 ± 7.3	26.78 ± 6.66	0.464b
Average diameter of stent	3.17 ± 0.32	3.16 ± 0.3	3.19 ± 0.36	0.753b
Causes of stent thrombosis				
Dissection	25% (25)	27.4% (20)	18.5% (5)	0.363a
Under deployed	74% (74)	74% (54)	74.1% (20)	0.992a
Malaposition	24% (24)	24.7% (18)	22.2% (6)	0.800a
Post dilation	87% (87)	89% (65)	81.5% (22)	0.329a
Medications Adherence				
Aspirin	100% (100)	100% (73)	100% (27)	–
Clopidogrel	96% (96)	94.5% (69)	100% (27)	0.572a
Statin	99% (99)	98.6% (72)	100% (27)	>0.999a
Number of involved vessels				
Single vessel disease	9% (9)	6.8% (5)	14.8% (4)	0.004e
Two vessel disease	49% (49)	58.9% (43)	22.2% (6)	
Three vessel disease	42% (42)	34.2% (25)	63% (17)	
Culprit Vessel				
Left main	4% (4)	0% (0)	14.8% (4)	0.003e
Left anterior descending artery	54% (54)	52.1% (38)	59.3% (16)	
Right coronary artery	25% (25)	30.1% (22)	11.1% (3)	
Left circumflex artery	17% (17)	17.8% (13)	14.8% (4)	
Management				
Export	97% (97)	95.9% (70)	100% (27)	0.561a
Balloon	76% (76)	72.6% (53)	85.2% (23)	0.191a
Stenting	22% (22)	26% (19)	11.1% (3)	0.110a
^Length of Stent	13.95 ± 3.23	14.21 ± 3.41	12.33 ± 0.58	0.362b
^Diameter of Stent	3.06 ± 0.28	3.04 ± 0.28	3.17 ± 0.29	0.474b
TIMI (thrombolysis in myocardial infarction) flow grade				
0	4% (4)	1.4% (1)	11.1% (3)	<0.001e
I	15% (15)	4.1% (3)	44.4% (12)	
II	49% (49)	50.7% (37)	44.4% (12)	
III	32% (32)	43.8% (32)	0% (0)	
Length of stay (days)	4.93 ± 2.46	5.71 ± 2.31	2.81 ± 1.39	<0.001b

a = Chi-square test, b = independent sample t-test, *c* = Mann–Whitney U test, d = Fisher's Exact test, e = Likelihood ratio test.
^based on patients in whom new stents were deployed.
CAD = coronary artery diseases, ER = emergency room, TIMI = thrombolysis in myocardial infarction.

Konstantinou K et al.[22] This study further reported that manual thrombus aspiration is not associated with improved reperfusion or long-term outcomes in contemporary PCI setting for ST patients.[22] Similarly, Noaman S et al[23] also reported very late ST in 64% of the cases. Compared to de novo STEMI, patients with STEMI due to ST were found to have a higher prevalence of dyslipidemia, hypertension, and diabetes mellitus, and outcomes after primary PCI in patients with STEMI due to ST were comparable to the outcomes of de novo STEMI.[23] However, in-hospital mortality in our study was observed to be much higher than the conventionally reported mortality rate after primary PCI. One of the possible reasons was the higher distribution of acute or sub-acute ST as a recent systematic review and meta-analysis reported significantly worst short- and long-term clinical outcomes among patients with early ST compare to late or very late ST.[24] Supporting these findings He C et al[25] also reported no significant differences in outcomes after late stent thrombosis compared to de novo lesion. However, contrary to the observations of these studies, Ergelen M et al[26] reported significantly higher in-hospital cardiovascular mortality among ST induced STEMI patients (10.2% vs. 5.3%; $p = 0.02$) as compared to de novo lesion, hence, less effectiveness of primary PCI for STEMI due to ST was concluded by this study.[26] A Japanese registry based study by Ohno Y et al[27] had similar observations regarding increased risk of adverse outcomes and complications and recurrent ST among acute coronary syndrome (ACS) patients presented with ST.[27] Similarly, Kim MC et al[28] reported death or recurrent ST in nearly 1/4th of the patients with ST during first year follow-up after ST event. Increased risk of adverse outcomes and failure to achieve the optimal TIMI flow after primary PCI in patients with ST can be attributed to several factors such as high burden of co-morbidities, older age, hemodynamically unstable presentation (high killip class), multi-vessel diseases, and high thrombus burden.

The present study has certain limitations, starting with inherent observational design and single-center coverage. Lack of follow-up for assessing short- or long-term outcomes is another main limitation. The intracoronary imaging is current gold standard, but our center is a high-burden center which provide free of cost services to all types of cardiovascular disease patients, hence, intracoronary imaging was not feasible in all patients and the mechanisms of ST was evaluated based on conventional angiogram which might be a less sensitive modality of the assessment. Finally, multivariable analysis for assessing associated factors with in-hospital mortality was not feasible due to the small sample size. Further longitudinal and randomized studies are required to access the primary efficacy of PCI in patients with STEMI due to ST.

5. Conclusion

In conclusion, for patients presenting to the hospital with STEMI due to ST and treated with primary PCI, the in hospital mortality was observed in more than 1/4th of the patients. Dissection, under deployment, and/or malposition were the commonly observed mechanism behind ST. Increased risk of adverse outcomes can be attributed to the high co-morbidities, older age, hemodynamically unstable presentation (high Killip class), multi-vessel diseases, and high thrombus burden. Further longitudinal and randomized studies are required to access the efficacy of primary PCI in patients with STEMI due to ST.

Declarations

Competing interest

All authors have no conflict of interest to disclose.

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