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OUTCOME OF STENT THROMBOSIS IN PATIENT UNDERGOING PERCUTANEOUS CORONARY INTERVENTION

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Abstract

OBJECTIVE

To determine the outcomes of stent thrombosis in patient undergoing percutaneous coronary intervention.

METHODOLOGY

A cross-sectional study was conducted at NICVD from August to December 2024 on 220 patients aged 18–65 years experiencing recurrent MI or having clinical suspicion of stent thrombosis. A non-probability purposive sampling method was applied, and PCI was done to assess the stent thrombosis. Demographic, comorbidity and outcome data were extracted. SPSS (version 26) was used for statistical analysis, and $p<0.05$ was considered statistically significant.

RESULTS

The mean age of the participants was found to be (54.31±12.52 years). The proportion of male patients was noted as 84.5%. The rate of mortality was documented in 21.81% of patients. The mean age of patients who died was significantly higher (58.42 ± 9.37 years) than the mean age of patients who did not die (53.16 ± 13.06 years, $p = 0.010$). No significant associations were found for stent type (DES 23.3% vs. 18.9%, $p=0.288$), culprit artery ($p=0.114$), ejection fraction (44.40% vs. 45.70%, $p=0.277$), or length of hospital stay (4.42±2.07 vs. 4.23±2.01 days, $p=0.579$).

CONCLUSION

It is to be concluded that insignificant associations were documented between mortality and factors such as stent type, culprit artery, or length of hospital stay. Although stent technology is right now getting better, the improved mortality, underscore the importance of better blood sugar monitoring and adjusted management. The present findings require further study to optimize prognosis and minimize complications in high-risk PCI patients.

INTRODUCTION

Emergency percutaneous coronary revascularization, with or without stenting, is the preferred method 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 findings [7]. While stent under-expansion, stent malposition, isolated uncovered struts, coronary evagination, neo-atherosclerotic 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].

According to the Academic Research Consortium criteria, ST can be stratified into early stent thrombosis (EST), occurring within 30 days after index PCI, late stent thrombosis (LST), occurring from 30 days to 1 year after index PCI, and very late stent thrombosis (VLST), occurring more than 1 year after index PCI [10]. Recently, several studies investigated the associations between the timing of ST occurrence and the clinical outcomes of ST, but the results were inconsistent [11-15].

The aim of our study is to determine the outcomes in patients with stent thrombosis undergoing percutaneous coronary intervention. Stent thrombosis is a rare complication after PCI. However, it is associated with poor prognosis. Various studies have been done at international level which shows inconsistent results [12-15]. The possible reason of inconsistency is results may be due to difference in type of stent thrombosis i.e. early or late. Findings of our study will help the cardiologist for developing some intervention in order to reduce risk of stent thrombosis and also help in risk stratification of patient which helps in timely counseling to the family regarding prognosis. Furthermore, findings of our study will open the pathway for future research regarding identification of factors associated with poor prognosis after PCI in patient with stent thrombosis.

METHODOLOGY

A cross-sectional study was carried out from August 2024 to December 2024 to elucidate the causes of stent thrombosis post-PCI on around 220 patients at National Institute of Cardiovascular Diseases (NICVD). Non-probability purposive sampling was used to include patients of age 18–65 years of both genders, presenting with recurrent myocardial infarction (MI) or present with suspected stent thrombosis. Exclusion criteria were patients with history of renal failure, chronic liver disease, arrhythmias, thyroid disorders, or pregnancy. All participants provided written informed consent, and enrollment occurred in the emergency department. For the patients with suspected stent thrombosis, PCI was performed to solidify the diagnosis.

Non-ST-elevation myocardial infarction (NSTEMI) was defined as an acute ischemic event resulting in myocyte necrosis, diagnosed in patients with retrosternal chest pain lasting longer than 20 minutes, radiating to the left arm or shoulder, and induced by effort or emotional stress. ECG ST depression (≥2 mm), transient ST elevation (≥2 mm), prominent T-wave inversions or drawn cardiac troponin was defined (> 0.05 ng/dL in males and > 0.03 ng/dL in females). Presence of ST-elevation

myocardial infarction (STEMI) among patients with reports of chest pain lasting more than 20 minutes, whereby an ECG evidenced ST elevation in two or more contiguous lead or a new left branch bundle block. ST elevation was defined as J-point elevation >2 mm in leads V2 and V3 and ≥1 mm in all other leads. Stent thrombosis was defined if thrombus formation located in implanted stent was identified by angiography.

Demographic characteristics (age, sex, BMI, smoking status, past history of diabetes, hypertension and dyslipidemia, family history of cardiovascular disease) were also obtained. Dyslipidemia (yes/no) was defined as total cholesterol >200 mg/dL, triglycerides >150 mg/dL, LDL >100 mg/dL, or HDL <40 mg/dL for men and <50 mg/dL for women. Patients had received anti-diabetic treatment for at least 1 year with a known history of diabetes mellitus. We regarded subjects to have a history of hypertension if they had a physician-diagnosed disease and received anti-hypertensive therapy for more than 1 year.

The outcomes studied were the length of stay in the hospital expressed as mean days from admission to discharge, and in-hospital mortality. We performed data entry and analysis using SPSS version 26. Mean and standard deviation for quantitative variables and frequency and percentage for qualitative variables were calculated by using descriptive statistics. A Chi-square test was performed to analyze the associations and a P-value < 0.05 was considered significant.

RESULTS

The baseline characteristics of patients with stent thrombosis and the associated mortality. The mean age of patients who died was significantly higher (58.42 ± 9.37 years) than the mean age of patients who did not die (53.16 ± 13.06 years, p = 0.010). In the mortality group, 23.7% were males, and 76.3% were males among the non-mortality group (p-value = 0.089). Forty-two (84.0%) patients with mortality had significant more hypertension than eight (16.0%) patients without mortality (p = 0.000) In respect to current smoking status, there was no significant difference (23.0% mortality group vs. 77.0% non-mortality group; p = 0.779). The mean weight and BMI were similar between groups (74.58 ± 9.37 kg vs 73.47 ± 10.49 kg, p = 0.507 and 25.83 ± 3.84 kg/m^2 vs 26.01 ± 3.74 kg/m^2; p = 0.761, respectively). The mortality group had an 18.6% incidence while the non-mortality group had an incidence of 81.4% (p = 0.490) of a previous history of coronary artery disease or myocardial infarction. Likewise, previously percutaneous coronary intervention was present in 15.4% of the death group and 84.6% of the no death group (p=0.135). (TABLE 1)

Mortality was compared with ejection fraction (EF), culprit artery, length of hospital stay (LOHS), and type of stent, using bivariate analysis with a threshold for statistical significance of $p < 0.05$. The mean ejection fraction was reported as 44.40 ± 8.21% in case of mortality and 45.70 ± 7.08% in absence of mortality with non-statistically significant differences (p = 0.277). For drug-eluting stents (DES), mortality was noted in 23.3% and in 18.9% of bare-metal stent (BMS) patients, not significantly different between groups (p = 0.288). Examining the culprit artery, mortality was most common in the right coronary artery (RCA) (26.0%), followed by the left anterior descending artery (LAD) (24.3%), and left circumflex artery (25.9%). The correlation of mortality was minimal with left main artery (8.7%). However, these differences were not statistically significant (p=0.114). The mean length of hospital stay was marginally longer in patients with mortality (4.42 ± 2.07 days) than the patients without mortality (4.23 ± 2.01 days); the difference was not significant (p = 0.579). (TABLE 2)

The analysis of predictors of stent thrombosis with mortality revealed heart failure was present in 17.6% of patients with mortality compared to 82.4% without mortality, with an odds ratio (OR) of 0.690 (95% CI: 0.33–1.42, p = 0.316). ST-segment deviation was observed in 22.4% of patients with mortality and 77.6% without mortality, with an OR of 1.119 (95% CI: 0.56–2.22, p = 0.748). Acute coronary syndrome occurred in 22.2% of patients who died and in 77.8% of those who survived (OR: 1.190 (95% CI:0.45–3.09, p = 0.464)). 21.7% of patients with mortality and 78.3% without mortality had elevated cardiac biomarkers (OR: 0.990; 95% CI: 0.52–1.87, p = 0.976). Furthermore, PCI for multivessel CAD was observed in 21.2% of patients with mortality and in 78.8% without mortality, OR (95% CI) 0.950 (0.47–1.91, p=0.887). TABLE 3)

TABLE 1: Baseline Features of Patients Who Had Stent Thrombosis with Mortality			
Features	**Mortality**		**P-Value**
	Yes (n=48)	No (n=172)	
Mean Age (years)	58.42±9.37	53.16±13.06	0.010
Male, n (%)	44 (23.7)	142 (76.3)	0.089
Hypertensive, n (%)	41 (32.0)	87 (68.0)	0.000
Current Smoker, n (%)	28 (23.0)	94 (77.0)	0.779
Mean Weight (kg)	74.58±9.37	73.47±10.49	0.507
Mean BMI (kg/m^2)	25.83±3.84	26.01±3.74	0.761
Prior History of CAD/MI, n (%)	11 (18.6)	48 (81.4)	0.490
Prior History of PCI, n (%)	10 (15.4)	55 (84.6)	0.135

TABLE 2: Comparison of Mortality with Ejection Fraction, Culprit Artery, LOHS & Type of Stent				
Features		**Mortality**		**P-Value**
		Yes (n=48)	No (n=172)	
Ejection Fraction (%)		44.40±8.21	45.70±7.08	0.277
Type of Stent	DES	34 (23.3)	112 (76.7)	0.288
	BMS	14 (18.9)	60 (81.1)	
Culprit Artery				
	LAD	18 (24.3)	56 (75.7)	0.114
	RCA	19 (26.0)	54 (74.0)	
	Left main	4 (8.7)	42 (91.3)	
	Left Circumflex	7 (25.9)	20 (74.1)	
Length of Hospital Stays (days)		4.42±2.07	4.23±2.01	0.579

LAD (Left Anterior Descending Artery), RCA (Right Coronary Artery), DES (Drug-Eluting Stent), BMS (Bare-Metal Stent)

TABLE 3: Factors Associated Among Predictors of Stent Thrombosis with Mortality				
Factors	**Mortality**		**OR (C.I.)**	**P-Value**
	Yes (n=48)	No (n=172)		
Heart Failure (n, %)	12 (17.6)	56 (82.4)	0.690 (0.33 ~ 1.42)	0.316
ST-segment deviation (n, %)	33 (22.4)	114 (77.6)	1.119 (0.56 ~ 2.22)	0.748
Acute Coronary Syndrome, n (%)	42 (22.2)	147 (77.8)	1.190 (0.45 ~ 3.09)	0.464
Elevated Cardiac Biomarkers, n (%)	25 (21.7)	90 (78.3)	0.990 (0.52 ~ 1.87)	0.976
PCI for Multivessel CAD, n (%)	14 (21.2)	52 (78.8)	0.950 (0.47 ~ 1.91)	0.887

PCI (Percutaneous Coronary Intervention), CAD (Coronary Artery Disease), OR (Odd Ratio)

DISCUSSION

Stent thrombosis (ST) is the most serious complication in patients with percutaneous coronary intervention (PCI) and stent implant [16]. This is the development of a thrombus within a coronary stent resulting in a very high risk of adverse cardiovascular events such as myocardial infraction (MI) and death. Knowledge of stent thrombosis results is important to optimize patient managements and decrease their incidence [17]. Stent thrombosis is a multifactorial phenomenon that can be determined by the characteristics of the stent, patient, procedural aspects or post-procedural approaches.

A key strength in the understanding of ST outcomes is the long-standing research that has been performed to elucidate its pathophysiology and risk factors [18]. There has been the transition from bare-metal stents (BMS) to drug-eluting stents (DES), which has greatly decreased ST incidence [19]. Drug-eluting stents (DES) are coated with a drug that inhibits neointimal hyperplasia and restenosis, and so have been linked to reduced rates of stent thrombosis. Nonetheless, late and very late stent thrombosis is still a concern even with DES, especially when there is early interruption of dual antiplatelet therapy (DAPT) or other comorbidities such as diabetes or chronic kidney disease [20]. Innovations in stented devices, including bioresorbable scaffolds and stents with enhanced coatings may represent a step forward in the reduction of ST and improved outcomes in patients who undergo PCI.

Stent thrombosis may be associated with varying clinical outcomes, but they are generally poor. ST results to acute coronary syndrome (ACS) and may present as ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI) or instability angina. Acute ST can lead to sudden cardiac death in extreme cases [21]. The timing of ST has an impact on the outcome, early ST (within 30 days after stent implantation) has a significantly worse mortality than does late ST, which generally manifests after one year of stent implantation [22]. Moreover, stent thrombosis can also result in revascularization procedures like coronary artery bypass grafting (CABG), and increase clinical complexity for patients.

In our study, mortality was noted in 21.81% and the mean length of hospital stay was 4.42±2.07 days (p=0.579). Another study reported in-hospital mortality in 26.1% of patients [12]. Kumar M, et al stated a mortality rate of 27%, with a hospital stay of 4.93±2.46 days [14]. In another study, in-hospital mortality was reported in 22.2% [23] whereas Braun E, et al noted in-hospital mortality in 14.3% and the length of hospital stay of 6 days [24]. The study of Juned H, et al found a mortality rate of 9.6%, and length of hospital stay of 8.04±0.963 [25].

Although there has been substantial progress in the understanding and management of stent thrombosis, significant shortcomings exist in the current approaches. Firstly, there continues to be an increased risk of stent thrombosis in high-risk patients, in particular in those with a clinical history of acute coronary syndrome or diabetes. Patients are still at risk even with the use of DES and improved stent technology. Another limitation is the lack of know- and information between precise biological mechanisms that can elicit thrombosis, making global prevention strategies a challenge. Another unresolved issue is the optimal duration of DAPT following PCI. DAPT might lower stent thrombosis risk in the short term but may also increase the risk of bleeding complications in the intermediate and long-term, especially in the elderly or in those with previous bleeding disorders. The clinical challenge of balancing the risk of bleeding against the benefit of thrombosis prevention continues to be a major concern.

Also, stent thrombosis can be attributed to less than optimal procedural technique, such as under-deployment of stent or wrong stent sizing. These mechanical defects might predispose to stent malapposition or under expansion, leading to thrombosis. Although procedural accuracy may be improved via modern imaging techniques, e.g. intravascular ultrasound (IVUS) and optical coherence tomography (OCT), such modalities are either not routinely employed in practice or not universally available, especially in settings equipped with limited resources. The wide spectrum of procedural techniques and patient populations also makes it difficult to identify and prevent stent thrombosis.

In light of these difficulties, we may suggest some tailored strategies to improve the outcome of patients treated by PCI and decrease the risk of stent thrombosis. First, there is an individual approach to DAPT management. It has been recommended that the DAPT duration should be individualized based on risk factors including diabetes, prior coronary event, and those at higher bleeding risk. Identifying high-risk patients early on will enable clinicians to use aggressive novel pharmacological strategies or interventions. Second, the continuous improvement of stent technology, for example, stents you have a better endothelial healing property or bio-absorbable stents should be tested. Challenges for future clinical trials must be to better define characteristics of thrombus-resistant stents. Also, improving procedural methods and encouraging advanced imaging modalities to prevent mechanical mechanisms of thrombosis by optimizing stent deployment.

Despite remarkable advances in the pathophysiological characterization and management of stent thrombosis in patients undergoing PCI, considerable challenges remain. Patient outcomes continue to be affected by the interaction of biologic, technical, and clinical factors. An approach that combines personalized therapy with technological advances and procedural standards is likely to be the secret to further reducing the incidence and improving the prognosis of stent thrombosis.

CONCLUSION

It is to be concluded that insignificant associations were documented between mortality and factors such as stent type, culprit artery, or length of hospital stay. Although stent technology is right now getting better, the improved mortality, underscore the importance of better blood sugar monitoring and adjusted management. The present findings require further study to optimize prognosis and minimize complications in high-risk PCI patients.

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