PREVALENCE OF EARLY SUBACUTE STENT THROMBOSIS AFTER PRIMARY PCI IN STEMI PATIENTS

Running Head: Interventional Cardiology, Angiographic representation in PCI, Stent

ABSTRACT

Background

In-hospital mortality data of patients who have been experiencing ST (stent thrombosis) shows 7.9% statistics for acute stent thrombosis (AST) within 24 hours of PCI, Sub-Acute Stent Thrombosis (SAST) occurs during 30 days of Percutaneous Coronary Intervention (PCI), Late Stent Thrombosis (LST) found 3.8% within the first year after PCI, and Very Late Stent Thrombosis found 3.6% in 1 year after PCI. From our study, we found out the frequency of Early Sub Acute Stent Thrombosis in STEMI Patients after Primary PCI.

Method

This descriptive-analytic case series was conducted in 06 months data collection time in the NICVD Karachi Pakistan. All the study sample were computed and analyzed by using SPSS (20.0 Version). Mean ± standard was be calculated for age, duration of surgery, weight, height, and BMI. Chi-square test by using $P \leq 0.05$ as significant on 95% Confidence Interval were used for other variables.

Result

Out of 142 patients of 55.56 years of mean age was evaluated with standard deviation ±12.24 and BMI mean was 27.56±6.28. The mean ± SD duration of surgery was 33.48±9.26. The mean height of study sample was 1.76 ± with S.D 0.48 beside, mean weight was 58.25 ± with S.D 8.48.

Conclusion

It is to be concluded that the frequency of EAST after primary PCI was found to be significant. Patients presenting with STEMI were at high risk of EST whether they were hemodynamically unstable or they had ACS stenting of multivessel coronary diseases.

Key Words

PCI, Stent Thrombosis (ST) , STEMI, Acute Stent Thrombosis (AST), Subacute ST(SAST) , Late ST(LST), Very Late ST(VLST)

INTRODUCTION

Acute stent thrombosis (AST) is an uncommon nevertheless very disastrous obstacle of percutaneous coronary intervention (PCI) events, which might happen even with new procedural methods and dual- antiplatelet therapy [1]. In 1977 the first balloon percutaneous intervention (PCI) introduced later in 1986 was an era of stent implantation turned the picture in PCI. The purpose of these stents is to reduce thrombosis and restenosis with other complications. Regardless of foremost apprehension of this extensive passion for stents globally, new ages of drug-eluting stents (DESs) declare as a real age to rule. Virchow was the first person who portrayed in his study the most acknowledged component for stent thrombosis (ST). Although his study exceptionally connected to an extremely realized set of three previously portrayed components i.e., pulmonary embolism, vascular injury after stent placement, and hypercoagulability state. All these factors are caused either by platelet activation because of disappointment in anticoagulation or antiplatelet treatment, balance blood deterioration by vessel dissection or malposition and sub expansion of stent struts [2]

In-hospital mortality data of patients who have been experiencing ST (stent thrombosis) shows 7.9% statistics for acute stent thrombosis (AST) within 24 hours of PCI, Sub-Acute Stent Thrombosis (SAST) occurs during 30 days of Percutaneous Coronary Intervention (PCI), Late Stent Thrombosis (LST) found 3.8% within the first year after PCI, and Very Late Stent Thrombosis found 3.6% in 1 year after PCI. [3]. Besides the effective agent which causes ST is still not known. However, a variety of effective stent design for of ST are available to reduce the comorbidities such as patient's consistency with APT, injury morphology, diabetes, or high on-treatment platelet reactivity. Moreover, all these components advancing the ST shift with expanding time from PCI. Today, the utilization of intravascular imaging has uncovered extra

reasons for ST. Under the expansion and malposition give off an impression of being driving reasons for AST and SAST. Besides, noeatherosclerosis has been demonstrated as a significant supporter of late ST (VLST) [4]

In 2016, two randomized trials were conducted to find out the reason behind early ST and mortality after PCI. They did the trial between procedural antithrombotic therapy and found out the amplified thread of acute ST with bivalirudin which was not converted inside 30 days of the mortality period. In contrast, a drastically low death rate was observed in whom acute or subacute ST occurred when treated with Bivalirudin rather than Heparin ± GPI. [5] Geraud S and his fellows in 2018 using optical coherence tomography (OCT) and analyzed the mechanism of stent thrombosis. They identify underlying morphological abnormality of ST in 97% of their study cases. The other major findings were malapposition and noeatherosclerosis rupture as the major reasons for LST and VLST as well as an important mechanism for AST and ASAT. Their study shows Bare metal stent (BMS) and Drug-eluting stent (DES) with no significant role in the allotment of ST types [6]

Another study in 2018 was done to find out the achievable function of hyperhomocysteinemia, high on-aspirin platelet reactivity, and inflammation in SAST. The investigation provided proof impacts of aspirin therapy diverge amongst patients going through PCI. No affiliation could not be set between a low reaction to aspirin and a repeat of SAST. There were also no distinctions in the high on-aspirin platelet reactivity (HPR) rates among ST and non-ST patients. They recommended that SAST was presumably not identified with diminished aspirin responsiveness yet related to expanded platelet affectability to adenosine diphosphate. However, a connection between the raised plasma concentrations of homocysteine and SAST was concluded result of their study [7] Case report of Heparin-induced thrombocytopenia (HIT) on recurrent arterial

thrombotic events was exhibits in 2017 argatroban as fundamental, followed by oral anticoagulant treatment was vital to anticipate thromboembolic complications. Their study aim to rule out HIT in coronary artery thrombosis during PCI especially in earlier heparin patients [8]. Similar case study before 2017 reported the reason for subacute stent thrombosis (SAST) was severe coronary vasospasm via DES. The study report the case of old man of 67 years of age arrive hospital with a complaint of chest pain, and later on diagnosed with effort angina by angiogram. After follow-up PCI was done, and after the dual antiplatelet therapy DES implanted in the mid-left ascending artery (LAD). After five days of successful intervention of the procedure, sub-acute ST occurred, which lead to immediate balloon angioplasty and aspiration thrombectomy. This case is the classical example of possible exposure of DES-induced coronary vasospasm [9].

Even though a few overlapping hazard factors for ST advancement despite stent-edge spasms were assumed a significant part in ST development [10]. Retrospective data of last 3 years patients who went through PCI with stenting and had SAST to find out the multiple causes of sub-acute ST (SAST). Variables were examination of vessel and lesion morphology, assessment between angioplasty procedure in acute coronary syndrome and stable patients, percentage of patients having SAST and ST in patients getting GPIIb/ IIIa receptor blockers were significant in this work lead. They found out SAST was certifiably not an exceptional Substance and could be diminished with legitimate patient assessment and preventive Procedures. As per their findings DM, HT, Before MI Vessel size < 2.5mm, Stent length > 15mm, Type C lesion have a high frequency of SAST [11]. In 2017, to find out the risk Factor of early stent thrombosis, a study was conducted on obstructive sleep apnea (OSA). The impact of OSA on EST was observed to be clearer within the sight of less ordinary cardiovascular risk factors. Along these lines, it might

be advantageous to test cardiovascular risk factors for OSA in patients with few conformists [12]. In the light of the previous studies and literature this study was designed, to sum up, the right now accesses information on the etiology, treatment, and visualization of early subacute ST(ESAST) with an end goal to bring issues to light and to improve the clinical results of AST after PCI. From our study, we found out the frequency of Early Sub Acute Stent Thrombosis in STEMI Patients after Primary PCI.

METHOD

This descriptive-analytic case series was conducted in 06 months data collection time in the National Institute of Cardiovascular Diseases Karachi Pakistan. By using W.H.O sample size calculator using the frequency of early subacute stent thrombosis after PCI in STEMI patients (62%)10, (d)=8% was considered as margin of error, 95% confidence interval was taken into account then the estimated sample size was n=142. The individual data of patients presenting with ST-segment elevation myocardial infarction and if they had 30 minutes or longer than 30 minutes chest pain which was later on confirmed on ECG changes of ST-elevation greater than 2mm in minimum two precordial leads or greater than 1mm in limb, recommended to admission to the hospital were selected as sample size. Furthermore, all patients who were under the age of 25-75 years of either gender admitted for primary percutaneous coronary intervention (PPCI) were prospectively recorded through nonprobability consecutive sampling. This study excluded all those patients who have a Prior history of ST-segment elevation myocardial infarction, Prior history of cardiac surgery and PCI, heart failure, and was not willing to be a part of this study i.e., not gave their consent.

All those patients admitted with STEMI NICVD Karachi, meeting the inclusion criteria was asking to give consent by themselves to be part of the study. Those who would give the consent by themselves were included in the study. All these patients were undergoing primary PCI by the researcher himself under the supervision of senior registrar or consultant cardiology having > 5 years of experience and stent either drug-eluting stents (coated with medication) or bare-metal stent was being placed. These patients were observed for 24 hours for early subacute stent thrombosis. Patients indicating acute ischemia in the distribution of the target vessel (in which stent is placed), developed sudden onset of typical chest pain, in these patient's angiography was be done (relook) if it shows complete occlusion within the stented segment with evidence of thrombus, the patient was being labeled as having acute stent thrombosis.

Exclusion criteria were be followed strictly to avoid confounding variables. All the study sampled data was entered and analyzed by using SPSS (20.0 version). Mean ± standard was be calculated for age, duration of surgery, weight, height, and BMI. Gender; hypertension, smoking status, diabetes mellitus, and outcome variable i.e., early subacute stent thrombosis (yes/no) were measured in frequency and percentage. Effect modifiers were controlled through stratification of age, gender, hypertension, diabetes mellitus, BMI, height, weight smoking status, and duration of surgery to seeing its effect on outcome followed by chi-square test. $P \leq 0.05$ was taken as significant.

RESULT

Out of 142 patients mean age of 55.56 year were evaluated with standard deviation ±12.24 and BMI mean was 27.56±6.28. The mean ± SD duration of surgery was 33.48±9.26. . The mean height of study sample was 1.76 ± with S.D 0.48 beside, mean weight was 58.25 ± with S.D 8.48. Among 142 study sample, 103 (72.53%) were male and 39 (27.4%) were female. Common predictors were 68 (47.88%) were hypertensive, 65 (45.77%) were diabetic, 89 (62.6%) patients with a family history of CAD. On the other hand, smoking status was also measured with the value of 100 (70.42%) presented as current smokers and 42 (29.58%) were currently non-smokers while 99 (69.7%) patients revealed a history of ex-smoking. The frequency of early subacute stent thrombosis was found to be 4 (2.82%). The stratification of age group 25—55 and > 55 years early sub-acute stent thrombosis was found to be 0% and 2.8% respectively which shows a highly significant association between age and early subacute stent thrombosis i.e. (P=0.047). Early sub-acute stent thrombosis was found 2.1% in males and 0.7% in the female which shows the non-significant difference between gender and early sub-acute stent thrombosis. In stratification for the duration of surgery (30-45) minutes 0.7% had early sub-acute stent thrombosis and in >45 minutes 2.1% had early sub-acute stent thrombosis and found a highly significant association between them i.e. (P=0.04). For the stratification of weight group

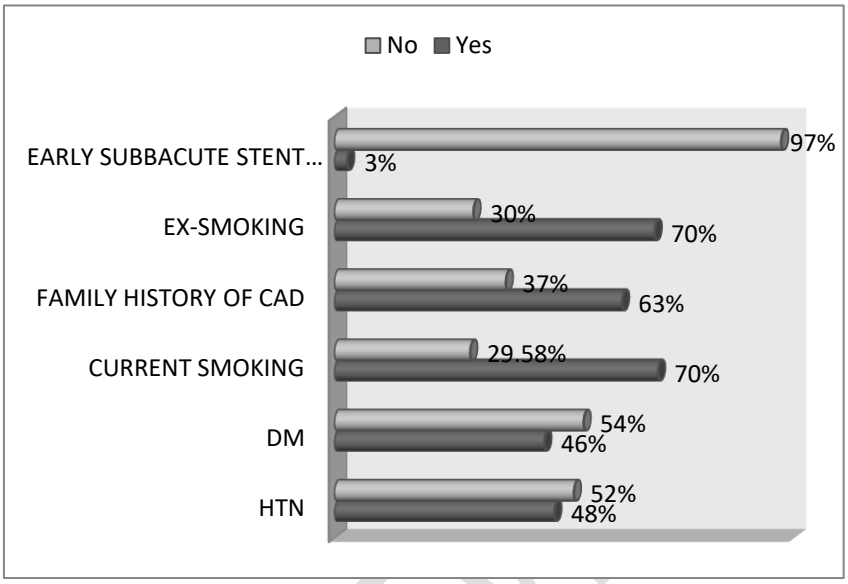

Figure 1: Early Subacute Stent Thrombosis in relation of Ex-Smoker, Family History of Coronary Artery Disease, Current Smoking, Diabetes Mellitus, and Hypertension.

45-60 and > 60 kg, early sub-acute stent thrombosis was found to be 0.7% and 2.1% respectively which shows a highly significant association between age and early sub-acute stent thrombosis i.e. (P=0.03).

	EARLY SUBACUTE STENT THROMBOSIS		P-Value
	YES	NO	
AGE GROUP IN YEARS			
25-55	0(0.0%)	75(52.8%)	0.047
>55	4(2.8%)	63(44.4%)	
GENDER			
Male	3(2.1%)	100(70.4%)	1.00
Female	1(0.7%)	38(26.8%)	
DURATION (In Minutes)			
30----45	1(0.7%)	106(74.7%)	0.046
>45	3(2.1%)	32(22.5%)	
WEIGHT (In kg)			
45---60	1(0.7%)	111(78.2%)	0.03
>60	3(2.1%)	27(19.0%)	
HEIGHT (In m)			
1.5---1.8	3(2.1%)	115(81%)	0.527
>1.8	1(0.7%)	23(16.2%)	
BODY MASS INDEX (In kg/m2)			
18.5---27	2(1.4%)	102(71.8%)	0.291
>27	2(1.4%)	36(25.4%)	
HYPERTENSION			
Yes	4(2.8%)	64(45.1%)	0.05
No	0(0%)	74(52.1%)	
DIABETES MELLITUS			
Yes	4(2.8%)	61(43.0%)	0.04
No	0(0%)	77(54.2%)	
SMOKING STATUS			
Yes	0(0%)	100(70.4%)	0.007
No	4(2.8%)	42(26.8%)	
ANTITHROMBOTIC THERAPY			
Yes	70(49.2%)	5(3.52%)	<0.00001
No	4(28.1%)	63(44.3%)	
TYPES OF STENTS			
Drug-Eluting Stent	3(2.11%)	60(42.2%)	0.653
Bare-Metal Stent	2(1.40%)	77(54.2%)	

Table 1: Stratification of age, gender, hypertension, diabetes mellitus, BMI, height, weight smoking status, antithrombotic therapy, types of the stent, and duration of surgery to seeing its

With respect to height group 1.5—1.8 and >, 1.8-meter early subacute stent thrombosis was found to be 2.1% and 0.7% respectively which shows a non-significant association between height and early sub-acute stent thrombosis i.e. (P=0.527). In stratification of BMI 18.5—27 and > 27 kg/m2, early sub-acute stent thrombosis was found to be 1.4% each which shows no significant association between BMI and early sub-acute stent thrombosis i.e. (P=0.291). Early sub-acute stent thrombosis was found at 2.8% in hypertensive and 0% in non-hypertensive patients which shows a significant association between hypertension and early sub-acute stent thrombosis i.e. (P=0.05). Early sub-acute stent thrombosis was found to be 2.8% in diabetes patients which shows a significant association and early sub-acute stent thrombosis i.e. (P=0.04). In Smoker Early sub-acute stent thrombosis was found 0% and 2.8% in a non-smoker which shows a highly significant association between smoking and early sub-acute stent thrombosis i.e. (P=0.007). Early sub-acute stent thrombosis was found 49.2% in antithrombotic therapy and 2.8% in non- antithrombotic therapy during PCI which shows a highly significant association between antithrombotic therapy and early sub-acute stent thrombosis i.e. (P=<0.00001). Early sub-acute stent thrombosis was found 2.11% in and 1.40% in a bare-metal stent which shows a non-significant difference between types of the stent and early sub-acute stent thrombosis

DISCUSSION

It is been computed that yearly around five million percutaneous coronary interventions (PCIs) are performed. Hence, stent-related intricacies, regardless of whether they happen at a genuinely low rate, are a significant worry for public wellbeing. Among these difficulties, ST, with 5–45% mortality and a 15–20% repeat rate at 5 years, is the most significant one.[13] The main findings of this study were: first, there was a significant association between hypertension and early sub-acute stent thrombosis, second, ST was associated with significantly diabetes mellitus and early

sub-acute stent thrombosis, and lastly outcome for patients with large coronary vessels show no differences when treated by bare-metal stents BMS or drug-eluting stents DES. Even after adjustment for stent diameter and stent length, BMS implantation in large coronary vessels appears equally effective as DES implantation. Patients with vessels >3.5 mm in diameter represent a low-risk population in whom BMSs confer a similarly low event rate as DESs. Their study resulted that in both group Stent thrombosis had not occur abundantly [14].

Our study supports the hypothesis that DESs do not have an edge over BMSs for larger vessels. It also stands that clinically or angiographicaly neointimal growth occurring in large vessels would given a similar degree of neointimal proliferation around a stent of any diameter. Concerning BMSs, a very rare observed case in new generation of BMS that in a ≥3.5 mm vessel would render a 2.5 mm diameter vessel even a late loss of 1 mm at long-term follow-up. This analysis would not require further intervention with no hemodynamic compensation as binary restenosis of <50% were observed on sufficient patency. The results of our study can be compared to David J Clark and colleagues who concluded that in the use of DES or BMS within 01 year or 30 days, large coronary arteries is associated with deployment of 3.5 mm diameter stents had low rate of mortality and major adverse cardiac events (MACE) [15]. The results of our study can be compared to Bryan P. Yana and colleagues who concluded that BMS implantation in large native coronary vessels ≥3.5 mm was associated with a low risk of MACE and repeat revascularization at 12 months that was comparable to DES. On 12 month mortality stats it showed no significant differences in MI (6.3 vs. 3.4%, p= 0.15), MACE (9.4 vs. 9.4%, p=0.90), TVR (3.6 vs. 4.8%, p=0.54), stent thrombosis (0.9 vs. 1.0%, p=0.88), or in patients who received DES vs. BMS [16].

Early sub-acute stent thrombosis was found 49.2% in antithrombotic therapy and 2.8% in non-antithrombotic therapy during PCI which shows a highly significant association between antithrombotic therapy and early sub-acute stent thrombosis. The studies have shown bivalirudin has a higher risk of acute stent thrombosis than heparin or planned provisional glycoprotein IIb/IIIa inhibitor GPI post-primary PCI. [5,12-13] Laura S.M et al reported that NSTEMI, younger age, stent diameter, and diabetes were discovered to be indicators of Stent thrombosis. [17] A case report was documented. In the case of sub-acute stent thrombosis after 4 days of PCI in the right coronary artery and the underlying cause of ST was stent under-sizing and malapposition. [18] Another case report explained the reasons behind ST after PCI. According to them, while addressing the technical challenges of stent under expansion could be the main reason of stent thrombosis. [19] Yuzo Kagawa et al conducted a case report and the report showed subacute stent thrombosis (SAT) happened after a drug-coated balloon (DCB) and the case highlighted the need for dual antiplatelet therapy DAPT after Drug-coated balloon DCB procedures. [20] Studies show dual antiplatelet therapy DAPT was a high risk of ST.[21] Another study conducted by Akram Saleh A et al; reported the predictors of acute stent thrombosis. Low LVEF, Younger age, ST-segment deviation, elevated blood levels of cardiac biomarkers, and heart failure were predictors of ST. This study also validates the outcome of our study.[22] Sahar et al conducted a study at a tertiary care university hospital in Karachi, Pakistan. Their data showed Diabetes mellites and hypertensive patients were observed on high risk of ST. [23] According to our study diabetes and hypertension are the risk factors for ST. Our results correlate with all national and international studies. The strength of our study was the use of consecutive sampling which compatibly suitable for inclusion and exclusion criteria, study design and sample selection.

CONCLUSION

It is to be concluded that the frequency of EAST after primary PCI was found to be significant. Patients presenting with STEMI were at high risk of EST whether they were hemodynamically unstable or they had ACS stenting of multivessel coronary diseases.

PATIENT CONSENT

Informed consent was taken from the patients or from the attendee. Senior Cardiologist or patient's cardiologist consent was also taken when extracting the data from file.

AVAILABILITY OF DATA AND MATERIAL

Confidentiality of the data was maintained by keeping the identity of the patient hidden and data is restricted to the primary investigator only. The data and material will be represented on request.

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