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PREDISPOSING FACTORS LEADING TO SUBACUTE STENT THROMBOSIS (SAT) IN PATIENTS WHO HAVE UNDERGONE PERCUTANEOUS CORONARY INTERVENTION (PCI) IN PATIENTS PRESENTING WITH ACUTE CORONARY SYNDROME

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ABSTRACT:

BACKGROUND: *Stent thrombosis is less common but life-threatening that in most cases leads to death or a big non-fatal ST-elevation myocardial infarction (STEMI). Past research data suggests multiple predisposing factors play role in sub-acute thrombosis (usually with ST elevation). However, very few studies have been conducted regarding the predisposing factors of subacute stent thrombosis (SST) in Pakistan, and hence, there is very limited knowledge regarding the trend of risk factors associated with SST.*

AIMS & OBJECTIVE: *This study will determine the predisposing factors that lead to subacute stent thrombosis in patients with the acute coronary syndrome who have undergone PCI.*

MATERIAL & METHODS: *Retrospective demographical and angiographical data of the patients who have undergone PCI and also were presented with ACS was gathered from the hospital registry. All the data were analyzed using SPSS and were presented as mean ± SD and percentages for continuous and categorical variables, respectively. Univariate and multivariate analysis was carried out to analyze the subacute stent thrombosis predictors.*

RESULTS: *The occurrence of subacute stent thrombosis was found to be 4..9 %. A significant higher number of patients who have developed subacute stent thrombosis were male (81.4 %, p = 0.037), suffered from diabetes mellitus (48.1 %, p = 0.034), had hypertension (59.2 %. P = 0.016), with pre-procedural decreased left ventricular ejection fraction (LVEF) (36.11 ± 6.86, p < 0.001) and Killip Class (p < 0.001). Significantly higher odds were observed among patients with diabetes (2.13 [1.01–4.34]), hypertension (2.33 [1.17–4.86]), and the Killip Class III or IV patients (6.4 [2.35–17.41]). The single independent predictor of the subacute stent thrombosis was found to be Killip Class III-IV with an adjusted ratio of 5.1 [1.81–15.32].*

CONCLUSION: *Subacute stent thrombosis in patients who have undergone PCI for acute myocardial infarction is not as infrequent as demonstrated by the previous studies accruing with a frequency of 4.9 % with a death rate of 7 % in the patients with SST. Diabetes and hypertension were observed to be associated and served as risk factors for the development of SST. Killip class III-IV was demonstrated to be the single independent predictor of subacute stent thrombosis.*

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Author's Contribution: KA: Intruduction, literature review, study design, paper drafting. KZ: Questionaire design, data collection, drafting. MAA: Data collection, analysis, interpretation. OMK: Questionaire design, data analysis. MZT: Data interpretation, drfating. MAR: Data collection, data analysis

INTRODUCTION

Percutaneous coronary intervention (PCI) by stenting is a modern and usually used technique for coronary revascularization. This technique is safe and greatly decrease the need for vessel revascularization compared to older technique Like balloon angioplasty so it becomes a first-line treatment for coronary revascularization.[1] As PCI is a catheter-based procedure that's why it's associated with the risk of vascular injury, especially at the endothelial level.[2] The notion of safe and efficient percutaneous coronary intervention (PCI) should include two features one is the opening of the targeted coronary vessel and the second is prevention or minimal vascular damage to decrease future pathological consequences. The life-saving technique is nonetheless associated with the risk of a complication, namely stent thrombosis which is a less common but fatal that in most cases lead to death or a big non-fatal ST-elevation myocardial infarction (STEMI). As per available data, approximately 10 percent of cardiac deaths after stent placement are associated with stent thrombosis, while the rest are attributable to disease progression.[3] Moreover, patients who are diagnosed with ACS are observed to have a greater likelihood of developing ST following percutaneous coronary intervention (PCI) compared to patients with stable ischemic heart disease. The 30-day rate of ST for ACS patients ranges from 1%-3%, whereas the rate for those with stable ischemic heart disease is 0.3%-0.5%.[4-7]

As per Academic Research Consortium (ARC) guidelines 2008, stent thrombosis was Classified based on the type of stent used, clinical presentation, and timing after initial stent placement.[8] Based on timing after initial stenting, if thrombosis formation occurs within 24 hours it is called acute thrombosis and if it occurs between 24h to 30 days it is known as sub-acute thrombosis.[9] Research data indicates that stent thrombosis frequency is similar within the first year among patients who receive bare-metal stents (BMS) or drug-eluting stents (DES) if both groups of patients get treatment with dual antiplatelet therapy (aspirin and PY12 antagonist) for the recommended period.[10-12] Patients with Drug eluted stents need dual antiplatelet therapy for a relatively longer duration as compared to BMS, this difference in therapy is reduced with newer-generation drug-eluting stents use. Previous research data suggests multiple predisposing factors play role in sub-acute thrombosis (usually with ST elevation). Stent thrombogenicity or device-related factors falls into the primary category and encompass factors such as materials from which the stent is made, its design, coating on the surface, and how it interacts with adjunctive therapy.[12,13] The second category considers factors specific to patients or lesions which include unstable angina/ ACS, ventricular ejection fraction, size of the vessel, coronary blood flow, coagulation or platelet activity at a local site, and characteristics of plaque.[14-17] The third category involves procedural factors such as malposition of the stent, improperly sized stents, suboptimal antithrombotic therapy, mechanical injury to the vessel, stent under-expansion and residual dissection.[18]

However, very few studies have been conducted regarding the predisposing factors of subacute stent thrombosis (SST) in Pakistan, and hence, there is very limited knowledge regarding the trend of risk factors associated with SST. Therefore, this study will determine the predisposing factors that cause subacute stent thrombosis in patients with the acute coronary syndrome who have undergone PCI.

METHODOLOGY:

STUDY DESIGN AND SETTING:

The retrospective records of those patients were obtained who were admitted at Punjab Institute of Cardiology (PIC) Lahore between 1st July 2019 to 30th June 2021 and were diagnosed with acute coronary syndrome and were subjected to PCI procedure. Non-probability consecutive sampling technique has been opted for. Availability of records of at least one month of follow-up was set as inclusion criteria and patients exhibiting unfavorable outcomes within a day after PCI including those who experienced either minor or major peri-procedural bleeding were subjected to exclusion from the study. Prior authorization of all concerning departments and the Ethical Review Committee was obtained before the study was conducted.

TREATMENT REGIMEN:

During the procedure, a medication regimen per recommended guidelines was maintained for patients which included 300 mg of aspirin, 600 mg of clopidogrel, and 70 to 100 units per kg of

heparin bolus unfractionated without glycoprotein IIb/IIIa inhibitor and 50 to 70 units per kg with glycoprotein IIb/IIIa inhibitor.In addition to that, right from the day the procedure began, every single patient was given 300 mg of soluble aspirin once per day and 75 mg clopidogrel twice per day as dual antiplatelet therapy for a period of one month as decided by the physician in charge. The procedure was followed up by providing patients with 75 mg of aspirin indefinitely and clopidogrel once per day for a year.

DATA COLLECTION AND FOLLOW-UP OUTCOMES:

A personalized database from a hospital registry was concerned to obtain demographic and procedural outcome data. Records of visits of outpatients on the 30th day of their treatment were also acquired to assess sub-acute stent thrombosis. To make up for the missing records of patients who received primary PCI treatment during the course of the study, telephonic calls were made to obtain information about their outcomes.

Sample size of 551 was calculated to achieve 95% power of the study while taking level of significance (α) as 0.05 with assuming the incidence of diabetes mellitus in patients with SST as (42.4 %) and in patients with no SST development as (25.7%) deduced from the findings of a recent study [19] in which diabetes was shown to be the independent predictor of stent thrombosis. However, we aimed to collect data of as many patients as possible within the duration of the study.

OPERATIONAL DEFINITIONS:

Percutaneous Coronary Intervention (PCI) is a minimally invasive procedure that uses a catheter to access the blocked coronary arteries and insert a stent to restore blood flow. The procedure is done under X-ray guidance and involves inserting a catheter through the artery in the arm or leg, up to the site of the blockage. A balloon is then inflated to open the artery and a stent is inserted to keep it open.[20]

This study focused on identifying subacute stent thrombosis in patients. Subacute stent thrombosis was elucidated as recurring MI and obstruction at the site where the stent was implanted for the treatment of the infarcted vessel after a day to a month (from 24 hour to 30 days) of successful balloon angioplasty.[21] Revascularization of TIMI flow grade-3 for the treated blood vessel and <50% angiographic residual stenosis was used as a basis for the definition of a successful angioplasty.

When describing the flow of blood beyond a coronary occlusion, TIMI 0 indicates there is no antegrade flow, TIMI 1 indicates faint antegrade flow but incomplete filling of the distal coronary bed, TIMI 2 indicates delayed or sluggish flow with complete filling of the distal territory, and TIMI 3 indicates normal flow that fully fills the distal coronary bed.[22]

Stent thrombosis was categorized as definite or probable per the definition of the Academic Research Consortium.[23]

DEFINITE STENT THROMBOSIS:

An angiographic confirmation of definite stent thrombosis is linked with one of the following criteria present within a 48-hour window: intense ischemic symptoms at rest, new ischemic electrocardiographic changes indicative of acute ischemia, or a noticeable increase/decrease in cardiac biomarkers. Additionally, a pathological confirmation of stent thrombosis (evidence of freshly formed thrombus within the stent examined at autopsy or with thrombectomy) is also associated with this phenomenon.[23]

PROBABLE STENT THROMBOSIS:

If there is an unexplained death within 30 days of stent implantation, or a myocardial infarction (MI) due to documented acute ischemia in the territory where the stent was implanted without angiographic evidence of stent thrombosis and without any other identifiable cause, then it is considered probable stent thrombosis, regardless of how much time has passed since the procedure.[23]

KILLIP CLASSIFICATION:

Patients were classified on the basis of Killip class

Killip class I encompasses individuals with no clinical evidence of heart failure. Killip class II is characterized by the presence of rales or crackles in the lungs, an S 3 gallop, and elevated jugular venous pressure. Killip class III describes individuals with frank acute pulmonary edema. Killip class IV is defined by cardiogenic shock or hypotension (systolic blood pressure <90 mmHg) and signs of low cardiac output (oliguria, cyanosis, or impaired mental status).[24]

STATISTICAL ANALYSIS:

The 24.0 version of IBM SPSS Statistics was used to analyze the gathered data. Continuous variable data was illustrated using mean ± S.D while for categorical data, percentages were used.

Factors that affect stent thrombosis were identified by using univariate and multivariate logistic regression analysis. The odds ratios with a 95% confidence interval were also computed to properly assess the potential predictors. Significant

variables from the univariate analytical data like family history of coronary disease, age, multi vessel disease (MVD), lesion length (mm), culprit left main (LM), stent length (mm), stent diameter (mm), bare-metal stents, hypertension, diabetes mellitus and Killip Class (III-IV) were used in multivariate analysis as explanatory variables. All the results were made known that were obtained from all the important variables used in multivariate analysis.

RESULTS:

A total of 551 patients were part of this study who underwent PCI. The proportion of 80.7 % (445) out of the total number of patients were male and the 19.2 % (106) were female patients. The mean age was 55.86 ± 11.12 for the total patients. Subacute stent thrombosis was observed to be developed in 27 (4.9 %) of the patients, among which 3 (11.1 %) were categorized under definite ST and 24 (88.8%) were under the category of probable ST.

The culprit artery among the total number of participants was LAD in 249 (45.2 %), RCA in 172 (31.2 %), LCX in 127 (23.0 %) and LM in 3 (0.5 %).

The demographic, baseline, and angiographic characteristics are summarized in table 1. A significantly higher number of patients who developed subacute stent thrombosis were male, suffered from diabetes mellitus, and had hypertension, with pre-procedural decreased left ventricular ejection fraction (LVEF) and Killip Class.

Table 2 summarized the procedural variables. A drug eluting stent was used in all patients. The TIMI flow grade after the procedure was assigned as 0 in 1 (0.1 %) of the patients, I in 2 (0.2 %) patients, II in 16 (3 %) of the patients, and III in 532 (96.5 %) of patients. No significant difference was observed in stent type, TIMI flow grade, stent length (mm), and stent diameter of the two groups.

Table 3. enlists the predisposing factors of sub-acute stent thrombosis. Among the patients related factors, Diabetes and hypertension are observed to be related to higher rates of sub-acute stent thrombosis ($p = 0.01$ and $p < 0.0001$, respectively). Smaller size of the vessel and presence of bifurcation lesion was significantly increased in patients with sub-acute stent thrombosis among the lesion related factors with a p value of < 0.0001 and 0.05, respectively. The stent related predisposing factors of sub-acute stent thrombosis were under sizing of the stent ($p < 0.0001$) and

Table 1. Demographic, clinical baseline and angiographic characteristics of the patients (n = 551)

Variable	Total	Subacute Stent Thrombosis		p - value
		No	Yes	
N	551	524	27	-
Gender				
Male	445 (81 %)	423 (81 %)	22 (81.4 %)	0.923
Female	106 (19.2 %)	101 (19.2 %)	5 (18.5 %)	
Age	55.86 ± 11.12	55.41 ± 10.89	58.45 ± 13.14	0.162
BMI (kg/m²)	25.56 ± 4.55	25.65 ± 4.59	25.16 ± 3.57	0.585
Multivessel disease	271 (49.1 %)	257 (49 %)	14 (51.8 %)	0.77
Culprit Artery				
LAD	249 (45.2 %)	236 (45 %)	13 (48.1 %)	0.06
RCA	172 (31.2 %)	162 (31 %)	10 (37 %)	
LCX	127 (23.0 %)	124 (23.6 %)	3 (11.1 %)	
LM	3 (0.5 %)	2 (0.3 %)	1 (3.7 %)	
Killip Class				
I	475 (86.2 %)	458 (87.4 %)	17 (63 %)	<0.001*
II	53 (9.6 %)	48 (9.1 %)	5 (18.5 %)	
III	13 (2.3 %)	11 (2.0 %)	2 (7.4 %)	
IV	10 (1.8 %)	7 (1.3 %)	3 (11.1 %)	
LVEF (%)	44.9 ± 10.03	45.4 ± 9.97	36.11 ± 6.86	<0.001*
Lesion length (mm)	19.08 ± 8.07	19.04 ± 8.18	19.7 ± 6.07	0.279

*Statistically significant

Table 2. Procedural characteristics of the patients				
Variable	**Total**	**Subacute Stent Thrombosis**		**p-value**
		No	Yes	
N	551	524	27	
Stent type				
Drug-eluting stents	551	524	27 (4.9%)	0.143
Stent length (mm)	16.85 ± 7.3	16.79 ± 7.27	17.51 ± 6.09	0.613
Stent diameter	3.15 ± 0.62	3.19 ± 0.65	3.01 ± 0.57	0.158
Post-procedural TIMI flow grade				
0	1 (0.1 %)	1 (0.2 %)	0 (0 %)	0.535
I	2 (0.4%)	2 (0.4%)	0 (0 %)	
II	16 (3 %)	14 (2.6 %)	2 (7.4 %)	
III	532 (96.5 %)	507 (96.7 %)	25 (92.5 %)	

Table 3. Predisposing factors of sub-acute stent thrombosis			
Variable	**With SAT n = 27**	**Without SAT n = 524**	**P-value**
Patient related			
DM	8 (29.6 %)	67 (12.7 %)	0.01*
Hypertension	22 (81.4 %)	219 (41.7 %)	< 0.0001*
Smoking	18 (66.6 %)	315 (60.1 %)	0.50
Heart failure	1 (3.7 %)	13 (2.4 %)	0.67
ACS	21 (77.7 %)	300 (57.2 %)	0.03
CKD	1 (3.7 %)	12 (2.2 %)	0.61
Poor or non-compliance to medication	3 (11.1 %)	31 (6 %)	0.28
Lesion related			
Long lesion	23 (85.1 %)	419 (80 %)	0.51
Diffuse dissection	1 (3.7 %)	19 (3.6 %)	0.97
Small vessel	11 (40.7 %)	78 (14.8 %)	< 0.0001*
Bifurcation lesion	11 (40.7 %)	127 (24.2 %)	0.05*
Thrombus	17 (62.9 %)	301 (57.4 %)	0.57
Stent related			
Under sizing	5 (18.5 %)	12 (2.2 %)	< 0.0001*
Under expansion	1 (3.7 %)	19 (3.6 %)	0.97
Edge dissection	5 (18.5 %)	32 (6.1 %)	0.01
*Statistically significant			

Table 4. Predictors of subacute stent thrombosis				
Variable	**Non-adjusted**		**Adjusted**	
	[95% CI] OR	**p-value**	**[95% CI] OR**	**p-value**
Diabetes Mellitus	2.13 [1.01–4.34]	0.037	2 [0.9–4.43]	0.085
Hypertension	2.33 [1.17–4.86]	0.02	1.63 [0.75–3.53]	0.221
Killip Class (III-IV)	6.4 [2.35–17.41]	<0.001	5.1 [1.81–15.32]	0.004

edge dissection (p = 0.01).

The adjusted and unadjusted odds ratio, with the 95 % confidence interval, of the significant variables are listed in Table 4. Significantly higher odds were observed among patients with diabetes (2.13 [1.01–4.34]), hypertension (2.33 [1.17–4.86]), and the Killip Class III or IV patients (6.4 [2.35–17.41]). The only independent predictor

of the subacute stent thrombosis was found to be Killip Class III-IV with an adjusted ratio of 5.1 [1.81–15.32].

DISCUSSION:

In this study we aimed to assess the potential predictors of sub-acute stent thrombosis. According to our findings, the Diabetes mellitus, hypertension and Killip Class (III-IV) were demostrated to have strong association in causing sub-acute stent thrombosis. However, only Killip Class (III-IV) was assessed to found as the independent predictor of sub-acute stent thrombosis. Acute and subacute stent thrombosis have some potential predictors, which have been investigated over the recent few years by different clinical studies.[16,19,25,26] These studies were aimed toward identifying and recognizing fundamental mechanisms for which stent thrombosis is credited and essentially in the long run coming up with a viable strategy to knock out the possibilities of this terrible complication. This goal, however, is hindered by several methodological obstacles such as extant patients' series yielding a low incidence of stent thrombosis and requiring a larger sample size. This retrospective that involved 551 patients who received primary PCI for STEMI found subacute stent thrombosis to occur in about 4.9 % of the patient population and is not so much of a rare event.

Even though the true pathophysiology of stent thrombosis has not been yet understood completely, there are seemingly various factors that may cause stent thrombosis. The most important category incorporates factors that are related to the device itself. According to our study, the type of implanted stent was not related to stent thrombosis in the course of one month. The next category encompasses factors specific to patients or lesions. Among those factors, hypertension and diabetes were associated with a greater risk of early stent thrombosis (within 30 days), as per our results.

The factors related to the procedure are included in the third category. However, in comparison with previous studies, the frequency of stent thrombosis was not found to be related to stent diameter and length or the length of the lesion. On the other hand, a mortality rate of 4.3% was reported with early stent thrombosis at thirty (30) days.

Stent thrombosis elevates the risk of MI and death. Keeping in view the emerging data, the extensive predictor of stent thrombosis is STEMI presentation. According to Swedish Coronary Angiography and Angioplasty Registry (SCAAR), patients without STEMI are at 2.5 fold reduced risk of stent thrombosis as compared to the patients with STEMI.[27] The rate of incidence of ST was found to be between 0.5 to 2.0%.[28-31] Several factors during the procedure or related to lesions like tissue protrusion, edge dissection compromised flow, under the expansion of the stent, independently and sometimes in a union were linked to these early events.

In our study, we used the retrospective data of consecutive present in the repository of the center which was collected irrespective of the stent type used for the patient's treatment. The use of bare metal stents (BMS) has been increasingly common in the treatment of patients with acute coronary syndrome (ACS). This is due to their low cost and efficacy in treating ACS.[32] Moreover, the decision to use BMS or DES in ACS was based on a patient's individual risk profile, and the physician's clinical judgment and the patient were informed and briefed about receiving BMS or DES. It was observed during our study that acute or subacute stent thrombosis was not related to either BMS or DES implanted stent type. ACUITY trials backed up our findings by showing no observable differences in an increased risk of early acute or subacute stent thrombosis between patients who got either a BMS or DES for the treatment of ACS.[6] A wide range of randomized trials and studies conducted to assess the difference between first-generation BMS and DES when it came to the rate of development of early stent thrombosis, concluded that the type of stent did not affect the rate of stent thrombosis in any way.[25,33-35]

Our study revealed some patient-, lesion- and stent-related factors that predispose patients to sub-acute stent thrombosis. Among the patient-related factors, diabetes mellitus, hypertension, and acute coronary syndrome were the predisposing factor to SAT, in line with other studies.[36] Among the lesion-related characteristics, Smaller vessel size, and the presence of bifurcation lesion are the observed predisposing factors as described by other trails.[16,36] Moreover, the stent-related predisposing factors are under-sizing of the stent and edge dissection, as also outlined by another study.[16]

Our study observed that diabetic patients have a higher risk of stent thrombosis, however, statistically significant values were not observed for diabetes mellitus for it to be considered as an independent predictor of stent thrombosis. Diabetes was reported as an independent predictor for stent thrombosis in

previous studies.[37-39] The significance of diabetes priorly reported can be ascribed to the impacts of confounders including smaller size of vessel, longer lesion length, higher residual dissections rate, and increase in platelet aggregation.[40-43]

Likewise, a relatively high risk of ST was noticed in patients with hypertension, but on its own, hypertension too proved inadequate to provide statistically significant data to be termed as an independent predictor of ST. This finding is oriented with the previous studies which also failed to state hypertension as a distinctive risk factor for stent thrombosis. Our study has demonstrated the final and only independent predictor for ST as presentation of patients in Killip Class III-IV. It was noted by Stone et al. in one of their studies in HORIZON-AMI trials that the presentation of patients with acute heart failure (Killip class $\geq$ 2) served as a significant independent predictor of re-infarction occurring in patients who underwent primary PCI therapy.[44]

The most frightening complication of PCI includes stent thrombosis. Before proceeding with PCI with the use of a stent, the risk factors associated with it and the capability of the patient for adhering to and tolerating DAPT should be precisely taken into consideration. The technicalities of optimizing stent implantation and deployment require assiduous care, especially in the case of complex diseases. Novel stents are also being introduced that inherently enable to lower the risk of stent thrombosis. Despite the enterprising management of ST, it still relates to crucial clinical events. The current study identified patient-related predisposing factors of SST which are diabetes and hypertension. Therefore, for preventing or at least minimizing the catastrophic complication of stent implantation, careful evaluation of an individual patient's risk associated with ST is essential.

One of the limitations of our study was it was a retrospective study that lacked the findings of acute, late, and very late stent thrombosis. Other than that, the process behind the greater risk of stent thrombosis is not studied in patients with heart failure, so we cannot be certain whether it is solely due to stent thrombosis.

CONCLUSION:

Subacute stent thrombosis in patients undergoing PCI for acute myocardial infarction is not as infrequent as demonstrated by the previous studies and is associated with a high mortality rate. Diabetes and hypertension were observed as risk factors for the development of SST. Killip Class III-IV was demonstrated to be the only independent predictor of subacute stent thrombosis.

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