PREDICTORS OF ACUTE STENT THROMBOSIS AFTER PRIMARY PERCUTANEOUS CORONARY INTERVENTION

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ABSTRACT

OBJECTIVES

To identify the specific predictors of acute stent thrombosis in patients after primary percutaneous coronary intervention.

METHODOLOGY

This retrospective study was carried out at the Department of Cardiology Hayatabad Medical Complex Peshawar from 1st January to 30th June 2022. All consecutive patients with an angiographically confirmed stent thrombosis were enrolled. Patients gave informed consent for the inclusion of data in this registry. Stent thrombosis was categorized according to the timing of the event as acute (occurrence within the first 24 hours after the index procedure).

RESULTS

A total of 400 patients were included in the study. Age ranged between 35-70 years, with a mean age of 52.5. There were 260(65%) males and 140(35%) females, with male to female ratio of 1.8:1. All patients underwent primary PCI with stent implantation. According to the elapsed time since stent implantation, 42(10.5%) patients presented with acute stent thrombosis after primary percutaneous coronary intervention. The mean time to develop acute stent thrombosis after primary PCI was ±4.5 hours (range 3-6 hours). In most STEMI patients, 340(85%) received a loading dose of clopidogrel at the time of the index PCI. In 23(54.7%) patients, acute stent thrombosis occurred within 6 hours, 10(23.8%) within 12 hours, 6(14.2%) within 18 hours and 3(7.1%) after clopidogrel loading.

CONCLUSION

Inadequate stent expansion or mal-opposition, diabetes mellitus, chronic kidney disease, and female gender were the strong predictors of acute stent thrombosis.

KEYWORDS: *Acute Stent Thrombosis, Myocardial Infarction, Risk Factors*

INTRODUCTION

Due to several challenges being overcome, percutaneous coronary intervention (PCI) has significantly increased in both safety and effectiveness over the past 40 years. Since the invention of coronary artery stents, stenting has been regarded as the major post-procedural complication of percutaneous coronary intervention stent thrombosis.[1,2] The development of drug-eluting stents (DES) was specifically one of the important developments in interventional cardiology.[3,4] With an initial higher risk of acute stent thrombosis, drug-eluting stents have been demonstrated to minimize in-stent restenosis and eventual target lesion revascularization.[5] These changes were consistent with improvements in stent manufacturing and design, percutaneous coronary intervention adjunctive medicine, and understanding of risk factors and the pathophysiology of stent thrombosis.[6] The effective management of stent thrombosis is an understudied subject in interventional cardiology; therefore, prevention and identification of the associated technical and clinical factors remain the pillars of stent thrombosis care.[7] The stent thrombosis risk was significantly higher in individuals with acute ST-elevation myocardial infarction (STEMI).[8] The major risk factors of stent thrombosis in the initial days of percutaneous stent implantation were technical flaws in the index PCI procedure. Technical shortcomings linked to increased stent thrombosis include residual stenosis, dissection, stent under-sizing, and sub-optimal final coronary flow.[9] Selection of type of antithrombotic medication during a procedure is another major factor of clinical outcomes such as stent thrombosis. To a certain degree, bleeding and stent thrombosis complications are minimized with the use

of DAPT and the selection of suitable ADP-receptor inhibitor.[10,11] Even though several studies have been reported, available data are skewed to specific geographies, and data from the South Asian region, especially Pakistan, almost ceased to exist.[12] To keep up the pace of developments in stent technology, research work should continue to improve our understanding of stent thrombosis's underlying mechanism and determinants.[13] Therefore, this study determined the factors that may predispose to stent thrombosis even with modern drug-eluting stents among patients with STEMI undergoing primary percutaneous coronary intervention.

METHODOLOGY

This retrospective study was carried out at the Department of Cardiology Hayatabad Medical Complex Peshawar from 1st January to 30th June 2022. All consecutive patients with an angiographically confirmed stent thrombosis were enrolled. Patients gave informed consent for the inclusion of data in this registry. Stent thrombosis was categorized according to the timing of the event as acute (occurrence within the first 24 hours after the index procedure). Detailed data on patient characteristics, concomitant medication and the index primary PCI procedure were collected. Two experienced interventional cardiologists reviewed the coronary angiograms of the index procedure and the procedure for stent thrombosis independently. All patients were given a 600-mg loading dose of clopidogrel during the primary PCI for STEMI. The use of glycoprotein IIb/IIIa therapy during the index PCI and at the time of the stent thrombosis was left to the discretion of the interventional cardiologist. Clinical follow-up information was obtained from telephone interviews with the patients, their relatives, and their general practitioners, as well as from pharmacy and hospital records. Information was also collected regarding the development of major adverse cardiac events, predefined as death or definite recurrent stent thrombosis. Statistical analysis was done using SPSS version 23.0 for windows. A p-value of ≤0.05 was considered statistically significant.

RESULT

A total of 400 patients were included in the study. Age ranged between 35-70 years, with a mean age of 52.5. There were 260(65%) males and 140(35%) females, with male to female ratio of 1.8:1. All patients underwent primary PCI with stent implantation. Figure-1 In our set-up, most cases of acute stent thrombosis were due to inadequate stent expansion or mal-opposition 13(31%) followed by edge dissection 9(21.4%), uncovered lesion 7(16.7%) and heavy clot burden 6(14.3%). Comorbid conditions like diabetes mellitus 3(7.1%), chronic kidney disease 2(4.8%) and female gender 2(4.8%) were also associated with acute stent thrombosis. Figure-2 The mortality rate in our study was 15(10%).

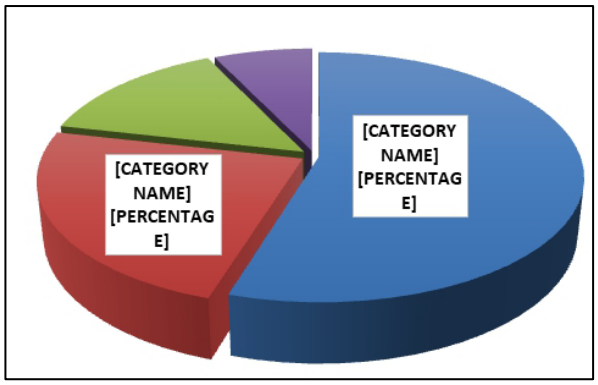

Figure 1: Time to Acute Stent Thrombosis after Clopidogrel Loading Dose

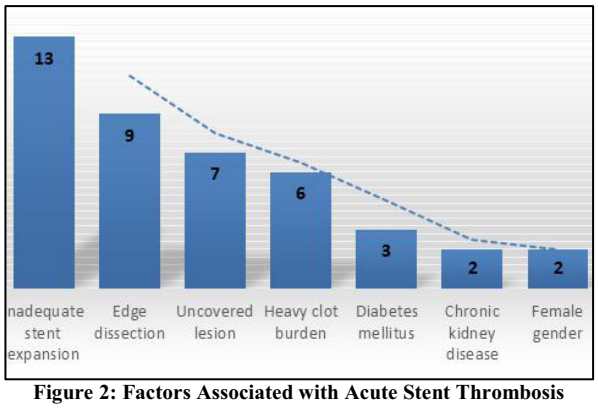

Figure 2: Factors Associated with Acute Stent Thrombosis

DISCUSSION

This study unequivocally demonstrates that the risk factor profiles for acute stent thrombosis vary significantly following primary percutaneous coronary intervention (PCI) for ST Elevation Myocardial Infarction. Although several correlates of acute stent thrombosis following primary PCI have been identified in other reports, a thorough risk factor analysis specifically for acute stent thrombosis is still missing.[14] The results of the current study are important because thrombus load may influence thrombolysis in myocardial infarction (TIMI) flow. Inadequate stent expansion or mal opposition are indicators of acute stent thrombosis. This is supported by the much greater incidence of visible thrombus in patients with acute stent thrombosis in the current analysis, which further emphasizes the significance of thrombus burden.[15]

Interestingly, most acute stent thromboses occurred within 6 hours after primary PCI (55%) and consequent clopidogrel loading. Uren NG et al. demonstrated that the maximal antiplatelet effect of a 600-mg loading dose of clopidogrel is achieved within 2 hours.[16] However, this was investigated in patients who underwent elective PCI for stable angina pectoris. In STEMI patients, the maximal antiplatelet effect of a 600-mg loading dose is impaired and delayed, mostly because of reduced gastrointestinal absorption of clopidogrel.[17] With predictors of acute ST these data, in addition to the findings of the present analysis, one might conclude that a single high-loading dose of clopidogrel in this setting of STEMI with substantially increased platelet activation is not sufficient to achieve optimal inhibition of platelet activation within the first 24 hours and therefore is not the optimal treatment to prevent acute stent thrombosis.[18] In support of this statement, treatment with the potent, immediately-acting antiplatelet glycoprotein IIb/IIIa was protective against the development of acute stent thrombosis. Large registry studies have shown that, in real-world daily practice, glycoprotein IIb/IIIa inhibitors are given to only 25–30% of patients with STEMI, often for bail-out situations.[19] The reported use in the present analysis of acute stent thrombosis was relatively low, reflecting real-world clinical practice. The severity of chronic kidney disease & diabetes mellitus was also associated with acute stent thrombosis, which agrees with previous reports.[20] In contrast to acute stent thrombosis, for which procedural correlates play a predominant role, the risk factors for subacute stent thrombosis are a more mixed collection of medication-related and lesion-related factors. The mortality rates in the present study are unfavorable 21(5.2%) but are relatively low, in contrast to those in previous reports on this subject, which have reported up to 40% mortality.[21] Our study used very strict definitions of definite angiographically confirmed stent thrombosis. In comparison, no additional deaths occurred within 30 days. Stent thrombosis is also known as a predictor of mortality. However, given the sample size, we could not perform an additional analysis with sufficient power to establish whether subacute stent thrombosis is a stronger predictor of mortality than acute stent thrombosis, although faster re-intervention is more likely in patients with acute stent thrombosis, and probably contributes to improved survival.

LIMITATIONS

Some limitations of our study need to be acknowledged. Previous reports have shown that TIMI major bleeding correlates with stent thrombosis development; however, in the present study, no data on TIMI major bleeding were obtained. The exact administration time of clopidogrel and glycoprotein IIb/IIIa inhibitors (prehospital or Cath lab) was not documented. Therefore, we could not analyze the effect of prehospital antiplatelet therapy in STEMI patients on the incidence of acute stent thrombosis.

CONCLUSION

Risk factor profiles for acute stent thrombosis vary greatly after primary PCI for ST elevated myocardial infarction patients. Inadequate stent expansion or mal-opposition, diabetes mellitus, chronic kidney disease and female gender were the strong predictors of acute stent thrombosis.

CONFLICT OF INTEREST: None

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