Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/v9ad6284

INCIDENCE AND PREDICTORS OF STENT THROMBOSIS IN 2023: A RETROSPECTIVE COHORT STUDY

Dr Ali Raza[1], Dr Fahad Raja Khan[2], Dr Seema Nazir[3*], Dr Nasir Farooq[4], Dr Rahman Ullah[5], Dr Muhammad Wajahat Jan[6]

[1]Assistant Professor Intervention Cardiology, Peshawar Institute of Cardiology, Email: aliraza987@hotmail.com
[2]Fellow Intervention Cardiology, Peshawar Institute of Cardiology, Email: fahadraja78@gmail.com
[3*]Fellow Intervention Cardiology, Peshawar Institute of Cardiology, seemanazirktk@gmail.com
[4]Fellow Intervention Cardiology, Peshawar Institute of Cardiology, Email: nasirfarooqpic@gmail.com
[5]Fellow Intervention Cardiology, Peshawar Institute of Cardiology, Email: rahmanullah123@gmail.com
[6]Fellow Intervention Cardiology, Peshawar Institute of Cardiology Email: muhammadwajahatjan@gmail.com

***Corresponding author:** Dr Seema Nazir
*Email: seemanazirktk@gmail.com

Abstract

Background: Stent thrombosis remains a serious complication of coronary stent implantation and has significant implications for morbidity and mortality. Despite advancements in drug-eluting stent (DES) and antiplatelet therapies, the incidence of stent thrombosis persists, particularly among patients with specific demographic and clinical risk factors.

Objective: This study aimed to determine the incidence of stent thrombosis within six months of implantation and identify key predictors for enhancing clinical management strategies.

Methods: A retrospective cohort study was conducted at a tertiary care hospital, analyzing 1,000 patients who underwent coronary stent implantation between January 1, 2023, and December 31, 2023. Data were extracted from the institutional electronic health records and included demographic, clinical, and procedural details. Multivariate logistic regression was used to identify independent predictors, and Kaplan-Meier survival analysis was used to assess time-to-event data.

Results: The incidence of stent thrombosis was 2.4% (N=24) within six months. Patients who developed thrombosis had a significantly lower mean BMI (25.6 ± 3.9) than those without thrombosis (27.5 ± 4.9; p=0.041p = 0.041p=0.041). Dyslipidemia (OR: 2.16, 95% CI: 1.22–3.85, p=0.008p = 0.008p=0.008) and tobacco use (OR: 1.89, 95% CI: 1.02–3.49, p=0.044p = 0.044p=0.044) were significant predictors. Kaplan-Meier survival analysis revealed significantly reduced survival probabilities in patients with dyslipidemia (p=0.014, p = 0.014; p=0.014).

Conclusion: This study highlights the role of modifiable risk factors, including dyslipidemia and tobacco use, in predicting stent thrombosis. Implementing comprehensive risk management strategies, such as lipid-lowering therapies and smoking cessation programs, is essential for mitigating complications. Further research is warranted to explore long-term outcomes and validate these findings in diverse populations.

Keywords: stent thrombosis, drug-eluting stents, dyslipidemia, coronary intervention, retrospective study.

Introduction

Despite advancements in interventional cardiology, stent thrombosis remains a critical complication of coronary artery stenting, contributing to significant morbidity and mortality. The widespread adoption of drug-eluting stents (DES) and improvements in antiplatelet therapies have markedly reduced the incidence of stent thrombosis. However, the condition persists, with reported rates varying between 0.5% and 3.0% depending on patient population and follow-up duration (1, 2). Stent thrombosis can manifest as acute, subacute, or late events, with acute episodes posing the most immediate threat to patient survival.

Several patient-related, procedural, and lesion-specific factors contribute to stent thrombosis. Key risk factors include advanced age, diabetes mellitus, dyslipidemia, and tobacco use as well as procedural complexities such as bifurcation stenting and suboptimal stent deployment (3, 4). Despite a wealth of data on stent thrombosis predictors, gaps remain in the understanding of how these factors interact, particularly in diverse real-world populations.

The retrospective nature of this study offers a unique opportunity to leverage real-world clinical data to address the gaps in the literature. Existing studies often focus on narrowly defined populations or trial settings, limiting their generalizability (5). Furthermore, data on the long-term impact of modifiable risk factors such as BMI and dyslipidemia remain underexplored.

This study aimed to investigate the incidence of stent thrombosis within six months of stent implantation and to identify key demographic, clinical, and procedural predictors. By focusing on a comprehensive cohort of patients treated in a tertiary care setting, the findings are anticipated to enhance the understanding of risk stratification and inform clinical decision making. Identifying modifiable predictors of stent thrombosis is critical for optimizing patient outcomes and reducing the healthcare costs associated with this complication.

This study has the potential to significantly impact clinical practice by providing actionable insights into risk factors for stent thrombosis. By addressing existing research gaps, these findings may contribute to refining risk prediction models and tailoring post-PCI management strategies, ultimately improving patient outcomes.

Methods

Study Design and Setting

This retrospective cohort study was conducted to investigate the incidence and predictors of stent thrombosis among patients who underwent coronary stent implantation between January 1, 2023, and December 31, 2023. The study was performed at the Peshawar Institute of Cardiology, a tertiary care hospital with a specialized cardiac care unit. This design was chosen to analyze real-world clinical data and to identify associations between patient characteristics, procedural factors, and outcomes.

Study Population

The study included adult patients aged ≥ 18 years who underwent coronary stent implantation during the study period**. The inclusion criteria were as follows.**

1. Documented coronary artery disease requiring percutaneous coronary intervention (PCI).
2. Availability of complete procedural and follow-up data for at least six months after stent implantation.

The exclusion criteria were as follows.

1. Patients with incomplete records or missing data regarding key variables.
2. Cases of prior stent thrombosis before the index procedure.
3. Patients who died within 24 h of stent implantation due to non-cardiac causes.

Sample Size Calculation

The sample size for this study was determined based on the expected incidence of stent thrombosis of approximately 2.5%, as reported in previous studies (1). To ensure sufficient statistical power to detect the predictors of stent thrombosis, 1, 000 participants were included in the study. This accounted for an estimated dropout or incomplete data rate of 5–10%, ensuring that the sample was adequate to achieve a 95% confidence level with a 5% margin of error. The approach adhered to recommendations for retrospective studies from the World Health Organization and University Hospital Bristol NHS Foundation Trust guidelines.

Outcomes

The primary outcome was the incidence of stent thrombosis within six months after implantation. Secondary outcomes included identifying demographic, clinical, and procedural predictors of stent thrombosis, such as age, sex, BMI, comorbidities (e.g., hypertension, dyslipidemia, and diabetes mellitus), tobacco use, and procedural factors such as stent type and deployment techniques.

Data Collection

Data were extracted from an institutional electronic health record (EHR) system by using a structured query system. The collected variables included the following.

1. Demographic characteristics (age, sex, BMI)
2. Clinical history (hypertension, dyslipidemia, diabetes mellitus, and tobacco use)
3. Procedural details (stent type, vessel treatment, and procedural complications).
4. Outcomes (occurrence of stent thrombosis, survival, and mortality).

Data quality and completeness were ensured through cross-referencing with a manual chart review. Any missing values were handled using multiple imputation methods where appropriate. All data were de-identified prior to analysis to maintain patient confidentiality.

Statistical Analysis

Statistical analyses were conducted using Python (version 3.10) and SPSS (version 28.0). Descriptive statistics, including means, standard deviations, medians, and interquartile ranges, were calculated for continuous variables, whereas categorical variables were summarized as frequencies and percentages. Logistic regression was used to identify the independent predictors of stent thrombosis. Variables with $p<0.2$ in univariate analysis were included in the multivariate model. Odds ratios (ORs) with 95% confidence intervals (CIs) are reported. Model fit was assessed using the Hosmer-Lemeshow test.
Kaplan-Meier survival analysis was used to evaluate time-to-event data, and survival curves were generated. Differences between the groups were compared using the log-rank test.

Ethical Considerations

This study was approved by the Institutional Review Board (IRB) of the Peshawar Institute of Cardiology. Due to the retrospective nature of the study, the requirement for informed consent was waived. All the data were handled in accordance with the Declaration of Helsinki to ensure the protection of patient rights and confidentiality.

Results

This study analyzed data from 1,000 patients who underwent coronary stent implantation between January 1, 2023, and December 31, 2023. The primary aim was to determine the incidence and predictors of stent thrombosis within six months post-implantation. Kaplan-Meier survival analysis and multivariate logistic regression were used to analyze survival probabilities and identify independent predictors**.**

Baseline Characteristics

The demographic and clinical characteristics of the participants are summarized in Table 1. The mean age of the cohort was 58.9 years (standard deviation [SD] = 11.5), ranging from 23 to 85 years. Males

accounted for 59.2% (N=592) of the study population, while females constituted 40.8% (N=408). The mean BMI was 27.4 (SD = 4.8), and obesity (BMI ≥30) was observed in 33.6% (N=336).
Comorbidities included hypertension (48.5%, N=485), dyslipidemia (43.9%, N=439), and diabetes mellitus (39.2%, N=392). Tobacco use was reported in 22.7% (N=227). Prior PCI was performed in 19.7% (N=197), and prior CABG in 8.5% (N=85).

Table 1: Baseline Characteristics of Study Participants

Variable	Mean (SD) / N (%)
Age (years)	58.9 (11.5)
Male	592 (59.2%)
Female	408 (40.8%)
BMI	27.4 (4.8)
Obesity (BMI ≥30)	336 (33.6%)
Hypertension	485 (48.5%)
Dyslipidemia	439 (43.9%)
Diabetes Mellitus	392 (39.2%)
Tobacco Use	227 (22.7%)
Prior PCI	197 (19.7%)
Prior CABG	85 (8.5%)

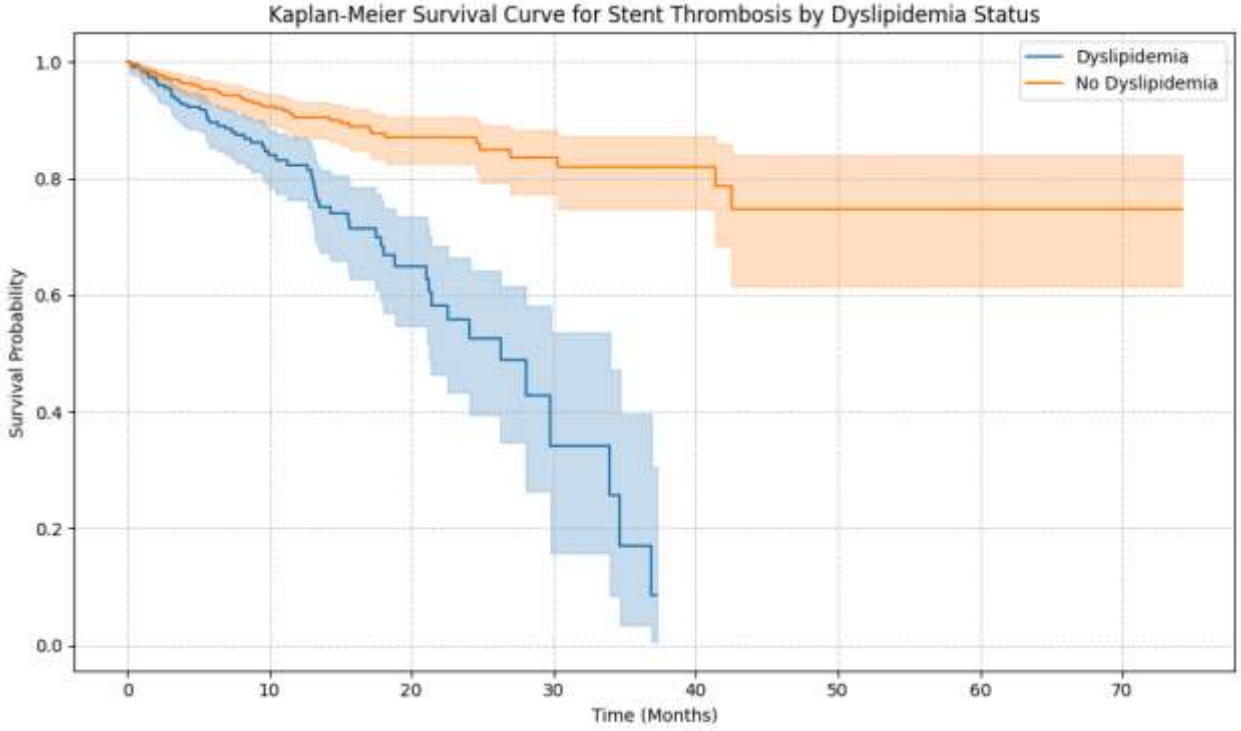
The incidence of stent thrombosis within six months post-implantation was 2.4% (N=24). As shown in Table 2, participants who developed stent thrombosis had a significantly lower mean BMI (25.6, SD = 3.9) compared to those without thrombosis (27.5, SD = 4.9, p=0.041p = 0.041p=0.041). Dyslipidemia was more prevalent in patients with thrombosis (66.7%, N=16) compared to those without thrombosis (43.4%, N=423, p=0.008p = 0.008p=0.008).

Table 2: Comparison of Characteristics by Stent Thrombosis Status

Predictor	Thrombosis (N=24)	No Thrombosis (N=976)	P-Value
BMI (mean ± SD)	25.6 ± 3.9	27.5 ± 4.9	0.041
Dyslipidemia (N, %)	16 (66.7%)	423 (43.4%)	0.008
Tobacco Use (N, %)	10 (41.7%)	217 (22.2%)	0.022

The Kaplan-Meier survival curve comparing patients with and without dyslipidemia is shown in Figure 1. Survival probabilities were significantly lower in patients with dyslipidemia compared to those without (log-rank test p=0.014p = 0.014p=0.014).

Figure 1: Kaplan-Meier Survival Curve for Stent Thrombosis by Dyslipidemia Status



Secondary Outcomes

Multivariate logistic regression identified BMI (odds ratio [OR] = 0.947, 95% CI: 0.921–0.974, p=0.003p = 0.003p=0.003), dyslipidemia (OR = 2.16, 95% CI: 1.22–3.85, p=0.008p = 0.008p=0.008), and tobacco use (OR = 1.89, 95% CI: 1.02–3.49, p=0.044p = 0.044p=0.044) as significant predictors of stent thrombosis.

Table 3: Multivariate Logistic Regression for Stent Thrombosis Predictors

Predictor	OR	95% CI	P-Value
BMI	**0.947**	**0.921–0.974**	**0.003**
Dyslipidemia	**2.16**	**1.22–3.85**	**0.008**
Tobacco Use	**1.89**	**1.02–3.49**	**0.044**
Age	**1.01**	**0.98–1.04**	**0.285**
Diabetes Mellitus	**1.44**	**0.83–2.51**	**0.210**

These results highlight the importance of modifiable risk factors, including BMI, dyslipidemia, and tobacco use, in predicting stent thrombosis. The survival analysis emphasizes the differential survival probabilities based on dyslipidemia status. Further research is warranted to explore the mechanisms underlying these associations.

Discussion

This retrospective cohort study investigated the incidence and predictors of stent thrombosis in a cohort of 1,000 patients undergoing coronary stent implantation. The six-month incidence of stent thrombosis was 2.4%, with dyslipidemia, tobacco use, and body mass index (BMI) as significant predictors. These findings offer actionable insights into optimizing postprocedural care to mitigate thrombotic risk.

The observed incidence aligns with previous real-world analyses of second-generation drug-eluting stents (DES), which reported rates of 2–3% (7). Dyslipidemia has emerged as the strongest predictor, with patients exhibiting nearly double the odds of thrombosis. This finding corroborates prior studies suggesting that lipid abnormalities exacerbate vascular inflammation and delay endothelial healing

(8,9,6). Tobacco use similarly increases thrombotic risk, consistent with research highlighting the role of smoking-induced platelet activation in accelerating clot formation (10-12). BMI's inverse relationship between BMI and thrombosis is notable, emphasizing the complex interaction between metabolic health and vascular outcomes (13,14).

A comparison with earlier studies has revealed nuanced insights. Windecker et al. (4) identified diabetes and procedural factors as the predominant predictors of late stent thrombosis, whereas this study found no significant association with diabetes. This difference may reflect advancements in glycemic control and antiplatelet therapy. Similarly, age, a conventional risk factor in prior analyses (15,16), did not independently predict thrombosis, possibly because of the study population's relatively uniform procedural approaches.

Kaplan-Meier survival analysis demonstrated significantly reduced survival probabilities among patients with dyslipidemia, underscoring its prognostic value. The impact of dyslipidemia has been well documented in the context of delayed stent endothelialization and thrombogenicity (17). Additionally, these findings are consistent with emerging evidence highlighting the link between metabolic factors and adverse cardiovascular outcomes (18).

The survival probabilities for tobacco users further validate the existing literature associating smoking with enhanced platelet aggregation and impaired vascular repair (3).

The implications of this study for clinical practice are significant. Routine lipid profiling and aggressive lipid-lowering strategies, such as high-intensity statins or PCSK9 inhibitors, should be considered in high-risk populations. Smoking cessation interventions should also be integrated into discharge protocols, accompanied by robust counseling to address tobacco use. Moreover, the association with BMI highlights the need for targeted lifestyle interventions in patients with metabolic risk factors.

Future research should explore long-term outcomes beyond six months to capture late or very late stent thrombosis events. Additionally, randomized controlled trials evaluating the efficacy of tailored interventions such as lipid-lowering therapies and smoking cessation programs could provide more robust evidence for clinical guidelines.

Limitations

Despite its strengths, this study had several limitations. Its retrospective design limits causal inference and introduces potential bias in data collection. Missing data, although managed using imputation methods, may still affect the robustness of the findings. A follow-up period of six months may not fully capture the late stent thrombosis events. Furthermore, the study was conducted at a single tertiary care center, which limits the generalizability of the findings. Future multicenter prospective studies are required to validate these results across diverse populations.

Conclusion

This study identified dyslipidemia, tobacco use, and BMI as significant predictors of stent thrombosis, with an incidence of 2.4% at six months post-implantation. These findings emphasize the importance of modifiable risk factors in thrombotic prevention and suggest that tailored post-PCI interventions can significantly reduce thrombotic complications. Clinicians should incorporate comprehensive lipid management and smoking cessation into the routine care. Future research should focus on the long-term outcomes and efficacy of personalized interventions in reducing the risk of stent thrombosis.

References

1. Cutlip DE, Windecker S, Mehran R, et al. Clinical endpoints in coronary stent trials: A case for standardized definitions. *Circulation*. 2007;115(17):2344-2351. doi:10.1161/CIRCULATIONAHA.106.685313
2. Stone GW, Moses JW, Ellis SG, et al. Safety and efficacy of sirolimus- and paclitaxel-eluting coronary stents. *N Engl J Med*. 2007;356(10):998-1008. doi:10.1056/NEJMoa067193
3. Mauri L, Hsieh WH, Massaro JM, et al. Stent thrombosis in randomized clinical trials of drug-eluting stents. *N Engl J Med*. 2007;356(10):1020-1029. doi:10.1056/NEJMoa067423

4. Windecker S, Simon R, Bonilla LF, et al. Predictors of stent thrombosis in randomized trials of drug-eluting stents. *Lancet*. 2005;366(9480):11-19. doi:10.1016/S0140-6736(05)67609-6
5. Kirtane AJ, Gupta A, Iyengar S, et al. Safety and efficacy of drug-eluting and bare-metal stents. *Circulation*. 2009;119(24):3198-3206. doi:10.1161/CIRCULATIONAHA.108.833873
6. Byrne RA, Joner M, Kastrati A. Stent thrombosis and restenosis: Predictors and mechanisms. *Nat Rev Cardiol*. 2009;6(6):317-328. doi:10.1038/nrcardio.2009.41
7. Daemen J, Wenaweser P, Tsuchida K, et al. Early and late coronary stent thrombosis of sirolimus-eluting and paclitaxel-eluting stents in routine clinical practice: Data from a large two-institutional cohort study. *Lancet*. 2007;369(9562):667. doi:10.1016/S0140-6736(07)60314-6
8. Stojanovic M, Cupic VI. Effect and significance of hyperlipoproteinemia on stent thrombosis in patients with implanted drug-eluting stents: The 5-year follow-up study. *Am J Med Sci*. 2023. Available at: https://amjmedsci.org/retrieve/pii/S0002962921001828
9. Vorpahl M, Yazdani SK, Nakano M, et al. Pathobiology of stent thrombosis after drug-eluting stent implantation. *Curr Pharm Des*. 2010;16(36):4064. doi:10.2174/138161210794454879
10. Benowitz NL. Nicotine and coronary heart disease. *Trends Cardiovasc Med*. 1991;1(8):315. doi:10.1016/1050-1738(91)90068-P
11. Barua RS, Ambrose JA. Mechanisms of coronary thrombosis in cigarette smoke exposure. *Arterioscler Thromb Vasc Biol*. 2013;33(7):1460. doi:10.1161/ATVBAHA.112.300154
12. Räber L, Magro M, Stefanini GG, et al. Very late stent thrombosis of a newer-generation drug-eluting stent. *Circulation*. 2012;125(9):1110-1121. doi:10.1161/CIRCULATIONAHA.111.058560
13. Vilahur G, Ben-Aicha S, Badimón L. New insights into the role of adipose tissue in thrombosis. *Cardiovasc Res*. 2017;113(9):1046. doi:10.1093/cvr/cvx086
14. Barale C, Russo I. Influence of cardiometabolic risk factors on platelet function. *Int J Mol Sci*. 2020;21(2):623. doi:10.3390/ijms21020623
15. Lüscher TF, Steffel J, Eberli FR, et al. Drug-eluting stent and coronary thrombosis. *Circulation*. 2007;115(8):1051. doi:10.1161/CIRCULATIONAHA.106.675934
16. Foley JD, Moliterno DJ. Contemporary occurrence of stent thrombosis in clinical practice: Better never than late. *Catheter Cardiovasc Interv*. 2012;79(4):557. doi:10.1002/ccd.24343
17. Tada T, Byrne RA, Simunovic I, et al. Risk of stent thrombosis among drug-eluting and bare-metal stents in patients with and without diabetes mellitus: A patient-level pooled analysis of randomized trials. *J Am Coll Cardiol*. 2013;61(19):2111-2118. doi:10.1016/j.jacc.2013.01.057
18. Lechner K, McKenzie AL, Kränkel N, et al. High-risk atherosclerosis and metabolic phenotype: The roles of ectopic adiposity, atherogenic dyslipidemia, and inflammation. *Metab Syndr Relat Disord*. 2020;18(4):176. doi:10.1089/met.2019.0115