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Original Article

The pattern and characteristics of acute coronary events in patients after percutaneous coronary intervention (PCI)

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Abstract

Objective: While PCI achieves high procedural success rates, persistent risks such as periprocedural thrombosis, inflammatory cascades, and myocardial hypoperfusion contribute to a bimodal pattern of early and late complications, underscoring gaps in current risk prediction models. This research focuses on identifying the risk factors contributing to recurrent coronary events, particularly stent thrombosis and in-stent restenosis, in patients admitted to the cardiac emergency department.

Methods: A retrospective cohort study was conducted on 204 patients at Shahid Rajaei Hospital to analyze their clinical records, including demographic data, cardiovascular risk factors, and treatment history. Data collection utilized a structured checklist encompassing demographics, cardiovascular risk factors, treatment history, procedural details, and outcomes. Statistical analyses (PRISM version 9.0) included descriptive statistics and multivariable logistic regression to assess predictors of outcomes.

Results: Multivariate logistic regression analysis revealed that for same-artery re-involvement, the duration after first myocardial infarction was significantly associated with increased risk ($P < 0.0001$, OR = 3.666). Regarding stent thrombosis, multiple factors including duration after myocardial infarction ($P < 0.001$), not taking Plavix ($P = 0.033$), MI type ($P = 0.001$), diabetes mellitus ($P = 0.002$), cigarette smoking ($P = 0.029$), and absence of family history ($P = 0.03$) all demonstrated significant effect. For in-stent restenosis, only the absence of diabetes ($P = 0.018$, OR = 0.432) was significantly associated with decreased likelihood of this outcome.

Conclusion: The study also emphasizes the importance of early detection, regular follow-ups, and effective management strategies, such as pharmacological intervention and lifestyle changes, to reduce the risk of adverse cardiac outcomes. Additionally, it was found that a prolonged duration since the initial myocardial infarction significantly elevates the risks of same-artery re-occlusion and stent thrombosis.

Keywords: Percutaneous coronary intervention (PCI), Acute coronary syndrome, Heart disease risk factors, Angioplasty

Introduction

Primary percutaneous coronary intervention (PCI) for the treatment of ST-segment elevation myocardial infarction (STEMI) is superior to thrombolytic therapy (1, 2). While procedural success rates have reached acceptable levels (3), significant challenges persist. Despite innovations in treatment strategies, patients with acute coronary syndrome (ACS) still experience significantly higher rates of coronary artery-related cardiac events, especially during PCI and within the first 30 days, compared to those with stable coronary artery disease (4). A recent report estimated that up to 16% of patients experience adverse cardiovascular events within the first year post-PCI (5).

It has also been shown that PCI induces a systemic inflammatory response and may contribute to long-term consequences, including the occurrence of restenosis (6). Additionally, myocardial tissue hypoperfusion persists after primary PCI in a significant proportion of patients despite the restoration of epicardial coronary circulation. The leading cause of this phenomenon is severe microvascular dysfunction or loss of integrity, leading to microvascular obstruction (7). However, the magnitude of myocardial damage after PCI varies greatly depending on clinical, angiographic, and pharmacotherapy characteristics, biomarkers, and the threshold applied for diagnosis (8).

Patients undergoing PCI are at risk of coagulation activation, intravascular thrombosis, and subsequent myocardial infarction (MI)(9). These periprocedural thrombotic events may occur due to vascular wall damage secondary to intervention or from thrombosis formation on equipment used for PCI (10). Periprocedural events

are associated with complications and mortality. Thus, adjunctive pharmacology is used during PCI to prevent thrombosis by inhibiting platelet activation and thrombin formation (11) .

Post-PCI complications follow a bimodal timeline: early events (<6 months) stem from procedural factors like neointimal hyperplasia (9), whereas late events arise de novo (9). Current prediction tools, however, remain inadequate. Most models focus narrowly on short-term outcomes or mortality , neglecting dynamic variables such as inflammatory biomarkers and ECG parameters (12-14).

Additionally, risk stratification is a valuable tool for planning early discharge and rehabilitation after STEMI (12). The rapid advancement of cardiac interventional measures ensures that predicting major cardiac event complications remains a goal for any risk classification model. Many models have been invented to predict adverse outcomes after PCI; currently, few of these models have been validated in bare-metal stents, but were only designed to predict mortality (13). Numerous research efforts have focused on creating predictive models for post-PCI clinical outcomes by employing traditional statistical approaches like logistic regression and machine learning techniques. However, these PCI prediction models have mainly focused on in-hospital or short-term outcomes (14).

Despite the high success rate of PCI in the management of acute coronary syndromes, the significant incidence of cardiac events after PCI, particularly in patients with uncontrolled metabolic risk factors, highlights the need to develop more accurate predictive tools and personalized approaches. This study highlights the need to revise current risk assessment models by identifying the key contributions of modifiable factors (e.g., adherence to treatment, glycemic control, and blood pressure) and non-modifiable factors (e.g., increasing age).

Methods

This retrospective cohort study analyzed the clinical records of 204 patients with acute coronary syndrome (ACS) and a history of percutaneous coronary intervention (PCI) with stenting admitted to the cardiac emergency department of Shahid Rajaei Hospital between 2021 and 2022. Inclusion criteria comprised: 1) confirmed ACS diagnosis (unstable angina or myocardial infarction) via ECG and angiography by a cardiologist; 2) documented history of PCI with stenting; and 3) availability of complete angiographic reports and medication records. Exclusion criteria included: 1) non-ACS diagnoses (e.g., non-cardiac chest pain); 2) incomplete medical records or missing key data (e.g., angiographic reports); and 3) the patient's refusal to participate.

Data were collected using a structured checklist developed from clinical guidelines for ACS and PCI complications. Variables included demographics (age, gender, and education), cardiovascular risk factors (hypertension, diabetes, smoking, and hypercholesterolemia), treatment history (antiplatelet agents and statins), procedural details (time since prior PCI), and outcome measures (stent thrombosis, restenosis, and involved vessel location).

Statistical analyses were performed using PRISM software (version 9.0). Descriptive statistics summarized categorical variables (e.g., sex and diabetes) as frequencies and percentages. Multiple logistic regression assessed crude associations between predictors (e.g., age and Plavix discontinuation) and outcomes (stent thrombosis and restenosis), reporting odds ratios (ORs), 95% confidence intervals (CIs), and area under the receiver operating characteristic curve values. The study protocol was approved by Kerman University of Medical Sciences Ethics Committee (IR.KMU.REC.1404.018), and patient confidentiality was maintained through anonymized data collection.

Results

Multivariable logistic regression analysis was conducted to examine predictors of reinfarction in the same artery. Among all variables examined, duration after first myocardial infarction (MI) showed high statistical significance ($P<0.001$), with longer duration strongly associated with increased risk of reinfarction in the same artery (OR = 3.66; 95% CI, 2.16–6.61). Additionally, family history approached statistical significance ($P=0.054$), with an odds ratio of 5.09 (95% CI, 1.14–36.6). The model demonstrated good discrimination ability (area under the ROC curve = 0.78; 95% CI, 0.72–0.86; $P<0.001$) and relatively good explanatory power (Tjur's $R^2=0.256$). The Hosmer-Lemeshow test confirmed good model fit ($P=0.714$). The overall classification accuracy of the model was 71.5%, with a sensitivity of 80.7% and specificity of 61.9%. The positive predictive value was 68.9% and the negative predictive value was 75.4%, indicating balanced classification capability (Table 1).

Multivariable logistic regression analysis was conducted to examine predictors of stent thrombosis. The analysis identified several statistically significant variables. Duration after first myocardial infarction with high statistical significance ($P<0.001$) was associated with decreased risk of stent thrombosis (OR = 0.16; 95% CI, 0.06–0.35). Not taking Plavix was statistically significant ($P=0.033$) and associated with increased risk of stent thrombosis (OR = 6.62; 95% CI, 1.35–46.33). MI type (increase in its severity) also showed significant statistical significance ($P=0.033$), associated with increased risk of stent thrombosis (OR = 2.06; 95% CI, 1.08–4.17). Diabetes mellitus was significantly associated with an increased risk of stent thrombosis ($P=0.001$). In contrast, the absence of diabetes markedly reduced the likelihood of thrombosis (OR = 0.10; 95% CI, 0.02–0.33). Cigarette

Table 1. Multivariable logistic regression analysis for predictors of same artery re-occlusion

Variable		Patients	Estimate (SE)	95% CI (estimate)	Odds ratio	95% CI (OR)	*P* value
Age			0.0001 (0.0383)	-0.07–0.07	1.00	0.92–1.07	0.997
Duration after MI			1.299 (0.282)	0.77–1.88	3.66	2.16–6.61	<0.001
Plavix	Y	59	-0.488 (0.762)	-2.01–0.99	0.61	0.13to 2.70	0.522
	N	147					
MI type	Unstable angina	93	0.269 (0.204)	-0.12–0.67	1.30	0.88–1.97	0.188
	Stemi	40					
	None stemi	69					
Diabetes (DM)	Y	64	-0.535 (0.391)	-1.31–0.22	0.58	0.26–1.25	0.171
	N	138					
Hypertension (Htn)	Y	158	0.798 (0.688)	-0.54–2.17	2.22	0.58–8.77	0.246
	N	44					
Cigarette	Y	61	0.032 (0.409)	-0.78–0.82	1.03	0.45–2.29	0.938
	N	141					
EF			-0.0149 (0.0238)	-0.06–0.03	0.98	0.93–1.03	0.531
Dyslipidemia	Y	65	-0.214 (0.391)	-0.99–0.55	0.80	0.37–1.73	0.584
	N	137					
Family history	Y	15	1.628 (0.846)	0.13–3.60	5.09	1.14–36.62	0.054
	N	187					

Model performance:
AUC (95% CI): 0.79 (0.72–0.86)
Tjur's R^2: 0.26
Hosmer-Lemeshow test: $P = 0.71$ (good fit)
Overall classification accuracy: 71.5%

smoking was statistically significant ($P = 0.043$), and non-smoking was associated with decreased risk of stent thrombosis (OR = 0.33; 95% CI, 0.11–0.95). Additionally, absence of family history was statistically significant ($P = 0.002$) and associated with decreased risk of stent thrombosis (OR = 0.05; 95% CI, 0.01–0.28). The model demonstrated excellent discrimination ability (area under the ROC curve = 0.920; 95% CI, 0.87–0.96; $p < 0.0001$) and high explanatory power (Tjur's $R^2 = 0.483$). The Hosmer-Lemeshow test confirmed excellent model fit ($P = 0.996$). The overall classification accuracy of the model was very good (88.4%), with high specificity (96.3%) but relatively lower sensitivity (58.3%). The positive predictive value was 80.8% and the negative predictive value was high at 89.7% (Table 2).

Multivariable logistic regression analysis, including demographic factors, clinical characteristics, and cardiac risk factors, was performed. Among all variables examined, only the absence of diabetes showed statistical significance ($P = 0.018$), associated with a decrease in the likelihood of in-stent restenosis occurrence (OR = 0.43; 95% CI, 0.21–0.86). Duration after first myocardial infarction (MI) approached statistical significance ($P = 0.055$), with longer duration associated with increased risk of restenosis (OR = 1.60; 95% CI, 1.01–2.65). The model demonstrated moderate discrimination ability (area under the ROC curve = 0.672; 95% CI, 0.59–0.75; $P = 0.000$) but relatively low overall explanatory power (Tjur's $R^2 = 0.088$). The Hosmer-Lemeshow test indicated adequate model fit ($P = 0.161$). The overall classification accuracy of the model was 64.5%, with a sensitivity of 33.9% and specificity of 83.2% (Table 3).

The study's findings revealed several significant associations. For same-artery reinfarction, a longer duration after the first myocardial infarction (MI) was strongly associated with increased risk, and family history approached significance. For stent thrombosis, significant predictors included shorter duration after first MI, not taking Plavix, more severe MI type, diabetes, smoking, and absence of family history. For in-stent restenosis, only the absence of diabetes was significant, and duration after MI approached significance. The models showed varying discrimination abilities, and stent thrombosis had the highest predictive accuracy, followed by same-artery reinfarction and in-stent restenosis.

Discussion

The study's findings revealed several significant associations: For same-artery reinfarction, a longer duration after the first myocardial infarction (MI) was strongly associated with increased risk, and family history approached significance. For stent thrombosis, the significant predictors included shorter duration after first MI, not taking Plavix, more severe MI type, diabetes, smoking, and the absence of family history. For in-stent restenosis, only the absence of diabetes was significant,

Table 2. Multivariable Logistic Regression Analysis for Predictors of Stent Thrombosis

Variable		Patients	Estimate (SE)	95% CI for β	OR estimate	95% CI for OR	*P* value
Age			-0.0144 (0.0460)	-0.10–0.076	0.98	0.89–1.08	0.75
Duration after MI			-1.784 (0.418)	-2.70—1.04	0.16	0.06–0.35	<0.001
Plavix	Y	59	1.891 (0.884)	0.30–3.83	6.62	1.35–46.3	0.033
	N	147					
MI type	Unstable angina	93	0.725 (0.340)	0.08–1.42	2.06	1.08–4.17	0.033
	Stemi	40					
	None stemi	69					
Diabetes mellitus	Y	64	-2.283 (0.647)	-3.67—1.09	0.10	0.02–0.33	<0.001
	N	138					
Hypertension	Y	158	0.503 (0.889)	-1.23to 2.29	1.65	0.29–9.91	0.572
	N	44					
Cigarette smoking	Y	61	-1.109 (0.549)	-2.22—0.05	0.33	0.11–0.95	0.043
	N	141					
Ejection Fraction			-0.0109 (0.0364)	-0.08–0.05	0.98	0.92–1.06	0.765
Dyslipidemia	Y	65	-0.0446 (0.619)	-1.27–1.18	0.95	0.28–3.27	0.943
	N	137					
Family history of CAD	Y	15	-2.990 (0.942)	-5.03—1.27	0.05	0.01–0.28	0.002
	N	187					

Model performance:
AUC (95% CI): 0.92 (0.88–0.96)
Tjur's R^2: 0.483
Hosmer-Lemeshow test: $P = 0.996$ (good fit)
Overall classification accuracy: 88.4%

Table 3. Results of Multivariable Logistic Regression for Predicting In-Stent Restenosis

Variable		Patients	Estimate (SE)	95% CI for β	OR (odds ratio)	95% CI for OR	*P* value
Age			0.0080 (0.0358)	-0.062–0.08	1.00	0.94–1.08	0.823
Duration after MI			0.4735 (0.2465)	0.01–0.97	1.60	1.01–2.65	0.054
Plavix	Y	59	-0.3024 (0.7181)	-1.72–1.10	0.73	0.17–3.01	0.673
	N	147					
MI Type	Unstable angina	93	0.0890 (0.1883)	-0.28–0.46	1.09	0.75–1.58	0.636
	Stemi	40					
	None stemi	69					
Diabetes mellitus (DM)	Y	64	-0.8393 (0.3548)	-1.54—0.14	0.43	0.21–0.86	0.018
	N	138					
Hypertension (HTN)	Y	158	0.3754 (0.6321)	-0.86–1.62	1.45	0.42–5.08	0.552
	N	44					
Cigarette smoking	Y	61	-0.0366 (0.3810)	-0.78–0.71	0.96	0.45–2.04	0.923
	N	141					
Ejection fraction (EF)			-0.0094 (0.0227)	-0.05–0.03	0.99	0.94–1.03	0.678
Dyslipidemia	Y	65	0.1959 (0.3691)	-0.51to 0.93	1.21	0.59–2.54	0.595
	N	137					
Family history	Y	15	1.073 (0.8123)	-0.35–2.9	2.92	0.7–19.97	0.186
	N	187					

Model performance:
AUC (95% CI): 0.67 (0.59–0.75)
Tjur's R^2: 0.088
Hosmer-Lemeshow test: $P = 0.16$ (good fit)
Overall classification accuracy: 64.5%

and duration after MI approached significance. The models showed varying discrimination abilities, and stent thrombosis had the highest predictive accuracy, followed by same-artery reinfarction and in-stent restenosis.

The duration after the first myocardial infarction (MI) was the strongest predictor of same-artery re-involvement ($P < 0.0001$). This aligns with previous studies linking increased risk of recurrent ischemic events to time elapsed post-MI (15-17). A notable proportion of patients exhibited vascular complications, with 12% showing progression in previously stenosed vessels and 10% developing new vessel stenoses, both contributing to early recurrent myocardial infarction (18). This association may be due to progressive atherosclerosis at the prior injury site, gradual decline in antiplatelet therapy efficacy, or patient-related factors (e.g., poor adherence to treatment). Family history, though marginally significant ($P = 0.054$), showed a high odds ratio, suggesting a potential role of shared genetic or environmental factors.

The two main reasons for treatment failure with stents are stent thrombosis (ST) and in-stent restenosis (ISR) (19). In our study, duration after first MI was inversely associated with stent thrombosis risk ($P < 0.001$), contrasting with the findings for same-artery re-involvement. However, a previous study highlighted that the risk of stent thrombosis during long-term follow-up was increased significantly (20). This discrepancy may arise from variations in study populations, stent types, or differences in adjunct antithrombotic therapy regimens. Also, Plavix has been identified as a crucial risk-reducing agent (21). We observed that patients who did not take Plavix in the first six months after PCI were associated with an increased risk of thrombosis, which was statistically significant. Bastiaan Zwart, in a multicenter study of a large population, reported that discontinuing Plavix in the first six months after PCI increases the risk of stent thrombosis (22). Diabetes ($P = 0.002$) and MI type ($P = 0.001$) emerged as strong risk factors, consistent with prior evidence on hyperglycemia (23) and systemic inflammation in thrombogenesis (24). Notably, no smoking was linked to reduced stent thrombosis risk ($P = 0.951$). The studies show that smoking creates a prothrombotic (clotting) environment by inducing systemic inflammation, platelet activation, and endothelial dysfunction, which significantly increases the risk of stent thrombosis (25, 26). Chen et al. demonstrated that patients who quit smoking before or after PCI are associated with a lower risk of mortality, and a reduction in mortality was also found in persistent smokers who attempted to decrease their cigarette consumption (27). Clinically, these findings support tighter glycemic control, personalized anti-inflammatory strategies, and antithrombotic agents in high-risk groups.

Absence of diabetes was identified as a protective factor against in-stent restenosis ($P = 0.018$), likely due to the detrimental effects of hyperglycemia on vascular smooth muscle cell proliferation(28, 29). This aligns with studies attributing restenosis to chronic inflammation and neointimal hyperplasia (30, 31). Another study revealed that diabetic patients exhibited an in-stent restenosis (ISR) incidence rate of up to 20% following drug-eluting stent (DES) implantation (32). The time course of the first MI showed a trend toward an increase in in-stent restenosis risk ($P = 0.055$). However, the model's weaker performance shows that unmeasured factors may have played significant roles. Several studies have shown that the greatest reduction in lumen diameter in stents occurs within the first 6–8 months after stenting (31, 33).

The findings of this study have important practical implications for clinical care and health policy. It is essential to design structured protocols for high-risk patients (e.g., elderly or diabetic) to have monthly visits in the first 6 months after PCI and to measure HbA1c regularly. It is also recommended to develop educational programs to educate patients about the importance of taking medications, quitting smoking, and eating a healthy diet, especially in underserved areas. Incorporating biomarkers (e.g., CRP or polymorphisms) into clinical decision-making can help personalize antiplatelet therapy. Finally, health policymakers should prioritize insurance coverage of highly effective antiplatelet drugs and lifestyle counseling sessions. These strategies will not only bridge the gap between research and clinical practice but also reduce the burden of cardiovascular disease in vulnerable populations.

This study has several limitations. Due to the retrospective nature of this study, some factors (incomplete records of medication adherence and lifestyle factors were not taken into account. The modest sample size ($N = 204$) restricts statistical power for detecting rare outcomes and subgroup analyses. Furthermore, we did not account for genetic predispositions or inflammatory biomarkers, which may influence post-PCI outcomes. Future prospective multicenter studies with larger cohorts, extended surveillance, molecular data, and biomarker integration are warranted to address these gaps.

Conclusion

In summary, this study identifies key predictors of adverse coronary events post-PCI. Prolonged duration since initial myocardial infarction significantly elevates risks of same-artery re-occlusion and stent thrombosis, while diabetes markedly increases susceptibility to stent thrombosis and in-stent restenosis. Critically, non-adherence to Pelavix therapy was associated with a rise in recurrent events. These findings underscore the imperative for stringent glycemic control, unwavering antiplatelet regimen compliance, and individualized risk stratification incorporating both modifiable (e.g., diabetes management and smoking cessation) and non-modifiable

factors. Structured follow-up protocols, prioritizing early symptom vigilance, routine biomarker monitoring, and patient education, are essential to mitigate recurrent events. Future research should validate these predictors in prospective cohorts and explore molecular mechanisms underlying observed associations.

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Validation: Ata Firouz.
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Competing Interests
The authors declare that they have no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

Ethical Approval
This study was approved by the Ethics Committee of Kerman University of Medical Sciences (ethical code: IR.KMU.REC.1404.018).

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References
1. Vogel B, Claessen BE, Arnold SV, Chan D, Cohen DJ, Giannitsis E, et al. ST-segment elevation myocardial infarction. Nat Rev Dis Primers. 2019;5(1):39. doi: 10.1038/s41572-019-0090-3.
2. Pascual I, Avanzas P, Almendárez M, Lorca R, Vigil-Escalera M, Arboine L, et al. STEMI, primary percutaneous coronary intervention and recovering of life expectancy: insights from the SurviSTEMI study. Rev Esp Cardiol (Engl Ed). 2021;74(10):829-37. doi: 10.1016/j.rec.2020.08.008.
3. Nafasi L, Rahmani R, Shafiee A, Salari A, Abdollahi A, Meysamie A. Can a high reloading dose of atorvastatin prior to percutaneous coronary intervention reduce periprocedural myocardial infarction? Curr Med Res Opin. 2014;30(3):381-6. doi: 10.1185/03007995.2013.834249.
4. Sawano M, Kohsaka S, Ishii H, Numasawa Y, Yamaji K, Inohara T, et al. One-year outcome after percutaneous coronary intervention for acute coronary syndrome - an analysis of 20,042 patients from a Japanese nationwide registry. Circ J. 2021;85(10):1756-67. doi: 10.1253/circj.CJ-21-0098.
5. Fanaroff AC, Zakroysky P, Wojdyla D, Kaltenbach LA, Sherwood MW, Roe MT, et al. Relationship between operator volume and long-term outcomes after percutaneous coronary intervention. Circulation. 2019;139(4):458-72. doi: 10.1161/circulationaha.117.033325.
6. Clare J, Ganly J, Bursill CA, Sumer H, Kingshott P, de Haan JB. The mechanisms of restenosis and relevance to next generation stent design. Biomolecules. 2022;12(3):430. doi: 10.3390/biom12030430.
7. Niccoli G, Scalone G, Lerman A, Crea F. Coronary microvascular obstruction in acute myocardial infarction. Eur Heart J. 2016;37(13):1024-33. doi: 10.1093/eurheartj/ehv484.
8. Ganesha Babu G, Malcolm Walker J, Yellon DM, Hausenloy DJ. Peri-procedural myocardial injury during percutaneous coronary intervention: an important target for cardioprotection. Eur Heart J. 2011;32(1):23-31. doi: 10.1093/eurheartj/ehq393.
9. Mangiacapra F, Bressi E, Di Gioia G, Pellicano M, Di Serafino L, Peace AJ, et al. Coronary microcirculation and peri-procedural myocardial injury during elective percutaneous coronary intervention. Int J Cardiol. 2020;306:42-6. doi: 10.1016/j.ijcard.2019.12.042.
10. Nakano M, Yahagi K, Otsuka F, Sakakura K, Finn AV, Kutys R, et al. Causes of early stent thrombosis in patients presenting with acute coronary syndrome: an ex vivo human autopsy study. J Am Coll Cardiol. 2014;63(23):2510-20. doi: 10.1016/j.jacc.2014.02.607.
11. Cavender MA, Bhatt DL, Stone GW, White HD, Steg PG, Gibson CM, et al. Consistent reduction in periprocedural myocardial infarction with cangrelor as assessed by multiple definitions: findings from CHAMPION PHOENIX (Cangrelor Versus Standard Therapy to Achieve Optimal Management of Platelet Inhibition). Circulation. 2016;134(10):723-33. doi: 10.1161/circulationaha.115.020829.
12. Pfoh ER, Hamilton A, Hu B, Stilphen M, Rothberg MB. The six-clicks mobility measure: a useful tool for predicting discharge disposition. Arch Phys Med Rehabil. 2020;101(7):1199-203. doi: 10.1016/j.apmr.2020.02.016.
13. Moscucci M, Kline-Rogers E, Shareet al D. Simple bedside additive tool for prediction of in-hospital mortality after percutaneous coronary interventions. ACC Curr J Rev. 2002;11(1):59. doi: 10.1016/s1062-1458(01)00578-5.
14. Wang J, Wang S, Zhu MX, Yang T, Yin Q, Hou Y. Risk prediction of major adverse cardiovascular events occurrence within 6 months after coronary revascularization: machine learning study. JMIR Med Inform. 2022;10(4):e33395. doi: 10.2196/33395.
15. Abtan J, Bhatt DL, Elbez Y, Sorbets E, Eagle K, Ikeda Y, et al. Residual ischemic risk and its determinants in patients with previous myocardial infarction and without prior stroke or TIA: insights from the REACH registry. Clin Cardiol. 2016;39(11):670-7. doi: 10.1002/clc.22583.
16. Li S, Peng Y, Wang X, Qian Y, Xiang P, Wade SW, et al. Cardiovascular events and death after myocardial infarction or ischemic stroke in an older Medicare population. Clin Cardiol. 2019;42(3):391-9. doi: 10.1002/clc.23160.
17. Lee SH, Jeong MH, Ahn JH, Hyun DY, Cho KH, Kim MC, et al. Predictors of recurrent acute myocardial infarction despite successful percutaneous coronary intervention. Korean J Intern Med. 2022;37(4):777-85. doi: 10.3904/kjim.2021.427.
18. Nair R, Johnson M, Kravitz K, Huded C, Rajeswaran J, Anabila M, et al. Characteristics and outcomes of early recurrent myocardial infarction after acute myocardial infarction. J Am Heart Assoc. 2021;10(16):e019270. doi: 10.1161/jaha.120.019270.
19. Byrne RA, Joner M, Kastrati A. Stent thrombosis and restenosis: what have we learned and where are we going? The Andreas Grüntzig Lecture ESC 2014. Eur Heart J. 2015;36(47):3320-31. doi: 10.1093/eurheartj/ehv511.
20. Doyle B, Rihal CS, O'Sullivan CJ, Lennon RJ, Wiste HJ, Bell M, et al. Outcomes of stent thrombosis and restenosis during extended follow-up of patients treated with bare-metal coronary stents. Circulation. 2007;116(21):2391-8. doi: 10.1161/circulationaha.107.707331.
21. Miyazaki Y, Suwannasom P, Sotomi Y, Abdelghani M,

Tummala K, Katagiri Y, et al. Single or dual antiplatelet therapy after PCI. Nat Rev Cardiol. 2017;14(5):294-303. doi: 10.1038/nrcardio.2017.12.
22. Zwart B, Godschalk TC, Kelder JC, Ten Berg JM. High risk of stent thrombosis in the first 6 months after coronary stenting: do not discontinue clopidogrel early after ACS. J Interv Cardiol. 2017;30(5):421-6. doi: 10.1111/joic.12413.
23. Nusca A, Piccirillo F, Bernardini F, De Filippis A, Coletti F, Mangiacapra F, et al. Glycaemic control in patients undergoing percutaneous coronary intervention: what is the role for the novel antidiabetic agents? A comprehensive review of basic science and clinical data. Int J Mol Sci. 2022;23(13):7261. doi: 10.3390/ijms23137261.
24. Triggle CR, Ding H, Marei I, Anderson TJ, Hollenberg MD. Why the endothelium? The endothelium as a target to reduce diabetes-associated vascular disease. Can J Physiol Pharmacol. 2020;98(7):415-30. doi: 10.1139/cjpp-2019-0677.
25. Sivagangailakshmi V, Rajkumar D. Effects of cigarette smoking on coagulation profile among smokers. Int Arch Integr Med. 2017;4(8):116-20.
26. Pujani M, Chauhan V, Singh K, Rastogi S, Agarwal C, Gera K. The effect and correlation of smoking with platelet indices, neutrophil lymphocyte ratio and platelet lymphocyte ratio. Hematol Transfus Cell Ther. 2021;43(4):424-9. doi: 10.1016/j.htct.2020.07.006.
27. Chen T, Li W, Wang Y, Xu B, Guo J. Smoking status on outcomes after percutaneous coronary intervention. Clin Cardiol. 2012;35(9):570-4. doi: 10.1002/clc.22004.
28. Xia J, Li Q, Liu Y, Ren Q, Gao J, Tian Y, et al. A GLP-1 analog liraglutide reduces intimal hyperplasia after coronary stent implantation via regulation of glycemic variability and NLRP3 inflammasome/IL-10 signaling in diabetic swine. Front Pharmacol. 2020;11:372. doi: 10.3389/fphar.2020.00372.
29. Ferraro RA, van Rosendael AR, Lu Y, Andreini D, Al-Mallah MH, Cademartiri F, et al. Non-obstructive high-risk plaques increase the risk of future culprit lesions comparable to obstructive plaques without high-risk features: the ICONIC study. Eur Heart J Cardiovasc Imaging. 2020;21(9):973-80. doi: 10.1093/ehjci/jeaa048.
30. Jakubiak GK, Pawlas N, Cieślar G, Stanek A. Pathogenesis and clinical significance of in-stent restenosis in patients with diabetes. Int J Environ Res Public Health. 2021;18(22):11970. doi: 10.3390/ijerph182211970.
31. Ullrich H, Olschewski M, Münzel T, Gori T. Coronary in-stent restenosis: predictors and treatment. Dtsch Arztebl Int. 2021;118(38):637-44. doi: 10.3238/arztebl.m2021.0254.
32. Wang JL, Qin Z, Wang ZJ, Shi DM, Liu YY, Zhao YX, et al. New predictors of in-stent restenosis in patients with diabetes mellitus undergoing percutaneous coronary intervention with drug-eluting stent. J Geriatr Cardiol. 2018;15(2):137-45. doi: 10.11909/j.issn.1671-5411.2018.02.011.
33. Alfonso F, Coughlan JJ, Giacoppo D, Kastrati A, Byrne RA. Management of in-stent restenosis. EuroIntervention. 2022;18(2):e103-23. doi: 10.4244/eij-d-21-01034.