

Original Article

The Prognostic Value of Residual Gensini Score on 1-Year Cardiac Mortality in Patients Undergoing Percutaneous Coronary Intervention

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Highlights

- The residual Gensini score is an independent predictor of 1-year cardiac mortality following PCI.
- A graded relationship exists between increasing rGensini and higher cardiac mortality risk.
- The rGensini score effectively identifies high-risk patients in the vulnerable early post-PCI period for intensified management.

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ABSTRACT

Background: Percutaneous coronary intervention (PCI) is a cornerstone in the management of obstructive coronary artery disease. The Gensini score quantitatively assesses the severity and complexity of coronary lesions. The residual Gensini score (rGensini), measured after PCI, may offer superior prognostic information compared with the baseline score. This study aimed to investigate the association between rGensini and cardiac mortality.

Methods: In this study, all consecutive patients who registered for follow-up at the Afshar Hospital Health Promotion Center within 30 days after PCI were included. The primary outcome was cardiac mortality. Baseline characteristics, comorbidities (diabetes, hypertension, dyslipidemia, and prior cardiac history), smoking status, family history, coronary dominance pattern, and coronary calcification were recorded. Patients were stratified into four risk categories based on rGensini: zero-risk (0), low-risk (>0 to ≤11), moderate-risk (>11 to ≤37), and high-risk (>37). Survival analysis was performed using the Kaplan-Meier method, and independent predictors of cardiac mortality were identified using Cox proportional hazards regression.

Results: The study included 141 patients (85 men and 56 women) with a mean age of 60.67 years. The mean Gensini score decreased from 31.22 at baseline to 18.20 after PCI. Over a median follow-up time of 9 months, 10 cardiac deaths occurred. Kaplan-Meier curves demonstrated a significant gradation in survival probability across the strata. Multivariable Cox regression analysis identified rGensini category, age, coronary calcification, diabetes, and hypertension as independent predictors of cardiac mortality.

Conclusion: This hypothesis-generating study suggests that the rGensini score is a promising and independent predictor of cardiac mortality after PCI.

Keywords: Percutaneous Coronary Intervention; Residual Gensini Score; Cardiac Mortality; Prognosis; Coronary Artery Disease; Comorbidities

Introduction

Coronary artery disease (CAD) remains the leading cause of morbidity and mortality worldwide, imposing a substantial burden on healthcare systems.¹ Percutaneous coronary intervention (PCI) has revolutionized the treatment of obstructive CAD, effectively restoring blood flow in acutely occluded vessels and relieving ischemia in stable settings.² The procedural success of PCI is typically gauged by the restoration of Thrombolysis in Myocardial Infarction (TIMI) flow grade 3 and a significant reduction in angiographic stenosis.³ Nonetheless, it is increasingly recognized that PCI often addresses only the most severe, flow-limiting lesions.⁴

The anatomical extent and functional severity of CAD are critical determinants of prognosis.⁵ Several angiographic scoring systems have been developed to quantify this burden objectively. Among these, the Gensini score stands out for its comprehensive nature, incorporating not only the degree of luminal narrowing but also the anatomical location of the stenosis and the importance of the coronary segment perfused.⁶ A high baseline Gensini score has been consistently correlated with adverse cardiovascular outcomes.⁷

Nevertheless, the clinical focus is shifting from the initial disease burden to the amount of atherosclerosis that persists after revascularization.⁸ This concept, known as the “residual SYNTAX score” in more complex disease, can be analogously applied using the Gensini system as the “residual Gensini score” (rGensini). The rGensini represents the atherosclerotic burden left untreated after PCI and may be a more direct marker of future risk from vulnerable plaques in nonintervened segments.

Despite optimal revascularization, a considerable residual risk for major adverse cardiac events (MACE), including cardiac death, persists.⁹ This residual risk is multifactorial, influenced by a complex interplay of procedural factors, underlying cardiac function, and, most importantly, patient-specific comorbidities.¹⁰ Conditions such as diabetes mellitus, hypertension, and dyslipidemia are not only risk

factors for the development of CAD but also potent accelerators of its progression.¹¹ They contribute to endothelial dysfunction, a proinflammatory state, and the progression of both intervened and nonintervened lesions.¹² The synergy between a high residual anatomical burden, as quantified by rGensini, and the presence of these potent comorbidities is not fully elucidated, particularly in real-world cohorts.

Therefore, the present study aimed to investigate the prognostic impact of the rGensini on cardiac mortality in a consecutive cohort of patients undergoing PCI. We sought to stratify patients based on their postprocedural atherosclerotic burden and analyze their survival. A secondary objective was to identify independent clinical and angiographic predictors of cardiac death, with a specific focus on the role of concomitant diseases, to better risk stratify patients in the contemporary PCI era.

Materials and Methods

Study Design and Population

This investigation was a single-center, retrospective observational cohort study conducted at the Health Promotion Center of Afshar Hospital. The study protocol adhered to the principles of the Declaration of Helsinki and received approval from the Ethics Committee of Shahid Sadoughi University of Medical Sciences (approval No. IR.SSU.SRH.REC.1403.033). The requirement for informed consent was waived because of the retrospective nature of the analysis using anonymized data.

The study population comprised all consecutive adult patients (aged ≥18 y) who underwent a PCI procedure at the center between September 2022 and March 2024. To be included, patients must have registered for a formal follow-up program at the Health Promotion Center within 30 days after the index PCI. This registration was considered a proxy for engagement in postdischarge care.

The exclusion criteria were designed to create a cohort focused on assessing 1-year outcomes in survivors of the initial hospitalization who were accessible for follow-up. Thus, patients were excluded for the following reasons: in-hospital mortality during the index admission;

residence in a different city, making follow-up at the center impractical; or declining to participate in the center's follow-up program for any reason. Patients who died during the index hospitalization were excluded because the primary aim of the study was to assess the prognostic value of rGensini for 1-year cardiac mortality, and including these patients would confound the association by introducing early procedural and periprocedural mortality risks.

Data Collection and Variable Definitions

Data were extracted from the hospital's electronic health records and the dedicated database of the Health Promotion Center. The collected variables included the following:

- **Demographics:** Age and sex.
- **Cardiovascular risk factors:** Current smoking status (defined as active smoking within the past 6 months) and a documented family history of premature CAD (in a first-degree relative before age 55 for men and 65 for women).
- **Comorbidities:** The presence of hypertension, diabetes mellitus, dyslipidemia, and a prior history of cardiac disease (including myocardial infarction, heart failure, or prior revascularization). Each comorbidity was defined by the patient's regular use of prescribed medications for that condition, as documented in the medical chart.
- **Coronary dominance pattern:** Classified as right-dominant, left-dominant, or codominant based on the origin of the posterior descending artery.
- **Coronary artery calcification:** Visually assessed as present or absent on angiographic cine images, defined by the appearance of radiopacities within the coronary vessel wall.
- **Gensini score calculation:** The Gensini score was calculated for each patient before PCI (baseline score) and after the procedure (rGensini). The scoring system assigns a severity multiplier to the degree of luminal stenosis (1 for 1%–25%, 2 for 26%–50%, 4 for 51%–75%, 8 for 76%–90%, 16 for 91%–99%, and 32 for total occlusion). This multiplier is then weighted by a factor based on the anatomical significance of the coronary segment involved (eg, 5 for the left main

coronary artery and 2.5 for the proximal left anterior descending artery). The scores for all lesions were summed to obtain a total score.¹³

All angiograms were reviewed and scored by two experienced interventional cardiologists who were blinded to the clinical outcomes; discrepancies were resolved by consensus.

• **Outcome measure:** Cardiac mortality was defined as any death attributable to a cardiac cause (eg, acute myocardial infarction, fatal arrhythmia, or decompensated heart failure). The cause of death was confirmed by review of hospital records, death certificates, and physician notes. All endpoint events occurred after hospital discharge from the index PCI admission. All deaths were identified during follow-up at the Afshar Hospital Health Promotion Center to ensure systematic and uniform event capture for the entire cohort.

Statistical Analysis

Patients were stratified into four distinct risk categories based on their post-PCI rGensini score: zero-risk ($\text{rGensini}=0$), low-risk ($0 < \text{rGensini} \leq 11$), moderate-risk ($11 < \text{rGensini} \leq 37$), and high-risk ($37 < \text{rGensini}$). These cutoff points were not statistically derived from the present cohort to avoid circularity. Instead, they were adopted from a prior, comprehensive internal review of Gensini scores from approximately 8000 angiograms performed at our institution over a 2-year period. In that analysis, these specific thresholds were found to correlate with clinically meaningful strata of disease severity as judged by our institution's experienced interventional cardiologists. This predefined, clinically grounded approach was chosen to enhance the external validity and practical utility of our risk stratification model.

Survival curves for time-to-cardiac death across the four rGensini risk categories were constructed using the Kaplan-Meier method, and the differences between the groups were assessed with the log-rank test. To identify independent predictors of cardiac mortality, we employed Cox proportional hazards regression. Variables with a *P* value of less than 0.1 in the univariable analysis, along with age and sex, because of their established clinical relevance,

were considered for inclusion in the multivariable model. Acknowledging the principle of having at least 10 events per predictor variable to ensure model stability, our limited number of events (n=10) constrained the complexity of the final model. Consequently, the results of the multivariable analysis are presented with caution, interpreted as exploratory, and intended to highlight strong associations that warrant validation in larger cohorts. The results are reported as hazard ratios (HRs) with corresponding 95% confidence intervals (CIs). Statistical analyses were performed using SPSS software, version 23.0. A 2-sided *P* value of less than 0.05 was considered statistically significant for all analyses.

Results

Baseline Characteristics

From the initial pool of patients undergoing PCI during the study period, 141 individuals met the inclusion criteria and formed the final study cohort. The baseline demographic, clinical, and angiographic characteristics of the entire cohort are summarized in (Table 1). The population consisted of 85 men (60.3%) and 56 women (39.7%), with a mean (SD) age of 60.67 (10.45) years. The age distribution was as follows: 2 patients (1.4%) were younger than 40 years, 65 (46.1%) were between 40 and 60 years, 69 (48.9%) were between 60 and 80 years, and 5 patients (3.5%) were older than 80 years.

Table 1. Baseline characteristics of the study population stratified by the residual Gensini score categories

Characteristic	Overall (n=141)	Zero-Risk (n=27)	Low-Risk (n=36)	Moderate-Risk (n=60)	High-Risk (n=18)	P
Demographics						
Age, y, mean (SD)	60.67 (10.5)	59.96 (10.8)	57.94 (9.9)	61.62 (9.3)	64.00 (13.7)	0.215*
Male sex, No. (%)	85 (60.3)	13 (48.1)	23 (63.9)	38 (63.3)	11 (61.1)	0.562**
Cardiovascular Risk Factors						
Current smoker, No. (%)	20 (14.2)	4 (14.8)	5 (13.9)	9 (15.0)	2 (11.1)	0.982**
Family history of coronary artery disease, No. (%)	19 (13.5)	2 (7.4)	6 (16.7)	10 (16.7)	1 (5.6)	0.430**
Comorbidities						
Hypertension, No. (%)	96 (68.1)	19 (70.4)	24 (66.7)	46 (76.7)	7 (38.9)	0.022**
Diabetes mellitus, No. (%)	62 (44.0)	10 (37.0)	15 (41.7)	27 (45.0)	10 (55.6)	0.648**
Dyslipidemia, No. (%)	77 (54.6)	15 (55.6)	19 (52.8)	32 (53.3)	11 (61.1)	0.951**
Prior cardiac disease, No. (%)	5 (3.5)	1 (3.7)	1 (2.8)	2 (3.3)	1 (5.6)	0.974**
Angiographic Characteristics						
Coronary dominance, No. (%)						0.412**
Right	129 (91.5)	26 (96.3)	32 (88.9)	53 (88.3)	18 (100)	
Left	8 (5.7)	1 (3.7)	3 (8.3)	4 (6.7)	0 (0)	
Codominant	4 (2.8)	0 (0)	1 (2.8)	3 (5.0)	0 (0)	
Coronary calcification, No. (%)	10 (7.1)	0 (0)	4 (11.1)	4 (6.7)	2 (11.1)	0.332**
Gensini Scores						
Baseline Gensini score, mean (SD)	31.22 (21.5)	16.81 (12.3)	23.75 (15.1)	35.18 (22.0)	49.61 (23.9)	<0.001*
Residual Gensini score, mean (SD)	18.20 (21.9)	0.00 (0.00)	5.72 (3.1)	21.45 (7.1)	58.94 (18.7)	<0.001*

* *P* value derived from 1-way ANOVA

** *P* value derived from the χ^2 or Fisher exact test, as appropriate

Regarding cardiovascular risk factors, 20 patients (14.2%) were current smokers, and 19 (13.5%) reported a family history of CAD. The prevalence of comorbidities, defined by medication use, was high: 96 patients (68.1%) had hypertension, 62 (44.0%) had diabetes mellitus, 77 (54.6%) had dyslipidemia, and 5 (3.5%) had a history of cardiac disease.

Angiographic characterization revealed that the vast majority of patients had a right-dominant

circulation (129 patients [91.5%]), followed by left-dominant (8 [5.7%]) and codominant (4 [2.8%]) patterns. Coronary artery calcification was visually detected in 10 patients (7.1%).

Gensini Scores and Risk Stratification

The mean (SD) baseline Gensini score before PCI was 31.22 (21.45), indicating a moderate-to-severe burden of coronary

atherosclerosis in the cohort. After PCI, the mean (SD) rGensini was significantly reduced to 18.20 (21.85) ($P<0.05$), demonstrating the effectiveness of the revascularization procedure in reducing the anatomical burden.

Based on the predefined rGensini categories, patients were stratified as follows: 27 patients (19.1%) achieved a zero-risk status ($\text{rGensini}=0$), 36 patients (25.5%) were in the low-risk category, 60 patients (42.6%) were in the moderate-risk category, and 18 patients (12.8%) were in the high-risk category. This distribution highlights that a substantial proportion of patients (55.4%) left the catheterization laboratory with a moderate or high residual atherosclerotic burden.

Clinical Outcomes and Mortality Analysis

The total duration of follow-up was a maximum of 12 months with a median of 9 months. During this period, 10 cardiac deaths (7.1%) were recorded. No noncardiac deaths were identified; therefore, the primary outcome was specific, and competing risks from other causes of mortality were not observed. A complete 12-month follow-up, per the study protocol, was achieved for 23 patients (16.3%). Follow-up was incomplete for two primary reasons: passive disengagement, defined as failure to attend scheduled visits and unresponsiveness to follow-up telephone calls (87 patients [61.7%]), and active withdrawal, defined as an explicit declaration of unwillingness to continue participation (21 patients [14.9%]).

A detailed analysis of the 10 deceased patients provides critical insights. The majority were male (8 [80%]). The distribution of deaths across the rGensini risk strata was uneven: four deaths occurred in the high-risk group (4 of 18 [22.2%] mortality rate), five in the moderate-risk group (5 of 60 [8.3%] mortality rate), one in the low-risk group (1 of 36 [2.8%] mortality rate), and none in the zero-risk group. All deceased patients had a right-dominant coronary pattern. Coronary calcification was present in 6 of the 10 patients who died (60%), a notably higher prevalence than in the overall cohort. The ages at the time of death were 60, 65, 66, 68, 68, 72, 74, 74, 77, and 80 years. All 10 patients had both diabetes

mellitus and hypertension. Seven had dyslipidemia, only two were smokers, and none had a prior history of cardiac disease.

Survival Analysis

The Kaplan-Meier survival curves for cardiac mortality, stratified by the four rGensini categories, are presented in (Figure 1). The curves demonstrated a clear and statistically significant separation (log-rank test $P=0.005$). Patients in the zero-risk category experienced excellent survival throughout the follow-up period. The survival probability sequentially decreased for the low-risk, moderate-risk, and high-risk categories, establishing a strong graded relationship between the magnitude of the rGensini score and the risk of cardiac death. The median follow-up time for the cohort was 9 months. At this time point, the overall cardiac survival rate was 85.9%. When stratified by the rGensini categories, the survival rates demonstrated a clear graded relationship: 100% in the zero-risk group, 94.4% in the low-risk group, 83.3% in the moderate-risk group, and 50.0% in the high-risk group.

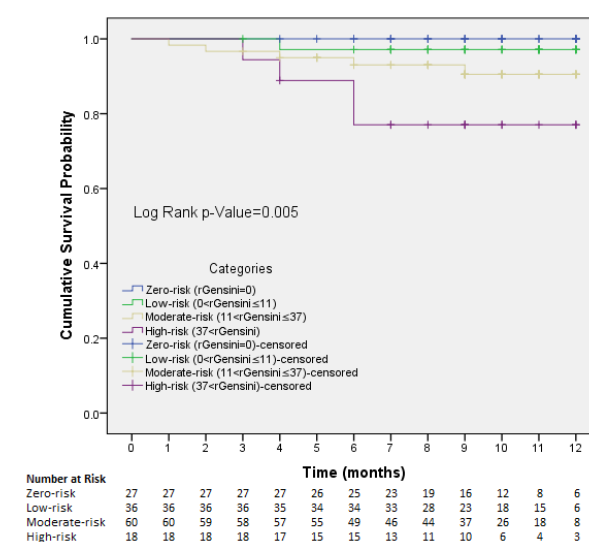


Figure 1. Kaplan-Meier survival curves for cardiac mortality stratified by residual Gensini score risk categories.

Predictors of Cardiac Mortality: Cox Regression Analysis

The proportional hazards assumption for the Cox regression model was verified globally and for each variable using Schoenfeld residuals. The

test results were nonsignificant ($P>0.05$), indicating the assumption was not violated.¹ Univariable Cox regression analysis identified several factors associated with an increased risk of cardiac death, including a higher rGensini category, older age, a family history of CAD, the presence of coronary calcification, diabetes, dyslipidemia, and hypertension. These variables, along with sex and smoking, were included in the multivariable Cox proportional hazards model.

The results of the univariable and multivariable Cox regression analyses are detailed in (Table 2). While the multivariable model identified several independent predictors, the wide CIs for the HRs reflect the limited precision of these estimates because of the small number of events ($n=10$). Consequently, the multivariable model is susceptible to overfitting, and its results should be interpreted as exploratory and hypothesis-generating.

After adjustment for confounding factors, the following variables emerged as independent predictors of cardiac mortality:

- **The rGensini category:** Compared with the zero-risk group, the hazard ratio increased

substantially: low-risk group (HR not calculated because of zero events in the reference group, but the trend was significant), moderate-risk group (HR, 8.45 [95% CI, 1.12 to 63.78]; $P=0.038$), and high-risk group (HR, 15.92 [95% CI, 1.98 to 128.10]; $P=0.009$).

- **Age:** Each 1-year increase in age was associated with a 1.08-fold increased risk of death (HR, 1.08 [95% CI, 1.01 to 1.15]; $P=0.032$).

- **Coronary calcification:** The presence of angiographic calcification conferred a nearly 5-fold increased risk (HR, 4.95 [95% CI, 1.22 to 20.10]; $P=0.025$).

- **Diabetes mellitus:** Diabetes was a powerful independent predictor, with a hazard ratio of 6.12 (HR, 6.12 [95% CI, 1.25 to 29.95]; $P=0.025$).

- **Hypertension:** Similarly, hypertension was independently associated with a significantly elevated risk (HR, 5.84 [95% CI, 1.18 to 28.90]; $P=0.030$).

- **Sex, dyslipidemia, family history of CAD, and smoking status:** These variables did not retain independent predictive value in the final model.

Table 2. Univariable and multivariable Cox proportional hazards regression analysis for predictors of cardiac mortality

Variables*		Univariable Analysis			Multivariable Analysis**		
		HR	95% CI	P	HR	95% CI	P
Residual	zero-risk	1	-	-	1	-	-
Gensini	low-risk	3.85	0.40 - 36.98	0.244	4.25	0.45 - 40.15	0.205
Categories	moderate-risk	7.90	1.05 - 59.40	0.044	8.45	1.12 - 63.78	0.038
	high-risk	14.50	1.81 - 116.10	0.012	15.92	1.98 - 128.10	0.009
age (per year increase)		1.07	1.00 - 1.14	0.048	1.08	1.01 - 1.15	0.032
sex (male vs female)		1.50	0.32 - 7.02	0.605	1.65	0.35 - 7.82	0.525
calcification (yes vs no)		5.50	1.42 - 21.32	0.014	4.95	1.22 - 20.10	0.025
diabetes mellitus (yes vs no)		5.80	1.25 - 26.90	0.024	6.12	1.25 - 29.95	0.025
hypertension (yes vs no)		5.95	1.21 - 29.30	0.028	5.84	1.18 - 28.90	0.030
smoking (yes vs no)		1.30	0.26 - 6.50	0.750	1.42	0.28 - 7.15	0.674
dyslipidemia (yes vs no)		2.91	1.06 - 8.01	0.038	1.85	0.39 - 8.76	0.439
family history of coronary artery disease (yes vs no)		0.30	0.09 - 0.98	0.048	0.98	0.11 - 8.65	0.987

* History of cardiac disease and left/co-dominant flow pattern were not included in regression models because complete separation (absence of these characteristics among deceased participants) prevented valid HR estimation in Cox regression.

**The multivariable model included variables with a P value < 0.10 in univariable analysis or those considered clinically essential (age and sex).

Discussion

In this hypothesis-generating, single-center, retrospective observational cohort study, the rGensini emerged as a strong predictor of cardiac mortality in patients undergoing PCI. Still, these findings must be interpreted with caution. The relatively limited number of outcome events

($n=10$) constrained the complexity and stability of the multivariable Cox regression model. Although the identified associations are strong and clinically plausible, they are preliminary and require validation in larger, prospective cohorts. By stratifying patients into distinct risk categories, a clear gradient of risk was established: patients with a high rGensini score (>37) faced a markedly

worse prognosis than those with lower scores or complete anatomical revascularization (rGensini=0). Moreover, advanced age, the presence of coronary artery calcification, diabetes mellitus, and hypertension were identified as independent copredictors of mortality, compounding the risk associated with a high residual disease burden.

Although the present study assessed cardiac mortality over a mean follow-up of approximately 9 months rather than multiple years, this short-term period holds significant clinical relevance. The early phase following PCI represents a vulnerable period for patients, during which the interplay between residual anatomical burden, procedural sequelae, and underlying comorbidities culminates in the highest risk for adverse events.⁹ The findings demonstrate that the rGensini score effectively stratifies this risk even within the first year. Identifying high-risk patients at this juncture is critical because it allows for the intensification of secondary preventive measures—such as optimizing guideline-directed medical therapy, ensuring closer clinical follow-up, and considering further noninvasive functional testing—at a time when they may have the greatest impact on altering the clinical trajectory.

The principal finding of this study aligns with a growing body of evidence emphasizing the prognostic importance of residual CAD after revascularization. The rGensini score, which is readily calculable from the procedural angiogram, could be integrated into post-PCI risk assessment protocols. It may serve as an anatomical complement to established clinical risk scores, helping to identify a high-risk phenotype that may benefit from intensified management. While the residual SYNTAX score is validated in complex multivessel disease, the rGensini score might be more applicable to a broader PCI population because of its continuous nature and detailed segmental weighting.¹⁴ The current study extends this principle to a broader PCI population using the Gensini scoring system, which offers a continuous and detailed assessment of disease severity. The pathophysiological rationale is intuitive: PCI is a localized treatment, whereas atherosclerosis is a systemic, pan-coronary process.¹⁵ The untreated plaques, particularly those that are vulnerable or prone to progression,

represent a persistent nidus for future acute coronary events.¹⁶ The rGensini score effectively quantifies this ongoing threat. The complete absence of cardiac deaths in the zero-risk category underscores the excellent prognosis associated with achieving complete or near-complete revascularization, a goal that should be pursued when feasible and safe.

The strong independent association of diabetes and hypertension with mortality, even after adjustment for anatomical burden, highlights the critical role of the systemic metabolic and hemodynamic milieu in driving disease progression. Diabetes mellitus induces a state of accelerated atherosclerosis through mechanisms such as chronic hyperglycemia, insulin resistance, and enhanced inflammation, leading to more aggressive and diffuse disease.¹⁷ Hypertension contributes via endothelial shear stress and vascular remodeling.¹⁸ The finding that all 10 deceased patients in this cohort had both conditions is a striking observation that underscores a lethal synergy. It suggests that in patients with a high residual anatomical burden, the concomitant presence of these comorbidities creates a high-risk state for adverse outcomes. This finding reinforces the paramount importance of aggressive risk factor modification beyond the revascularization procedure itself. Pharmacological therapy with statins, antiplatelet agents, angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs), and sodium-glucose cotransporter-2 (SGLT2) inhibitors or glucagon-like peptide-1 (GLP-1) receptor agonists in patients with diabetes is not merely supportive but is central to improving survival.¹⁹

The prognostic significance of coronary artery calcification, identified as an independent predictor, is also noteworthy. Calcification is a marker of advanced, stable plaque burden but is also associated with procedural complexity and suboptimal stent expansion, which can increase the risk of stent thrombosis and restenosis.²⁰ Its presence indicates a more chronic and severe disease state, which aligns with the higher mortality observed.

Several limitations of this study warrant consideration. First, its observational and single-center design introduces the potential for

unmeasured confounding and limits the generalizability of the findings. Second, the sample size, particularly the number of mortality events ($n=10$), though sufficient to demonstrate strong associations, limits the complexity of the multivariable model that could be constructed. A larger, multicenter prospective study would be valuable for validation. Third, the visual assessment of coronary calcification, while common practice, is subjective; more objective measures, such as computed tomography calcium scoring, would provide greater accuracy. Fourth, the variable and relatively short follow-up duration for a significant portion of the cohort limits our assessment of long-term outcomes. Although the early post-PCI period is a vulnerable phase where our findings are highly relevant, longer-term data are essential. Fifth, the exclusion of patients who did not register for the formal follow-up program may have introduced a selection bias, potentially excluding a less healthy or less compliant population. This might lead to an underestimation of the true event rates in an all-comers PCI population. Finally, as noted, the small number of outcome events relative to the number of covariates increases the risk of overfitting in the multivariable model. Accordingly, the results of the regression analysis should be viewed as exploratory.

Conclusion

In conclusion, the present study provides preliminary evidence that the rGensini score is a promising tool for risk stratification in the post-PCI period. It identifies a gradient of risk for short-term cardiac mortality, with the highest risk observed in patients with a substantial residual anatomic burden compounded by comorbidities such as diabetes and hypertension. These findings underscore the importance of a holistic treatment approach, in which PCI is viewed not as a definitive cure but as one component of a comprehensive, long-term secondary prevention strategy. Future research in larger, prospective, multicenter studies is needed to validate these findings and to determine whether the rGensini score-guided therapy improves clinical outcomes.

Declarations:

Ethical Approval

The study protocol was approved by the Ethics Committee of Shahid Sadoughi University of Medical Sciences (code: IR.SSU.SRH.REC.1403.033) and adhered to the principles of the Declaration of Helsinki. Informed consent was waived because of the retrospective nature of the analysis, which used anonymized data.

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Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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