



Predictors of no-reflow phenomenon in patients with type 2 diabetes mellitus undergoing elective percutaneous coronary intervention: the emerging role of the triglyceride–glucose index

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Abstract

Objective: To determine the prevalence and predictors of the no-reflow phenomenon in patients with type 2 diabetes mellitus (T2DM) undergoing elective percutaneous coronary intervention and to evaluate the predictive value of the triglyceride–glucose (TyG) index.

Methods: This prospective observational cohort study included 137 patients with T2DM scheduled for elective percutaneous coronary intervention at two tertiary centres. Patients were classified according to postprocedural Thrombolysis in Myocardial Infarction flow grade into normal reflow and no-reflow groups. Clinical, laboratory, echocardiographic, angiographic and procedural variables were compared. Receiver operating characteristic curve analysis and multivariable logistic regression were performed to assess predictive performance and independent predictors.

Results: The no-reflow phenomenon occurred in 13 patients (9.5%). Patients with no-reflow had a significantly longer diabetes duration, higher fasting blood glucose, random blood glucose, glycated haemoglobin, low-density lipoprotein cholesterol, triglyceride and TyG index values, and lower left ventricular ejection fraction compared with patients with normal reflow. Severe coronary stenosis and pre-dilatation were significantly more frequent in the no-reflow group. The TyG index demonstrated the highest predictive performance for no-reflow (area under the curve [AUC] = 0.870, 95% confidence interval [CI]: 0.753–0.987, $p < 0.001$). A TyG cutoff value of approximately 9.01 yielded 76.9% sensitivity and 89.5% specificity. Multivariable logistic regression identified the TyG index as an independent predictor of no-reflow (odds ratio [OR] = 2.05 per 0.1-unit increase, 95% CI 1.26–3.32, $p = 0.004$), together with duration of diabetes and reduced ejection fraction.

Conclusions: No-reflow occurred in approximately 1 in 10 diabetic patients undergoing elective percutaneous coronary intervention. The TyG index is a simple, inexpensive and effective marker for preprocedural risk stratification.

Keywords

no-reflow phenomenon, percutaneous coronary intervention, type 2 diabetes mellitus, triglyceride-glucose index, insulin resistance

Introduction

Percutaneous coronary intervention (PCI) has become the cornerstone of coronary revascularisation in patients with obstructive coronary artery disease and is associated with significant improvements in myocardial perfusion, symptom burden and cardiovascular outcomes [1,2]. Despite successful restoration of epicardial coronary artery patency, a subset of patients experience inadequate myocardial tissue perfusion, a condition referred to as the no-reflow phenomenon [3]. This complication is characterised by impaired myocardial reperfusion in the absence of mechanical (epicardial) coronary obstruction and reflects obstruction at the microvascular rather than the epicardial level. It is associated with larger infarct size, impaired ventricular function, heart failure, arrhythmias and increased mortality [4]. The phenomenon has been studied predominantly in the context of primary PCI for acute coronary syndromes, particularly ST-elevation myocardial infarction,

where thrombus burden and ischaemia–reperfusion injury are greatest. Its frequency, predictors and clinical relevance in elective PCI for stable coronary artery disease—the setting of the present study—are less well characterised, and the two contexts should not be conflated.

The pathophysiology of no-reflow is multifactorial and involves distal embolization, ischaemia–reperfusion injury, endothelial dysfunction, inflammatory activation, oxidative stress, platelet aggregation and coronary microvascular obstruction [5]. Patients with type 2 diabetes mellitus (T2DM) are particularly vulnerable because of diffuse atherosclerosis, chronic inflammation, endothelial dysfunction, insulin resistance and impaired coronary microvascular integrity [6].

T2DM represents one of the most important global cardiovascular risk factors and is associated with increased morbidity and mortality [7–9]. Persistent hyperglycaemia and insulin resistance contribute to vascular inflammation, oxidative stress and endothelial injury, all of which may predispose to impaired myocardial reperfusion following PCI [10].

The triglyceride–glucose (TyG) index, derived from fasting triglyceride and fasting glucose levels, has emerged as a simple and

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reliable surrogate marker of insulin resistance [11]. Recent studies have demonstrated associations between elevated TyG index and adverse cardiovascular outcomes, coronary artery disease severity and microvascular dysfunction [12]. However, limited data are available regarding its role in predicting no-reflow in diabetic patients undergoing elective PCI [13].

Therefore, the present study aimed to evaluate the prevalence of the no-reflow phenomenon in patients with T2DM undergoing elective PCI and identify clinical, laboratory, angiographic and procedural predictors of no-reflow, with particular emphasis on the predictive value of the TyG index.

Patients and methods

1. Study design and population

This prospective observational cohort study was conducted at the Cardiology Department of Menoufia University Hospital and Shebin El-Kom Teaching Hospital between September 2023 and December 2025. Consecutive patients with T2DM undergoing elective PCI for stable coronary artery disease were prospectively enrolled. A total of 137 diabetic patients undergoing elective PCI and coronary stenting were included after obtaining written informed consent.

2. Sample size calculation

The sample size was estimated using the single-population proportion formula, $n = Z^2 \cdot \frac{p(1-p)}{d^2}$. At the time of study design, robust incidence data for no-reflow specifically among diabetic patients undergoing elective PCI were lacking; the estimate of 22.46% reported by Pantea-Roşan et al. [14] in a diabetic population was, therefore, used as the best available figure. With a 95% confidence level ($Z = 1.96$) and absolute precision $d = 0.07$, the minimum required sample size was 137. We acknowledge that this estimate derives from a primary-PCI/STEMI population and likely overestimates the incidence in the elective setting; the correspondingly lower observed event rate (9.5%) reduced the statistical power available for multivariable modelling.

3. Inclusion criteria

Age ≥ 18 years; confirmed diagnosis of T2DM according to the 2022 American Association of Clinical Endocrinology criteria [15]; scheduled for elective PCI for stable coronary artery disease.

4. Exclusion criteria

Acute coronary syndrome; structural heart disease; advanced heart failure and/or pulmonary oedema; severe renal impairment ($\text{eGFR} < 30 \text{ mL/min/1.73 m}^2$); haematological disorders; active hepatobiliary disease; active infection or inflammatory disease; malignancy; thyroid dysfunction; recent major surgery or trauma; and incomplete clinical or angiographic data.

5. Ethical approval

The study protocol was approved by the local Institutional Ethics Committee (Approval No. 8/2023CARD 11), and all procedures

complied with the Declaration of Helsinki. Written informed consent was obtained from all participants.

6. Clinical and laboratory assessment

All patients underwent comprehensive clinical evaluation, including detailed history taking, physical examination, electrocardiography, transthoracic echocardiography and laboratory investigations. Clinical variables included age, sex, body mass index (BMI), smoking status, hypertension, dyslipidaemia, previous ischaemic heart disease and duration of diabetes. Laboratory investigations included fasting blood glucose, random blood glucose, glycated haemoglobin (HbA1c), lipid profile, complete blood count and serum creatinine.

The TyG index was calculated as: $\text{TyG index} = \ln [(fasting triglycerides [\text{mg/dL}] \times fasting glucose [\text{mg/dL}])/2]$.

7. Angiographic and procedural assessment

Coronary angiography and PCI were performed using radial or femoral access according to operator discretion. Coronary flow was graded using the Thrombolysis in Myocardial Infarction (TIMI) classification on the final angiogram, obtained after stent implantation and any procedural optimisation (post-dilatation). No-reflow was defined as TIMI flow grade ≤ 2 in the absence of angiographically evident mechanical obstruction, dissection or thrombus. When impaired flow was observed, intracoronary nitroglycerine (Tridil) was administered to exclude epicardial spasm; no-reflow was diagnosed only when TIMI flow grade ≤ 2 persisted despite intracoronary vasodilator administration. Adenosine, verapamil and sodium nitroprusside were not routinely used. The degree of coronary stenosis was determined by visual angiographic estimation by the operating interventional cardiologist; quantitative coronary angiography was not performed.

Patients were categorised into Group I (no-reflow, TIMI ≤ 2) and Group II (normal reflow, TIMI 3). Procedural variables included target vessel, degree of stenosis, pre- and post-PCI TIMI flow, stent number, stent diameter, stent length and use of pre-dilatation or post-dilatation.

8. Statistical analysis

Data were analysed using IBM SPSS Statistics and MedCalc software. Continuous variables were expressed as mean $\pm$ standard deviation and compared using the independent-samples *t*-test; categorical variables as frequencies and percentages, compared using the Chi-square or Fisher's exact test as appropriate. Exact *p*-values are reported wherever generated by the software. Receiver operating characteristic (ROC) curve analysis assessed the discriminatory performance of metabolic biomarkers for no-reflow, with the optimal TyG cutoff determined by the Youden index. Because the cutoff was derived and evaluated within the same cohort, internal validation was performed using bootstrap resampling (2000 resamples) to obtain bias-corrected estimates of the area under the curve (AUC) and cutoff. Given the limited number of no-reflow events ($n = 13$), multivariable logistic regression was pre-specified as an exploratory, hypothesis-generating

analysis rather than a clinical prediction model. To respect the events-per-variable constraint, a parsimonious model was fitted, including TyG index, duration of diabetes and left ventricular ejection fraction (LVEF). Pre-dilatation was not entered because all no-reflow events occurred in pre-dilated patients (complete separation), precluding stable maximum-likelihood estimation; this association is described descriptively. A fully adjusted model, additionally including demographic and cardiovascular risk covariates and degree of stenosis, was examined as a sensitivity analysis. A two-sided $p < 0.05$ was considered statistically significant.

Results

1. Baseline characteristics

The study included 137 patients with T2DM undergoing elective PCI, including 103 males (75.2%) and 34 females (24.8%), with a mean age of $65.4 \text{ years} \pm 5.88 \text{ years}$. The no-reflow phenomenon occurred in 13 patients, yielding a prevalence of 9.5%. No significant differences were observed between the reflow and no-reflow groups regarding age, sex, BMI, smoking status, hypertension, dyslipidaemia, known ischaemic heart disease, or baseline cardiovascular medications. However, patients with no-reflow had a significantly longer duration of diabetes compared with patients with normal reflow ($13.77 \text{ years} \pm 4.83 \text{ years}$ vs. $8.08 \text{ years} \pm 3.98 \text{ years}$, $p = 0.001$) (Table 1).

2. Laboratory and echocardiographic findings

Patients with no-reflow exhibited significantly higher random blood glucose, fasting blood glucose, HbA1c, low-density lipoprotein

cholesterol, triglycerides and TyG index. In contrast, LVEF was significantly lower in the no-reflow group (Table 2, Figure 1).

3. Angiographic and procedural findings

The no-reflow group demonstrated significantly more severe coronary stenosis and a higher frequency of pre-dilatation. All patients with no-reflow exhibited postprocedural TIMI 2 flow, whereas all patients in the reflow group achieved TIMI 3 flow. No significant differences were observed regarding target vessel distribution, coronary dominance, stent diameter or stent length (Table 3).

4. Predictive performance of the TyG index

ROC analysis demonstrated that the TyG index had the highest predictive performance for no-reflow among all studied metabolic markers (AUC 0.870, 95% confidence interval [CI] 0.753–0.987, $p < 0.001$). The optimal cutoff by the Youden index was 9.01, yielding a sensitivity of 76.9% and a specificity of 89.5%. Because the cutoff was derived and evaluated within the same cohort, internal validation was performed using 2000 bootstrap resamples: the bootstrap 95% CI for the AUC was 0.738–0.977, and the optimism-corrected AUC was 0.870, indicating negligible optimism—as expected for a single continuous marker. The bootstrap 95% CI for the optimal cutoff was 8.95–9.20, supporting its stability; nonetheless, the sensitivity and specificity at this cutoff are internal estimates and require external validation (Table 4, Figures 2 and 3).

5. Multivariable logistic regression analysis

Because only 13 no-reflow events occurred, multivariable logistic regression was pre-specified as an exploratory analysis. In the parsimonious model, the TyG index remained independently associated with no-reflow (odds ratio [OR] 2.05 per 0.1-unit increase,

Table 1 - Baseline demographic, clinical characteristics and medication use according to reflow status

		Reflow ($n = 124$)	No-reflow ($n = 13$)	p -value
Age (years)	Mean $\pm$ SD	65.31 ± 5.83	66.69 ± 6.47	0.471
BMI (kg/m^2)	Mean $\pm$ SD	28.31 ± 1.70	29.89 ± 2.93	0.078
Duration of diabetes	Mean $\pm$ SD	8.08 ± 3.98	13.77 ± 4.83	0.001*
Sex—male	n (%)	93 (75.00)	10 (76.92)	1.000
Sex—female	n (%)	31 (25.00)	3 (23.08)	
Smoking	n (%)	65 (52.42)	7 (53.85)	0.922
HTN	n (%)	93 (75.00)	10 (76.92)	1.000
Dyslipidaemia	n (%)	83 (66.94)	9 (69.23)	1.000
Known IHD	n (%)	17 (13.71)	2 (15.38)	1.000
Aspirin	n (%)	105 (84.68)	11 (84.62)	1.000
Clopidogrel	n (%)	97 (78.23)	10 (76.92)	1.000
Beta-blocker	n (%)	86 (69.35)	10 (76.92)	0.754
Statin	n (%)	79 (63.71)	8 (61.54)	1.000
ACEi/ARBs	n (%)	57 (45.97)	6 (46.15)	1.000

ACEi, angiotensin-converting enzyme inhibitor; ARBs, angiotensin receptor blockers; BMI, body mass index; HTN, hypertension; IHD, ischaemic heart disease; SD, standard deviation

*Significant

Table 2 - Haemodynamic parameters, laboratory findings and cardiac function according to reflow status

		Reflow (n = 124)	No-reflow (n = 13)	p-value
HR	Mean ± SD	87.40 ± 11.55	88.38 ± 12.36	0.788
Systolic BP	Mean ± SD	132.83 ± 12.15	134.62 ± 15.87	0.700
Diastolic BP	Mean ± SD	86.65 ± 10.53	88.08 ± 10.91	0.660
SpO ₂	Mean ± SD	98.63 ± 0.49	98.69 ± 0.48	0.658
RBG (mg/dL)	Mean ± SD	170.23 ± 25.73	198.85 ± 34.39	0.012*
Serum creatinine (mg/dL)	Mean ± SD	0.99 ± 0.32	1.00 ± 0.18	0.849
CrCl (mL/min)	Mean ± SD	129.10 ± 11.98	129.00 ± 15.04	0.982
Hb (g/dL)	Mean ± SD	13.30 ± 0.81	12.56 ± 1.38	0.080
HbA1c (%)	Mean ± SD	7.19 ± 0.71	8.59 ± 1.71	0.012*
Cholesterol (mg/dL)	Mean ± SD	178.48 ± 19.51	181.92 ± 22.51	0.604
LDL (mg/dL)	Mean ± SD	126.00 ± 17.27	151.62 ± 35.91	0.025*
HDL (mg/dL)	Mean ± SD	37.10 ± 4.25	36.83 ± 3.62	0.807
TGs (mg/dL)	Mean ± SD	145.27 ± 20.46	167.31 ± 21.97	0.004*
FBG (mg/dL)	Mean ± SD	96.98 ± 12.35	124.77 ± 34.62	0.014*
TyG index	Mean ± SD	8.84 ± 0.20	9.21 ± 0.26	<0.001*
Ejection fraction (%)	Mean ± SD	50.91 ± 6.11	43.77 ± 4.53	<0.001*

BP, blood pressure; CrCl, creatinine clearance; FBG, fasting blood glucose; Hb, haemoglobin; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; HR, heart rate; LDL, low-density lipoprotein; RBG, random blood glucose

*Significant

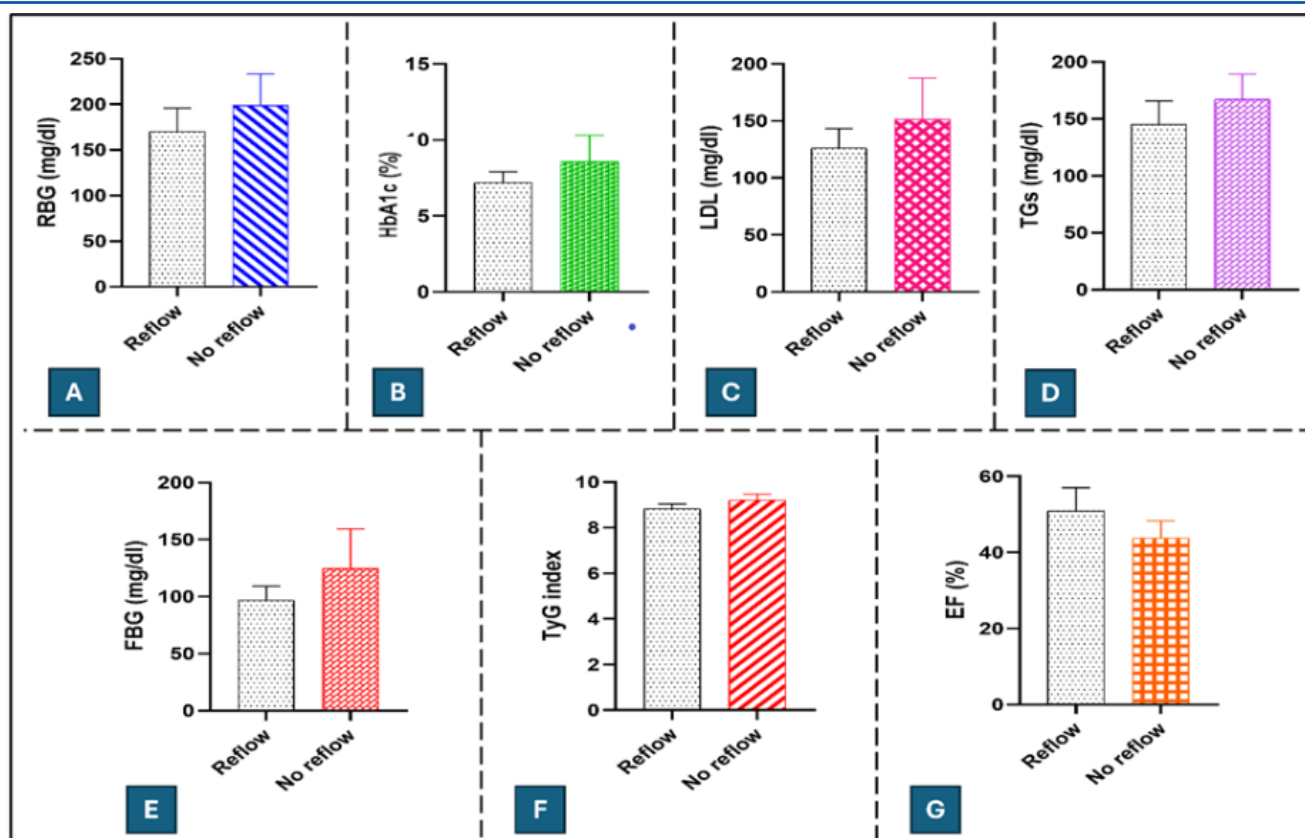


Figure 1 - RBG (A), HbA1c (B), LDL (C), TGs (D), FBG (E), TyG index (F) and EF (G) by reflow status. FBG, fasting blood glucose; HbA1c, glycated haemoglobin; LDL, low-density lipoprotein; RBG, random blood glucose; TyG, triglyceride–glucose

Table 3 - Angiographic characteristics and procedural details according to reflow status

		Reflow (<i>n</i> = 124)	No-reflow (<i>n</i> = 13)	<i>p</i> -value
Ischaemic changes present	<i>n</i> (%)	104 (83.87)	11 (84.62)	1.000
Affected vessel—LAD	<i>n</i> (%)	73 (58.87)	8 (61.54)	1.000
Affected vessel—LCX	<i>n</i> (%)	31 (25.00)	3 (23.08)	
Affected vessel—RCA	<i>n</i> (%)	20 (16.13)	2 (15.38)	
Dominance—Co-dominant	<i>n</i> (%)	25 (20.16)	3 (23.08)	0.898
Dominance—LCX	<i>n</i> (%)	12 (9.68)	1 (7.69)	
Dominance—RCA	<i>n</i> (%)	87 (70.16)	9 (69.23)	
Stenosis 80%	<i>n</i> (%)	1 (0.81)	0 (0.00)	0.001*
Stenosis 85%	<i>n</i> (%)	8 (6.45)	0 (0.00)	
Stenosis 90%	<i>n</i> (%)	110 (88.71)	8 (61.54)	
Stenosis 95%	<i>n</i> (%)	5 (4.03)	5 (38.46)	
Pre-PCI TIMI 1	<i>n</i> (%)	1 (0.81)	0 (0.00)	0.768
Pre-PCI TIMI 2	<i>n</i> (%)	31 (25.00)	4 (30.77)	
Pre-PCI TIMI 3	<i>n</i> (%)	92 (74.19)	9 (69.23)	
Pre-dilatation	<i>n</i> (%)	45 (36.29)	13 (100.00)	<0.001*
Number of stents = 1	<i>n</i> (%)	62 (50.00)	3 (23.08)	0.064
Number of stents = 2	<i>n</i> (%)	62 (50.00)	10 (76.92)	
Post-PCI TIMI 2	<i>n</i> (%)	0 (0.00)	13 (100.00)	<0.001*
Post-PCI TIMI 3	<i>n</i> (%)	124 (100.00)	0 (0.00)	
Stent diameter	Median (IQR)	3 (2.5–3)	3 (2.63–3.13)	0.626
Stent length	Median (IQR)	24 (20.5–32)	30 (23–34.5)	0.292

IQR, interquartile range; LAD, left anterior descending; LCX, left circumflex; PCI, percutaneous coronary intervention; RCA, right coronary artery; TIMI, Thrombolysis in Myocardial Infarction

*Significant

Table 4 - ROC analysis of metabolic laboratory parameters for predicting the no-reflow phenomenon

Parameter	AUC	SE	<i>p</i> -value	95% CI
TyG index	0.870	0.059	<0.001	0.753–0.987
RBG (mg/dL)	0.761	0.067	0.002	0.629–0.893
HbA1c	0.770	0.088	0.001	0.597–0.943
Cholesterol (mg/dL)	0.564	0.093	0.447	0.382–0.746
LDL (mg/dL)	0.755	0.105	0.003	0.550–0.960
HDL (mg/dL)	0.500	0.078	0.997	0.348–0.653
TGs (mg/dL)	0.772	0.078	0.001	0.619–0.925
FBG (mg/dL)	0.808	0.069	<0.001	0.673–0.943

AUC, area under the curve; CI, confidence interval; FBG, fasting blood glucose; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; RBG, random blood glucose; ROC, receiver operating characteristic; SE, standard error.

95% CI 1.26–3.32, $p = 0.004$), as did duration of diabetes (OR 1.42 per year, 95% CI 1.10–1.85, $p = 0.008$) and LVEF (OR 0.76 per 1%, 95% CI 0.60–0.95, $p = 0.019$). The association of the TyG index was directionally consistent in a fully adjusted sensitivity model (OR 2.30 per 0.1-unit increase, $p = 0.027$), although that model was over-parameterised for the available events and is reported for transparency only. Pre-dilatation could not be modelled because all no-reflow events occurred in pre-dilated patients (Table 5).

Discussion

In this cohort of diabetic patients undergoing elective PCI, no-reflow occurred in approximately 1 in 10 patients and was associated with poorer glycaemic control, an adverse lipid profile, longer diabetes duration, lower ejection fraction and more severe coronary stenosis. Among all metabolic markers examined, the TyG index showed the strongest discriminatory and independent association with no-reflow.

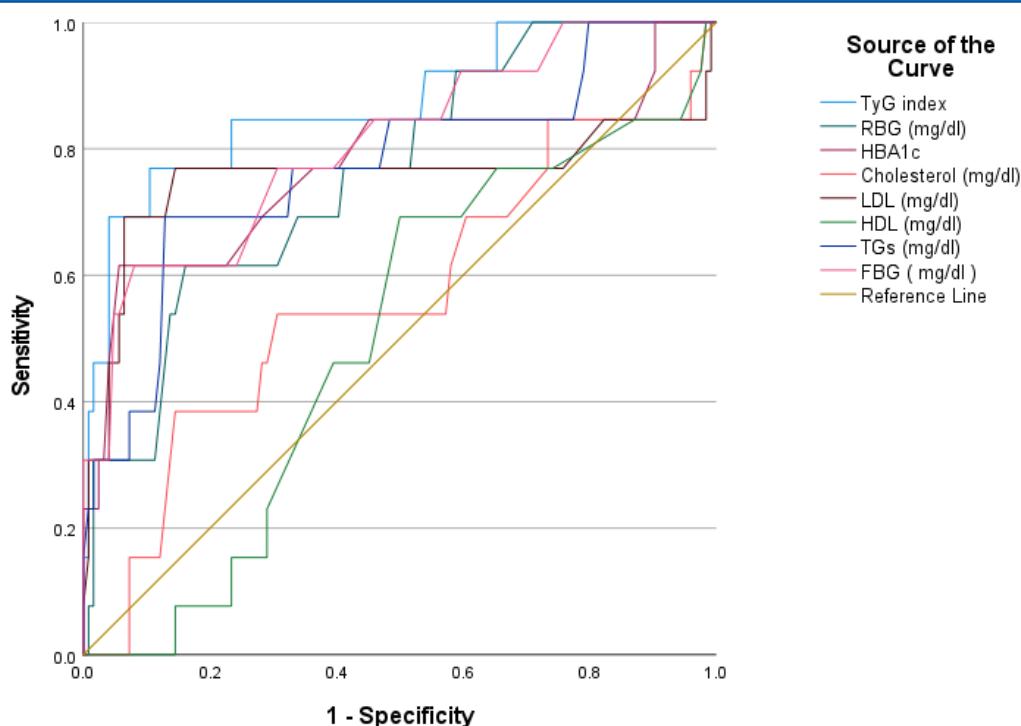


Figure 2 - Area under ROC curves for predicting reflow. FBG, fasting blood glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; RBG, random blood glucose; ROC, receiver operating characteristic; TyG, triglyceride–glucose

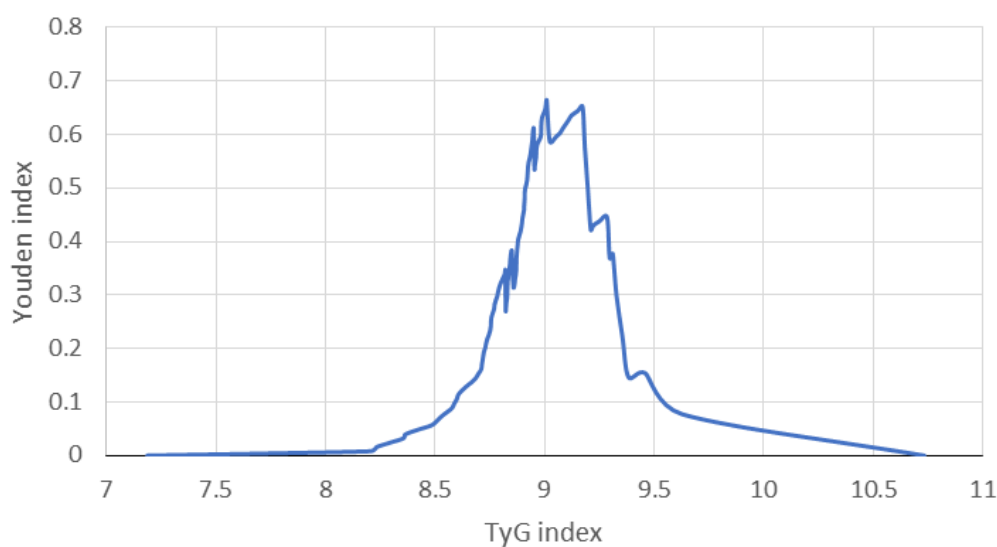


Figure 3 - Optimising cutoff for TyG index by Youden index. TyG, triglyceride–glucose

The mechanisms of no-reflow—distal embolization, ischaemia–reperfusion injury, endothelial and microvascular dysfunction, inflammation and platelet activation—are well described, predominantly in the setting of primary PCI for STEMI [3–5]. That context differs materially from elective PCI for stable disease, where thrombus burden and ischaemic time are lower; findings from the STEMI literature are, therefore, cited here only for mechanistic comparison and should not be assumed to transfer directly to

the elective setting examined in this study. Elective PCI for stable coronary disease is increasingly performed as a routine, often ambulatory, procedure [16], so anticipating even relatively infrequent complications such as no-reflow remains clinically important.

Because prevalence is our primary outcome, the observed incidence warrants direct comparison. Our rate of 9.5% is higher than that reported in other elective PCI series—2.4% by Jeon et al. [13] in non-acute patients and 5% by Waqar et al. [17] in stable

Table 5 - Multivariable logistic regression for predicting the no-reflow phenomenon

Model	Variable	β	SE	p-value	OR (95% CI)
<i>Parsimonious (primary)</i>	TyG index (per 0.1-unit)	0.717	0.247	0.004*	2.05 (1.26–3.32)
	Duration of diabetes (yr)	0.353	0.134	0.008*	1.42 (1.10–1.85)
	Ejection fraction (%)	–0.280	0.119	0.019*	0.76 (0.60–0.95)
<i>Full (sensitivity)</i>	TyG index (per 0.1-unit)	0.833	0.377	0.027*	2.30 (1.10–4.82)
	Ejection fraction (%)	–0.483	0.221	0.029*	0.62 (0.40–0.95)
	Duration of diabetes (yr)	0.582	0.254	0.022*	1.79 (1.09–2.94)
	Degree of stenosis (%)	0.586	0.333	0.078	1.80 (0.94–3.45)
Age, sex, BMI, smoking, HTN, dyslipidaemia, known IHD		—	—	>0.16 (NS)	—

BMI, body mass index; CI, confidence interval; HTN, hypertension; IHD, ischaemic heart disease; NS, not significant; OR, odds ratio; SE, standard error; TyG, triglyceride–glucose

Pre-dilatation was excluded from both models: all 13 no-reflow events occurred in pre-dilated patients (complete separation), precluding stable estimation

*Significant

coronary disease—and lower than rates in primary PCI STEMI series (Omar et al. [18] 11.1%; Elrayes et al. [19] 14.6%). The higher rate relative to other elective cohorts is consistent with our exclusively diabetic, higher-risk population, in whom diffuse atherosclerosis and microvascular vulnerability are more pronounced. This intermediate position, between unselected elective and acute STEMI populations, is the expected pattern for an all-diabetic elective cohort.

Metabolic status was strongly linked to no-reflow: affected patients had significantly higher fasting and random glucose, HbA1c, LDL cholesterol, triglycerides and TyG index, and a lower ejection fraction. These associations align with Qu and Guan [20] and with Zhao et al. [6], who likewise reported worse glycaemic control and more severe stenosis in patients developing no-reflow, and with Eldamhory et al. [5], who linked elevated glucose to impaired systolic function.

Among procedural factors, severe stenosis was significantly more frequent in the no-reflow group, whereas stent length (median 30 mm vs 24 mm, $p = 0.292$) and stent diameter ($p = 0.626$) were not associated with no-reflow. This differs from Jeon et al. [13], who reported longer lesion length among no-reflow patients, and indicates that in our cohort, lesion severity, rather than stent geometry, drove the association. All no-reflow events occurred in pre-dilated patients; however, because pre-dilatation is preferentially performed on more complex, severe lesions, it is best interpreted as a marker of lesion complexity rather than an independent contributor, and its complete association with the outcome precluded formal modelling.

The central finding is the performance of the TyG index. It integrates glycaemic and lipid abnormalities into a single fasting-derived surrogate of insulin resistance, a plausible driver of endothelial and microvascular dysfunction. In our data it showed the highest discrimination among metabolic markers (AUC 0.870) and remained independently associated with no-reflow in the parsimonious model (OR 2.05 per 0.1-unit increase, 95% CI 1.26–3.32, $p = 0.004$), alongside diabetes duration and ejection fraction. This is directionally concordant with prior reports: Yang et al. [21] (STEMI-diabetic) found the TyG index an independent predictor (adjusted OR 2.98), Ma et al. [11] reported an OR of 3.23 in diabetic PCI and Altunova et al.

[12] described good discrimination (AUC 0.821). A meta-analysis by Fajar et al. [22] similarly identified diabetes, hyperglycaemia, low ejection fraction and multivessel disease as predictors of no-reflow. The consistency of direction across differing clinical settings supports biological plausibility, while the differences in effect size underline the need for setting-specific validation.

Study limitations

Several limitations should be acknowledged. First, this was a single-centre observational study with a modest sample size, limiting generalisability; moreover, the sample-size estimate was based on a STEMI-derived incidence because elective-PCI diabetic data were unavailable at design, and the lower observed event rate (9.5%) reduced the statistical power for multivariable modelling. Second, only 13 no-reflow events occurred, yielding a low events-per-variable ratio; the multivariable model should, therefore, be regarded as exploratory and hypothesis-generating, and the identified associations require confirmation in larger cohorts. Third, the optimal TyG cutoff was both derived and evaluated within the same cohort; although bootstrap resampling indicated a stable cutoff and negligible optimism in the AUC, the cutoff-specific sensitivity and specificity may be optimistic and require external validation. Fourth, no-reflow was defined using angiographic TIMI flow grade alone; more sensitive indices of microvascular perfusion—myocardial blush grade and the corrected TIMI frame count—were not assessed or were cardiac magnetic resonance or myocardial contrast echocardiography [23], so subclinical microvascular dysfunction may have been underestimated. Fifth, only intracoronary nitroglycerine (Tridil) was administered when impaired flow was encountered; because nitrates act predominantly on epicardial vessels, persistence of impaired flow argues against epicardial spasm but does not exclude a potentially reversible microvascular component that dedicated agents (adenosine, verapamil, sodium nitroprusside) might have addressed. Sixth, stenosis severity and TIMI flow were assessed by visual estimation rather than quantitative coronary angiography

or core-laboratory adjudication, introducing potential inter-observer variability. Direct measures of insulin resistance, such as HOMA-IR, were not collected, precluding head-to-head comparison with the TyG index. Finally, long-term clinical outcomes were not evaluated, and residual confounding cannot be excluded; causality cannot be inferred from these observational data.

Future multicentre studies with larger populations and longer follow-up are warranted to validate these findings and determine whether interventions targeting insulin resistance and metabolic optimisation can reduce no-reflow and improve outcomes.

Conclusion

No-reflow occurred in approximately 1 in 10 patients with type 2 diabetes undergoing elective PCI and was associated with longer diabetes duration, poorer glycaemic control, an adverse lipid profile, reduced ejection fraction and severe coronary stenosis. Among all studied biomarkers, the triglyceride–glucose index demonstrated the highest predictive accuracy and remained an independent predictor of no-reflow after multivariable adjustment. The TyG index may, therefore, represent a simple, inexpensive and clinically useful tool for preprocedural risk stratification in diabetic patients undergoing elective PCI. Further large-scale multicentre studies are recommended to validate these findings and determine whether targeted metabolic interventions can improve myocardial reperfusion and long-term cardiovascular outcomes.

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Conflict of interest

The authors declare no conflicts of interest.

Consent to Participate

All participants were provided an informed written consent to participate in the study in accordance with the code of ethics of the World Medical Association (Declaration of Helsinki) for experiments on humans.

List of abbreviations

AUC, area under the curve; BMI, body mass index; CAD, coronary artery disease; CI, confidence interval; ECG, electrocardiography; HbA1c, glycated haemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; ROC, receiver operating characteristic; T2DM, type 2 diabetes mellitus; TIMI, Thrombolysis in Myocardial Infarction; TyG, triglyceride–glucose.

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