ORIGINAL ARTICLE

AL-ANBAR MEDICAL JOURNAL
Anb. Med. J. 22(3): 185–191, 2026

Prevalence and Clinical Correlates of TIMI Flow Grade Zero Culprit-artery Occlusion in Patients with NSTEMI Undergoing Early Percutaneous Coronary Intervention

Raad Hassan Najim[1] and Mufeed Akram Taha[2,*]

[1] *Department of Internal Medicine/Cardiology, College of Medicine, University of Kirkuk, Kirkuk, Iraq.*
[2] *Department of Internal Medicine/Neurology, College of Medicine, University of Kirkuk, Kirkuk, Iraq.*
(Received : 7 March 2026; Accepted : 3 June 2026; First published online: 1 July 2026)

ABSTRACT

Background: A substantial proportion of patients with non-ST-elevation myocardial infarction (NSTEMI) have an acutely occluded culprit artery despite the absence of diagnostic ST-segment elevation on the presenting electrocardiogram.
Objectives: To determine the prevalence of thrombolysis in myocardial infarction (TIMI) flow grade 0 total culprit-artery occlusion in percutaneous coronary intervention (PCI) treated NSTEMI and to identify its clinical, laboratory, echocardiographic, and angiographic correlates.
Materials and methods: This prospective single-center observational study included consecutive patients with NSTEMI who underwent coronary angiography within 24 hours of presentation and culprit-vessel PCI at Kirkuk Cardiac Centre, Iraq, from September 2023 to September 2025. NSTEMI was diagnosed by ischemic symptoms, absence of persistent ST-segment elevation, and cardiac troponin I above the 99th-percentile upper reference limit (0.04 μg/L) with a serial rise/fall pattern. Patients were classified into the TIMI 0 and TIMI $\geq$1 groups based on pre-PCI culprit-vessel flow. Echocardiography was performed within 24 hours of admission and repeated before discharge. Group comparisons and multivariable logistic regression were performed.
Results: Among 225 patients, TIMI 0 total occlusion was present in 78 (34.7%). Compared with TIMI $\geq$1, TIMI 0 was associated with male sex, higher body mass index (BMI), smoking, arrhythmia, family history of coronary artery disease, higher troponin, lower high-density lipoprotein (HDL) cholesterol, lower left ventricular ejection fraction, and more frequent left circumflex culprit lesions. In multivariable analysis, higher BMI, family history of coronary artery disease, higher admission troponin, lower HDL cholesterol, and lower admission ejection fraction remained independently associated with TIMI 0, whereas diabetes mellitus showed an inverse association.
Conclusion: TIMI 0 total occlusion was common in PCI-treated NSTEMI and was associated with a distinct clinical and angiographic profile, including higher BMI and troponin levels, lower HDL cholesterol and left ventricular ejection fraction, and more frequent left circumflex culprit lesions.
Keywords: Non-ST elevated myocardial infarction; Coronary occlusion; Coronary angiography; Percutaneous coronary intervention; Myocardial infarction.

DOI: 10.33091/amj.2026.170406.2635
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INTRODUCTION

Non-ST-elevation myocardial infarction (NSTEMI) is a major presentation of acute coronary syndrome and includes a heterogeneous spectrum of coronary pathology. Although NSTEMI is defined by ischemic symptoms, myocardial injury biomarkers, and absence of persistent diagnostic ST-segment elevation, the initial electrocardiogram does not always reflect the severity of the culprit lesion. Contemporary evidence supports early invasive evaluation in selected high-risk NSTEMI patients; however, clinical presentation and electrocardiographic findings alone may not reliably identify whether the culprit artery is totally occluded [1].

Earlier studies and meta-analyses have shown that a clinically important proportion of NSTEMI patients have a to-

* Corresponding author: E-mail: mufeedakram@uokirkuk.edu.iq
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tally occluded culprit artery at angiography. Khan et al. reported that NSTEMI patients with total culprit-vessel occlusion had higher mortality and major adverse cardiac events than those without total occlusion [2]. Similarly, Hung et al. found that approximately one-third of NSTEMI patients had an occluded culprit artery, with more frequent left circumflex artery involvement and worse outcomes [3]. Recent evidence has confirmed that acute total culprit-vessel occlusion remains common in NSTEMI and is associated with greater myocardial injury, impaired ventricular function, and poorer prognosis [4].

Recognition of occlusive NSTEMI before angiography remains challenging. Standard 12-lead electrocardiography has limited sensitivity for detecting posterior, lateral, and circumflex-territory ischemia, and acute left circumflex artery occlusion may occur without classical ST-elevation criteria [5]. This limitation has contributed to increasing interest in the occlusion myocardial infarction/non-occlusion myocardial infarction framework, which emphasizes culprit-artery patency rather than relying only on ST-segment elevation [6].

Despite the growing literature, important gaps remain. Many studies have focused mainly on mortality and adverse outcomes, whereas fewer have evaluated the combined clinical, laboratory, echocardiographic, angiographic, and procedural correlates of TIMI flow grade 0 total occlusion among percutaneous coronary intervention (PCI) treated NSTEMI patients. Variables such as troponin level, lipid profile, left ventricular ejection fraction, culprit-vessel distribution, collateral circulation, thrombus burden, and final angiographic reperfusion require further clarification, particularly in different regional clinical settings.

Regional data from Iraq remains limited. A recent study from Duhok addressed occlusive myocardial infarction in non-ST-elevation acute coronary syndrome, but data specifically focused on PCI-treated NSTEMI patients from other Iraqi centers remain sparse [7]. Therefore, the present study aimed to determine the prevalence of TIMI flow grade 0 total culprit-artery occlusion among NSTEMI patients undergoing early coronary angiography and culprit-vessel PCI at Kirkuk Cardiac Centre, Iraq. It also aimed to compare the clinical, laboratory, echocardiographic, angiographic, and procedural characteristics of patients with TIMI 0 versus TIMI $\geq$1 flow.

MATERIALS AND METHODS

This prospective single-center observational study was conducted at Kirkuk Cardiac Centre, Kirkuk, Iraq, from September 2023 to September 2025. Consecutive patients admitted with NSTEMI and managed with an early invasive strategy were screened. The final analytic sample included 225 patients who underwent coronary angiography within 24 hours of presentation and culprit-vessel PCI during the index admission. Participant demographic data, including age, sex, and body mass index (BMI), were collected at baseline and included in the comparative analysis between the TIMI 0 and TIMI $\geq$1 groups.

NSTEMI was diagnosed by ischemic symptoms, absence of persistent ST-segment elevation or recognized STEMI equivalent, and cardiac troponin I above the 99th-percentile upper reference limit of 0.04 μg/L with a serial rise-or-fall pattern. Troponin was measured at admission and after 3 hours when needed. Eligible patients had confirmed NSTEMI, early coronary angiography, and culprit-vessel PCI. Patients with pericarditis, myocardial infarction with non-obstructive coronary arteries, or coronary anatomy requiring referral for coronary artery bypass grafting rather than culprit-vessel PCI were excluded.

Pre-PCI culprit-vessel flow and culprit-vessel identification were independently assessed by two interventional cardiologists who were blinded to echocardiographic measurements and laboratory summaries. Disagreements were resolved by consensus. Patients were classified as TIMI 0, no antegrade culprit-vessel flower TIMI $\geq$1, any residual antegrade flow. The culprit vessel was categorized as left anterior descending, left circumflex, right coronary, or left main coronary artery. Multivessel disease, collateral circulation, thrombus burden, and final TIMI 3 flows were recorded. Multivessel disease was recorded as a patient-level indicator of significant non-culprit coronary artery disease; detailed non-culprit lesion severity, plaque morphology, and SYNTAX score were not collected. Collateral circulation was graded using the Rentrop classification, which ranges from grade 0, indicating no visible collateral filling, to grade 3, indicating complete collateral filling of the distal vessel. Thrombus burden was graded using the TIMI thrombus grading system, which ranges from grade 0, indicating no angiographic thrombus, to grade 5, indicating total occlusion with thrombus and no antegrade flow [8].

Transthoracic echocardiography was performed by one of two non-interventional cardiologists using the biplane Simpson method. Left ventricular ejection fraction was calculated from apical two-chamber and four-chamber views according to the biplane Simpson method. The echocardiography cardiologists were not involved in culprit-vessel identification or TIMI-flow assessment and were blinded to the angiographic TIMI-flow classification at the time of measurement. Left ventricular ejection fraction was measured within 24 hours of presentation and repeated 48–96 hours after PCI before discharge. These cardiologists were not involved in culprit-vessel identification or TIMI-flow assessment and were blinded to angiographic TIMI-flow classification at the time of measurement. Left ventricular ejection fraction was measured within 24 hours of presentation and repeated 48-96 hours after PCI before discharge.

The analyzed variables included demographic characteristics, comorbidities, cardiovascular risk factors, admission and 3-hour troponin I, lipid profile, admission and discharge left ventricular ejection fraction, culprit vessel, multivessel disease, symptom-onset-to-angiography time, door-to-PCI time, pre-PCI medications, collateral grade, thrombus burden, and final TIMI 3 flow. The anatomical location of presenting electrocardiogram (ECG) changes and regional wall-motion abnormalities were not analyzed as predefined categorical variables. There were no missing values for the primary analytic variables, and complete-case analysis was applied. Management followed the local early-invasive acute coronary syndrome protocol, compatible with contemporary ESC and ACC/AHA guideline recommendations [9, 10].

The study was approved by the Research Ethics Committee of the College of Medicine, University of Kirkuk, Kirkuk, Iraq (Approval number 29B, dated 27-3-2023). Informed consent for participation and publication was obtained from all patients.

Statistical analysis was performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). Normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed variables were summarized as mean $\pm$ standard deviation and compared using Welch's t-test. In contrast, skewed variables were summa-

http://doi.org/10.33091/amj.2026.170406.2635

rized as median and interquartile range and compared using the Mann-Whitney U test. Categorical variables were presented as numbers and percentages and compared using the chi-square test or Fisher's exact test. A multivariable logistic regression model was constructed with TIMI 0 total occlusion as the dependent variable. Clinically plausible early variables were selected, and admission troponin was log-transformed because of right-skewed distribution. A two-sided P-value < 0.05 was considered statistically significant.

The overall study design, eligibility pathway, final analytic cohort, and grouping according to pre-PCI culprit-vessel TIMI flow are summarized in Figure 1.

RESULTS

A total of 225 patients with NSTEMI who underwent early coronary angiography and culprit-vessel PCI were included. TIMI 0 total culprit-artery occlusion was present in 78 patients (34.7%; 95% CI 28.5%-41.3%), while 147 patients had TIMI flow grade ≥1.

Baseline clinical, laboratory, and echocardiographic characteristics are shown in Table 1. Compared with the TIMI ≥1 group, patients with TIMI 0 were more frequently male and had higher body mass index, arrhythmia, smoking, family history of coronary artery disease, admission and 3-hour troponin levels, and lower high-density lipoprotein (HDL) cholesterol and left ventricular ejection fraction (LVEF) at admission and discharge. Type 2 diabetes mellitus was less frequent in the TIMI 0 group, whereas age, arterial hypertension, dyslipidemia, total cholesterol, and LDL cholesterol were similar between groups.

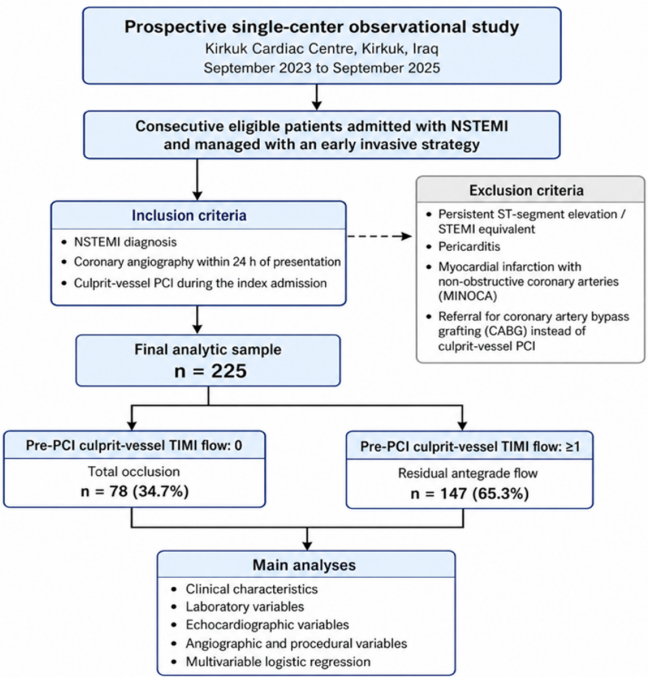

Figure 1. Flow diagram of the study design, patient selection, and grouping according to pre-percutaneous coronary intervention culprit-vessel Thrombolysis in Myocardial Infarction flow. Abbreviations: CABG, Coronary artery bypass grafting; MINOCA, Myocardial infarction with non-obstructive coronary arteries; NSTEMI, Non-ST-elevation myocardial infarction; PCI, Percutaneous coronary intervention; TIMI, Thrombolysis in myocardial infarction.

Angiographic and procedural characteristics are summarized in Table 2. Culprit-vessel distribution differed significantly between groups. TIMI 0 was associated with more frequent left circumflex artery (LCx) culprit lesions and less frequent left anterior descending artery (LAD) culprit lesions. In contrast, involvement of the right coronary artery (RCA) and the left main coronary artery (LM) did not differ significantly. The lower frequency of LAD involvement indicates an inverse association with TIMI 0 total occlusion (OR 0.40, 95% CI 0.23-0.70; P-value = 0.001). Multivessel disease, symptom-onset-to-angiography time, door-to-PCI time, and pre-PCI medications were similar between groups. Collateral circulation and thrombus burden were significantly greater in the TIMI 0 group, while final TIMI 3 flow was achieved in most patients in both groups.

In multivariable logistic regression (Table 3), higher BMI, family history of coronary artery disease, higher log-transformed admission troponin, lower HDL cholesterol, and lower admission LVEF remained independently associated with TIMI 0 total occlusion. Type 2 diabetes mellitus remained inversely associated with TIMI 0, whereas male sex showed borderline significance and arrhythmia and smoking were not independently associated.

DISCUSSION

In this prospective single-center study of PCI-treated NSTEMI patients, TIMI 0 total culprit-artery occlusion was identified in 34.7% of cases. This finding confirms that complete epicardial coronary occlusion is not uncommon in NSTEMI despite the absence of diagnostic ST-segment elevation. It also supports previous reports showing that a considerable proportion of NSTEMI patients have an acutely occluded culprit artery and may represent a higher-risk phenotype with greater myocardial injury and worse outcomes [11, 12].

TIMI 0 total occlusion was associated with a distinct clinical and biochemical profile. On multivariable analysis, higher BMI, family history of coronary artery disease, higher admission troponin, lower HDL cholesterol, and lower admission left ventricular ejection fraction remained independently associated with TIMI 0. In contrast, smoking and arrhythmia were no longer significant after adjustment, suggesting that their univariate associations may have been influenced by overlapping with other risk variables. The association with higher troponin and lower ejection fraction is biologically plausible, as complete interruption of antegrade coronary flow may produce greater ischemic burden and myocardial injury. Similar findings have been reported in NSTEMI cohorts in which reduced pre-PCI coronary flow was associated with worse ventricular function and adverse clinical outcomes [13].

Culprit-vessel distribution differed significantly between groups. TIMI 0 was associated with more frequent culprit lesions in the left circumflex artery and less frequent LAD involvement. This finding is clinically important because LCx occlusion is often under-recognized on the standard 12-lead ECG and may occur without classical ST-elevation criteria [5, 14, 15]. Previous studies have also shown that acute coronary occlusion may be missed when traditional STEMI criteria are used alone, particularly in patients with posterior, lateral, or circumflex-territory ischemia [16, 17]. Therefore, some patients classified clinically as NSTEMI may have an

Table 1. Baseline clinical, laboratory, and echocardiographic characteristics according to initial thrombolysis in myocardial infarction (TIMI) flow grade[¶].

Characteristic	TIMI 0 (n = 78)	TIMI 1–3 (n = 147)	P-value
Clinical characteristics			
Age, years	61.8 ± 9.3	60.9 ± 11.0	0.517*
Male sex, n (%)	60 (76.9)	92 (62.6)	0.029§
BMI, kg/m^2	31.8 ± 5.0	27.3 ± 4.6	0.001*
Arterial hypertension, n(%)	58 (74.4)	107 (72.8)	0.800§
Arrhythmia, n(%)	35 (44.9)	46 (31.3)	0.043§
Type 2 diabetes mellitus, n(%)	35 (44.9)	95 (64.6)	0.004§
Smoking, n(%)	48 (61.5)	70 (47.6)	0.047§
Dyslipidemia, n(%)	24 (30.8)	40 (27.2)	0.573§
Family history of CAD, n(%)	35 (44.9)	40 (27.2)	0.007§
Laboratory variables			
Admission troponin, μg/L	0.152 [0.110–0.255]	0.096 [0.054–0.154]	0.001†
Troponin at 3 h, μg/L	0.293 [0.178–0.424]	0.142 [0.082–0.231]	0.001†
Total cholesterol, mg/dL	182.3 [142.4–204.4]	171.4 [139.9–200.8]	0.401†
Triglycerides, mg/dL	103.7 [79.4–135.3]	116.9 [86.5–158.2]	0.023†
HDL, mg/dL	35.0 ± 9.8	43.0 ± 10.4	0.001*
LDL, mg/dL	97.0 ± 31.0	93.0 ± 29.0	0.348*
Echocardiographic variables			
LVEF at admission, %	40.5 ± 10.8	46.3 ± 11.1	0.001*
LVEF at discharge, %	43.8 ± 11.4	48.1 ± 11.3	0.008*

¶ Data are presented as mean ± SD, median [IQR], or n (%), as appropriate. * Welch's t test; † Mann–Whitney U test; § Chi-square test. Abbreviations: BMI, Body mass index; CAD, Coronary artery disease; HDL, High-density lipoprotein; LDL, Low-density lipoprotein; LVEF, Left ventricular ejection fraction; TIMI, Thrombolysis in myocardial infarction.

Table 2. Angiographic and procedural characteristics according to initial TIMI flow grade[¶].

Characteristic	TIMI 0 (n = 78)	TIMI 1–3 (n = 147)	P-value
Angiographic characteristics			
Culprit-vessel distribution, overall	—	—	0.001§
Left anterior descending artery, *n* (%)	33 (42.3)	95 (64.6)	0.001§
Left circumflex artery, *n* (%)	25 (32.1)	18 (12.2)	0.001§
Right coronary artery, *n* (%)	20 (25.6)	32 (21.8)	0.512§
Left main coronary artery, *n* (%)	0 (0.0)	2 (1.4)	0.545*
Multivessel disease, *n* (%)	24 (30.8)	51 (34.7)	0.552§
Collateral Rentrop grade	1.0 [1.0–2.0]	0.0 [0.0–1.0]	0.001†
Collateral circulation present (Rentrop $\geq$ 1), *n* (%)	63 (80.8)	65 (44.2)	0.001§
Thrombus burden, TIMI thrombus grade 0–5	3.0 [3.0–4.0]	2.0 [1.0–3.0]	0.001†
Procedural characteristics			
Symptom onset to angiography, h	10.2 [8.4–13.4]	10.8 [8.6–14.2]	0.409†
Door-to-PCI time, h	5.2 [3.5–6.8]	4.6 [3.8–5.8]	0.489†
Pre-PCI aspirin, *n* (%)	78 (100.0)	143 (97.3)	0.301*
Pre-PCI P2Y12 loading, *n* (%)	73 (93.6)	143 (97.3)	0.282*
Pre-PCI heparin, *n* (%)	76 (97.4)	135 (91.8)	0.146*
Pre-PCI high-intensity statin, *n* (%)	72 (92.3)	135 (91.8)	0.901§
Final TIMI 3 flow, *n* (%)	77 (98.7)	135 (91.8)	0.037*

¶ Data are presented as median [IQR] or n (%), as appropriate. Individual culprit-vessel rows are exploratory descriptive comparisons; the primary comparison for culprit-vessel distribution is the overall row. * Fisher exact test., † Mann–Whitney U test., § Chi-square test. Abbreviations: LAD, Left anterior descending artery; LCx, Left circumflex artery; LM, Left main coronary artery; PCI, Percutaneous coronary intervention; RCA, Right coronary artery; TIMI, Thrombolysis in myocardial infarction .

occlusive infarction pattern that is less apparent on electrocardiography. This supports the growing interest in the occlusion myocardial infarction/non-occlusion myocardial infarction concept, which emphasizes culprit-artery patency rather

http://doi.org/10.33091/amj.2026.170406.2635

Table 3. Multivariable logistic regression analysis of factors independently associated with TIMI 0 total occlusion at initial angiography[¶].

Predictor	Adjusted OR (95% CI)	P-value
Male sex	2.29 (0.97 to 5.42)	0.059
BMI, per 1 kg/m^2	1.29 (1.17 to 1.42)	0.001
Arrhythmia	0.89 (0.39 to 2.05)	0.789
Type 2 diabetes mellitus	0.35 (0.15 to 0.78)	0.011
Smoking	1.70 (0.76 to 3.82)	0.196
Family history of CAD	2.76 (1.18 to 6.46)	0.020
Log-transformed admission troponin	3.92 (2.08 to 7.39)	0.001
HDL, per 1 mg/dL	0.91 (0.87 to 0.95)	0.001
LVEF at admission, per1%	0.95 (0.91 to 0.98)	0.002

[¶] Dependent variable: TIMI 0 total occlusion. Model performance: pseudo-R2 = 0.434; likelihood-ratio test P-value < 0.001. For continuous variables, odds ratios are expressed per 1-unit increase unless otherwise specified. Abbreviations: BMI, Body mass index; CAD, Coronary artery disease; CI, Confidence interval; HDL, High-density lipoprotein; LVEF, Left ventricular ejection fraction; OR, Odds ratio; TIMI, Thrombolysis in myocardial infarction.

than ST-segment elevation alone [6, 16].

The angiographic findings further support this interpretation. Greater collateral circulation and higher thrombus burden in the TIMI 0 group suggest a more advanced occlusive and thrombotic phenotype. The higher frequency of collateral circulation may reflect recruitment or angiographic visibility of collateral channels in response to severe impairment or absence of antegrade flow. In contrast, higher thrombus burden supports intracoronary thrombosis as an important mechanism of total culprit-artery occlusion. Angiographic thrombus burden is an established marker of intracoronary thrombotic severity during PCI and may increase procedural complexity [8]. Importantly, symptom-onset-to-angiography time, door-to-PCI time, multivessel disease, and pre-PCI medication use were similar between groups, arguing against delayed presentation or less aggressive early management as the main explanation for the observed differences.

The similar frequency of multivessel disease suggests that significant non-culprit coronary artery disease, assessed at the patient level, did not account for the differences between the TIMI 0 and TIMI ≥1 groups. However, detailed non-culprit lesion severity, plaque characteristics, and SYNTAX score were not assessed; therefore, the influence of overall coronary anatomical complexity cannot be fully excluded. Final TIMI 3 flow was achieved in most patients in both groups, indicating that worse baseline angiographic status did not prevent successful immediate epicardial reperfusion after PCI. Nevertheless, restoration of final TIMI 3 flow does not eliminate the potential prognostic importance of presenting with an occluded culprit artery, which has been associated with adverse short- and long-term outcomes in previous studies [18, 19].

The inverse association between type 2 diabetes mellitus and TIMI 0 should be interpreted cautiously and should not be considered protective. Diabetes may be associated with a more diffuse coronary disease pattern, different lesion morphology, or different revascularization pathways within a PCI-treated cohort. Previous studies have shown that diabetes and multivessel coronary artery disease may influence revascularization decisions and outcomes in non-ST-elevation acute coronary syndrome patients [20, 21]. Because of the observational single-center design, residual confounding remains possible.

Taken together, these findings add local Iraqi data to the growing literature on angiographically occult total occlusion in NSTEMI. The combination of higher troponin, lower HDL cholesterol, lower ejection fraction, more frequent left circumflex culprit lesions, greater collateral circulation, and higher thrombus burden suggests that TIMI 0 total occlusion represents a distinct occlusive NSTEMI phenotype. These findings support careful early risk assessment and guideline-based invasive evaluation in selected higher-risk NSTEMI patients [9, 10, 22].

Several limitations should be acknowledged. The study was conducted at a single center. It included only NSTEMI patients who underwent culprit-vessel PCI, which may limit generalizability to conservatively managed patients, those with myocardial infarction with non-obstructive coronary arteries, or those referred for coronary artery bypass grafting. The observational design does not allow causal inference, and long-term clinical outcomes were not assessed. In addition, although ECG and echocardiography were performed clinically, the anatomical location of ECG changes and regional wall-motion abnormalities were not analyzed as predefined categorical variables. Finally, detailed non-culprit lesion severity, plaque characteristics, and the SYNTAX score were not collected; therefore, multiple univariate comparisons require cautious interpretation. Larger multicenter studies with outcome assessment are needed to validate these findings.

CONCLUSION

TIMI 0 total culprit-artery occlusion was common among PCI-treated NSTEMI patients, occurring in 34.7% of cases. It was independently associated with higher BMI, family history of coronary artery disease, higher admission troponin, lower HDL cholesterol, and lower admission left ventricular ejection fraction. At the same time, type 2 diabetes mellitus showed an inverse association. Angiographically, TIMI 0 was characterized by more frequent LCx culprit involvement, greater collateral circulation, and higher thrombus burden, supporting its recognition as a distinct occlusive NSTEMI phenotype. Larger multicenter studies with clinical outcome assessment are needed to validate these findings and clarify their prognostic significance.

ETHICAL DECLARATIONS

Acknowledgments

The authors thank the staff of Kirkuk Cardiac Centre for their assistance during patient care and data collection.

Ethics Approval and Consent to Participate

The study was approved by the Research Ethics Committee of the College of Medicine, University of Kirkuk, Kirkuk, Iraq (Approval number 29B, dated 27-3-2023). Informed consent to participate was obtained from all patients.

Consent for Publication

Not applicable (No identifying patient information is included in the manuscript).

Availability of Data and Material

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request and subject to institutional approval.

Competing Interests

The authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Use of Artificial Intelligence

Artificial intelligence (AI) tools were used only for language editing, formatting support, and the preparation of the revision package. The authors reviewed and approved all edited content, verified all scientific statements, and confirmed that AI tools were not used to generate, fabricate, analyze, or manipulate study data, citations, images, or research findings.

Authors' Contributions

Najim RH contributed to study conception, data acquisition, clinical interpretation, and manuscript drafting. Taha MA contributed to study design, data interpretation, critical revision, correspondence, and approval of the final manuscript. Both authors reviewed and approved the final version and agree to be accountable for all aspects of the work.

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