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ORIGINAL RESEARCH

Leuko-Glycemic Index in the Prognosis of Acute Myocardial Infarction; a Cohort Study on Coronary Angiography and Angioplasty Registry

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Abstract: **Introduction:** The leuko-glycemic index (LGI), a combined index of patient leukocyte counts and blood glucose levels, has been shown to predict the prognosis of myocardial infarction (MI) patients. Our study aims to investigate the performance of LGI in prediction of outcomes in a population of diabetic and non-diabetic MI patients. **Methods:** This observational registry-based cohort study was performed on acute myocardial infarction (AMI) patients. Participants were sub-grouped according to their diabetes status and the calculated optimal LGI cut-off value. The outcomes of the study were the length of hospital stay, and in-hospital and 30-day mortality. **Results:** A total of 296 AMI (112 diabetic and 184 non-diabetic) patients were included in the study. The optimal cut-off value of LGI in the diabetic and non-diabetic groups was calculated as 2970.4 mg/dl.mm^3 and 2249.4 mg/dl.mm^3, respectively. High LGI was associated with increased hospital admission duration in non-diabetic patients (p = 0.017). The area under the curve (AUC) of LGI for prediction of in-hospital mortality was 0.93 (95% CI: 0.87 to 1.00) in the diabetic group and 0.92 (95% CI: 0.85 to 0.99) in the non-diabetic group. LGI had a sensitivity and specificity of 90.00%, and 93.14% in prediction of in-hospital mortality in the diabetic group compared to 77.77% and 90.85% in the non-diabetic group. We observed 4 post-discharge mortalities in our patient group. **Conclusion:** Our study demonstrated that higher LGI predicts in-hospital mortality in both diabetic and non-diabetic patients, while the length of hospital stay was only predicted by LGI levels in non-diabetic patients.

Keywords: Glycemic index; Leukocyte count; Myocardial infarction; Risk assessment; Prognosis

1. Introduction

Cardiovascular diseases (CVDs) are the leading cause of death and premature death worldwide (1). It has been estimated that 18.6 million people died due to CVDs in 2019, 58% of which were in Asia. Ischemic heart diseases (IHDs) have been reported to be the cause of almost half of CVD deaths and the median cost of IHD care in low- and middle-income countries has been estimated to be 10% of the total healthcare spending (2, 3). Risk stratification and timely identification of high-risk IHD patients can aid physicians in appropriate patient management and prognostication, leading to improvements in patient care and outcome.

Various scoring systems and biomarkers have been shown to be of use in IHD patient risk stratification (4, 5). However, the current tools do not account for the inflammatory response subsequent to myocardial infarction (MI). Inflammation plays a key role in the development of atherosclerotic diseases and studies have demonstrated that inflammatory biomarker levels are associated with the outcome of MI pa-

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tients (6, 7). Besides the high economic cost of most inflammatory markers, their unavailability limits their clinical pertinence (8).

In 2010, Quiroga et al. (9) proposed that a combined index of patient leukocyte count and blood glucose levels can predict the prognosis of MI patients. The so-called leuko-glycemic index (LGI) has since been shown to be a fair predictor of mortality and adverse outcomes in various conditions (10-13). LGI consists of two routinely assessed laboratory variables and considering its straightforwardness and ease of calculation, it can be implemented in clinical practice with no significant cost (9).

A recent systematic review (14) has demonstrated that LGI can be a fair predictor of mortality and acute cardiac complications after MI. In their systematic review, Sadeghi et al. have noted that most current LGI studies have been performed on Latino and Hispanic populations with no distinctions between diabetic and non-diabetic patients. Our study aims to investigate the performance of LGI in prediction of outcomes in an Asian population of diabetic and non-diabetic MI patients.

2. Methods

2.1. Study design and patients

This prognostic accuracy study was performed using Coronary Angiography and Angioplasty Registry (CAAR). CAAR contains a prospective follow-up of consecutive acute myocardial infarction (AMI) patients, presenting to Imam Hossein Hospital, one of the main referral hospitals in Tehran, Iran. Patients presenting with myocardial infarction between 27 July 2021 and 27 February 2023, with a subsequently performed coronary angiography, were included in the study.

The protocol of CAAR has been enlisted at Shahid Beheshti University of Medical Sciences (SBMU), Vice-Chancellor for Research and Technology, and has been approved by the Ethics Committee (IR.SBMU.RETECH.REC.1400.256). This study was approved by the research ethics committee of the School of Medicine, SBMU (IR.SBMU.MSP.REC.1402.034), and written consent was obtained from all the participants.

2.2. Participants

AMI was diagnosed based on the fourth universal definition of MI (15) and included both ST segment elevation MI (STEMI) and non-STEMI patients. Being younger than 18 years of age, having no data on leukocyte count and plasma glucose and no follow-up data, and presence of coinciding infection such as pneumonia, diabetic foot and sepsis were the exclusion criteria of this study. Participants were divided into groups of diabetic and nondiabetic patients, according to their past medical histories or admission HbA1c ≥ 6.5%. Participants were further sub grouped to high- and low-LGI

groups, according to the calculated optimal LGI cut-point.

2.3. Data gathering

The patient information consisting of demographic and baseline characteristics, chief complaint, admission hemodynamic assessments, admission laboratory tests, AMI presentation, angiography results, final diagnosis, and outcomes were obtained using the registry database.

Laboratory variables including admission time, white blood cell count (WBC, cells/mm^3), and blood glucose level (mg/dl) were measured in peripheral venous sample. LGI was calculated as a multiplication of admission time WBC count and glucose level, divided by 1000, and reported as mg/dl.mm^3.

2.4. Outcomes

Patients were followed during their hospital admission and for 30 days after discharge through a telephone interview with the patient. The outcomes of the study were length of hospital stay, in-hospital mortality, and 30-day mortality. 30-day mortality was defined solely as post-discharge mortality and does not include in-hospital mortality.

2.5. Statistical analysis

The normality of the quantitative variables was assessed using Q-Q plot. Quantitative variables are reported as mean and standard deviation (SD) and qualitative variables are reported as frequency and percentage. The mean of quantitative variables was compared between LGI groups using student's t-test or Mann-Whitney U test, depending on the normality of variables. The frequency of qualitative variables was compared between LGI groups using Fisher's exact test or chi-square test. The optimal LGI cut-point for the prediction of in-hospital mortality among MI patients was determined using the maximum Youden's index (sensitivity + specificity 1). Validity indices such as sensitivity, specificity, Negative Predictive Value (NPV), Positive Predictive Value (PPV), accuracy, and discrimination and calibration indices such as area under the receiver operating characteristic curve (AUC, ROC) and Hosmer-Lemeshow's test were used to assess the predictive ability of LGI using a multivariable logistic regression model in diabetic and non-diabetic patients. After determining the optimal cut-point of LGI, survival probability was calculated using the Kaplan-Meier method and the P values for comparison of survival probability between different LGI values were estimated using log-rank's test. The univariate and multivariable Cox regression models were used to identify the association between LGI and other factors with the occurrence of in-hospital mortality. For selecting the best variables to enter the last multivariable model, stepwise selection method with backward approach (with p value ≤ 0.5) was used. The proportional hazards assumption was as-

sessed using Schoenfeld residual's test based on p value ≥ 0.05. Results of Cox regression models were reported as crude Hazard Ratio (HR) and adjusted HR (aHR) with 95% Confidence Interval (CI). All the analyses were conducted using STATA software version 14.

3. Results

3.1. Baseline characteristics of studied cases

A total of 296 AMI patients were included in this study, comprising 112 diabetic (mean age 63.54 years) and 184 non-diabetic patients (mean age 58.66 years). The patients were further sub-grouped according to the calculated optimal LGI cut-off point. In the diabetic patients, the high-LGI subgroup had an LGI of >2970.4 mg/dl.mm^3 (n=37) and in the non-diabetics, the high-LGI subgroup had an LGI of >2249.4 mg/dl.mm^3 (n=18).

In the diabetic group, high-LGI patients had lower BMI, lower systolic blood pressure, higher WBC, and higher HbA1c levels compared to low-LGI diabetic patients (p < 0.001). In the non-diabetic group, high-LGI patients had higher urea and higher WBC levels (p < 0.001) compared to low-LGI non-diabetic patients. The patients had no significant differences in age, gender, smoker status, past medical histories, heart rate, diastolic blood pressure, and other lab values such as creatinine, lipid profile values, troponin I, C-reactive protein, and pro-brain natriuretic peptide. The most common chief complaints were typical angina and dyspnea in both diabetic and non-diabetic groups. In the diabetic group, weakness was more observed in high-LGI patients (p < 0.05). Table 1 demonstrates the baseline characteristics, chief complaints, and laboratory variables at admission time.

High LGI was associated with lower left ventricular ejection fraction (LVEF) in non-diabetic patients (p = 0.027). There was no significant difference between the angiographic profile of high- and low-LGI patients in the diabetic or the non-diabetic groups and three-vessel disease was the most common manifestation in both groups. In the non-diabetic group, the prevalence of high LGI in STEMI patients was higher than NSTEMI patients (p = 0.043). Table 2 provides further details on the CAD presentations, angiographic results, final diagnosis, and outcome of patients.

3.2. Value of LGI in outcome prediction of AMI patients

The length of hospital stay was not affected by LGI levels in diabetic patients, while in the non-diabetic patients, the high-LGI group had increased hospital admission duration (p = 0.017). High-LGI patients had a higher rate of in-hospital mortality in both diabetic and non-diabetic patients. We observed 4 post-discharge mortality cases in our patient group. In diabetic patients, in-hospital mortality was more preva-

lent in the high-LGI group (24.32% vs 1.33%; p < 0.001). The same association was observed between high LGI and in-hospital mortality in non-diabetic patients (33.33% vs 1.81%; p < 0.001). Our analysis revealed that high LGI independently predicted in-hospital mortality in both the diabetic (aHR = 9.50, 95% CI: 1.03 to 87.67; p value = 0.047) and the non-diabetic groups (aHR = 24.36, 95% CI: 3.83 to 154.72; p value = 0.001). C-reactive protein (CRP) was also found to be an independent predictor of in-hospital mortality in non-diabetic patients (aHR = 1.02) (Table 3).

The AUC of LGI for the prediction of in-hospital mortality was 0.93 (95% CI: 0.87 to 1.00) in the diabetic group and 0.92 (95% CI: 0.85 to 0.99) in the non-diabetic group (Figure 1). LGI had a sensitivity, specificity, and NPV of 90.00%, 93.14%, and 98.96%, respectively, in prediction of in-hospital mortality in the diabetic group compared to 77.77%, 90.85%, and 98.75%, respectively, in the non-diabetic group. The optimal cut-off value of LGI in the diabetic and non-diabetic groups was calculated as 2970.4 mg/dl.mm^3 and 2249.4 mg/dl.mm^3, respectively. Table 4 provides the validity indices of LGI.

In-hospital survival probability was shown to be lower in the high-LGI group in both the diabetic (p = 0.0007) and non-diabetic patients (p = 0.0002) (Figure 2).

4. Discussion

The results of the current study demonstrated that higher LGI predicts in-hospital mortality in both diabetic and non-diabetic patients, while the length of hospital stay was only affected by LGI levels in non-diabetic patients. Our results indicate that LGI has a higher sensitivity in prediction of outcomes in diabetic patients, with similar specificities for both diabetic and non-diabetic patients. In a similar study, Qi et al. have also investigated the predictive value of LGI in diabetic and non-diabetic patients (16). In line with our findings, LGI levels were shown to be predictive of in-hospital mortality in non-diabetic patients (adjusted odds ratio (OR) = 1.001, p-value = 0.001). However, in contrast to our results, Qi et al. argue that LGI levels cannot predict in-hospital mortality in diabetic patients (adjusted OR = 1.000, p-value: 0.807). Qi et. al. have reported that higher LGI levels are predictive of 15-month major adverse cardiovascular events only in non-diabetic patients.

Studies have established the prominence of blood cell counts and glucose levels in the prognostication of AMI patients (17-20) and many mechanisms have been speculated to explain such an effect. Stimulated neutrophils have been shown to increase the infarction size through release of free radicals and proteolytic enzymes, which in turn contribute to electrical instability of the heart through endothelial damage (21, 22). Leukocytes have also been shown to induce progressive capillary plugging and a hypercoagulable state, leading

to adverse outcomes after ischemic cardiac injuries (17). The pathophysiology of hyperglycemia in AMI is not completely known; however, it has been proposed to contribute to endothelial damage through an increase in vasoconstriction and inflammatory factors, which has been shown to lead to an increase in infarct size and overall mortality of AMI patients (23, 24). This evidence suggests that a combined index of leukocyte count and blood glucose levels could be a useful predictor of adverse outcomes in AMI patients.

In their systematic review, Sadeghi et al. (14) reported an AUC of 0.77 with sensitivity and specificity of 0.75 and 0.66, respectively, for the value of LGI in prediction of mortality (in-hospital and post-discharge mortality) regardless of the diabetic status of the patients. We demonstrated a better predictive value for LGI through separation of diabetic and non-diabetic patients. This might indicate that the dichotomization of patients based on diabetes status can improve the performance capabilities of LGI. It should also be noted that our results solely focus on in-hospital mortality and so LGI might be a better predictor of in-hospital mortality than combined in-hospital and post-discharge mortality. Moreover, almost all the populations included in the systematic review were of Latino and Hispanic origin and the possible ethnic differences between our studies should also be taken into account. Studies have utilized varying cut-offs for LGI. The reported cut-offs diverge from as low as 656 to as high as 2200 mg/dl.mm^3 irrespective of the diabetic status of the patient population (14, 25, 26). Considering that diabetic patients have higher average blood glucose levels, the utilized threshold should be different for diabetic and non-diabetic patients. In our study, the optimal cut-off value of LGI in the diabetic and non-diabetic groups was calculated as 2970.4 and 2249.4 mg/dl.mm^3, respectively. Qi et al. (16) reported an optimal cut-off value of 3593 mg/dl.mm^3 for diabetic and 1402 mg/dl.mm^3 for non-diabetic patients. Further studies are required to determine the optimal cut-off value of LGI in diabetic and non-diabetic patients.

A few studies have investigated the incorporation of LGI in classical risk scores. Hirschon Prado et al. (8) argue that LGI improves risk assessment in patients underestimated by Thrombolysis in Myocardial Infarction (TIMI) score and demonstrated that the addition of LGI (with a cut-off point > 1000) to the TIMI score improves its discriminatory capacity of ST-elevation MI patients. Bearing in mind the fairly acceptable predictive performance of LGI, it can be feasible to incorporate LGI into classical risk scores to improve their performance.

5. Limitations

This study has its limitations. The small sample size, especially for non-diabetic high-LGI patients could have caused bias. Moreover, we could not assess the predictive value of LGI for post-discharge mortality due to a very low event rate. We also did not explore the combined value of LGI with other classical risk scores. There was also no data on the treatment plan for the AMI and diabetes of the patients.

6. Conclusion

Our study demonstrated that higher LGI predicts in-hospital mortality in both diabetic and non-diabetic patients, while the length of hospital stay was only predicted by LGI levels in non-diabetic patients. Further studies, with separate diabetic and non-diabetic patient groups, are required for the determination of the predictive value of LGI in each respective population and its optimal cut-off value.

7. Declarations

7.1. Acknowledgments

7.2. Conflict of interest

The authors declare they have no conflicts of interest.

7.3. Research funding

7.4. Authors' contribution

Study design: RS, MHA
Data gathering: RP, NT
Analysis: NT, AS
Interpreting the results: RS, MHA, AS, KA
Drafting: KA, AS
Critically revised: All authors

7.5. Availability of data and materials

Qualified researchers with approved research proposals can contact the Prevention of Cardiovascular Diseases Research Center to request access to the gathered data.

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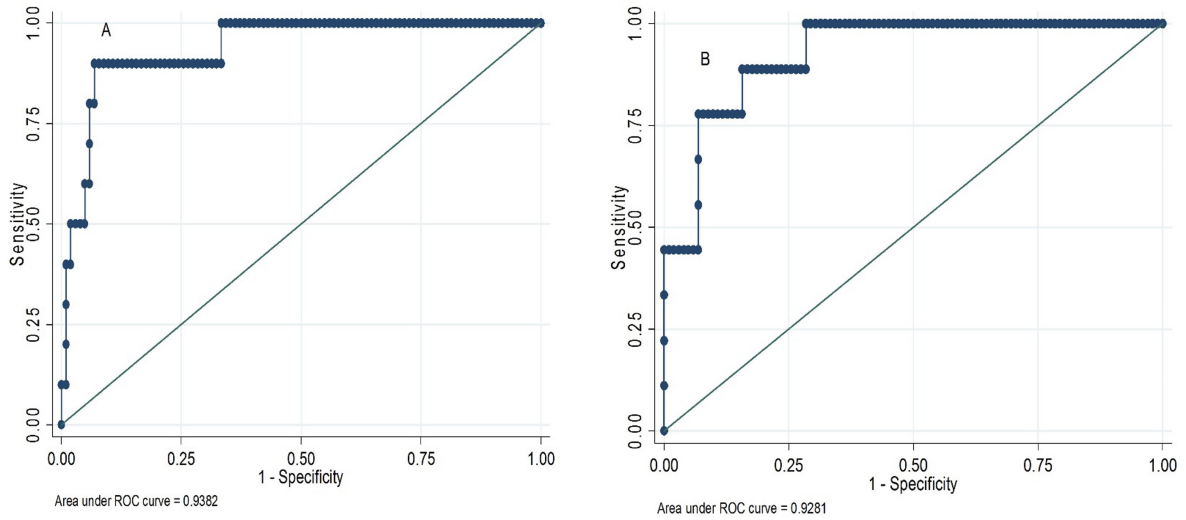**Figure 1:** The receiver operating characteristic (ROC) curve of leuko-glycemic index in prediction of in-hospital mortality among diabetic (A) and non-diabetic (B) myocardial infarction patients.
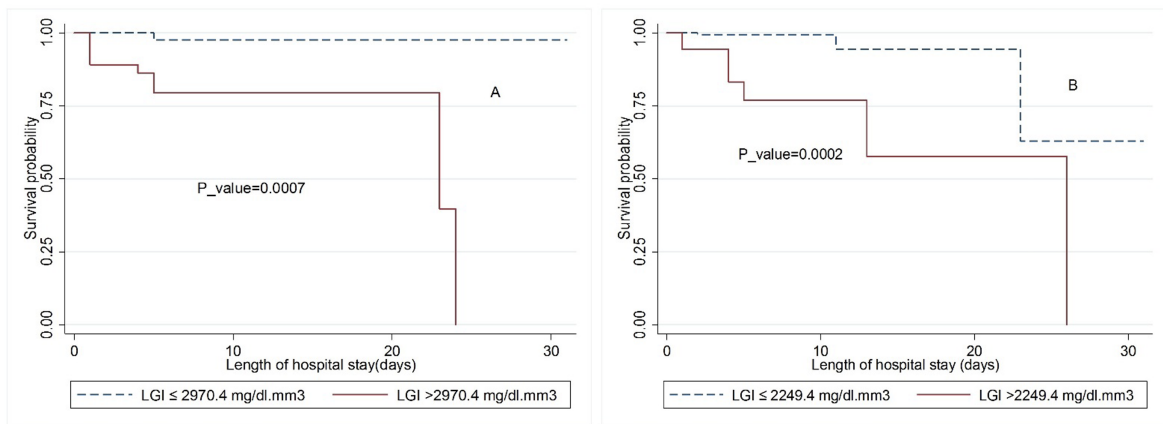

Figure 2: Kaplan-Meier time to event analysis of leuko-glycemic index (LGI) for the occurrence of in-hospital mortality during hospitalization in the diabetic (A) and non-diabetic (B) patients.

Table 1: Patients' general and baseline information

Variables	Diabetics				Non-diabetics			
	Low LGI (n= 75)	High LGI (n= 37)	Total (n=112)	P value	Low LGI (n= 166)	High LGI (n= 18)	Total (n=184)	P value
General information								
Age (years)								
Mean ± SD	63.96±11.92	62.67±12.73	63.54±12.15	0.600	58.51±11.44	60.08±17.13	58.66±12.06	0.707
Gender								
Male	42 (56.00)	25 (67.57)	67 (59.82)	0.240	132 (79.52)	16 (88.89)	148 (80.43)	0.533
Female	33 (44.00)	12 (32.43)	45 (40.18)		34 (20.48)	2 (11.11)	36 (19.57)	
BMI (kg/cm^2)								
Mean ± SD	28.68 ± 4.99	26.34 ± 4.19	27.92 ± 4.86	0.011	27.52 ± 5.24	27.50 ± 4.21	27.51 ± 16.49	0.653
Current smoker (yes)								
Mean ± SD	17 (22.67)	12 (32.43)	29 (25.89)	0.267	81 (48.80)	10 (55.56)	91 (49.46)	0.586
Medical history (yes)								
Myocardial infarction	10 (13.33)	8 (21.62)	18 (16.07)	0.261	28 (16.87)	1 (5.56)	29 (15.76)	0.315
Ischemic heart disease	37 (49.33)	15 (40.54)	52 (46.43)	0.380	44 (26.51)	3 (16.67)	47 (25.54)	0.569
Hypertension	51 (68.00)	19 (51.35)	70 (62.50)	0.087	57 (34.34)	7 (38.89)	64 (34.78)	0.700
Dyslipidemia	47 (62.67)	19 (51.35)	66 (58.93)	0.252	47 (28.31)	2 (11.11)	49 (26.63)	0.162
Heart failure	22 (30.14)	9 (25.00)	31 (28.44)	0.576	30 (19.11)	6 (33.33)	36 (20.57)	0.157
CVI/TIA	7 (9.46)	4 (10.81)	11 (9.91)	1.000	10 (6.10)	1 (5.56)	11 (6.04)	1.000
PCI	12 (16.67)	9 (25.00)	21 (19.44)	0.302	29 (17.79)	2 (11.11)	31 (17.13)	0.742
Chief complaint (yes)								
Non-angina chest pain	2 (2.67)	2 (5.41)	4 (3.57)	0.598	8 (4.82)	0 (0.00)	8 (4.35)	1.000
Atypical chest pain	5 (6.67)	1 (2.70)	6 (5.36)	0.662	10 (6.02)	0 (0.00)	10 (5.43)	0.602
Typical angina	58 (77.33)	26 (70.27)	84 (75.00)	0.488	140 (84.34)	13 (72.22)	153 (83.15)	0.192
Dyspnea	30 (40.00)	18 (48.65)	48 (42.86)	0.384	65 (39.16)	7 (38.89)	72 (39.13)	0.982
Palpitation	5 (6.67)	3 (8.11)	8 (7.14)	1.000	14 (8.43)	1 (5.56)	15 (8.15)	1.000
Weakness	9 (12.00)	10 (27.03)	19 (16.96)	0.046	14 (8.43)	1 (5.56)	15 (8.15)	1.000
Syncope	0 (0.00)	0 (0.00)	0 (0.00)	N/A	1 (0.60)	2 (11.11)	3 (1.63)	0.026
Faint	0 (0.00)	2 (5.41)	2 (179)	0.107	0 (0.00)	3 (16.67)	3 (1.63)	0.001
Hemodynamic assessments								
SBP (mmHg)	135.02 ± 26.59	120.78 ± 27.94	130.32 ± 27.75	0.010	129.48 ± 25.98	134.61 ± 38.36	129.98 ± 27.35	0.586
DBP (mmHg)	80.34 ± 16.29	78.89 ± 15.75	79.86 ± 16.06	0.774	80.81 ± 15.77	83.05 ± 25.18	81.03 ± 16.85	0.784
Heart rate (beat/min)	82.06 ± 18.08	87.16 ± 22.15	83.75 ± 19.57	0.331	78.29 ± 15.45	85.88 ± 18.29	79.04 ± 15.86	0.179
Laboratory results during admission								
WBC × 10^9/L	9.10 ± 2.57	13.35 ± 3.22	10.50 ± 3.43	<0.001	9.56 ± 2.71	31.78 ± 53.26	11.73 ± 17.72	<0.001
Blood sugar (mg/dl)	176.20 ± 63.85	355.51 ± 104.66	235.43 ± 115.96	<0.001	127.94 ± 28.67	179.63 ± 67.71	131.30 ± 34.70	0.018
HbA1c (%)	7.77 ± 1.87	10.28 ± 2.10	8.53 ± 2.25	<0.001	6.05 ± 0.35	6.08 ± 0.34	6.05 ± 0.35	0.716
Creatinine (mg/dL)	1.27 ± 0.61	1.15 ± 0.36	1.23 ± 0.54	0.510	1.12 ± 0.30	1.26 ± 0.33	1.13 ± 0.30	0.085
Urea(mg/dL)	44.86 ± 21.48	48.14 ± 25.00	45.92 ± 22.62	0.478	35.06 ± 18.40	42.95 ± 15.69	35.84 ± 18.27	0.004
Triglycerides (mg/dL)	138.41± 95.69	188.04± 188.67	154.32 ±133.50	0.450	129.54 ±71.52	129.26 ± 100.03	129.50 ± 74.77	0.358
Cholesterol (mg/dL)	149.67± 44.33	158.28± 45.27	152.43 ± 44.52	0.374	160.27 ± 44.82	154.14 ± 35.63	159.63 ± 43.87	0.528
LDL (mg/dL)	89.18 ± 40.84	98.08 ± 38.26	91.98 ± 40.00	0.287	107.88 ± 33.68	102.66 ± 34.61	107.27 ± 33.69	0.601
HDL (mg/dL)	42.82 ± 27.78	44.37 ± 33.07	43.31 ± 29.34	0.897	36.73 ± 7.55	39.14 ± 7.43	36.99 ± 7.54	0.256
Troponin I (ng/mL)	3.26 ± 6.12	4.19 ± 9.18	3.58 ± 7.26	0.935	4.60 ± 10.71	4.41 ± 7.31	4.58 ± 10.43	0.629
CRP (mg/L)	16.15 ± 30.30	21.30 ± 27.70	17.80 ± 29.44	0.667	14.71 ± 25.72	18.25 ± 21.88	15.12 ± 25.27	0.166
Pro-BNP (pg/mL)	2902.50±5163.8	4129.4±6118.7	3296.3±5480.0	0.349	1408.1±2632.3	3385.3±5477.3	1641.7±3133.1	0.213

Data presented as n (%) or mean ± standard deviation. BMI: Body mass index; CRP: C-reactive protein;
CVI/TIA: Cerebrovascular incident/ Transient ischemic attack; HDL: High density lipoprotein; LDL: Low density lipoprotein;
LGI: Leuko-glycemic index; PCI: Percutaneous coronary intervention; Pro-BNP: Pro- brain natriuretic peptide;
WBC: White blood cell; SBP: Systolic blood pressure; DBP: Diastolic blood pressure. In diabetics low LGI was ≤ 2970.4 mg/dl.mm^3
and high LGI was > 2970.4 mg/dl.mm^3 and in non-diabetics low LGI was ≤ 2249.4 mg/dl.mm^3 and high LGI was > 2249.4mg/dl.mm^3.

Table 2: Patients' clinical information and outcomes

Variables	Diabetics				Non-diabetics			
	Low LGI (n= 75)	High LGI (n= 37)	Total (n=112)	P value	Low LGI (n= 166)	High LGI (n= 18)	Total (n=184)	P value
General information								
Angiography and echocardiography results								
Normal/Mild CAD	6 (8.00)	1 (2.70)	7 (6.25)	1.000	9 (5.42)	1 (5.56)	10 (5.44)	1.000
Single-vessel disease	13 (17.33)	3 (8.11)	16 (14.29)	0.256	45 (27.11)	6 (33.33)	51 (27.72)	0.575
Two-vessel disease	15 (20.00)	8 (21.62)	23 (20.54)	0.842	44 (26.51)	6 (33.33)	50 (27.17)	0.536
Three-vessel disease	41 (54.67)	25 (67.57)	66 (58.92)	0.192	68 (40.96)	5 (27.78)	73 (39.67)	0.277
LVEF (%)	38.82 ± 12.27	35.30 ± 13.91	37.75 ± 12.83	0.224	40.90 ± 11.72	33.75 ± 12.44	40.25 ± 11.93	0.027
Final diagnosis								
NSTEMI	40 (53.33)	15 (40.54)	55 (49.11)	0.203	70 (42.17)	3 (16.67)	73 (39.67)	0.043
STEMI	35 (46.67)	22 (59.46)	57 (50.89)		96 (57.83)	15 (83.33)	111 (60.33)	
Outcomes								
LOS (days)	6.44 ± 4.69	8.16 ± 6.15	7.00 ± 5.25	0.157	6.25 ± 4.67	8.94 ± 6.25	6.51 ± 4.89	0.017
In-hospital mortality								
No	74 (98.67)	28 (75.68)	102 (91.07)	<0.001	163 (98.19)	12 (66.67)	175 (95.11)	<0.001
Yes	1 (1.33)	9 (24.32)	10 (8.93)		3 (1.81)	6 (33.33)	9 (4.89)	
30-day mortality								
No	72 (97.30)	24 (85.71)	96 (94.12)	0.063	156 (95.71)	11 (91.67)	167 (95.43)	0.440
Yes	1 (1.35)	2 (7.14)	3 (2.94)		1 (0.61)	0 (0.00)	1 (0.57)	
Unknown	1 (1.35)	2 (7.14)	3 (2.94)		6 (3.68)	1 (8.33)	7 (4.00)	

Data presented as n (%) or mean ± standard deviation. CAD: Coronary Artery Disease; LGI: Leuko-glycemic index; LVEF: Left ventricular ejection fraction; NSTEMI: Non-ST elevation myocardial infarction; STEMI: ST elevation myocardial infarction; LOS: Length of hospital stay. In diabetics low LGI was ≤ 2970.4 mg/dl.mm^3 and high LGI was > 2970.4 mg/dl.mm^3 and in non-diabetics low LGI was ≤ 2249.4 mg/dl.mm^3 and high LGI was > 2249.4mg/dl.mm^3.

Table 3: Cox regression analysis1 for the prediction of in-hospital mortality in patients

Variables	Diabetics				Non-diabetics			
	Crude HR (95% CI)	p value	Adjusted HR (95% CI)	P value	Crude HR (95% CI)	p value	Adjusted HR (95% CI)	P value
Age (years)	1.03 (0.98 – 1.08)	0.213	0.99 (0.93 – 1.06)	0.942	1.08 (1.02 – 1.14)	0.007	1.003 (0.92 – 1.08)	0.935
Gender								
Female	Reference	0.544	Reference	0.394	Reference	0.043	Reference	0.127
Male	0.65 (0.16 – 2.60)		0.40 (0.04 – 3.26)		0.23 (0.05 – 0.95)		0.16 (0.01 – 1.67)	
BMI (kg/cm^2)	0.92 (0.77 – 1.09)	0.346	0.93 (0.75 – 1.15)	0.539	-	-	-	-
Current smoker								
No	Reference	0.938	Reference	0.508	Reference	0.218	Reference	0.314
Yes	0.93 (0.18 – 4.60)		2.15 (0.22 – 20.99)		0.41 (0.10 – 1.68)		0.34 (0.04 – 2.77)	
CRP (mg/L)	1.006 (0.98 – 1.02)	0.526	1.004 (0.97 – 1.03)	0.731	1.01 (1.004 – 1.03)	0.013	1.02 (1.0006 – 1.05)	0.045
LGI (mg/dl.mm^3)								
Low LGI	Reference	0.011	Reference	0.047	Reference	0.001	Reference	0.001
High LGI	14.86 (1.86 – 118.40)		9.50 (1.03 – 87.67)		10.38 (2.46 – 43.64)		24.36 (3.83 – 154.72)	

BMI: Body mass index; CRP: C-reactive protein; HR: Hazard ratio; LGI: Leuko-glycemic index. In diabetics low LGI was ≤ 2970.4 mg/dl.mm^3 and high LGI was > 2970.4 mg/dl.mm^3 and in non-diabetics low LGI was ≤ 2249.4 mg/dl.mm^3 and high LGI was > 2249.4mg/dl.mm^3. Leuko-glycemic Index (LGI) was adjusted with age, gender and STEMI/NSTEMI status using multivariable logistic regression model. 1Based on maximum value of Youden's index.

Table 4: Performance values of the leuko-glycemic index in prediction of in-hospital mortality

Indices	Values (95%CI)	
	Diabetics	**Non-diabetics**
Optimal cut point values of LGI (mg/dl.mm^3)	2970.4	2249.4
True positive (n)	9	7
True negative (n)	95	159
False positive (n)	7	16
False negative (n)	1	2
Sensitivity (%)	90.00 (55.50 – 99.70)	77.77 (40.00 – 97.20)
Specificity (%)	93.14 (86.40 – 97.20)	90.85 (85.60 – 94.70)
Positive Predictive Value (%)	56.25 (29.90 – 80.20)	30.43 (13.20 – 52.90)
Negative Predictive Value (%)	98.96 (94.30 – 100)	98.75 (95.60 – 99.80)
Positive Likelihood ratio	13.11 (6.23 – 27.60)	8.51 (4.75 – 15.24)
Negative Likelihood ratio	0.11 (0.02 – 0.69)	0.24 (0.07 – 0.83)
Accuracy (%)	92.86 (85.29 – 96.26)	90.22 (84.98 – 94.10)
Discrimination index		
AUC	0.93 (0.87 – 1.00)	0.92 (0.85 – 0.99)

AUC: Area under the receiver operating characteristic curve; LGI: Leuko-glycemic index; CI: confidence interval.