RESEARCH ARTICLE / *Araştırma Makalesi*

Leukoglycemic Index may be a Unique Parameter to Predict Mortality in Patients with Acute Myocardial Infarction: Single Operator Experience

Lökoglisemik İndeks Akut Miyokard Enfarktüsünde Mortaliteyi Öngördürücü Benzersiz Bir Parametre Olabilir

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Abstract

Introduction	Predicting high-risk patients is crucial in acute myocardial infarction (AMI). We aimed to investigate whether the leukoglycemic index (LGI) has a unique ability and to compare it with other inflammatory parameters in predicting in-hospital mortality in AMI.
Materials and Methods	In this single-center study, we retrospectively analyzed all AMI patients hospitalized and followed by a single operator. Patients were divided into two according to in-hospital outcomes. Other inflammatory parameters (systemic immune-inflammatory index, platelet-lymphocyte ratio, neutrophil-lymphocyte ratio, triglyceride-HDL ratio, and LDL-HDL ratio), C-reactive protein (CRP), and LGI were calculated according to previously described criteria. Univariable and multivariable logistic regression analyses were used to find independent predictors. The receiver operating characteristic (ROC) curve was used to find the cut-off point of LGI and other parameters in predicting mortality.
Results	A total of 304 patients with AMI were included in the study. The mean age was 62.18±11.89 and 74 (24.3%) of patients were female. The total death rate was 19 (6.3%). In univariate variable analysis, LGI was found as a significant predictor of mortality (p<0.001). After adjusting risk factors (age, coronary artery disease history, ejection fraction, CHA2DS2VASc score, creatinine, and CRP) in multivariable analysis, LGI was still found as a significant predictor of short-term mortality. ROC curve analysis showed that the area under the curve was 0.837 (0.704-0.971) with a sensitivity of 76.5% and with a specificity of 91.5% with a 3.39 cut-off value.
Conclusion	Our study showed that LGI might be a unique parameter in predicting short-term mortality in AMI.
Keywords	leukoglycemic index, acute myocardial infarction, mortality

Öz

Amaç	*Akut miyokard enfarktüsünde (AME) yüksek riskli hastaları öngörmek çok önemlidir. Bu çalışmada lökoglisemik indeksin (LGI) AME'de hastane içi mortaliteyi öngörmede benzersiz bir yeteneği olup olmadığını araştırmayı ve diğer inflamatuar parametrelerle karşılaştırmayı amaçlanmıştır.*
Yöntem ve Gereçler	*Bu tek merkezli çalışmada, hastaneye yatırılan ve tek operatör tarafından takip edilen tüm AME hastalarını retrospektif olarak inceledik. Hastalar hastane içi ölüm sonuçlarına göre ikiye ayrıldı. Diğer inflamatuar parametreler (sistemik immun-inflamatuar indeks, platelet-lenfosit oranı, nötrofil-lenfosit oranı, trigliserit-HDL oranı ve LDL-HDL oranı), C-reaktif protein (CRP) ve LGI daha önce açıklanan kriterlere göre hesaplandı. Bağımsız yordayıcıları bulmak için tek değişkenli ve çok değişkenli lojistik regresyon analizi kullanıldı. Mortaliteyi öngörmede LGI ve diğer parametrelerin kestirim noktasını bulmak için receiver operator characteristic (ROC) eğrisi kullanıldı.*
Bulgular	*Çalışmaya toplam 304 AMI hastası dahil edildi. Ortalama yaş 62,18±11,89 olup hastaların 74'ü (%24,3) kadındı. Toplam ölüm oranı 19 (%6,3) idi. Tek değişkenli değişken analizinde LGI, mortalitenin anlamlı bir yordayıcısı olarak bulundu (p<0.001). Çok değişkenli analizde risk faktörleri (yaş, koroner arter hastalığı öyküsü, ejeksiyon fraksiyonu, CHA2DS2VASc skoru, kreatinin ve CRP) modele eklendikten sonra LGI, kısa vadeli mortalitenin önemli bir göstergesi olarak bulundu. ROC eğrisi analizi, eğri altında kalan alanın 0,837 (0,704-0,971) olduğunu, duyarlılığın %76,5 ve özgüllüğün %91,5 olduğunu ve 3,39 cut-off değerini gösterdi.*
Sonuç	*Çalışmamız, LGI'nin AMI'de kısa vadeli mortaliteyi tahmin etmede diğer parametrelere göre benzersiz bir parametre olabileceğini gösterdi.*
Anahtar Kelimeler	*lökoglisemik indeks, akut miyokard enfaktüsü, mortalite*

INTRODUCTION

Acute myocardial infarction (AMI) has high morbidity and mortality worldwide despite advances in medical treatment and interventional techniques. Predicting acute and serious effects of AMI and taking action against it is highly important to prevent harmful outcomes. Therefore, risk factors associated with coronary artery disease (CAD) and AMI were investigated in detailed. Moreover, risk scoring systems have been developed to categorize the high risk and low risk patients.[1,2] Quick and easy markers as well as risk scores and clinical status of the patients have been studied in predicting short and long term outcomes of AMI patients.[3–5]

It is known that inflammation plays a crucial role in the course and prognosis of different acute and chronic diseases.[6–8] Acute MI is strongly associated with inflammatory process and inflammatory markers measured in blood stream can easily reflect this pathophysiological state.[9] Based on this data, different inflammatory markers have been studied in the prognosis of AMI. Neutrophil-lymphocyte ratio (NLR), platelet-lypmhocyte ratio (PLR), systemic immune-inflammatory index (SII), triglyceride-HDL ratio and LDL-HDL ratio and C-reactive protein (CRP) are some of the inflammatory markers of which predictive values have been shown.[3,5,10–12] Leukoglycemic index (LGI) which constitutes blood glucose level and white blood cell (WBC) count has been previously studied in different populations including ST elevation myocardial infarction (STEMI) and thought to be a prognostic marker in prognosis.[13]

Although it was shown that inflammatory markers have prognostic data in predicting short and long term outcomes of AMI in separate studies, LGI has not been compared with other inflammatory markers previously. In this study, we wanted to investigate the ability of LGI and to compare it with other inflammatory markers in predicting in-hospital mortality of AMI patients.

MATERIALS and METHODS

In this retrospective study, patients with AMI followed by a single operator in a tertiary hospital between October 2019 and October 2021 were included in the analysis. NonSTEMI and STEMI diagnoses were based on the European Society of Cardiology guidelines.[14,15] All patients were followed in coronary care unit (CCU) under the supervision of interventional cardiologist and medical and interventional treatments were administered according to the discretion of the physician. Demographic characteristics, laboratory data and discharge status were recorded. Blood tests were taken from patients upon arrival to the CCU and complete blood count samples were collected in dipotassium EDTA tubes. Other biochemical measurements including lipid parameters were also checked.

Inflammatory Parameters

Basic inflammatory parameters (e.g. CRP) were measured in previously taken blood samples and other parameters were calculated according to previously described methods. Triglyceride-HDL ratio, LDL-HDL ratio, SII, PLR and NLR were separately calculated and LGI was calculated by multiplying blood glucose level and WBC count and dividing by a thousand. All parameters were separately tested in univariable logistic regression analysis to test the ability of predicting in-hospital mortality.

Statistical Analysis

SPSS software package (Version 23.0, SPSS, Inc., Chicago, IL) was used for analyzing the data. Shapiro-Wilk test was used to test the distribution of numerical values and mean ± standart deviation was used for normally distributed valus and the median (interquartile range) was used for the non-normal ones. Chi-square test was used to test the categorical variables which were expressed as frequencies (%). Independent samples t-test and Mann-Whitney U test were used to test normally and non-normally distributed variables, respectively. Univariable and multivariable logistic regression analysis was used to test the significance of the variables in predicting the outcomes. First, variables

were tested in univariable logistic regression. Next, LGI was tested in multivariable logistic regression with adjusted model by adding clinically and statistically significant variables to the model. Area under the receiver operating characteristic (ROC) curve was used to test the sensitivity and specificity and find the cut-off value of the LGI.

RESULTS

A total of 304 patients were included in the study. Mean age was 62.18±11.89 and 74 (24.3%) of patients were female. While non-STEMI constituted 169 (55.6%) of patients, STEMI was 135 (44.4%). A total of 19 (6.3%) of patients died in hospital follow-up and there was not statistically significant difference between STEMI and nonSTEMI and between STEMI subgroups in mortality (p=0.176). Table 1 illustrates some of the basal demographic characteristics and laboratory findings of both groups (survivors and nonsurvivors). There was not statistically significant difference between survivors and nonsurvivors in terms of age, gender, cardiovascular risk factors and several laboratory parameters including lipid parameters (all p values >0.005). Ejection fraction was significantly lower in nonsurvivors (Table 1, p<0.001). Previously calculated inflammatory parameters were also compared between groups and SII, PLR, NLR, TG-HDL ratio and LDL-HDL ratio were all similar between groups (Table 1). Only LGI was significantly higher in nonsurvivors (5.3 (2.25-6.85) vs 1.57 (1.19-2.15), p<0.001).

Binary logistic regression analysis was used to find the significant predictors of in-hospital mortality. First, variables were tested in univariable logistic regression analysis. Table 2 showed the univariable analysis results of CRP, LGI and other inflammatory parameters. In unadjusted model, LGI and CRP were significantly related to in-hospital mortality (2.345 (1.759-3.126), p<0.001 and 1.012 (1.003-1.022), p=0.006, respectively). But other inflammatory parameters were failed to predict short-term outcomes. In adjusted model of logistic regression, we added CRP, LGI and clinically important parameters (age, CAD history, ejection fraction, CHA2DS2VASc score (congestive HF or left ventricular dysfunction, HT, age 75 years and older or between 65-74 years, DM, thromboembolism or stroke history, vascular disease, and female gender), creatinine and CRP) to the multivariable model. Adjusted model showed that EF and LGI are the only significant predictors of in-hospital mortality in AMI patients (Table 3). Area under the ROC curve (AUC) showed that LGI has a 76.5% sensitivity and 91.5% specificity with the cut-off value of 3.39 (AUC: 0.837, p<0.001, Figure). Moreover, there was no difference in the prognostic value of LGI in mortality in subgroup analyzes with and without DM.

Table 1. Demographic characteristics and laboratory findings of survivors and nonsurvivors

	Survivors (n=385)	Nonsurvivors (n=19)	p value
Age (years)	62±11.9	64.9±11.7	0.295
Female, n (%)	66 (23.2)	8 (42.1)	0.093
STEMI, n (%)	123 (43.2)	12 (63.2)	0.176
Hypertension, n (%)	97 (34)	5 (26.3)	0.619
Diabetes Mellitus, n (%)	80 (28.1)	5 (26.3)	0.895
CAD, n (%)	70 (24.6)	4 (21.1)	0.900
Hyperlipidemia, n (%)	17 (6)	1 (5.3)	0.955
Smoking, n (%)	116 (40.7)	7 (36.8)	0.720
Body Mass Index	26.4 (24.7-28.3)	25.3 (23.9-27)	0.990
EF (%)	55 (45-55)	30 (25-45)	<0.001
CHA2DS2VASc	2 (1-3)	3 (1-4)	0.198
WBC	10 (8.4-12.6)	15.3 (10.3-19)	<0.001
Hemoglobin	14.5±0.9	13.4±1.1	0.145
Platelet count	287±30	219±46	0.595
Neutrophil count	7±3.7	9.4±7.3	0.046
Lymphocyte count	2.1 (1.5-2.9)	2.5 (1.9-3.8)	0.189
Glucose	147 (117-200)	375 (175-484)	0.001
Creatinine	1.1±0.2	1.7±1.1	0.142
Sodium	140±3.5	138±3.8	0.067
Potassium	4±1	4.3±0.4	0.294
Albumin	4.0±0.4	3.2±0.8	0.298
AST	28 (21-45)	37 (32-90)	0.158
ALT	21 (16-31)	33 (22-76)	0.155
CRP	5 (2-12)	12 (6-76)	0.036
LDL	125±49	132±32	0.328
HDL	40±15	41±14	0.377
Total cholesterol	191±45	189±48	0.178
Triglyceride	125±89	79±17	0.327
LGI	1.57 (1.19-2.15)	5.3 (2.25-6.85)	<0.001
SII	787 (473-1225)	1045 (491-2662)	0.283
PLR	111.8 (81.3-153.8)	105.4 (78-149)	0.365
NLR	3.1 (2.04-5.25)	4.1 (2.5-9.5)	0.286
TG-HDL ratio	3 (2-4.6)	2.2 (1.5-3.8)	0.318
LDL-HDL ratio	3.1 (2.5-3.8)	2.9 (1.8-3.1)	0.175

Continuous variables are presented as mean ± SD or median (IQR), categorical variables are presented as frequency (%)
ALT: alanine transaminase; AST: aspartate transaminase; CAD: Coronary Artery Disease; CRP: C-reactive protein; EF: ejection fraction; HDL: high density lipoprotein; IQR: interquartile range; LDL: low density lipoprotein; LGI: leukoglycemic index; SD: standard deviation; NLR: Neutrophil lymphocyte ratio; PLR: platelet lymphocyte ratio; SII: systemic immune-inflammatory index; STEMI: ST Elevation Myocardial Infarction; TG: triglyceride; WBC: white blood cell

Table 2. Univariable logistic regression analysis of several inflammatory markers

Variable	Odds ratio (Confidence Interval)	p value
CRP	1.012 (1.003 – 1.022)	0.006
SII	1.153 (0.637 – 2.088)	0.639
NLR	1.080 (0.981 – 1.190)	0.118
PLR	0.996 (0.989 – 1.004)	0.363
TG-HDL ratio	0.875 (0.678 – 1.129)	0.304
LDL-HDL ratio	0.678 (0.386 – 1.191)	0.176
LGI	2.345 (1.759 – 3.126)	<0.001

Abbreviations: CRP: C-reactive protein, HDL: high density lipoprotein, LDL: low density lipoprotein, LGI: leukoglycemic index, NLR: neutrophil lymphocyte ratio, PLR: platelet lymphocyte ratio, SII: systemic immune inflammatory index, TG: triglyceride

Table 3. Adjusted and unadjusted logistic regression analysis of LGI in predicting mortality

Variables	Odds ratio (95% CI)	P value
Leukoglycemic index		
Unadjusted	2.345 (1.759 – 3.126)	<0.001
Adjusted	2.159 (1.522 – 3.061)	<0.001

Risk factors adjusted by age ,CAD history, EF, CHA2DS2VASc score, creatinine and CRP
Abbreviations: CAD: coronary artery disease, CRP: C-reactive protein, EF: ejection fraction, LGI: leukoglycemic index

DISCUSSION

Our single operator study showed that LGI may be a unique parameter and has better prediction ability than previuosly studied inflammatory parameters to show in-hospital mortality in AMI.

High mortality risk in AMI led clinicans to investigate the clinical and laboratory predictors of both short-term and long-term mortality. Inflammatory parameters have been tested for several years. White blood cell count, CRP and lymphocyte to monocyte ratio were separately tested and found as significantly correlated with the extent of atherosclerosis and long-term outcomes in AMI.[16–18] Besides, Oylumlu et al. showed that PLR is strongly associated with in-hospital mortality with acute coronary syndrome.[19] Furthermore, the newly described inflammatory parameter SII was tested in several studies and proven that it may

predict short and long term outcomes in stable CAD and AMI patients. Yang et al. investigated the ability of SII in predicting clinical outcomes in patients with CAD. They found that SII has a better predictive ability in major cardiovascular outcomes than traditional risk factors in CAD patients after coronary intervention.[20] Huang et al. revealed the same findings in elderly patients.[21] Moreover, two independent studies proved that increased LDL/HDL ratio and decreased TG/HDL ratio are associated with worse clinical outcomes in patients with STEMI.[11,12] These studies clearly indicate that different inflammatory parameters can be used as a prognostic tool both in chronic coronary syndromes and in AMI. Neverthless, the research to find better parameters to predict the prognosis is still going.

Leukocyte count which is directly related to inflammatory state of the body is a very good prognostic factor in AMI to predict heart failure, cardiogenic shock and death.[22] Besides, hyperglycemia is promoted by activated inflammatory mediators regardless of diabetes and it may also trigger the inflammatory response.[23,24] These pathophysiological effects of leukocyte and vlood glucose levels necessitated investigation of the combination of these parameters. The LGI parameter was obtained as a result of this research and its effects in different clinical scenarios were examined. Padella-Cueto et al. documented the prognostic effects of LGI in Cuban patients with STEMI in a retrospective study.[13] Qi et al. investigated the LGI in an observational and multicenter study including AMI patients and they showed that LGI is a significant predictor of all-cause mortality in non-diabetics, but not in diabetics.[22] Investigators also categorized LGI in their study rather than taking it as a numerical value. Kilic et al. showed that LGI is a predictor of CAD severity and it is highly correlated with the Gensini score.[25] Although these three studies have demonstrated the prognostic value of LGI in different patient groups, LGI was not compared with other inflammatory markers which have proven prognostic value in AMI. Therefore, comparing the prognostic value of LGI with other inflammatory markers will provide a more objective evaluation of the results. Therefore, in our study, besides testing the prognostic value of LGI in AMI patients, we demonstrated its superiority over other inflammatory markers. Based on these findings, it may be rational to use LGI instead of other inflammatory markers in AMI process.

Limitations Of The Study

Despite considerable findings, our study has several limitations. First, our study was designed in a retrospective manner which may lead the investigators to the bias. Second, our population included only single operator patients. Third, data acquired from study includes only single center patients. Therefore, prospectively designed studies involving several centers may provide more comprehensive and valuable information. Fourth, we did not compare the clinical significance of LGI with GRACE risk score which has proven prognostic value in ACS. However, despite these limitations, our study is important in that it demonstrates the superiority of LGI over other inflammatory parameters.

CONCLUSION

This study showed that LGI may be a unique parameter to predict in-hospital mortality in AMI patients including STEMI and nonSTEMI regardless of DM and superior to other inflammatory parameters that have previously proven clinical importance.

Confict of Interest

No conflict of interest was declared by the authors.

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