Turk Kardiyol Dern Ars 2018;46(4):260-267 doi: 10.5543/tkda.2018.36151

ORIGINAL ARTICLE

The relationship between monocyte/high-density lipoprotein ratio and Selvester QRS score in patients with STEMI

STEMİ'li hastalarda monosit / yüksek dansiteli lipoprotein oranı ve Selvester QRS skoru arasındaki ilişki

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ABSTRACT

Objective: The ratio of monocytes to high-density lipoprotein (MHR) has recently been recommended as a new prognostic factor in cardiovascular disease. Studies have documented the value of the Selvester QRS score for prediction of prognosis of ST-elevation myocardial infarction (STEMI). However, no study has examined the association between the QRS score and MHR in patients with STEMI. The present study analyzed the relationship between MHR and QRS score in patients with STEMI.

Methods: A cohort of 99 consecutive patients who experienced STEMI between June and September 2016 was retrospectively evaluated. Serial electrocardiogram, monocyte count, and lipid panel measurements (day 1, day 2, and after discharge) were performed in all patients, and MHR was calculated. The patients were classified into 2 groups based on the median values according to the estimated infarct size: QRS score <6 and QRS score ≥6.

Results: The MHR was higher in the high QRS score group on day 1 in hospital (p=0.001). The MHR value was associated with QRS score in univariate logistic regression analysis and was found to be an independent predictor of the QRS score (Odds ratio: 0.390, 95% Confidence interval: 0.252–0.605; p<0.001).

Conclusion: A higher MHR serves as an indicator of inflammation and oxidative stress and was reported to be associated with a high QRS score. In addition, it was found to be an independent predictor of such scores during follow-up in patients with STEMI.

ÖZET

Amaç: Monositlerin yüksek yoğunluklu lipoproteine (MHR) oranı kardiyovasküler hastalıkta yeni bir prognostik faktör olarak son zamanlarda önerilmektedir. Çalışmalar ST yükselmeli miyokart enfarktüsü (STEMİ) prognozunun tahmini için Selvester QRS skorunun değerini belgeledi. Bununla birlikte, STEMİ hastalarında QRS skoru ile MHR arasındaki ilişkiyi inceleyen bir çalışma bulunmamaktadır. Bu çalışmada STEMİ'li hastalarda MHR ve QRS skoru arasındaki ilişki incelenmiştir.

Yöntemler: ST yükselmeli miyokart enfarktüslü 99 ardışık hasta Haziran ve Eylül 2016 yılları arasında geriye dönük olarak değerlendirildi. Tüm hastalarda EKG, monosit sayısı ve lipid panellerinin (1. gün, 2. gün ve sonra) seri ölçümleri yapıldı ve ardından MHR bu zaman noktalarında hesaplanmıştır. Hastalar, tahmin edilen enfarktüs boyutuna göre medyan değerlere göre iki gruba ayrıldı: QRS skoru <6 ve QRS skoru ≥6.

Bulgular: Monositlerin yüksek yoğunluklu lipoproteine oranı hastanede 1. günde yüksek QRS skor grubunda daha yüksekti (p=0.001). Tek değişkenli lojistik regresyon analizinde MHR değeri QRS skoruyla ilişkiliydi ve QRS skorunun bağımsız tahmincisi olarak bulundu (OO: 0.390, %95 GA: 0.252–0.605, p<0.001).

Sonuç: Daha yüksek bir MHR, enflamasyon ve oksidatif stresin bir göstergesi olarak kullanılır ve yüksek bir QRS puanı ile ilişkili olduğu bildirilmiştir. Buna ek olarak, STEMİ hastalarında izlem sırasında bu skorların bağımsız bir önbelirleyicisi olduğuna karar verdik.

The initiation of atherosclerosis involves an influx of monocytes and differentiated macrophages, key players responsible for vascular inflammation, to the sites of inflammation. In atherosclerotic lesions, these immune cells lead to an inflammatory response and tissue remodeling.[1] A high monocyte count, especially, plays a significant role in plaque progression during the acute phase of ST-elevation myocardial

Received: December 04, 2017 *Accepted:* March 05, 2018

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infarction (STEMI).[2] Consequently, monocytes have been identified as an independent marker for the prognosis of STEMI.[3] The protective role of serum high-density lipoprotein (HDL) during STEMI has also been proven; HDL molecules prevent macrophage migration, thus reducing the chance of vascular inflammation.[4,5] Recently, the monocyte count/HDL ratio (MHR) has emerged as an independent predictor of major cardiovascular events in patients with chronic kidney disease (CKD).[6] Clinicians frequently use the standard 12-lead electrocardiogram (ECG) to determine the simplified Selvester QRS score, which is significant in studying the symptoms of potential coronary artery disease (CAD) and estimating myocardial infarct size.[7] Durrer et al.[8] performed a computer simulation of the electrical activation sequence of the human heart. The current 50-criteria, 31-point version of the QRS score corresponding to 3% of the left ventricular (LV) mass has a high level of specificity in normal populations.[9] However, the predictive value of the QRS score decreases in cases of multiple myocardial infarctions (MIs).[10]

The present study examined the MHR and utilized the QRS score to predict the myocardial infarct size in patients with STEMI.

METHODS

Study population

A total of 99 patients who presented at the center with STEMI between June and September 2016 and underwent primary percutaneous coronary intervention (pPCI) were retrospectively enrolled in the study. STEMI was defined as chest pain or angina equivalent to within 30 minutes over the last 15 hours, suggesting myocardial ischemia. This was accompanied by a 1-mm (0.1-mV) ST-segment elevation in 2 or more adjacent derivations followed by an increase in creatine kinase (CK) and CK-MB-troponin. Hypertension was defined as an arterial blood pressure >140/90 mmHg and/or use of antihypertensive medication, as stated in the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.[11] Diabetes was defined as a blood glucose (GLU) level >200 mg/dL and/or diabetic medication.[12] Hyperlipidemia (HL) was diagnosed as cholesterol levels greater than 200 mg/dL: a low-density lipoprotein (LDL) level >130 mg/dL or a triglyceride (TG) level >150 mg/dL.[13] Patients who had smoked in the previous year were considered smokers.

The patients were grouped into high and low Selvester score categories according to the baseline ECG. Individuals were excluded if they had a previous history of CAD or received any cardiac medication, suffered from any infection, any known autoimmune or blood disorder, cancer, renal/hepatic disease, heart issues, or received any coagulant medication before the pPCI, or had experienced resuscitated cardiac arrest or cardiogenic shock.

Abbreviations:

CAD	*Coronary artery disease*
CI	*Confidence interval*
CK	*Creatine kinase*
CKD	*Chronic kidney disease*
ECG	*Electrocardiogram*
ED	*Emergency department*
EF	*Ejection fraction*
GLU	*Glucose*
HDL	*High-density lipoprotein*
HL	*Hyperlipidemia*
LDL	*Low-density lipoprotein*
LV	*Left ventricular*
MHR	*Monocyte count/HDL ratio*
MI	*Myocardial infarction*
MRI	*Magnetic resonance imaging*
OR	*Odds ratio*
pPCI	*Primary percutaneous coronary intervention*
STEMI	*ST-elevation myocardial infarction*
TC	*Total cholesterol*
TG	*Triglyceride*
WBC	*White blood cell*

The current study complied with the Declaration of Helsinki and was approved by the ethics review board of Adiyaman University Education and Research Hospital.

Laboratory parameters

Peripheral venous blood was collected in the emergency department (ED) before the pPCI in dry tubes for biochemical testing and in tubes with ethylenediaminetetraacetic acid for hematological tests. An automated blood cell counter (Beckman Coulter, Inc.; Brea, CA, USA) was used to measure monocytes using the impedance method. Total cholesterol (TC), TG, HDL, and blood glucose plasma concentration ranges were measured using the Cobas 6000 (Roche Diagnostics AG, Basel, Switzerland) enzymatic chemical method. The Friedewald formula was used to calculate LDL levels.[14] The biochemical parameters measured included GLU, creatinine, aspartate aminotransferase, alanine aminotransferase, TG, TC, HDL, and LDL. The complete blood count parameters were hemoglobin, white blood cell (WBC), platelets, and monocytes. The MHR was calculated as the ratio of monocytes to the HDL level. A serial measurement

of monocyte count and lipid panels (day 1, day 2, and 2 weeks after discharge) was performed in all patients and the day-1 MHR, day-2 MHR, and post-discharge MHR were calculated.

Electrocardiographic and echocardiographic analysis

The infarct size was estimated using the QRS score, which is based on 50 criteria capable of generating a total of 31 points. The original complete Selvester score had 54 criteria and 32 points. In the ED, a 12-lead ECG was recorded to determine the QRS score.[15] The ECG trace was ensured to be of good quality, with no left or right bundle branch block, left anterior or posterior fascicular block, left or right ventricular hypertrophy, Wolff-Parkinson-White syndrome, low voltage, or ventricular pacing, which can confound QRS score determination. Patients with such results were excluded from the study. Two observers who were blinded to the clinical data calculated the patients' QRS scores. The ejection fraction (EF) was calculated according to the modified Simpson's method,[16] both in the hospital and 2 weeks after discharge. Serial measurements of ECG (day 1, day 2, and 2 weeks after discharge) were performed in all patients.

The patients were grouped according to high and low Selvester scores according to the baseline ECG. The patients were classified into 2 groups using the median value of infarct size: a small infarct size was assigned a low QRS score, and a large infarct size had a high QRS score.

Coronary angiography and percutaneous coronary intervention

Our hospital is a tertiary clinic with facilities for PCI available 24 hours/day and 7 days/week. Every patient who is admitted to the ED with chest pain is initially assessed by a cardiologist. During PCI processing, all patients received 300 mg aspirin, a 600-mg loading dose and 75-mg maintenance dose of clopidogrel, and a 70 IU/kg dose of unfractionated heparin. PPCI was performed only in the culprit artery, except in cases of cardiogenic shock. Those patients who underwent implantation of a bare metal stent or balloon angioplasty were administered aspirin (100 mg/day indefinitely). Patients who underwent treatment with sirolimus and the implantation of a paclitaxel-eluting stent were administered the same dose of clopidogrel, but for 1 year instead of 1 month. Every patient, no matter the procedure used, was administered beta-blockers during the hospital stay, as well as high-intensity statin therapy (atorvastatin 80 mg/day) unless contraindications appeared.[17] All of the patients were discharged and prescribed a statin (atorvastatin 20 mg/day). A final Thrombolysis in Myocardial Infarction flow grade of 3 was achieved in all participants. Interventional cardiologists who were blinded to the patients' laboratory and clinical follow-up data evaluated the coronary angiograms and determined the Syntax score using the online score calculator (version 2.1).

Statistical analysis

All statistical calculations were performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). Categorical variables were expressed as numbers and proportions. Normally distributed continuous data are shown as mean±SD, and non-normally distributed data are shown as median (interquartile range). The normality of data distribution was analyzed using the Kolmogorov-Smirnov test. The significance of the differences between groups in terms of mean values was determined using the Student's t-test. A chi-squared test was used to compare categorical variables between groups. The Mann-Whitney U test was applied for the comparison of non-normally distributed variables. Variables with a significance level of $p<0.25$ in univariate logistic regression analysis were identified as potential risk markers and included in the full multivariate model as covariates. The final model was constructed by determining the most discriminating factors among the groups using forward logistic regression. Odds ratios (ORs) and confidence intervals (CIs) were determined. A p value <0.05 was considered statistically significant.

RESULTS

The study included 99 patients with STEMI: 52 patients (mean age: 61.3±12.2 years, 84.6% male) were assigned to the low QRS score group, and 47 patients (mean age: 59.5±11.4 years, 63.8% male) were assigned to the high QRS score group. All clinical, demographic, and biochemical characteristics were similar between the QRS score groups except HL and gender ($p>0.05$). The incidence of HL in males was significantly greater in the high QRS score group when compared with the low QRS score group ($p=0.031$ and $p=0.017$, respectively; Table 1).

Among these patients with STEMI, it was observed that the MHR and monocyte count were significantly higher in the high QRS score group on day 1 in the hospital than in the low QRS score group (p=0.001 and p=0.006, respectively). The LVEF was significantly lower in the high QRS score group, both in the hospital and 2 weeks after discharge (p<0.001 and p=0.028, respectively). The other serial measurements of ECG and biochemical characteristics were similar for day 1 and day 2 in the hospital and after discharge (Table 2).

The OR and 95% CI values using the univariate logistic regression model for each parameter are listed in Table 3. According to these results, smoking, MHR,

Table 1. Baseline and biochemical characteristics of the study population

Parameter	Low QRS score (n=52)	High QRS score (n=47)	*p*
Gender (male), (%)	44 (84.6)	30 (63.8)	0.017*
Age, (years)	61.3±12.2	59.5±11.4	0.539
Hypertension (%)	36 (69.2)	39 (83.0)	0.111
Diabetes (%)	15 (28.8)	10 (21.3)	0.387
Smoking (%)	35 (67.3)	31 (66.0)	0.887
Hyperlipidemia (%)	8.1	16.2	0.031*
Infarct-related artery			
Left anterior descending artery (%)	18 (34.6)	24 (51.1)	
Circumflex artery (%)	22 (42.3)	13 (27.7)	0.211
Right coronary artey (%)	12 (23.1)	10 (21.3)	
Location of myocardial infarction			
Anterior myocardial infarction (%)	18 (34.6)	24 (51.1)	0.098
Inferior myocardial infarction (%)	34 (65.4)	23 (48.9)	
Syntax scores	19.2±8.8 (2–42)	17.3±9.8 (1–51.5)	0.161
Glucose (mg/dL)	167.54±84.8 (63–426)	166.09±94.2 (76–428)	0.616
Creatinine (mg/dL)	0.89±0.15 (0.60–1.4)	0.83±0.27 (0.2–1.5)	0.217
Aspartate aminotransferase (U/L)	41±30.1 (13–156)	51.5±31.8 (9–167)	0.093
Alanine aminotransferase (U/L)	32.4±19.7 (6–131)	38.0±21.6 (7–129)	0.182
Platelet ($10^3 \times \mu L$)	237±61.6 (111–385)	236.9±60.6 (113–393)	0.990
White blood cell ($10^3 \times \mu L$)	11.4±3.8 (5.3–20.8)	12.9±3.6 (7.1–21.5)	0.058
Hemoglobin (g/dL)	13.9±1.8	13.4±1.6	0.156

*P value <0.05.

Table 2. In-hospital day 1 clinical and biochemical characteristics of the study participants

Paramaters	Low QRS score (n=52)	High QRS score (n=47)	*p**
Left ventricular ejection fraction (%)	51.8±3.2	42.2±4.2	<0.001*
Triglycerides (mg/dL)	141.5±97.9 (46–405)	144.6±71.2 (37–437)	0.139
Total cholestrol (mg/dL)	184.8±38.1 (127–488)	193.4±57.9 (110–259)	0.715
Low density lipoprotein (mg/dL)	123.4±36.0 (58–306)	130.6±44.4 (55–196)	0.222
High density lipoprotein (mg/dL)	34.4±6.3 (22–50)	32.5±6.5 (20–62)	0.194
Monocytes ($10^3 \times \mu L$)	0.65±0.24 (0.3–1.6)	0.91±0.36 (0.3–1.8)	0.006*
Monocytes/high density lipoprotein ratio	0.01±0.008 (0.007–0.06)	0.029±0.012 (0.009–0.06)	<0.001*

*P value <0.05.

Table 3. Factors associated with QRS score

Variable	Odds ratio*	95% Confidence interval*	*p*
Gender (male, %)	-0.138	-0.348–0.071	0.193
Age, (years)	0.006	-0.001–0.013	0.079
Hypertension (%)	0.334	0.206–0.638	0.696
Diabetes (%)	-0.049	-0.199–0.102	0.466
Smoking (%)	0.026	-0.160–0.212	0.024
Monocytes/ high density lipoprotein ratio	1.545	0.394–5.645	<0.001
Left ventricular ejection fraction (%)	-0.743	-0.897–-0.050	<0.001
Low density lipoprotein (mg/dL)	-0.034	-0.206–0.134	0.474
Triglycerides (mg/dL)	0058	0.012–0.356	0.550
White blood cell ($10^3 \times \mu L$)	0.022	0.006–0.038	0.009

*Values were obtained using univariate logistic regression analysis.

Table 4. Multivariate logistic regression result for QRS score

	Odds ratio*	95% Confidence interval*	*p*
Step 1			
Monocyte/high density lipoprotein ratio	0.395	0.255–0.612	<0.001
Step 2 (final step)			
Monocyte/high density lipoprotein ratio	0.390	0.252–0.605	<0.001
Left ventricular ejection fraction (%)	1.307	1.015–1.683	0.038

Final results of the logistic regression using the forward logistic regression method. Smoking, MHR and LFEF were covariates.

LVEF, and WBC were associated with QRS score. Only LVEF (OR: 1.307, 95% CI: 1.015–1.683; p=0.038) and MHR (OR: 0.390, 95% CI: 0.252–0.605; p<0.001) were determined to be independent predictors of QRS score in the multivariate analysis (Table 4).

DISCUSSION

The current study investigated the association between QRS score and MHR in a STEMI population. Based on the literature, this study is the first to demonstrate a relationship between a high QRS score and MHR, and that an increased MHR could serve as an independent predictor for a high QRS score in patients with STEMI.

In STEMI, pPCI guards against both mortality and infarction during a 6-month period. Since acute myocardial infarction increases the risk of mortality, patient prognosis is required.[18] In this regard, the circulating monocyte count, particularly the proinflammatory monocyte subpopulations (CD14+ CD16++ [intermediate] and CD14+ CD16++ [non-classical]), has emerged as an excellent prognostic marker for plaque development.[19–21] The count, in turn, serves as an important determinant of potential atherosclerosis.[22] Murphy et al.[23] demonstrated the anti-inflammatory role of HDL and AP–1.

During inflammation, monocytes are recruited to the inflamed site and secrete high levels of proinflammatory molecules, including tumor necrosis factor alpha and interleukin 1 beta. In previous studies, a high monocyte count has been shown to be significantly associated with the cardiovascular prognosis, including mortality, in STEMI cases.[24] For instance, increased circulating platelet–monocyte aggregates were observed in patients with acute coronary syndromes,[25] which may induce the expression and release of chemotactic factors, such as monocyte chemoattractant protein 1 and/or interleukin 8.[26] In addition to the facilitation of monocyte adhesion to endothelial cells, monocyte chemoattractant protein 1 induces the expression of tissue factor and superoxide anions, and exerts prothrombotic effects.[27,28] A previous study pi-

oneered the investigation of human monocyte subset dynamics after STEMI.[29] In the present study, the monocyte count was found to be significantly related to the QRS score, an observation that plays an integral role in estimating the infarct size.

As discussed above, mounting evidence demonstrates that an increased monocyte count is associated with both the development and progression of atherosclerotic and proinflammatory processes. However, there is no study in the literature, to our knowledge, that has reported increased monocyte counts due to reperfusion. We, for the first time, have demonstrated that the monocyte count increased significantly on day 2 due to reperfusion. However, the monocyte count decreased significantly after 2 weeks of follow-up.

Kalantar-Zadeh et al.[30] measured and monitored the HDL proinflammatory index in 189 patients for 2½ years. They discovered that a higher index was associated with a higher hazard ratio for death. Interestingly, the HDL level was not different between the groups and was not related to the QRS score in our study. Recently, MHR was identified as a marker for heart disease outcomes.[31–33] Another study showed a relationship between MHR and isolated coronary ectasia.[34] Kanbay et al.[6] monitored 340 patients with stage 1–5 chronic kidney disease (CKD) for 33 months and found that the glomerular filtration rate was not associated with MHR Furthermore, the MHR was shown to be an independent indicator of a major adverse cardiac event in CKD patients with poor heart prognoses. In addition, MHR independently predicted stent thrombosis in STEMI patients and was related to in-hospital major adverse cardiac events.[35,36]

The examination date, set at 8 days post event, was strategically planned, as it was important to observe the initial injury and determine the infarct QRS changes. In addition, it assisted in eluding the post-infarction rate where hyperenhanced myocardium and QRS score were reduced.[37] In our study, the in-hospital day 1, day 2, and 2-weeks post-discharge infarct size was evaluated using QRS scoring.

Selvester et al.[38] established a QRS score to determine infarct size and position using an ECG. This score has been demonstrated as a prognostic marker in several studies.[39,40] The QRS score significantly correlated with the anatomically measured size of single MIs in the anterior, inferior, and posterolateral thirds of the left ventricle and could be used for patients admitted with first episodes of chest pain suggestive of an acute coronary syndrome.[41–43] Kim et al.[44] reported an association between the infarct extent and magnetic resonance imaging (MRI) findings. Jones et al.[45] reported a higher QRS score in relationship to death. Recently, Kalogeropoulos et al.[46] demonstrated the role of the QRS score as a predictor of heart failure in patients with STEMI. For patients surviving a first STEMI, the pre-discharge QRS score demonstrated prognostic value for predicting short-term mortality and/or re-hospitalization due to heart failure.

Limitations

The primary limitations of this study are its single-center design and the limited cohort. A 6-month follow-up might have been more beneficial in terms of prognosis. The Selvester QRS scoring could be improved with the use of MRI or scintigraphy. In addition, we could not analyze all of the factors involved in monocyte interaction. Furthermore, other inflammatory and/or thrombotic markers, including, but not limited to C-reactive protein, fibrinogen, and plasma coagulation factors, were not analyzed for any confounding complications. Future experiments accessing the characterization of the various monocyte subgroups would augment our findings. Further, long-term and larger studies may be required in terms of prognosis.

Conclusions

This study is the first to focus on the relationship between the QRS score and the MHR ratio in patients with STEMI. Of note, we also found a significant relationship between the QRS score and the MHR ratio. Further studies are required to examine this hypothesis.

Peer-review: Externally peer-reviewed.

Conflict-of-interest: None.

Authorship contributions: Concept: L.A, M.Ç., S.T., H.T., E.A.; Design: L.A, M.Ç., S.T., H.T.; Supervision: S.T., H.T.; Materials: L.A, M.Ç., S.T.; Data: L.A, E.A.; Analysis: M.Ç., S.T.; Literature search: L.A, E.A., H.T.; Writing: L.A, M.Ç., S.T., H.T., E.A.; Critical revision: M.Ç., E.A.

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Keywords: Ejection fraction; monocyte to HDL ratio; Selvester QRS score; ST-elevation myocardial infarction.

Anahtar sözcükler: Ejeksiyon fraksiyonu; monosit/HDL oranı; Selvester QRS skor; ST elevasyonlu miyokart enfarktüsü.