# Hematological Inflammatory Markers and Fragmented QRS Complexes in Patients Presenting With Acute Myocardial Infarction

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## ABSTRACT

**Introduction:** Inflammation plays a crucial role in the pathophysiology of acute myocardial infarction (AMI). Recent data suggest that some inflammatory markers may predict presence of fragmented QRS (fQRS) on electrocardiography (ECG) in AMI patients. However, data regarding which inflammatory marker predicts the presence of fQRS more accurately remains unclear. In this study, we aimed to define the strongest predictor of the presence of fQRS on EGC among various hematological inflammatory markers in patients presenting with AMI.

**Patients and Methods:** A total of 906 patients with AMI were included into the study. The association between fQRS and various hematological inflammatory markers such as white blood cell (WBC) count, monocyte count, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte to high-density lipoprotein ratio (MHR) were investigated.

**Results:** The frequency of fQRS was found to be 44.4% in the study population. Patients with fQRS had significantly higher values of WBC, MHR, neutrophil and monocyte counts compared to those without fQRS. The value of NLR and PLR were similar in patients with and without fQRS. In the Receiver operating characteristics (ROC) curves analyses, a monocyte count > 0.79 ($\times 10^3/\mu L$) was found to be a predictor of presence of fQRS on ECG with a specificity of 80.36% and a sensitivity of 60.95% (AUC: 0.754, p< 0.001). Furthermore, multivariate analysis demonstrated that only monocyte count was an independent predictor of fQRS among all hematological inflammatory markers (p< 0.001, odds ratio:1.221, 95% confidence interval:1.078-1.447).

**Conclusion:** Monocyte count is the strongest predictor of fQRS among all hematological inflammatory markers and may be useful in risk stratification of patients with AMI.

**Key Words:** Fragmented QRS; inflammation; acute myocardial infarction; monocyte count

## Akut Miyokard İnfarktüsü ile Başvuran Hastalarda Hematolojik İnflamatuar Belirteçler ve Fragmente QRS

## ÖZET

**Giriş:** İnflamasyon, akut miyokard infarktüsünün (AMI) patofizyolojisinde önemli bir rol oynamaktadır. Yakın zamanlı veriler bazı inflamatuar belirteçlerin AMI hastalarında elektrokardiyografide (EKG) fragmente QRS (fQRS) varlığını öngördüğünü düşündürmektedir. Ancak, hangi inflamatuar belirtecin fQRS varlığını daha doğru öngördüğüne ilişkin veriler net değildir. Çalışmamızın amacı, AMI hastalarında çeşitli hematolojik inflamatuar belirteçler arasında EKG'de fQRS varlığının en güçlü göstergesi olan belirteci tanımlamaktır.

**Hastalar ve Yöntem:** AMI tanısı almış toplam 906 hasta çalışmaya dahil edildi. Beyaz kan hücresi sayısı, monosit sayısı, nötrofil/lenfosit oranı (NLR), platelet/lenfosit oranı (PLR) ve monosit/yüksek yoğunluklu lipoprotein oranı (MHR) gibi inflamatuar belirteçler ile fQRS arasındaki ilişki araştırıldı.

**Bulgular:** Çalışma hastalarında fQRS sıklığı %44.4 olarak saptandı. EKG'de fQRS olan hastalar, olmayanlara göre daha yüksek beyaz kan hücresi, MHR, nötrofil ve monosit sayısına sahipti. NLR ve PLR değerleri, fQRS olan ve olmayan hastalarda benzerdi. Receiver operating characteristics (ROC) curve analizinde monosit sayısının >0.79 ($\times 10^3/\mu L$) olmasının, %80.36 spesifite ve %60.95 sensitivite ile EKG'de fQRS varlığını öngördürdüğü tespit edildi (AUC: 0.754, p< 0.001). Ayrıca, çok değişkenli analizde hematolojik inflamatuar belirteçler arasından sadece monosit sayısının EKG'de fQRS varlığının bağımsız bir göstergesi olduğu saptandı (p< 0.001, odds ratio:1.221, 95% confidence interval: 1.078-1.447).

**Sonuç:** Monosit sayısı, hematolojik inflamatuar belirteçler arasında fQRS varlığının en güçlü göstergesi olup AMI hastalarının risk sınıflandırılmasında kullanılabilir.

**Anahtar Kelimeler:** Fragmente QRS; inflamasyon; akut miyokard infarktüsü; monosit sayısı

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## INTRODUCTION

Inflammation has essential roles in all stages of coronary artery disease (CAD) and is one of the most powerful predictors of adverse cardiovascular events, in particular, due to its effect on the progression of CAD and being a triggering factor for acute coronary syndromes (ACS)[1-3]. As markers of inflammation, circulating white blood cell (WBC) count, its subtypes and their ratios are strong predictors of ACS and future cardiac events[4,5]. Nevertheless, data regarding which inflammatory marker has a stronger relationship with sign and symptoms of cardiac events remains unclear.

A narrow fragmented QRS complex (fQRS) on a 12-lead electrocardiography (ECG) is a sign of myocardial fibrosis and scar tissue and is a prognostic marker in various cardiovascular diseases, and describes different QRS morphologies and notches in the original QRS complexes in the absence of typical bundle branch block[6,7]. Recent studies have demonstrated that increased systemic inflammation and elevated levels of hematological inflammatory markers may be associated with the presence of fQRS on ECG in patients with acute myocardial infarction (AMI)[8,9]. However, no study has investigated the comparison of inflammatory markers for the prediction of the presence of fQRS on ECG in these patients. Therefore, the main goal of our study was to investigate and compare the importance and value of WBC derived hematological inflammatory markers such as WBC count, monocyte count, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte to high-density lipoprotein (HDL) ratio (MHR) for the prediction of the presence of fQRS in patients presenting with AMI. Thus, the present study aimed to investigate the relationship of inflammation with fQRS as an ECG sign of myocardial damage and fibrosis and to describe the strongest predictor of presence of fQRS on ECG in AMI patients among all WBC derived hematological inflammatory markers.

## PATIENTS and METHODS

In this cross-sectional study, 1179 consecutive patients who had received a diagnosis of AMI between September 2016 and September 2018 were retrospectively screened for the study. Among these patients, 273 patients were excluded from the study (patients with complete or incomplete bundle branch block (n= 96), atrial fibrillation at admission (n= 72), active infection, inflammatory disease or malignancy (n= 64), previous cardiac surgery (n= 19), severe valvular heart disease (n= 18), and pacemaker implantation (n= 4)). As a consequence, the remaining 906 patients were included into the study. Patients' demographics recorded at baseline were obtained from medical records. The diagnosis of AMI was made as ST-segment elevation myocardial infarction (STEMI) or Non-ST-elevation myocardial infarction (NSTEMI) according to the current guidelines[3,10]. The treatment of AMI was also made in consistent with the current guidelines including interventional therapy, medical treatment or both according to the patients' clinical status. Vast majority of the study population underwent interventional therapy and subsequent long term optimal medical therapy. Hypertension was defined as systolic blood pressure of ≥ 140 mmHg and/or diastolic blood pressure of ≥ 90 mmHg or known treatment with antihypertensive drugs, diabetes mellitus was defined as at least two fasting plasma glucose levels of ≥ 126 mg/dL and/or 2-hour plasma glucose levels of ≥ 200 mg/dL or HbA1c ≥ 6.5% or known treatment with antidiabetic drugs. All patients underwent a detailed echocardiographic examination. The study was conducted in full accordance with the Declaration of Helsinki and approved by the Usak University Medical School Ethics Committee on 01.07.2020 with reference number '152-05-23'.

Laboratory analyses were performed from venous blood samples obtained immediately after admission. The counts of circulating WBCs and its subtypes were detected by an automated blood cell counter. PLR was calculated as the ratio of the platelet count to lymphocyte count, NLR was calculated as the ratio of neutrophil count to lymphocyte count, and MHR was calculated as dividing the monocyte count by the HDL cholesterol count.

ECGs were obtained from all patients within 10 minutes of admission at the emergency service. In ECG analysis, admission ECGs were used, and the presence of fQRS was defined as an additional R wave leading various RSR′ patterns or notching in R or S waves in the absence of typical bundle branch block in two contiguous leads in one of the major coronary artery territories in the original QRS complex[6]. Two independent experienced cardiologists blindly analyzed all ECGs. In case of disagreement in the definition of fQRS, final decision was achieved by consensus. Figure 1 demonstrates an example of fQRS.

### Statistical Analysis

Statistical analyses were performed using SPSS software version 25.0 (IBM Corparation, Armonk, New York, United States). Normality distribution was tested with Kolmogorov-Smirnov test. Continuous variables were presented using medians and interquartile range for non-normally distributed variables, and categorical variables were presented as number and percentages. Since the continuous variables were not normally distributed, Mann-Whitney U test was used in comparison of these parameters. The proportions of categorical variables were compared with the chi-square test or Fisher's exact test. Receiver operating characteristics (ROC) curves analyses were performed to determine the area under the curve (AUC) of

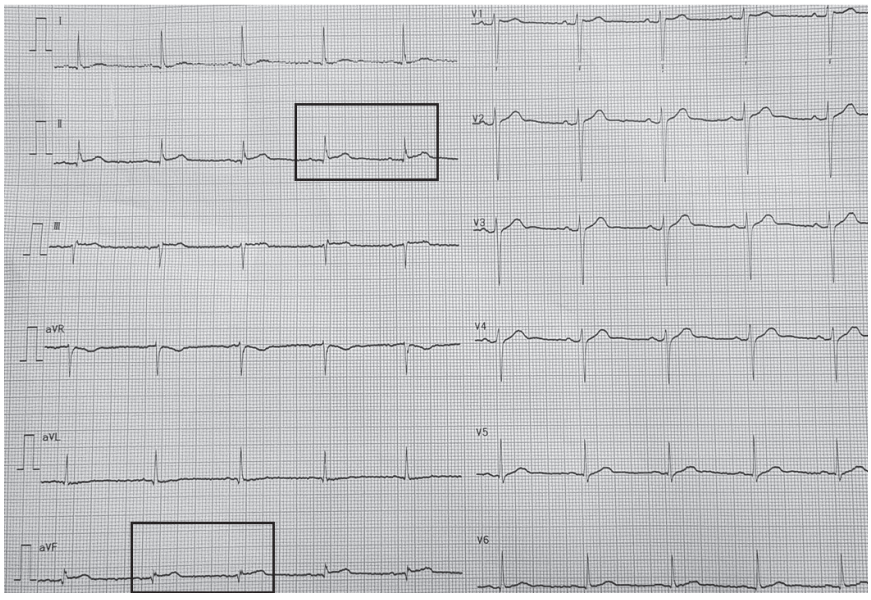


**Figure 1.** An example of fragmented QRS on electrocardiography.

the hematological-inflammatory parameters for predicting the presence of fQRS on ECG. A multivariate logistic regression analysis was performed to define the independent predictors of the presence of fQRS on ECG. All variables with p< 0.05 in the univariate analysis were included into the model. A p-value of < 0.05 was considered to show statistical significance.

## RESULTS

Among the 906 patients with AMI included into the study, 488 (54%) patients presented as STEMI and 418 (46%) patients presented as NSTEMI. Mean age of the study population was 63 years with a predominant male population (73.1%), and the frequency of fQRS was found to be 44.4%. There was no significant difference between STEMI and NSTEMI patients regarding frequency of fQRS (p= 0.071). Patients with fQRS had significantly lower left ventricular ejection fraction (LVEF) and had significantly higher SYNTAX (Synergy Between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery) score compared to those without fQRS. In laboratory analysis, patients with fQRS had significantly higher values of WBC, neutrophil and monocyte counts and troponin I levels compared to the patients without fQRS. When the ratios of hematological inflammatory markers were analyzed, only MHR was significantly higher in patients with fQRS compared to those without fQRS. NLR and PLR were similar in patients with and without fQRS. Table 1 demonstrates clinical and laboratory characteristics of the patients with and without fQRS.

In the ROC curve analysis for defining the predictors of the presence of fQRS on ECG, monocyte count had the highest AUC among all hematological inflammatory markers. A monocyte count > 0.79 ($\times 10^3/\mu L$) was found to be a predictor of the presence of fQRS on ECG with a specificity of 80.36% and a sensitivity of 60.95% (AUC: 0.754, p< 0.001). Additionally, a MHR value of > 0.021 was found to predict the presence of fQRS with a specificity of 81.3% and a sensitivity of 51.5% (AUC: 0.710, p< 0.001 ), and a WBC count of >9.56 ($\times 10^3/\mu L$) was found to predict the presence of fQRS with a specificity of 60.71% and a sensitivity of 60.2% (AUC: 0.621, p< 0.001). Besides the hematological inflammatory markers, SYNTAX score >20 (AUC: 0.759, specificity:79.17%, sensitivity: 62.44 %, p< 0.001) and LVEF ≤ 45 (AUC: 0.626, specificity: 69.25%, sensitivity: 50.5%, p< 0.001) were also found to be predictors of fQRS in the ROC curve analysis. Figure 2 shows the ROC curve of monocyte count (A) and MHR(B) for predicting the presence of fQRS.

Importantly, multivariate logistic regression analysis demonstrated that only monocyte count independently predicted the presence of fQRS among all hematological inflammatory markers (p< 0.001, odds ratio:1.221, 95% confidence interval:1.078-1.447). The other independent predictors of the presence of fQRS were LVEF and SYNTAX score (Table 2).

## DISCUSSION

The present study points out the impact of inflammation on ventricular conduction in patients with AMI. Our study revealed that monocyte count, as a traditional inflammatory marker, may be the strongest predictor of ventricular conduction disturbance detected by fQRS in patients with AMI, among various hematological inflammatory markers. To our knowledge, this is the first study comparing the predictive value of various hematological inflammatory markers regarding the presence of fQRS in the setting of ACS.

**Table 1. Clinical and laboratory characteristics of the patients with and without fragmented QRS**

| | Total (n= 906) | Fragmented QRS (-) (n= 504) | Fragmented QRS (+) (n= 402) | P |
|---|---|---|---|---|
| Age, years (median [IQR]) | 63 (18) | 62 (18.50) | 64 (19) | 0.184 |
| BMI (kg/m$^2$), (median [IQR]) | 26 (3.60) | 26 (3.50) | 26 (3.50) | 0.146 |
| LVEF, % (median [IQR]) | 50 (20) | 50 (15) | 45 (15) | < 0.001 |
| SYNTAX score, (median [IQR]) | 17 (15) | 13 (11) | 23 (16.50) | < 0.001 |
| Male sex, n (%) | 662 (73.1) | 366 (72.6) | 296 (73.6) | 0.763 |
| Diabetes mellitus, n (%) | 540 (59.6) | 300 (59.5) | 240 (59.7) | 0.999 |
| Hypertension, n (%) | 396 (43.7) | 218 (43.3) | 178 (44.3) | 0.788 |
| Previous CAD, n (%) | 269 (29.6) | 121 (24) | 148 (36.8) | < 0.001 |
| STEMI, n (%) | 488 (54) | 284 (58.2) | 204 (41.8) | 0.071 |
| NSTEMI, n (%) | 418 (46) | 219 (52.4) | 199 (47.6) | |
| Troponin I (ng/mL), (median [IQR]) | 4.28 (21) | 3.76 (15.19) | 5.24 (29.45) | 0.001 |
| Triglycerides (mg/dl), (median [IQR]) | 142 (101) | 142.5 (100) | 139.5 (98) | 0.718 |
| Total cholesterol (mg/dl), (median [IQR]) | 183 (63) | 183 (59) | 183 (67) | 0.862 |
| LDL (mg/dl), (median [IQR]) | 109.9 (51) | 111.1 (48.75) | 109 (55.3) | 0.825 |
| HDL (mg/dl), (median [IQR]) | 40 (13) | 39.8 (12.85) | 40.2 (13.9) | 0.679 |
| Creatinine, mg/dl (median [IQR]) | 0.86 (0.28) | 0.86 (0.26) | 0.87 (0.29) | 0.099 |
| WBC count (×10$^3$/μL), (median [IQR]) | 9.50 (4.18) | 8.96 (3.81) | 10.17 (4.24) | < 0.001 |
| Hemoglobin (g/dL), (median [IQR]) | 13.20 (2.99) | 13.45 (2.92) | 13.01 (3.01) | 0,031 |
| Platelet count (×10$^3$/μL), (median [IQR]) | 215 (93) | 211.50 (82.50) | 222 (103) | 0,051 |
| Neutrophil count (×10$^3$/μL), (median [IQR]) | 6.31 (3.97) | 5.94 (3.46) | 6.90 (4.08) | < 0.001 |
| Monocyte count (×10$^3$/μL), (median [IQR]) | 0.68 (0.42) | 0.57 (0.30) | 0.87 (0.45) | < 0.001 |
| Lymphocyte count (×10$^3$/μL), (median [IQR]) | 1.91 (1.22) | 1.89 (1.15) | 1.94 (1.30) | 0.250 |
| NLR, (median [IQR]) | 3.29 (3.25) | 3.07 (3.02) | 3.53 (3.58) | 0.061 |
| PLR, (median [IQR]) | 111.74 (78.04) | 110.06 (74.54) | 112.88 (83.04) | 0.905 |
| MHR, (median [IQR]) | 0.02 (0.01) | 0.01 (0.01) | 0.02 (0.02) | < 0.001 |

IQR: Interquartile range, BMI: Body mass index, LVEF: Left ventricular ejection fraction, CAD: Coronary artery disease, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-ST-elevation myocardial infarction, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, WBC: White blood cell, NLR: Neutrophil-to-lymphocyte ratio, PLR: platelet-to-lymphocyte ratio, MHR: Monocyte to HD ratio.

Although the presence of fQRS on ECG seems to be also associated with non-cardiac conditions[11] and cardiac diseases other than CAD[12,13], its importance and prognostic value is well demonstrated in patients with CAD and AMI[14,15]. The importance of fQRS was first described in patients with CAD, and as a sign of inhomogeneous ventricular conduction, the presence of fQRS on ECG indicates myocardial damage, fibrosis and scar tissue in CAD patients[6,7]. Furthermore, fQRS seems to be associated with impaired cardiac structure, unfavorable left ventricular remodeling, increased risk of adverse events and more severe CAD in patients with AMI[16-18]. Moreover, further studies have indicated that inflammation may be associated with the presence of fQRS on ECG independent of underlying cardiovascular status[8,9]. In this sense, the relationship of inflammation with the ECG signs of myocardial fibrosis is worth to be investigated in detail. Thus, we aimed to investigate which hematological inflammatory marker was the strongest predictor of the effect of inflammation on myocardial fibrosis and conduction disturbances, and we found that monocyte count seemed to be the strongest predictor of fQRS among all hematological inflammatory markers.

Inflammation is a potent trigger of vascular damage and plays a key role in all stages of atherosclerosis from its begin-

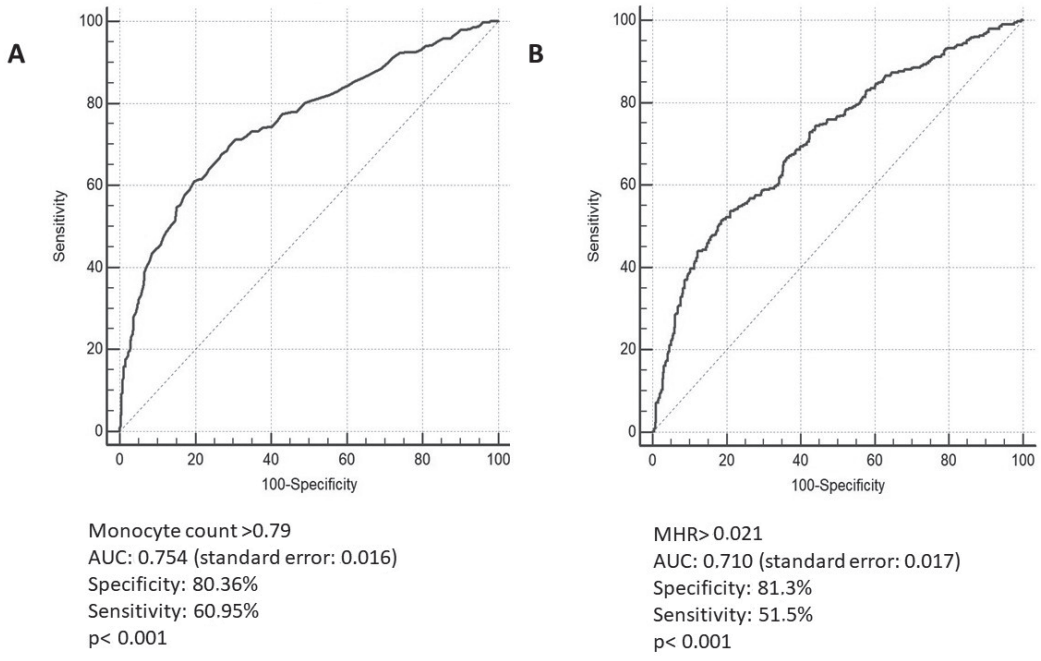


**Figure 2.** ROC curve of monocyte count (A) and MHR (B) for predicting the presence of fragmented QRS.

**Table 2. Independent predictors of the presence of fragmented QRS in multivariate logistic regression analysis**

| Dependent variable | Independent predictors | p | Odds Ratio | 95% Confidence Interval |
|---|---|---|---|---|
| Presence of fragmented QRS | LVEF | < 0.001 | 0.794 | 0.494-0.867 |
| | Monocyte count | < 0.001 | 1.221 | 1.078-1.447 |
| | SYNTAX score | < 0.001 | 1.478 | 1.114-1.592 |

LVEF: Left ventricular ejection fraction.

ning to plaque disruption and subsequent cardiac events[1,2]. Hereby, it seems reasonable to use inflammatory markers for the risk stratification of patients with ACS. As a marker of chronic inflammation, WBC count is a strong predictor of vascular injury, atherogenesis and AMI[19]. Importantly, monocytes have multiple essential roles in all stage of coronary atherosclerosis and seems to have stronger relationship with initiation, progression and complications of CAD, compared to other WBC subtypes[20-22]. Furthermore, circulating monocytes are the main substances of the inflammation in atherosclerotic plaques, and they play a key role in plaque destabilization and subsequent ACS through the secretion of proinflammatory molecules and proteases[23]. Besides the pathophysiologic roles of monocytes, monocyte count is associated with abnormal myocardial perfusion in the setting of AMI, and it also seems to be a predictor of inadequate reperfusion and impaired coronary flow in patients with AMI[24]. Additionally, monocyte count also seems to be associated with endothelial dysfunction and deformation in the coronary vasculature even in the absence of stenotic CAD[25]. Hence, the current evidence strongly suggest that both monocyte count and fQRS are associated with impaired cardiac functions and structure, which is supportive of the key finding of our article that indicates a significant relationship of monocyte count with the presence of fQRS on ECG in patients with AMI.

Previous studies have reported the importance of some other hematological inflammatory markers such as NLR, PLR and MHR in patients with CAD[26-28]. However, we did not find a relationship between these markers and fQRS in our study population. These markers may be useful in patients with CAD and ACS in some conditions; however, the results of our study suggest that these hematological inflammatory markers are not comparable to monocyte count in predicting the presence of fQRS in patients with AMI. We believe that this finding may be mainly due to the unique role of the circulating monocytes in the pathophysiology of coronary atherosclerosis and AMI.

The present study has some limitations. Although the presence of fQRS is a well described sign of fibrosis within myocar-

dium, it may be affected by some other cardiac and non-cardiac conditions, and lack of confirmation of myocardial fibrosis by cardiac magnetic resonance imaging (CMRI) may be the major limitation. However, it is not reasonable to perform CMRI routinely in the setting of AMI, and we tried to include all well described factors associated with the presence of fQRS into the analysis to eliminate confounding factors. Another limitation may be the lack of data regarding the levels of C-Reactive Protein (CRP). The importance of CRP is well described in patients with ACS, and hence it is not routinely measured in clinical practice. Finally, data regarding the localization of fQRS on ECG and number of leads with fQRS were not investigated in this study. The majority of the study population had fQRS in two contiguous leads, and localization of fQRS was mainly in inferior leads. fQRS predicts myocardial fibrosis and impaired cardiac structure independent of its localization on ECG and the number of leads affected, and we mainly aimed to define the association of inflammatory markers with only the presence of fQRS as previously described.

## CONCLUSION

The present study revealed that monocyte count is the strongest predictor of the presence of fQRS among various hematological inflammatory markers in patients with AMI. Numerous inflammatory markers are described and studied in patients with CAD. On the other hand, monocytes seem to have unique roles in the inflammation of atherosclerotic plaques that leads to an acute coronary event. Monocyte count also seems to be the most powerful predictor of deteriorated cardiac functions and myocardial damage among all WBC subtypes. Therefore, as an easily accessible hematological inflammatory marker from routine hemogram measurement, monocyte count may be used to define myocardial fibrotic burden defined via fQRS and may be useful for risk stratification of patients with AMI.

**Ethics Committee Approval:** The approval for this study was obtained from Uşak University Medical School Ethics Committee (Decision no: 152-05-23 Date: 01.07.2020).

**Informed Consent:** This is retrospective study, we could not obtain written informed consent from the participants.

**Peer-review:** Externally peer-reviewed.

**Author Contributions:** Concept/Design - İK, ME; Analysis/Interpretation - İK, ME; Data Collection - İK, ME; Writing - İK, ME; Critical Revision - İK, ME; Final Approval - İK, ME; Statistical Analysis - İK, ME; Overall Responsibility - İK, ME.

**Conflict of Interest:** The authors declared that there was no conflict of interest during the preparation and publication of this article.

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