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# Relation between CRP-Albumin ratio and left atrial volume index for prediction of new onset atrial fibrillation in patients with STEMI treated with percutaneous coronary interventions

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**Abstract**

New onset atrial fibrillation (NOAF) in the case of acute myocardial infarction appears to be associated with inflammation. However, its influence alongside other risk factors is currently unknown. In this study, the effect of inflammation in predicting NOAF independent of left atrial volume index (LAVi) was investigated with a new marker, the C-reactive protein/Albumin ratio (CAR). We included 945 ST-elevation myocardial infarction (STEMI) patients who underwent pPCI. Two groups were defined according to the presence of NOAF and the groups were compared for demographic, clinical and angiographic findings. Predictors of the NOAF were assessed by multivariable regression analysis. Fifty-five (5.8%) patients had NOAF after the procedures. CAR was substantially higher in patients with NOAF (5.9 [4.9] vs 0.46 [1] p<0.01). CAR was shown as an independent marker for NOAF (OR:1.19 95% CI: 1.11-1.27 p<0.01) development in multivariable regression analysis. In receiver operating curve characteristics, the sensitivity and the specificity of the value CAR>4.2 were 64% and 81% (AUC: 0.86) respectively. In subgroup analysis, OR of CAR in LAVi ≥26.7 ml/m² was 19.5 and 22.1 in LAVi<26.7 ml/m² (p for interaction=0.02). A novel inflammatory marker, CAR has the potential to predict the development of NOAF regardless of LAVi in patients with STEMI treated by PPCI.

**Keywords:** Crp/albumin ratio, STEMI, atrial fibrillation, LAVi

## Introduction

New-onset atrial fibrillation (NOAF) in the setting of ST segment elevation myocardial infarction (STEMI), occurs frequently. The incidence ranges between 2.3-21% and patients with NOAF have a poorer prognosis [1]. Left atrial volume index (LAVi) is a powerful marker for atrial fibrillation (AF) development in patients with STEMI [2]. Left atrial enlargement is associated with aging, heart failure, hypertension or diabetes [3]. Changes in atrial architecture serve as a substrate for AF development.

AF occurrence in the course of STEMI has several explanations [4]. An association between AF and inflammation was demonstrated both in population-based studies and acute myocardial infarction patients (AMI) [5,6]. C-reactive protein (CRP) is a good indicator for inflammatory process and was shown to be associated with NOAF in AMI [7]. Recently, studies examining the role of CRP/albumin ratio (CAR) in various cardiovascular diseases, have revealed a stronger relationship between inflammation than CRP alone [8,9].

LAVi is the sign of chronically increased hemodynamic burden and atrial remodeling. However, all STEMI patients with increased LAVi do not develop AF and vice versa. This scenario indicates the potential role of inflammation as a trigger for AF development independent from LAVi. In this regard, we investigated the predictive performance of CAR and interaction with LAVi in patients with STEMI who developed AF after primary percutaneous coronary interventions (PCI).

## Materials and Methods

### Study Design and Patient Population

STEMI patients treated with pPCI between the years 2014-2017 were enrolled in this current study. Patients with a previous history of atrial fibrillation/flutter or on anti-arrhythmic medications

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were not included. Active infection, liver disease, sepsis, chronic inflammatory diseases, nephrotic syndrome, malignancy or steroid usage and need for emergency surgery were considered as exclusion criteria. After these exclusion, the remaining 945 patients were included. The study population, then divided into two groups according to the absence or presence of NOAF. Demographic, clinical characteristics and laboratory parameters were collected from hospital records. The study protocol was approved by the XXX Local Ethics Committee (approval number: 28001928-604.01.01, date: 23.12.2021).

NOAF developed after pPCI during hospitalization was defined as the primary outcome. Atrial fibrillation was characterized with irregular RR intervals and no visible P waves on ECG or telemetry records. NOAF was the arrhythmia which was lasted more than ≥30 seconds and self-terminated or cardioverted electrically or medically. Continuous monitoring was done for all patients in the intensive care unit (ICU). After discharge from ICU, daily ECGs were performed and additional ECGs were recorded when patients reported symptoms. STEMI was defined as typical chest pain associated with classical ECG findings according to the universal guidelines [10].

Major adverse cardiovascular events (MACE) compounded by heart failure, cardiogenic shock, acute stent thrombosis and re-infarction. Mortality was composed from death occurred from any cause in 30 days of admission.

**Revascularization Procedure and Medications**

Invasive evaluation was performed for all patients using standard angiographic techniques as recommended by the latest guidelines. Clopidogrel (600mg) and acetylsalicylic acid (300mg) were the standard antiplatelet therapy. After angiographic evaluation 100u/kg heparin or 1mg/kg low molecular weight heparin was given. Bare metal or drug eluting stents were used for revascularization procedures and other treatments including thrombus aspiration or additional antithrombotic medications was individualized by the operator. Dual antiplatelet treatment was consisted of clopidogrel 75 mg and acetylsalicylic acid at hospital discharge and β blocker, angiotensin converting enzyme inhibitor or statin treatment have been given unless contraindicated. Patients with documented NOAF were also anticoagulated according to their ischemic risk.

**Laboratory Parameters and Echocardiography**

Blood samples were taken at first admission in the emergency department before primary PCI via antecubital vein. CRP and serum albumin level measurements were performed by Cobas Integra Analyzer (Roche Diagnostic Turkey). CAR was calculated by dividing CRP levels to albumin levels. Normal reference values for CRP were between the range 0-5 mg/dl and was 3.4-5.5g/dl for serum albumin. Transthoracic echocardiographic evaluation was performed to evaluate LVEF after primary PCI procedure (Vivid-5 (General Electric Company Milwaukee, WI). The formula A1 (parasternal long axis; anteroposterior) × A2 (apical four-chamber; mediolateral)×A3 (apical four-chamber; apicobasal)×0.524 was used to calculate the left atrial volume and after then LAVI was calculated based on body surface area.

**Statistics**

Normally distributed variables were demonstrated as mean ± standard deviation (SD) and variables without normal distribution on a median [inter-quantile range] in statistical analysis. Numbers and percentages were used to specify the categorical variables and Kolmogorov-Smirnov statistics was the choice to check the normal distribution for continuous variables. Categorical variables were analyzed by Pearson's $\chi 2$ test and Fisher's Exact tests. Mann–Whitney U test or the Student t-tests were used to compare for differences between the NOAF (+) versus NOAF (-) groups. Independent predictors of NOAF were evaluated with univariable and multivariable binary logistic regression analyses. Variables which have P values <0.10 in univariable analyses were put into the multivariable regression analyses. The AUC and cut-off values of CAR for NOAF prediction was done by receiver operating curves analysis. P-values <0.05 indicated statistical significance. The ROC curves of CAR and CRP were compared with DeLong test using the MEDCALC software program (Softwarebvba 13, Ostend, Belgium). Univariable and multivariable regression analyses of CAR for predicting NOAF were performed in both LAVi subgroups and OR's were demonstrated. Interaction p values were found from general linear model regression analyses. Statistical Package for Social Sciences software (SPSS 22.0 for Windows, SPSS Inc., Chicago, Illinois) program was used for statistical analyses.

**Results**

Nine hundred and forty-five STEMI patients (mean age: 57.1±12.3 male n=704, 74.5%) who treated by pPCI were reviewed. NOAF was present in 55 patients (5.8%). Older patients and (66.7±11.8 vs 56.5±12.5 p<0.01) and patients with poorer LVEF (35.1±6.9 vs 44.3±11.5 p<0.01) were seen in NOAF (+) group more frequently. In addition, presentation in Killip class >3-4(16.4% vs 7.4% p=0.01) was more frequent in NOAF (+) patients. NOAF (+) patients had higher LAVi (34.1±10.9 vs 28.2±4.9 p<0.01) and heart rates (95.5±24.3 vs 79.6±17.3 p<0.01) when compared with NOAF (-) patients. RCA involvement was observed more often in the NOAF (+) group (47.3% vs 31.2% p=0.01) at angiographic evaluation. In comparison with NOAF (-) group, patients in the NOAF (+) group had higher MACE (17.3% vs 6.3% p=0.02) and mortality (13.5% vs 2.8% p<0.01) rates. The other parameters were comparable between the groups and baseline features of those groups were provided in Table-1.

Comparison of laboratory parameters was shown in Table-2. Patients in the NOAF (+) group had higher serum glucose, CRP and creatinine and lower albumin levels. CAR was higher in the NOAF (+) group (5.9 [4.9] vs 0.46 [1] p< 0.01). Other laboratory parameters were similar between groups.

All variables were investigated with univariable and multivariable binary logistic regression analysis for identifying the independent predictors of NOAF. In univariable regression analyses age, heart rate, Killip class, LAVI, LVEF, serum glucose, RCA involvement, creatinine and CAR were found to be correlated with the occurrence of NOAF. After putting these variables into the multivariable regression analysis age (OR:1.04 95% CI: 1.01-1.07 p<0.01), LV ejection fraction (OR:0.94 95% CI: 0.91-0.96 p<0.01), LAVI (OR: 1.12 95% CI:1.07-1.17 p<0.01), CAR (OR:1.19 95% CI:1.11-1.27 p<0.01) and RCA involvement (OR:3.03 95% CI: 1.50-6.12 p<0.01) were detected as predictors of new onset atrial fibrillation. These data were represented in Figure-1.

CAR levels ≥4.2 had a sensitivity of 64% and a specificity of %81 (AUC: 0.86; 95% CI:0.81-0.90 p<0.01) for the prediction of NOAF development in ROC curve analysis. This was demonstrated in the Figure-2. In addition, the comparison of the ROC curves demonstrated that, CAR had a better predictive performance rather than CRP alone (AUCCAR: 0.86; 95% CI:0.81-0.90 vs AUCCRP: 0.79; 95% CI:0.75-0.83 p<0.001). This data was also depicted in Figure-3.

**Table 1.** Demographic, clinic, echocardiographic and angiographic characteristics of patients

| Characteristics | Overall | NOAF (+) | NOAF (-) | P value |
|---|---|---|---|---|
| **n** | **945** | **55** | **890** | |
| **Demographics** | | | | |
| Age, years | 57.1±12.3 | 66.7±11.8 | 56.5±12.1 | **<0.001** |
| Males, n (%) | 704 (74.5%) | 37 (67.3%) | 667 (74.9%) | 0.20 |
| **Medical History** | | | | |
| Hypertension, n (%) | 400 (42.4%) | 27 (49.1%) | 373 (42%) | 0.29 |
| Diabetes Mellitus, n(%) | 170 (18%) | 10 (18.2%) | 160 (18%) | 0.96 |
| Smoking, n (%) | 616 (65.4%) | 30 (54.5%) | 586 (66.1%) | 0.08 |
| Hyperlipidemia, n (%) | 134 (14.2%) | 9 (16.4%) | 125 (14.1%) | 0.63 |
| PAH, n (%) | 15 (1.6%) | 0 (0%) | 15(1.7%) | 0.33 |
| CVA, n (%) | 21 (2.2%) | 1 (1.8%) | 20 (2.2%) | 0.83 |
| Previous CAD, n (%) | 163 (17.2%) | 10 (18.2%) | 153 (17.2%) | 0.85 |
| **Previous Medications** | | | | |
| β-blockers, n (%) | 78 (8.3%) | 8 (14.3%) | 70 (7.9%) | 0.08 |
| ACEI-ARB, n (%) | 206 (21.8%) | 17 (30.9%) | 189 (21.2%) | 0.09 |
| Statin, n (%) | 79 (8.4%) | 5 (9.1%) | 74 (8.3%) | 0.09 |
| Acetylsalicylic asid | 116 (12.3%) | 10 (18.2%) | 106 (11.9%) | 0.16 |
| Clinic and echo findings | | | | |
| SBP, (mmHg) | 121.7±20.6 | 121.0±21.8 | 121.7±20.6 | 0.79 |
| Heart rate, (bpm) | 80.5±18.1 | 95.5±24.3 | 79.6±17.3 | **<0.001** |
| Killip class 3-4, n (%) | 75 (7.9%) | 9 (16.4%) | 66 (7.4%) | **0.017** |
| LAVI (ml/m²) | 28.6±5.6 | 34.1±10.9 | 28.2±4.9 | **<0.001** |
| LVEF (%) | 43.8±11.5 | 35.1±6.9 | 44.3±11.5 | **<0.001** |
| **Angiographic findings** | | | | |
| Culprit vessel | | | | |
| LAD, n (%) | 436 (46.1%) | 23 (41.8%) | 413 (46.4%) | 0.50 |
| LCx, n (%) | 176 (18.6%) | 6 (10.9%) | 170 (19.1%) | 0.13 |
| RCA, n (%) | 304 (32.2%) | 26 (47.3%) | 278 (31.2%) | **0.013** |
| Multivessel disease, n(%) | 426 (45.1%) | 29 (52.7%) | 397 (44.6%) | 0.24 |
| Post-procedural TIMI3 | 154 (16.3%) | 14 (25.5%) | 140 (15.7%) | 0.058 |
| 0 | 13 (1.3%) | 1 (1.8%) | 12 (1.3%) | 0.67 |
| 1 | 43 (4.6%) | 2 (3.6%) | 41 (4.6%) | 0.24 |
| 2 | 97 (10.3%) | 8 (%14.5) | 89 (10%) | 0.41 |
| 3 | 791 (83.7%) | 44 (80%) | 747 (83.9%) | 0.70 |
| Use of stents n(%) | 894 (94.6%) | 52 (94.5%) | 842 (94.6%) | 0.48 |
| GpIIb/IIIa inhibitor n (%) | 375 (39.7%) | 20 (36.4%) | 355 (39.9%) | 0.60 |
| MACE n (%) | 65 (6.3%) | 9 (17.3%) | 56 (6.3%) | **0.021** |
| Mortality n (%) | 28 (3.5%) | 7 (13.5%) | 21 (2.8%) | **<0.001** |

ACEI/ARB Angiotensin converting enzyme inhibitor/Angiotensin receptor blocker, CAD coronary artery disease, CVA cerebrovascular accident, LAVI; left atrium volume index, LAD left anterior descending, LCx left circumflex, LVEF left ventricular ejection fraction, MACE; major cardiovascular events, PAH peripheric arterial disease, RCA right coronary artery disease, SBP; systolic blood pressure TIMI; thrombolysis in myocardial infarction

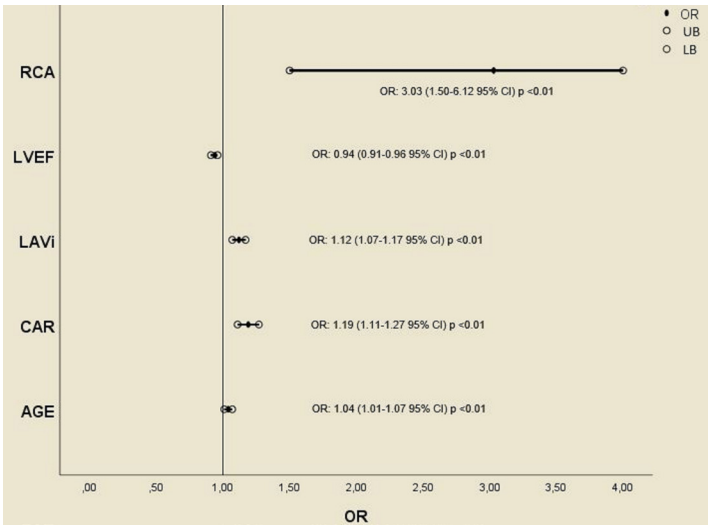


**Figure 1.** Forest plots of independent predictors of NOAF development in multivariate regression analysis

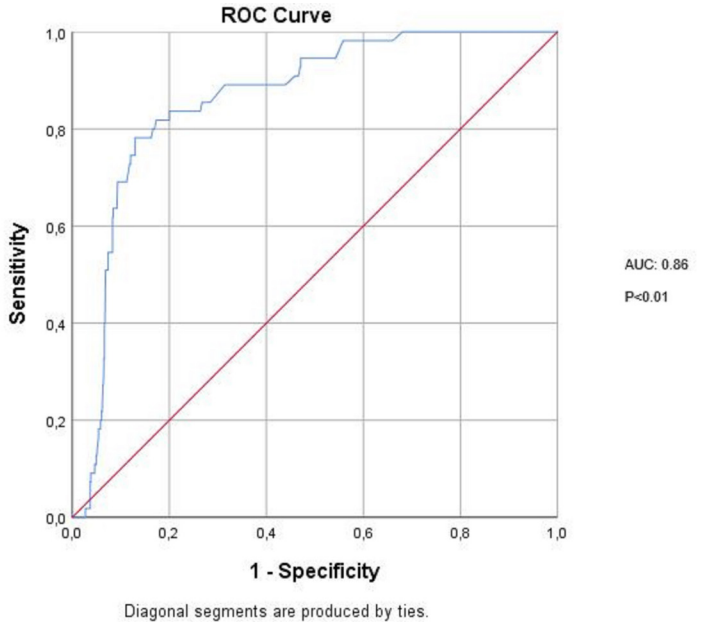


**Figure 2.** Receiving operator curve analysis of the C-reactive protein/Albumin ratio for new-onset atrial fibrillation

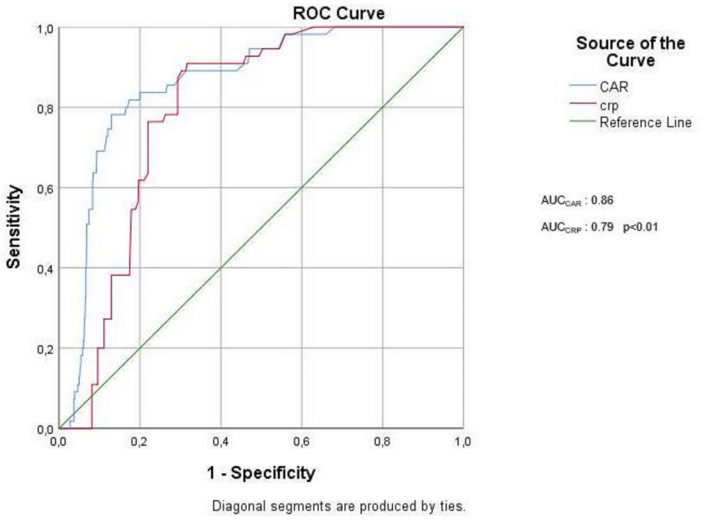


**Figure 3.** Comparing receiving operator curve analysis of C-reactive protein and C-reactive protein/Albumin ratio

We divided the study population into two subgroups according to the median LAVI value 26.7ml/m² and performed univariable and multivariable regression analyses for predicting NOAF in both subgroups. We also determined a median value for CAR (0.51) and used this categorical value for this regression analyses. In this way, we obtained OR's of categorical CAR predicting NOAF in both subgroups. (Categorical CAR was corrected with age, heart rate, Killip class, LVEF, serum glucose, RCA involvement and creatinine in multivariable regression analyses). (Table 3) In subgroup analysis, we determined significantly greater increase in NOAF risk in patients with high CAR in LAVI<26.7ml/m² compared to LAVI>26,7ml/m² subgroup. (LAVI<26.7ml/m² adjusted OR: 22.1 vs. LAVI>26.7ml/m² adjusted OR: 19.5, p-value for interaction=0.02)

**Table 2.** Comparsion of laboratory parameters between NOAF (+) vs NOAF (-) groups

| Laboratory Parameters | Overall | NOAF (+) | NOAF (-) | P value |
|---|---|---|---|---|
| n | 945 | 55 | 890 | |
| WBC 10^3 µ/L | 12.3±3.7 | 13.2±3.5 | 12.2±3.7 | 0.06 |
| Hemoglobin g/dl | 13.9±1.6 | 13.6±1.7 | 13.9±1.6 | 0.14 |
| Platelet 10^3µ/L | 249.1±74.5 | 236.3±77.1 | 249.9±74.3 | 0.18 |
| Glucose mg/dl | 157.2±75.8 | 202.1±113.3 | 154.4±72.5 | **<0.001** |
| Sodium mEq/L | 137.1±4.1 | 136.3±5.1 | 137.1±4.1 | 0.13 |
| Potassium mEq/L | 4.1±0.5 | 4.2±0.6 | 4.1±0.4 | 0.30 |
| Creatinine mg/dl | 0.93±0.42 | 1.22±1.0 | 0.91±0.34 | **0.024** |
| C-reactive protein mg/dl | 2.1 [4.3] | 20 [16.5] | 1.8 [4.1] | **<0.001** |
| Albumin mg/dl | 3.8 [0.4] | 3.6 [0.8] | 3.9 [0.4] | **<0.001** |
| CAR | 0.51 [1.1] | 5.9 [4.9] | 0.46 [1] | **<0.001** |
| Total cholesterol mg/dl | 186.6±42.4 | 184.1±96.3 | 186.8±42.7 | 0.65 |
| LDL cholesterol mg/dl | 118.4±35.1 | 113.9±29.2 | 118.7±35.5 | 0.34 |
| HDL cholesterol mg/dl | 36.1±5.8 | 35.1±5.5 | 35.8±5.7 | 0.20 |
| Triglyceride mg/dl | 152.1±90.2 | 131.1±70.5 | 153.3±11.9 | 0.08 |
| c-Troponin I ng/ml | 6.7 [43] | 8.8 [49.2] | 6.6 [40.9] | 0.62 |

CAR C-reactive protein albumin ratio, HDL high density lipoprotein, LDL low density lipoprotein.

**Table 3.** The OR' s of CAR derived from univariable and multivariable regression analyses for predicting NOAF in LAVi subgroups in patients with STEMI

| | Unadjusted OR (%95 CI) | p value | Adjusted* OR (%95 CI) | p value |
|---|---|---|---|---|
| | **LAVI<26.7 ml/m²** | | **LAVI<26.7 ml/m²** | |
| CAR | 18.5 (2.4-141.2) | **<0.001** | 22.1 (5.2-92.9) | **<0.001** |
| | **Unadjusted OR (%95 CI)** | | **Adjusted* OR (%95 CI)** | |
| | **LAVI≥26.7 ml/m²** | | **LAVI≥26.7 ml/m²** | |
| CAR | 9.9 (1.2-80.6) | **0.03** | 19.5 (4.3-87.5) | **<0.001** |

CAR, C-Reactive protein albumin ratio, LAVI; left atrium volume index. *Adjusted for age, heart rate, Killip class, LVEF, serum glucose, RCA involvement

## Discussion

In this study our findings are as follows 1) CAR and LAVI were independent predictors for the development of new onset atrial fibrillation in patients with STEMI treated by primary PCI 2) predictive value of CAR for NOAF development was better than CRP alone 3) the subgroup analysis revealed that high CAR predicted NOAF development in both LAVI subgroups 4) the predictive power of high CAR was more pronounced in LAVI<26.7 ml/m² group.

In the STEMI setting, new-onset atrial fibrillation is a relatively frequent complication and leads to a worse prognosis by reducing left ventricular contraction, exacerbating heart failure symptoms and attenuating coronary perfusion. The incidence of NOAF in this study was 5.8% as consistent with the literature [1]. In agreement with previous studies, we identified several risk factors for NOAF development including age, low LVEF, RCA involvement and LAVI [2,11-13]. In addition, we found out higher 30-days MACE rates and mortality in NOAF patients.

Atrial fibrillation is a multifactorial process and the link between inflammation and AF pathophysiology has gained attention [14]. Inflammatory infiltrates were shown in histological evaluation of atrial tissue samples in AF patients compared with normal subjects [15]. Chung et al. demonstrated two folds increased CRP levels in AF patients [16]. Their studies also revealed an association between CRP levels and AF burden where patients with higher CRP levels had more persistent AF. Moreover, increased CRP levels were demonstrated to be a predictor for unsuccessful cardioversion [17]. In addition, statin use through its anti-inflammatory effects was supported to decrease the electrical cardioversion failure and AF recurrence in several studies [18]. Despite, large volume data, whether inflammation is a trigger for AF development or the arrhythmia itself produces inflammation is controversial.

Distinct mechanisms exist for AF development in STEMI patients such as atrial infarct and an acute rise in left atrial pressures due to LV dysfunction [19]. However, these mechanisms can not be expanded to all NOAF patients and the precise mechanism is not fully understood. The association between inflammation and AF development was also shown in AMI patients. Aronson et al. studied patients with AMI and revealed positive correlation between increasing CRP tertiles and NOAF after adjusting for clinical variables and left ventricular ejection fraction [20]. Necrosis of the myocytes induce inflammatory response both in the myocardium and systemic circulation [21]. The inflammatory reaction is not confined to the ischemic myocardium and CRP bound to phosphocholine on cell membranes activates complement cascade resulting more accumulation of inflammatory infiltrates [22]. Thus, increased local inflammation and tissue necrosis in atrial myocardium may precipitate AF development. A relationship between inflammation and atrial electrical properties was shown in clinical studies. Increased CRP levels were associated with frequent atrial ectopies [23]. In a study, the prolonged P wave duration after exercise, was accompanied with increased levels of CRP which suggest that acute changes in inflammatory markers are associated with atrial electrical conduction disturbances despite atrial volume remains unchanged [24]. Atrial remodeling may include some degree of low-grade inflammation beside atrial fibrosis, as reflected by population-based studies [16]. However, inflammatory response in STEMI patients is much higher than stable coronary artery disease [25] and may be the trigger for AF development even in patients not susceptible for the arrhythmia. In this regard, we evaluated the potential role of CAR on top of LAVi for predicting NOAF development in patients STEMI. A novel marker, CRP/albumin ratio, has been proved to reflect the underlying inflammatory state better than CRP or albumin alone [8,9]. Considering the latest data, we preferred to use CAR as an inflammatory marker instead of CRP.

In this current study, CAR was superior to CRP for predicting NOAF in STEMI patients according to the ROC analysis. A cut off point 4.2 predicted the development of NOAF in patients with STEMI treated by pPCI. A study which was performed in cardiac surgery patients has shown an independent association between CAR and postoperative atrial fibrillation similar to our results [26]. LAVi is a well-defined risk factor for NOAF [2] as it has been in our study. LAVi reflects atrial enlargement due to atrial remodeling and has a direct effect in the occurrence and maintenance of AF. Interaction analysis in this study demonstrated that CAR was effective to predict NOAF development in both LAVi subgroups. Moreover, the efficacy of CAR was higher in patients with LAVi< 26.7 ml/m². Our study is far from establishing a clear mechanism for AF development however, we can assume that the high burden of inflammation in STEMI patients may be responsible for inducing NOAF even in patients whom have LAVi<26.7 ml/m².

A few limitations existed for our study. The mode of detection of the arrhythmia may lead us to underestimate the exact number of patients. Atrial fibrillation was detected in daily surface electrocardiography or when patients reported symptoms. Due to this, short or asymptomatic episodes of AF might be missed. Laboratory parameters before pPCI were recorded once and no consecutive measurements done which may change over time. Additionally, paroxysmal episodes of atrial fibrillation before admission might be missed, which may interfere with our results. Finally, our study had a relatively small sample size and designed as a retrospective study in single center.

## Conclusion

In conclusion, elevated CAR and LAVI were independently associated with an increased risk of AF development in STEMI patients treated by PPCI. The effect of CAR appeared in both LAVI subgroups. Those patients with high CAR were more prone to NOAF development regardless of LAVI, thus reflecting the importance of inflammation in NOAF pathogenesis. However, more comprehensive studies are needed to define the cause-effect relationship between inflammation and NOAF in STEMI and whether therapies targeting to reduce CRP levels can prevent AF development.

**Conflict of interests**
*The authors declare that there is no conflict of interest in the study.*

**Financial Disclosure**
*The authors declare that they have received no financial support for the study.*

**Ethical approval**
*The study protocol was approved by the University of Health Sciences, Dr Siyami Ersek Cardiovascular and Thoracic Center ethical committee in 23.12.2021 with the number E-28001928-604.01.01.*

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