Cukurova Medical Journal
ÇUKUROVA ÜNİVERSİTESİ TIP FAKÜLTESİ

Cukurova Med J 2019;44(2):416-424
DOI: 10.17826/cumj.444433

ARAŞTIRMA / RESEARCH

Arrhythmias in patients with acute ST elevation myocardial infarction

Akut ST elevasyonlu miyokard enfarktüslü hastalarda aritmiler

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Cukurova Medical Journal 2019;44(2):416-424

Abstract

Purpose: Arrhythmias and conduction disturbances are common during acute myocardial infarction (AMI) and a major cause of death in the pre-hospital phase. The aim of this study was to investigate common predictors for all arrhythmias in patients with ST elevation AMI (STEMI) during in-hospital phase.

Materials and Methods: Ninety patients (74 male, 55 ±11 years) with acute STEMI were included. Clinical charesteristics were recorded and laboratory parameters including serum C- reactive protein (CRP), creatinine kinase MB (CKMB) and potassium levels were measured. The patients were divided into two groups according to development of arrhythmias.

Results: Group 1 (n=42) patients had new onset arrhythmias and Group 2 (n=48) patients had without arrhythmias. Median baseline CRP levels were significantly higher in group 1 (36.6 (21.8-77) mg/dl vs. 21.8 (24.2-30.7) mg/dl), especially in patients with atrial fibrillation and ventricular arrhythmias. Logistic regression analysis showed that baseline higher CRP level, peak CKMB level and inferior localization of AMI were significantly associated with the development of arrhythmia following AMI.

Conclusion: Levels of CRP and CKMB and inferior infarct localization have predictive values for all the arrhythmic events during AMI. CRP levels were found to be associated with both atrial and ventricular arrhythmias. The assessment of CRP levels can be used to detect patients at high risk for arrhythmic events after STEMI.

Keywords: Myocardial infarction, cardiac arrhythmias, inflammation

Öz

Amaç: Aritmiler ve iletim bozuklukları akut miyokard enfarktüsünde (AMİ) yaygındır ve hastane öncesi dönemde önemli bir ölüm nedenidirler. Bu çalışmanın amacı, hastane içi dönemde ST yükselmeli AMI (STEMI) hastalarında tüm aritmiler için ortak öngördürücüleri araştırmaktı.

Gereç ve Yöntem: Akut STEMI olan 90 hasta (74 erkek, 55±11 yıl) çalışmaya dahil edildi. Hastaların klinik özellikler kaydedildi ve serum C-reaktif protein (CRP), kreatinin kinaz MB (CKMB) ve potasyum düzeyleri gibi laboratuvar parametreleri ölçüldü. Hastalar aritmilerin gelişip gelişmemesine göre hastalar iki gruba ayrıldı.

Bulgular: Grup 1 (n=42) hastalar yeni başlangıçlı aritmi ve Grup 2 (n=48) olan hastalar aritmisi olmayan hastalardı. Medyan bazal CRP düzeyleri, özellikle atriyal fibrilasyon ve ventriküler aritmiler olan hastalarda grup 1'de (36.6 (21.8-77) mg/dl'ye karşı 21.8 (24.2-30.7) mg/dl, P = 0.002) anlamlı olarak yüksek bulundu. Lojistik regresyon analizi, başlangıç yüksek CRP düzeyi, tepe CKMB düzeyi ve inferior lokalizasyonlu AMI'nin AMI sonrası aritmi gelişimi ile anlamlı olarak ilişkili olduğunu gösterdi.

Sonuç: CRP ve CKMB düzeyleri ile inferior lokalizasyonlu infarktüsün, AMI sırasında tüm aritmik olaylar için öngördürücü değere sahip olduğunu bulduk. CRP düzeylerinin hem atriyal hem de ventriküler aritmiler ile ilişkili olduğu bulunmuştur. CRP düzeylerinin değerlendirilmesi, STEMI'den sonra aritmik olaylar için yüksek risk taşıyan hastaları tespit etmek için kullanılabilir.

Anahtar kelimeler: Miyokard infarktüsü, kardiyak aritmiler, inflamasyon

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Geliş tarihi/Received: 17.07.2018 Kabul tarihi/Accepted: 29.10.2018 Çevrimiçi yayın/Published online: 24.02.2019

INTRODUCTION

Arrhythmias and conduction abnormalities are common complications of AMI, which are a major cause of death within the first 3 days in AMI[1-4]. It's primarily a consequence of heightened adrenergic nervous system tone, ischemia and myocardial necrosis, hypokalemia, hypomagnesemia, intracellular hypercalcemia, acidosis, free fatty acid production from lipolysis, and increased free radical production from reperfused ischemic myocardium (Figure 1)[5,6].

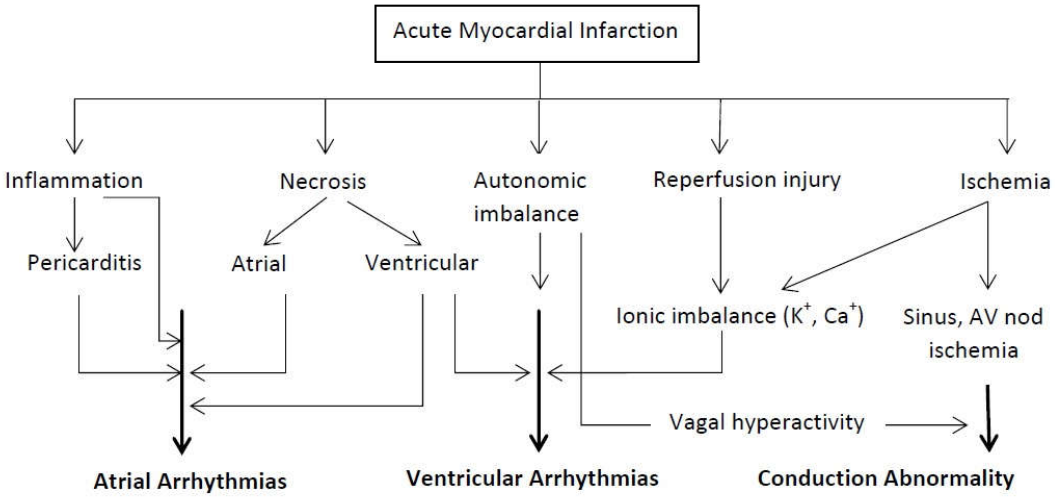

Figure 1. Possible mechanism of arrhythmias in setting of AMI.

Inflammation is associated with short-and long-term prognosis following the AMI[7-9]. Many reports have been published in association between inflammation and atrial fibrillation (AF) in patients with[10,11] and without[12-15] AMI. However, the relation of inflammation and other type of arrhythmias after AMI were not evaluated in these studies. The association of potassium levels and infarct location with arrhythmia is already known. Potassium (K^+) levels at admission had a significant relation with ventricular arrhythmias[16,17] and patients with inferior wall AMI are more prone to development of conduction abnormalityt[18]. In previous studies, single type of arrhythmias were primarily investigated, rather than examining all arrhythmias after AMI. Therefore, the aim of this study was to investigate common predictors for all arrhythmias during AMI.

MATERIALS AND METHODS

In this prospective study, 96 patients with their first STEMI were recruited and finally 90 patients (74 male, 55 ±11 years) were included. The patients were followed up for the development of new onset arrhythmias during their hospitalization. Accordingly, patients were assigned into two groups: group 1 (n=42); patients with new onset arrhythmias and group 2 (n=48); control group, patients without arrhythmias.

All patients had been admitted to our clinic within 12 hours of STEMI. Diagnosis of STEMI was made on the basis of a new ST-segment elevation of 2 mm or more on at least two adjacent leads and either of the following criteria: typical chest pain or increase in serum cardiac enzymes. Infarct localization was determined in all patients. We excluded patients who died prior to the laboratory investigation, and those with dilated cardiomyopathy, prior AMI, prior antiarrhythmic therapy, prior statin therapy, collagen diseases, advanced liver disease, renal failure, malignancy, any infectious disease. The study protocol was approved by the institutional Ethical

Committee and written informed consent was obtained from all patients.

All patients were monitored continuously for new onset arrhythmias throughout their hospitalization period and all arrhythmias were recorded. The appearance of reperfusion arrhythmias during thrombolytic therapy or primary angioplasty were not accepted as new onset arrhythmia. New onset arrhythmias were assigned to the following categories: atrial arrhythmias (atrial fibrillation/flutter), ventricular arrhythmias (ventricular tachycardia VT, sustained or nonsustained, ventricular fibrillation VF) or conduction abnormalities (AV blocks 1º, 2 º and complete AV blocks, left anterior or posterior fascicular blocks, left or right bundle branch blocks BBB, and sinus bradycardia with heart rate < 50 / min).

Serum CRP and potassium levels were measured within the first 12 hours and upon admission. The CRP levels were measured in plasma samples by a sensitive nephelometric method, whereby a value from 0–5 mg/dl is regarded as a normal value. Potassium concentration (mEq/L) was measured in venous blood samples in all patients on admission. The normal range of potassium was defined 3.50 to 5.50 mEq/L. Venous blood samples were obtained on admission and every 6 hours until the peak CKMB level was determined.

Left ventricular ejection fraction (LVEF) and left atrial diameter were measured in all patients by two-dimensional echocardiography. Two dimensional echocardiographic examinations were performed with a Vingmed cardiac ultrasound unit using a 2.5 MHz transducer in 3 (2–4) days after AMI and the results were assessed by two cardiologists. LVEF was estimated using Simpson's method[19].

Statistical analysis

The statistical analyses were performed using the Statistical Package for Social Sciences software (SPSS Inc. Chicago, Illinois, USA). For continuous variables, the data are reported as mean±SD if it shows normal distribution and median (interquartile range) if it is skewed distribution. For the comparison of group means Student's *t*- test and Mann Whitney nonparametric test were used. Group frequencies were compared by chi-square test. Spearman correlation analysis was carried out to investigate the correlation between CKMB levels and LVEF. The cutoff points for CRP level were determined using receiver-operating characteristics (ROC). Logistic regression was performed to determine the most common predictors of arrhythmias. Two-tailed p values of less than 0.05 was considered as statistically significant.

RESULTS

In group 1; ventricular arrhythmias developed in 19 patients (VF in 8 patients, VT in 10 patients, both VT and VF in 3 patients), atrial arrhythmias in 7 patients (AF in 7 patients) and conduction abnormalities in 16 patients (AV blocks in 10 patients [complete AV block in 9, Mobitz type 2 block in 1 patient], RBBB in 3 patients and sinus bradycardia in 3 patients). Baseline clinical and demographic characteristics are shown in Table 1. There is no difference between the groups in terms of baseline clinical parameters except the location of AMI. Fifty-three patients had anterior and thirty-seven patients had inferior wall AMI. Arrhythmias developed in 60% of patients with inferior wall AMI and 38% of patients with anterior wall AMI (P=0.049). Compared to patients with an anterior wall AMI, conduction abnormalities were more common in patients with an inferior wall AMI (32% vs 8%, P<0.05).

Twenty-nine patients were treated by primary angioplasty and forty-four patients had thrombolytic therapy. There is no difference between two groups in terms of reperfusion strategy (P=0.252). Revascularization therapy was not performed in seventeen patients; they received medical therapy alone. Oral acetylsalicylic acid, intravenous nitroglycerin, beta-blocker, angiotensin converting enzyme inhibitor and statin were administered to patients without contraindications in each group. Antiarrhythmic drugs except beta-blocker were given 10 patients (amiodaron in 8 patients and lidocain in 2 patients). D&C cardioversion was applied in 14 patients, temporary pacemaker was implanted in 8 patients. No permanent pacemaker and implantable cardioverter defibrillator (ICD) were implanted in any of the patients. During hospitalization, pericarditis developed in one patient (in group II), two patients died due to VF and another two died due to non-arrhythmic causes. Potassium chloride infusions were given to patients with a potassium level less than 3.5 mEq/l.

Median baseline CRP levels were higher in group 1 compared to group 2 (36.6(21.8-77) mg/dl vs.

21.8(24.2-30.7) mg/dl, P=0.002) (Table 2). Figure 2 shows the receiver operating characteristic (ROC) analysis for the determination of CRP cutoff level as a predictor of arrhythmia. The cutoff point was determined as 26.35 mg /dl by ROC analysis (sensitivity: 63%, specificity: 65%, P=0.002). CRP levels >26.35 mg/dl were associated with higher risk of arrhythmia (odds ratio: 3.0, 95% CI: 1.2–7.3, P= 0.015).

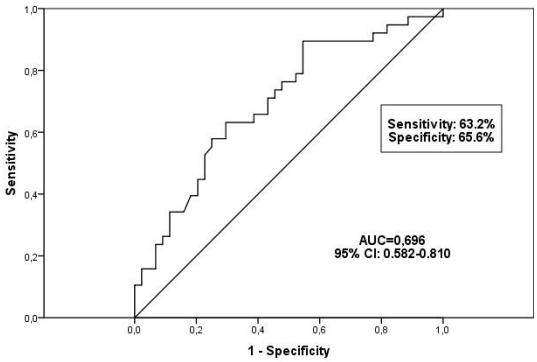

Figure 2. Receiver operating curves of CRP for prediction of arrhythmia

Mean baseline K^+ levels were similar in two groups (3.95 ± 0.61 mEq/l and 4.15±0.43 mEq/l, P=0.10, respectively) (Table 2). In group 1, potassium levels were significantly lower in patients developing ventricular arrhythmias compared to patients developing other types of arrhythmias (3.7mEq/l vs. 4.1 mEq/l, P=0.04) (Table 3).

Twelve out of 90 patients had hypokalemia (serum potassium <3.50 mEq/l). The rate of hypokalemia was 26% among arrhythmic patients (P=0.001). Ventricular arrhythmias were the most common type of arrhythmia in hypokalemic patients. Severe hyperkalemia (serum potassium >5.5 mEq/l) did not observe.

LVEF did not associate with arrhythmia. LVEF were similar in two groups (47±9% in group 1, 46±9% in group 2, P=0.548). But, median CKMB levels were higher in group 1 (301(155-561)IU/l, 206(105-377) IU/l, respectively, P=0.018) (Table 2). Whereas, a significant negative corellation was found between CKMB levels and LVEF (rho = -0.292, P=0.007).

Table 1. Baseline clinical characteristics

	Group 1 (n=42)	Group 2 (n=48)	*P* value
Age (years)	57 ±12	55 ±11	0.501
Male sex (n,%)	35 (83%)	39 (81%)	0.796
Diabetes mellitus (n,%)	10 (24%)	10 (21%)	0.735
Hypertension (n,%)	14 (33%)	10 (21%)	0.181
Family history of CAD (n,%)	13 (31%)	15 (31%)	0.976
Smoking (n,%)	27 (64%)	30(62%)	0.861
Body mass index (kg/m^2)*	25.9(23.9-28.1)	28(24.2-30.7)	0.111
Systolic blood pressure (mmHg)	110±12	112±12	0.882
Killip class II (n,%)	21(50)	18(38)	0.233
Heart rate	79 ±18	75 ±12	0.242
Inferior location of AMI	22 (52%)	15 (31%)	0.042
Revascularization	35 (83%)	38 (79%)	0.252
Reperfusion arrhythmia	19 (45%)	13 (27%)	0.073

Values are mean±SD, median (interquartile range)*; CAD: coronary artery disease; AMI: Acute myocardial infarction

The subgroup analysis of the comparisons between the group 1 and 2 in terms of atrial arrhythmias, ventricular arrhythmias, and conduction disturbance were performed and shown in Table 3. In subgroup analysis, CRP levels was significantly higher only in ventricular arrhythmia and atrial arrhythmia subgroups (P=0.006, P= 0.003, respectively). Higher CKMB levels and lower potassium levels were found significantly in the ventricular arrhythmia subgroup (P=0.001, P=0.004, respectivelly). Also localization of AMI was significantly differ in the conduction abnormalities subgroup, in which related with inferior AMI (P=0.003).

Multivariate logistic regression analysis showed a baseline CRP level >26.35 mg/dl (P=0.018, OR:4.0, 95% CI: 1.2-12.90.7), peak CKMB level (P=0.009, OR: 1.00, 95% CI: 1.00-1.00) and inferior localization of AMI (P=0.025, OR:4.9, 95% CI:1.2-20.4) were significantly associated with arrhythmia development during in-hospital phase of AMI regardless of types of arrhythmia (Table 4).

Table 2. Laboratory and echocardiographic findings

	Group 1 (n=42)	Group 2 (n=48)	P value
C-reactive protein (mg/dl)*	36.6(21.8-77)	21.8(8.5-34.2)	0.002
Hypokalemia (n, %)	11(26%)	1(2%)	0.001
Potassium levels (mEq/l)	3,95 ± 0,61	4,15 ± 0,43	0.086
Creatinine kinase- MB (IU/l)*	301(155-561)	206(105-377)	0.018
Ejection fraction (%)	47 ± 9	46 ± 8	0.548
IVS (mm)*	12(10-13)	12(10-14)	0.391
LA diameter (mm)	38±4	36±6	0.236

Values are mean±SD, median (interquartile range)*; IVS: interventricular septum; LA: Left atrium; Hypokalemia: serum potassium < 3.50 mEq/l.

Table 3. Subgroup analysis

Variables	Ventricular arrhythmias (n=19)	Atrial arrhythmias (n=7)	Conduction disturbances (n=16)	No Arrhythmia (n=48)
CRP (mg/dl)	42.4 (23.4-69.3) ‡	120 (45.4-189.5) ¥	23 (17.2-52)	21.8 (8.5-34.2)
K^+ (mEq/l)	3.7±0.6 +	4.3±0.5	4.0±0.5	4,1 ± 0,4
CKMB (IU/l)	462(274-750) *	184(140-256)	299(142-453)	206(105-377)
LVEF	47±10	44±11	48±11	46±8
Inferior AMI (n,%)	9(47)	1(14)	12(75) ¥	15 (31%)

‡ :P=0.006 ; ¥: P= 0.003 (Fischer's exact test); *:P=0.001; + :P=0.004; Values are mean±SD, median (interquartile range); CRP: C-reactive protein; K^+: Potassium ; CKMB: Creatine kinase MB; LVEF: Left ventricular ejection fraction.

Table 4. The Predictors of all arrhythmias in logistic regression analysis

Variables	odds ratios, (95% CI)	P value
Male sex	1.6(0.3-7.0)	0.504
Age	1.02(0.9-1.0)	0.271
LVEF	1.01(0,9-1.0)	0.859
Hypokalemia	7.6(0.6-89.2)	0.103
Creatinine kinase MB	1.00(1.00-1.00)	0.009
CRP >26.35 mg/dl	4.0 (1.2-12.9)	0.018
Inferior infarct localization	4.9(1.2-20.4)	0.025

LVEF: Left ventricular ejection fraction; CRP : C-reactive protein; Hypokalemia: serum potassium < 3.50 mEq/l

DISCUSSION

In our study, we examined the association between CRP, potassium, cardiac enzyme plasma concentrations and clinical charesteristics and arrhythmias to determine common predictors of all arrhythmias in patients with STEMI. In our study, we found that elevated CRP levels, peak CKMB levels, hypokalemia and inferior localization of AMI were associated with new onset arrhythmia in patients with STEMI. Also, in logistic regression analysis, higher CRP levels (>26.35mg/dl), peak CKMB levels and inferior localization of AMI were found to be predictors of new-onset arrhythmias in patients with AMI regardless of type of arrhythmia. In addition, according to subgroup analysis, ventricular arrhythmias were related with CRP, CKMB and potassium levels, atrial arrhythmias (atrial fibrillation) were related with only CRP level and conduction disturbances were related with only localization of AMI. Inflammation were found to be associated with both atrial and ventricular arrhythmias. The association of potassium levels, infarct location and infarct size with arrhythmia was already known in patients with AMI. The remarkable finding of our study was the relationship between CRP level and new onset arrhythmias.

Arrhythmias and conduction disturbances may occur in the setting of AMI. Sustained ventricular tachyarrhythmias may be associated with sudden cardiac death in the early phase of AMI[3]. AF seen during AMI is reported to be associated with longer

duration of hospitalization, higher in-hospital and long-term mortality and an increased incidence of thromboembolic events compared with patients without AF[20]. Among conduction arrhythmias, high-degree atrioventricular block is associated with short- and long-term mortality an AMI[18]. Although the exact pathogenetic mechanisms of arrhythmias in the setting of AMI have not been established yet, it has been reported complex mechanisms including adrenergic nervous system tone, ischemia and myocardial necrosis, inflammation, hypokalemia, hypomagnesemia, intracellular hypercalcemia, acidosis, free fatty acid production from lipolysis, and increased free radical production from reperfused ischemic myocardium[5,6,10].

New onset AF is one of the most common supraventricular arrhythmia type in the setting of AMI, with a prevalence ranging from 7 to 20%[20,21]. The relationship between arrhythmias and inflammation has been shown in AF patients without AMI[12-15]. Aviles et al. reported that CRP is not only associated with the presence of AF but, may also predict patients at increased risk for future development of AF[12]. In a couple of studies, the association between inflammation and AF have been demonstrated in AMI patients. Gedikli et al. have showed that elevated CRP levels were related to risk of developing AF in patients with anterior location AMI[10]. Also, they were found to be age and CRP levels independent predictors of AF in patients with acute anterior AMI. Aronson et al. have reported that the positive association between increased CRP and new-onset AF in AMI patients[11]. Parashar et al. had found that for every two fold increase in hs-CRP, there was a 15% increase in the rate of AF in patients with AMI[22]. In our study we found that elevated CRP levels were associated with new-onset AF in patients with STEMI. Our findings are compatible with the studies mentioned above. Although the significance of CRP elevation for the pathogenesis of AF remains unclear, inflammation may contribute to structural and /or electrical remodelling in the atria[14].

Ventricular arrhythmias were the most common type of arrhythmias in our study. The reported incidence of VF varies from 2.1% to 12.4%, and that of VT ranges from 1% to 9.9%[23]. Although inflammation plays an important role in pathogenesis of AF, there is few information about the relationship between other arrhythmias and inflammation in AMI[10-15]. In our study, baseline CRP levels, peak CKMB levels and hypokalemia rate were significantly higher in patients with ventricular arrhythmia. Ventricular arrhythmias following AMI were being shown in related K^+ levels and left ventricular function[24-26]. Grade et al. determined the relationship between the ventricular arrhythmias and infarct size estimated on the basis of CKMB levels in patients with AMI[27]. Additionally, the larger infarct size is associated with the occurrence of sinus bradycardia, accelerated idioventricular rhythm and sustained VT but not VF[6]. Khairy et al. reported that VT in the post-AMI phase is associated with infarct extension, heart failure and increased mortality[28]. Although, we found that significantly negative correlation between CKMB and LVEF, LVEF did not differ in two group. In our study, we concluded that, both the magnitude of CRP response due to inflammation and larger infarct size measured by CKMB could be related to ventricular arrhythmias in addition to potassium levels.

Many studies have suggested that statins may have antiarrhythmic effects in patients with coronary artery disease. Statin therapy has been shown to be protective against both AF after cardiac[29] and non-cardiac surgery[30] and ventricular tachyarrhythmias[31,32]. These antiarrhythmic effects, at least in some part, may be explained with anti-inflammatory actions and antioxidant or cell membrane fatty acids - modulating activities of statins. The benefits of statin treatment, especially in patients with ventricular arrhythmias show that inflammation might play a role in the development of ventricular arrhythmias.

Potassium plays a critical role in cellular electrophysiology. Several studies of patients with acute myocardial infarction have shown an association between hypokalemia and cardiac arrhythmias[16,34]. The findings of our study about potassium were similar to those of previous studies. In our study, K^+ levels at the time of admission were only associated with the development of ventricular arrhythmias (Table 3). K^+ levels at admission had a significant relation with the presence of ventricular arrhythmias, but this relationship was not observed with supraventricular arrhythmias and AV blocks[16,17]. Although the rate of hypokalemia was significantly higher in group 1, hypokalemia did not find to be a predictor of arrhythmias in logistic regression analysis.

The overall incidence of complete atrioventricular block in AMI patients is reported to 2–13%, depending on the type and anatomical location of the AMI[18]. AV conduction disturbances are more

frequent in patients with inferior AMI than with anterior AMI[18,35]. We found that inferior AMI were significantly higher in group 1. In addition, the rate of conduction abnormalities was higher in patients with inferior AMI compared to anterior AMI. Biomarkers have been used increasingly to determine the severity of cardiac disorders, for instance; recent studies suggest that natriuretic peptide biomarker screening may helpful the prevent HF[36,37]. We conclude that assays for CRP levels can be used to detect patients at high risk for arrhythmic events after STEMI. Thus, these high-risk patients may be more carefully monitoring.

The present study was conducted with a relatively small number of patients. An important limitation of our study was the investigating all type arrhythmias by coronary care unit monitoring system and electrocardiography. Non sustained VT and short-term AF are likely to be missed. Ideally, patients should be investigated by Holter monitoring system. In our study, we did not measure other inflammation markers such as cytokines, etc. measurement of other inflammation markers could have increaseed the strength of our study. Apart from measurement on admission, CRP levels were not consecutively measured throughout in-hospital period, thus the peak serum CRP level was not determined.

In our study we found that higher levels of CRP, peak CKMB levels and inferior localization of the infarct have predictive values for all the arrhythmic events during AMI. Also, CRP levels significantly increased in patients with atrial fibrillation and ventricular arrhythmias. We conclude that assays for CRP levels can be used to detect patients at high risk for arrhythmic events after STEMI. We think that predicting the patients with high risk of arrhythmia by laboratory tests (i.e CKMB and CRP) contributes to the literature. Thus, these patients can be monitored more carefully for arrhythmia development. Large prospective studies are required to establish the association between these parameters and all new-onset arrhythmias during AMI.

Yazar Katkıları: Çalışma konsepti/Tasarımı: İD; Veri toplama: İD, MBT; Veri analizi ve yorumlama: İD; Yazı taslağı: İD; İçeriğin eleştirel incelenmesi: MBT; Son onay ve sorumluluk: İD, MBT; Teknik ve malzeme desteği: İD; Süpervizyon: MBT; Fon sağlama (mevcut ise): yok.
Bilgilendirilmiş Onam: Katılımcılardan yazılı onam alınmıştır.
Hakem Değerlendirmesi: Dış bağımsız.
Çıkar Çatışması: Yazarlar çıkar çatışması beyan etmemişlerdir.
Finansal Destek: Yazarlar finansal destek beyan etmemişlerdir.
Teşekkür: Yazarlar Serkan Yüksel, Dr. Mahmut Şahin, Dr.Osman Yeşildağ, Dr.Okan Gülel and Dr. Korhan Soylu'ya katkılarından ndolayı teşekkür eder.

Author Contributions: Concept/Design : İD; Data acquisition: İD,MBT; Data analysis and interpretation: İD; Drafting manuscript: İD; Critical revision of manuscript: MBT; Final approval and accountability: İD,MBT; Technical or material support:; İD; Supervision: MBT; Securing funding (if available): n/a.
Informed Consent: Written consent was obtained from the participants.
Peer-review: Externally peer-reviewed.
Conflict of Interest: Authors declared no conflict of interest.
Financial Disclosure: Authors declared no financial support
Acknowledgement: Authors thank Serkan Yüksel, Dr. Mahmut Şahin, Dr.Osman Yeşildağ, Dr.Okan Gülel and Dr. Korhan Soylu who had the support in the preparation of this research.

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