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Role of C -reactive protein as an inflammatory marker in STEMI prognosis in patients receiving thrombolytic (a single center cohort study)

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ABSTRACT

Background: Proper prognostic biomarker is of a great importance for clinical decision-making in patients with acute myocardial infarction undergoing thrombolytic during admission. Although a lot of significant inflammatory biomarkers have emerged recently, the inflammatory mediator C-reactive protein still plays an important role in predicting adverse post-infarction complications.

Aims: to evaluate the role of C -reactive protein as an inflammatory marker in STEMI prognosis in patients receiving thrombolytic therapy

Patients and methods: This study enrolled around 100 consecutive patients in cohort study design. Those patients with acute myocardial infarction received thrombolytic therapy and have reason not to perform primary Percutaneous Coronary Intervention without non-cardiovascular causes of inflammation correlated with admission levels of C -reactive protein (more than 5 mg/dl considered significant) during hospitalization. Data collection occurred from January to September 2024 at a single center Al Imamain Alkadhumain Medical City, Baghdad, Iraq. Clinical follow up and echocardiography for left ventricular function were performed.

Results: Hundred patients were included to illustrate the therapeutic response and thrombus resolution following actylase administration in STEMI patients, stratified by C -reactive protein status. Patients with normal C -reactive protein demonstrated significantly better thrombus (STE) resolution, with 62.5% achieving >70% resolution compared to 36.5% in the increased C -reactive protein group P=0.024. Similarly, a complete chest pain response to actylase therapy was more frequent in the normal C -reactive protein group 75.0% than in those with 48.1%. Ejection fraction post-thrombolysis was also significantly higher in the normal C -reactive protein group compared to the elevated C -reactive protein group suggesting a better preservation of left ventricular function in patients with lower inflammatory status. Ejection fraction post-thrombolysis was also significantly was higher in the normal C -reactive protein group compared to the elevated group suggesting a better preservation of left ventricular function in patients with lower inflammatory status.

Discussion: This study demonstrated that STEMI patients with normal C-reactive protein (CRP) levels had significantly better thrombus resolution in response to thrombolytic therapy than those with elevated CRP, supporting the notion that systemic inflammation impairs thrombolytic efficacy. Additionally, better preservation of left ventricular ejection fraction was observed in the normal CRP group, indicating less myocardial injury and improved cardiac function. These results align with prior investigations showing elevated CRP levels predict worse clinical outcomes after myocardial infarction, including increased thrombus burden and reduced reperfusion success. However, this study found no significant association between CRP levels and clinical variables such as Killip class, smoking, diabetes, or premedication use, differing from some reports that describe relationships between CRP and these factors, possibly due to demographic or methodological differences. The lack of correlation between CRP and post-thrombolysis ECG abnormalities further suggests that CRP's prognostic value is more related to infarct extent than to acute electrical changes

Conclusion: C reactive protein tends to be a valuable prognostic marker in STEMI patients as patients with normal C reactive protein level demonstrating significantly better thrombus (ST elevation) resolution and a better preservation of left ventricular function in patients as evidenced by ejection fraction.

KEYWORDS: prognostic biomarker, C -reactive protein, inflammatory biomarkers, canonical inflammatory mediator, STEMI prognosis, thrombolytic therapy.

Abbreviations

AMI: Acute Myocardial Infarction

CRP: C - reactive protein

DM: Diabetes Mellitus

ECG: Electrocardiogram

EF: Ejection Fraction

HF: Heart Failure

HTN: Hypertension

IHD: Ischemic Heart Disease

LV: Left Ventricular

MI: Myocardial Infarction

PCI: Percutaneous Coronary Intervention

STE: ST elevation

STEMI: ST elevation myocardial infarction

TIA: Transient Ischemic Attack.

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INTRODUCTION

C-reactive protein (CRP) is a substance produced by the liver in response to inflammation [1]. Its levels in the bloodstream can rise in response to conditions that cause inflammation, including infections, chronic inflammatory diseases, and acute injury [2]. Elevated CRP levels have been studied extensively in relation to cardiovascular diseases, including myocardial infarction (MI). CRP is commonly used by clinicians in acute bacterial diseases for both the detection of the inflammatory process and for the quantization of its intensity [3]. Certain CRP isoforms activate the complement pathway, induce phagocytosis, and promote apoptosis, while different isoforms promote the chemotaxis and recruitment of circulating leukocytes to areas of inflammation and can delay apoptosis [4]. MI is often a consequence of atherosclerosis, the process of plaque formation in the arterial walls. Inflammation plays a critical role in the development and rupture of these plaques. CRP is considered a marker of systemic inflammation and is associated with the presence of atherosclerosis. Numerous epidemiological studies have demonstrated that high-sensitivity CRP (hs-CRP) levels can predict the risk of future cardiovascular events, including myocardial infarction. Increased hs-CRP levels have been associated with a higher incidence of MI in both men and women, independent of traditional cardiovascular risk factors such as cholesterol levels, hypertension, and smoking [5]. Some studies have assessed whether reducing inflammation through pharmacological means (e.g., using statins or anti-inflammatory drugs) can lower the risk of myocardial infarction. Statins, which lower cholesterol and have anti-inflammatory effects, have been shown to reduce CRP levels and are associated with a decreased risk of MI [6]. CRP which displays CRP level changes over time, has been suggested as a very early and more sensitive parameter for more serious outcomes following STEMI [7]. A study demonstrates association between CRP level dynamics and adverse cardiovascular events and death after acute coronary syndromes has been suggested [8]. According to many studies, changes in CRP concentrations during STEMI might serve as a risk marker for post-infarct LV systolic dysfunction [9]. Also an elevated peak C-reactive protein (CRP) levels are associated with reduced LV ejection fraction (LVEF, more severe myocardial tissue injury), and worse outcome in the setting of acute myocardial infarction [10]. Whether measurement of CRP contributes clinically relevant incremental information over and above clinical risk scores and other biomarkers is still controversial, and studies have shown contradictory results [11-14]. The present study aims to evaluate the role of CRP as an inflammatory marker in STEMI prognosis in patients receiving thrombolytic therapy.

PATIENTS AND METHODS

Study Design

This single center cohort study enrolled 100 consecutive patients diagnosed with acute ST-segment elevation myocardial infarction receiving thrombolytic (alteplase 100 mg over 90 minutes) within 12 hr. of typical ischemic chest pain & having a reason not to perform primary PCI (because of lack of facilities, time factor, allergy to contrast or patient preference). Data collection occurred from January to September 2024 at the Coronary Care Unit of Al Imamain Alkadhumain Teaching Hospital, Baghdad. The study analyzed the correlation between admission and peak C-reactive protein (CRP) levels during hospitalization, focusing on CRP levels greater than 5 mg/dl (as lab machine consider above 5mg/dl as a positive result), when used for cardiac risk stratification, CRP levels are interpreted as follows:

Below 1 mg/dL: Low cardiovascular risk

Above 3 mg/dL: High cardiovascular risk [15]

Population:

Inclusion Criteria

- Adult patients aged 18 years and above
- Presented with acute STEMI within 12 hours of symptom onset
- Eligible and fit for thrombolytic therapy

Exclusion Criteria

- Patients with heart failure
- Contraindications to thrombolytic therapy

Patient Data Collection

Demographic data (age, sex), comorbidities including diabetes mellitus (DM), hypertension (HTN), ischemic heart disease (IHD), heart failure (HF), and history of stroke or transient ischemic attack (TIA) were collected using a structured questionnaire. Detailed drug history and social habits such as smoking and alcohol consumption were also documented. Duration and characteristics of chest pain were assessed

Clinical and Laboratory Evaluation

Vital signs & full clinical evaluation was performed, Killip classes was assessed in each patient, Killip classification helps categorize the severity of STEMI and heart failure and determines the risk of adverse outcomes. Following the administration of thrombolytic (Actylase), the outcomes regarding chest pain response were as follows: a complete response, partial response or lack of response

The Killip classification (a clinical evaluation of heart failure severity) is as follows: [16]
Killip class I: This class indicates no evidence of heart failure.
Killip class II: This class represents mild signs of heart failure, such as bibasal rales, a third heart sound (S3), and raised JVP.
Killip class III: This class denotes the presence of acute pulmonary edema.
Killip class IV: This class represents cardiogenic shock
A baseline electrocardiogram (ECG) was performed on all patients prior to thrombolytic administration prior to & after thrombolytic administration. Complete, partial or no STE resolution if STE reduction is by 70 %, 30-70 % or <30 % respectively Blood samples were collected to measure inflammatory markers, including CRP and cardiac biomarkers such as troponin levels. CRP levels were recorded both on admission and at peak during hospitalization to assess inflammatory response and predict clinical outcomes.

Echocardiographic Assessment

Echocardiography was performed on all patients using M-mode and Simpson's biplane method to assess left ventricular wall motion abnormalities and calculate ejection fraction (EF), providing an objective evaluation of myocardial injury and cardiac function

Statistical analysis

Continuous variables were expressed as means and standard deviations. Categorical variables were expressed as frequency and percentages. The Welch's t-test (for normally distributed variables) was performed to test the differences in means. The difference between categorical variables was investigated using either the $\chi 2$ test with Yates' correction or Fisher's exact test, depending on the context. A univariate logistic regression analysis was conducted to study the risk of no response or partial response to actylase therapy in patients with STEMI. A P value less than 0.05 was considered statistically significant. R software packages were used for data processing, visualization, and statistical analysis ("R version 4.5.2, R Foundation for Statistical Computing, Vienna, and Austria").

Ethical consideration

Ethical and scientific approval for the research was obtained from the Scientific Committee at the Department of cardiology, Board for Medical Specialization. Verbal consent was obtained from all patients before starting data collection and after explaining the details of the study and assuring confidentiality.

RESULTS

The demographic and past medical history characteristics of patients with STEMI, stratified by CRP status, are summarized in table (1). The mean age was higher in the increased CRP group compared to the normal CRP group (58.2 ± 10.8 vs. 53.6 ± 13.4 years), although the difference did not reach statistical significance P=0.067. Gender distribution, BMI, and smoking history were similar between groups, with no statistically significant differences P>0.05. Additionally, histories of alcoholism, diabetes, hypertension, ischemic heart disease (IHD), and stroke showed no significant differences between patients with normal and elevated CRP levels (all P>0.5). Overall, no baseline demographic or medical history variable demonstrated a statistically significant association with CRP status.

Table 1: Demographic and past-medical history in patients with STEMI stratified by the CRP status

Characteristic	Overall, N = 100[1]	Normal CRP, N = 48[1]	Increased CRP, N = 52[1]	P-value[2]
Age in years	56.0 ± 12.2	53.6 ± 13.4	58.2 ± 10.8	0.067
Gender				
Male	84 (84.0%)	41 (85.4%)	43 (82.7%)	0.7
Female	16 (16.0%)	7 (14.6%)	9 (17.3%)	
BMI (kg/m^2)	28.5 ± 4.1	27.9 ± 4.1	29.1 ± 4.0	0.2
History of exposure to smoking	70 (70.0%)	32 (66.7%)	38 (73.1%)	0.5
History of alcoholism	4 (4.0%)	2 (4.2%)	2 (3.8%)	>0.9
History of diabetes	35 (35.0%)	17 (35.4%)	18 (34.6%)	>0.9
Duration of diabetes (years)	10.4 ± 8.6	8.9 ± 7.7	12.0 ± 9.4	0.3
History of hypertension	48 (48.0%)	23 (47.9%)	25 (48.1%)	>0.9
History of IHD	15 (15.0%)	7 (14.6%)	8 (15.4%)	>0.9
History of stroke	1 (1.0%)	1 (2.1%)	0 (0.0%)	0.5

[1]Mean ± SD; n (%)
[2]Welch Two Sample t-test; Pearson's Chi-squared test; Fisher's exact test

In table (2), when comparing between CRP groups, there were no statistically significant differences in the usage rates of any drug category before STEMI, as all p-values exceeded the conventional significance threshold ($p > 0.05$).

Table 2: Description of drug history stratified by the CRP status

Characteristic	Overall, N = 100[1]	Normal CRP, N = 48[1]	Increased CRP, N = 52[1]	P-value[2]
Aspirin	15 (15.0%)	6 (12.5%)	9 (17.3%)	0.5
Statin	16 (16.0%)	6 (12.5%)	10 (19.2%)	0.4
Plavix	7 (7.0%)	3 (6.3%)	4 (7.7%)	>0.9
Others	36 (36.0%)	16 (33.3%)	20 (38.5%)	0.6

[1]Mean ± SD; n (%)
[2]Welch Two Sample t-test; Pearson's Chi-squared test; Fisher's exact test

Table (3) presents a comparison of vital signs and myocardial infarction (MI) characteristics at admission, stratified by CRP status. No statistically significant differences were observed between the normal and increased CRP groups regarding pulse rate categories P=0.8, systolic blood pressure (133.7 ± 23.6 mmHg vs. 132.3 ± 23.3 mmHg; P=0.5), or diastolic blood pressure (81.8 ± 15.2 mmHg vs. 82.9 ± 14.9 mmHg; P=0.5). Killip class distribution did not significantly differ P>0.9, with most patients in Class 1 across both groups. The mean duration of symptoms prior to admission was comparable (6.4 ± 11.2 hours in the normal CRP group vs. 5.6 ± 6.2 hours in the increased CRP group; P=0.6). History of angina, pain severity, presence of reciprocal changes, and ECG-detected site of injury showed no significant variation across CRP strata (all P>0.05). A marginal trend was noted in the presence of Q-waves prior to thrombolysis, which was more frequent in the increased CRP group (48.1% vs. 29.2%), although this did not reach statistical significance (P=0.053).

Table 3: description of vital signs, characteristics of MI at admission to the hospital

Characteristic	Overall, N = 100[1]	Normal CRP, N = 48[1]	Increased CRP, N = 52[1]	P-value[2]
Pulse rate at admission				
Less than 50 b/m	7 (7.0%)	3 (6.3%)	4 (7.7%)	0.8
50 -100 b/m	80 (80.0%)	40 (83.3%)	40 (76.9%)	
More than 100 b/m	13 (13.0%)	5 (10.4%)	8 (15.4%)	
SBP at admission	133.7 ± 23.6	135.2 ± 24.1	132.3 ± 23.3	0.5
DBP at admission	81.8 ± 15.2	80.6 ± 15.6	82.9 ± 14.9	0.5
KILLIP classification				
Class 1	80 (80.0%)	39 (81.3%)	41 (78.8%)	>0.9
Class 2	11 (11.0%)	5 (10.4%)	6 (11.5%)	
Class 3	8 (8.0%)	4 (8.3%)	4 (7.7%)	
Class 4	1 (1.0%)	0 (0.0%)	1 (1.9%)	
Duration of symptoms (hours)	6.0 ± 8.9	6.4 ± 11.2	5.6 ± 6.2	0.6
History of angina before MI	42 (42.0%)	21 (43.8%)	21 (40.4%)	0.7
Pain severity				
Mild	4 (4.0%)	1 (2.1%)	3 (5.8%)	0.6
Moderate	19 (19.0%)	8 (16.7%)	11 (21.2%)	
Severe	77 (77.0%)	39 (81.3%)	38 (73.1%)	
Reciprocal changes before actylase	59 (59.0%)	28 (58.3%)	31 (59.6%)	0.9

Characteristic	Overall, N = 100[1]	Normal CRP, N = 48[1]	Increased CRP, N = 52[1]	P-value[2]
Q-wave before actylase	39 (39.0%)	14 (29.2%)	25 (48.1%)	0.053
Site of injury on ECG				0.4
Inferior	38 (38.0%)	21 (43.8%)	17 (32.7%)	
Anteroseptal	22 (22.0%)	11 (22.9%)	11 (21.2%)	
Anterior	16 (16.0%)	9 (18.8%)	7 (13.5%)	
Anterolateral	16 (16.0%)	5 (10.4%)	11 (21.2%)	
Extensive	3 (3.0%)	0 (0.0%)	3 (5.8%)	
Anteroinferior	2 (2.0%)	1 (2.1%)	1 (1.9%)	
Septal	2 (2.0%)	1 (2.1%)	1 (1.9%)	
Lateral	1 (1.0%)	0 (0.0%)	1 (1.9%)	

[1]Mean ± SD; n (%)
[2]Welch Two Sample t-test; Pearson's Chi-squared test; Fisher's exact test

Table (4) illustrates the therapeutic response and thrombus resolution following actylase administration in STEMI patients, stratified by CRP status. Patients with normal CRP demonstrated significantly better thrombus resolution, with 62.5% achieving >70% resolution compared to 36.5% in the increased CRP group P=0.024. Similarly, a complete response to actylase therapy was more frequent in the normal CRP group 75.0% than in those with elevated CRP (48.1%), indicating a statistically significant difference in treatment efficacy P=0.005. Ejection fraction post-thrombolysis was also significantly higher in the normal CRP group 52.3 ± 6.8% compared to the elevated CRP group (46.7 ± 7.0%; P<0.001), suggesting a better preservation of left ventricular function in patients with lower inflammatory status. Other parameters, including the presence of Q-waves, reciprocal changes, and ectopic arrhythmias after therapy, did not differ significantly between the groups (all P>0.3).

Table 4: response to therapy and resolution of the thrombus after thrombolytic therapy in patients with STEMI

Characteristic	Overall, N = 100[1]	Normal CRP, N = 48[1]	Increased CRP, N = 52[1]	P-value[2]
% of STE resolution after thrombolytic				0.024
Resolution > 70%	49 (49.0%)	30 (62.5%)	19 (36.5%)	
Resolution 30% - 70%	27 (27.0%)	8 (16.7%)	19 (36.5%)	
Resolution <30%	24 (24.0%)	10 (20.8%)	14 (26.9%)	
Presence of Q-wave after actylase	19 (19.0%)	7 (14.6%)	12 (23.1%)	0.3
Reciprocal changes after actylase	9 (9.0%)	4 (8.3%)	5 (9.6%)	>0.9
Presence of ectopic arrhythmia	7 (7.0%)	4 (8.3%)	3 (5.8%)	0.7
Response after thrombolytic				
Complete response	61 (61.0%)	36 (75.0%)	25 (48.1%)	0.005*
Partial response	30 (30.0%)	7 (14.6%)	23 (44.2%)	
No response	9 (9.0%)	5 (10.4%)	4 (7.7%)	
Ejection fraction after thrombolytic	49.4 ± 7.4	52.3 ± 6.8	46.7 ± 7.0	<0.001

[1]Mean ± SD; n (%)
[2]Welch Two Sample t-test; Pearson's Chi-squared test; Fisher's exact test

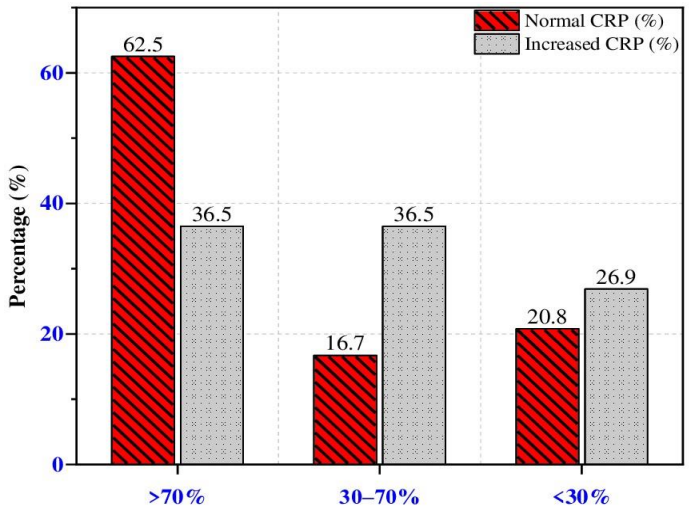

Figure 1: Distribution of STE resolution among STEMI patients receiving Actylase therapy, stratified by CRP level. Patients

with normal CRP more frequently achieved >70% resolution compared to those with elevated CRP (P=0.024)

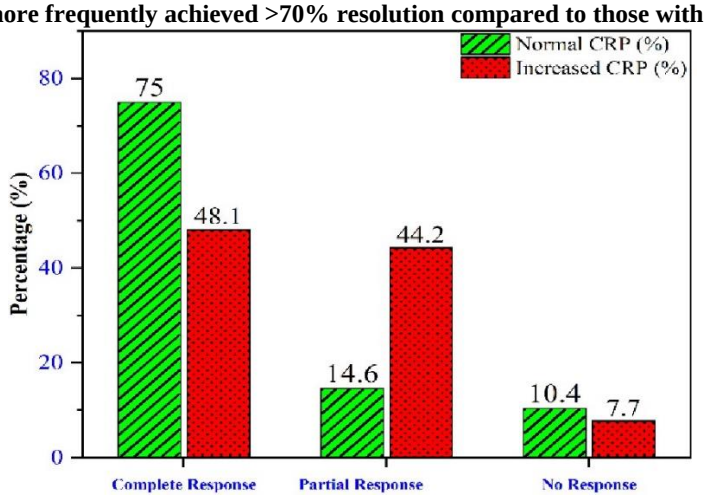

Figure 2: Clinical therapeutic response after thrombolytic, categorized by CRP status. Complete responses were more prevalent in the normal CRP group; partial responses dominated in the elevated CRP group (P=0.005)

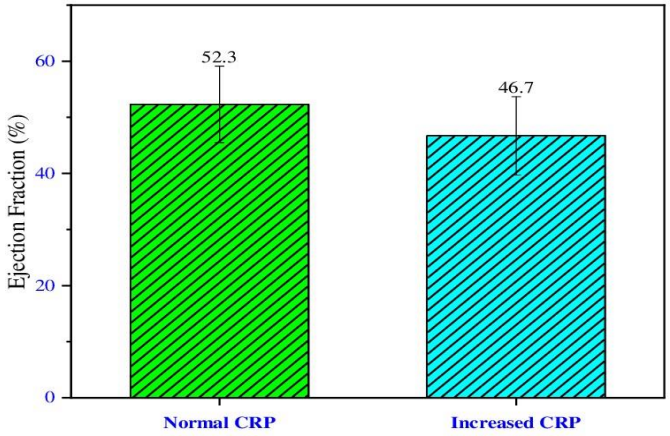

Figure 3: Comparison of mean ejection fraction after thrombolytic between CRP groups. Normal CRP patients showed significantly better LVEF than those with elevated CRP (P<0.001)

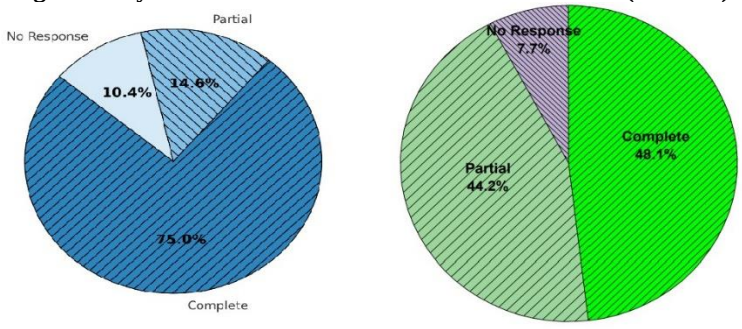

Figure 4: Therapeutic response to thrombolytic in STEMI patients stratified by CRP level. Patients with normal CRP (left) showed a higher rate of complete response (75.0%) than those with elevated CRP (right; 48.1%), indicating reduced thrombolytic efficacy in the presence of systemic inflammation

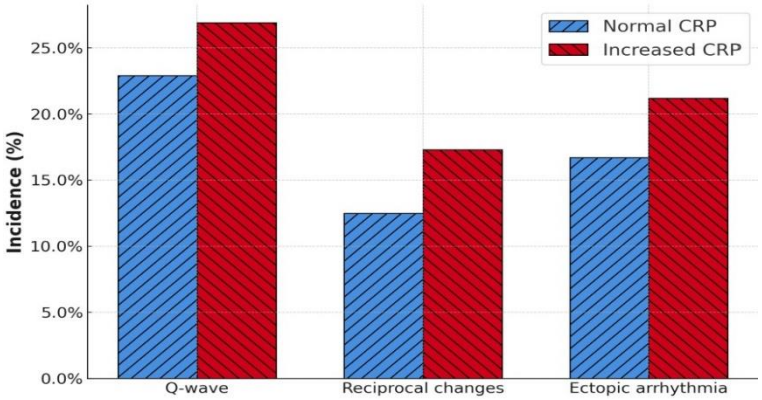

Figure 5: Bar chart comparing the incidence % of Q-waves, reciprocal changes, and ectopic arrhythmias after thrombolytic therapy in patients with normal and increased CRP. No statistically significant differences were observed between groups P>0.3 Figure (5) clarifies Post-Treatment ECG Abnormalities and other variables by CRP Status comparing post-thrombolysis incidence of three ECG findings-Q-waves, reciprocal changes, ectopic arrhythmias and other variable— between STEMI patients with normal versus elevated CRP levels. Although the rates of each abnormality were numerically higher in the increased CRP group (e.g., ectopic arrhythmias: 21.2% vs. 16.7%), none of the differences reached statistical significance (all P>0.3). These data suggest that CRP status may not strongly influence electrical recovery patterns post-thrombolysis.

DISCUSSION

In this study data demonstrates that STEMI patients with STEMI & normal CRP levels achieved significantly better STE resolution after thrombolytic therapy, with 62.5% achieving >70% resolution compared to 36.5% in the elevated CRP group (P=0.024). This finding suggests that elevated systemic inflammation, as indicated by higher CRP levels, may impair thrombolytic efficacy and thrombus resolution. This agrees with studies showing that baseline CRP can predict response to thrombolytic therapy and is associated with microvascular obstruction and thrombus burden, which can limit reperfusion success Krasniqi et al [17]. Similarly, a complete clinical response to thrombolytic therapy was more frequent in the normal CRP group 75.0% than in those with elevated CRP (48.1%), indicating a statistically significant difference in treatment efficacy (P=0.005), agrees with many studies showing elevated CRP levels serve as an early marker of inflammation and are closely associated with the extent of myocardial injury with a better outcome if CRP was not elevated Mitsis A et al., [18] & also aligns with meta-analyses showing that elevated CRP on admission predicts worse clinical outcomes post-thrombolysis or PCI, including no-reflow and mortality [19]. Also in this study, had been noticed Ejection fraction post-thrombolysis was significantly higher in the normal CRP group compared to the elevated CRP group, suggesting a better preservation of left ventricular function in patients with lower inflammatory status, In contrary a cross sectional study showed that high CRP levels are associated with greater EF in STEMI [20] while other studies [21,22] showed that overall increase in inflammation adversely affects left ventricular remodeling in myocardial infarction, which results in accelerated cell apoptosis after myocardial infarction Together, this leads to further damage to the cardiac muscle cells and impairs cardiac function & this shows CRP's utility as an inflammatory marker reflecting infarct severity and ventricular function prognosis. In spite of non-significant relation regarding Killip classification, current smoking and increasing age in our study, Holzknecht M et al., study showed coexistence related matter especially Killip classes 1 and 2 (p=0.001) and smoking & showed that CRP increase with increasing of age [21], variation in the results between current study & previous one can be attributed to patients number. The relationship between diabetes, CRP, and myocardial infarction may highlights the complex interplay between metabolic disorders and cardiovascular health, in our study no significant relation between dm state & CRP level was illustrated while there were contraditatory results in which some goes with our results [23] & others show positive proportional relation between CRP level & presence of dm [24-25], this might be correlated to timing of sampling after STEMI. Premedication before STEMI and is effect on CRP was not significant finding and statins use, antiplatelet was not having an impact on CRP in this study, although many facts showed in different studies contradictory results to ours & observed that high-dose statins, use of aspirin & clopidogrel markedly accelerated CRP declines post-ACS [26], possible explanation is that study enrolled single-center Iraqi patients& might differ in genetic, environmental, or clinical characteristics influencing inflammatory responses and drug effects, or timing of CRP measurement or medication adherence and dosing information which was lacking in our study. Localization of MI classified by ECG findings was not associated with significant relation to CRP levels, consistent with previous observations that systemic inflammatory markers such as CRP are more closely related to infarct extent rather than the anatomical location of infarction, while anterior MI is generally associated with larger infarcts and higher CRP in some reports, this association is not always consistently observed, possibly due to variability in infarct extent within each site and patient-specific inflammatory responses [18]. No statistically significant difference in the incidence of key post-thrombolysis ECG abnormalities-including Q-waves, reciprocal changes, and ectopic arrhythmias-between STEMI patients with normal versus elevated CRP levels, despite numerically higher rates in the elevated CRP group (P>0.3 for all comparisons). This suggests that systemic inflammation evidenced by CRP is a well-known marker of infarct severity g, it may not independently or strongly influence the acute electrical changes or arrhythmogenic substrate detectable by ECG after thrombolytic therapy & this agrees with other study in this context [27] showing that myocardial injury rather than inflammation may play a pathophysiological role in ventricular arrhythmia post MI & sudden cardiac death, this might be attributed to sample size, diversity in time of presentation, different population demographics or genetics.

CONCLUSION

C reactive protein tends to be a valuable prognostic marker in STEMI patients as patients with normal C reactive protein level demonstrate significantly better thrombus (STE) resolution and a better preservation of left ventricular function in patients as evidenced by ejection fraction. DM status didn't affect CRP values. Premedication with statins & antiplatelates were not associated with reduction in CRP levels. Electrical status of the heart was consistent across patients with acute STEMI & variable CRP.

Limitations: single center study with limited number of patients

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