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Measurement of C reactive protein concentration in myocardial ischemia and infarction

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Abstract:

Background: Myocardial ischemia and infarction represent significant health concerns. Inflammation plays a crucial role in the pathophysiology of these conditions. The elevation of CRP levels, an acute-phase protein, signifies the inflammatory status and has been associated with the severity and prognosis of myocardial damage. Measuring C-reactive protein (CRP) concentration in myocardial ischemia and infarction has garnered significant attention due to its potential role as a biomarker for cardiovascular risk stratification.

Patients and Methods: Subjects for the measurement of CRP concentration in myocardial ischemia and infarction include patients who present with symptoms of acute chest pain, such as angina pectoris or myocardial infarction. Blood samples are typically drawn on admission to the hospital and at various time intervals throughout hospitalization to measure CRP concentration. CRP concentration is one of several markers that are used to diagnose acute myocardial infarction, along with troponin levels, electrocardiography, and clinical symptoms Patients presenting with chest pain, suggestive of acute myocardial infarction or unstable angina. The study aimed to evaluate the diagnostic value of high-sensitivity crp (hs-crp) testing in this population, as well as the prognostic value of crp concentration in predicting adverse cardiovascular events. The study included 200 patients with chest pain and high sensitive crp concentration

Result: This cross-sectional study included 200 patients who suggestive of acute myocardial infarction or unstable angina. 53% of included patients was diagnosed with myocardial ischemia and infraction and 47% was diagnosed with angina. The median age of included patients was 55 years old. The patients with MI were older than patients with angina (p=0.005). 64% of included patients were male. There was statistically significant difference between both groups regarding age, sex and job occupations. There is marked elevation in crp level in old patient. the present study observed elevated levels of CKMB in patients experiencing acute MI compared to those with chronic or subacute MI

Conclusion: In conclusion, this study provided compelling evidence supporting the utility of C-reactive protein (CRP) as a biomarker for myocardial ischemia and infarction. The findings demonstrate a significant elevation in CRP levels among patients with myocardial infarction compared to those with angina. This aligned with previous research underscoring the role of CRP as an indicator of inflammation intricately linked with atherosclerotic disease progression and adverse cardiovascular outcomes. this study underscored CRP's utility as a predictor and prognosticator of myocardial infarction, independently or in conjunction with other biomarkers. It substantiated the role of inflammatory processes in MI pathogenesis and the need to incorporate CRP evaluation in the diagnosis and risk stratification of patients with suspected acute coronary events

Keywords: Coronary artery disease (CAD), risk factors, ischemic heart disease, high sensitive CRP, and Myocardial ischemia, Biomarker are the key focal points of the study.

1. Introduction

Myocardial infarction: The term "myocardial infarction" focuses on the myocardium (the heart muscle) and the changes that occur in it due to the sudden deprivation of circulating blood. The main change is necrosis (death) of myocardial tissue.

The word "infarction" comes from the Latin "infarcire" meaning "to plug up or cram." It refers to the clogging of the artery.

C-reactive protein (CRP) is an acute-phase protein produced by the liver in response to inflammation or tissue damage. It is a nonspecific marker of inflammation that can be measured in blood serum.

CRP levels increase rapidly in response to the onset of acute inflammation, and hence, this protein is used as a diagnostic marker in many inflammatory and infectious conditions. High-sensitivity CRP (hs-CRP) assays are now available that can detect CRP levels at much lower concentrations, which allows for more sensitive detection of inflammation and also helps in the prediction of future cardiovascular disease.

CRP levels are elevated in a number of conditions, including infections, inflammatory diseases such as rheumatoid arthritis, and in certain cancers. In addition, CRP has been found to be a predictor of cardiovascular disease, with levels of CRP being higher in individuals with atherosclerosis, hypertension, and hyperlipidemia.

Overall, CRP is an important tool in the diagnosis and monitoring of inflammatory conditions and chronic diseases, including cardiovascular disease.

Aim and objectives:

1. To determine the association of CRP concentration with the severity of myocardial ischemia and infarction.
2. To assess the diagnostic accuracy of CRP concentration in distinguishing between acute and chronic myocardial ischemia and infarction.

study design:

Retrospective and prospective descriptive study

site of study:

The study will conduct at Alabas hospital cardiology center, Taiz city Yemen.

study group:

The study included 200 patients with chest pain

Inclusions criteria:

We study The pt with stable Angina, unstable Angira, Acute M.I

Exclusion criteria

- Pregnancy: CRP levels will typically increase during pregnancy, especially in the third trimester, making it difficult to interpret results accurately.
- Acute injuries and surgeries: CRP levels may increase in response to acute injuries or surgical procedures, making it difficult to distinguish between inflammation caused by these factors and other inflammatory conditions.
- Chronic inflammatory diseases: Patients with chronic inflammatory diseases such as rheumatoid arthritis may have consistently elevated CRP levels, making it difficult to use CRP as a diagnostic marker for infections or other acute inflammatory conditions.

- Liver disease: Patients with liver disease may have elevated CRP levels due to impaired liver function, making it difficult to use CRP levels to detect inflammation caused by other factors.

Data collection: Data collected through structured questionnaire wich included demographic data (age,sex, marital status, and education) ,clinical examination, investigations include high sensitive crp , electrocardiography and echocardiogeaphy

Ethical considerations were paramount throughout the study, with informed consent obtained from all participating patients. Procedures and data collection were designed to avoid physical or emotional harm to the subjects. Data analysis involved the use of IBM SPSS software version 20.0, with qualitative data presented in percentages and numbers, and the normality of distribution verified using the Kolmogorov-Smirnov test. Quantitative data were described in terms of range, mean, standard deviation, and median, with significance determined at the 5% level. Various statistical tests were employed including the Chi-square test for categorical variables, the Mann Whitney test for abnormally distributed quantitative variables, and the two-sample T-test for comparing means of independent groups. Spearman's correlation was utilized for ranked data analysis, and univariate regression analysis examined the relationship between independent variables such as cognitive function tests and sleep disorders scale, and gender. R-squared values were used to assess the percentage of variance explained, while p-values indicated statistical significance, with values below 0.05 considered significant.

Ethical Statement: The present study runs in concordance with international ethical standards and applicable local regulatory guidelines. The study does not have any physical, psychological, social, legal, economic, or any other anticipated risks to study's participants. The study conserves participants' privacy. Investigators are responsible for keeping the security of the data. We also confirm that the participants' data were not used for any other purpose outside this study. Personal data (e.g., Name, contact information) were not entered in our data

entry software to conserve the participants' privacy, however, each subject got a unique identifier code.

Result:

This cross-sectional study included 200 patients who suggestive of acute myocardial infarction or unstable angina. 53% of included patients was diagnosed with myocardial ischemia and infraction and 47% was diagnosed with angina. The median age of included patients was 55 years old. The patients with MI were older than patients with angina (p=0.005). 64% of included patients were male. The most prominent jobs was worker and house wife. There were statistically significant difference between both groups regarding sex and job occupations. The socio-demographic parameters of included patients were further illustrated in table (1) and figures (1&2)

Table (1): Socio-demographic parameters of included patients

	Total (N=200)		Angina (N=94, 47%)		Myocardial ischemia and infraction (N=106, 53%)		P value
Age*	5.0	45- 62	50	43-60	58.5	46-65	**.005**
	No.	%	No.	%	No.	%	
SEX							
male	129	64.5%	50	53.2%	79	74.5%	**.002**
female	71	35.5%	44	46.8%	27	25.5%	
status MARRIED	200	100%	94	100%	106	100%	-------
Job	130	100.0%					
House wife	66	50.8%	44	64.7%	22	35.5%	**.000**
Captin	4	3.1%	0	0.0%	4	6.5%	
A judge	4	3.1%	0	.0%	4	6.5%	
Worker	34	26.2%	20	29.4%	14	22.6%	
Engineer	5	3.8%	1	1.5%	4	6.5%	
Driver	2	1.5%	2	2.9%	0	0.0%	
Farmer	5	3.8%	0	0.0%	5	8.1%	
Tailar	1	0.8%	0	0.0%	1	1.6%	
Teacher	7	5.4%	0	0.0%	7	11.3%	
Doctor	1	0.8%	0	0.0%	1	1.6%	
Police man	1	0.8%	1	1.5%	0	0.0%	

*represented as median and IQR

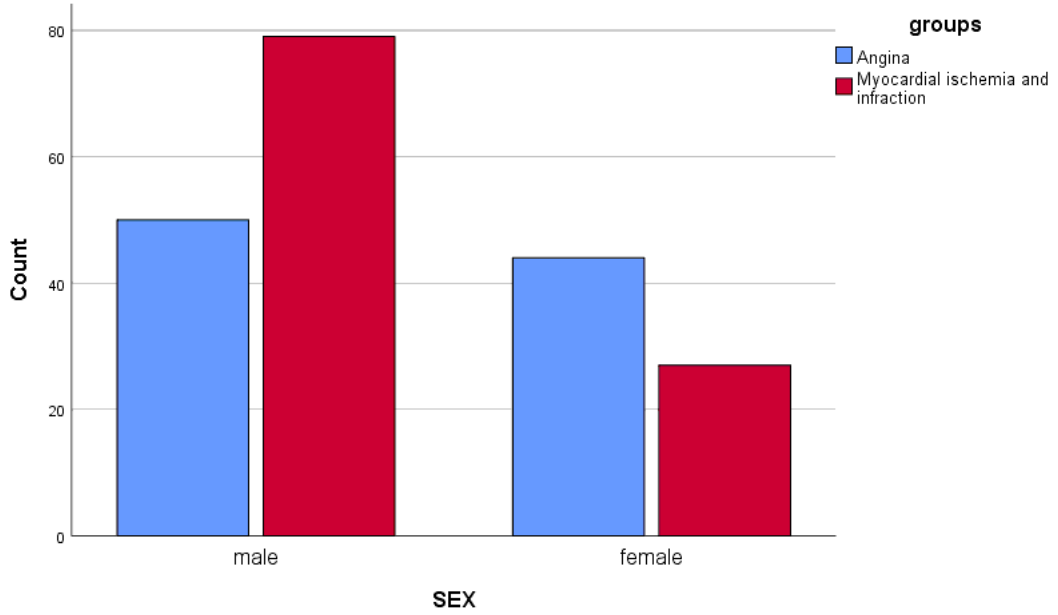

Figure (1): Gender of included patients in both groups

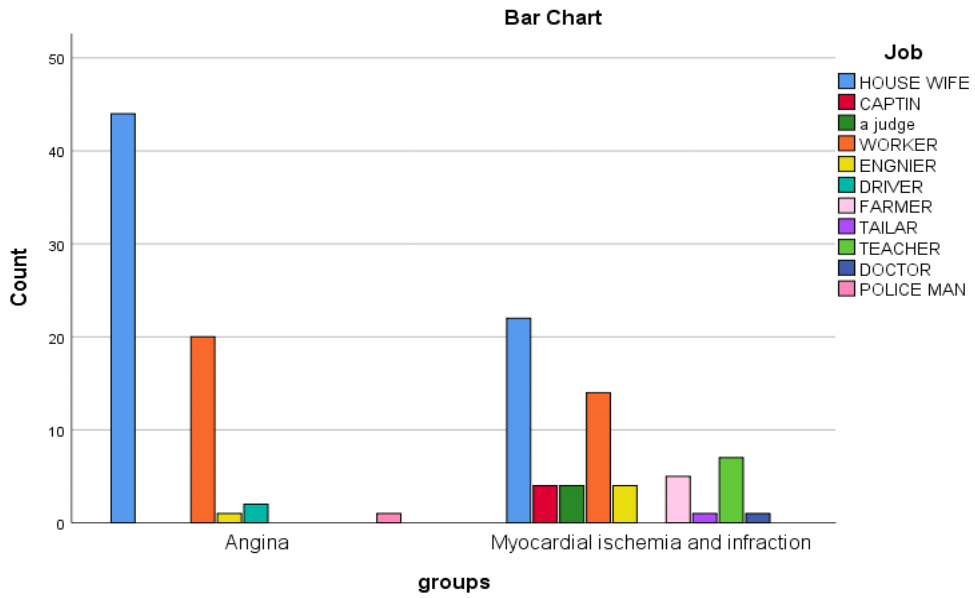

Figure (2): Jobs of included patients in both groups

Complains and its duration were illustrated in table (2). The median duration was 60 days. Duration was lower in patients with MI than patients with angina (P=0.00). While, SOB without chest pain was highly reported in patients with angina. There were statistically significant difference between both groups regarding complains and its duration.

Table (2): Complains and its duration

	Total (N=200)		Angina (N=94, 47%)		Myocardial ischemia and infraction (N=106, 53%)		P value
	median	IQR	median	IQR	median	IQR	
Duration of symptoms (days)	60.0	5.0-247.5	180	45, 365	7	2, 60	**.000**
Complains	No.	%					
Chest pain and SOB	29	14.5%	17	18.1%	12	11.3%	**0.17**
Chest pain	156	78.0%	62	66.0%	94	88.7%	**0.17**
SOB	15	7.5%	**15**	**16.0%**	0	0.0%	**0.001**

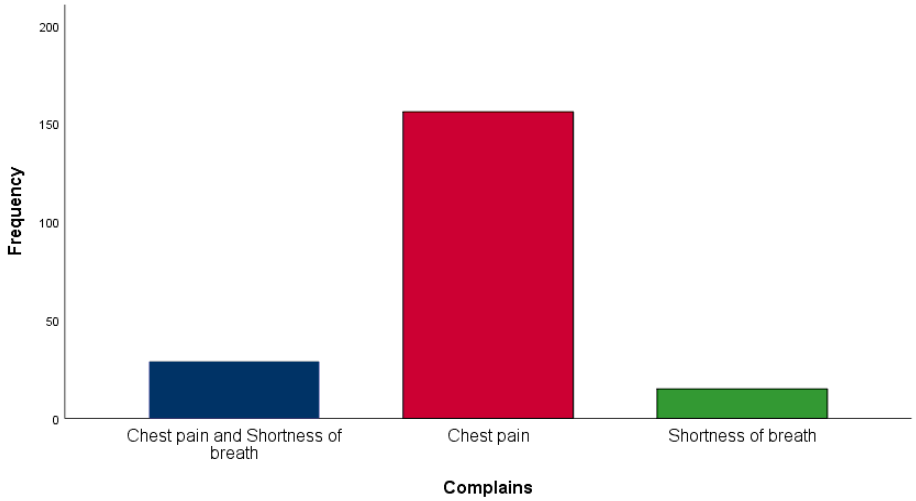

Figure (3): Complains of included patients

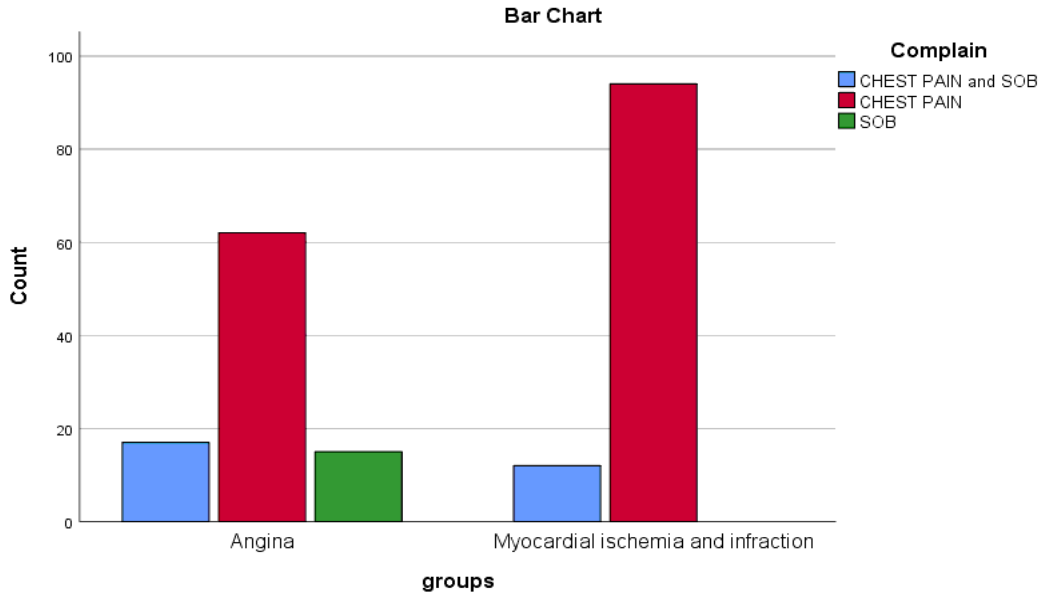

Figure (4): Complains of included patients in both groups

Table 3 shows echocardiography Findings, 23.5% had normal echocardiogram. Approximately, 59% had hypokinesia in various walls. There were statistically significant difference between both groups regarding Echocardiography Findings. The Echocardiography Findings were further illustrated in table (3) and figures (5)

Table (3): Echocardiography Findings

	Total (N=200)		Angina (N=94, 47%)		Myocardial ischemia and infraction (N=106, 53%)		P value
	N	%	N	%	N	%	
Normal	64	32.5%	44	47.8%	20	19.0%	**.000**
VHD	12	6.1%	2	2.2%	10	9.5%	**0.036**
Hypokinesia of septal and anterior wall	9	4.6%	0	0.0%	9	8.6%	0.003
Hypokinesia of anterolateral wall	5	2.5%	1	1.1%	4	3.8%	0.37
Hypokinesia of anterior wall	23	11.7%	9	9.8%	14	13.3%	0.5
Hypokinesia of inferior wall	35	17.8%	13	14.1%	22	21.0%	0.19
Hypokinesia of posterior wall	3	1.5%	0	0.0%	3	2.9%	0.24
Hypokinesia of inferiolatral wall	8	4.1%	3	3.3%	5	4.8%	0.72
Hypokinesia of septal wall	11	5.6%	4	4.3%	7	6.7%	0.54
Hypokinesia of apical wall	3	1.5%	3	3.3%	0	0.0%	0.12
ICMP	3	1.5%	3	3.3%	0	0.0%	0.12
Hypokinesia of posterior and inferior wall	7	3.6%	2	2.2%	5	4.8%	0.45
Hypokinesia of lateral wall	2	1.0%	1	1.1%	1	1.0%	0.99
Hypokinesia of inferiolatral and posterior walls	1	0.5%	0	0.0%	1	1.0%	0.98
Hypokinesia of distal 2/3 of septum and inferior wall	1	0.5%	0	0.0%	1	1.0%	0.98
Hypokinesia of all walls	3	1.5%	3	3.3%	0	0.0%	0.24
Hypokinesia of mid and basal and inferoseptal wall	1	0.5%	0	0.0%	1	1.0%	0.99
Hypokinesia of anteriorseptal wall	1	0.5%	0	0.0%	1	1.0%	0.98

Hypokinesia of anterior and anteroseptal wall	2	1.0%	1	1.1%	1	1.0%	0.99
VHAV	2	1.0%	2	2.2%	0	0.0%	0.22
Severe M.R	1	0.5%	1	1.1%	0	0.0%	0.99

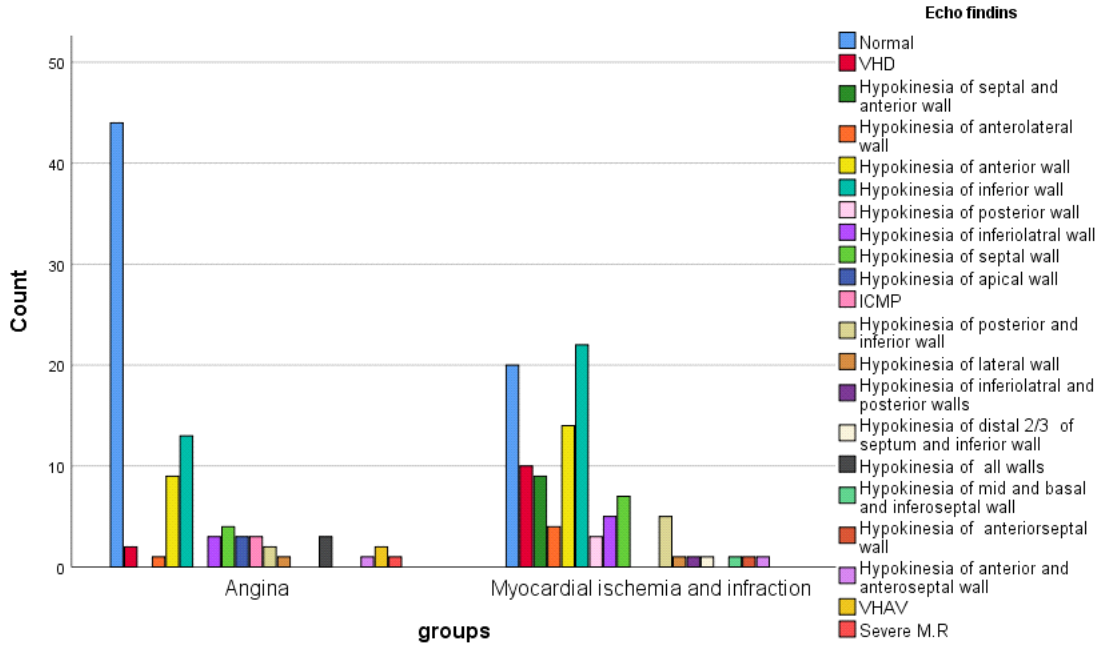

Figure (5): Echocardiography Findings of included patients in both groups

Considering electrocardiography Findings, 27.1% had normal ECG. Approximately, 64% had changes in ST (elevation or depression). . There were statistically significant difference between both groups regarding Electrocardiography Findings. The electrocardiography Findings were further illustrated in table (5) and figures (6&7).

Table (4): Electrocardiography findings

ECG	Total (N=200)		Angina (N=94, 47%)		Myocardial ischemia and infraction (N=106, 53%)		P value
	N	%	N	%	N	%	
Normal	54	27.1%	38	40.9%	16	15.1%	**.000**
ST elevetion in inferior leads	12	6.0%	0	0.0%	12	11.3%	**0.004**
T wave invention in inferior leads	45	22.6%	38	40.9%	7	6.6%	**0.00**
hypokinesia of postrior wall	2	1.0%	0	0.0%	2	1.9%	0.49
St elevation in anterior leads	32	16.1%	0	0.0%	32	30.2%	**0.00**
St elevation in inferiolatral lead	16	8.0%	0	0.0%	16	15.1%	**0.02**
LBBB	9	4.5%	8	8.6%	1	0.9%	**0.013**
AF	3	1.5%	3	3.2%	0	0.0%	0.246
St elevation in v2,3,4,5,6	5	2.5%	0	0.0%	5	4.7%	0.06
WBW syndrom, st elevation in v1,v2	3	1.5%	0	0.0%	3	2.8%	0.169
RBBB	3	1.5%	3	3.2%	0	0.0%	0.246
St depression in v3,4,5,6	3	1.5%	0	0.0%	3	2.8%	0.248
St depression in v2,3,4	4	2.0%	0	0.0%	4	3.8%	0.198
T wave invention in lead ii , iii	2	1.0%	1	1.1%	1	0.9%	0.99
St elevation in anterolateral	3	1.5%	0	0.0%	3	2.8%	0.169
DEPRESSED T wave in inferior lead	1	0.5%	1	1.1%	0	0.0%	0.98
St depression in inferior lead ii , iii	2	1.0%	1	1.1%	1	0.9%	0.99

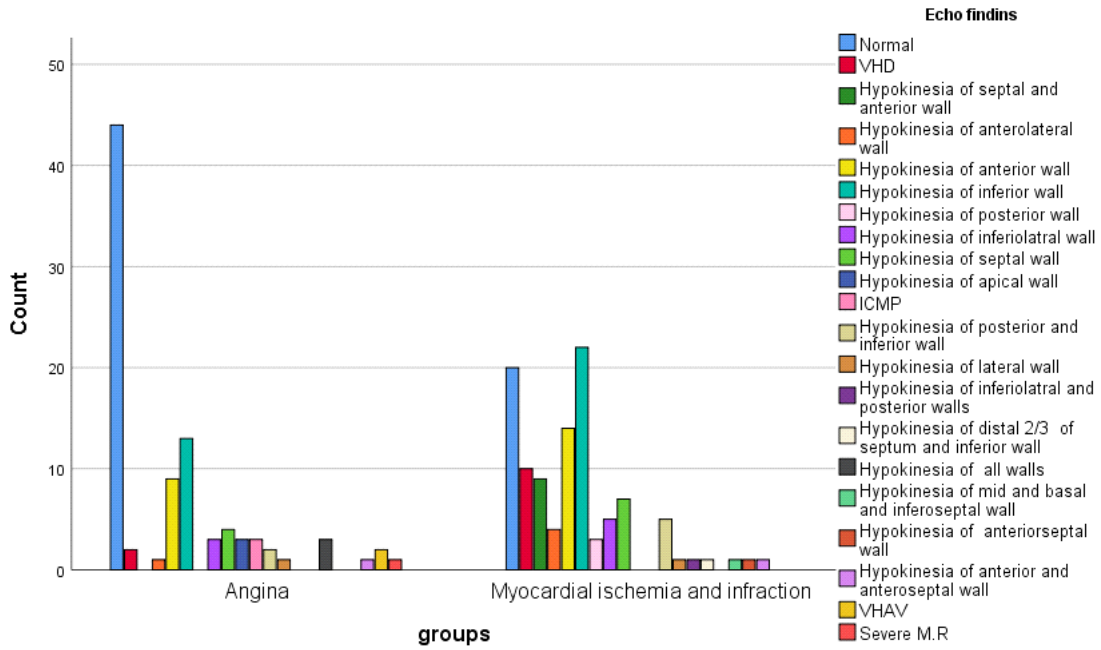

Figure (6): Electrocardiography findings of included patients in both groups

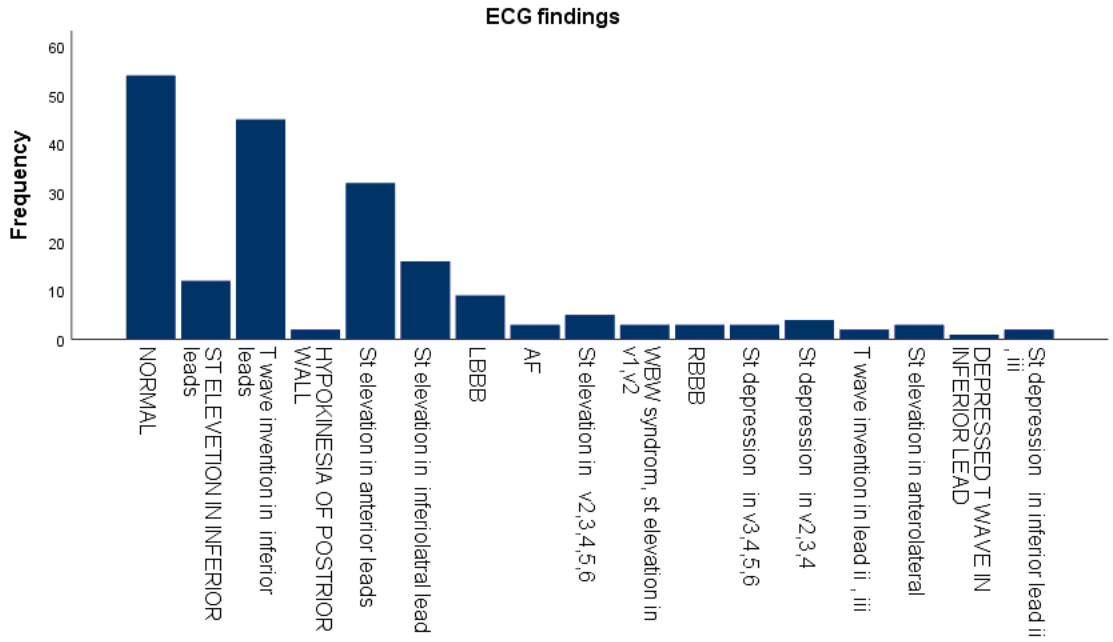

Figure (7): Electrocardiography findings of included patients

Tropinin, CK-MB and CRP were higher in patients with MI than patients with angina (P=0.00), (table 5and figures 8,9).

Table (5): laboratory signs

	Total (N=200)		Myocardial ischemia and infraction (N=106, 53%)		Angina (N=94, 47%)		Mann-Whitney U	P value
	median	IQR	median	IQR	median	IQR		
troponin	0.1	0.0, 5.8	5.445	0.635, 13.4	0.01	0.006, 0.04	732.5	**.000**
CKMB	4.6	2.2, 43.4	31.7	3, 78	3	2, 5	2391.5	**.000**
CRP	7.4	4, 22.7	9.7	5.4, 33.75	6	2.5, 11.1	3055.5	**.000**

Regarding vital signs and ejection fraction (EF), there were no statistically significant difference between both groups regarding BP, Pulse and Oxygen saturation. EF was lower in patients suffered from MI than in patients with angina (table 6& figure 10).

Table (6): vital signs and ejection fraction (EF)

	Total (N=200)		Myocardial ischemia and infraction (N=106, 53%)		Angina (N=94, 47%)		Mann-Whitney U	P value
	median	IQR	median	IQR	median	IQR		
Systolic BP	110	100-120	110	100, 120	108	100, 120	4303.5	.602
Diastolic BP	70.0	60-80	70	60, 87.5	70	60, 78.75	3861	.111
Pulse	84.0	74-93	81	73, 93	87	76, 93.25	3907	.144
Oxygen saturation	96.0	94.0-96	96	94, 97	95.5	92, 96	3889.5	.125
EF	54.5	44.0-65.0	51.5	43, 63.5	64.5	53, 73	803.5	.004

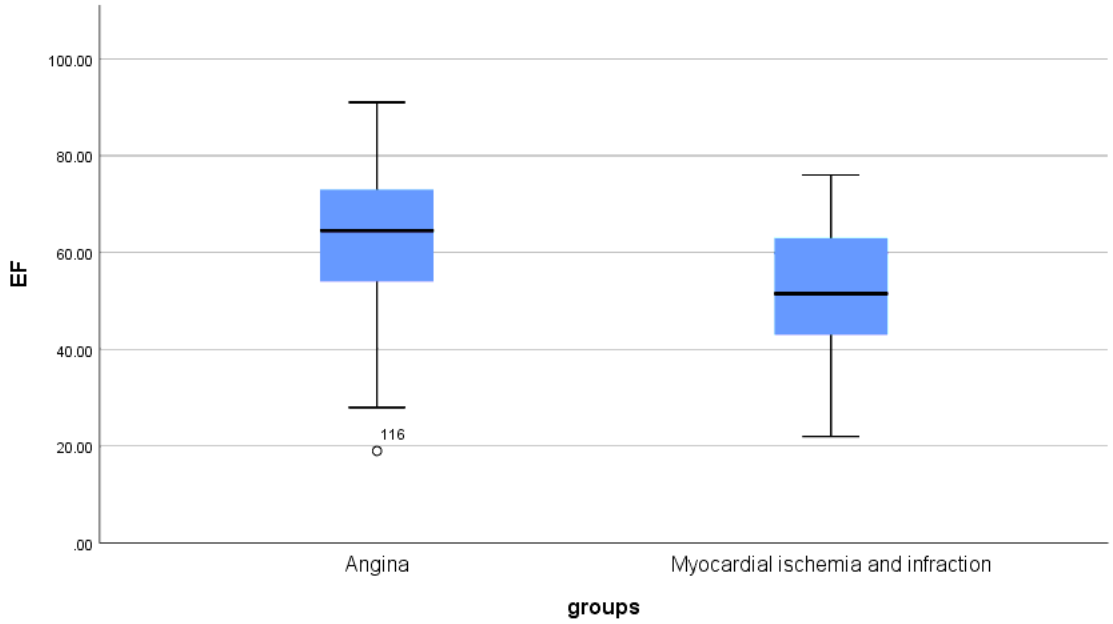

Figure (10): Boxplot for EF in both groups

Regarding MI classifications, the majority of included patients had acute MI. 23.6% had subacute MI.

Table (7): Classification of patients with MI according to duration of symptoms

	Frequency	Percent
Acute	73	68.9%
sub-acute	25	23.6%
chronic	8	7.5%

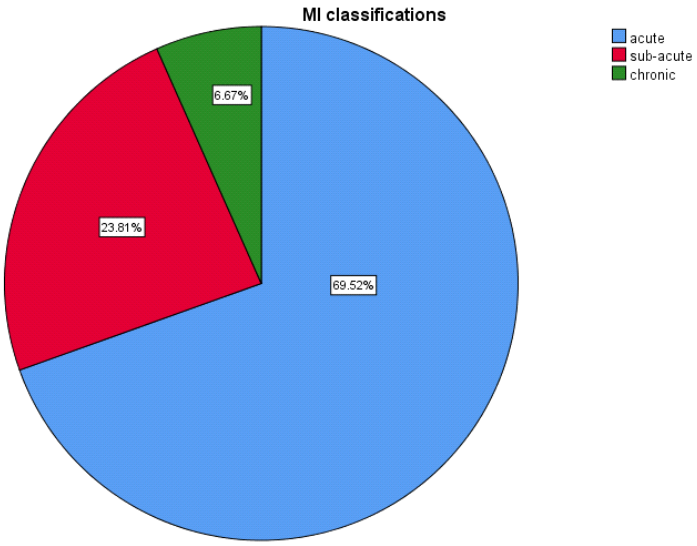

Figure (11): Pie chart for MI classifications

Regarding laboratory signs in patients with MI, CKMB was higher in patients with acute MI than patients with chronic or subacute MI (p=0.02). There were no statistically significant difference between stages of MI regarding troponin and CRP.

Table (8): laboratory signs in patients with MI

	Acute Myocardial ischemia and infraction (N=%)		Subacute Myocardial ischemia and infraction (N=%)		Chronic Myocardial ischemia and infraction (N=%)		Mann-Whitney U	P value
	median	IQR	median	IQR	median	IQR		
troponin	5.8	0.8, 13.0	2,	0.1, 13.2	8.1	0.1, 15.3	224.	.591
CKMB	43.5	6.0-83.5	4.0	2-31.7	4.0	4.0-17.5	121.5	**0.022**
CRP	9.7	5.1-31.0	8.6	5.9-26.1	68.0	2-85	173	.173

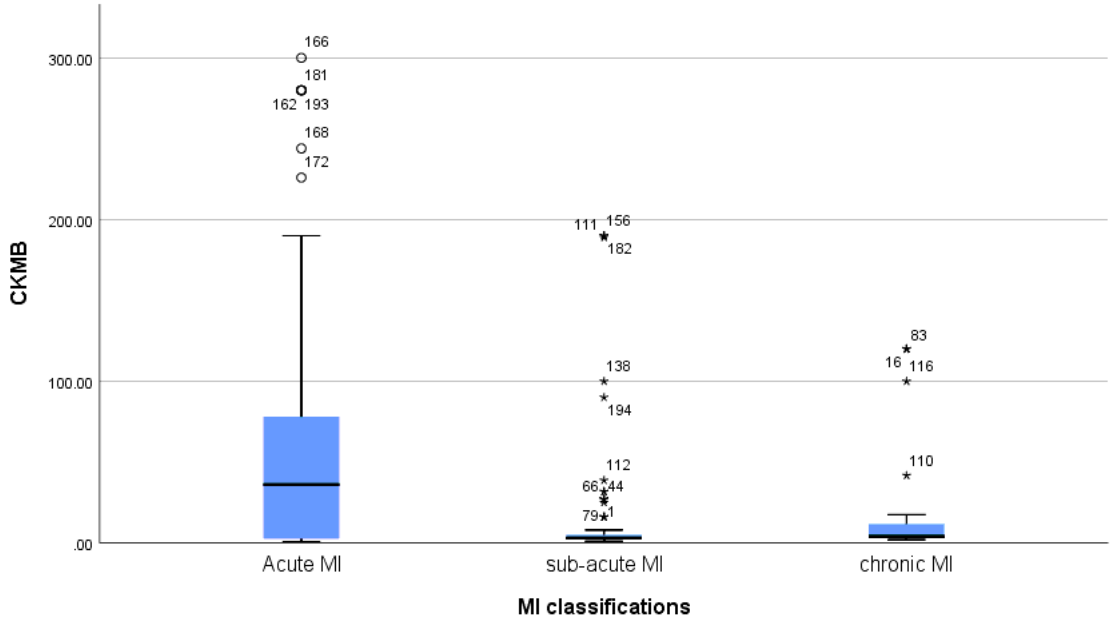

Figure (12): Boxplot for CK-MB in stages of MI

There were no statistically significant difference between stages of MI regarding vital signs and ejection fraction (EF) in patients with MI (P>0.05).

Table (9): vital signs and ejection fraction (EF) in patients with MI

	Acute Myocardial ischemia and infraction (N=%)		Subacute Myocardial ischemia and infraction (N=%)		Chronic Myocardial ischemia and infraction (N=%)		Mann-Whitney U	P value
	median	IQR	median	IQR	median	IQR		
EF	52	45, 63	43	43,64	56	36,62	131.5	.730
systolic	110	100-120	110-	90-160	120	100, 137.5	149.5	.276
diastolic	69	50-70	70	47-82	80	56-82	105.	.062
Pulse	84	67-93	76	74-92	74	73-81.3	159.5	.404
Oxygen	95	94, 97	96	94, 96,	98	93.3, 98.3	126.5	.130

There were a negative correlation between EF and MI. There were a positive

correlation between MI and CRP, troponin and CKMB.

Table (10): correlation between MI and various parameters

	r	P value
CRP	.316**	**.000**
troponin	.308**	**.000**
CKMB	.359**	**.000**
Systolic BP	.052	.474
Pulse	-.110	.130
Diastolic BP	-.121	.096
EF	-.262**	**.007**

Point biserial correlation or phi correlation

The decrease in ejection fraction one degree increase the incidence of MI by 0.98 with 95% confidence interval (0.927, 0.989). The increase in CRP one degree increase the incidence of MI by 1.043 with 95% confidence interval (1.021, 1.065). The increase in troponin one degree increase the incidence of MI by 1.99 with 95% confidence interval (1.49, 2.66). The increase in troponin one degree increase the incidence of MI by 1.023 with 95% confidence interval (1.02, 1.034).

Table (11): Prediction with MI

	Odds ratio	95% C.I	P value
EF	.958	.927, .989	**.009**
CRP	1.043	1.021, 1.065	**.000**
troponin	1.994	1.491, 2.666	**.000**
CKMB	1.023	1.012, 1.034	**.000**

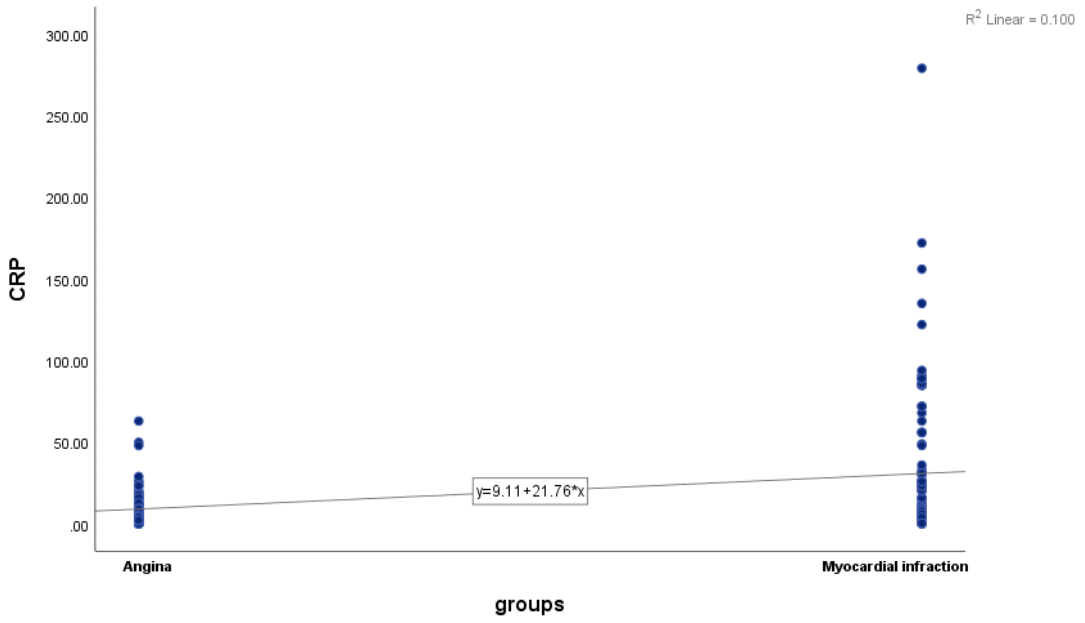

Figure (13): Scatter plot for the association between CRP and MI

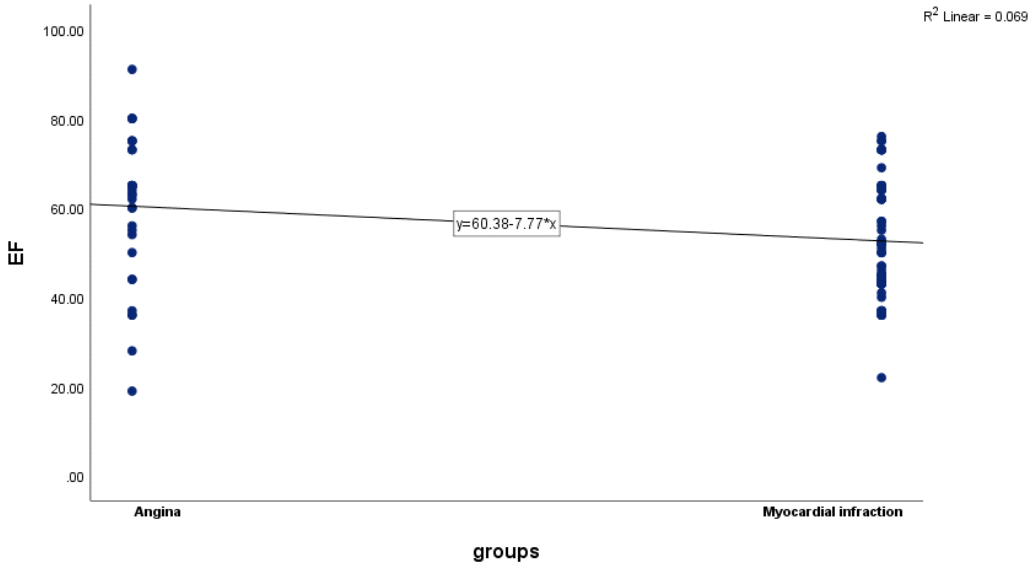

Figure (14): Scatter plot for the association between EF and MI

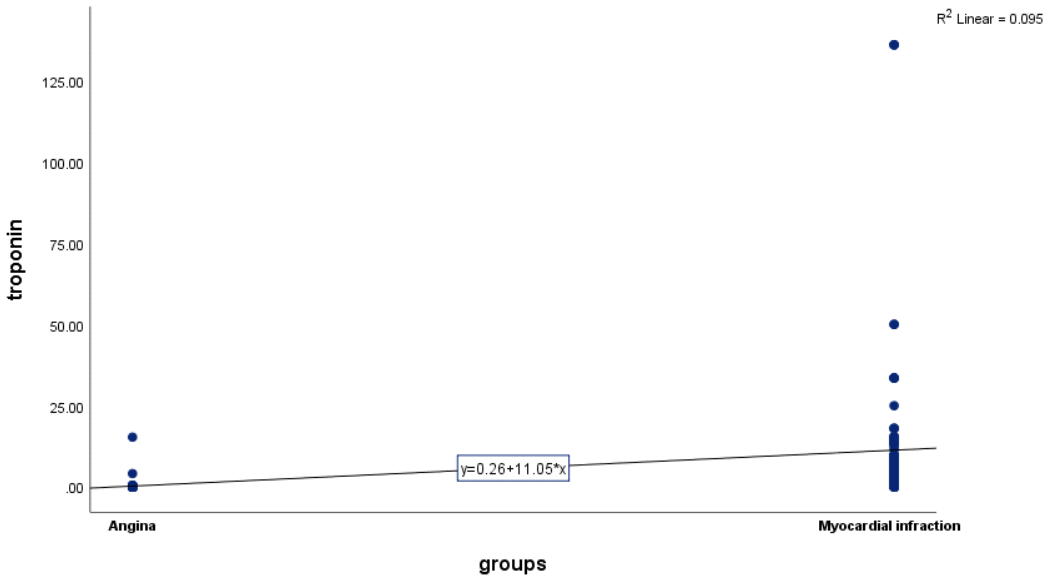

Figure (15): Scatter plot for the association between troponin and MI

CRP more than 20 could predict MI with sensitivity 42.16%, specificity 91.21%. CK-MB more than 15 could predict MI with sensitivity 59.43%, specificity 87.78%. troponin more than 0.5 could predict MI with sensitivity 75.47%, specificity 97.87%.

Table (12): Accuracy for prediction with MI

	AUC	Cut-off	Sensitivity	Specificity	Youden index J	95% CI	P value
CRP	0.671	>20	42.16%	91.21%	0.3337	0.600 to 0.737	0.0001
CK-MB	0.749	>15	59.43%	87.78%	0.4721	0.683 to 0.808	0.0001
troponin	0.923	>0.5	75.47%	97.87%	0.7334	0.877 to 0.956	0.0001

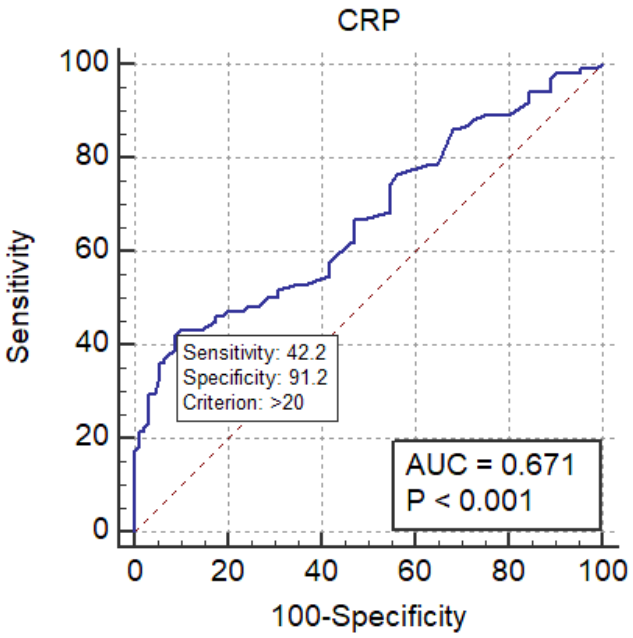

Figure (16): ROC curve for for prediction of MI using CRP

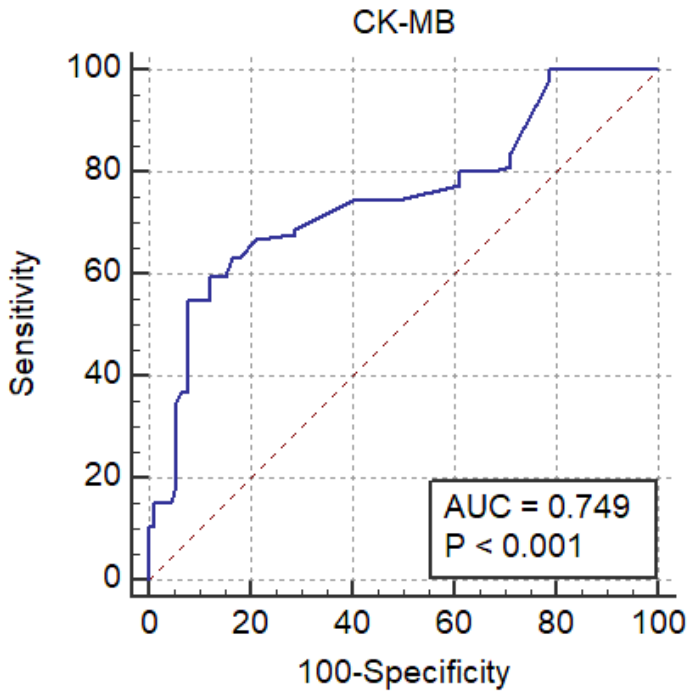

Figure (17): ROC curve for prediction with MI using CK-MB

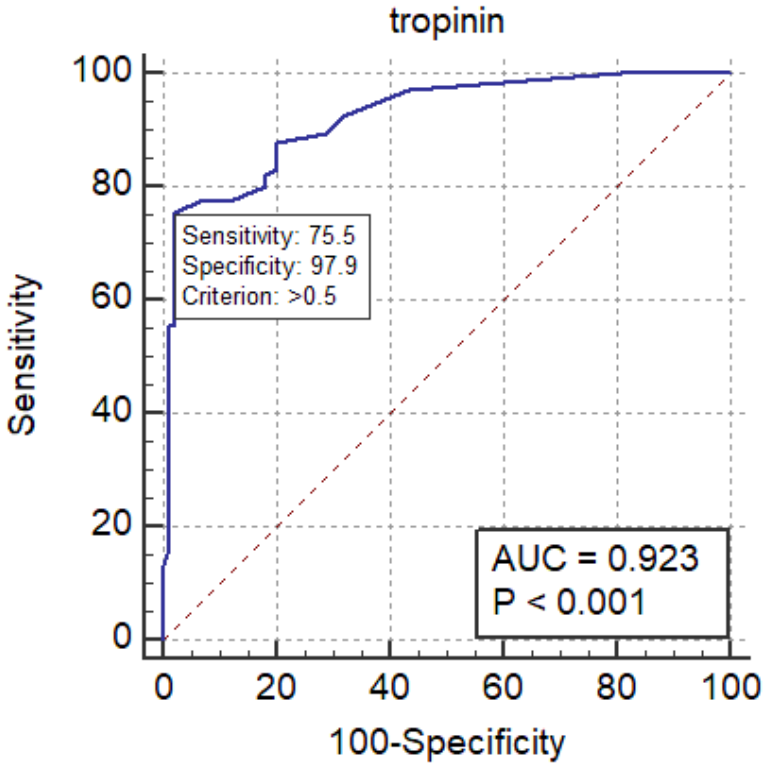

Figure (18): ROC curve for prediction of MI using tropinin

Troponin had the highest accuracy in the prediction with MI. There were no statistically significant difference between CK-MB and CRP in the prediction with MI.

Table(13):Comparison between Accuracy of various signs for prediction with MI

	Difference in AUC	95% CI	P value
CK-MB versus CRP	0.102	-0.00719 to 0.210	0.0672
CK-MB versus troponin	0.163	0.104 to 0.222	P < 0.0001
CRP versus troponin	0.264	0.177 to 0.352	P < 0.0001

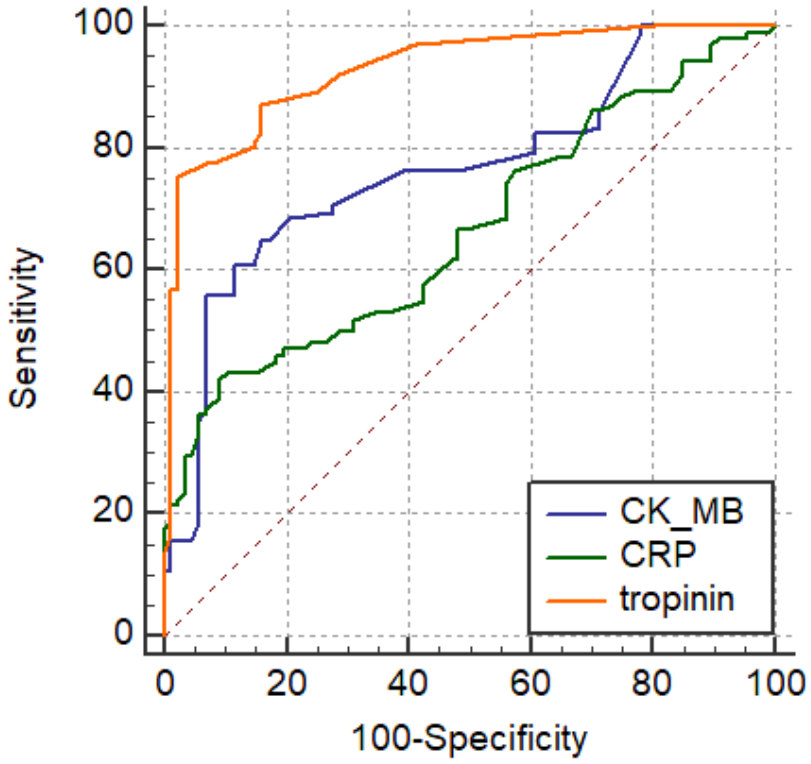

Figure (19): ROC curve for comparison between accuracy of various signs for prediction with MI

CK-MB more than 27 could predict acute MI with sensitivity 67.12, specificity 87.70.

Table (14): Prediction with acute MI using CKMB

	AUC	Cut-off	Sensitivity	Specificity	Youden index J	95% CI	P value
CK-MB	0.785	>27	67.12	87.70	0.5483	0.720 to 0.840	<0.0001

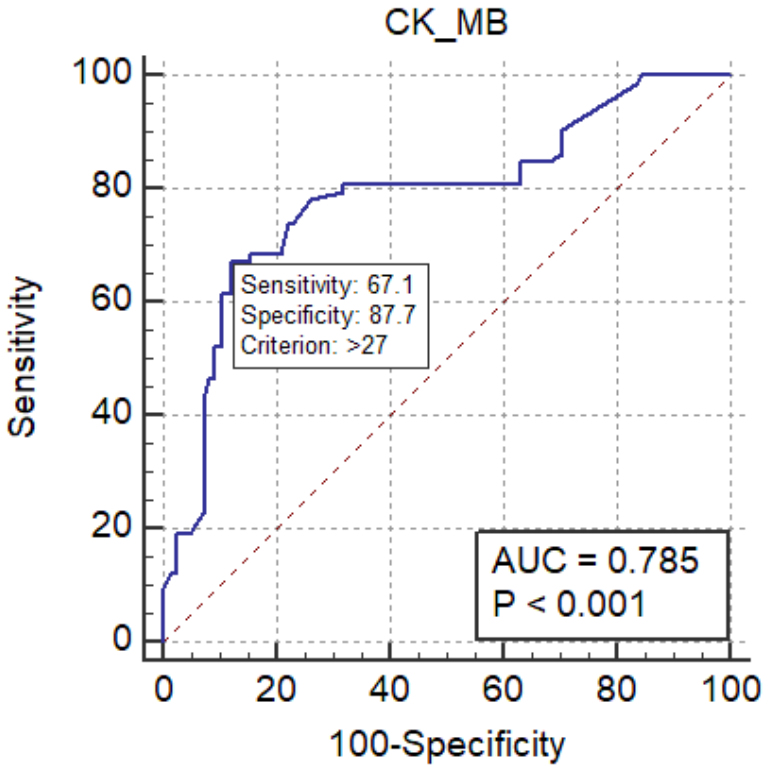

Figure (20): ROC curve for prediction with acute MI using CKMB

Discussion:

Myocardial ischemia and infarction represent significant health concerns. Inflammation plays a crucial role in the pathophysiology of these conditions. The elevation of CRP levels, an acute-phase protein, signifies the inflammatory status and has been associated with the severity and prognosis of myocardial damage. Measuring C-reactive protein (CRP) concentration in myocardial ischemia and infarction has garnered significant attention due to its potential role as a biomarker for cardiovascular risk stratification (Biasucci et al., 2013; Liu et al., 2022).

Several studies have demonstrated that elevated CRP levels are associated with adverse cardiovascular outcomes. For instance, Eggers et al. (2021) found that CRP independently predicted all-cause mortality in patients with myocardial infarction with nonobstructive coronary arteries (MINOCA), indicating its prognostic value beyond traditional risk factors (Eggers et al., 2021). Another study by Sabanoglu and Inanc (2022) further supported the link between inflammation markers like CRP and coronary artery disease severity assessed by Syntax score—a method quantifying coronary artery disease complexity (Sabanoglu & Inanc, 2022). Similarly, Ozdemir et al. (2020) corroborated these findings by demonstrating a correlation between elevated serum Fe and CRP concentrations with increased Syntax score,

reinforcing that systemic inflammation is intricately linked with atherosclerosis progression (Ozdemir, 2020).

Additional meta-analysis on the impact of preprocedural CRP levels on in-stent restenosis post-percutaneous coronary intervention revealed an association between higher baseline CRP levels and a greater likelihood of restenosis, highlighting the role of systemic inflammation even after revascularization procedures (Li et al., 2010). Furthermore, An et al. (2021) investigated ABCA1 promoter methylation and highlighted how epigenetic modifications influenced by inflammatory processes can affect cholesterol transport mechanisms critical for atherogenesis prevention (An et al., 2021). This finding suggested that inflammatory mediators like CRP may actively participate in pathophysiological pathways leading to CAD development beyond serving as mere biomarkers.

Therefore, the current study aimed to determine the association of CRP concentration with the severity of myocardial ischemia and infarction. Also, it seeks to assess the diagnostic accuracy of CRP concentration in distinguishing between acute and chronic myocardial ischemia and infarction. The study included 200 patients who were suspected of having acute myocardial infarction or unstable angina. Of the included patients, 53% were diagnosed with myocardial ischemia and infarction, and 47% were diagnosed with angina.

The present study investigated the median duration of complaints 'at 60 days, underscoring a pivotal distinction in the clinical presentations of myocardial infarction (MI) and angina. Notably, shorter symptom duration was observed in MI patients compared to those with angina, P=0.00. This difference highlights the acute nature of MI, potentially facilitating quicker medical intervention due to the severity or abrupt onset of symptoms. It also sheds light on the chronic or less acute manifestations of angina, which may contribute to delays in seeking medical attention.

Furthermore, the shortness of breath (SOB) without accompanying chest pain was predominantly noted in patients with angina, which introduces an essential diagnostic insight. It underscores the importance of a comprehensive symptom assessment in the clinical evaluation of cardiac conditions, challenging the conventional reliance on chest pain as a primary symptom of cardiac distress. This divergence in symptomatology and its duration between MI and angina patients enriches our understanding of these conditions. It underscores clinicians' need to adopt a nuanced approach to diagnosing and managing cardiac diseases, recognizing the broad spectrum of symptom presentations and their implications for patient care.

The presence of anginal symptoms or myocardial ischemia has been associated with clinical outcomes in patients with stable coronary artery disease. Interestingly, most cardiovascular events occurred in patients without angina or ischemia, suggesting that silent ischemia may not always predict adverse outcomes as strongly as symptomatic ischemia. However, anginal symptoms (with or without ischemia on noninvasive testing) but not silent ischemia appear to be associated with an increased risk for adverse cardiovascular outcomes (Steg et al., 2014).

In addition, the echocardiography and electrocardiography findings from our study revealed critical insights into the cardiac function and electrical activity of the heart among the participants. 23.5% of the patients exhibited a normal echocardiogram, and a significant 59% displayed hypokinesia across various walls. This variation in echocardiographic findings indicates that a considerable proportion of the subjects experience compromised heart wall motion, highlighting the severity of cardiac impairment.

Similarly, the electrocardiography results further emphasized the cardiac challenges for the patients in the study. Only 27.1% showed a normal ECG, while a substantial 64% exhibited ST changes, including elevation or depression, pointing towards significant myocardial ischemia or injury. The statistically significant differences observed between both groups in terms of echocardiography and electrocardiography findings corroborate the diversity in cardiac manifestations among the patients and underscore the essential role of these diagnostic tools in identifying and categorizing the extent of cardiac involvement.

These findings were consistent with several other research. For example, Joyce et al. (2015) explored the accuracy of two-dimensional speckle-tracking echocardiography in detecting significant coronary artery disease after ST-segment elevation myocardial infarction (STEMI), finding it a promising technique for identifying significant angiographic CAD. This finding aligns with the hypokinesia finding in our study, suggesting that advanced echocardiographic techniques can enhance the diagnostic accuracy for myocardial damage (Joyce et al., 2015). Also, Prastaro et al. (2017) discussed the prognostic role of echocardiography after acute myocardial infarction, highlighting its utility in evaluating myocardial walls involved in the ischemic process and predicting short- and long-term outcomes (Prastaro et al., 2017).

On the other hand, the current study found a significant elevation in Tropinin, CK-MB, and CRP levels in patients with MI than patients with angina, P-value< 0.001. This finding aligned with previous studies, underscoring the clinical value of these biomarkers in distinguishing between acute coronary syndromes. For example, it was found that Troponin,

CK-MB, and CRP levels positively correlated with the severity of myocardial infarction in aged patients, indicating their importance in assessing the condition's severity (Apple et al., 1995).

Likewise, Vyas et al. (2017) explored the changes in serum cardiac markers post-acute MI, emphasizing the diagnostic and prognostic utility of Troponin I, CK-MB, and CRP levels. Their findings highlight the markers' role in early detection and risk stratification of patients with acute coronary syndrome (ACS) (Vyas et al., 2017). Furthermore, Kouvelos et al. (2011) investigated the predictive value of these biomarkers for cardiovascular outcomes post-vascular surgery. Their study underscored the superiority of troponin levels over CK-MB and CRP in predicting adverse cardiovascular events, supporting their critical role in patient management following MI (Kouvelos et al., 2011).

Additionally, the present study observed elevated levels of CKMB in patients experiencing acute MI compared to those with chronic or subacute MI, p-value= 0.02. This differentiation in CKMB levels highlights its sensitivity to acute cardiac events, offering a valuable diagnostic tool for distinguishing the temporal onset of myocardial infarction. Conversely, the lack of statistically significant differences in troponin and C-reactive protein (CRP) levels across different stages of MI suggests a broader range of myocardial injury or inflammation that these biomarkers can indicate, not confined to the acute phase. This observation points to the complex interplay of biochemical markers in MI, where CKMB is a more acute phase indicator. At the same time, Troponin and CRP provide a broader lens on myocardial injury and inflammatory processes without specificity to the injury's timing (Biasucci et al., 2013).

The higher CKMB levels in patients with acute MI, compared to those with chronic or subacute MI, aligned with findings from previous research, suggesting that CKMB is a sensitive marker for early myocardial injury. For instance, Kurniawan et al. (2021) reported that troponin I and CK-MB values were significantly higher in STEMI Acute myocardial infarction patients compared to NSTEMI patients, highlighting the markers' capacity to indicate MI severity based on ST-elevation presence (Kurniawan et al., 2021).

Also, Chen et al. (2023) highlighted the prognostic value of hs-cTnT and CK-MB in AMI patients with chronic kidney disease (CKD), where both markers were high and predicted in-hospital mortality, suggesting the biomarkers' relevance extends to prognostication in specific patient subsets (Chen et al., 2023). Furthermore, Eyyupkoca et al. (2021) explored the cTnT/CK-MB ratio as a predictor of left ventricular function post-AMI, finding it a valuable indicator of adverse cardiac remodeling, thereby suggesting the dynamic interplay of these markers in post-MI recovery and remodeling processes (Eyyupkoca et al., 2021).

On the other hand, the present study found a significant negative correlation between ejection fraction (EF) and MI, P= 0.007. Conversely, a significant positive correlation was found between MI and CRP, Troponin, and CKMB. The analysis of the impact of ejection fraction, CRP, and troponin levels on the incidence of MI elucidates significant associations that are paramount in understanding the pathophysiological mechanisms underlying MI and its risk factors.

The negative association between the ejection fraction and the incidence of MI (95% CI: 0.927, 0.989) highlights the critical role of cardiac function in MI risk. Contrarywise, an increase in CRP levels by one unit is associated with a 1.043-fold increase in MI incidence (95% CI: 1.021, 1.065), underscoring the significance of inflammation in the pathogenesis of MI. Moreover, the study presents two distinct findings related to troponin levels; initially, a one-unit increase in troponin is associated with a 1.99-fold increase in MI incidence (95% CI: 1.49, 2.66), and subsequently, a slight adjustment in the model indicates a 1.023-fold increase with a 95% CI of (1.02, 1.034).

These results affirm the prognostic value of Troponin in indicating myocardial damage and suggest a nuanced relationship between troponin levels and MI incidence. Collectively, these associations between ejection fraction, CRP, and troponin levels with MI incidence underscore the multifactorial nature of MI risk, integrating cardiac functional status, inflammatory response, and myocardial injury markers in the comprehensive assessment of MI risk, thereby informing targeted interventions and management strategies in clinical practice (Pandey et al., 2017).

Lastly, the present study investigated the diagnostic accuracy of CRP, CK-MB, and Troponin in predicting myocardial infarction (MI). CRP levels exceeding 20 units offer a sensitivity of 42.16% and a specificity of 91.21%, indicating a moderate ability to correctly identify MI cases with a high capacity to exclude non-MI conditions. Conversely, CK-MB levels above 15 units enhance predictive sensitivity to 59.43% while maintaining a specificity of 87.78%, suggesting a better detection rate of MI than CRP, yet with a slight decrease in specificity. Notably, troponin levels surpassing 0.5 units emerge as the most precise biomarker, with an impressive sensitivity of 75.47% and the highest specificity at 97.87%, underscoring its superior accuracy in diagnosing MI.

The comparative analysis revealed no statistically significant difference between CK-MB and CRP in MI prediction, highlighting their complementary roles in the diagnostic process. However, Troponin's outstanding accuracy distinctly positions it as the paramount biomarker for MI detection, reflecting its critical role in clinical decision-making and

emphasizing the importance of integrating these biomarkers within a comprehensive diagnostic framework to optimize MI diagnosis and patient management strategies.

The diagnostic accuracy of CRP, CK-MB, and Troponin in predicting MI has been extensively studied; each biomarker offers unique insights into cardiac events. Nevertheless, several studies were consistent with our findings and detected that Troponin, due to its high sensitivity and specificity, is considered a cornerstone in diagnosing myocardial infarction. High-sensitivity Troponin (hsTn) assays could identify patients at risk for myocardial infarction more accurately than traditional biomarkers. For instance, the diagnostic accuracy of high-sensitive Troponin for identifying myocardial infarction in patients presenting with acute heart failure has been highlighted, underscoring its pivotal role in early detection (Ledwoch et al., 2022). Likewise, hsTn has a very high diagnostic accuracy for myocardial infarction (MI), with a negative predictive value of 81% to exclude obstructive coronary artery disease (CAD) (Paiva et al., 2024).

Additionally, the combination of Copeptin and Troponin has been recently found to rapidly rule out acute myocardial infarction and assess the prognostic value of post-myocardial infarction outcomes (Elseidy et al., 2023). Moreover, Troponin I has been shown to have a tremendous diagnostic ability for Acute Myocardial Infarction, with an area under the curve (AUC) of 0.99, indicating almost perfect accuracy (Pyati et al., 2016). Also, Troponin T and I have demonstrated high sensitivity and specificity, with Troponin I showing 88% sensitivity and 100% specificity, and Troponin T showing 93% sensitivity and 92% specificity in predicting MI (Hawkins & Tan, 1999).

In addition, CK-MB has been traditionally used in MI diagnosis; however, its role appears secondary to troponin in the diagnostic accuracy of AMI. The combination of CK-MB and POC-cTn or c-cTn might be valuable for the early diagnosis of AMI, especially when hs-cTn is unavailable. This combination of CK-MB with troponin could increase diagnostic sensitivity (56.1% vs. 63.9%, $P<0.001$; 82.7% vs. 84.3%, $P=0.025$) (G. Wang et al., 2022).

On the other hand, the diagnostic accuracy of C-reactive protein (CRP) in predicting myocardial infarction (MI) has been explored in several studies. CRP levels, especially high-sensitivity CRP (hsCRP), could predict recurrent cardiovascular events after MI and are associated with inflammation post-MI. Specifically, Wang et al. (2020) found significant decreases in cTnI and hs-CRP levels, as well as their extent of change (C) and change rate (Cr) in the effective group of MI patients. These parameters were found to be independent factors for evaluating treatment efficacy. The authors concluded that C and Cr values of cTnI and hs-

CRP are more valuable in assessing early treatment efficacy than absolute values (L. Wang et al., 2020).

Moreover, a comparison between hs-CRP (AUC= 0.572, 95% CI [0.470–0.675]) and CRP (AUC= 0.565, 95% CI [0.462–0.669]) showed similar diagnostic accuracy in predicting mortality in patients with acute myocardial infarction, suggesting that both markers can be useful in clinical settings (Hofer et al., 2021). Another study highlighted the CRP/troponin ratio as an effective tool for differentiating perimyocarditis from acute myocardial infarction (AMI), showing reasonable specificity when the ratio is above 500 (Kobo et al., 2021). In a specific patient population with Type 2 diabetes mellitus, CRP demonstrated a sensitivity of 84.6% and a specificity of 75.9% for predicting AMI, with a critical value of 7.34 mg/L according to ROC analysis (Li et al., 2018).

In conclusion, this study provided compelling evidence supporting the utility of C-reactive protein (CRP) as a biomarker for myocardial ischemia and infarction. The findings demonstrate a significant elevation in CRP levels among patients with myocardial infarction compared to those with angina. This aligned with previous research underscoring the role of CRP as an indicator of inflammation intricately linked with atherosclerotic disease progression and adverse cardiovascular outcomes.

Furthermore, comparative analysis reveals CRP's complementary role alongside other biomarkers like CK-MB and, most notably, troponin in diagnosing acute myocardial infarction. Though troponin emerges as the most accurate predictor of MI, CRP levels above 20 mg/L maintain reasonable sensitivity and high specificity for MI detection. The study enriches our understanding of how these cardiac biomarkers intersect to provide clinical insights into myocardial injury's presence, severity, and temporal onset.

Additionally, investigating the correlations between ejection fraction, CRP, troponin, and CK-MB levels with MI incidence focuses on the multifaceted pathophysiology underpinning myocardial infarction risk. The findings highlighted the integration of cardiac function, inflammatory status, and myocardial injury markers in comprehensively evaluating and stratifying MI risk to guide targeted therapeutic interventions.

Overall, this study underscored CRP's utility as a predictor and prognosticator of myocardial infarction, independently or in conjunction with other biomarkers. It substantiated the role of inflammatory processes in MI pathogenesis and the need to incorporate CRP evaluation in the diagnosis and risk stratification of patients with suspected acute coronary events. Further research is warranted to continuously refine the application of CRP and other biomarkers in the clinical management of myocardial ischemia and infarction.

Conclusion: In conclusion, this study provided compelling evidence supporting the utility of C-reactive protein (CRP) as a biomarker for myocardial ischemia and infarction. The findings demonstrate a significant elevation in CRP levels among patients with myocardial infarction compared to those with angina. This aligned with previous research underscoring the role of CRP as an indicator of inflammation intricately linked with atherosclerotic disease progression and adverse cardiovascular outcomes. this study underscored CRP's utility as a predictor and prognosticator of myocardial infarction, independently or in conjunction with other biomarkers. It substantiated the role of inflammatory processes in MI pathogenesis and the need to incorporate CRP evaluation in the diagnosis and risk stratification of patients with suspected acute coronary events

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