



ORIGINAL ARTICLE

Novel Correlation among C3, h-CRP, and WBC in the Pathogenesis of ST-elevation Myocardial Infarction

Hassan Sarhan Sachit¹, Ali Mohammed Barakat¹, Ghassan Jabbar Auda¹, Raed Fanoukh Aboqader^{*2}, Adnan Taan Thamer¹

¹College of Medicine, Al-Ayen Iraqi University, AUIQ, An Nasiriyah, Iraq

²Al-Aouadi College of Medicine, Al-Ayen Iraqi University, AUIQ, An Nasiriyah, Iraq

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ABSTRACT: ST-segment elevation myocardial infarction (STEMI) is a major contributor to global morbidity and mortality, necessitating a deeper understanding of its pathophysiology. This study aimed to evaluate the correlation between white blood cell (WBC) counts, complement component C3, high-sensitivity C-reactive protein (hs-CRP), and high- and low-density lipoprotein levels in patients diagnosed with ST-segment elevation myocardial infarction (STEMI) compared to a healthy control group. A total of 60 participants were included, comprising 30 individuals diagnosed with STEMI and 30 healthy controls, who were recruited from outpatient clinics in Nassirya Province, Iraq, between January and December 2024. Blood samples were collected and analyzed for WBC counts, hs-CRP, C3, and LDL levels using standardized laboratory techniques, including ELISA and spectrophotometry. The results showed a significant increase in hs-CRP ($P < 0.0001$), WBC ($P < 0.0001$), and LDL ($P < 0.0001$) levels in the STEMI group compared to the control group, accompanied by a notable decrease in C3 levels ($P < 0.0001$). A positive correlation was notably observed between BMI and increased levels of LDL ($r = 0.939$, $P < 0.01$). Additionally, a positive correlation was remarkably observed between BMI and increased levels of WBC ($r = 0.815$; $P < 0.01$) and h-CRP ($r = 0.805$; $P < 0.01$), but a negative correlation was detected between BMI and levels of HDL ($r = -0.937$; $P < 0.01$) and C3 ($r = -0.733$; $P < 0.01$). These findings suggest that elevated inflammatory markers and lipid profiles may play a crucial role in the immunopathogenesis of STEMI. The study highlights the importance of monitoring these biomarkers in clinical settings, as they may provide insights into the inflammatory processes associated with myocardial infarction. Furthermore, the correlation among these markers could inform potential therapeutic strategies to mitigate inflammation and improve patient outcomes post-STEMI. Overall, this research contributes to the understanding of the complex interplay between lipid metabolism and inflammatory responses in the context of acute myocardial infarction, emphasizing the need for further investigation into targeted interventions for high-risk populations.

INTRODUCTION

The concept of acute myocardial infarction has undergone significant changes to incorporate more sensitive indicators of myocardial damage and advanced imaging techniques that enhance our comprehension of

the underlying mechanisms associated with acute coronary syndrome. Consequently, the universal definition of myocardial infarction suggests categorizing patients based on the underlying causes of their

*Corresponding author: Raed.fanoukh@alayer.edu.iq (R. Fanoukh Aboqader)
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condition. While this classification has been utilized in clinical trials to refine both primary and secondary endpoints, its application in routine clinical practice remains limited, leading to uncertainty regarding the prevalence and consequences of various subtypes of acute myocardial infarction[1, 2].

In contemporary clinical practice, there is an acknowledgment of a continuum of both acute and chronic myocardial injury stemming from diverse cardiac and non-cardiac factors. The prognosis for both patient groups is poor, and the approaches to investigation and management are often inconsistent. This inconsistency may be partly attributed to variations in the interpretation of existing guidelines. ST-segment elevation myocardial infarction (STEMI) is a life-threatening condition caused by the abrupt occlusion of an epicardial coronary artery. Timely reperfusion, ideally achieved through primary angioplasty or systemic fibrinolysis, significantly improves survival rates in patients affected by this condition [3, 4].

The significance of thrombosis as a precipitating factor for acute myocardial infarction (AMI) has been a subject of extensive discourse throughout the 20th century, culminating in a consensus by the 1970s, which identified it as the predominant cause of nearly all AMIs identified during autopsies, along with the majority of clinically evident large AMIs. The primary and most critical instigator of thrombosis is atherosclerosis, often accompanied by subsequent inflammatory responses [5]. The initiation of atherosclerotic lesions manifests as intimal thickening within the walls of coronary arteries or as lipid streaks[6]. Over time, some lesions progress into either robust, fibrous-capped atheromas or vulnerable, thin, fibrous-capped atheromas characterized by a lipid-rich core. These atherosclerotic formations are susceptible to acute exacerbations, which may occur either through asymptomatic thrombosis or intraplaque hemorrhage [2, 7].

The onset of atherosclerosis is instigated by the uptake and oxidation of low-density lipoprotein (LDL) within the intima, triggering a sequence of inflammatory cytokine release, enzymatic activity, and the expression of cell adhesion molecules. This cascade leads to the recruitment of T lymphocytes and monocytes into the subintimal compartment[8]. The buildup of oxidized

LDL causes further damage to endothelial cells, leading to the increased production of cytokines and reactive oxygen species within the subintimal area. The deleterious effects of oxidized LDL extend beyond cardiovascular disease, contributing to various human pathologies as highlighted in recent comprehensive reviews on its role across multiple disease pathways[9]. Subsequently, oxidized LDL is phagocytosed by macrophages derived from monocytes, transforming them into foam cells. Over an extended period, smooth muscle cells migrate from the media to the intima, where lipid accumulates beneath a fibrous cap composed of vascular smooth muscle cells, elastin, and collagen. Additionally, low-grade inflammation, as indicated by elevated levels of C-reactive protein—regardless of LDL concentration—has been implicated in myocardial incidents and is believed to play a role in both the development and progression of atherosclerotic disease [10].

Hyperlipidemia and increase in LDL remain the primary cause of acute myocardial infarction. Acute myocardial infarction initiates an intense inflammatory response[11]. The relationship between low-density lipoprotein cholesterol (LDL-C) levels and myocardial infarction (MI) is complex, with recent studies revealing both potential risks and benefits associated with LDL-C management. While lower LDL-C levels are generally considered beneficial, paradoxically, they may correlate with worse outcomes in MI patients. This overview will examine the implications of LDL-C levels in the context of myocardial infarction [12].

At the cellular signaling level, factors involved in various cell death programs are activated. During the ensuing necrosis, cytoplasmic membranes become unstable, leading to the dispersal or exposure of intracellular contents into the extracellular space[13]. This triggers an immune response, as evidenced by the release of pro-inflammatory cytokines, activation of the complement system, and infiltration of inflammatory cells. Myocardial infarction (MI) is a critical condition characterized by the death of heart tissue due to ischemia, and recent research highlights the role of complement C3 in its pathophysiology. Studies indicate that C3 is integral to the innate immune response, influencing myocardial necrosis and subsequent cardiac

fibrosis. This response can significantly impact post-infarction outcomes [14, 15].

Prior animal research has indicated that under conditions of ischemia/reperfusion (I/R) injury, circulating C3 is found in the ischemic heart tissue that is subsequently reperfused with oxygenated blood. Recently, researchers discovered that in a mouse heart model, the level of myocardial necrosis was reduced in C3-deficient (C3^{-/-}) mice. This suggests that in wild-type (WT) mice subjected to I/R, C3 contributes to the promotion of necrosis. In addition to necrosis, fundamental studies have demonstrated that apoptosis also plays a significant role in cardiac ischemia-reperfusion (I/R) injury. However, it remains uncertain whether C3 is involved in apoptosis during myocardial I/R in humans [16].

C-reactive protein (CRP) is a significant biomarker in the context of myocardial infarction (MI), particularly about inflammation and prognosis. Elevated CRP levels post-MI have been associated with larger infarct sizes and increased mortality risk [17]. CRP is a well-established marker of inflammation and is elevated in patients with coronary artery disease and myocardial infarction. Studies have shown that elevated h-CRP levels correlate with the severity of coronary artery disease and can predict adverse outcomes in STEMI patients.

The combination of elevated h-CRP and WBC count provides a more comprehensive assessment of the inflammatory state and risk stratification in STEMI patients. These markers, along with other novel biomarkers, can enhance the prediction of complications and guide therapeutic intervention. While the focus is often on h-CRP and WBC count, the role of C3 in STEMI pathogenesis remains less explored. Further research is needed to elucidate the specific contributions of C3 and its potential interactions with other inflammatory markers in the context of myocardial infarction. Understanding these relationships could lead to the development of improved diagnostic and therapeutic strategies for managing STEMI.

The following sections elaborate on these associations. Together with the intracellular changes, these inflammatory responses lead to the cell death of ischemic tissues and subsequent long-term consequences;

therefore, studying the correlation among WBC, C3, hs-CRP, and LDL is an important subject.

MATERIALS AND METHODS

This case-control investigation comprised 60 participants, including 30 individuals diagnosed with ST-elevation myocardial infarction (STEMI) and 30 healthy control subjects with the same age range and gender-matched, recruited from outpatient clinics in Nassiryia Province, Iraq, between January and December 2024. Fasting blood samples were obtained to analyze various lipid profiles, including high-density lipoprotein (HDL), low-density lipoprotein (LDL), complement component C3, high-sensitivity C-reactive protein (h-CRP), and white blood cell (WBC) counts. The participants' ages ranged from 39 to 72 years, and they were categorized into two distinct groups. A volume of 5 ml of blood was drawn from the patients. At the same time, the control samples were divided into two portions: one portion was collected in an EDTA tube for hematological analysis of WBC using an automated analyzer, and the other was centrifuged for evaluation of serum hs-CRP, C3, and LDL levels. The hs-CRP levels were quantified using the DRG CRP HS ELISA kit, while serum C3 was assessed with a commercially available immunoassay kit from Milano, Italy. LDL levels were determined manually via spectrophotometry using a Randox kit from the UK, and WBC counts were analyzed using a Beckman Coulter hematology autoanalyzer. The Body Mass Index (BMI) was calculated in kg/m² by dividing the weight in kilograms by the square of the height in meters. A detailed questionnaire was employed to collect demographic information, including gender, age, smoking status, physical inactivity, and family history of acute coronary syndrome (ACS) for both groups. Rigorous exclusion criteria were applied to eliminate individuals with chronic illnesses such as diabetes, liver cirrhosis, end-stage renal failure, acute heart failure, strokes, musculoskeletal injuries, cancer, endocrine disorders, and other inflammatory diseases that could potentially influence the study results.

Statistical analysis

Data analysis was conducted utilizing IBM's Statistical Package for the Social Sciences, version 27.0 (SPSS, Chicago, Illinois, USA). The Shapiro-Wilk test was applied to assess the normality of the data distribution. Independent T-tests were employed to compare the mean values of biomarkers across different groups. By employing receiver operating characteristic (ROC) analysis, the Area under the Curve (AUC) and the 95% confidence interval lower and upper limits of C3, CRP, and WBC were determined in critical scenarios. The association between acute coronary syndrome (ACS) and lipid profiles, C3, CRP, and WBC, was investigated using Pearson's correlation coefficient. Scale variables that exhibited a normal distribution were expressed as mean $\pm$ standard deviation. A p-value of less than 0.05 was deemed statistically significant.

RESULTS

The analysis revealed that the average age of participants in the MI cohort was 59 ± 1.80 years. In comparison, the control group had a mean age of 54.90 ± 1.87 years, a difference that was not statistically significant ($P = 0.121$, see Table 1). The average BMI of the MI group was 28.03 ± 0.14 kg/m², whereas the control group had a mean BMI of 23.17 ± 0.12 kg/m², showing a statistically significant difference ($P < 0.0001$). The HDL and LDL levels in the MI group were measured at 32.63 ± 0.39 and 173.92 ± 1.3 , respectively. In contrast, the control group exhibited HDL and LDL levels of 55.67 ± 0.78 and 102.89 ± 1.23 , respectively, with both variables being statistically significant ($P < 0.0001$) (Table 1 and Figure 1).

The findings of this research indicate that the concentrations of C3 significantly declined in the patients suffering from MI when compared to those in the control group, with mean $\pm$ SE values recorded at 19.13 ± 2.43 mg/dL for the MI patients and 60.44 ± 3.25 mg/dL for the individuals in the control group, a difference that reached statistical significance as indicated by the P-value of less than 0.0001. Moreover, the outcomes from this study demonstrated that the levels of h-CRP were similarly elevated in the MI patient group

in comparison to the control group, with mean $\pm$ SE values of 13.30 ± 0.67 mg/dL for the MI patients contrasted with 4.38 ± 0.34 mg/dL for the control group, confirming a statistically significant difference as denoted by the P-value of less than 0.0001, which can be found in both Table 1 and Figure 1. Additionally, the WBC count elevated in the MI patient group in comparison to the control group, with mean $\pm$ SE values of $14.96 \pm 0.38 \times 10^9/L$ for the MI patients contrasted with $8.61 \pm 0.40 \times 10^9/L$ for the control group, confirming a statistically significant difference as denoted by the P-value of less than 0.0001 (Table 1 and Figure 1).

A positive correlation was notably observed between body mass index (BMI) and increased levels of LDL, with a correlation coefficient (r) of 0.939 and a P-value of less than 0.01, as indicated in Table 2. Additionally, a positive correlation was remarkably observed between BMI and increased levels of WBC ($r = 0.815$; $P < 0.01$) and h-CRP ($r = 0.805$; $P < 0.01$), but a negative correlation was detected between BMI and levels of HDL ($r = -0.937$; $P < 0.01$) and C3 ($r = -0.733$; $P < 0.01$) (Table 2).

In addition to this finding, a positive correlation was also identified between advancing age and elevated BMI ($r = 0.188$), LDL ($r = 0.225$), WBC ($r = 0.163$), and h-CRP ($r = 0.211$) while conversely, the correlation between BMI and age with high-density lipoprotein (HDL) ($r = -0.172$) and C3 ($r = -0.205$) demonstrated a negative relationship, as detailed in Table 2. The receiver operating characteristic (ROC) analysis for WBC indicated an area under the curve (AUC) of 0.954 with a 95% confidence interval (CI) ranging from 0.901 to 1. The ROC analysis for C3 indicated a 95% confidence interval (CI) ranging from 0.001 to 0.098 and an area under the curve (AUC) of 0.049. The analytical assessment, known as the receiver operating characteristic (ROC) analysis, specifically focused on h-CRP, revealed a 95% confidence interval (CI) that ranged from a lower limit of 0.967 to an upper limit of 1. This was further substantiated by an area under the curve (AUC) value of 0.987, indicating robust performance of the biomarker in question (Table 3 and Figure 2).

Table 1. The mean SE of various variables in two different examined groups carried out by Independent-T test.

	Group	N	Mean $\pm$ SE	P-value
BMI(kg/m²)	MI	30	28.03 $\pm$ 0.14	0.0001
	Control	30	23.170 $\pm$ 0.12	
Age(Year)	MI	30	59.00 $\pm$ 1.80	0.121
	Control	30	54.90 $\pm$ 1.87	
HDL(mg/dl)	MI	30	32.63 $\pm$ 0.39	0.0001
	Control	30	55.68 $\pm$ 0.78	
LDL(mg/dl)	MI	30	173.92 $\pm$ 1.3	0.0001
	Control	30	102.89 $\pm$ 1.23	
WBC($\times 10^9$/L)	MI	30	14.96 $\pm$ 0.38	0.0001
	Control	30	8.61 $\pm$ 0.40	
C3 (mg/dl)	MI	30	19.13 $\pm$ 2.43	0.0001
	Control	30	60.44 $\pm$ 3.25	
h-CRP(mg/dl)	MI	30	13.30 $\pm$ 0.67	0.0001
	Control	30	4.38 $\pm$ 0.34	

Table 2. Correlation analysis among variables, including profile lipids, BMI, Age, C3, WBC, and h-CRP, using Pearson's correlation coefficient.

	Group	BMI	Age	HDL	LDL	WBC	C3	h-CRP
Group	1	-0.961**	-0.202	0.961**	-0.981**	-0.831**	0.800**	-0.838**
BMI	-0.961**	1	0.188	-0.937**	0.939**	0.815**	-0.733**	0.805**
Age	-0.202	0.188	1	-0.172	0.225	0.163	-0.205	0.211
HDL	0.961**	-0.937**	-0.172	1	-0.943**	-0.763**	0.727**	-0.813**
LDL	-0.981**	0.939**	0.225	-0.943**	1	0.823**	-0.792**	0.817**
WBC	-0.831**	0.815**	0.163	-0.763**	0.823**	1	-0.666**	0.763**
C3	0.800**	-0.733**	-0.205	0.727**	-0.792**	-0.666**	1	-0.705**
h-CRP	-0.838**	0.805**	0.211	-0.813**	0.817**	0.763**	-0.705**	1

** . Correlation is significant at the 0.01 level (2-tailed).

Table 3. Area Under the Curve (AUC), 95% CI Lower and Upper of WBC, C3, and h-CRP obtained by Roc Curve in patients.

Variable(s)	AUC	S.E	P-value	95% CI	
				Lower	Upper
WBC	0.954	0.027	0.000	0.901	1.000
C3	0.049	0.025	0.000	0.001	0.098
h-CRP	0.987	0.010	0.000	0.967	1.000

The test result variable(s) (WBC, C3) have at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption; b. Null hypothesis: actual area = 0.5

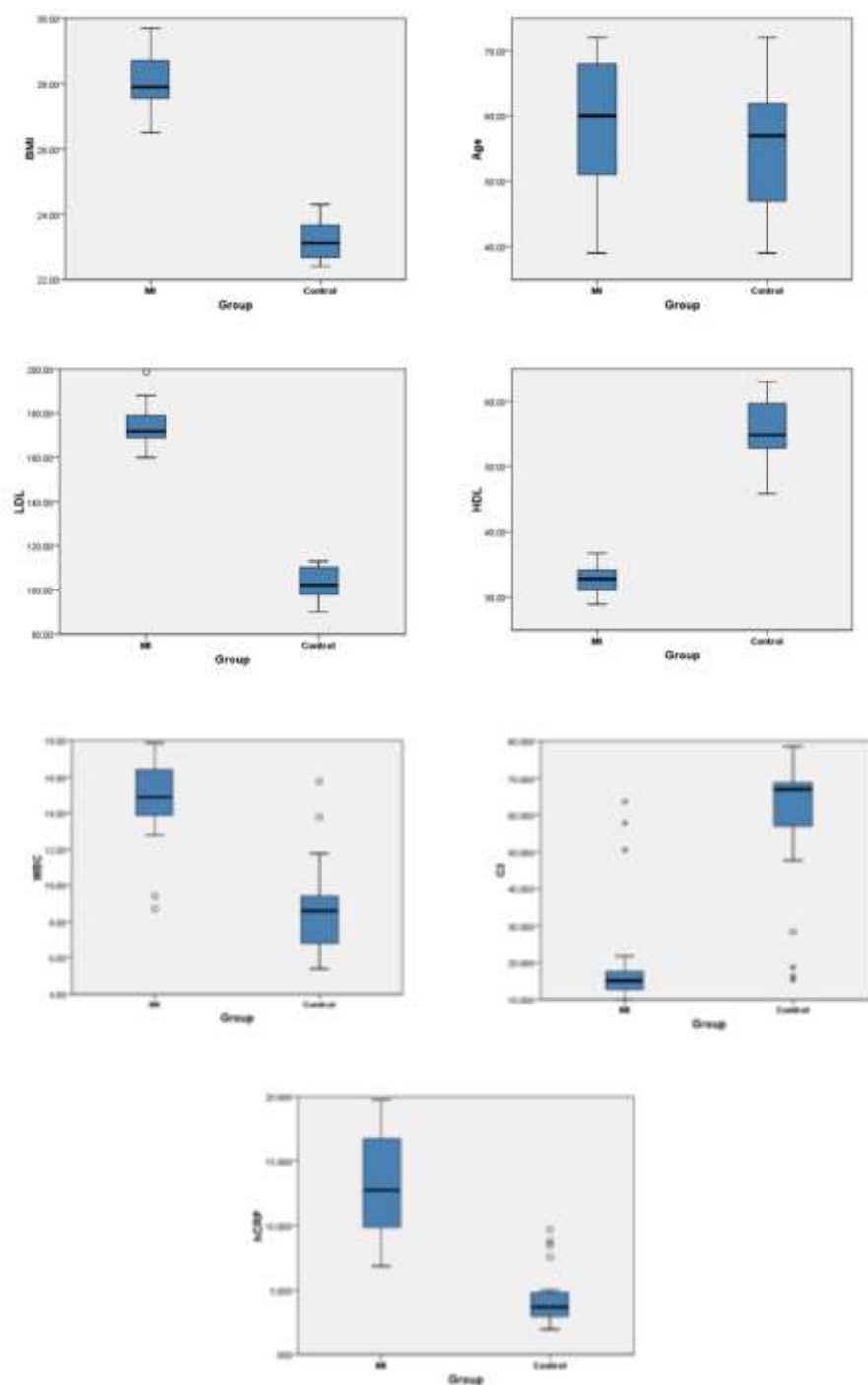


Figure 1. The comparison of BMI, age, LDL, HDL, WBC, C3, and h-CRP in MI and control groups.

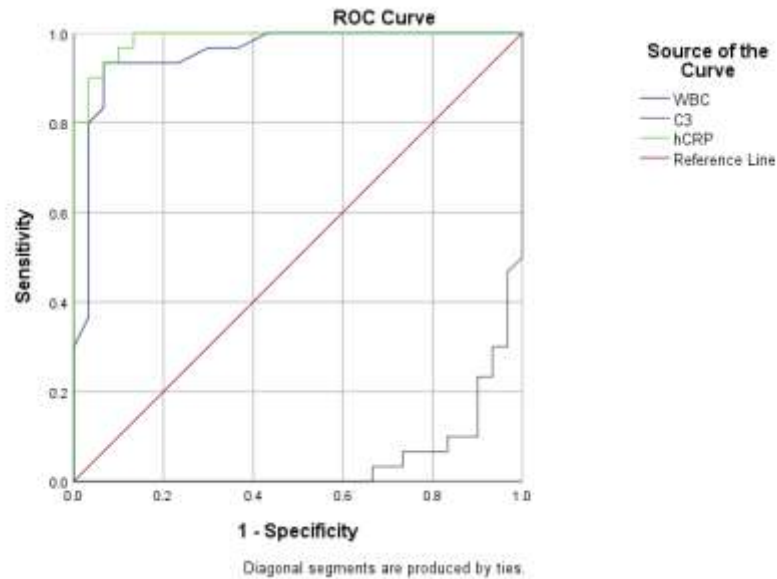


Figure 2. Receiver Operating Characteristic (ROC) Curve of C3, WBC, and h-CRP in patients with MI.

DISCUSSION

The pathogenesis of acute myocardial infarction (AMI) is multifactorial; however, several studies have implicated impaired lipid metabolism as one of the crucial factors in the development of this disease[18]. The investigation revealed markedly elevated levels of high-sensitivity C-reactive protein (hs-CRP) and white blood cell (WBC) counts in patients with ST-elevation Myocardial Infarction (STEMI) when compared to healthy individuals. Additionally, the study noted a significant reduction in complement component C3 levels among STEMI patients, indicating that C3 may play a multifaceted role in myocardial injury and recovery, potentially influencing adverse remodeling following an infarction.

Obesity is known to trigger a condition of chronic low-grade inflammation, which significantly contributes to cardiovascular diseases, including myocardial infarction (MI). This inflammatory condition is marked by elevated levels of pro-inflammatory cytokines like CRP, which are linked to negative cardiovascular outcomes in MI patients with visceral obesity[19]. It results in the buildup of adipose tissue, which functions as an endocrine organ that releases inflammatory mediators, thereby fostering a pro-inflammatory environment. This inflammation is correlated with heightened oxidative stress and mitochondrial dysfunction, which further exacerbates cardiovascular issues[20].

C3 is an indicator of chronic inflammation, and its levels are heightened in obese individuals, linking it to insulin resistance and an increased risk of cardiovascular problems. In the scenario of myocardial infarction, lower levels of C3 may suggest a malfunctioning immune response, which could impact the evolution and recovery from MI. The NLRP3 inflammasome pathway, activated by obesity, plays a role in the inflammatory response and may also influence C3 levels.[20, 21]

Though the relationship between obesity, inflammation, and decreased C3 levels in MI patients is clear, it's crucial to recognize the wider implications of these observations. The inflammatory state caused by obesity not only impacts cardiovascular health but also has repercussions for other disorders, such as insulin resistance and metabolic syndrome. Gaining insight into these connections can aid in developing targeted treatments to alleviate the detrimental effects of obesity on cardiovascular health[19, 20]. Although the specific diagnostic significance of lowered C3 levels in patients with myocardial infarction is not extensively documented, the involvement of complement activation in heart-related diseases suggests a possible, albeit indirect, importance. Components of the complement system, such as C3, may indicate the presence of inflammatory processes that contribute to cardiovascular incidents, including myocardial infarction (MI)[22].

However, in the present study, based on ROC analysis, the AUC for C3 is surprisingly low, indicating an almost little diagnostic value.

This inflammation is linked to increased oxidative stress and mitochondrial dysfunction, further exacerbating cardiovascular conditions. A study involving 1,987 MI patients found that those with the lowest LDL-C levels had a significantly higher risk of major adverse cardiovascular events (MACE) compared to those with higher levels, suggesting that very low LDL-C may indicate a poorer prognosis [23]. Conversely, a personalized lipid-lowering approach targeting LDL-C levels below 55 mg/dL resulted in improved outcomes, with a lower incidence of MACE observed in patients achieving this goal [24].

Elevated levels of oxidized LDL (Ox-LDL) were associated with a 1.70-fold increased risk of premature myocardial infarction (MI), suggesting that not all LDL-C are equal; the oxidized form may play a more detrimental role in atherosclerosis. The study found a significant association between elevated circulating oxidized low-density lipoprotein cholesterol (Ox-LDL) levels and premature myocardial infarction (MI), indicating that higher Ox-LDL levels increase the risk of MI, particularly in younger individuals [24]. In agreement with previous studies, the serum level of LDL in the MI group (173.92 ± 1.3) was found to be higher than that in the control group (102.89 ± 1.23), which is recognized as a major risk factor in MI patients.

A significant proportion of post-myocardial infarction (MI) patients fail to achieve the recommended low-density lipoprotein cholesterol (LDL-C) targets, with only 31% reaching levels below 55 mg/dL, as reported in a recent study. This underutilization of effective therapies highlights the need for improved management strategies. In contrast, some researchers argue that focusing solely on lowering LDL-C may overlook other critical factors influencing cardiovascular health, such as inflammation and overall lipid profiles. This perspective suggests a more nuanced approach to lipid management in MI patients. Patients with a history of myocardial infarction should aim for LDL-cholesterol levels <55 mg/dL. In the study, only 31% of post-myocardial infarction patients achieved this target, highlighting significant

underutilization of available therapies for LDL-C management [24].

In murine models, C3 deficiency resulted in decreased myocardial necrosis compared to wild-type (WT) mice following ischemia/reperfusion injury. C3^{-/-} mice exhibited better cardiac function and less fibrosis four weeks post-infarction, suggesting that C3 contributes to adverse remodeling following MI. The findings suggest that targeting the complement system, particularly C3 may offer new therapeutic avenues to mitigate heart failure after myocardial infarction (MI) by reducing fibrosis and improving cardiac function. Myocardial infarction leads to necrotic cell death and cardiac fibrosis. The study demonstrates that mice deficient in complement C3 exhibit reduced myocardial necrosis, less cardiac fibrosis, and improved cardiac function compared to wild-type mice, underscoring the crucial role of C3 in the progression of heart failure [24]. Conversely, while C3 plays a detrimental role in myocardial injury, other factors, such as thyroid hormones and inflammatory markers like C-reactive protein (CRP), also significantly influence outcomes in MI patients, indicating that a multifaceted approach is necessary for effective treatment strategies [24].

In 2024, Gregory found that baseline apolipoprotein C3 (ApoC3) levels were not significantly related to the risk of myocardial infarction ($p = 0.50$) in patients with acute coronary syndrome who received optimized statin treatment, indicating no clinically meaningful predictive value [24]. High CRP levels after discharge in patients with ST-segment elevation myocardial infarction (STEMI) correlate with larger final infarct sizes, indicating more extensive myocardial damage. Patients with elevated CRP levels showed a final myocardial salvage index (MSI) that was significantly lower, indicating poorer recovery [24]. A study found that higher CRP levels during hospitalization were associated with increased long-term mortality in MI patients, with the highest quartile of CRP showing a nearly threefold increase in risk compared to the lowest quartile. The velocity of CRP increase during the acute phase of STEMI also correlates with higher mortality rates, indicating that rapid inflammation may exacerbate myocardial injury [24]. In the present study, an elevation in the h-CRP level was observed in MI patients. In

addition to this finding, a positive correlation was also identified between advancing age and elevated BMI ($r = 0.188$), LDL ($r = 0.225$), WBC ($r = 0.163$), and h-CRP ($r = 0.211$).

The CRP-troponin test has been proposed as a tool to stratify mortality risk in non-ST-elevation myocardial infarction (NSTEMI) patients, emphasizing the importance of both biomarkers in assessing patient outcomes. While elevated CRP levels are generally associated with worse outcomes in MI, it is essential to consider individual patient factors, such as comorbidities and the timing of CRP measurements, which may influence the prognostic value of this biomarker[25].

On the other hand, Rafael Y Brzezinski et al. (2024) reported high CRP levels (>54 mg/L), indicating an increased 30-day mortality risk. Additionally, normal CRP levels with high troponin levels (4,918 ng/L) also correlate with higher mortality rates. Early CRP values after acute myocardial infarction are predictive of one-year mortality[25]. According to the Alkouri report of 2022, higher CRP levels at 12, 24, and 48 hours post-AMI are associated with an increased mortality risk, suggesting the potential for identifying patients who may benefit from anti-inflammatory therapies [25]. This observation highlights the importance of effective lipid management strategies for individuals at risk of ST-segment elevation myocardial infarction (STEMI). Additionally, given the central role of oxidative stress in propagating both lipid peroxidation and inflammation, the incorporation of antioxidant therapies may provide a complementary approach, as discussed in detail in recent literature addressing antioxidant mechanisms across various diseases [26].

The interaction between complement C3 and cytochrome c plays a significant role in modulating myocardial apoptosis during ischemia-reperfusion (I/R) injury. Complement C3, a central component of the complement system, has been shown to interact with cytochrome c in the cytosol of cardiomyocytes, potentially sequestering cytochrome c and reducing apoptosis[27]. This interaction suggests a novel mechanism where a circulating immune factor influences intracellular processes to affect cell death outcomes during ischemic events. Beyond apoptosis, C3 is involved in preserving myocardial function and reducing fibrosis post-

infarction. C3 deficiency leads to increased fibrosis and impaired cardiac function, suggesting its role in myocardial preservation and regeneration[16]. The complement system, including C3, is implicated in the inflammatory response during reperfusion, which can exacerbate myocardial injury. However, maintaining C3 activity while inhibiting later complement events may offer therapeutic benefits. While the interaction between C3 and cytochrome c provides a protective mechanism against apoptosis, the broader role of C3 in myocardial ischemia/reperfusion involves complex interactions with inflammatory and regenerative processes. The balance between these roles is crucial for therapeutic strategies aimed at reducing myocardial injury and improving cardiac outcomes[16, 27].

CONCLUSIONS

The study emphasizes the need for a comprehensive approach to managing STEMI, which includes addressing both inflammatory and lipid-related factors. This holistic view can lead to improved patient outcomes and may involve interdisciplinary collaboration among cardiologists, dietitians, and primary care providers. This observation underscores the necessity for effective lipid management strategies in individuals at risk for ST-segment elevation myocardial infarction (STEMI). The research also identified positive correlations between body mass index (BMI) and levels of LDL, WBC, and h-CRP, while a negative correlation was observed between BMI and C3 levels. These associations suggest that obesity may intensify inflammatory responses and lipid imbalances, thereby complicating the clinical management of STEMI patients. The results emphasize the importance of a comprehensive approach to STEMI management, which should encompass both inflammatory and lipid-related factors. Such an approach could lead to the development of enhanced therapeutic strategies aimed at minimizing myocardial damage and promoting recovery. Overall, the study advocates for further investigation into the roles of these biomarkers in STEMI to facilitate the development of targeted interventions that could improve patient outcomes.

Medicinal plants with cardiac tonic properties can significantly contribute to cardiovascular health, prevent heart diseases, and mitigate the side effects of chemical

drugs, offering a natural and effective treatment option. By integrating traditional knowledge with modern scientific advancements, we can develop innovative therapeutic approaches and reduce the incidence of heart diseases. Continued comprehensive and systematic research is essential to fully harness the potential of these natural resources.

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Conflict of interests

No conflict of interest

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