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Investigation of the Association between Chemerin, Resistin, and Selected Cardiac Enzymes in Patients with Acute Myocardial Infarction

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ABSTRACT

Background & Objective: Myocardial infarction (MI), commonly known as a heart attack, refers to the irreversible necrosis of cardiac muscle cells due to insufficient oxygen delivery resulting from reduced or obstructed coronary blood flow. Severe myocardial cell loss is a major cause of mortality in MI patients. Inflammatory markers such as chemerin and resistin, alongside cardiac enzymes, may play a key role in MI diagnosis and monitoring. This study aimed to evaluate the clinical significance of inflammatory markers (chemerin and resistin) and cardiac enzymes, including lactate dehydrogenase (LDH), aspartate aminotransferase (AST), creatine kinase-MB (CK-MB), and cardiac troponin I, by assessing their serum levels in patients with myocardial infarction compared to healthy controls.

Materials & Methods: A total of 120 subjects participated in this study. Sixty were MI patient's diagnosed based on clinical presentation, symptoms, and electrocardiogram (ECG) findings, with a mean age of 59.31 ± 1.64 years. The remaining 60 were healthy control subjects with a mean age of 37.30 ± 2.10 years. The study was conducted in the Cardiac Care Unit of Baqubah Teaching Hospital, Diyala Governorate, Iraq. Serum levels of chemerin, resistin, troponin I, LDH, CK-MB, and AST were measured and statistically analyzed.

Results: This study was conducted in Cardiac Care Unit of Baqubah Teaching Hospital in Diyala governorate, Iraq. The study revealed a significant decrease ($P < 0.001$) in the serum levels of chemerin and resistin among MI patients compared to controls. Conversely, there was a significant increase ($P < 0.001$) in troponin I, LDH, CK-MB, and AST levels. Additionally, overweight MI patients exhibited significantly higher levels ($P < 0.001$) of troponin I, chemerin, and resistin compared with normal-weight MI patients.

Conclusion: Chemerin and resistin levels significantly decreased following admission and treatment in MI patients, suggesting that these inflammatory markers may serve as potential indicators of therapeutic response in myocardial infarction management.

Keywords: Chemerin, Resistin, Myocardial Infarction, Electrocardiogram

1. Introduction

Myocardial infarction occurs when blood flow to the heart muscle is reduced or completely blocked, resulting in damage to the cardiac tissue (1). Statistics indicate that approximately 10% of patients presenting to emergency departments with chest pain are ultimately diagnosed with a heart attack (2). MI typically develops following the rupture of an atherosclerotic plaque, which leads to thrombus formation and causes coronary artery disease or ischemic heart disease. A blockage either partial or complete within the coronary artery restricts blood flow to the myocardium (3, 4). Clinically, MI is diagnosed based on elevated cardiac biomarkers, electrocardiographic abnormalities, or imaging evidence consistent with myocardial ischemia (5). Myocardial necrosis occurs when the interruption of blood flow leads to irreversible damage of cardiac muscle cells (6). ST-segment elevation myocardial infarction (STEMI) accounts for roughly 40% of all MI cases. Various diagnostic methods are used by clinicians, including

electrocardiography (ECG), angiography, and chest X-rays. Following myocardial injury, several cardiac biomarkers become detectable in the bloodstream, such as cardiac troponin I (TnI), lactate dehydrogenase, creatine kinase-MB, aspartate aminotransferase, and other inflammatory markers (7).

The event triggers both a systemic inflammatory response and local tissue degradation at the necrotic site, eventually leading to scar formation. The typical clinical presentation involves sudden, intense, and persistent chest pain that may radiate to the shoulder or arm (8). MI is associated with impaired blood flow to the heart wall, increased enzyme levels, and elevated biological markers indicating cardiac stress or injury. When myocytes are damaged, they release these biomarkers into the bloodstream, which is of diagnostic and therapeutic importance (9).

Several biomarkers are valuable in identifying cardiovascular diseases. Among the earliest markers of myocardial necrosis are LDH, AST, CK-MB, and cardiac troponin I (10). Resistin, a 12.5 kDa polypeptide hormone composed of 114 amino acids, belongs to the resistin-like molecule family (11). Initially identified as a hormone secreted by adipocytes, resistin was found to induce insulin resistance in animal studies (12). These findings support the hypothesis that resistin may play a role in the development of diabetes and obesity-related insulin resistance. However, due to significant interspecies differences in structure and biochemistry, further research is required to clarify resistin's role in human insulin sensitivity and obesity (11). In vitro studies have shown that resistin stimulates human endothelial cells, increasing the number of human aortic muscle cells and the expression of adhesion molecules (13, 14).

This suggests that resistin may promote atherosclerosis, thereby linking it to human cardiovascular disease (CVD) (15). Evidence from cross-sectional and case-control studies indicates that patients with coronary heart disease (CHD) exhibit higher plasma resistin levels than healthy controls (16, 17). These findings imply that resistin contributes to the progression of atherosclerosis and may be associated with CVD in humans (18-20).

Adipocytokine chmerin (16kDa) is made up of 137 amino acid sequences (21); the liver, adipose tissue, and circulatory system produce the majority of its receptors (22). The protein chemerin is essential for numerous physiological functions, including maturation, adipose differentiation, immune system modulation, inflammation and insulin resistance, as well as metabolism (21). Studies have demonstrated a connection between chemerin and obesity, metabolic syndrome, and diabetes (18-22). Chemerin has been linked in the past to a number of CVD, such as the onset of hypertension and the formation of atherosclerotic plaques (20), Patients with dilated cardiomyopathy have a reduced cardiac output (20).

2. Materials and Methods

The study was conducted in the intensive cardiac care unit of Baqubah Teaching Hospital in Diyala governorate/ Iraq. The study included 120 people, 60 with myocardial infarction with an average age of (59.3±1.64) years, and 60 healthy subjects with an average age of (47.30±2.10) years. The director of Diyala Teaching Hospital approved the study to be conducted, and the patients agreed to have blood drawn from them to conduct the study. A careful history was obtained and an appropriate clinical examination was performed. Blood samples were collected from each subject by drawing (5ml) of venous blood into test tube with a gel material and left for half an hour at a temperature of 22 ◦C, after which the samples were centrifuged and serum for each sample was divided into two parts: first part used to estimation cardiac enzymes LDH, AST, CK-MB and troponin I, while the second part was stored in small Eppendorf tubes at -20 ◦C for a quantitative measurement of chemerin and resistin.

2.1 Enzyme measurements

The effectiveness of cardiac enzymes was estimated using kits prepared from Roche, Germany, and using an automatic device Cobas C311 of Roche company, Germany. Which works on serum and plasma.

2.2 Estimation of chemerin and resistin

The enzyme linked immunosorbent assay (ELISA), according to the manufacturer's examination kit (Shanghai), was used to test the levels of resistin and chemerin in the blood of patients who experienced a myocardial infarction, as well as the control group. This methodology is based on the antibody sandwich ELISA method.

2.3 Statistical Data

Statistical Package for Social Sciences (SPSS) version 24 and Microsoft Office Excel 2010 were used in the collection, compilation, analysis, and presentation of the data. One-way analysis of variance, or ANOVA, was used to quantify the variation in the mean of numerical variables among more than four. The level of significance was established by using a p-value of less than 0.05. A significant degree of significance was shown for a p-value less than 0.001. Using Pearson correlation analysis, the degree of linking between the variables was determined, and the results were expressed using the correlation coefficient and significant level. The Receiver Operator Characteristic (ROC) curve analysis was used to determine the cutoff value that denotes a successful outcome. We considered area under the curve (AUC), accuracy level, sensitivity, specificity, and significance level.

3. Result

3.1 Characterization of samples under current study

Table 1 illustrate the general anthropometric characteristics in total study samples which include (sex,

age, weight, length, and BMI). The total samples were (120) divided into two groups, first one was control group, include (60) healthy individual, second group included (60) patients with myocardial infraction admitted into cardiac care unit.

3.2 Comparison for AST, LDH, CKMB, and Troponin in study groups

AST, LDH, CKMB and Troponin increased significantly ($p<0.05$) in patients (135.18±19.17, 592.95±54.57, 1319.96±193.25 and 38664.68±15634.70 respectively) when compared with control group (23.46±1.27, 175.23±4.47, 15.30±1.17 and 2.07±0.22 respectively) as showed in Figure 1, Figure 2 and Table 2.

3.3 Comparison of Resistin (RETN), and Chemerin (CHEM) in study groups

The RETN and CHEM decrease significantly in patients (267.16±34.32 and 507.84±34.57) when compared with RETN and CHEM in control group (723.79±86.37 and 1316.86±189.17) with p-value<0.05 as shown in Figure 3 and Table 3.

3.4 Body Mass Index (BMI)

BMI has been calculated according to a specific formula which includes weight divided by the square of height.

BMI=Weight (Kg)/Square height $(m)^2$

The weight status divided according to the value of BMI is shown in Table 4 (23).

3.5 Comparative Troponin, RETN, and CHEM in patients according to BMI

The troponin, RETN, and CHEM increased significantly in patients with BMI less than 25kg/m2 (24122.70±3197.17, 174.70±36.36 and 447.24±19.20 respectively) when compared to patients with BMI more than 25kg/m2 (49051.80 ± 2672.26, 333.20 ± 50.29 and 551.13±56.90 respectively) p-value<0.05 as showed in Figure 4, Table 5.

Table 1. Characterization of healthy and myocardial infraction patients.

Sex			
Control	Men	Number = 30	Percentage (50 %)
	Women	Number = 30	Percentage (50 %)
Myocardial infraction patients	Men	Number = 41	Percentage (68 %)
	Women	Number = 19	Percentage (32 %)
Age (year) Mean ± SE			
Control		47.30 ± 2.10	
Myocardial infraction patients		59.31 ± 1.64	
Weight Mean ± SE			
Control		71.90 ± 1.87	
Myocardial infraction patients		78.18 ± 2.04	
Length Mean ± SE			
Control		165.60 ± 1.65	
Myocardial infraction patients		171.80 ± 1.60	
BMI Mean ± SE			
Control		25.58 ± 0.29	
Myocardial infraction patients		26.25 ± 0.68	

Table 2. Comparison of AST, LDH, CKMB and Troponin levels in study groups.

Parameters	Groups	Mean ± SE	T-test P. Value
AST U/L	Control	23.46 ± 1.27	0.001
	Patients	135.18 ± 19.17***	
LDH U\L	Control	175.23 ± 4.47	0.001
	Patients	592.95 ± 54.57***	
CKMB U\L	Control	15.30 ± 1.17	0.001
	Patients	1319.96±193.25***	
Troponin ng\L	Control	2.07 ± 0.22	0.001
	Patients	38664.68 ± 15634.70***	

*p-value <0.05, **p-value <0.01, ***p-value <0.001

Table 3. Comparison of RETN and CHEM levels in studied groups.

Parameters	Groups	Mean ± SE	T-test P-Value
RETN pg\L	Control	267.16 ± 34.32	0.001
	Patients	723.79 ± 86.37***	
CHEM pg\L	Control	507.84 ± 34.57	0.001
	Patients	1316.86 ±189.17***	

Table 4. BMI Weight statues categories.

BMI range – Kg/ m^2	Weight Status
18 or less	Under weight
18.5 to 25	Normal weight
25 to 30	Over weight
30 to 35	Obesity (Class 1)
35 to 40	Obesity (Class 2)
40 or great	Morbid obesity

Table 5. Comparison Troponin, RETN, and CHEM in patients according to BMI.

Parameters	Groups	Mean ± SE	T-test P-Value
Troponin ng\L	BMI less 25	24122.70 ± 3197.17	0.001
	BMI more 25	49051.80 ± 2672.26**	
RETN pg\L	BMI less 25	174.70 ± 36.36	0.021
	BMI more 25	333.20 ± 50.29 *	
CHEM pg\L	BMI less 25	447.24 ± 19.20	0.043
	BMI more 25	551.13 ± 56.90 *	

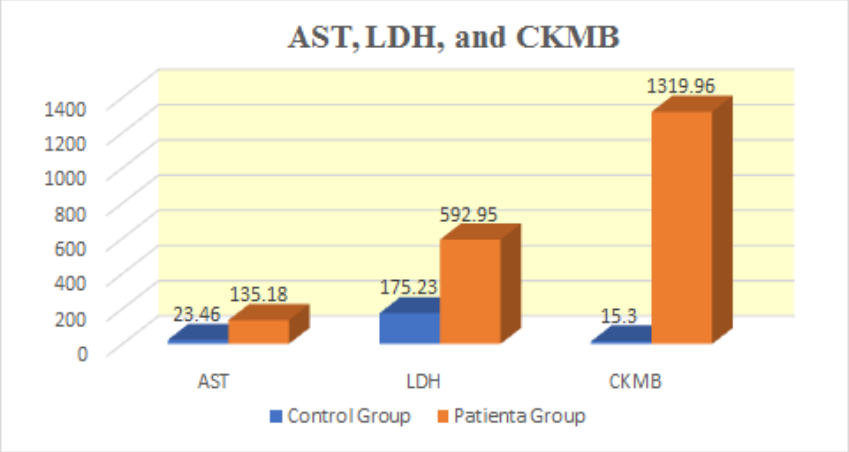

Figure 1. AST, LDH, and CKMB in study groups (**Prepared by Authors, 2025**).

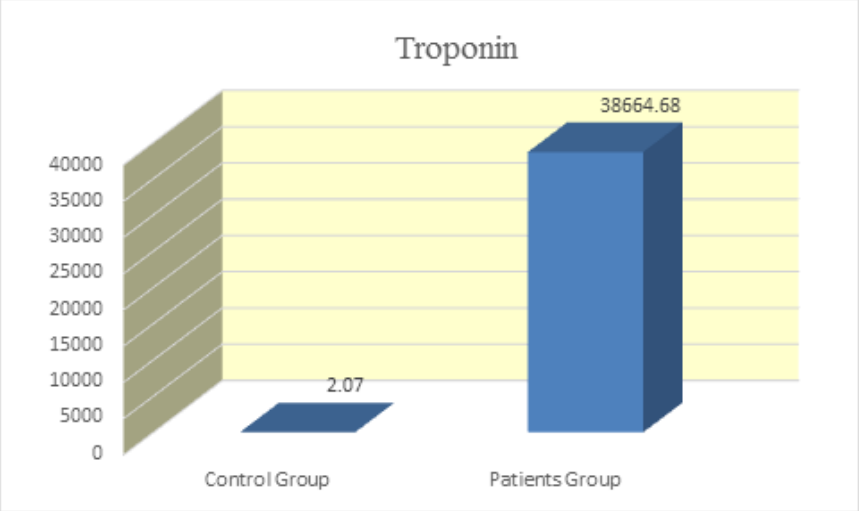

Figure 2. Troponin in study groups (**Prepared by Authors, 2025**).

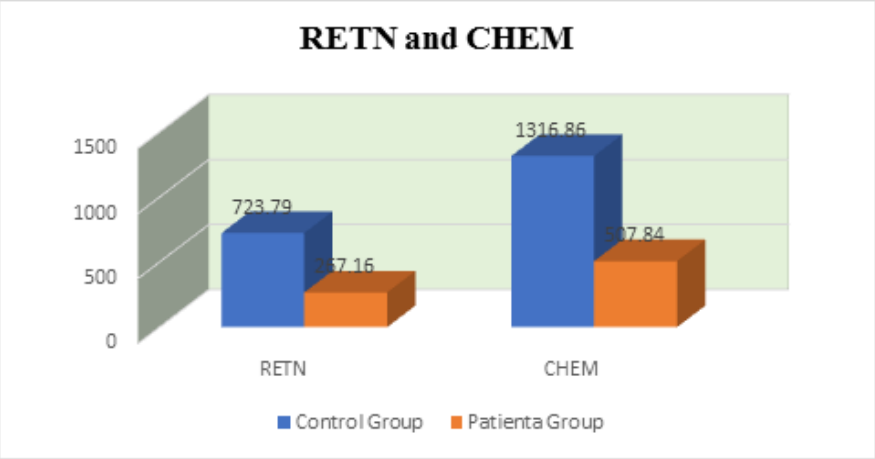

Figure 3. Levels of RETN and CHEM in study groups (**Prepared by Authors, 2025**).

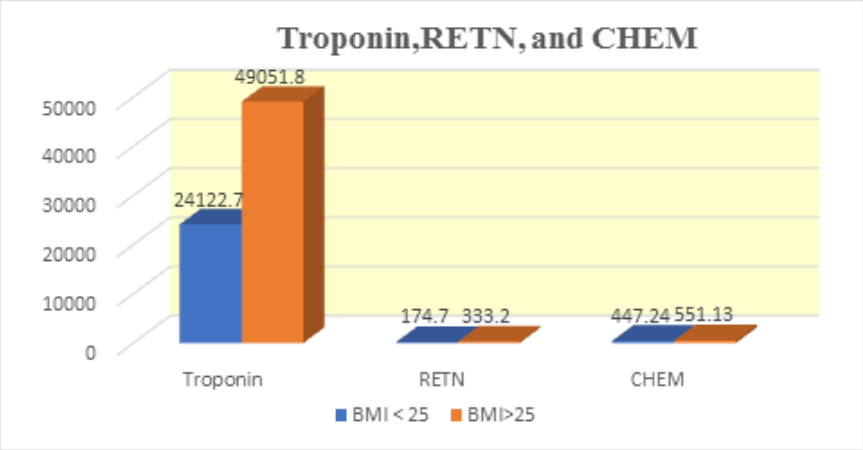

Figure 4. Troponin, RETN, and CHEM in patient groups (**Prepared by Authors, 2025**).

4. Discussions

The present findings showed a highly significant difference ($P < 0.001$) in aspartate transaminase levels between myocardial infarction patients and healthy individuals. The mean values were (135.18 ± 19.17) and (23.46 ± 1.27), respectively. These outcomes are consistent with Saleem's study, which reported a very significant elevation ($P < 0.001$) in the activities of AST and creatine kinase among patients with various cardiac disorders compared to healthy controls **(11)**.

Aspartate transaminase (AST), also known as glutamate oxaloacetate transaminase (GOT), is an enzyme broadly distributed across heart tissue, skeletal muscles, red blood cells, the liver, and kidneys. It catalyzes the reversible conversion of α-ketoacids to amino acids **(12)**. Although found in several organs, AST is considered less specific for liver disease due to its wide tissue distribution **(20)**.

The study revealed a highly significant increase ($P<0.001$) in lactate dehydrogenase (LDH) levels among MI patients compared to healthy controls, with mean values of (592.95 ± 54.57) and (175.23 ± 4.47), respectively. These findings are consistent with those of Shamil **(10)**, who observed a notable rise ($P \leq 0.01$) in serum LDH activity in MI and unstable angina patients compared with healthy individuals **(10)**. Similarly, studies by Zahid et al **(20)** and Bodor **(21)** identified LDH as one of the most frequently used enzymes for detecting myocardial infarction. The development of cardiovascular disease is primarily linked to endothelial dysfunction, which triggers atherosclerosis and ultimately leads to cardiac damage. This injury causes the release of intracellular myocardial enzymes into the bloodstream, resulting in elevated plasma enzyme concentrations **(20, 21)**. The current findings also align with those of Khalil et al **(19)**, and Allwsh and Aziz **(24)** who reported a significant rise ($P \leq 0.01$) in LDH activity among MI patients. The elevation is attributed to myocardial cell damage, which releases LDH into circulation. Serum LDH levels typically begin to increase 4–12 hours after myocardial injury, peak around 48 hours, and serve as an important diagnostic marker for MI **(19, 24)**.

The current results demonstrated a highly significant difference ($P < 0.001$) in creatine kinase-MB (CK-MB)

levels between patients and healthy individuals, with mean values of (1319.96 ± 193.25) and (15.30 ± 1.17), respectively. These findings are consistent with Shamil's results, which showed a significant increase ($P \leq 0.01$) in serum CK activity among MI patients compared to unstable angina and healthy subjects (19).

Brewster's study further explored the role of creatine kinase, an enzyme responsible for ATP regeneration, and found that CK activity is an important predictor of blood pressure. Elevated CK levels may be associated with sodium retention and other physiological factors that contribute to hypertension. Increased CK activity in the serum is commonly observed in cardiac and skeletal muscle disorders, making it a highly sensitive biomarker for acute myocardial infarction (AMI) and muscle injury (18).

The results of the study revealed a highly significant increase ($P< 0.001$) in troponin T (TnT) levels among patients compared to the healthy control group. The mean TnT concentrations were (38664.68 ± 15634.70) in patients and (2.07 ± 0.22) in healthy individuals. This marked elevation in TnT levels among patients indicates a strong association between myocardial infarction (MI) and increased troponin T concentrations. Cardiac troponin type (T, I) has advantages that are balanced by the rest of the cardiac indicators, because the troponin level in unaffected people is very low, and therefore a noticeable increase in troponin indicates cardiac muscle injury, and an increase in the troponin level also appears in cases of irregularity of heart. Heart palpitations and high blood pressure in patients with chronic kidney failure they are at increased risk of cardiovascular disease (25). These, agreed with results of Roffi et al (26) Amsterdam et al (27) studies which refers to considered (Tn-I) and (Tn-T) as a vital sign to detect a heart muscle injury and MI (26, 27). Infarct size can be estimated by troponin, indicating that cardiac troponin (T) measured in acute coronary syndrome (ACS) is more sensitive and indicative of myocardial infarction (MI) (28).

The present study found a highly significant difference ($P < 0.001$) in resistin (RETN) levels between MI patients and healthy individuals, with mean values of (723.79 ± 86.37) and (267.16 ± 34.32), respectively. These findings are consistent with Dera et al (29) who reported that plasma RETN concentrations significantly increase within the first week after the onset of acute myocardial infarction symptoms, suggesting that RETN can serve as a prognostic biomarker for AMI (29). Additionally, higher RETN levels have been observed in patients with cardiovascular disease (CVD). Resistin contributes to CVD progression by promoting inflammation, endothelial cell dysfunction, and smooth muscle cell apoptosis. Therefore, reducing serum RETN concentrations may represent a novel therapeutic approach due to its clinical significance in cardiovascular pathology (30).

The study findings revealed a highly significant elevation ($P < 0.001$) in CHEM levels among patients compared to healthy individuals, with mean values of (1316.86 ± 189.17) and (507.84 ± 34.57), respectively. Elevated CHEM concentrations are considered an independent predictor of coronary artery disease. Increased plasma CHEM levels were observed in CAD patients and were linked to a greater risk of major adverse cardiovascular events (31). As shown in Table 5, there were significant positive correlations between RETN and CHEM, triglycerides, very low-density lipoprotein cholesterol, and CK-MB among the patient group. Conversely, HDL-C demonstrated a significant negative correlation with RETN. Additionally, CHEM exhibited a significant positive correlation with total cholesterol (23).

Table 5 indicates that there is a highly significant difference in the Troponin I with a probability level of $P<0.001$ when comparing patients with myocardial infarction with a body mass index more than 25 and less than 25. These results are in agreement with the results of Abdullah et al., which the higher BMI was positively correlated with in-hospital mortality in patients with acute STEMI. BMI is an important predictor of death in AMI. Excess weight should be avoided as possible in those patients (31). High Troponin I in ST-segment elevation myocardial infarction permits early identification of patients at increased risk of major cardiac complications and death (32). These results are in agreement with the results of Piestrzeniewicz et al (33) study. In patients with AMI, obesity is positively related to the blood resistin concentration. Resistin is likely to play a major role in the atherogenesis and its complications (33).

The serum results of chemerin in our study are in agreement with Baig et al (32) results which significantly correlated with weight (BMI) in their patients. Chemerin levels in another study were slightly higher in the group of patients with atherosclerotic disease with a BMI greater than 30 kg/m² compared to those with a BMI less than 25 kg/m² (34). Obese people are more likely to suffer from myocardial infarction due to excess body fat. Resistin and chemerin are increased in people suffering from obesity and people suffering from myocardial infarction, as shown in Table 5.

5. Conclusion

The findings of this study revealed that the serum levels of chemerin and resistin were significantly elevated in myocardial infarction patients compared to healthy individuals. These elevations were associated with changes in several other biochemical parameters, suggesting that CHEM and RETN may serve as potential biomarkers for assessing the severity and progression of myocardial infarction. Moreover, significant increases were observed in the levels of troponin I, lactate dehydrogenase, creatine kinase-MB, and aspartate aminotransferase within 24–72 hours after patients were admitted to the CCU, confirming their diagnostic importance during the acute phase of MI. Additionally, myocardial infarction patients with a body mass index greater than 25 kg/m² exhibited higher concentrations of troponin, resistin, and chemerin compared to those with lower BMI values, indicating a possible association

between obesity and enhanced inflammatory as well as cardiac injury responses. In conclusion, the combined evaluation of CHEM, RETN, and conventional cardiac enzymes may provide a more comprehensive approach for monitoring disease progression and assessing cardiovascular risk in myocardial infarction patients.

6. Declarations

6.1 Acknowledgments

The authors would like to express their sincere gratitude to the doctors and nurses at Baquba General Hospital for their valuable assistance during sample collection, as well as to all the patients who kindly participated in this study.

6.2 Ethical Considerations

This study was approved by the Ethics Committee of the University of Diyala, Baquba, Iraq (24/09/16/5034).

6.3 Authors' Contributions

Conceptualization, methodology, formal analysis, investigation, data curation, writing-original draft, AAS, writing-review and editing, A.A.S. and K.S.S.; Supervision, K.S.S. All authors reviewed, edited, and approved the final version of the manuscript.

6.4 Conflict of Interest

The authors declare no conflict of interest.

6.5 Fund or Financial Support

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

6.6 Using Artificial Intelligence Tools (AI Tools)

The authors were not utilized AI Tools.

7. Publisher's Note

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