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Chemerin, Resistin, and Various Biochemical Markers in the Serum of Individuals Affected by Myocardial Infarction

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Abstract

Myocardial infarction (MI) is a widespread disease that may lead to death. Atherosclerosis is one of the main causes of a heart attack. Due to the narrowing of the arteries, it leads to the aggregation of LDL. As a result of this aggregation, a clot is formed. This clot leads to a complete or partial blockage of these arteries, followed by the death of a section of the heart muscle. Then, ischemic heart disease occurs because the heart's need for oxygen is greater than the amount it receives. The study aimed to discover the relationship between the MI and alteration of chemerin, resistin and some myocardial biomarkers by evaluating this biomarkers in MI patients and to compare them with those of controls. The parameters which evaluated in study were cardiac enzymes including lactate dehydrogenase (LDH), aspartate aminotransferase (AST), creatine kinase MB (CK-MB), troponin I (TnI) and lipid profile (cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C), and very low density lipoprotein (VLDL-C)) in addition to chemerin and resistin. This study was conducted in Baqubah Teaching Hospital / Diyala Governorate from Jan/2023 until May /2023, Included the collection of samples were collected from patients lying in the cardiac care unit (CCU) who had suffered from myocardial infarction (MI). The

study included 60 patients with an average age of (59.31±1.64) years, and the patients were compared with 60 healthy people as a control group, with an average age of (54.86±0.88) years. The result showed an increase in the levels of chemerin, resistin, TnI, LDH, AST and CK-MB significantly in patients of MI after 24-72hrs of entering attack. The ievels of lipid profile parameters were not changed significantly in patients of MI after admitted to Cardiac Care Unit (CCU) and this may due to receive of Hyperlipidemia drug. A correlation in a significant positive manner between resistin with chemerin, TG, VLDL-C, and CK-MB was observed, while HDL-C showed significant negative correlation with resistin in the patients group. Chemerin also has a significant positive correlation with TC in. No conclusion in the abstract the Levels of chemerin and resistin were observed to be significantly higher in patients compared to healthy controls, but the increase in chemerin and resistin was associated with changes in other parameters studied in patients. Therefore, the significant increase in chemerin and resistin may be used as new factors in monitoring or to indicate the extent of MI patients.

Keywords: Myocardial infarction, chemerin, resistin, myocardial biomarkers, lipid profile.

Introduction

Myocardial infarction (MI), an acute ischemia of the heart muscle caused by a lack of or restriction of coronary blood flow, damages heart muscle tissue [1]. The "right and left" main coronary arteries anatomically supply the heart muscle with blood. A small branch at the end of each of them transports nutrients from the side of the heart to the myocardium. The two primary terminal branches of the left coronary artery are the left circumflex artery and the left anterior descending "anterior interventricular" artery. The tiny terminal branches of the coronary arteries anastomose, but if one of them is obstructed, this anastomosis is insufficient [2]. Evokes a systemic inflammatory response and locally the degradation of the necrotic tissue, followed by scar formation, with the classic symptoms of sudden, severe, and persistent pain in the back of the chest (accompanied by pain radiating to the shoulder and sometimes the arm) [3]. Myocardial ischemia may occur either from increased demand of oxygen by the myocardium, or decreased oxygen supply to the myocardium, or both[4]. MI is myocardial necrosis occurs when blood flow stops to a fragment of the heart leading to the damage of the cardiac muscle [5]. ST-segment elevation myocardial infarction (STEMI) make up about 40%

of MI. Doctors use many methods to diagnose MI, such as (ECG), Angiography, chest x-ray and others. After myocardial injury many cardiac biomarker become detectable into venous circulation such as cardiac enzymes TnI, LDH, CK-MB, AST, and many other inflammatory markers [6].

The hormone resistin has a molecular weight of 12.5 kDa and 114 amino acids. It shares a chemical family with resistin [7]. Insulin resistance in animal studies is assumed to originate from resistin, which is produced by adipocytes [8], the results validated the hypothesis that this hormone could have a role in the development of diabetes and the fat-related insulin resistance. Even though the structure and biochemistry of reaction differ significantly between species, more investigation is needed to understand how reaction influences human insulin sensitivity and how obesity is connected to it [7]. Human aortic muscle cell numbers and adhesion molecule expression rose in response to resistin-stimulated human endothelial cells in vitro [9-10]. Resistin may promote atherosclerosis, indicating a potential connection between resistin and human cardiovascular disease (CVD) [11]. Patients with coronary heart disease (CHD) exhibited greater plasma resistin levels than controls, according to research integrating cross-sectional and case-control data [12-13].

Adipocytokine chmerin (16kDa) is made up of 137 amino acid sequences [14]; the liver, adipose tissue, and circulatory system produce the majority of its receptors [15]. The protein chemerin is essential for numerous physiological functions, including maturation, adipose differentiation, immune system modulation, inflammation and insulin resistance, as well as metabolism [16]. Studies have demonstrated a connection between chemerin and obesity, metabolic syndrome, and diabetes [17-18]. Chemerin has been linked in the past to a number of CVD, such as the onset of hypertension and the formation of atherosclerotic plaques [19], Patients with dilated cardiomyopathy have a reduced cardiac output [15].

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) 0f them are having myocardial infarction with an average age of (59.3±1.64) years, and (60) healthy subjects with an average age of (54.30±2.10) years were recruited into this 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 a 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 of each sample was divided into two parts: first part used to estimate the cardiac enzymes LDH, AST, CK-MB, troponin I, cholesterol, TG and HDL, while the second part was stored in small eppendorf tube at -20 ◦C for a quantitative measurement of chemerin and resistin.

Study 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.

Lipid profile tests (cholesterol, TG and HDL) were assessed using an automated chemical analyzer Cobase411 of Roche company, Germany. LDL is calculated according to the equation: LDL-C = [Chol] - [HDLDirect]-{[TG]/5}, While VLDL-C was calculated by using formula: VLDL-C ={[TG]/5}.

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 MI, as well as the control group. This methodology is based on the antibody sandwich ELISA method.

The body mass index (BMI) as parameter for each participant was calculated by dividing weight in kg on height in m^2.

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.

Results

Table 1 displays the mean ± standard deviation (M±SD) of age and BMI for the patients and healthy controls group. The patients' age and BMI were matched to that of the healthy controls group.

Table 1: The comparison of M±SD for Age and BMI of patients with healthy controls

Parameter	M±SD of control (n=60)	M±SD of Patients (n=60)	*P*-value
Age (years)	54.86 ± 0.88	59.31 ± 1.64	0.068
BMI (Kg/m²)	25.58±0.29	26.25±0.68	0.292

Comparison levels of lipid profile in studied groups

The levels of lipid profile which include (TC), (TG), (HDL-C), (LDL-C), and (VLDL-C) were changed non-significantly ($p>0.05$) in patients compared with healthy controls, as shown in Table 2 and Figure 1.

Table 2: The comparison of lipid profile of patients with healthy controls

Parameters	Groups	Mean ± SE	*P*. Value
TC mg\dL	Control	171.40 ± 6.26	0.098
	Patients	175.40 ± 5.15	
TG mg\dL	Control	128.46 ± 5.49	0.077
	Patients	147.28 ± 6.88	
HDL-C mg\dL	Control	50.80 ± 1.63	0.064
	Patients	40.56 ± 1.13	
LDL-C mg\dL	Control	99.73 ± 2.83	0.092
	Patients	104.95 ± 5.14	
VLDL-C mg\dL	Control	25.66 ± 1.09	0.270
	Patients	29.46 ± 1.36	

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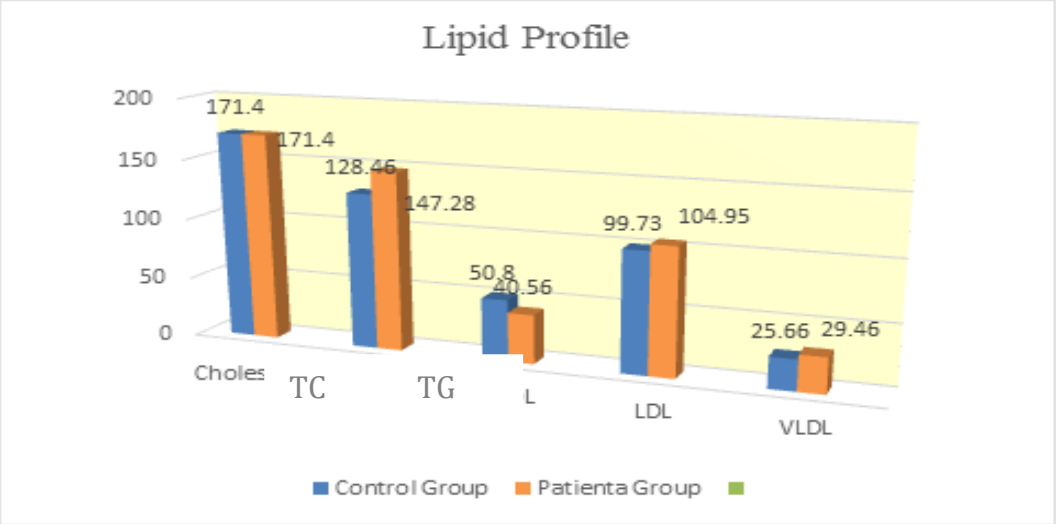

Figure 1: Lipid profile comparison in study groups

Comparison of AST, LDH, CK-MB and Troponin I levels in study groups

Table 3 the M±SD values obtained for AST, LDH, CK-MB and TnI for patients in comparison with healthy controls group. Statistically, patients of MI had significantly higher levels of AST, LDH, CK-MB and TnI than control group (p<0.05). As shown in Figure 2 and Figure 3.

Table 3: Comparison of AST, LDH, CK-MB and TnI for patients with controls

Parameters	Groups	Mean ± SE	*P*. Value
AST U/L	Control	23.46 ± 1.27	0.000
	Patients	135.18 ± 19.17	
LDH U\L	Control	175.23 ± 4.47	0.000
	Patients	592.95 ± 54.57	
CKMB U\L	Control	15.30 ± 1.17	0.000
	Patients	1319.96±193.25	
Troponin ng\L	Control	2.07 ± 0.22	0.000
	Patients	38664.68 ± 15634.70	

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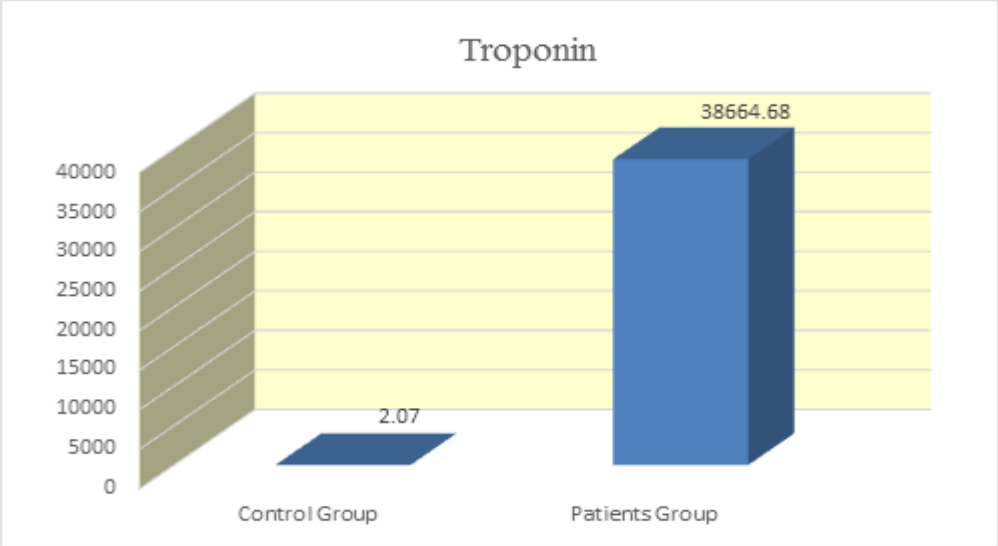

Figure 2: Comparison of Troponin in study groups

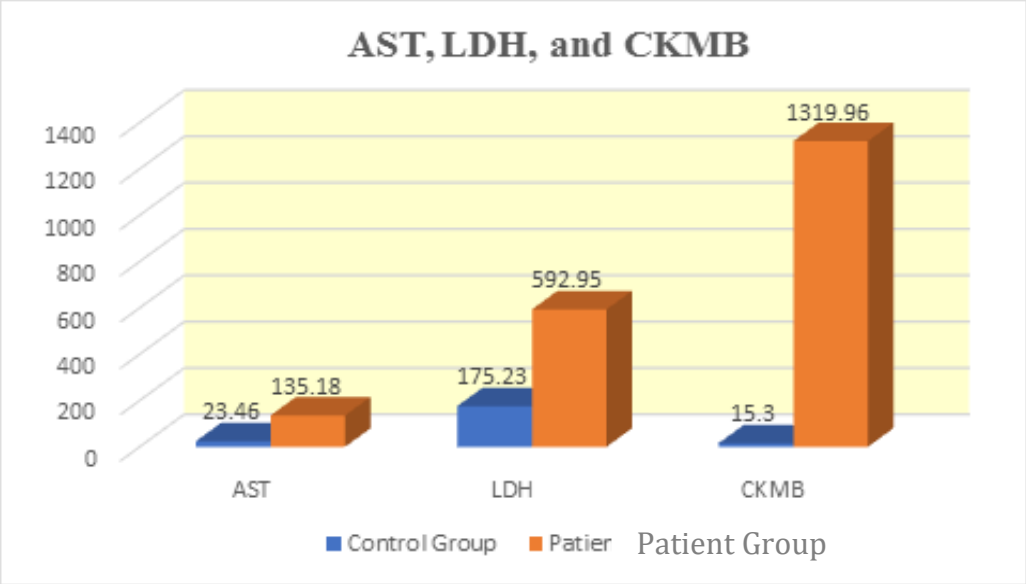

Figure 3: AST, LDH, and CKMB in study groups

Comparison of (RETN), and (CHEM) in study groups

The levels of RETN and CHEM ware increased significantly (p<0.05) of patients in comparison to healthy controls as shown in Table 4 below.

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Table 4: The comparison of RETN and CHEM of patients with healthy controls

Parameters	Groups	Mean ± SE	*P*-Value
RETN pg\L	Control	267.16 ± 34.32	0.000
	Patients	723.79 ± 86.37	
CHEM pg\L	Control	507.84 ± 34. 757	0.000
	Patients	1316.86 ± 189.1	

Correlations

The level of RETN showed positive significant correlation (p<0.05) with other studied parameters in patients of MI including TG, VLDL and K-MB, while HDL showed negative significant correlation (p<0.05) as shown in Table 5. Also as shown in Table 5 below, CHEM td demonstrated a positive significant correlation (p<0.05) with cholesterol and RETN.

Table 5: The correlation of CHEM and RETN with other studied parameters in patients

Parameters		RETN	CHEM
TC	Pearson Correlation	0.080	0.041*
	Sig. (2-tailed)	0.541	0.757
TG	Pearson Correlation	0.032*	0.099
	Sig. (2-tailed)	0.808	0.452
HDL-C	Pearson Correlation	-0.050*	-0.103
	Sig. (2-tailed)	0.704	0.435
LDL-C	Pearson Correlation	0.104	0.057
	Sig. (2-tailed)	0.427	0.663
VLDL-C	Pearson Correlation	0.034*	0.094
	Sig. (2-tailed)	0.798	0.477
AST	Pearson Correlation	0.054	-0.165
	Sig. (2-tailed)	0.732	0.209
LDH	Pearson Correlation	0.083	-0.165
	Sig. (2-tailed)	0.529	0.209
CK-MB	Pearson Correlation	0.047*	-0.127
	Sig. (2-tailed)	0.723	0.335
Troponin I	Pearson Correlation	0.238	-0.055
	Sig. (2-tailed)	0.067	0.679
RETN	Pearson Correlation	1	0.052
	Sig. (2-tailed)	1	0.762*

Discussion

In table (1) there were no significant changes between patients and healthy controls regarding age and (BMI). In the table (2) the results of the study indicated that there was no significant increase (p> 0.05) in the level of (TC) when compared between patients and healthy subjects.

The values of the arithmetic mean for the patients were (175.40±5.15), while the healthy group (171.40±6.26), the TC level of the patients was close to that of the healthy control group may due to lipid-lowering treatment. The results agreed with AL-Azzawy who indicated the high concentration of TC in patients with MI compared with control have increased at the beginning of the injury and then decreased after the condition stabilized and as a result of using hyperlipidemia drug [20]. The results of the current study are consistent with the study of Naresh Kunar, which showed that the level of lipids after acute myocardial infarction (AMI) decreased, includes (TC), (LDL-C) and Slight increase in (TG) , Because of the lipid-lowering treatment that they take in the first hours periodically until a stable health condition is reached[21].

The results of the current study indicate that there is no significant increase (P>0.05) in the level of (TG) in patients compare with healthy subjects, and the arithmetic mean values were (147.28±6.88), (128.46±5.49), respectively. This study not agreed with Velho study who indicate that concentration of TG were higher significantly in patients with CVD and considered as risk factor for heart and vascular diseases[22].

The results showed that there were non-significant decrease in the levels of HDL-C (P>0.05) when compared between the patients and healthy group and the arithmetic mean values were (40.56±1.13), (50.80±1.63), respectively. This study agrees with Velho study who indicate that concentration of HDL-C was lower in patients with CVD and decreases level of HDL-C considered as risk factor for heart and vascular diseases and CVD [22]. Studies indicated that decrease levels of HDL-C is associated as risk factor for IHD, CHD [23].

The results showed that there was no significant elevation in the level of LDL-C (P>0.05) when compared between the patients and healthy group, and the mathematic mean values were (104.95±5.14), (99.73±2.83), respectively. The results of the current study showed a consistent somewhat with the Naresh Kunar study results which showed that the level of lipids after acute myocardial infarction (AMI) includes a decrease in TC, LDL-C, HDL-C and an increase in (TG) [21]. Mentioned that LDL-C is the main cause atherosclerosis. Increase concentration of LDL-C is associated with as increased risk of MI [24].

The results of the study demonstrated non-significant difference (P>0.05) in the level of VLDL-

C when balancing between patients and healthy people, and the arithmetic mean values were (29.46±1.36), (25.66±1.09), respectively. This study not agreed with Allwsh study which showed a significant increase in concentration of VLDL-C in heart diseases patients group (MI) as compared with control group [25].

In the table (3) there was a highly significant difference (P<0.001) in the level of (AST) between the patients and healthy people. The mean values were (135.18±19.17) and (23.46±1.27), respectively. This results agreed with results of Saleem study, which showed a very highly significant increase in the activities of AST and CK-MB (P< 0.001) in different heart diseases when compared with normal healthy subjects [26]. AST is widely distributed in heart tissues, skeletal muscles, red blood cells, liver and kidneys. It is less specific for liver diseases [27].

The study results indicated that there was a very high significant increase (P<0.001) in the level of (LDH) between the patients and healthy group, and the mean values were (592.95±54.57), (175.23±4.47), respectively. This study agrees with Allwsh study who showed a significant increase (P≤0.01) in the activity of LDH enzyme compared with control group, also these results agreed with this of Khalil study which showed increases the activity of LDH with MI and the reason may be due to pathological condition that led to damage of heart cells, causing the release of the enzyme and hence the high activity of LDH enzyme in blood serum. The effectiveness of serum LDH enzyme increases after MI and begins after 4-12 hours after injury and reaches a maximum height after 48 hour and is a useful clinical sign for patients with MI [25,28]. The cardiovascular damage leads to a detectable rise in the plasma concentration of myocardial enzymes normally confined within cardiac cells [29,30].

The results of the current study indicated that there was a highly significant difference (P<0.001) in the level of (CK-MB) between the patients and healthy people. The average values reached (1319.96±193.25) and (15.30±1.17), respectively. These results were in agreement with Shamil study results which showed a significant increase (P≤0.01) in the mean level of serum CK of MI patients compared with unstable angina patients and healthy subjects [31,32].

The results of the study indicated that there was a high significant increase (P>0.001) in the level of TnI between the patients and healthy group, and the average values were (38664.68±15634.70), (2.07±0.22), respectively, and this means that there is a relationship

between MI disease with elevated TnI levels. The agreed with results of (Roffi, *et al.*; Amsterdam, *et al.*) studies which refers to considered (Tn-I) and (Tn-T) as a vital sign to detect a heart muscle injury and MI [33,34]. Cardiac troponin type (T, I) has advantages that are balanced by the rest of the cardiac indicators, because the TnI level in unaffected people is very low, and therefore a noticeable increase in TnI indicates cardiac muscle injury, and an increase in the TnI level also appears in cases of irregularity of heart. Heart palpitations, high blood pressure and in patients with chronic kidney failure they are at increased risk of CVD [35].

In table (4) The results indicated presence a highly significant increase ($P<0.001$) in the RETN level between patients group and healthy people, reaching (723.79±86.37) and (267.16±34.32), respectively. The results agreed with Dear,A.A et al. study results which showed that plasma RETN levels in patients with (AMI) are elevated significantly within the first week after symptoms onset. RETN can be used as a prognostic marker associated with AMI [36]. There were significantly increasing in levels of RETN in (CVD) patients.

RETN has a role in developing CVD by promoting inflammation, vascular endothelial cell dysfunction, and apoptosis in smooth muscle cells. Reducing serum RETN levels could be a new therapeutic target because of its clinical significance in CVD [37].

The results of the study demonstrated existing a highly significant increase ($P<0.001$) in the CHEM level between the group of patients and healthy people, reaching (1316.86±189.17), (507.84±34.57), respectively. High CHEM levels are an independent predictor of coronary artery disease. It was observed that plasma CHEM levels were increased in patients with coronary artery disease and were associated with an increased risk of significant adverse cardiovascular effects in these patients [38].

In table (5) the results indicate that there are positive significant correlations between RETN with CHEM, TG, VLDL-C, and CK-MB, while HDL-C showed negative significant correlation with RETN in the pateints group. CHEM also has a positive significant correlation with TC [38].

Conclusion

The levels of CHME and RETN were found to be significantly higher in patients compared to healthy controls, but the increase in CHEM and RETN was associated with changes in other parameters studied. Therefore, the significant increase in CHEM and RETN may be used as new factors to monitor or to indicate the extent of MI patients. A significant increase have been appeared in the levels of TnI, LDH, CK-MB and AST in patients with MI during a period of 24-72hrs of entering CCU. A decrease in the levels of TC, TG, LDL-C, HDL-C and VLDL-C in patients with MI after entering to CCU and received the treatments.

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