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Original Research Article

Evaluation Serum Levels of Resistin and Interleukins-6 as Predictors of Left Ventricular Remodelling in Patients with Acute ST Elevation Myocardial Infarction

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Abstract: ***Objectives*:** To identify some biochemical parameters that help predict LV remodelling after STEMI. By determining serum levels of Resisten and IL-6 in patients with ST-segment elevation MI (STEMI) and apparently healthy controls. ***Patients & Methods*:** This prospective case-controlled study was conducted from beginning May 2023 to end of October 2023. This study was conducted on 110 individuals, including sixty-two patients (50 males and 12 females) who had undergone a STEMI and forty-eight healthy control subjects. The information about patients in this study was retrieved from patients' hospital records. The samples were collected from Azadi Teaching Hospital/ Kirkuk on patients with STEMI. Forty-eight of apparently healthy sex and age-matched controls were included in this study**.** The data were collected from the cases group as (G2) means: From the moment the patient arrives to internal resuscitation and up to 48 hours and at the baseline of the STEMI (ST elevation of the myocardial fraction), and (G3) means: After 4-6 months follow up, These states were also compared to the control group composed of (48) individuals who were apparently healthy. About five ml of blood was collected from the antecubital vein for both patients and healthy individuals in an anticoagulant tube to estimate the Resisten and IL-6 levels. ***Results:*** The results of the study showed that the serum level of resisten were significantly higher in group G2 (patients group) in comparison to those of the A1 (control group) P <0.01. The current research demonstrated non-significant differences in IL-6 levels between (G1 and G2), while showing highly significant differences between A1 and A3; this was also applicable for G2 and G3. ***Conclusion*:** The current study concluded that there are significantly increasing of Resistin levels in patients in matching to control groups. Also, it was noticed that IL-6 mean concentration levels were lower in A2 in comparison to A1 but higher than in A3.

Keywords: Biochemical Parameters, Resistin and Interleukins-6, Myocardial Infarction, Ventricular Remodelling.

INTRODUCTION

Cardiovascular diseases are a leading cause of mortality in humans, and nearly 20 million individuals worldwide die from acute cardiovascular events every year. Myocardial infarction commonly known as heart attack leads to changes in size, shape, and functions of the heart. These changes are referred to as cardiac remodeling and encompass a vast array of pathophysiological alterations, including electrophysiological changes, ventricular dilatation, myocyte hypertrophy, and interstitial fibrosis [1-4].

Adipose tissue is a complex, essential, and highly active metabolic and endocrine organ [4], that releases many metabolically active peptide hormones, bioactive cytokines, chemokines, and adipokines, including leptin, visfatin, resistin, apelin, omentin, sex steroids, and various growth factors, which are cell signaling proteins [5-7].

Resistin, is a member of the resistin-like molecule hormone family with a 108-amino acid polypeptide, encoded by the RETN gene on chromosome 19p13. Synthesized in adipocytes, pancreatic cells, muscle, and peripheral-blood

mononuclear cells, it is characterised by 10 conserved cysteine residues and a conserved cysteine residue located near the amino terminal end. Resistin is secreted as a disulphide-linked homotrimer and circulates in plasma as such or a hexamer. Resistin is a pro-inflammatory adipocytokine with an important role in the pathogenesis and development of atherosclerosis It also upregulates the expression of other pro-inflammatory cytokines, including TNF-α, IL-6, IL-1β and monocyte chemoattractant protein-1 resistin in an NF-KB signalling dependent mechanism, thus promoting the inflammatory process. Resistin levels are high in patients with ACS, its levels increasing early at 3–6 h after onset making it a potentially useful diagnostic marker [8, 9].

IL-6 is an inflammatory cytokine involved in vascular inflammation, the initiation and progression of atherosclerosis and degradation of fibrous cap contributing to plaque instability. It propagates inflammation in patients with AMI, and its levels at admission are associated with infarct size and cardiac function, making it a predictor of in-hospital prognosis, but also of LVR and long term outcome/mortality. IL-6 levels are increased during the first 2 weeks and reach a steady-state afterward. On the one hand, IL-6 has protective effects—such as myocytes protection against oxidative stress. On the other hand, it has been shown that IL-6 signalling can lead to hypertrophy and depressed cardiac function [10-12].

PATIENTS AND METHODS

This prospective case-controlled study was conducted from beginning May 2023 to end of October 2023. This study was conducted on sixty two individuals including thirty one patients who had undergone a STEM. After 3 months this patients follow-up after STEMI and thirty one healthy control subjects. The information about patients in this study was retrieved from patients hospital records. The samples were collected from Azadi teaching Hospital/ Kirkuk on patients with STEMI. Thirty one of apparently healthy sex and age matched control were included in this study. Apparently healthy sex and age matched control with any autoimmune, inflammatory disease or infection were excluded from this study. The data was collected from the study participants directly and filled in prepared questionnaire. The questionnaire was designed by support of supervisor on previous literatures.

Routine baseline laboratory investigations, in addition to cardiac troponin I (cTnI), galectin-3, Ischeamia modified albumin (IMA), Brain natriuretic peptide (BNP), Resisten, Interleukin 6 (Il-6), Interleukin 1RA (IL-1RA).

Follow-up after STEMI will include a clinical visit and blood sampling for Gal-3, Brain natriuretic peptide (BNP), Interleukin 6 (Il-6), Interleukin 1RA (IL-1RA) measurements at 6 months, when a twodimensional echocardiogram will be again obtained.

A total number of Thirty one patients (males & females) who have STEMI, (Females 19% - Males 81%) were included in the study. There ages were between (40-75) years and BMI range (24-35). The criteria of exclusion include the following: malignant disease, infectious disease, and liver disease.

The results of the patient groups were compared with Thirty one comparable age and sex-matched healthy subjects, with the age ranged between (18-53) years old used as control group, with exclusion criteria that include a history of infection, inflammation, cancer, and liver disease. Control group were clinically diagnosed to be free from symptoms and signs of any diseases including renal disease, liver disease, diabetes, hypertension and malignancy which affect STEMI. Control subjects did not receive any treatment.

About five milliliters of blood were collected from the antecubital vein of the patients and health control have been kept in gel tubes without any anticoagulant at room temperature for 10-15 minutes then allowed to clot. The samples in gel tubes then were centrifuged (3000 rpm) for 15min. The clear serum was pipetted into clear dry Eppendorf's tubes then stored at (-20C^{o}) until used for the various investigations.

The serum was thawed at (20-25C^{o}) temperature for two hours then submitted to the centrifuged for five minutes at 3000 rpm.

RESULTS

The current research demonstrated non-significant differences for IL-6 levels between (G1 and G2), while showed high significant differences between G1 and G3, this was also applicable for G2 and G3, where the highest mean levels of IL-6 (50.7±10.4) was detected in A1 group in comparison to the lowest mean level of IL-6 (36.9±8.8) which was found in G3 group. Table 4.2. Figure 4.4.

It was noticed that IL-6 mean concentration levels was lower in the A2 in comparison to A1, but higher than A3, this showed some disagreement with a study conducted in AL-Qadisiyah city, Iraq, which was reported by Alzamily *et al.*,

[13], who found higher levels of IL-6 in patients in matching to control, these variances may be attributed to the difference in the kinetic of IL-6 response in these patients [14, 15].

Furthermore, our study demonstrated higher levels of Resistin in patients in matching to control group, this was close to what was reported earlier by Wasyanto and , Febrilia [15], who found that Resistin and hs troponin I levels were related in STEMI patients with systolic heart failure. Resistin levels were associated with systolic heart failure in participants in the STEMI group. The levels of hs troponin I were not correlated with systolic heart failure in STEMI patients.

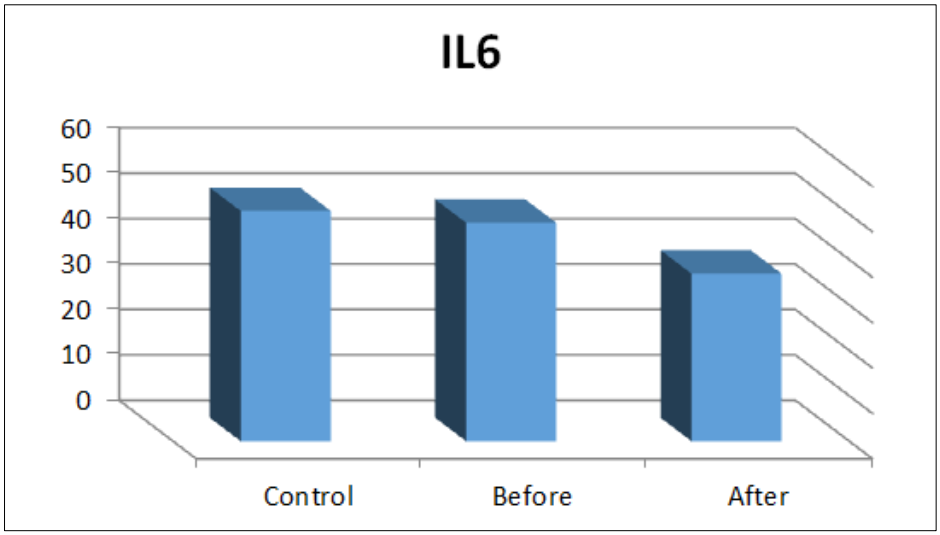

Figure 1: Levels of IL-6 in control group and patients groups

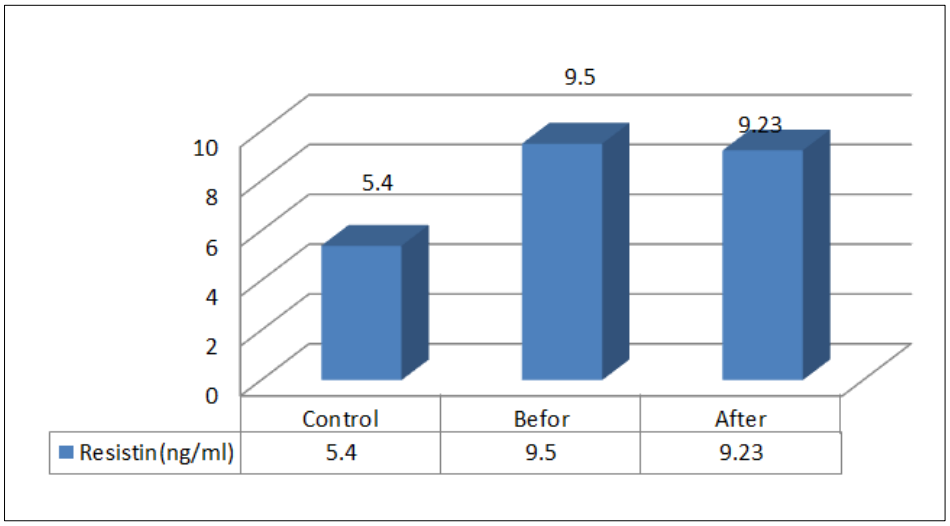

Figure 2: Levels of Resistin in control and patient groups.

There were insignificant differences (P>0.05) between diabetes and non-diabetes patients at the G1 stage, while showed significant results (P<0.05) at the G2 stage. The highest level of IL-6 (46.7±9.4) was seen in diabetes patients before resuscitation, while the lowest (33.1±7.9) was detected after resuscitation in non-diabetes patients. Table 1.

Table 1: Comparison between diabetes and non-diabetes on the basis of IL-6 levels and acute myocardial infarction before (G1) and after (G2)

IL6(Interleukin-6)		Before (G1)	After (G2)
Non diabetes n=36	Mean	43.8	33.1
	SD	8.5	7.9
Diabetes n=26	Mean	46.7	41.2
	SD	9.4	7.5
		>0.05	<0.05

Diabetes patients revealed highest level of IL-6 in comparison to non-diabetes, these were close to a prior study performed in Kerbala city, Iraq by Qalaf *et al.*, [16], who found elevation in IL-6 levels in patients with acute myocardial infarction and diabetes mellitus.

An insignificant increment in the mean levels of Resistin (9.6±0.8 vs. 9.5±0.6) was noted among diabetes patients and non-diabetes, respectively. The results were non-significant statistically (P>0.05). Table 2

Table 2: Comparison between diabetes and non-diabetes on the basis of Resistin levels and acute myocardial infarction

Resisten		**Before**	**After**
Non diabetes n=36	Mean	9.5	9.3
	SD	0.6	0.6
Diabetes n=26	Mean	9.6	9.73
	SD	0.8	0.8
		>0.05	>0.05

There was insignificant increase in the mean levels of Resistin, this was somehow disagreed with Ghanem *et al.*, [17], who found significant increase in Resistin levels among diabetes patients with myocardial infarction, these variances may be due to the differences in the Resistin kinetic response in these patients, or may be due to the differences of sample size.

It was noted that the differences between smokers and non-smokers in regards to IL-6 levels at G1 stage were non-significant (P.value greater than 0.05), whereas the mean levels of IL-6 at G2 stage showed high significant (P<0.01) variances between smokers and non-smokers. Table3.

Table 3: Mean levels of IL-6 in smokers and non-smokers patients before and after IR

Smokers for Patients			
IL6		**Before (G1)**	**After (G2)**
NO n=38	Mean	42.6	30.8
	SD	9.9	9.3
YES n=24	Mean	46.8	38.1
	SD	7.6	9.0
		>0.05	<0.01

An insignificant increase in the levels of the IL-6 levels was seen in smokers in comparison to non-smokers, these were close to what was previously reported by Tiller *et al.*, [18], who found increase in the levels of IL-6 among smokers patients with ST elevation myocardial infarction in matching to non-smokers patients. Our findings were also in line with other studies conducted by Ghamri *et al.*, [19], and Gager *et al.*, [20].

Table 4. Shows insignificant changes (P>0.05) in the levels of Resistin between smokers and non-smokers patients with ST. Elevation myocardial infarction

Table 4: Serum Resistin levels in smokers and non-smokers patients with ST-elevation myocardial infarction

Resisten		**Before**	**After**
NO n=38	Mean	9.5	9.17
	SD	0.6	0.6
YES n=24	Mean	9.5	
	SD	0.7	
		>0.05	>0.05

Moreover, non-significant increase was seen in the levels of Resistin between smokers and non-smokers, this was in disagreement with Zhu *et al.*, [28], who recorded significant increase of Resistin in smokers patients with ST-elevation myocardial infarction, these dissimilarities could be because of the dynamic responses of Resistin in these patients in comparison to our patients, another reason may be due to the ratio of smokers to non-smokers.

Non-significant differences of IL-6 levels were noticed between hypertensive and non-hypertensive patients before and after internal resuscitation. The highest concentration mean level of IL-6 (49.9±10.6) was demonstrated in hypertensive in comparison to 47.4±5.7 in non-hypertensive patients Table 5.

Table 5: The relationship between IL-6 and hypertension in ST-elevation myocardial infarction

IL6		Before (G1)	After (G2)
NO n=38	Mean	47.4	36.4
	SD	5.7	5.3
YES n=24	Mean	49.9	40.5
	SD	10.6	10.5
		>0.05	>0.05

The mean concentration levels of IL-6 was slightly higher in hypertensive than non-hypertensive, these were in accordance with Zegeye *et al*., [22], who noticed increase in IL-6 levels in hypertensive patients with ST-elevation myocardial infarction.

Table 6. Demonstrates slight differences in the concentration levels of Resistin between hypertensive and non-hypertensive patients. The P. value >0.05

Table 6: Serum resistin levels in hypertension and among ST-elevation myocardial infarction

Resisten		Before	After
NO n=38	Mean	9.6	9.09
	SD	0.6	0.6
YES n=24	Mean	9.5	9.2
	SD	0.6	0.6
		>0.05	>0.05

The Resistin mean levels was insignificantly elevated in hypertensive patients, which is disagreed with an earlier work of Zhu *et al.,* [23] , which showed a significant correlation between hypertension and Resistance levels. These differences could be due to the differences in the rates of hypertensive and non-hypertensive patients in these studies.

The current study shows non-significant differences (P>0.05) in regards to IL-6 mean levels between patients with BMI (24-29) and (30-35) before internal resuscitation, while significant differences (P<0.05) were noticed after internal resuscitation, as revealed in Table 7.

Table 7: Association between BMI and IL-6 levels

IL6		Before (G1)	After (G2)
BMI1(24-29) n=38	Mean	44.8	39.5
	SD	10.4	9.1
BMI2 (30-35) n=24	Mean	43.2	31.8
	SD	13.7	8.7
		>0.05	<0.05

IL-6 mean levels were lower in patients with BMI (30-35), than patients with BMI (24-29), these were in disagreement with was reported by Zegeye *et al.,* [22], who detected higher levels of IL-6 among obese patients in comparison to non-obese, these differences may be due to the negative impact of obesity on IL-6 levels. Insignificant changes was detected in the levels of Resistin between obese and non-obese patients, (P>0.05) Table 8.

Table 8: Resistin level in obese and non-obese patients

Resisten	Before	After
BMI1(24-29) n=38	9.5±0.4	9.35
BMI2(30-35) n=24	9.5±0.7	9.26±0.7
	>0.05	>0.05

The levels of Resistin was insignificantly higher in obese than non-obese patients, this agreed to some degree with Scărlătescu *et al.,* [24], who noticed significant relationship between Resistin levels and obesity. Our work was also close to a study done in Lahore and performed by Rashid *et al.,* [25], who also detected significant link between Resistin and obesity in patients with ST-elevation myocardial infarction.

The current study showed insignificant differences in IL-6 levels between remodeling and non-remodeling at G1 time, whereas significant differences were noted after internal resuscitation, as shown in Table 9.

Table 9: Serum IL6 levels in remodeling and non-remodeling

IL6		Before	After
REM(10-14hr) n=36	Mean	45.8	39.6
	SD	11.4	8.8
Non Remodeling (4-9hr) n=26	Mean	43.4	30.8
	SD	10.0	8.9
		>0.05	<0.05

The present study was in line with Tiller *et al.*, [18], found that patients with IL-6 were more likely to experience left ventricular remodeling.

The presented study showed insignificant changes between remodeling of the patients and non-remodeling on the basis of Resistin levels Table 10.

Table10: Serum resistin levels in remodeling and non-remodeling patients

Resisten		Before	After
REM(10-14hr) n=36	Mean	9.5	9.24
	SD	0.6	0.5
Non Remodeling (4-9hr) n=26	Mean	9.6	9.3
	SD	0.5	0.5
		>0.05	>0.05

Our results were close to Scărlătescu *et al.*, [24] , who detected association between remodeling and Resistin levels.

The levels of IL-6 increased insignificantly in H1 in comparison to H2 at the time of IR, while showed significant elevation after IR Table 11.

Table 11: Serum IL6 levels in LVEF for IMA patients

IL6		Before (G1)	After (G2)
H1(31-49%) n=30(Mild /Moderate)	Mean	46.9	31.0
	SD	12.8	8.2
H2(50-62%) n=32(Normal)	Mean	44.3	31.7
	SD	9.9	9.1
		>0.05	<0.05

Our findings were disagreed with Turkyilmaz *et al.*, [26], and Acet *et al.*, [27], who detected the opposite, these differences might be attributed to the status of the patients who were severe cases. The current insignificant increment of Resistin levels was observed between H1 and H2, as shown in Table 12.

Table 12: Connection between LVEF and Resistin levels

Resisten		Before
H1(31-49%) n=30(Mild/Moderate)	Mean	9.5
	SD	0.6
H2(50-62%) n=32(Normal)	Mean	9.5

CONCLUSIONS

It was noticed that IL-6 mean concentration levels was lower in the G2 in comparison to G1, but higher than G3. Furthermore, our study demonstrated higher levels of Resistin in patients in matching to control group.

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