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Relationship of omentin-1 with oxidative stress in Iraqi patients with myocardial infraction

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***Abstract*---**A recently recognized adipokine mediator omentin-1 mainly expressed in human epicardial adipose tissue (EAT) has been found to inhibit the inflammatory response and atherosclerosis factors that has potential beneficial effects on cardiovascular disorders. Recent clinical studies reported that the serum omentin-1 levels were negatively related to the severity of Ischemic heart diseases. In this study, we characterized the clinical significance of omentin-1 in myocardial infarction (MI) patients.Investigated its association with oxidative stress, and demonstrated its protective effect as an antioxidant. The study contained 120 subjects who were divided evenly into patients and control groups for the period between September and December 2021. The serum levels of omentin-1 were assessed by enzyme-linked immunosorbent assays methods and serum zinc and copper were measured using atomic absorption spectroscopy. Also, protein oxidation markers and malondialdehyde (MDA) serum levels were measured by spectrophotometer. The study showed that serum omentin-1 is associated inversely with MI when the concentration of omentin-1had been reduced significantly (89.95 ± 34.43 ng /ml) when compared with the control group (164.39 ± 39.58 ng/ml), as well as it confirmed that there is a significant ($P<0.05$) positive correlation was found between omentin-1 and thiol, total thiol, and disulphide, and a significant ($P<0.05$) negative correlation was also found in free amine and C-reactive protein.The study results established the prognostic and diagnostic role of omentin-1 as a cardioprotective adipokine against oxidative stress suggesting that a decrease in serum omentin-1 levels may contribute to a high risk of MI.

***Keywords*---**Myocardial Infraction, Omentin-1, protein oxidation, Necrosis, Antioxidant.

1. Introduction

Myocardial infarction (MI) is known as the permanent death (necrosis) of heart muscle caused by a shortage of oxygen flow to myocardial tissue. Furthermore, it accompanied by acute loss of practical myocardium which is the main source of death in industrialized nations [1]. Cardiovascular failure happens when the bloodstream diminishes or stops flowing to the coronary corridor of the heart (tissue demise or localized necrosis of the myocardium brought about by ischemia) [2]. MI is described as a quick increase in cytokines and chemokines and a flood of leukocytes into the weak district lining the infarcted site [3].Omentin-1 (Intelectin-1), is a novel hydrophilic adipokine produced by visceral adipose tissue and a secretory protein of 313 amino acids, was discovered in 2005 [4]. Omentin-1 is secreted by omental adipose tissue, the lungs, the heart, and the intestines in little amounts, but it is most abundant in human plasma. Many adipokines are secreted by adipose tissue, including leptin, resistin, adiponectin, chemerin, tumor necrosis factor-α (TNF-α), and interleukin-6 (IL-6) [5]. These adipokines have important roles in carbohydrate and lipid metabolism, homeostasis, insulin resistance, diabetes, atherosclerosis, inflammation, and cardiovascular function [6]. Omentin-1 is extensively produced in human epicardial adipose tissue and it has an anti-inflammatory activity which is essential in cardiometabolic disorders [7]. Omentin-1 plays a significant role in influencing the synthesis of toll-like receptor-4. As a result, omentin-1 has a strong anti-inflammatory impact on inflammatory conditions associated with MI, in contrast, low levels of omentin-1 may induce inflammatory diseases [8]. Furthermore, omentin-1 inhibits the generation of reactive oxygen species (ROS) such as superoxide ($O_2^{\bullet -}$) and other free radicals, which are known to have a role in the onset of vascular smooth muscle inflammation, making it a protective adipokine in Ischemic Heart Disease (IHD) [9]. Omentin-1 stimulates the release of nitric oxide ($NO^{\bullet}$), which causes a significant vasodilation effect; hence, no other inhibitor will reduce the vasodilation effect generated by omentin-1 [10]. It has been reported that serum omentin levels are a significant predictor of cardiovascular events in patients with suspected MI, and heart failure [11].

ROS can attack vital cell components (polyunsaturated fatty acids, proteins, nucleic acids, carbohydrates) ultimately casing in cell death [12]. Myocardium faces an oxidative challenge in all forms of heart diseases and the oxidative modified molecules act as the determinant in the extent of injury and may be useful biomarkers in the diagnostic and prognostic measures [13]. The oxidant and antioxidant imbalance lead to cellular damage in the cardiomyocytes due to the accumulation of oxidants that constitutes oxidative stress [14]. The accumulation of oxidized glutathione (GSSG) was found to be negatively correlated with the myocardium's functional recovery [15]. Because reduced glutathione (GSH), a tripeptide containing a free thiol group, is one of the most significant scavengers of ROS in the heart and the ratio of (GSSG) to (GSH), it is employed as a marker of oxidative stress [12], [16]. As a result, a variety of amino acid modifications might occur, resulting in proteins that are damaging. The free radicals can attack both the peptide backbone and the amino acid side chains of proteins, leading to different radical protein derivatives [17]. In respect of the peptide backbone cleavage of the peptide backbone, the free radicals abstract the hydrogen at the α-carbon position followed by a reaction with $O_2^{\bullet -}$ to give peroxyl

radicals, which results in the cleavage of the protein's backbone and the formation of free amine and carbonyl fragments [18]. Malondialdehyde (MDA) is a dialdehyde of three carbon atoms highly reactive compounds that are produced by the peroxidation process of polyunsaturated fatty acids, it has been used as a marker of oxidative stress to determine the damage of cell membrane lipid by ROS [19]. The study's main goals were to investigate and evaluate the relationship between serum levels of omentin-1 and oxidative stress parameters in patient with myocardial infraction.

2. Experimental

The study involved 120 subjects divided into two groups; group-C included 60 as healthy control and group-A 60 patients diagnosed with AMI based on patient history, symptoms, ECG. The study included only men aged between 43-66 years and the healthy subjects were collected from Mustansiriyah University, as well as AMI patients were collected from Baghdad-Teaching Hospital-Medical City, Baghdad, Iraq and Karbala cardiac center-Alhusaini hospital, Karbala, Iraq between October and December 2021. The laboratory portion of the study was carried out at the Biochemistry Research Laboratory of Mustansiriyah University, department of chemistry science. Using disposable syringes, 5 ml of blood was drawn slowly through a vein puncture and then the blood translocated into gel tube carefully and slowly then left for 15 min at room temperature to clot. The gel tubes' samples were centrifuged for 10 minutes at 3000 rpm and four Eppendorf tubes used to stored the obtained serum at -30 °C until the time of analysis. Some information was taken from patients and control, which include height, weight and age.

This study was conducted on men under 70 years old, excluding any associated disease with MI, especially renal failure and NSTEMI as well as those who were obese or smokers. 5 ml of blood was drawn slowly through a vein puncture without using tourniquet then translocated into gel tubes then left for 15 min at room temperature to clot. After that, the samples were centrifuged for 10 minutes at 3000 rpm and four eppendorf tubes used to stored the obtained serum at -30 °C until the time of analysis.

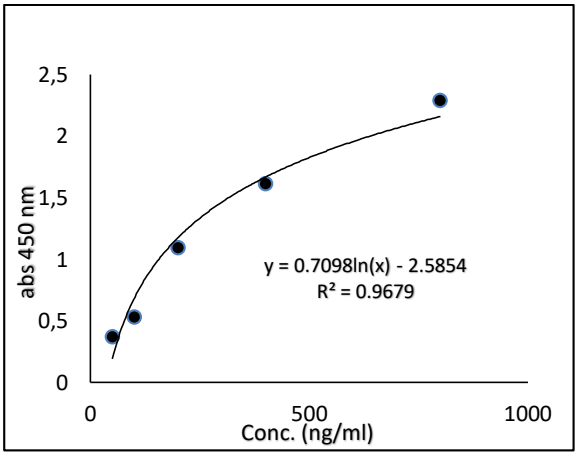
Serum concentrations of omentin-1 were measured by a solid-phase sandwich ELISA kit (Bioassay Technology Laboratory/ China) with Eliza reader (ELX800, BioTech / USA). According to the kit protocol, five standard concentrations (800, 400, 200,100, 50 ng/ml) diluted from the original standard concentration of 1600 ng/ml were used to estimate 120 samples at the wavelength of 450 nm. C-reactive protein was estimated by standard biochemical kits supplied by Abbott using Nycocard reader, a fully autoanalyzer. Flam Atomic Absorption Spectrophotometer (FAAS) model AA646, Shimadzu Corporation, Kyoto, Japan was used to determine Zinc and Copper at wavelength 213.9 nm, 324.7 nm respectively and acetylene was the gas flow. The method of Zaia et al. was used for spectrophotometric measurement of free amino groups [20]. Also, Ellman's technique for determining thiol groups was used [18]. MDA serum levels were determined by TBARs spectrophotometric test [21]. A sample of serum (200µL) was mixed with 2mL of solution contain a 15 % (w/v) trichloroacetic acid, 0.38% (w/v) thiobarbituric acid and 0.25N of hydrochloric acid (HCl). The mixture was

heated to 100°C for 30 minutes, and the absorbance was measured at 535 nm after centrifugation and total samples MDA determined by the difference in absorbance between test and standard in µmol TBARs/L. Statistical analysis Values were explained as mean ± standard deviation (SD). The comparison of study variables was performed using Independent-Samples student's t-test where the difference is considered as highly significant when ($p<0.001$), significant when ($p<0.05$) and non-significant when ($p>0.05$). In addition, Pearson's correlation analysis was carried out to determine the relationships between all study variables.

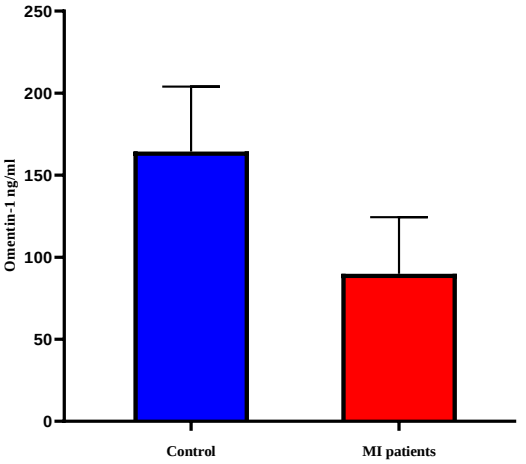
3. Results

(Table-1) showed that the mean age of MI patients and control were (55.45 ± 6.93), (53.91 ± 5.7) with a non-significant p-value ($p>0.01$). Also, weight, height, and BMI mean values were non-significant p-value ($p>0.01$). These non-significant obtained results for age and BMI provide a unique opportunity to conduct a comparative study accurately.

The logarithmic equation (y= 0.7098ln(x)-2.5854) used to estimate omentin concentration as shown in (Figure-1). The study showed that serum omintin-1 levels were reduced significantly ($P<0.001$) in MI patients (89.95 ± 34.43 ng /ml) than those of control group (164.39 ± 39.58 ng/ml) (Figure-2).While CRP level increased significantly in patients compared to the control group ($P<0.001$). Also, the results in (Table-1) indicated that there was significant ($P<0.001$) increase in free amine, MDA, Copper, LDH and ($P<0.012$) for triglyceride (TG). Finally, significant ($P<0.05$) positive correlation was found between omentin and thiol, total thiol, and disulphide while, significant ($P<0.05$) negative correlation was found in free amine and CRP as shown in (Table-2).



Figuer-1: Standard curve used to determine omentin-1 levels.



Figuer-2: Comparisons of serum omentin-1 levels between control subjects and MI patients.

Discussion

The current study shows that patients with acute myocardial infarction had significantly lower omentin-1 serum levels than healthy subjects. Many clinical studies agree with our findings and reported that the serum omentin-1 levels were adversely associated with the severity of Ischemic heart disease [8], [11], [22].Omentin-1 has a strong vasodilator impact on blood vessels stimulated by endothelium-derived nitric oxide, which is considered a powerful vasodilator. It also has an important role as an antioxidant and anti-inflammatory, especially for those with myocardial ischemia [23].

Table 1: The statistics differences between patients and controls regarding to the parameters.

Parameters	Group-C Mean± SD	Group-A Mean± SD	P-value
Age (year)	53.91±5.7	55.45±6.93	0.183 NS
BMI (kg/m^2)	25.52±3.51	26.49±3.95	0.216 NS
Omentin (ng/ml)	164.39±39.58	89.95±34.43	0.001**S
Crp (mg/L)	8.3±4.2	25.44±8.11	0.001**S
Free amine (mmol/l)	35.96±10.15	49.88±13.61	0.001**S
Native thiol (µmol/L)	311.30±93.84	190.90±79.18	0.001**S
Total thiol (µmol/L)	404.55±122.16	267.29±110.87	0.001**S
Disulphide (µmol/L)	46.63±14.17	38.20±15.84	0.003*S
% Native thiol/Disulphide	14.97±0.30	20.01±0.04	0.001**S
MDA (µmol/L)	0.54±0.19	1.83±0.77	0.001**S
Zinc (µg/dl)	98.27±22.67	70.15±6.69	0.001* S
Copper (µg/dl)	128.71±16.32	152.65±9.84	0.001**S
% Zinc/Copper	77.11±18.16	46.17±5.53	0.001**S
LDH (U/L)	300.31±104.71	809.14±180.61	0.001** S
TG (mg/dl)	121.53±26.44	133.23±23.60	0.012*S

*Significant at P<0.05, **highly significant P<0.001, NS: non-significant.

In microvascular endothelial cells, Omentin-1 inhibitsvascular endothelial growth factors that stimulate endothelial cell migration and angiogenesis.

Table 2: Correlation between the studied serum parameters in patients group.

Parameters	*r*	*p*-value
Omentin/crp	-.415-**	0.001
Omentin/free amine	-.345-**	0.007
Omentin/native thiol	.468**	0.000
Omentin/total thiol	.468**	0.000
Omentin/disulphide	.467**	0.000
Omentin/(%thiol,disulphide)	-0.109	0.405
Omentin/mda	-.263-*	0.043
Omentin/zinc	-0.119	0.364
Omentin/(%zinc/copper)	0.047	0.723
Omentin/copper	-0.124	0.345
Omentin/ldh	0.049	0.708
Omentin/tg	0.065	0.621
Crp/native thiol	-.300-*	0.020
Free amine/mda	.701**	0.000
Nativethiol/disulphide	1.000**	0.000
MDA/copper	.352**	0.006
Copper/ldh	.306*	0.018
% Zinc,Copper/ldh	-.270-*	0.037
LDH/tg	.606**	0.000

*Correlation is significant at the 0.05 level (2-tailed). **Correlation is significant at the 0.01 level (2-tailed).

As a result, omentin-1's powerful vasodilation action inhibits norepinephrine's impact, resulting in prolonged heart vasodilation [6, 22]. The recently recognized role of omintin-1 as an anti-inflammatory agent has been suggested as a potential mechanism of anti-atherogenic effect in the affected coronary artery. The anti-inflammatory activity of omentin-1 has been postulated as a possible mechanism of anti-atherogenic impact, based on the inverse association established between omentin-1 and inflammatory cytokines like IL-6, TNF-, and C-reactive protein [24]. Omentin-1 also has a role in preventing the development of ischemic heart disease by inhibiting the generation of reactive oxygen species and free radicals. This suggests that omentin-1 has an antiangiogenic action, making it a powerful protective agent in the formation of atherosclerosis and in the prevention of ischemic heart disease [25]. This current study reveals that there is a significant reduction in omentin-1 serum levels in patients with MI. The current study shows

that oxidative stress markers like protein oxidation and lipid peroxidation have a significant effect in MI patients indicating the role of oxidative stress as a risk factor for injury and this demonstrates the protective effect of omentin-1. This study found that serum MDA levels are much higher in MI patients than in the control group, which is in line with many other studies that have revealed that MDA serum levels are significantly higher, implying that oxidative stress is a risk factor for MI and this study confirms that there is a significant negative relationship between the serum MDA and omentin-1 concentrations where, many studies suggested that omentin-1 has a negative correlation with the serum oxidative stress levels and demonstrate the protective effect of omentin-1 to prevent the damage caused by oxidative stress [26], [27], [28]. The results obtained show that serum thiol levels significantly decrease as well as the increase in free amine levels that agreeing with several studies that prove that protein oxidation is an important indicator of increased oxidative stress [29], [30]. Total thiols and free amine in serum have been explored as a measure of oxidative stress (protein oxidation) in a variety of systemic diseases like ischemic heart disease, acute renal failure, diabetes, pulmonary diseases and preeclampsia, thus chemistry of thiol has become more important [31]. Thiol molecules are very important in the antioxidant system that contains sulfhydryl groups (-SH) by work as a scavenger of reactive oxygen and nitrogen species that cause oxidative damage in cardiac muscle cells [32]. Thiols are oxidatively converted to disulfides in a double-sided balance reaction and both sides are measured in which the excess of disulfides and free amine or thiols deficiency are important biomarkers of oxidative stress [29]. The study confirms that omentin-1 has a significant negative correlation with free amine levels and a significant positive for free thiol and that demonstrates the protective effect of omentin-1 to prevent the damage caused by oxidative stress through protein oxidation. The study appears a significant difference in serum trace zinc, and copper elements. The results show agreement with several studies that demonstrate the adverse effect of zinc and copper as an oxidative stress marker in MI patients [33], [34], [35]. MI is associated with an intelligible imbalance in zinc, and copper trace elements, which are significant for immune and cardiovascular function [36]. These trace elements have an important role in regulatory, catalytic, and structural ions for enzymes, transcription factors, and proteins [37]. These trace elements are linked with a reduced antioxidants potential in organisms and also related to protection against ROS and nitrogen RNS, which are believed to possibly underlie the onset of cancer, AMI, atherosclerosis, accelerated aging, developmental retardation in children, and immunological abnormalities [38], [39].

4. Conclusions

In conclusion, serum omentin-1 levels may have a good diagnostic role in MI patients. These data indicate that omentin-1 may serve as a biomarker for the early detection of ischemia and the reduction in the serum level of omentin-1 may cause the induction of ischemic heart attack and increased oxidative stress in these patients.

5. Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

6. Funding source

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