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Serum Level Of Irisin And Some Biochemical Parameters In Patients With Acute Myocardial Infarction

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Abstract

Acute myocardial infarction (AMI) is the most common form of cardiovascular disease (CVD), that leads to death. Myocardial infarction might directly increase irisin secretion through damaged cardiomyocytes. This study investigated the role of serum irisin level in myocardial infarction patients compared to control and explored its correlation with myocardial infarction in human patients. Sixty patients with acute myocardial infarction and 60 healthy control subjects were comprised in this study. Irisin level was measured using the ELISA method. The mean age of AMI patients was 61.30, 62.38, and 59.21 years for STEMI, NSTEMI, and control, respectively. The ratio of males and females with AMI was 2:1.6, where 37 (61.6%) and 23 (39.4%) were male and female, respectively. The mean irisin levels were 69.83, 72.63 and 204.8 ng/ml for STEMI, NSTEMI, and control groups, respectively. In conclusion, this study approved a significant decrease in the level of the irisin AMI patients group in comparison to the healthy control group.

Keywords: Serum irisin, Biomarker, Acute Myocardial Infarction.

Introduction

Acute myocardial infarction (MI) inflicts massive injury to the coronary microcirculation, leading to vascular disintegration and capillary rarefaction in the infarct region. (1). Acute myocardial infarction is a part of ACS, which includes UA, NSTEMI, and STEMI, are characterized by a sudden reduction in blood supply to the heart (2). Coronary artery disease deaths, most of them caused by myocardial infarction. Early notice is required instantly in patients with stable coronary artery disease (3). The high risk of fatality happens in the preliminary hours of starting acute—myocardial infarct pain. The medical history, physical examination, proper ECG findings, and heart biomarkers are essential in discovering



acute ischemia(4). Within the last three decades, obesity has turned into an international epidemic; in the world, the most severe reason for death is heart disease, especially in the coronary arteries. A substantial risk factor for coronary artery disease is obesity (5). Irisin is a myokine derived from the splitting of fibronectin type III. Irisin regulates glucose metabolism and other biochemical functions. Muscles (skeletal and cardiac) produce irisin, which inspires various cardiac functions. In the early phase of myocardial infarction, a rising irisin level can diminish damage within endothelial cells by inhibiting inflammation and oxidative stress (6). The disturbance of the lipid profile is called dyslipidemia, which includes both hyperlipidemia and hypolipidemia. Fat-soluble vitamin deficiency leads to hypolipidemia, for example, hypobetalipoproteinemia, which is characterized by a lack of apo B in plasma(7). This type of dyslipidemia doesn't have a clinical manifestation In the heterozygous style and, therefore, is unusually diagnosed. Others, like hypo alfa1ipoproteinemia (low HDL-C), have a relation to increased cardiovascular risk (8). In clinical practice, the main recurrent and then the main relevant dyslipidemias are hyperlipidemias. These dyslipidemias are related to an increased risk of coronary disease (9). Review of literature revealed scarce publications regarding the irisin levels in patients diagnosed with acute myocardial infarction in Iraq. Therefore, this study designed to investigate the role of serum irisin level in myocardial infarction patients compared to control and explored its correlation with myocardial infarction in human patients.

Materials & Methods

Ethical approval

Subjects

This is a case-control research on myocardial infarction patients done from June 2019 until April 2020. Sixty (60) patients were selected from individual suffering from chest pain and seeking medical care at Al-Imamin AL-Kadhimeen Hospital. Depending on diagnosis , clinical history and symptoms of ischaemic heart disease (IHD), that confirmed by heart enzymes within sera and Electro cardio Graph (ECG), the patients classified either to be ST-elevation or non-ST-elevation Myocardial infarction (MI).

Blood samples

Venous blood samples (7 milliliter) were collected under aseptic condition from AMI patients and the control groups. Serum were separated from blood after centrifugation at 3000 RPM for 15 minutes. Then, aliquot of serum were kept into a 1.5 ml Eppendorf tube; this portion was used for immediate assay (lipid profile with blood sugar). The remaining part were transferred to small tubes and stored at -20°C for irisin test estimation. Repeated freezing and thawing was avoided.

Methods

Serum irisin was estimated by ELISA using a commercial kits (Bioassay, china)

Results

This study comprised 60 patients and 60 controls. According to gender, the numbers and percentages of female and male in STEMI group were 12 (40.00%) and 18 (60.00%) respectively. The numbers and percentages of female and male in control group was 26(43.33%) and 34(56.67%) respectively, while the ratio of male: female was (2:1.6). Additionally, the recognized frequencies in the male group were more than in the female. However, statistically was no significant difference between the frequency of the patients in male and female groups compared to the control group (P=0.82). Table. (1).

Table. 1: General characteristics and the risk factors of the studied groups

Parameter	Control Mean values ±SD (n=60)	STEMI Mean values ±SD (n=30)	NSTEMI Mean values ±SD (n=30)	P value
Age (years)	59.21±10.12 ^a	61.30±9.18 ^a	62.36±7.84 ^a	0.28
Gender				
Male	34(56.67%)	18(60.00%)	19(63.33%)	0.82
Female	26(43.33%)	12(40.00%)	11(36.67%)	

.Sig. P < 0.05; high .Sig. .P < 0.01; no asterisk: P> 0.05

"Similar small letters indicate unimportant differences while different lowercase letters indicate large differences"

Serum lipid profiles were significantly increased in the AMI groups, indicating a more clearly defined lipid disorder. Serum HDL-C was found to diminish significantly compared to the control group (Table .2). Serum FBS in the STEMI group did not show any significant correlation with other parameters (Table .2).

Table. 2: The Mean values ±SD lipid profile and FBS of the studied groups

Parameter	Control Mean values ±SD (n=60)	STEMI Mean values ±SD (n=30)	Non-STEMI Mean values ±SD (n=30)	P value
FPG (mg/dl)	99.43±8.09 ^a	99.16±10.50 ^a	97.06±11.25 ^a	0.52
TC (mg/dl)	165.40±25.61 ^a	203.90±66.27 ^b	189.73±42.99 ^b	0.001
TG (mg/dl)	131.91±11.86 ^a	168.93±45.14 ^b	152.56±36.16 ^b	0.001
HDL-C (mg/dl)	50.10±14.14 ^a	33.60±4.59 ^b	36.00±5.97 ^b	0.001
VLDL-C (mg/dl)	26.38±2.37 ^a	33.78±9.02 ^b	30.51±7.23 ^b	0.001
LDL-C (mg/dl)	76.48±13.22 ^a	136.54±62.53 ^b	123.22±38.81 ^b	0.001



.Sig. P < 0.05; high .Sig. .P < 0.01; no asterisk: P> 0.05

"Similar small letters indicate unimportant differences while different lowercase letters indicate large differences"

Table (3) revealed a highly significant decrease in omentin-1 levels in (the STEMI and NSTEMI) group of patients in comparison with the control group (p=0.01).

Table (3): Mean values and ±SD of omentin-1 levels of the three studied groups

Parameter	STEMI(G1) (n=30)	NSTEMI(G2) (n=30)	Control(G3) (n=60)	P value
irisin	3.51± 1.54	3.36± 1.46	9.95± 4.42	0.01

Table (4). The person correlation analysis of STEMI group

	S. Irisin (ng/ml)	TG (mg/dl)	TC (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
S. Irisin (ng/ml)	r=1	0.28 0.13	0.29 0.11	-0.17 0.35	-0.31 0.09	0.28 0.13
TG (mg/dl)	r= 0.28 p= 0.13	1	0.45 0.01	-0.35 0.06	0.35 0.05	1.00 0.001
TC (mg/dl)	r= 0.29 p= 0.11	0.45 0.01	1	-0.00 0.98	-0.99 0.00	0.45 0.01
HDL-C (mg/dl)	r= -0.17 p= 0.35	-0.35 0.06	-0.00 0.98	1	-0.02 0.88	-0.35 0.06
LDL-C (mg/dl)	r= -0.31 p= 0.09	0.35 0.05	-0.99 0.00	-0.02 0.88	1	0.35 0.05
VLDL-C (mg/dl)	r= 0.28 p= 0.13	1.00 0.001	0.45 0.01	-0.35 0.06	0.35 0.05	1

Table.5: The person correlation analysis of NSTEMI group

	S. Irisin (ng/ml)	TG (mg/dl)	TC (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
S. Irisin (ng/ml)	r=1	-0.23 0.22	-0.19 0.32	-0.17 0.36	-0.14 0.46	-0.23 0.22
TG (mg/dl)	r= -0.23 p= 0.22	1	0.55 0.001	0.32 0.08	0.37 0.03	1.00 0.001
TC (mg/dl)	r= -0.19 p= 0.32	0.55 0.001	1	0.21 0.24	0.97 0.001	0.55 0.001
HDL-C (mg/dl)	r= -0.17 p= 0.36	0.32 0.08	0.21 0.24	1	0.02 0.88	0.32 0.08
LDL-C (mg/dl)	r= -0.14 p= 0.46	0.37 0.03	0.97 0.001	0.02 0.88	1	0.37 0.03
VLDL-C (mg/dl)	r= -0.23 p= 0.22	1.00 0.001	0.55 0.001	0.32 0.08	0.37 0.03	1

Table.6: The person correlation analysis of control group

	S. Irisin (ng/ml)	TG (mg/dl)	TC (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
S. Irisin (ng/ml)	r=1	0.02 0.83	0.00 0.94	-0.06 0.64	-0.05 0.65	0.02 0.83
TG (mg/dl)	r= 0.02 p= 0.83	1	0.03 0.78	-0.45 0.00	-0.30 0.01	1.00 0.00
TC (mg/dl)	r= 0.00 p= 0.94	0.03 0.78	1	-0.06 0.61	-0.06 0.62	0.03 0.78



HDL-C (mg/dl)	r= -0.06 p= 0.64	-0.45 0.00	-0.06 0.61	1	0.98 0.00	-0.45 0.001
LDL-C (mg/dl)	r= -0.05 p= 0.65	-0.30 0.01	-0.06 0.62	0.98 0.00	1	-0.30 0.01
VLDL-C (mg/dl)	r= 0.02 p= 0.83	1.00 0.00	0.03 0.78	-0.45 0.001	-0.30 0.01	1

Discussion

Irisin is believed to be linked with the increase of thermogenesis because of the activation of "browning" for fat tissue and improving glucose profile via lowering of insulin resistance (10)(11). Circulating irisin concentrations have been correlated with a broad series of metabolic diseases, starting from increased BMI (obesity) and insulin resistance to diabetes mellitus(11), cardiovascular diseases, thyroid function, and bone metabolism (12). Irisin, a myokine, is incised from the extracellular portion of FNDC5 in striated and cardiac muscle and secreted as a hormone into circulation during exercise (13). The average value for serum irisin in the control group was (9.95±4.42). The average value for serum irisin in the STEMI patient group was(3.51±1.54). The average value for serum irisin in the NSTEMI patient group was(3.36±1.46). There was a considerable variance between control and patient collection(P value=0.01). The data results of this study show that the irisin level was significantly decreased in the patient group(P value=0.01). This result agrees with a previous study done by (14), which found that Irisin concentration is decreased in patients who have myocardial infarction. Irisin is a circulating hormone (myokine) that promotes the health advantages of physical exercise and organizes energy homeostasis (15). Irisin concentrations are decreased in CAD and MI because their manufacturing may rely on blood supplying myocardial muscle (16). Acute myocardial infarction is one of the most important causes of death in the world. The occurrence of the disease almost three million people worldwide, with over inevitable million deaths in the USA annually. (17). Atherosclerosis is a chronic vascular disease related to endothelial dysfunction. Oxidation of LDL cholesterol is one of the key factors for atherosclerosis. (18) Serum lipid profiles were significantly elevated in all the MI groups, with more pronounced lipid disturbances, except for the serum HDL-C, which was found to be reduced significantly when matched with the control group, as shown in Table(2). This study agrees with a past study done by (19), which also found a significantly low HDL level in non-diabetic patients with AMI. Nevertheless, previous reports show that dyslipidemia is one of the conventional risk factors in patients together with ACD (20). Research by (4) predicted that most patients had risen LDL cholesterol concentrations and depressed HDL cholesterol concentrations within 24 hours of the onset of MI. In the preliminary studies, rising LDL and VLDL concentrations and diminished HDL concentration signaled a rise in arterial hardness and carotid intima-media density. (21)

Conclusion

Irisin has a function that is important in protective action in the heart, and the measurement of irisin in Acute myocardial infarction patients may support diagnosis and prevent complications in myocardial infarction.

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Competing interests statement

No conflict of interest related with publishing of this article.

Ethics statement

All authors approved that this research follows the journal's ethical guidelines as appeared on the journal's author guidelines page.

Author contributions

All authors contribute equally in doing this research.

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