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Evaluation of Adiponectin hormone and some biochemical parameters with unstable angina and acute myocardial infarction patients

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Abstract

The acute coronary syndromes [unstable angina (UA) and acute myocardial infarction (AMI)] are more dangerous than other ischemic heart diseases (IHD) due to acute morphological changes in atherosclerotic plaques which cause (acute ischemia) severe imbalance between myocardium demand and oxygen supply. In this study we investigated adiponectin hormone, creatine kinase(CK), lactate dehydrogenase(LDH), malondialdehyde(MDA), nitric oxide(NO), superoxide dismutase(SOD), and glutathione(GSH) in patients with unstable angina and acute myocardial infarction, This study has been carried out on 100 patients with unstable angina and acute myocardial infarction and 50 healthy subjects. the study shows that there is a significant difference in the concentration of serum Adiponectin, CK, LDH, MDA, NO, SOD, and GSH between (controls, AMI and UA) in the (male and female) groups ($p \leq 0.05$).

KEYWORDS: Unstable Angina, Myocardial Infarction, Adiponectin, Creatine Kinase, Lactate dehydrogenase

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1. INTRODUCTION:

Coronary artery disease (CAD) is commonly classified clinically into two subtypes: stable angina pectoris (SAP) and acute coronary syndrome (ACS). Acute coronary syndrome, which includes unstable angina pectoris (UAP) and acute myocardial infarction (AMI), is the medical term for the advanced stage of coronary artery disease. It is the main reason of the rupture of a coronary atherosclerotic plaque, which results in partial or complete vessel occlusion (Ghattas *et al*. 2013, Goldstein *et al.* 2000). AMI is a disorder that may occur as a result of ischemic heart disease or coronary artery disease, and it manifests itself when an atherosclerotic plate ruptures and a developing thrombus completely or partly occludes the coronary artery, decreasing the flow of blood to the cardiac muscle (Liakos and Parikh 2018, Aydin and Aydin 2016). Patients with ACS are divided into two groups: Patients with and without ST-segment elevation. ACS without ST-segment elevation (NSTEACS) also includes unstable angina pectoris (UA) and non-ST elevation myocardial infarction (NSTEMI). It is important to note that UA is defined as ischemic chest pain at rest without a rise in serum cardiac biomarkers, while the establishment of

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NSTEMI diagnosis requires a rise in serum cardiac biomarkers. ACS with ST-segment elevation (STEMI) includes both ST-segment elevation and a rise in serum cardiac biomarkers (**Bergovec** et al., 2009). The connection between UA, NSTEMI, and STEMI is based on the fact that these are closely connected conditions with similar pathogenesis and clinical presentation, but they do differ by the level of severity(**Falconnet** et al., 2009, Majid et al., 2014). When individuals have sudden chest pain or overwhelming shortness of breath, with neither ST-elevation nor abnormal heart enzymes they are thought to have an acute coronary syndrome (ACS) known as unstable angina pectoris (UAP) (Amsterdam *et al.* 2010). UAP has <50% atherosclerotic plaque, which does not obstruct the flow of blood or induce angina symptoms (Maddox *et al.* 2014). Important risk factors are earlier cardiovascular disease, old age, tobacco smoking, high blood levels of certain lipids (LDL and TG) and small levels of (HDL) cholesterol, diabetes, high blood pressure, lack of physical activity, obesity, chronic kidney disease, excessive alcohol consumption and the use of cocaine and amphetamines (Graham *et al* ., 2007, Auda et al., 2018, Majid et al., 2018). Myocardial infarction [MI] is manifested with impaired systolic and diastolic function, ventricular dilatation, and ultimately congestive HF [Shiny et al., 1989, Majid and Sayer, 2018]. Adiponectin is a 244 amino acid collagen-like protein that was discovered in 1995. It is produced by adipocytes and has anti-inflammatory and insulin-sensitizing effects. Obesity has been linked to adiponectin dysregulation, cardiovascular disease, metabolic syndrome, type 2 diabetes, and hypertension (Pessin and Kwon 2013, Lopez 2016). Oxidative stress is a condition in which the body undergoes pathogenic alteration; this is followed by an increase in the generation of free radicals. Oxidative stress causes a variety of factors to act with biological processes, connecting adversity in the environment to depressed emotional states (Huynh and Alderson 2009). This current study aimed to estimate serum of adiponectin in patients with ACS AMI, UA, and healthy individuals were both enrolled. Accurately measure the levels of creatine kinase, lactate dehydrogenase, and oxidant-antioxidant parameters in patients with AMI, UA, and healthy individuals.

2. MATERIALS AND METHODS

The present study was performed in Nasiriyah Heart Center, especially in the coronary care unit (CCU) and the laboratories during the period from 10/ 6/ 2021 to 31/ 9/ 2021. The study included (150) subjects, (100 patients). The patients were divided into two groups: a group1 of 50 patients with unstable angina pectoris (UA) 32 males and 18 females with an age range (of 35-80), group 2 patients: 50 patients with acute myocardial infarction (AMI) 36 males and 14 females with age range (35-80). And group 3 control group, consists of 50 healthy individuals 28 males and 22 females without a history of systemic disease in the age range (35 -80).

2.1.Statistical Analysis: Data were statistically analyzed using Package Social Sciences (SPSS) for Windows version 12.0 software. All experimental data were expressed as mean ± standard deviation(SD).

RESULTS AND DISCUSSION

Table 1: Changes in serum Adiponectin and other parameters in different groups under study

Parameters	Gender	Control	AMI	UA
Adiponectin	Male	9.02±1.10 [a]	2.65±0.72 [c]	3.57±0.72 [b]
	Female	9.34±0.96 [a]	2.16±0.42 [c]	3.52±0.31 [b]
CK	Male	96.32±13.45 [c]	278.56±32.47 [a]	150.28±14.09 [b]
	Female	100.00±9.88 [c]	317.84±33.92 [a]	150.52±18.74 [b]
LDH	Male	154.16±13.54 [c]	358.00±45.27 [a]	233.16±24.40 [b]
	Female	149.24±15.41 [c]	336.36±38.11[a]	243.68±21.75 [b]
MDA	Male	1.84±0.41 [c]	6.05±0.99 [a]	3.76±0.37 [b]
	Female	1.72±0.47 [c]	6.65±1.13 [a]	4.34±0.71[b]
NO	Male	6.92±0.81 [c]	13.86±1.09 [a]	11.76±1.56 [b]
	Female	7.46±0.78 [c]	13.46±1.19 [a]	10.70±1.86 [b]
SOD	Male	1522.05±109.58 [a]	927.21±57.58 [c]	1237.35±90.54 [b]
	Female	1606.20±97.15 [a]	873.83±92.77 [c]	1222.80±98.41 [b]
GSH	Male	525.76±57.68 [a]	331.08±53.98 [c]	374.56±82.02 [b]
	Female	517.28±37.74 [a]	339.60±69.89 [b]	341.60±24.36 [b]

In this study, we measured the level of Adiponectin, Creatine Phosphokinase, Lactate Dehydrogenase, Malondialdehyde, Superoxide dismutase, Nitric Oxide, and Glutathione. Table1 show in the control groups it was found no significant difference in the concentration of serum Adiponectin between male and female ($p \geq 0.05$). While in the AMI group, it was found a significant difference in the concentration of serum Adiponectin between males and females ($p \leq 0.05$). In the UA group, it was found a significant difference in the concentration of serum Adiponectin between males and females ($p \leq 0.05$). Also show a significant difference in the concentration of serum Adiponectin between (controls, AMI and UA) groups in the (male and female) groups($p \leq 0.05$). Also, the table1 show in the control groups it was

found no significant difference in the concentration of serum CK, LDH, and NO between male and female ($p \geq 0.05$). Also in the AMI group, it was found no significant difference in the concentration of serum CK between males and females ($p \geq 0.05$). In the UA group, it was found no significant difference in the concentration of serum CK between males and females ($p \geq 0.05$). Also show a significant difference in the concentration of serum CK between (controls, AMI and UA) groups in the (male and female) groups ($p \leq 0.05$). Table1 show in the control group it was found no significant difference in the concentration of serum MDA between male and female ($p \geq 0.05$). Also in the AMI group, it was found no significant difference in the concentration of serum MDA between males and females ($p \geq 0.05$). While in the UA group it was found a significant difference in the concentration of serum MDA between males and females ($p \leq 0.05$). Also show a significant difference in the concentration of serum MDA between (controls, AM and UA) groups in the (male and female) groups ($p \leq 0.05$).

Also, show in control groups it was found no significant difference in the concentration of serum SOD between males and females ($p \geq 0.05$). Also in the AMI group, it was found a significant difference in the concentration of serum SOD between males and females ($p \leq 0.05$). In the UA group, it was found no significant difference in the concentration of serum SOD between males and females ($p \geq 0.05$). Also show a significant difference in the concentration of serum SOD between (controls, AMI and UA) groups in the (male and female) groups ($p \leq 0.05$).

Table 1 show in control groups it was found a significant difference in the concentration of serum GSH between male and female($p \leq 0.05$). But in the AMI group, it was found no significant difference in the concentration of serum GSH between males and females ($p \geq 0.05$). In the UA group, it was found a significant difference in the concentration of serum GSH between males and females ($p \leq 0.05$). Also show a significant difference in the concentration of serum GSH between (controls, AMI and UA) groups in the (male and female) groups ($p \leq 0.05$).

CONCLUSION

Gender has an effect on each Adiponectin and SOD with AMI patients, Also affects each Adiponectin, MDA, and GSH with UA patients. In all studied groups, it was found no significant difference in the concentration of serum CK, LDH, and NO between males and females.

REFRENCES :

Amsterdam, E. A., Kirk, J. D., Bluemke, D. A., Diercks, D., Farkouh, M. E., Garvey, J. L. and American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee of the Council on Clinical Cardiology, Council on Cardiovascular Nursing, and Interdisciplinary Council on Quality of Care and Outcomes Research. 2010. Testing of low-risk patients presenting to the emergency department with chest pain: a scientific statement from the American Heart Association. Circulation., 122: 1756-1776.

Auda, M. A., Majid, A., Al-Fartosi, K, G. 2018. Protective role of polyphenolic compounds extracted from *cyperus rotundus* rhizomes and taurine on troponin-I and some oxidant/antioxidants parameters of female rats
treated with isoproterenol-induced myocardial infarction**.** Journal of Thi-Qar University. 13(2). 61:76.

Aydin, S. and Aydin, S. 2016. Irisin concentrations as a myocardial biomarker. Biomarkers in Cardiovascular Disease., pp. 489-504.

Bergovec, M.; Vrazic, H. and Rajcan Spoljaric, I. Features and pathophysiology of acute coronary syndrome [Oblici patofiziolgija akutnog koronarnog sindroma].Acute Medica Croatica : Casopis Hravatske Akademije Medcinskih Znanosti .,63(1):3-7;2009.

Falconnet, C.; Carballo, S. and Roffi, M . Acute coronary syndrome: Guidelines and geriatrics specificity (syndrome coronarien aigu: Guidelines et specificite geriatrique). Revue Medicale Suisse.,5(204):1137-1147;2009

Ghattas, A., Griffiths, H. R., Devitt, A., Lip, G. Y., and Shantsila, E. 2013. Monocytes in coronary artery disease and atherosclerosis: where are we now. Journal of the American College of Cardiology., 62: 1541-1551.

Goldstein, J. A., Demetriou, D., Grines, C. L., Pica, M., Shoukfeh, M., and O'Neill, W. W. 2000. Multiple complex coronary plaques in patients with acute myocardial infarction. New England Journal of Medicine., 343: 915-922.

Graham, I.; Atar, D.; Borch-Johnsen, K.; Boysen, G.; Burell, G.; Cifkova, R.; Dallongeville,J.; De Backer, G.; Ebrahim, S.; Gjelsvik, B .“European Guidelines on Cardiovascular Disease Prevention in Clinical Practice.” *Heart J* ., 28: 2375–414, 2007

Huynh, T. and Alderson, M. 2009. Concept analysis of human ecology. In Nursing Forum. Malden, USA: Blackwell Publishing Inc., 44: 115-128

Liakos, M. and Parikh, P. B. 2018. Gender disparities in presentation, management, and outcomes of acute myocardial infarction. Current cardiology reports., 20: 1-9.

Lopez, J. P. 2016. The role of adiponectin in cardiometabolic diseases: effects of nutritional interventions. The Journal of nutrition., 146: 422S-426S.

Maddox, T. M., Stanislawski, M. A., Grunwald, G. K., Bradley, S. M., Ho, P. M., Tsai, T. T. and Rumsfeld, J. S. 2014. Nonobstructive coronary artery disease and risk of myocardial infarction. Jama., 312: 1754-1763

Majid, A. Faraj, H. R. and Affat, A. S. 2014. Clinical Study Of Some Physiological Parameters in Patients With Acute Coronary Syndrome (ACS) in Thi-Qar Governorate / Iraq. International Journal of Scientific & Engineering Research., 5: 645-657.

Majid, A. Sayer. S A. 2018. Protective effect of phenolic extract of Cyperus rotundus rhizomes on myocardial infarction induced by isoproterenol in female rats. IOP Conf. Ser.: Mater. Sci. Eng. **454** 012005.

Majid, A., Auda, M. A., Al- Fartosi, K. G. Protective Role of Polyphenolic Compounds of *Cyperus Rotundus* and Taurine on Troponin –I and Lipid Profile of Female Rats treated with Isoproterenol – Induced Myocardial Infraction.Thi-Qar Univ. J. for Agric. Res. 7(2). 14:22

Shiny K S, Kumar S H, Farvin K H, Anandan R and Devadasan K 1989 Protective effect of myocardial infraction *J. Physiol Pharmacol* 1235

Pessin, J. E., and Kwon, H. 2013. Adipokines mediate inflammation and insulin resistance. Frontiers in endocrinology., 4: 71.