The Association between Adiponectin, Insulin and Troponin I in Patients with Acute Myocardial Infarction

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Abstract

Adipose tissue is increasingly recognized as a key regulator of energy balance, playing an active role in lipid storage and buffering, and synthesizing and secreting a wide range of endocrine products that may be directly involved in the pathogenesis of the complications associated with obesity the most important adiposcyte deviated hormone is adiponectin. Insulin is a hormone that is central to regulating the energy and glucose metabolism in the body. Troponin is a complex of three regulatory proteins that is integral to muscle contraction in skeletal and cardiac muscle.

This study was conducted during the period from Decenber2009 to April 2010, includes fifty patients with Acut Myocardial Infarction (AMI) were admitted to Cardiac Care Unit (CCU) at Medical City Teaching Hospital and Ibn –ALbetar Hospital in Baghdad. Levels of adiponectin, insulin and troponin were measured by ELISA method.The levels of adiponectin, insulin and troponin were significantly elevated with ($p<0.001$), A negatively significant correlation between adiponectin with insulin and troponin in acute myocardial infarction patients was found.

Adiponectin was negatively associated with insulin in patients with AMI.

Keywords: adiponectin, insulin and Acute Myocardial Infarction.

Introduction

Acute Myocardial Infarction (AMI), commonly known as a heart attack, is the interruption of blood supply to part of the heart, causing some heart cells to die. This is most commonly due to occlusion (blockage) of a coronary artery following the rupture of a vulnerable atherosclerotic plaque, which is an unstable collection of lipids (fatty acids) and white blood cells (especially macrophages) in the wall of an artery. The resulting ischemia (restriction in blood supply) and oxygen shortage, if left untreated for a sufficient period of time, can cause damage or death (infarction) of heart muscle tissue. [1]

Important risk factors are previous cardiovascular disease (such as angina, a previous heart attack or stroke), old age (especially men over 40 and women over 50), tobacco smoking, high blood levels of certain lipids (triglycerides, low-density lipoprotein or "bad cholesterol") and low levels of high density lipoprotein (HDL, "good cholesterol"), diabetes, high blood pressure, obesity, chronic kidney disease, heart failure, excessive alcohol consumption, the abuse of certain drugs (such as cocaine and methamphetamine), and chronic high stress levels. [2]

Adipose tissue is increasingly recognized as a key regulator of energy balance, playing an active role in lipid storage and buffering, and synthesizing and secreting a wide range of endocrine products that may be directly involved in the pathogenesis of the complications associated with obesity. So obesity is considered the major independent risk factor for atherosclerotic cardiovascular disease.

Initially, adiponectin was thought to be exclusively synthesized by adipocytes; however, a recent study suggests that adiponectin is also synthesized and secreted by human cardiomyocytes. [3] This may explain why adiponectin is increased in obese subjects with AMI while it is supposed to decrease in obese subjects, but how much this cardiomyocytes effect on the total concentration of adiponectin needs more study.

M. Vlasova et al.2010 [4] reported that is found as two forms in serum, as a lower molecular weight trimer-dimer and a (High

Molecular Weight) HMW complex. Females display significantly higher levels of the HMW complex in serum than do males. The levels of the HMW complex appeared to be negatively regulated by insulin.

Adiponectin gene regulation includes a number of hormonal and environmental factors. It's expression in white adipose tissue is decreased by obesity, glucocorticoids, β-adrenergic agonists and TNF-α and increased by leanness, cold exposure, adrenalectomy and IGF-1. [5]

Insulin is a hormone that is central to regulating the energy and glucose metabolism in the. [6]

Troponin is a complex of three regulatory proteins that is integral to muscle contraction in skeletal and cardiac muscle, but not muscle. Discussions of troponin often pertain to its functional characteristics and/or to its usefulness as a diagnostic marker for various heart disorders. [7]

Subjects

This study was conducted during the period from Decenber2009 to April 2010, includes fifty patients with Acut Myocardial Infarction (AMI) who were admitted to Cardiac Care Unit (CCU) at Medical City Teaching Hospital and Ibn –ALbetar Hospital in Baghdad. patients with age range of 20-78 years, were included in this study. Blood samples were taken from the patients after having thoroughly examined after exclusion of subjects with a history a MI or diabetes mellitus or any chronic diseases. Control group contain fifty age, sex and BMI matched, apparently healthy individuals, were included in this study as control group.

Blood collection and laboratory analysis

From each patients and control, five ml venous blood were aspirated from a suitable vein. Samples were collected between (8-9 A.M.) after 10 hours fast. Blood samples transferred to plain tubes for storage to measure the adiponectin and insulin. The non heparinized blood in the plain tubes were left to clot and then centrifuged at 4000 rpm for 5 minutes to separate the serum and dispensed into tightly closed Eppendorf tubes in 1.0 ml and stored at -20 C° until assayed. Each serum sample was analyzed for urea and creatinin to excluded kidney diseases. Adiponectin, insulin and troponin were measured by using ELISA kits from United States Biological Company.

Statistical Analysis

Statistical analysis was performed by statisticians with the SPSS 15.01 Statistical Package for Social Sciences and also Excel 2003. Data analysis was done using chi-square test for tables with frequencies, while we used independent sample t-test for tables with means and standard deviations. *p* value of ≤ 0.05 was used as the level of significance. Correlation coefficient used to find the correlation between studied markers by using Pearson correlation. Descriptive statistics for the clinical and laboratory results were formulated as mean and standard error.

Results

Serum levels of adiponectin, insulin and troponin I were compared among the studied groups as in Table (1). The patients with MI were found to have significantly higher serum insulin and adiponectin with ($p<0.001$).

Table (1)
Comparison between groups for adiponectin, insulin and troponin in studied groups.

parameters	*Female patients Mean±SR NO.=16*	*Female Control Mean±SR NO.=16*	*P-value*	*Male Patient Mean±SR NO.=24*	*Male Control Mean±SR NO=34*	*p-value*	*Total Patients Mean±SR N0.=50*	*Total Control Mean±SR N0.40*	*P-value*
Adiponectin g/ml	59.46 ±18.09	13.769± 5.095	<0.001	50.01 ±10.93	13.279 ±6.817	<0.001	54.68 ±13.88	13.48 ±2.09	<0.001
Insulin ulU/ml	71.99 ±6.05	29.792 ±8.309	<0.001	71.74 ±9.70	34.092 ±7.98	<0.001	71.83 ±11.09	32.39 ±4.50	<0.001
Troponin ng/ml	34.63 ±6.51	0.0	--	38.18 ±9.95	---------	--	30.21 ±8.36	0.0	-----
BMI Kg/m2	26.97 ±2.99	26.62 ±3.65	0.803	29.03 ±5.28	29.1 ±2.7	0.943	28.19 ±4.88	28.47 ±3.03	0.763
Age Years	61.70 ±10.19	60.18 ±9.938	0.523	53.37 ±12.20	54.043 ±9.14	0.620	56.26 ±12.11	56.51 ±9.19	0.916

Correlation between adiponectin with insulin

A negative correlation was found between adiponectin and insulin in female patient group (r=-0.476), in female control group (r=-0.403), in male patient group (r=-0.400), in male control group (r=-0.387), in total patient (r= -0.419) and in total control (r=-0.375) and with troponin in female patient group (r=-0.641) in male patient group (r=-0.619) and in total patient (r=-0.637)

Table (2)
The correlation between adiponectin with insulin and troponin for studied groups.

Parameters	*Female Patients NO.=16*	*Female Control NO.=16*	*Male Patients NO.=34*	*Male Control NO.=24*	*Total Patients NO.=50*	*Total Control NO.=40*
Insulin ulU/ml	-0.476*	-0.403*	-0. 400*	-0.387*	-0. 419*	-0. 215*
Troponin I ng/ml	-0.641*	--	-0. 619*	---	-0.637*	--

**= P‹0.001*

Discussion

Kobayashi et al., in 2009 demonstrated that adenovirus mediated increase of plasma adiponectin significantly suppressed the progression of atherosclerotic lesions in apoE ob/ob mice. These mice develop hyperlipidemia and vascular lesions similar to human atherosclerosis. Adenovirus-derived adiponectin accumulated in the fatty streak lesions composed of macrophages and foam cells in apoE ob/ob mice.[5]

Animal data support adiponectin as a cardiovascular protective molecule. In a mouse model of acute MI, adiponectin null mice responded with larger infarct sizes, greater myocardial cell apoptosis, and increased tumor necrosis factor expression when compared with wild-type controls. Rescue attempts with adiponectin delivered by adenovirus, and recombinant adiponectin infusion prior to or during the ischaemia-reperfusion procedure, ameliorated all the associated damaging effects, suggesting that exogenous adiponectin protects the heart against ischaemic insults. [8] That agrees with our notice that the patients with higher levels of adiponectin have good prognosis. Adiponectin was also demonstrated to inhibit strongly the expression of adhesion molecules, including intracellular adhesion molecule-1, vascular

cellular adhesion molecule-1, and E-selectin Adiponectin was also shown to inhibit TNF-α-induced nuclear factor-κB activation through the inhibition of κB phosphorylation.

Suppression of nuclear factor-κB by adiponectin might be a major molecular mechanism for the inhibition of monocyte adhesion to endothelial cells (VCAM-1 play a pivotal role in the development of atherosclerosis. The expression of VCAM-1 localized over the surface of endothelial cells in lesion-prone sites). Adiponectin also inhibits the expression of the scavenger receptor class A-1 of macrophages, resulting in markedly decreased uptake of oxidized low-density lipoprotein by macrophages and inhibition of foam cell formation. In addition, in cultured smooth muscle cells, adiponectin attenuated DNA synthesis induced by growth factors including platelet-derived growth factor, heparin-binding epidermal growth factor (EGF)-like growth factor, basic fibroblast growth factor, and EGF, as well as cell proliferation and migration induced by heparin binding EGF-like growth factor. [9]

(Wolfgang Koenig,et al,2009) [10] study on adiponectin-deficient mice have shown a 2-fold increase in neointimal thickening and increased proliferation of vascular SMCs in arteries after mechanical injury.that agree with what we noticed with patients, that the thickness of artery in ECO monitoring is may be associated negatively with adiponectin level. In addition, adiponectin knockout mice showed high levels of TNF-alpha mRNA in adipose tissue. Thus suggest an antidiabetic and antiatherogenic role of increased concentrations of adiponectin and that hypoadiponectinemia, in particular in combination with low HDL-C, therefore might be associated with a strongly increased risk of T2DM and atherosclerotic disease. The effects of both, low adiponectin and low HDL-C on endothelial dysfunction, and their enhancement of an inflammatory response may represent plausible arguments for their additive effect on risk.

Atul Singhal studied adiponectin in vitro and suggested that adiponectin reduces the development of atherosclerosis by stimulating the production of nitric oxide from vascular endothelial cells. [11]

On the other hand, low adiponectin levels are associated with reduced expression of nitric oxide, and increased expression of angiotensin II and cellular adhesion molecules from the endothelium. [10]

In humans, there are many offensive factors present, including oxidized LDL, inflammatory stimuli and chemical substances that may induce vascular injuries. At that time, adiponectin secreted from adipose tissues may go into the injured arteries and protect against the development of atherogenic vascular changes. Therefore, adiponectin might be likened firefighters who put out the fire of the vascular walls while it is still small. When the plasma levels of adiponectin are decreased in the subjects with visceral fat accumulation, the small fire may become bigger and bigger because of the shortage of firefighters [12].

Despite all the counter-regulatory mechanisms that are mobilized in the high-risk patients, including up-regulation of plasma adiponectin levels, it is intuitive that the reparative processes of the body may be overwhelmed, translating into higher cardiovascular morbidity. The anti-inflammatory effects of adiponectin indicate that it is an interesting protective factor for atherosclerosis development, particularly in those clinical situations associated with low plasma concentrations of adiponectin. It is conceivable that the use of recombinant adiponectin may become beneficial in the prevention of cardiovascular disease in selected patients. That suggest the increasing plasma adiponectin might be useful in preventing vascular restenosis after vascular intervention.[13]

Further investigations in patients with the above-mentioned states and other hypoadiponectinemic conditions are required to clarify these aspects of the potential therapeutic applications of this adipocytokine. [14]

Osamu et al 2009 [15] demonstrated a novel effect of natriuretic peptides (ANP and BNP) on the production of adiponectin by adipocytes in both experimental and clinical studies. By:

-First, they clearly demonstrated that pathophysiological and pharmacological concentrations of either ANP or BNP

increased adiponectin synthesis by primary cultured human adipocytes.

-Second, they showed that administration of recombinant ANP increased the plasma adiponectin level.

This study showed a negative correlation between adiponectin with insulin in Table (2).

Takashi Kadowaki and Toshimasa Yamauchi, 2005 demonstrated that the amount of HMW adiponectin complex, but not the total amount of adiponectin, was recently reported to be correlated with a thiazolidinedione-mediated improvement in insulin sensitivity and that proof that HMW is the most reactive form of adiponectin. Thiazolidinediones, a class of insulin-sensitizing antidiabetic drugs, increase adiponectin in insulin-resistant patients. In addition, high adiponectin concentrations are associated with a reduced risk of type II diabetes. [14]

For instance, adiponectin-deficient mice are both insulin resistantand prone to diabetes, and replenishment of adiponectin reverses insulin resistance in mice models of lipoatrophy, obesity, and type 2 diabetes. Furthermore, in humans, a diabetes susceptibility locus has been mapped to chromosome 3q27, the location of the gene encoding adiponectin whose expression is reduced in adipose tissue of obese Caucasians with type 2 diabetes.

Adiponectin's downstream metabolic effects include stimulation of glucose utilization and fatty-acid oxidation by the activation of AMP-activated protein kinase. Adiponectin decreases lipid synthesis and glucose production in the liver and causes a decrease in glucose and free fatty acid concentrations in the blood. In addition, triglyceride production is decreased and fat oxidation and energy dissipation in the muscle are increased Increased serum adiponectin concentrations are associated with increased insulin sensitivity and glucose tolerance.[14]

(Hakan Ekmekci, and Ozlem B. Ekmekci, 2009) [16] have shown that secretion of adiponectin by 3T3-L1 adipocytes requires phospatidylinositol 3-kinase (PI-3K), a major intermediate of insulin signalling activity. Insulin stimulated insulin receptor substrate 1 (IRS-1) associated PI-3K activity has been shown to be decreased in adipocytes of type 2 diabetic subjects.

Hanif Wasim et al 2009 [17] have shown that insulin infusion leads to decreased circulating adiponectin levels, consistent with the interpretation that insulin exerts an acute effect on adipocytes to decrease production and/or secretion of this adipocytokines.[17]

The vasodilation evoked by insulin is mediated by NO released from the endothelium by eNOS. It is well known that insulin can induce Akt phosphorylation through the activation of the IRS-1/phosphatidylinositol 3-kinase (PI3K) cascade that occurs after the physical interaction between IRS-1 and the PI3K subunit p85. Insulin able to stimulate interaction between IRS-1 and p85. [18]

So the action of adiponectin as insulin-sensitizor is important to reduce the damage of blocked aretery, the suggested mechanism of Action of Adiponectin as Insulin-sensitizing actions are:

Combs's et al., [19] in 2009 also reported that in adiponectin transgenic mice, reduced expression of gluconeogenic enzymes such as phosphoenolpyruvate carboxykinase and glucose- 6-phosphatase was associated with elevated phosphorylation of AMPK in liver. [19]

As shown in Table (1) it was found that insulin was significantly higher in patients group than control groups. This is first study for our knowledge that reported insulin level in AMI patients in Iraq.

Mechanisms accounting for the progressively worse coronary circulatory functional abnormalities observed in more severe states of IR remain unclear. They may be related to an increased number of vascular stressors, including elevated plasma levels of free fatty acids, triglycerides, oxidized low-density lipoprotein cholesterol, and oxidation-prone small dense low-density lipoproteins. Pro-inflammatory cytokines and inflammatory markers such as C-reactive protein and adipocyte-derived adipokines contribute to further decreases in production and inactivation of NO. Formation of reactive oxygen species in hyperglycemia, together with inactivation of protein kinase C, may further impair vascular function. [20]

Endothelial dysfunction, even in the absence of macrovascular coronary artery lesions, may be responsible for reduction in or failure to appropriately augment coronary flow, leading to myocardial ischemia. When coexisting with obstructive coronary artery disease, endothelial dysfunction may cause more severe and extensive stress-induced perfusion abnormalities, possibly because of stress-related sympathetically mediated vasoconstriction. [20], that agree with our noticed, from our observation that the patients with elevated level of insulin have high blood pressure.

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Adiponectin with Troponin

To our knowledge we are the first in our country who studied a relationship between adiponectin and troponin, as Table (2) shows that there is a negative correlation between adiponectin and troponin, as troponin referred to the infarction size this negative correlation gives a good idea about adiponectin as it may have good effect (as it explained previously in adiponectin and its effect on artery wall by increasing NO production in addition to its ability to decrease Intra Cellular Adhesion Molecular) on heart by decreasing the infarction area size.

Conclusions

- The significant increase in adiponectin in AMI may be related to inflammation.
- Adiponectin is negatively associated with insulin from this negative correlation may be due to ability of adiponectin to improvement of sensitivity of insulin.

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الخلاصة

النسيج الدهني يُعْرَفُ على نحو متزايد كمنظّم رئيسي لميزانِ الطاقةِ، يَلْعبُ دوراً نشيطاً في خزنِ الدهون و ويفرز تشكيلة واسعة من المُنتَجاتِ الإفرازيةِ التي قَدْ تُشتَرَكُ في النشوء المرضي لكثير مِنْ التعقيداتِ المرتبطة بصورة مباشرة بالسمنةِ من اهم المركبات التي يفرزها النسيج الدهني هرمون

adiponectin. الأنسيولين هو الهورمون الذي ينظيم أيضِ الجلوكوزَ والطاقةَ في الجسمِ.

Troponin هو مركّب مِنْ ثلاثة بروتينِات تنظيم الإنكماشِ العضلي في العضلات الهيكليةِ والقلبيةِ.

تَضمّنتْ الدراسةُ ٥٠ مريضُ بالذبحة الصدرية اما المجموعة المسيطرة فقد تضمنت أربعون من الأشخاص الأصحاء. مستويات adiponectin والأنسيولينِ و troponin تم قياسها

مستويات adiponectin والأنسولين و troponin كانت مرتفة بشكل معنوي ($p <0.001$)، كان هناك إرتباطُ سلبيُ بين adiponectin والأنسولين و troponin في مرضى الذبحةّ الصدرية.

وجود زيادة معنوية في مستويات adiponectin، في مرضى الذبحةّ الصدرية وهذه قَد تكون مُتَعَلّقة بالالتهاب. هنالك علاقة عكسية بين adiponectin والأنسولين.