EFFECT OF ACUTE MYOCARDIAL INFARCTION ON SOME PRO-INFLAMMATORY CYTOKINES AND THEIR RELATIONSHIP TO SERUM LIPIDS

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Abstract

The effect of acute myocardial infarction (AMI) on some pro-inflammatory cytokines, as well as their relationship to serum lipids needs to be clarified. The current work was carried on 45 male subjects. Their ages ranged from 43-56 years. They were 25 patients with AMI compared with 20 healthy subjects matched for the same age and sex as controls. For all subjects, serum interleukin-1 (IL-1), interleukin-6 (IL-6) and tumor necrosis factor –alpha (TNF-α) and lipid profile were assayed 24 hours post infarction and 6 weeks later on.

The results of this work showed that serum IL-1, IL-6 and TNF-α were significantly increased 24 hours post infarction and 6 weeks later compared with the control group ($p<0.05$). However, serum IL-1, IL-6 and TNF-α were significantly decreased 6 weeks later after the onset of infarction compared with the same parameters 24 hours post infarction ($p1 < 0.05$).

Also, lipid profile showed a significant change in serum low density lipoprotein-cholesterol(LDL-c), very low density lipoprotein-cholesterol(VLDL-c) and triglycerides 24 hours post infarction and 6 weeks post infarction while serum total cholesterol and high density lipoprotein-cholesterol(HDL-c) showed a significant change 6 weeks post infarction($p<0.05$) ..However, serum total cholesterol and HDL-c had non-significant change 24 hours post infarction when they compared with the control group.

Comparative study of serum lipid profile 6 weeks post infarction versus 24 hours post infarction, the data revealed that; a significant increase in serum total cholesterol and serum LDL-c while a significant

decrease was found in serum HDL-c, VLDL-c and triglycerides ($p1<0.05$)

Correlation study at 24 hours post infarction, revealed that serum IL-1, IL-6 and TNF-α had non-significant correlation with serum lipid profile .On the other hand serum IL-6 had a significant negative correlation with serum LDL (r= - 0.550) and serum TNF-α had a significant positive correlation with serum VLDL (r=0.582) and triglycerides (r=0.498) 6 weeks later after the onset of AMI

So, this study revealed that ,serum IL-1, IL-6 and TNF-α were prominent markers for inflammation following AMI either at the onset or shortly thereafter . They had been implicated in abnormal lipid metabolism. Serum cholesterol levels were no longer valid 24 hours after AMI while serum triglycerides is preferred for diagnosis of hyperlipidaemia . However, it remains to be determined whether these markers of inflammation actually have a causal relation with the cardiovascular disease, or simply reflect the underlying disease process. Such determination becomes important with the potential use of these markers in targeting preventive therapies.

INTRODUCTION & AIM OF WORK

In the past years coronary atherosclerosis and plaque rupture have been characterized not only as a problem of growth of smooth vascular cells, of activated thrombocytes and coagulation but also of inflammation[1] **.** Inflammatory process within the vessel wall is considered to be crucial for initiation and progression of atherosclerosis[2]**.** Atherosclerosis is a chronic disease that, from its origins to its ultimate complications, involves inflammatory cells (T cells, monocytes, macrophages), inflammatory proteins (cytokines, chemokines), and inflammatory responses from vascular cells (endothelial cell expression of adhesion molecules) [3] .

Human express two distinct molecular forms of IL-1, called IL-1α and IL-1β . These are peptides,159 and 153 amino acids long, respectively, that are encoded by separate genes and share only 26 % amino acid sequence similarity, but have virtually identical potency and biological activities and bind with about the same affinity to the same cell surface receptors. Furthermore, IL-6 is a single polypeptide, but can be glycosylated and phosphorylated to various degree. It is produced by many cell types , including activated B and T cells, monocytes and endothelial cells. Moreover, tumor necrosis factor-alpha (TNF-α) is synthesized as a propeptide and then processed intracellularly by an enzyme called TNF- α converting enzyme (TACE) to its mature , secreted form, which is 157 amino acids long [4].

However, the effect of AMI on some pro-inflammatory cytokine responses especially IL-l, IL-6 and TNF-α as well as their relationship to serum lipids needs to be clarified.

SUBJECTS AND METHODS

This study was conducted on 45 subjects throughout the period from May to December 2002, in Benha faculty of medicine and Benha University hospital, Zagazig University. All subjects were males & their ages ranged from 43 to 56 years. They were classified into 2 groups:

Group I: They comprised 20 healthy subjects their ages ranged from 43-56 years with the mean value (49.7±4.05) served as a control group.

Group II: They comprised 25 patients suffering from acute myocardial infarction. Their ages ranged from 43-56 years with the mean value (51.08± 4.02)**.**

Acute myocardial infarction was diagnosed when the following criteria were present : typical prolonged chest pain, typical evolutionary changes in ECG characteristic of infarction and transient significant elevation of the serum cardiac enzymes [5]

All subjects included in this study were subjected to the followings : Full history , through clinical examination, determination of blood pressure, 12-lead surface resting E.C.G tracing and laboratory investigations for serum assay of glucose [6] ,cholesterol [7], HDL-c [8], LDL-c [8], VLDL-c[8], triglycerides[9], aspartate aminotransferase (AST) [10] ,alanine aminotransferase (ALT) [10], creatinine[11],creatine kinase-MB[12] ,IL-1 by radioimmunoassay using advanced magnetics Inc. kit [13] **,** IL-6 and TNF-α by ELISA technique according to the instructions of kit manufacture (Bio-source, Fleurus, Belgium*)* and as described elsewhere[14,15]

Sampling

About 7 cc. venous blood were taken after admission "stress period" by 24 hours post infarction while fasting and six weeks later on "non-stress period" from patients with AMI and their corresponding controls.

These fasting samples were divided into 2 parts. The first part (0.5 cc) was taken on small amount of Ethylene Diamine Tetra-acetic Acid (EDTA) powder for determination of blood picture. The second part (6.5 cc) was left to be clotted, centrifuged and the sera separated were used for determination of creatine kinase isozyme (CK-MB) , fasting glucose, AST, ALT, creatinine, cholesterol ,HDL-c, LDL-c, VLDL-c and triglycerides while the remaining sera were kept frozen at – 70^{o}C for later determination of IL-1, IL-6 and TNF-α .

Exclusion criteria:

All patients with haematological disorders, diabetes mellitus, cancer, autoimmune disease, obesity , heart failure as well as patients on hypolipidaemic drug therapy and smokers were excluded from the study**.**

Statistical analysis:

The results of this work were tabulated and statistically analyzed using mean values , standard deviation , student t- test, paired t- test and correlation coefficient (r). p values more than 0.05 were considered insignificant while p values less than 0.05 were considered significant [16] .

RESULTS

Table (1) showed that, the mean values of serum IL-1(6.82± 0.974 Pg/ml vs 1.95 ± 0.272 Pg/ml), IL-6 (7.12± 0.335 Pg/ml vs 2.84 ± 0.143 Pg/ml) and TNF-α (4.05± 0.228 Pg/ml vs 2.37 ± 0.296 Pg/ml) were increased 24 hours post infarction compared with the control group with statistical significant differences ($p<0.05$). Also , the mean values of serum IL-1 (5.45±0.851 Pg/ml vs 1.95±0.272 Pg/ml) , IL-6 (5.42±0.407 Pg/ml vs 2.84±0.143 Pg/ml) and TNF- α (3.63±0.273 Pg/ml vs 2.37±0.296 Pg/ml) were increased 6 weeks later after AMI compared with the control group with statistical significant differences ($p<0.05$).

On the other hand, comparative study of patients 6 weeks after AMI versus 24 hours post infarction , the mean values of serum IL-1 (5.42±0.851 Pg/ml vs 6.82± 0.974 Pg/ml), IL-6 (5.42 ± 0.407 Pg/ml vs 7.12 ± 0.335 Pg/ml) and TNF- α (3.63 ± 0.273 Pg/ml vs 4.05 ± 0.228 Pg/ml) were decreased with statistical significant differences ($p1 < 0.05$).

Furthermore, table (2)) showed that, the mean values of serum VLDL-c (35.54 ±2.537 mg/dl vs 15.80 ±3.073 mg/dl) ,triglycerides (177.72 ±12.687 mg/dl vs 78.85 ±15.170 mg/dl) were increased with statistical significant difference ($p<0.05$) while there were non-significant changes in serum total cholesterol (190.96 ±6.611 mg/dl vs 187.95 ±5.155 mg/dl) and HDL-c (64.68 ±2.734 mg/dl vs 66.00 ±2.294 mg/dl) in 24 hours post infarction compared with the control group. However, the mean values of serum LDL-c (90.74 ±8.590 mg/dl vs 106.15 ±3.405 mg/dl) was decreased with statistical significant differences ($p1< 0.05$) when compared with the control group.

On the other hand, comparative study of patients 6 weeks after AMI versus 24 hours post infarction , the mean values of serum total cholesterol (216.04 ±16.134 mg/dl vs 190.96 ±6.611 mg/dl) and LDL-c

(151.09 ±15.834 mg/dl vs 90.74 ±8.590 mg/dl) were increased with statistical significant differences ($p1 < 0.05$) while the mean values of serum HDL-c (31.68 ±6.290 mg/dl vs 64.68 ±2.734 mg/dl) ,VLDL-c (23.27 ±3.058 mg/dl vs 35.54 ±2.537 mg/dl) ,and triglycerides (166.36 ±15.290 mg/dl vs 177.72 ±12.687 mg/dl) were decreased with statistical significant differences ($p1 < 0.05$)

Moreover, table (3) showed the correlation study at 24 hours post infarction. The data revealed that, serum IL-1 and cholesterol ($r = -0.218$), HDL-c ($r= - 0.341$), LDL-c ($r=0.216$), VLDL-c ($r=0.139$) and triglycerides ($r=0.111$) had non –significant correlation . Also, serum IL-6 and cholesterol ($r = 0.231$) HDL-c($r= - 0.310$), LDL-c ($r=0.198$), VLDL-c ($r=0.201$) and triglycerides ($r = 0.210$) showed non-significant correlation . Additionally, serum TNF- α and cholesterol ($r = 0.246$) HDL-c ($r=0.278$), LDL-c ($r=0.225$), VLDL-c ($r=0.311$) and triglycerides ($r = 0.190$) showed non-significant positive correlation.

In addition, table (4) showed the correlation study 6 weeks after AMI. The data revealed that, serum IL-1 and cholesterol ($r = 0.222$), HDL-c ($r=0.125$), LDL-c ($r= -0.214$), VLDL-c ($r=0.132$) and triglycerides ($r=0.289$) had non – significant correlation . Also, serum IL-6 and cholesterol ($r = 0.206$) HDL-c ($r= - 0.312$), LDL-c ($r= -0.550$), VLDL-c ($r=0.210$) and triglycerides ($r = 0.159$) showed non-significant correlation . Additionally, serum TNF- α and cholesterol ($r = 0.325$) HDL-c ($r=0.195$), LDL-c ($r= -0.232$), VLDL-c ($r=0.582$) and triglycerides ($r = 0.498$) showed non-significant correlation.

Table (1): Mean ± SD and P values of IL-1, IL-6 and TNF- α in patients with acute myocardial infarction (AMI) at 24 hours post infarction and 6 weeks later compared with the control group.

AMI / Immuno-chemical parameters	Control group (n = 20)	24 hours post infarction (n = 25)	6 weeks later (n = 25)
Serum IL-1 (Pg/ml)	1.95 ± 0.272	6.82 ± 0.974 p < 0.05	5.45 ± 0.851 p < 0.05 p1 < 0.05
Serum IL-6 (Pg/ml)	2.84 ± 0.143	7.12 ± 0.335 p < 0.05	5.42 ± 0.407 p < 0.05 p1 < 0.05
Serum TNF-α (Pg/ml)	2.37 ± 0.296	4.05 ± 0.228 p < 0.05	3.63 ± 0.273 p < 0.05 p1 < 0.05

p : probability versus control group.

p1 : Probability versus AMI at the onset of stress

Table (2): Mean ± SD and p values of serum lipid profile in patients with acute myocardial infarction (AMI) at 24 hours post infarction and 6 weeks later compared with the control group.

AMI / Serum lipid profile	Control group (n = 20)	24 hours post infarction (n = 25)	6 weeks later (n = 25)
S. total cholesterol (mg/dl)	187.95 ±5.155	190.96 ±6.611 p: N.S	216.04 ±16.134 p <0.05 p1<0.05
Serum HDL-c (mg/dl)	66.00 ±2.294	64.68 ±2.734 p: N.S	31.68 ±6.290 p <0.05 p1<0.05
Serum LDL-c (mg/dl)	106.15 ±3.405	90.74 ±8.590 p <0.05	151.09 ±15.834 p <0.05 p1<0.05
Serum VLDL-c (mg/dl)	15.80 ±3.073	35.54 ±2.537 p <0.05	33.27 ±3.058 p <0.05 p1<0.05
Serum triglycerides (mg/dl)	78.85 ±15.170	177.72 ±12.687 p <0.05	166.36 ±15.29 p <0.05 p1<0.05

p : Probability versus control group.

p1 : Probability versus AMI at the onset of stress

Table (3): correlation coefficient (r) between different cytokines (IL-1, IL-6 and TNF-α) and serum lipids profiles at 24 hours post infarction.

Groups / Immuno-chemical parameters		24 hours post infarction				
		S.cholesterol	**S.HDL-c**	**S.LDL-c**	**S.VLDL-c**	**S.triglycerides**
IL-1	(r)	- 0.218	- 0.341	0.216	0.139	0.111
	(p)	NS	N.S	N.S	N.S	NS
IL-6	(r)	0.231	- 0.310	0.198	0.201	0.210
	(p)	NS	N.S	N.S	N.S	NS
TNF-α	(r)	0.246	0.278	0.225	0.311	0.190
	(p)	NS	N.S	N.S	N.S	NS

N.S = Non significant.

Table (4): correlation coefficient (r) between different cytokines (IL-1, IL-6 and TNF-α) and serum lipids profiles in patients with AMI at 6 weeks later .

Groups / Immuno-chemical parameters		6 weeks post infarction				
		S.cholesterol	**S.HDL**	**S.LDL**	**S.VLDL**	**S.triglycerides**
IL-1	(r)	0.222	0.125	- 0.214	0.132	0.289
	(p)	N.S	N.S	N.S	N.S	NS
IL-6	(r)	0.206	0.312	- 0.550	0.210	0.159
	(p)	N.S	N.S	<0.05	N.S	NS
TNF-α	(r)	0.325	0.195	- 0.232	0.582	0.498
	(p)	N.S	N.S	N.S	<0.05	<0.05

N.S = Non significant.

DISCUSSION

Our results showed a significant elevated levels of serum IL-1, IL-6 and TNF-α 24 hours post infarction (AMI). "Stress period" and also six weeks later "Non-stress period". Moreover, their levels continued to be relatively high as they did not reach the normal level values detected in the healthy matched control group (Table 1).

Proinflammatory cytokines are implicated in the etiology of coronary heart disease (CHD), and are also sensitive to emotional stress[17].

Plasma IL-1 is produced by myocardial cells in response to injury. IL-1 is not acting alone under circumstances of myocardial injury, but in concert with other proinflammatory molecules and their effectors [18].

The proinflammatory cytokine IL-1 is expressed mainly within the endothelium of atherosclerotic plaques and may be linked with inflammatory mechanisms of anthropogenesis. IL-1 action is complex and regulated in part by its naturally occurring inhibitor, the IL-1 receptor antagonist (IL-1 ra). IL-1 causes multiple responses within the endothelium but most notably induces the expression of adhesion molecules, which promote monocyte recruitment and infiltration into the arterial wall [19] .

Also, IL-1 plays a role in mediating acute inflammation during ischaemia-reperfusion injury in the heart, which leads to both necrosis and apoptosis of cardiomyocytes [20].

Dewberry et al.,[19] reported that IL-1 has been detected in plaque cells, in luminal extracellular cells and macrophages, in the sera of patients with ischaemic heart disease and unstable angina and in myocytes and infiltrating leukocytes in human dilated cardiomyopathy.

The increase of serum IL-1 in patient with acute myocardial infarction was in accordance with the results of ***Hasdai et al .,***[21] ***Pudil et al.,***[22] ***Biasucci et al.***[23]***, and Balbay et al.,***[24]**.**

Moreover, ***Yudkin et al.,***[25] proposed a key role for the proinflammatory cytokine IL-6 in several mechanism that contribute to the development of coronary heart disease (CHD). Firstly, IL-6 is a powerful inducer of hepatic acute phase response like C-reactive protein & fibrinogen. Elevated levels of fibrinogen, a strong risk factors for CHD with autocrine and paracrine activation of monocytes by IL-6 in the vessel wall contributing to the deposition of fibrinogen. Secondly, IL-6 decreases lipoprotein lipase (LPL) activity and monomeric LPL levels in plasma which increases macrophage uptake of lipids. In fatty streaks and in the atheromatous "Cap" and "Shoulder" regions, macrophage foam cells and smooth muscle cells express IL-6, suggesting a role for this cytokine along with IL-1 and TNF-α in the progression of arteriosclerosis. Both of these cytokines induce the release of IL-6 from several cell types including smooth muscle cells (SMC). During vascular injury SMC are exposed to platelets or their products and cytokine reduction by SMC further contribute to vascular damage. Thirdly, IL-6 stimulates the hypothalamo- pituitary – adrenal (HPA) axis, activation of which is associated with central obesity, hypertension and insulin resistance. So, IL-6 was proposed to play a role in the pathogenesis of CHD through a combination of autocrine, paracrine and endocrine mechanisms.

However, the increased serum level of IL-6 in patients with AMI of this study , may be explained through the experimental study of ***Burger et al.,***[26] They found that catecholamines are increased during stressful condition with subsequent induction of cardiac hypertrophy.

Only small amount of IL-6 mRNA was detected in unstimulated rat cardiac fibroblasts. There was an increase of IL-6 after stimulation with norepinephrin by 50 folds.. Addition of alpha and beta adrenergic receptor antagonist, prevented almost completely the norepinephrin induced synthesis of IL- 6 m RNA.

These data confirm the previous report of ***Yudkin et al.,***[25] as IL-6 play an important autocrine and paracrine role in cardiac disease states associated with hypertrophy.

The increase of serum IL-6 in our patients with AMI was in agreement with the results of ***Gabriel et al.,***[27] ***Deliargyris et al.,***[28] ***Kucharz and Wilk ,***[29] ***Kanda et al.,***[30] ***Biasucci et al.,***[23] ***Balbay et al.,***[24] ***Ikeda et al.,***[31] ***Blankenberg et al.,***[2] ***and Buratti et al.,***[32] **.**

In addition, the heart is a tumor necrosis factor (TNF) - producing organ. Both myocardial macrophages and cardiac myocytes themselves synthesize TNF. Accumulating evidence indicates that myocardial TNF is an autocrine contributor to myocardial dysfunction and cardiomyocyte death in ischaemia-reperfusion (IS/R) injury. TNF contributes to post ischemic myocardial dysfunction via direct depression of contractility and induction of myocyte apoptosis. IS/R activates myocardial P38 mitogen-activated protein (MAP) kinase and nuclear factor- kappa B (NF-KB), which lead to TNF production.[33]. Also, NF-KB activation inducing adhesion molecules and facilitating leukocyte infiltration. Meanwhile, myocardial IS/R injury had been improved in mice lacking TNF-α [34].

However, plasma concentrations of TNF-α are persistently elevated among post-myocardial infarction (MI) patients at increased risk for recurrent coronary events [35]. These data support the hypothesis that a persistent inflammatory instability is present among stable patients at increased vascular risk. ***Irwin et al.,***[36] observed that, TNF-α expression in

myocardium of rats after ligation of the left anterior descending coronary artery was persisted after infarction even in otherwise normal myocardial segments. In that study, TNF-α gene and protein expression persisted in myocytes over time, which suggests a possible long-term role of this cytokine in vascular remodeling. Anyhow, ischemia alone is sufficient to induce TNF- α mRNA expression and peptide synthesis in the myocardium [37].

The previous reports explained the persistence elevation of serum TNF-α found in our patients with AMI . The current data were compatible with the results of ***Halawa et al.,[38] Pudil et al.,[22] and Balbay et al.,[24].***

At 24 hours post infarction; a significant increase in serum VLDL-c and triglycerides ($p<0.05$) was observed while non-significant change was found in serum total cholesterol and HDL-c. However, serum HDL-c was significantly decreased ($p<0.05$) when they compared with the control group. On the other hand, serum total cholesterol, LDL-c, VLDL-c, and triglycerides were significantly increased while serum HDL-c was significantly decreased 6 weeks post infarction when compared with the control group ($p<0.05$) (Table 2).

Comparative study of serum lipid profile 6 weeks post infarction versus 24 hours post infarction, the data revealed ; a significant increase in serum total cholesterol and serum LDL-c but a significant decrease in serum HDL-c, VLDL-c and triglycerides ($p1<0.05$).

The correlation study at 24 hours post infarction, revealed that, serum IL-1, IL-6 and TNF-α had non-significant correlation with serum lipid profile (table 3) .On the other hand serum IL-6 had a significant negative correlation with serum LDL-c (r=0.550) and serum TNF-α had a significant positive correlation with serum VLDL-c (r=0.582) and triglycerides (r=0.498) 6 weeks later after the onset of AMI (table 4).

It was found that; in the acute phase response to myocardial injury or other trauma or surgery, total and LDL cholesterol levels are markedly decreased [39]**.** Cytokines like IL-1, IL-6 , TNF- α and the interferons can decrease lipoprotein lipase and increase lipolysis in cultured fat cells. In vivo many cytokines increase serum triglycerides by increasing very-low – density lipoprotein production[40]. Also, C-reactive protein binds selectively with VLDL-c and interferes with its catabolism, thereby increasing serum triglycerides concentration[41] and this may explain the significant increase in serum VLDL-c and triglycerides **.** The significant decrease in serum LDL-c in our patients 24 hours post infarction may be due to the upregulation of LDL-receptor in hepatic cells by IL-6 which lead to a decrease in serum LDL-cholesterol level [39]**.**

These results were nearly similar to the findings of ***Chamsi-Pasha et al.,[42] and Schumacher et al***.,[43] who found a non-significant change of serum total cholesterol in patients 24 hours post infarction compared with the control group. However the current results as regard serum HDL and LDL were in agreement with the finding of ***Schumacher et a***l.,[43] and not compatible with the results of ***Carlsson et al.,[44]and Wattanasuwan et al.[45]***

In addition, the hypertriglyceridaemia seen in these patients after admission and 6 weeks later was in agreement with the results of ***Rowe***

et al.,[41] and ***Wattanasuwan et al***.,[45]. Some reports[42, 47]showed that serum triglycerides had non - important changes either after admission or 3 months later on while other report[46] showed non-significant changes at admission but marked increase at discharge after four to five weeks.

The cause of this discrepancy between our results and the others may be due to the time of taken the samples , the duration of follow up, the type of thrombolytic therapy taken as well as many studies did not implicate the role of cytokines in abnormal lipid metabolism in patients with AMI.

We could conclude that, serum IL-1, IL-6 and TNF-α were prominent markers for inflammation following AMI either at the onset or shortly thereafter . They had been implicated in abnormal lipid metabolism .Serum cholesterol levels were no longer valid 24 hours after AMI while serum triglycerides were preferred for diagnosis of hyperlipidaemia .However, it remains to be determined whether these markers of inflammation actually have a causal relation with cardiovascular disease, or simply reflect the underlying disease process. Such determination becomes important with the potential use of these markers in targeting preventive therapies.

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الملخـــص العـــربى

تأثير الإحتشاء القلبى الحاد على بعض السيتوكينات المسببة للإلتهابات وعلاقتهم بالدهون فى مصل الدم .

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تلعب الإلتهابات دورا كبيرا فى حدوث مرض تصلب الـشرايين وبالتـالى حـدوث الجلطة الدموية التى تتسبب فى حدوث الإحتشاء القلبي الحاد .وهناك العديـد مـن الخلايـا المسئولة عن حدوث الإلتهابات مثل الخلايا الليمفاوية "ت" والتي تفرز السيتوكينات .

يهدف هذا البحث إلى دراسة تأثير الإحتشاء القلبي الحاد على بعـض الـسيتوكينات المسببة للإلتهابات وعلاقتهم بالدهون فى مصل الدم .

وقد أجريت هذة الدراسة على 45 رجلا تتراوح أعمارهم من 43 – 56 سـنة وهـم عبارة عن 25 مريضا بالإحتشاء القلبى الحاد و20 رجلا من الأصحاء كمجموعـة ضـابطة .وتم قياس مستوى الإنترلوكين –1 والإنترلوكين –6 وعامل تآكل الورم – ألفا وصورة كاملة للدهون بالدم وذلك بعد 24 ساعة من حدوث المرض ثم بعد 6 أسابيع أخرى .

وقد أظهرت نتائج هذا البحث وجود زيادة ذات قيمة إحصائية فى مستوى الإنترلوكين –1 والإنترلوكين –6 وعامل تآكل الورم –ألفا بمصل الدم بعد 24 ساعة من بداية الأحتـشاء القلبى الحاد وكذلك بعد 6 أسابيع عند مقارنتهم بالمجموعة الضابطة .أمـا بالنـسبة للـدهون بمصل الدم فقد حدثت زيادة ذات قيمة إحصائية فى الدهون الثلاثية بينما لم يتأثر الكوليسترول بدرجة كافية بعد 24 ساعة على حين حدثت زيادة فيهما وذات دلالة إحصائية بعد 6 أسـابيع من حدوث المرض . كما وجد أن هناك علاقة عكسية وذات قيمة إحصائية بين الإنترلوكين – 6 والبروتينات المحملة بالدهون ذات الكثافة المنخفضة وعلى الجانب الآخر فأن هناك علاقة طر دية بين عامل تآكل الورم – ألفا والدهون الثلاثية والبروتينات المحملـة بالـدهون ذات الكثافة المنخفضة جدا وذلك بعد 6 أسابيع من حدوث الإحتشاء القلبى الحاد .

نستخلص من هذا البحث أن الإنترلوكين –1 والإنترلوكين –6 وعامل تآكل الورم – ألفا له دلالة واضحة فى حدوث الإلتهابات التى تلى الإحتشاء القلبى الحاد سواء عنـد بدايتـة أوحتى بعد مرور 6 أسابيع على حدوثة .كما أنه يخلص إلى أن السيتوكينات تلعب دورا مهما فى الخلل الذى يحدث فى التمثيل الغذائى للدهون . وأيضا يوضح هذا البحث أن قياس مسنوى الدهون الثلاثية تفضل على الكوليسترول فى تشخيص زيادة الدهون بمصل الدم عنـد بدايـة

المرض. ويبقى السؤال هل دلالات الإلتهاب هذة لها علاقة سببية فى حدوث الإحتشاء القلبـــى الحاد أم أنها تعكس فقط مجرد حدوث المرض ؟