CIRCULATING LEVELS OF sCD40L, sFas, VCAM-1 and ICAM-1 IN ACUTE AND CHRONIC ISCHEMIC HEART FAILURE IN MALE PATIENTS

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ABSTRACT

Background and aim of work: Immunomodulatory mediators play a crucial role in the pathogenesis of heart failure (HF). Vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-l (ICAM-1) are important inflammatory mediators of leukocyte adhesion to vascular endothelium and their plasma levles increase in chronic and acute inflammation. Furthermore, the immune modulators CD40 ligand (CD40L) and sFas have been receiving increased attention, since they play a key role in the pathophysiology of multicellular vascular events such as thrombosis, inflammation, and atherosclerosis. Based on the previous facts, we planned to evaluate the role of plasma VCAM-1, ICAM-1, and serum soluble CD40L (sCD40L) and sFas in HF. **Subjects and methods:** The present study was done on 30 male patients with HF who were classified according to the type of HF to: 15 patients with chronic HF (CHF) due to ischemic causes and 15 patients with acute myocardial infarction (AMI) complicated with HF during acute phase (AHF). Ten age-matched healthy male volunteers were taken as controls. Plasma levels of VCAM-1, ICAM-1, and serum levels of sCD40L and sFas were measured in all groups. Lipid profile, creatine phosphokinase and its MB fraction were also measured. **Results:** There were significant increase in plasma levels of VCAM-1 ($P<0.05$), ICAM-1 ($P<0.001$), and serum levels of sCD40L ($P<0.001$) and sFas ($P<0.001$) in both CHF and AHF patients compared to control subjects. There was a significant positive correlation between VCAM-1 and sCD40L, as well as sFas in CHF ($r = 0.46$, $P = 0.03$ and $r = 0.47$, $P = 0.02$ respectively) and a significant positive correlation between ICAM-1 and sCD40L**,** as well as sFas in AHF ($r = 0.551$, $P<0.01$ and $r = 0.49$, $P = 0.012$ respectively). Also, there was a positive significant correlation between sCD40L and low-density lipoprotein cholesterol in both CHF and AHF cases ($P<0.05$). Conclusion: sCD40L, sFas, VCAM-1, and ICAM-1 could be used as markers that might predict cardiovascular events in patients with chronic and acute heart diseases.

Key words: *sCD40L, sFas, VCAM-1 and ICAM-1, Heart failure.*

INTRODUCTION

Coronary heart disease is a major cause of hospitalization and mortality worldwide. Chronic inflammation and endothelial activation may be present in patients with heart failure (HF) and may contribute to the pathogenesis

and progression of that disease[1]. Cellular adhesion molecules**,** namely, intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1), members of immunoglobulin superfamily genes, are poorly expressed by the resting endothelium [2]. They are upregulated during inflammatory atherogenesis and might be an index of endothelial activation or even a molecular marker of early atherosclerosis[3]. A variety of stimuli may impair endothelial function rendering it susceptible to leucocytes adhesion with expression of large amount of VCAM-I and ICAM-I [4]. Each molecule of ICAM-1 (also known as CD154) is characterized by five distinct immunoglobulin-like domains, a transmembrane domain, and cytoplasmic tail. VCAM-1 (also known as CD 106) is a seven domains. In atherosclerosis, it has been shown that ICAM-1 is upregulated in sites prone to atherosclerosis development[5]. Baseline levels of ICAM-1 are increased many years before a first myocardial infarction (MI) occurs[6,7].

CD40 ligand (CD40L), a transmembrane protein structurally related to tumor necrosis factor, was originally identified on $CD4^+$ T cells, and can be expressed by macrophages, T cells, endothelium, and smooth muscle in atherosclerotic lesions in vivo. It has been estimated that more than 95% of the circulating soluble CD40L (sCD40L) is derived from platelets and has been shown to activate endothelium[8]. Both membrane-bound and sCD40L forms of that ligand may interact with CD40, which is constitutively expressed on B cells, macrophages, endothelial cells, and vascular smooth muscle cells resulting in various inflammatory responses, matrix degradation, and also thrombus formation [9].

The pro-apoptotic protein apoptosis-stimulating fragment (Fas) may exert an adverse effect on the progression of HF, whereas Fas ligand induces apoptosis when binding to cell-surface bound Fas [10]. Fas and Fas ligand are transmembrane proteins of the tumor necrosis factor family of receptors and ligands [11]. Soluble Fas (sFas) may competitively inhibit the binding of Fas ligand to surface-bound Fas, thereby exerting a potential anti-apoptotic effect [12]. The risk of a clinical event increases with sFas concentrations in patients with advanced coronary disease [13]. Experimental findings have indicated a protective effect of sFas in the myocardium. sFas inhibits apoptosis of muscle cells in cell culture and improves the survival of animals with HF[14].

In the present study, we planned to examine serum levels of sCD40L and sFas, and plasma levels of ICAM-1 and VCAM-1; and their roles in the pathogenesis of coronary heart disease in patients with acute HF (AHF) following MI and chronic HF (CHF) of ischemic origin.

SUBJECTS & METHODS

The study was carried out on 30 male patients who were admitted to Cardiology Department of Zagazig University Hospital and diagnosed as HF, and 10 age-matched normal healthy men volunteers were taken as controls. Patients with HF were

classified according to the type of HF to: 15 patients with CHF due to ischemic causes and 15 patients with MI complicated with HF during acute phase (AHF). CHF was defined as dyspnea, fatigue at rest or on exertion for more than 3 months, and The diagnosis of AMI was determined by the finding of typical rise and fall of creatine phosphokinase (CPK)-MB with at least one of the followings criteria: (a) ischemic symptoms, (b) development of pathologic Q waves on the ECG, (c) ECG changes indicating ischemia (ST segment elevation or depression), or coronary arterial intervention [(15)]. The age of patients with AHF ranged from 47-69 years (mean 58±6 years). The age of CHF patients ranged from 48-71 years (mean 58±8 years). The age of controls ranged from 47-70 years (mean 57±7 years). There were no significant differences as regard age in the three groups.

Eight ml blood sample was withdrawn from each subject after overnight fasting. Blood samples from patients with AHF were collected within the first 24 hours of diagnosis of MI. Plasma samples were used for detection of VCAM-1 and ICAM-1, and serum samples were used for determination of sCD40L, sFas, lipid profile, CPK, and CPK-MB. All samples were stored at -20°C till used.

Plasma levels of VCAM-1 and ICAM-1 were measured using commercially available kits for quantitative enzyme-linked immunosorbent assay (ELISA) techniques (Diaclone Research, France). These kits are solid phase sandwich ELISA. Monoclonal antibodies specific for each component had been coated onto the wells of the microtitre strips [(7)].

Measurement of serum levels of sCD40L was done by using a commercially available ELISA kit based on the sandwich principle, according to the manufacturer's instructions (sCD40L ELISA test kit; Bender Medsystems, Vienna, Austria). This assay is highly specific for the quantitative determination of sCD40L in human sera and has a minimum sensitivity of 0.095 ng/ml. Intra- and inter-assay coefficients of variation were 4.0 and 6.8% respectively, and mean recovery was 91%.

Serum levels of sFas were determined by ELISA method using sFas ELISA kit (Mitsubishi Bioclinical Laboratories, Inc., Tokyo, Japan). This ELISA system adopted a specific sandwich method using peroxidase labeled monoclonal antibody to the extracellular domain of Fas and polyclonal antibody to the intracellular domain[(17)].

Measurements of lipid profile; total cholesterol (TC) was measured according to **Flegg, (1973)**[(18)], triglycerides (TG) were measured according to **Buccolo** and **David, (1973)**[(19)], high-density lipoprotein-cholesterol (HDL-C) was measured according to **Kostner, (1977)**[(20)] and low-density lipoprotein-cholesterol (LDL-C) was calculated by using Friedwald formula [(21)].

CPK and CPK-MB fraction were measured by colorimetric method using kit by Sclavo (Italy) according to **Lang** and **Würzburg (1982)** [(22)].

Statistical Analysis:

The data was processed by the SPSS (SPSS Inc., Chicago, IL,

version 11, USA) statistical package to analyze the data. All results were expressed as mean ± SD. Comparison among groups was made by student's *t*-test (unpaired). P-value ≤0.05 was considered significant.

RESULTS

There were highly significant increase in CPK and CPK-MB activities in patients with AHF as compared to either controls or patients with CHF (P<0.001 in both groups), but there was no statistically significant difference in these parameters between patients with CHF and controls. There were no statistically significant differences between patients with AHF and patients with CHF or either of these groups compared to controls as regard TC, TG, and HDL-C. But; as regard LDL-C, there were highly significant increase the mean serum level in either patients with AHF or patients with CHF compared with controls (P<0.001), and significant increase in patients with AHF compared to patients with CHF (P = 0.02), Table 1.

There were highly significant increase in the mean plasma levels of VCAM-1 and ICAM-1 and in mean serum levels of sCD40L, and sFas in either patients with AHF or patients with CHF compared with controls, but no significant differences were found in these parameters comparing patients with AHF vs patients with CHF, Table 2.

Pearson's correlation (r) was done to find relations between the studied parameters in patients with AHF and patients with CHF. In patients with AHF: as regard serum sCD40L, a highly significant positive correlation was found with both TC (r = 0.657, P=0.002) and plasma ICAM-1 (r = 0.551, P<0.003), and a significant positive correlation with plasma VCAM-1 (r = 0.473, P = 0.028) and LDL-C (r = 0.511, P = 0.011), and insignificant correlation with CPK, CPK-MB, TG, HDL-C, and serum sFas; as regard plasma VCAM-1 a significant positive correlation was found only with serum sCD40L and insignificant correlation with other parameters; and as regard plasma ICAM-1 a significant positive correlation was found with TC (r = 0.439, P = 0.040), LDL-C (r = 0.442, P = 0.040), and serum sFas (r = 0.493, P=0.012), and insignificant correlation with CPK, CPK-MB, TG, HDL-C, Table 3.

In patients with CHF: as regard serum sCD40L, a significant positive correlation was found with TC (r = 0.491, P = 0.014), LDL-C (r = 0.440, P = 0.038), plasma VCAM-1 (r = 0.462, P = 0.028), and plasma ICAM-1 (r = 0.410, P = 0.041), and an insignificant correlation with CPK, CPK-MB, TG, HDL-C, and serum sFas; as regard plasma VCAM-1 a significant positive correlation with serum sFas (r = 0.473, P = 0.021) and insignificant correlation with CPK, CPK-MB, TC, TG, HDL-C, LDL-C, and plasma ICAM-1; and as regard plasma ICAM-1 a significant positive correlation was found only with TG (r = 0.471, P = 0.026) and insignificant correlation with other parameters, Table 4.

Table (1): Biochemical profile (mean ± SD) in controls, chronic heart failure (CHF) patients, and acute heart failure (AHF) patients

Parameters	Controls, n=10	CHF patients, n=15	AHF patients, n=15
CPK, (IU/L) P-value	70.91±40.1 -- --	72.87± 35.29 0.139* --	739.3±136.54 <0.001* <0.001†
CK-MB, (IU/L) P-value	2.95 ±1.2 -- --	3.15±1.4 0.128* --	76.93± 14.002 <0.001* <0.001†
TC, (mg/dl) P-value	197.3±20.32 -- --	216.4±38.64 0.212* --	215.67±27.9 0.186* 0.10†
TG, (mg/dl) P-value	154.3±36.6 -- --	164.47±41.38 0.592* --	155.13±39.13 0.640* 0.76†
HDL-C, (mg/dl) P-value	43.8±5.88 -- --	35.93±6.69 0.173* --	32.07±5.34 0.191* 0.81†
LDL-C, (mg/dl) P-value	106.64±24.09 -- --	147.32±28.11 0.001* --	154.79±29.89 0.001* <0.02†

TC = total cholesterol, HDL-C = high-density lipoprotein-cholesterol; LDL-C = low-density lipoprotein-cholesterol.
**P versus controls. *P versus CHF patients*

Table (2): Serum levels (mean ± SD) of sFas, sCD40L, and plasma levels (mean ± SD) of VCAM-1, ICAM-1 in controls, chronic heart failure (CHF) patients, and acute heart failure (AHF) patients

Parameters	Controls, n=10	CHF patients, n=15	AHF patients, n=15
sFas, (µg/ml) P-value	2.16 ±0.83	5.11 ± 0.94 <0.001*	5.62±1.1 <0.001* 0.85†
sCD40L, (ng/ml) P-value	4.25±1.05	9.4±2.81 <0.001*	8.82±2.64 <0.001* 0.76†
VCAM-1 (ng/ml) P-value	550.90±52.29	667.71±66.14 0.02*	682.43±73.73 0.02* 0.73†
ICAM-1 (ng/ml) P-value	234.06±27.69	340.22±62.05 <0.001*	328.51± 49.76 <0.001* 0.82†

sCD40L = soluble CD40 ligand; VCAM-1 = vascular cell adhesion molecule-1; ICAM-1 = intercellular adhesion molecule-l
**P versus controls. *P versus CHF patients.*

Table (3): Pearson's Correlation (r) between the studied parameters in acute heart failure patients (n=15).

Parameters	Serum sCD40L		Serum VCAM-1		Serum ICAM-1	
	R	P- value	r	P-value	r	P- value
CPK	-0.018	0.949	-0.305	0.270	-0.155	0.581
CK-MB	0.258	0.353	-0.055	0.846	0.111	0.693
TC	0.657	0.002	0.231	0.408	0.439	0.040
TG	0.463	0.082	0.101	0.720	0.386	0.156
HDL-C	0414	0.125	0.388	0.153	0.152	0.589
LDL-C	0.511	0.011	0.213	0.445	0.442	0.040
Serum sCD40L	--------	--------	0.473	0.028	0.551	0.003
Serum VCAM-1	0.473	0.028	--------	-------	0.346	0.070
Serum ICAM-1	0.551	0.003	0.346	0.070	--------	--------
Serum sFas	0.192	0.391	0.239	0.283	0.493	0.012

TC = total cholesterol, HDL-C = high-density lipoprotein-cholesterol; LDL-C = low-density lipoprotein-cholesterol; CD40L = CD40 ligand; VCAM-1 = vascular cell adhesion molecule-1; ICAM-1: intercellular adhesion molecule-l.
P<0.05 significant; P<0.01 High significant

Table (4): Pearson's correlation (r) between the studied parameters in chronic heart failure patients (n=15).

Parameters	Serum sCD40L		Plasma VCAM-1		Plasma ICAM-1	
	R	P- value	r	P -value	r	P- value
CPK	-0.173	0.538	-0.311	0.259	0.120	0.669
CK-MB	-0.36	0.898	-0,253	0.364	-0.154	0.585
TC	0.491	0.014	-0.046	0.871	0.116	0.680
TG	0.318	0.249	0.003	0.992	0.471	0.026
HDL-C	-0.319	0.246	-0.308	0.264	0.027	0.923
LDL-C	0.440	0.038	0.022	0.939	-0.140	0.619
Serum sCD40L	-----	------	0.462	0.028	0.410	0.041
Plasma VCAM-1	0.462	0.028	-----	-------	0.116	0.681
Plasma ICAM-1	0.410	0.041	0.116	0.681	-------	-------
Serum sFas	0.296	0.213	0.473	0.021	0.103	0.812

TC = total cholesterol, HDL-C = high-density lipoprotein-cholesterol; LDL-C = low-density lipoprotein-cholesterol; CD40L = CD40 ligand; VCAM-1 = vascular cell adhesion molecule-1; ICAM-1 = intercellular adhesion molecule-l.
P<0.05 significant; P<0.01 High significant

DISCUSSION

In the present study, the small sample size was due to the critical condition of the patients with AHF, and we chose male patients and excluded female patients to exclude the protective effect of the female sex hormone on coronary heart disease in order to unify the risk factors in the studied subjects.

In the present study, the mean plasma levels of ICAM-1 and VCAM-1 were significantly higher in patients with either AHF or CHF compared to controls, but no significant difference in their mean levels between patients with AHF and patients with CHF. Thus the increase in the cellular adhesion molecules is depending on HF. The extent to which the underlying disease processes of atherosclerosis and cardiomyopathy contribute to the increased plasma level of adhesion molecules in patients with HF is unclear. Increased levels of ICAM-1 and VCAM-1 in AHF or CHF have been found in other studies[23-25]. **Tanne *et al.* (2002)**, in a large epidemiological study, have demonstrated that healthy subjects with elevated levels of ICAM-I are at increased risk of developing coronary heart disease, peripheral vascular disease, carotid atherosclerosis, and stroke [4]. **Casey *et al.* (2008)** have found that patients with advanced congestive HF have elevated circulating levels of pro-inflammatory cytokines as interleukin-1 and TNF-α [26]. Adhesion of circulating leucocytes to the endothelial cells is thought to be mediated by cellular adhesion molecules in response to these cytokines. **Rizzoni *et al.* (2003)** have found no significant differences in mean levels of ICAM-1 and VCAM-1 in the three groups of patients with hypertension alone, patients with type II diabetes mellitus alone, and patients with both conditions, but all were significantly higher than levels in healthy controls [27]. Elevated levels of both ICAM-1 and VCAM-1 have also been found in patients with essential hypertension without atherosclerotic disease [28]**.** But, **de Lemos *et al.* (2000)** have found no association between VCAM-1 and the future risk of cardiovascular events through eight years of follow-up [29]. Nonspecific indicators of inflammation such as C-reactive protein are elevated in patients with unstable coronary disease. It remains uncertain however whether this is also the source of soluble cellular adhesion molecules or whether they reflect a more generalized activation of the vascular tree [30]. Soluble cellular adhesion molecules are generated by alternative splicing, are considered to be indicators of an activation of endothelial cells, platelets or leucocytes. They have been shown to increase in patients with acute coronary syndrome, after atherosclerotic plaque rupturing, as well as in ischemic and reperfused areas [31, 32].

Our results revealed a significant increase in sCD40L in patients with CHF as well as in patients with AHF compared to controls, but no significant difference between patients with AHF vs patients with CHF. The high levels of sCD40L were not restricted to those with MI, but rather

represent a common feature of HF regardless the etiology. So, the high levels in patients with MI with AHF could not be attributed to underlying factors of MI alone, but also to HF. Therefore, plasma level of sCD40L might be used as a marker that may predict the prognosis of cardiovascular events in patient with chronic and acute heart diseases. High serum levels of sCD40L in patients with either AHF following MI or CHF secondary to either ischemic or idiopathic dilated cardiomyopathy have been found in previous studies [33-35]. **Heeschen *et al* (2003)** have found that elevation of sCD40L plasma levels indicate an increased risk of cardiovascular events in patients with unstable coronary artery disease [36]. It could be suggested that high serum levels of CD40L are not only a marker of HF, but also it may be involved in the pathogenesis of failure by activating leucocytes and releasing the proinflammatory mediators. In cases of MI, the engagement of the sCD40L on endothelial cells or monocytes might lead to synthesis of adhesion molecules and chemokines that might contribute to contribute to atherothrombotic pathophysiological changes. While sCD40L is only weakly expressed in normal myocardial tissue, this receptor is strongly expressed in cardiomyocytes during acute myocarditis and idiopathic dilated cardiomyopathy [37]. Both CD40 and CD40L have been shown to be present in human atheroma [38]. sCD40L was shown to be associated with an increased risk of cardiovascular event in apparently health women[39,40]. On contrary to our results, **Liang *et al.* (2006),** in a retrospective study, have found sCD40L and adhesion molecules did not play significant roles in predicting coronary artery disease progression in the general population [41]. On the other hand, they have found conventional risk factors as TC, LDL-C, male sex, and the inflammatory markers hs-CRP were predictive of coronary artery disease progression. But, the retrospective nature of the study might have an explanation of that difference. Also, **Vivona *et al.*, (2009)** have found no association between sCD40L levels and atherosclerotic risk factors in patients with MI and ischemic heart[42].

In the present study, the mean serum levels of sFas were significantly higher in patients with HF compared to controls. The increase in sFas serum levels was related to the HF not the cause of the failure. Increase of sFas in CHF has occurred in the absence of biochemical evidences of myocardial damage. Therefore, the increase in serum sFas might be related to the pathophysiologic mechanisms of HF. sFas could have a role in sustaining inflammatory response and in prolonging the detrimental effects correlated with it in HF. Increased levels of sFas has been found in patients with chronic congestive HF[43]; in patients with dilated cardiomyopathy [44], in MI [45], and in patients affected by CHF and unstable angina [46]. **Nishigaki *et al.* (1997)**[43] and **Kawakami *et al.* (1998)**[44] have also found plasma sFas were increasing in proportion to the severity of HF. Production of sFas might primarily reflect the activation

of cytotoxic lymphocytes which contribute to the development of cardiomyopathy [47, 48]. Accordingly, sFas correlated with the soluble IL-2 receptor, a marker of activated lymphocytes[46], and was elevated in auto-immune diseases triggered by cytotoxic lymphocytes[12]. In addition to the potential effects of sFas on the myocardium, circulating sFas may also contribute to multi-organ dysfunction in HF patients[49]. High sFas indicated a poor prognosis and reflecting ongoing apoptotic activity and may help to identify high-risk patients who may benefit from an intensified treatment.

CPK and CK-MB were significantly higher in patients with AHF following MI in comparison to patients with CHF or controls and no significant difference was found between patients with CHF vs controls. Because our patients of CHF were of ischemic origin, so the levels of CPK and CK-MB increase in response to MI and do not reflect the coronary ischemia and could not be used as a marker of ischemic heart state. LDL-C is the only parameter of lipid profile that showed significant increase in patients with CHF and in patients with AHF vs controls. Therefore; drugs that lower LDL-C levels may improve the ischemic heart state.

In the current work we found no significant correlation between the three studied inflammatory markers (sCD40L, VCAM-1 and ICAM-1) and markers of myocardial necrosis; CPK, CK-MB in either patients with AHF or patients with CHF. **Yoshioka *et al*., (2010)** have found the plasma log CD40L levels in the culprit coronary arteries correlated positively with maximal serum CK-MB after AMI [50].

In the present study, correlation analysis revealed that serum sCD40L levels were positively associated with plasma levels of both ICAM-1 and VCAM-1, implying that elevated serum adhesion molecules in patients with ischemic heart disease might be related to the increased sCD40L concentrations. These findings are comparable to those of **Peng *et al.* (2002)** [51] and **Tsakiris *et al*. (2000)**[52].

It could be concluded that the sCD40L, sFas and adhesion molecules (VCAM-1 and ICAM-1) could be used as markers that might predict cardiovascular events in patient with chronic and acute heart diseases as the increase in the VCAM-1, ICAM-1 and CD40L are depending on HF. Moreover, the increase in serum sFas might be related to the pathophysiologic mechanisms of CHF. Therefore, sFas is a promising biomarker in pathophysiological relevance in medical diagnosis of HF and may be possibly a marker for the prognosis of HF treatment.

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ربيطة المكيف المناعي وجزئ التصاق الخلية الوعائية – ١ وجزئ التصاق داخل الخلية –١ في حالات قصور القلب الحاد والمزمن

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تلعب وسائط التكيف الخلوي المناعي دوراً محورياً في تولد مرض قصور القلب . إن جزئ التصاق الخلية الوعائية–١ (VCAM-1) ، وجزئ التصاق داخل الخلية–١ (ICAM-1) هي وسائط التهابية هامة لالتصاق الكريات البيضاء للبطانة الوعائية الفارشة. يرتفع مستوى أشكال هذه الجزيئات القابلة للذوبان في حالة الالتهاب الحاد و المزمن.

إضافةً إلى ذلك ، فإن ربيطة المكيف المناعي CD40 ligand (CD40L) والفاس الذائب sFas قد حظيتا باهتمام متزايد ، حيث أنهما تلعبان دوراً رئيسياً فى فسيولوجيا أمراض الأوعية والأحداث الوعائية متعددة الخلايا مثل الالتهاب ، وتصلب الشرايين . بناءً على الحقيقة السابقة ، فإننا رأينا تقييم دور جزئ التصاق الخلية الوعائية – ١ (VCAM-1) ، وجزئ التصاق داخل الخلية – ١ (ICAM-1) وربيطة التكيف المناعي (CD40L) فى حالات قصور القلب لدى الإنسان .

أُجريت هذه الدراسة على ٣٠ مريضاً ذكراً أعمارهم ما بين 48 ، 71 سنة كان لدى خمسة عشر منهم تاريخ مرضى سابق للإصابة بذوي العضلة القلبية ، ويعانون من قصور مزمن بالقلب ، وخمسة عشر منهم يعانون من احتشاء حاد بعضلة القلب (MI) ، مضاعف بقصور القلب أثناء الطور الحاد .

أُختبر 10 ذكور طبيعيون متطوعون كمجموعة ضابطة في نفس المجال العمري.

تم قياس مستويات جزئ التصاق الخلية الوعائية – ١ (VCAM-1) وجزئ التصاق داخل الخلية – ١ (VCAM-1) ، وربيطة التكيف المناعي sCD40 القابلة للذوبان فى مصل الدم والفاس الذائب sFas ، وقد تم قياسها جميعاً في كل المجموعات . دهون الدم، ومنشط الفوسفوكرياتين (CPK) وجزء (MB) الخاص به ، تم قياسها . وجدت زيادة ذات دلالة احصائية في جزئ التصاق الخلية الوعائية – ١ (VCAM-1) وجزئ التصاق داخل الخلية فى البلازما ، وربيطة التكيف المناعي القابلة للذوبان sCD40L والفاس الذائب sFas في كل من حالات قصور القلب المزمنة والحادة بالمقارنة في مجموعة الضبط.

أوضحت نتائج هذا البحث أنه توجد علاقة ارتباط إيجابية ذات دلالة إحصائية بين ربيطة التكيف المناعي القابلة للذوبان (sCD40L) ، وجزئ التصاق الخلية الوعائية-١ (VCAM-1) فى حالات قصور القلب الحادة ($P = 0.028$ ، $r = 0.473$) وأيضاً توجد علاقة ارتباط إيجابية ذات دلالة إحصائية بين ربيطة التكيف المناعي القابلة للذوبان (sCD40L) والبروتينات الدهنية خفيضة الكثافة (LDL-C) في كل من حالات قصور القلب الحادة ($P = 0.011$ ، $r = 0.511$) والمزمنة ($P = 0.038$ ، $r = 0.440$) .