The Role of Cystatin C and Galectin-3 in Cardiorenal Syndrome

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Abstract

Acute heart disease patients often go on to develop worsening renal function, termed as cardiorenal syndrome. The growing breadth of studies has shown the implications of combining multiple biomarkers to better chart outcomes between heart and kidneys, and produce desirable results in such patients, as cystatin C and galectin-3

The aim: To estimate the levels of cystatin C and galectin-3, and their relationship with cardiorenal syndrome.

Methods: Cystatin C and galectin-3 were obtained from 144 samples: 50 sample with cardiorenal syndrome, 25 sample with heart disease, 25 sample with kidney disease and 44 normal healthy.

Results: A highly significant increase ($p<0.0001$) in the levels of cystatin C in serum of patients with cardirenal syndrome, heart disease and kidney disease compared with normal individuals. A highly significant ($p<0.0001$) increase in the serum levels of galectin-3 in patients with cardirenal syndrome and patients with heart disease, significant increase in the serum levels of galectin-3 and in patients with kidney disease when compared with control group

Conclusions: Cystatin C and galectin-3 have higher diagnostic validity values in the current study, which may be useful as a diagnostic tool to identify recurrence of the cardiorenal syndromes.

Keywords: Cardiorenal biomarkers, Cystatin C, galectin-3

List of Abbreviation:- ROS=reactive oxygen species, NO=nitric oxide, GFR=glomerular filtration rate, AKI=acute kidney injury, CAD=coronary artery disease, CVD =cardiovascular disease, CKD= Chronic kidney disease, ROC =Receiver operating characteristics, AUC =The area under the curve

Introduction

The tight and delicate coordination of physiological functions among organ systems in the human body is such that a dysfunction in one could lead to malfunction of one or more organ systems, this relationship know as cardio-renal syndrome (CRS)[1]. Both heart and the kidneys are richly vascular (the kidneys are more vascular than the heart) and both organs are supplied by sympathetic and parasympathetic innervations. These two organs act in tandem to regulate blood pressure, vascular tone, diuresis, natriuresis, intravascular volume homeostasis, peripheral tissue perfusion and oxygenation[2]. They have endocrine functions with interdependent physiological hormonal actions regulated by arterial natriuretic peptide, a vasodilator secreted from the heart and renin-angiotensin-aldosterone system (RAAS). Also, vitamin D3, erythropoietin and renalase are all secreted from the kidneys, and are capable of cellular and humoral signaling. Dysfunction of either of the two organs can cause dysfunction of the other[3].

Cystatin C (also cystatin 3; formerly gamma trace, post-gamma globulin, neuroendocrine basic polypeptide) is a monomeric protein , a 122-amino acids 13.3 kD a globular, nonglycosylated protein. It has a high isoelectric point (pI=9.3) and in all bodily fluids, is positively charged. The third amino acid residue, proline, is hydroxylated to hydroxyproline in approximately every second cystatin C molecules. No other posttranslational modifications occur, physiologically. Cystatin C has a stable production rate nucleated cells and the low molecular weight and the positive charge of cystatin C at physiological pH help its removed from the blood circulation by glomerular filtration. After filtration cystatin C is reabsorbed and catabolized by the tubular epithelial cells. Hence, urinary clearance of cystatin C cannot be measured. In healthy individuals cystatin C is completely reabsorbed and degraded in the tubules. Cystatin C is active as a monomer. It may, however, undergo dimerization through domain swapping, and the formed dimer is inactive. During intracellular trafficking, prior to secretion from the endoplasmic reticulum in a monomeric form, the precursor of cystatin C is transiently dimerized and becomes inactive[4]. Only a few circumstances have been identified that have an impact on the production of cystatin C, such as very large doses of glucocorticoids and thyroid dysfunction serum or plasma cystatin C (later: "cystatin C") fulfills many criteria that are set for an ideal endogenous biomarker of kidney function. As a low molecular- weight globular protein with a high isoelectric point, cystatin C is freely filtered through the glomerular membrane and then almost completely reabsorbed and degraded by the proximal tubular cells. Therefore, the plasma concentration of cystatin C is almost exclusively determined by the glomerular filtration rate (GFR), making cystatin C an excellent indicator of GFR[5].

Galectin-3 is the most widely studied member of the galectin protein family. Galectin-3 is a mid-size protein (29 to 35 kDa) consisting of an N-terminal domain with tandem repeats of short amino acid segments (a total of approximately 120 amino acids) linked to a single C-terminal CRD of about 130 amino acids. It can interact with carbohydrates, which

involves the C-terminal domain, and with unglycosylated molecules, such as cell surface and extracellular receptors, which involves the N-terminal domain.

As other galectins, galectin-3 does not contain a signal sequence, therefore it is localized primarily in the cytoplasm, and occasionally, in the nucleus and mitochondria. Galectin-3 is found in epithelial cells, fibroblasts, dendritic cells and inflammatory cells[6].

Materials and Methods

Patients and control were divided into four groups:

Group 1: It included a total of 50 patients (30 males and 20 females); their ages ranged from 40-65 years for females and (38-70) years for males. All of them are suffering from heart with kidney diseases.

Group 2: It included a total of 25 patients (13 males and 12 females); their ages ranged from 38 to 70 years for males and for 40 to 65 years for females. They are suffering from heart disease.

Group 3: It included a total of 25 patients (13 male and 12 female); their ages ranged from 38 to 68 years for males and for 40 to 65 years for females . they are suffering from kidney diseases.

Group 4: This group is including 44 apparently healthy controls (22 males and 22 females); their ages ranged from 38 to 70 years .

The cystatin C and galectin-3 assay employs the quantitative sandwich enzyme immunoassay technique suppliers by cusabio companies[7,8].

Results and discussion

Serum cystatin C

The mean (±SD) of cystatin C concentration in serum of control group (healthy individuals), serum of patients with cardiorenal syndrome group, serum of patients with heart disease group and in serum of patients with kidney disease group are shown in table 1 . There are a highly significant increase ($p<0.0001$) in the serum levels of cystatin C in cardiorenal syndrome group, heart disease group and in kidney disease group when compared with control group, while there was no significant differences between males and females, or between age groups as shown in the tables 2. There are a highly significant increase ($p<0.0001$) in the serum levels of cystatin C in cardiorenal syndrome group when compared with heart disease group and significant increase ($p<0.05$) in the serum levels of cystatin C in cardiorenal syndrome group when compared with kidney disease group, and significant increase ($p<0.05$) between heart disease group and kidney disease group.

Table 1: Mean ±SD of cystatin C µg/ml concentration in studied groups according to gender.

Cystatin C	Control	CRS	Heart disease	Kidney disease
Total	**1.02±0.19**	**5.88±1.24⋆**	**3.8±0.88⋆**	**4.34±0.91⋆**
Males	**1.045±0.22**	**5.89±1.38**	**3.89±0.97**	**4.36± 0.72**
Females	**0.99±0.15**	**5.87±1.11**	**3.7±0.79**	**4.31± 1.12**

⋆ high Significant $p<0.0001$ compared with control

Table 2: Mean (±SD) cystatin C µg/ml concentration in studied groups according to age.

Cystatin C	Control	CRS	Heart disease	Kidney disease
38-50 years	**0.976±0.125**	**5.69±1.17**	**3.66±0.86**	**4.22±1.12**
>50 years	**1.060±0.23**	**6.16±1.316**	**3.90±0.91**	**4.46± 0.652**

Table 3 shows the criteria of statistical diagnosis validity of cystatin C level in cardiorenal syndrome group compared with control by using 1.88, 1.23 and 1.28 µg/ml for cardiorenal syndrome group, heart disease group and kidney disease group as cut-off value respectively ((The optimal cut-off value for cystatin C in all groups estimated from ROC curves)). According to these results, test is positive if test > threshold value (cut off values). Figures (1,2,3) explained the ROC curve for cystatin C concentration in all groups.

Table 3 : Predictive values of serum cystatin C level studied groups.

Groups	Sensitivity	Specificity	PPV	NPV	Accuracy	AUC
cardiorenal syndrome	**99%**	**98%**	**99%**	**95%**	**99%**	**1**
Heart disease	**79%**	**81%**	**77%**	**80%**	**89%**	**0.971**
Kidney disease	**99%**	**97%**	**0.96%**	**100%**	**98%**	**0.999**

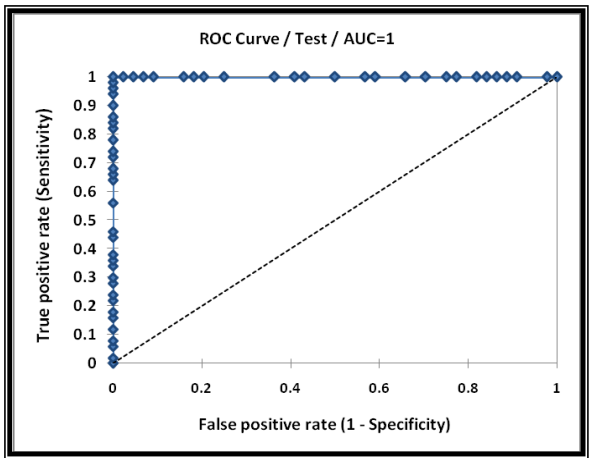

Figure 1: ROC curve for cystatin C concentration in cardiorenal syndrome group

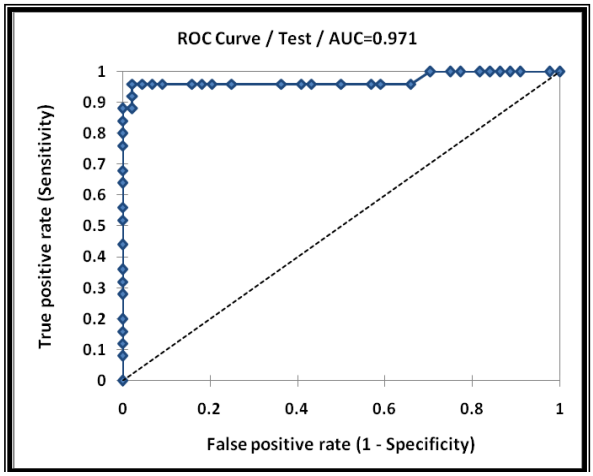

Figure 2: ROC curve for cystatin C concentration in heart disease group

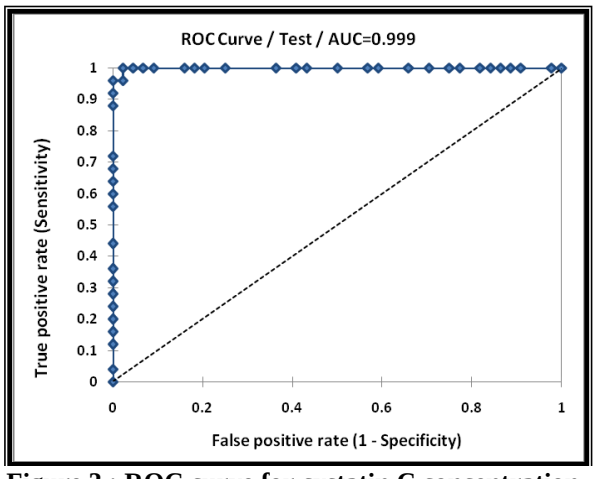

Figure 3 : ROC curve for cystatin C concentration in kidney disease group

There was a decrease in the values of sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and Accuracy tests from 99%, 98%, 99%,95%, 99% in cardiorenal syndrome group and 99%, 97%, 96%, 100%, 98% in kidney disease group to 79%, 81%, 77%, 80%, 89% in heart disease group, in addition to changes the AUC values from 1 in cardiorenal syndrome group and 0.999 in kidney disease group to 0.971 in heart disease group. These results indicate that, the cystatin C is a very sensitive for cardiorenal syndrome and kidney disease more than that in heart disease.

These results are in disagreement with *Kim et al.*(2010) who reported that no differences were found in serum cystatin C level was between coronary artery disease (CAD) and non-CAD groups[9], and also in disagreement with others who have reported that there was increased cystatin C values with advancing age[10,11]. Results of this study were consistent with many others studies who found that, decreased renal perfusion (in addition to nephrotoxic agents and over-diuresis) eventually leads to acute kidney injury (AKI) in heart disease patients. Evaluating renal function is an essential part of the assessment of every cardiac patient[12,13]. They also found that cystatin C is a diagnostic index in the early kidney damage and can estimate cardiac function damage early[14]. According to *Sonia Triki et al*. (2013) study, increased cystatin C levels are strongly and independently associated with future secondary cardiovascular (CV) events in patients with newly diagnosed coronary heart disease[15]. Others showed that high cystatin C concentration predicts all cause of mortality, CV events and incident heart failure independently of traditional CV risk factors among ambulatory persons with cardiovascular disease (CVD)[16]. They demonstrated that the risk of death during follow up increased with increasing levels of cystatin C in patients with acute coronary syndrome. and that cystatin C is a best predictive factor in patients with heart failure and CVD. *Carmen et al* .(2011) found that only subjects with confirmed decreased GFR by cystatin C had elevated risk of death, cardiovascular disease, and heart failure, which extremely elevated risk of kidney failure[17]. Others showed that cystatin C was a reliable predictor in patients with cardiac dysfunction of any severity and of AKI in post-cardiac-surgery patients; therefore, cystatin C predicts latent low renal function and acute renal failure[18]. The data lead to the hypothesis that cystatin C may be a better marker of kidney function in elderly persons and in persons with mild reductions in kidney function. Cystatin C may be a better measure of kidney function than is actual GFR because of considerable variability in iothalamate clearance due to short-term changes in true GFR and to measurement error. Mechanisms for a greater association of cystatin C with mortality than kidney failure could include direct toxicity or a reflection of some other deleterious process that may parallel reductions in kidney function[19]. *Jolanta Malyszko et al*. (2014) demonstrated that, the usefulness of cystatin C as an ideal marker of renal function and the relationship that exists between cystatin C values and increased cardiovascular and renal risk in various population groups and associated diseases. Serum cystatin C levels in patients with chronic renal disease are associated with cardiovascular disease mortality

and all cause mortality, and were shown to predict cardiovascular death, myocardial infarction and stroke in a prospective cohort of adults aged ≥ 65 years[20].

3.1.3 Serum Galectin -3

The mean (±SD) of galectin-3 concentration in serum of control group (healthy individuals), serum of patients with cardiorenal syndrome group, serum of patients with heart disease group and in serum of patients with kidney disease group are illustrated in table 4. There are a highly significant increase ($p<0.0001$) in the serum levels of galectin-3 in cardiorenal syndrome group and heart disease group, significant increase ($p<0.05$) in the serum levels of galectin-3 in kidney disease group when compared with control group, no significant differences between males and females, and no significant differences between age groups as shown in the table.

Table 4 : Mean ±SD of galectin-3 ng/ml concentration in studied groups according to gender.

Galectin-3	Control	CRS	Heart disease	Kidney disease
Total	**6.7±1.57**	**25.72±2.07⋆**	**20.22±1.37⋆**	**11.33±2.43#**
Males	**6.67±1.73**	**24.78±2.0**	**20.26±1.39**	**11.32± 2.86**
Females	**6.72±1.43**	**26.6±2.17**	**20.17±1.41**	**11.35± 1.99**

⋆ high Significant $p<0.0001$ compared with control

\# Significant $p<0.05$ compared with control

There are a highly significant increase ($p<0.0001$) in the serum levels of galectin-3 in cardiorenal syndrome group when compared with heart disease group and kidney disease group, and a highly significant increase ($p<0.0001$) in the serum levels of galectin-3 in heart disease group when compared with kidney disease group.

Table 5 : Mean (±SD) galectin-3 ng/ml concentration in studied groups according to age.

Galectin-3	Control	CRS	Heart disease	Kidney disease
38-50 years	**6.86±1.34**	**25.18±1.69**	**20.36±1.34**	**11.33±3.16**
>50 years	**6.53±1.79**	**26.53±2.35**	**20.11±1.44**	**11.34± 1.42**

Table 6 shows the criteria of statistical diagnosis validity of galectin-3 level in cardiorenal syndrome group compared with control by using 13.6, 11.3 , 9.50 ng/ml for cardiorenal syndrome group, heart disease group and kidney disease group as cut-off value respectively ((The optimal cut-off value for galectin-3 in all groups estimated from ROC curves)). According to these results, test is positive if test > threshold value (cut off values). Figures (4,5,6) explained the ROC curve for galectin-3 concentration in all groups.

Table 6 : Predictive values of serum galectin-3 level studied groups.

Groups	Sensitivity	Specificity	PPV	NPV	Accuracy	AUC
cardiorenal syndrome	**98%**	**100%**	**98%**	**97%**	**98%**	**1.000**
Heart disease	**92%**	**97%**	**95%**	**95%**	**95%**	0.989
Kidney disease	**62%**	**73%**	**85%**	**75%**	**75%**	**0.807**

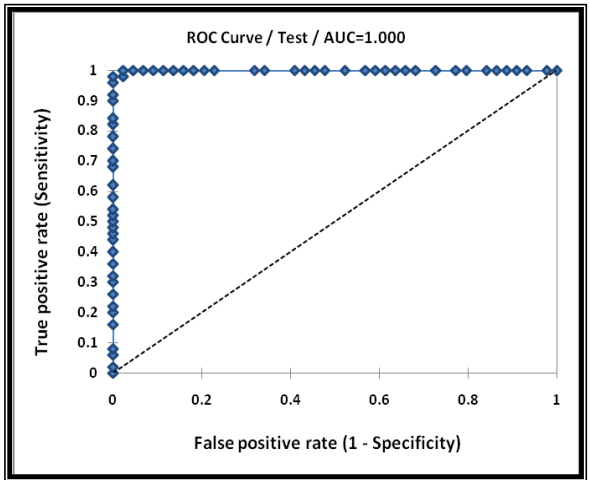

Figure 4 : ROC curve for galectin-3 concentration in cardiorenal syndrome group

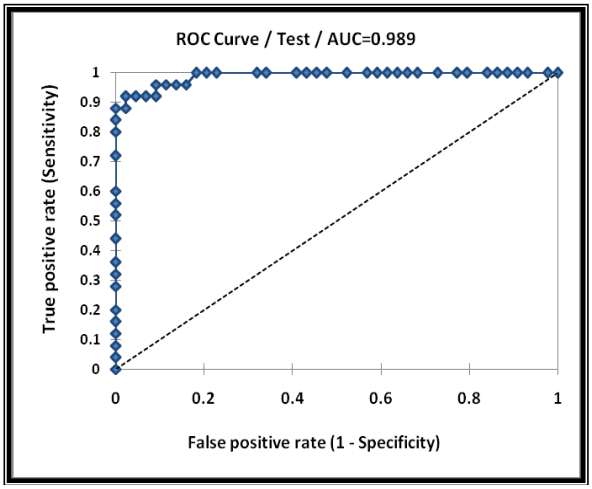

Figure 5 : ROC curve for galectin-3 concentration in heart disease group

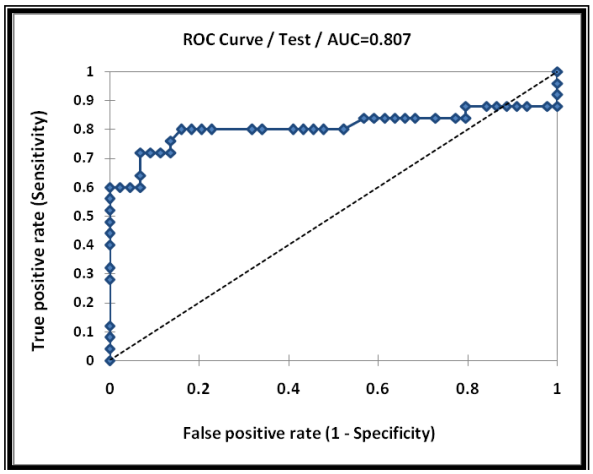

Figure 6 : ROC curve for galectin-3 concentration in kidney disease group

There was a decrease in the values of sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and Accuracy tests from 98%, 100%, 98%,97%, 98% in cardiorenal syndrome group and 92%, 97%, 95%, 95%, 95% in heart disease group to 62%, 73%, 85%, 75%, 75% in kidney disease group, in addition to changes the AUC values from 1 in cardiorenal syndrome group and 0.989 in heart disease group to 0.807 in kidney disease group. These results indicate that, the galectin-3 its very sensitive for heart disease with cardiorenal syndrome more than in kidney disease. From these results one can estimate that the galectin-3 is good biomarker in cardiorenal syndrome and heart disease better than that in kidney disease.

The main findings in this study are that circulating galectin-3 concentrations are inversely related to heart and kidney function, exceeding the reference range by as much as 3 fold in heart or kidney disease and 4 fold in heart disease with kidney complication, and the circulating galectin-3 concentrations are associated with clinical outcomes in patients with impaired kidney and heart function. jointly, evidence is accumulating that increased plasma galectin-3 levels are associated with cardiovascular disease and renal dysfunction[21-23]. In pre-clinical studies, increased expression of galectin-3 is associated with subsequent development of cardiac dysfunction[22]. In previous study, galectin-3 has been demonstrated to be a biomolecular mediator of the processes of fibrosis and inflammation, and in animal models, inhibition of galectin-3 has been shown to improve conditions associated with organ fibrosis. They hypothesized that galectin-3 would be associated with myocardial infarction, because galectin-3 expression has been detected in atherosclerotic plaques[21,24]. However, the role for galectin-3 in atherosclerosis may be ambiguous; *Iacobini C et al*.(2009) were suggested that galectin-3 attenuates the progression of atherosclerosis, whereas others have suggested the opposite[25]. There is ample evidence that galectin-3 is important in infection and in the inflammatory response, involving mast cells, neutrophils, monocytes, and T cells. Mice that lack the gene encoding galectin-3 have an impaired inflammatory response[21]. As a multifunctional biomarker, galectin-3 promotes macrophage migration, myofibroblast activation and collagen synthesis, all involved in organ fibrogenesis process. The relationship of galectin-3 with other cardiovascular markers and renal function suggests a role of galectin-3 in integrating these mechanisms in the progression of heart failer[26]. Because the liver primarily excretes galectin-3, elevations of its levels before overt kidney disease would potentially make it a useful biomarker to identify people at risk for Chronic kidney disease (CKD) (e.g., those with diabetes or hypertension). Researchers hypothesized that galectin-3 may predict new-onset CKD and progression of CKD in the general population, CKD progression is characterized by development of tubulointerstitial fibrosis and galectin-3 is a proven profibrotic mediator, including in renal tissues[27]. They observed that galectin-3 concentrations were strongly and inversely correlated with GFR, and were further elevated in patients on dialysis: it is possible that galectin-3 is, at least in part, handled or cleared by the kidney. Within the kidney, galectin-3 is found in the ureteric bud, in the collecting ducts, and in tubules[28-30].

Conclusion

Hormones (cystatin C and galectin-3) levels measurement showed a significant of validity values in the cardiorenal syndrome, heart disease and kidney patients while more sensitive in cardiorenal syndrome which give: valuable information for diagnosis, good monitoring disease status and progression of the disease.

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دور هرمون الستاتين سي والكاليستين-3 في المتلازمة القلبية الكلوية

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الملخص

أمراض القلب الحادة تؤدي غالبا إلى تدهور الوظيفة الكلوية, وهذه الحالة تعرف بالمتلازمة القلبية الكلوية. إن الدراسات المتزايدة بينت إن نتائج الدالات الكيموحيوية المتعددة التي تم جمعها تعطي أفضل توضيح للمخطط بين القلب والكلى وتعطي نتائج مرغوبة في هؤلاء المرضى , كما في الستاتين سي والكاليستين-3.

الهدف من الدراسة :- لاستنتاج مستويات الستاتين سي والكاليستين-3, ومعرفة علاقتها بالمتلازمة القلبية الكلوية.

طرق العمل:- تم دراسة الستاتين سي والكاليستين-3 في مصل دم 144 عينة :-50 عينة لمرضى المتلازمة القلبية الكلوية و25 عينة لمرضى القلب و25 عينة لمرضى الكلى و44 عينة للأصحاء تم استخدامها كمجموعة سيطرة للمقارنة.

النتائج:- وجود ارتفاع معنوي عالي ($p<0.0001$) في تركيز ن هرمون الستاتين سي في مصل دم مرضى المتلازمة القلبية الكلوية ومرضى القلب ومرضى الكلى مقارنة مع مجموعة السيطرة (الأصحاء). وجود ارتفاع معنوي عالي ($p<0.0001$) في تركيز هرمون الكاليستين-3 في مصل دم مرضى المتلازمة القلبية الكلوية ومرضى القلب وزيادة معنوية في مصل دم مرضى الكلى , مقارنة مع مجموعة السيطرة (الأصحاء).

الاستنتاجات:- الستاتين سي والكاليستين-3 يمتلكان صحة تشخيصية عالية في الدراسة الحالية , والتي قد تكون مفيدة كدالة تشخيصية لتمييز المتلازمة القلبية الكلوية.