Association between the Levels of B- type Natriuretic Peptide, and C-reactive protein in Patients with Chronic Renal Failure

*Sahar M El-deek, Mohamed M Hasan *and Ramadan A Said*
Biochemistry and Department of Internal medicine * Departments
Faculty of Medicine, Assiut University

ABSTRACT

Introduction: *Chronic kidney disease is associated with increased morbidity and mortality in cardiovascular disease. Apart from traditional risk factors, chronic inflammation, and increased left-ventricular wall tension related to hypervolemia are important in cardiovascular disease development in renal patients. B-type natriuretic peptide (BNP) is a cardiac neurohormone specifically secreted by cardiac ventricles in response to volume expansion and pressure overload. High sensitivity C-reactive (hsCRP) have been found to reflect chronic inflammation and significantly elevated in hemodialysis patients.* ***Aim of the work:*** *To assess the relationship between left ventricular filling pressure (using plasma BNP levels) and inflammation (using plasma hsCRP levels) in patients with chronic renal failure and their relationship with renal echogenisity detected by ultrasonography.* ***Patients and methods:*** *Plasma BNP and hsCRP were measured on the same day in 38 pre-dialysis patients. Patients were classified into 5 groups according to ultrasonographic renal echogenisity into Group 1(no=3) with grade 0, group 2 (no=2) with grade I, group 3 (no=15) with grade II, group 4 (no=14) with grade III renal echogenisity and group 5 (no=4) with complete loss of the medulla and cortex of the kidney.* ***Result:*** *Plasma levels of BNP and hsCRP were significantly higher in patients with chronic renal failure in comparison with controls (274.3±97.1 pg/ml versus 33.7±8.0 pg/ml and 11.4±3.9 mg/L versus 2.7±1.0 mg/L respectively P <0.0001 for each). Comparing plasma levels of BNP and hsCRP with ultrasonograghic renal echogenisity, which reflect severity of renal disease, showed that the plasma levels of BNP were 104.7±15.0 pg/ml, 148.0±67.9 pg/ml, 248.5±72.0 pg/ml, 310.1±39.2 pg/ml and 436.0±10.0 pg/ml in the five groups of patients respectively and hsCRP were 4.7±0.6 mg/L, 5.5±0.7 mg/L, 10.3±3.2 mg/L, 13.6±1.8 mg/L and 16.0±0.8 mg/L in the same previous groups respectively. It was clear that the plasma levels of both biomarkers were significantly higher in patients with more severe renal affection (P< 0.0001 for each). There was, also, highly significant positive correlation between plasma levels of BNP and hsCRP in all groups of patients (r= 0.9, P<0.0001) and significant negative correlation between both markers and serum albumin (r=-0.4, P<0.001 and r=-0.5, P<0.0001 respectively) and significant negative correlation between both markers and hemoglobin levels (r=-0.8, P< 0.0001 and r=-0.8, P< 0.0001 respectively).* ***Conclusion:*** *The present results suggest a link between left ventricular pressure and inflammation in patients with chronic renal failure. The importance of strict volume control in these patients, in order to reduce left ventricular pressure and therefore inflammation, should be considered. Both BNP and hsCRP could provide complementary diagnostic and prognostic information regarding future cardiovascular disorders in renal patients.*

INTRODUCTION

It has long been known that among patients with chronic renal failure, cardiac disease was the single greatest cause of mortality, accounting for nearly one half of all deaths[(1)]. Among community-based population, renal insufficiency was an independent predictor of the risk of subsequent ischemic heart disease, conferring a risk equivalent to that of diabetes[(2)]. Furthermore, the presence of even moderate renal impairment in a patient presenting with an acute coronary syndrome was strong short-term prognostic indicator, significantly increasing the 30-day risk of myocardial infarction, heart failure, and cardiac death[(3)]. Thus, a strong and pervasive link clearly exists between kidney failure and cardiac disease. The common challenge to the nephrologists and cardiologist therefore lies in successfully recognizing those patients within their scope of practice who are most at risk, thereby allowing targeted application of interventions designed to circumvent adverse outcomes [(4)]. A variety of individual biomarkers have been evaluated to predict adverse events among patients with various degrees of kidney dysfunction. These included markers of ventricular dysfunction as B-type natriuretic peptide (BNP) and markers of systemic inflammation as high sensitivity C-reactive protein (hsCRP)[(5)].

BNP had recently gained a great deal of attention, not only for their clear diagnostic value in patients with heart failure but, also, as tools for risk stratification in patients with suspected acute coronary syndrome [(4)]. Patients receiving hemodialysis are usually exposed to increased peripheral vascular resistance and volume abnormalities and, thus, chronically increased ventricular afterload, significant increase in the level of BNP [(4)] and [(6)]. Also, expansion of extra-cellular volume causing myocardial stretching and increased left ventricular pressures, which is the principal cause of increased level of BNP. The left ventricular hypertrophy and endothelial dysfunction in severe chronic renal failure, systolic and diastolic dysfunction, the associated cardiac disease (usually ischaemic), also contribute to that increase [(7)].

hsCRP was considered to be the prototype marker of inflammation. In the general population, there were ample clinical and epidemiological data that indicate its usefulness both in predicting the prognosis for various forms of cardiovascular and renal disease and in monitoring response to treatment. There was, also, evolving evidence that hsCRP may be directly involved in the pathological disease process itself[(8)]. In patients with chronic renal failure, increased level of hsCRP was accounted for both traditional and non-traditional risk factors. However, since it is a non-specific inflammatory marker and acute phase reactant, hsCRP may become elevated as a result of other dialysis-related and dialysate-unrelated factors[(8)].

Erythrocyte sedimentation rate (ESR) was widely used in the general population. It can be used in heamodialysis patients much in the same way as in general population, as

it was influenced by the same factors[9].

Aim of the Work

1) To clarify of the importance of estimation of plasma levels of BNP and hsCRP in patients with chronic renal failure. 2) To investigate the presence of link between left ventricular filling pressure and inflammation in patients with chronic renal failure. 3) To assess the relationship between plasma BNP and hsCRP levels and severity of renal disease detected by renal echogenisity in ultrasonography.

PATIENTS & METHODS

Patients:

The present study was carried out on 38 patients with chronic renal failure and 15 age and sex matched healthy controls. The patients were selected from Inpatients of Renal Unit, Internal Medicine Department of Assiut University Hospital whom underwent hemodialysis. An informed consent was obtained from each subject. The study was approved by Ethical Committee of Faculty of Medicine, Assiut University. Patients with the following condition were excluded: heart failure, coronary artery diseases, overt active inflammatory state, infection, infectious disease, fever within 30 days before the study, autoimmune disease, malignancy and patients taking immunosuppressive therapy.

To all patients renal history was taken and complete physical examination was performed. The following data were included: age, sex, arterial hypertension, diabetes mellitus and smoking. Hypertension was defined as blood pressure >140/90 on two occasional measurements or being under treatment for hypertension. Diabetes mellitus was diagnosed as fasting blood glucose level >126 mg/dl or if the patients were taking antidiabetic treatment.

Methods:

Blood Sampling:

Venous blood samples were withdrawn from venous end of an AV fistula at the beginning of the hemodialysis session. Each blood sample was divided into two halves. One half was collected in a tube containing K_2 EDTA and immediately centrifuged in 500 rpm for 15 minutes. The separated plasma was stored at – 70°C until measurement of BNP and hsCRP. The second half was allowed to clot at room temperature and centrifuged at 500 rpm for 10 min. The serum was collected and stored at – 70°C and used for determination of urea, creatinine and albumin.

Urine Sampling:

Urine was collected in plastic container and stored in a refrigerator at 4°C until the estimation of urinary albumin.

Biochemical measurement:

The following conventional laboratory tests were performed by laboratory of Assiut University Hospital, serum creatinine by commercial assay Kit (Stanbio Laboratory USA) according to the method of Jaffe reaction[10], blood urea by commercial assay kit according to Wybenga et al.[11] and[12], serum albumin by commercial assay

Kit according to Bromocresol green method[13]. ESR by westergren methods. Also, urinary albumin was estimated by turbidimetry[14].

Plasma BNP was determined by an ELISA kit for the quantitative determination of BNP. Cat. No. S-1136 manufactured by: Peninsula Laboratories Inc. a member of the Bachem Group USA. The measurement was performed according to the method described by Porstman and Kiessig., [15].

Plasma hsCRP was determined by an ELISA kit for the quantitative determination of hsCRP manufactured by: DiaMed EuroGen, 2300 Turnhout, Belgium. The measurement was performed according to the method described by Mitra and Panja., [16].

Kidney Ultrasound:

Ultrasonography to the kidney was performed to all patients and any abnormalities were recorded. The severity of renal affection was assessed by renal echogenisity according to the grading that was described by Rosenfield et al., [17].

Statistical analysis:

It was performed using SPSS statistical program (statistical package for social science, version 11) for analyzing the resulted data. All the studied parameters concentrations were presented as mean ±SD. The one-way ANOVA was used to determine the significance of the difference between the groups. Student t-test was used to compare between two groups. Qualitative data were presented in the form of number and percentage. Chi-square test was used to compare between qualitative data and Pearson correlation coefficient to study the relation between variables. P-value < 0.05 was considered significant.

RESULTS

In the present study, the patients were divided into five groups according to the renal echogenisity assessed by ultrasonography. Group 1 with grade 0 (n=3, 7.9%), group 2 with grade I (n=2, 5.3%), group 3 with grade II (n=15, 39.5%), group 4 with grade III renal echogenisity (n=14, 36.8%) and group 5 with complete loss of the medulla and cortex of the kidney (n=4, 10.5%).

The baseline clinical characteristics of the patients and controls are presented in (Table 1). The patients and controls were similar for distribution of age and gender. In particular, higher percent of patients had hypertension and diabetes mellitus. Patients had significantly higher values of blood urea 40.4±11.3 mmol/L, higher value of serum creatinine 1133.4±397.0 umol/L, lower value of serum albumin 36.3±6.6 gm/L, lower value of hemoglobin 7.7±1.3 g/dl ($p<0.0001$ for each) (Table 2). Albumin detected in the urine of patients and there were 6 patients with no albumin in urine, 17 patients with (+) urinary albumin, 12 patients with (++) urinary albumin and 3 patients with (+++) urinary albumin. In addition patients had significantly higher value of the ESR in the first and second hours than controls, 67.9±21.3 mm and 90.1±24.4 mm respectively ($p<0.0001$ for each) (Table 3).

The mean plasma levels of BNP and hsCRP were 274.3±97.1 pg/ml and 11.4±3.9 mg/L in patients and

33.7±8.0 pg/ml and 2.7±1.0 mg/L in controls. It was clear that the plasma levels of both markers were significantly higher in patients than controls (p< 0.0001for each) (Table 3).

In the present study, also, plasma levels of BNP and hsCRP were compared with ultrasonograghic renal echogenisity in patients with chronic renal failure. The plasma mean levels of BNP were 104.7±15.0 pg/ml, 148.0±67.9 pg/ml, 248.5±72.0 pg/ml, 310.1±39.2 pg/ml and 436.0±10.0 pg/ml in patients with grade 0, grade I, grade II, grade III renal echogenisity and patients with complete loss of the medulla and cortex respectively.

hsCRP were 4.7±0.6 mg/L, 5.5±0.7 mg/L, 10.3±3.2 mg/L, 13.6±1.8 mg/L and 16.0±0.8 mg/L in the same previous groups respectively. The plasma levels of both biomarkers were significantly higher in patients with more severe renal affection (Table 4 and 5).

There was highly significant positive correlation between plasma levels of BNP and hsCRP in all groups of patients (r=0.9, P< 0.0001) (figure 1). Also there was highly significant negative correlation between plasma levels of BNP and hsCRP with serum albumin (r=-0.4, P<0.001 and r=-0.5, P<0.0001 respectively), highly significant negative correlation between both markers and Hb levels (r=-0.8, P<0.0001 and r= 0.8, P< 0.0001 respectively) and highly positive correlation with urea, creatinine levels and ESR in both first and second hours. There was significant positive correlation between the levels of BNP and hsCRP and the degree of renal echogenisity that detected by ultrasonography and reflect severity of renal affection (r= 0.8, P< 0.001 and r= 0.9, P< 0.001 respectivily).

Table (1): Baseline characteristics of patients with chronic renal failure and controls.

Characteristic	**Patients**	**Controls**	**P-valu**
Numbers	38	15	
Age(years)	47.8 ± 15	43.4 ± 9.8	NS
Male(no/%)	20 (52.6%)	9 (60%)	NS
Female(no/%)	18 (47.4%)	6 (40%)	NS
Hypertension	23 (60.5%)	--	P< 0.0001
Diabetes mellitus	31 (81.6%)	--	P< 0.0001
Systolic blood pressure (mm Hg)	139 ± 62	121.3 ± 7.4	P< 0.0001
Diastolic blood pressure (mm Hg)	85.8 ± 12.9	77. 3 ± 5.9	P< 0.001

Table (2): The studied biochemical parameters in patients with chronic renal failure and controls.

Parameters (mean ± SD)	**Patients (No = 38)**	**Controls (No = 15)**	**P-value**
Serum urea (mmol/L)	40.4 ±11.3	5.9 ± 0.6	P< 0.0001
Creatinine (umol/L)	1133.4 ± 397.0	121.3 ± 6.7	P< 0.0001
Serum albumin (g/L)	36.3 ± 6.6	49.7 ± 4.6	P< 0.0001
Hemoglobin (g/dl)	7.7 ± 1.3	14.2 ± 1.2	P<0.0001

Table (3): The plasma levels of BNP, hsCRP, and ESR in patients with chronic renal failure and controls.

Blood parameters (mean ± SD)	**Patients (no= 38)**	**Controls (no= 15)**	**P-value**
BNP (pg/ml)	274.3 ± 97.1	33.7 ± 8.0	P< 0.0001
hsCRP (mg/L)	11.4 ± 3.9	2.7 ± 1.0	P <0.0001
ESR			
First hours	67.9 ± 21.3	7.6 ± 2.4	P< 0.0001
Second hours	90.1 ± 24.4	17.3 ± 3.2	P< 0.0001

Table (4): Type of ultrasonographic involvement of the kidney and plasma levels of BNP in patients with chronic renal failure.

Kidney echoginesity	**Patients (no/ %)**	**BNP (pg/ml) (mean ±SD)**	**P-value**
Grade 0	3 (7.9 %)	104.7 ± 15.0	NS
Grade I	2 (5.3 %)	148.0 ± 67.9	*P<0.0001
Grade II	15 (39.5 %)	248.5 ±72.0	**P< 0.0001
Grade III	14 (36.8 %)	310.1 ± 39.2	***P< 0.0001
Lost cortex	4 (10.5 %)	436.0 ±10.0	

NS patients with grade I versus patients with grade 0
**Patients with grade II versus grade I*
***Patients with grade III versus grade II*
****Patients with grade III versus patients with complete loss of the cortex*

Table (5): Type of ultrasonographic involvement of the kidney and plasma levels of hsCRP in patients with chronic renal failure.

Kidney echoginesity	Patients (no/ %)	hsCRP(mg/L) (mean ± SD)	P-value
Grade 0	3 (7.9 %)	4.7 ± 0.6	NS
Grade I	2 (5.3 %)	5.5 ± 0.7	*P<0.0001
Grade II	15 (39.5 %)	10.3 ± 3.2	**P< 0.0001
Grade III	14 (36.8 %)	13.6 ± 1.8	***P< 0.0001
Lost cortex	4 (10.5 %)	16.0 ± 0.8	

NS patients with grade I versus patients with grade 0
**Patients with grade II versus grade*
***Patients with grade III versus grade*
****Patients with grade III versus patients with complete loss of the cortex*

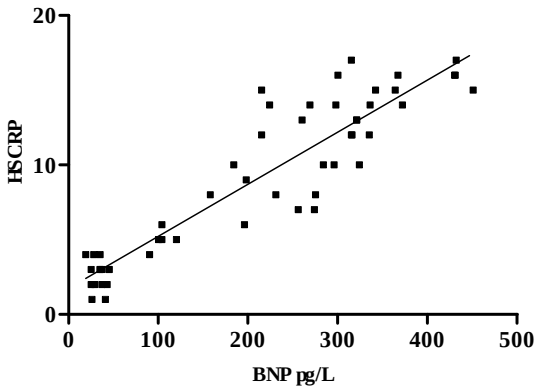

Fig. (1): Correlation between BNP and hsCRP in chronic renal failure patients.

DISCUSSION

Cardiovascular complications are the major cause of death in patients with end stage renal disease (ESRD), accounting for approximately one half of all deaths and the mortality risk attributable to coronary heart disease in these patients is about 100 times higher than that in the general population[18]. Even though cardiovascular risk in patients with chronic kidney diseases now constitutes a major concern in the nephrology community, ESRD patients are treated much less intensively than needed [19]. Because of the daunting burden of cardiovascular disease and the still insufficient emphasis on appropriate treatment, cardiovascular risk stratification is a fundamental issue in strategies aimed at bettering clinical management of chronic kidney disease patients[20].

BNP is a hormone, synthesized by myocardium and it is important in regulation of blood pressure, electrolyte and volume homeostasis [21]. In addition, it has emerged as a biomarker for altered myocardial function and structure and it has been shown to be increased in patients with renal insufficiency and reflect cardiac preload and afterload as well as intrinsic heart disease, thus providing an index of cardiac stress. Furthermore, circulating BNP levels are predictive of cardiovascular outcome in ESRD patients[22].

In the present study, a significant increase in the mean plasma levels of BNP in patients with chronic renal failure before dialysis was found than in control. This result was in agreement with that of Apple et al.[6] and Safley et al.[23] who found that

BNP concentrations are increased in ESRD dialysis patients. Also, Zeng et al.[24] reported that BNP was elevated in patients with end stage renal failure and the plasma BNP offers a good sensitivity and specificity in diagnosing left ventricular dysfunction in patients with dialysis-dependent renal failure. In addition, Austin et al.[25] reported that plasma levels of BNP was elevated in patients with renal dysfunction, regardless of degree of renal impairment or type of left ventricular dysfunction and their levels increased as glomerular filtration rate declines, indicating a great effect of glomerular filtration rate on BNP levels. Moreover, Sheen et al., [26] found that BNP levels were elevated in patients with chronic renal failure and declined after each dialysis session.

The principal cause of increased BNP in haemodialysis patients is that the expansion of extra-cellular volume causing myocardial stretching and increased left ventricular pressure[7]. The left ventricular hypertrophy and endothelial dysfunction in severe chronic renal failure, systolic and diastolic left ventricular dysfunction, the associated cardiac disease (usually ischaemic), also contribute to this increase[7]. On the other hand, Anwaruddin et al.[27] clarify that renal clearance is likely one mode of removal of BNP from the circulation, and the more elevated levels of BNP that were detected in patients with chronic renal failure may simply reflect decreased clearance.

Advancement in the understanding of the pathogenesis of atherosclerotic vascular disease now suggest a central contribution of inflammation to morbidity and mortality, and hsCRP, a circulating marker of inflammation, has been closely linked through epidemiologic data to increased cardiovascular disease[28]. hsCRP has been shown to be increased in a substantial numbers of ESRD patients and associated with clinical outcome, including all causes of death. Over the past decade, numerous studies have, also, demonstrated that hsCRP is increased in substantial numbers of ESRD patients without apparent clinical reason [28]. The presence of increased hsCRP confirms the existence of chronically activated acute phase response in the ESRD population[29].

In the present study, a significant increase was found in the mean plasma levels of hsCRP in patients with chronic renal failure before dialysis than in control. These results were in agreement with those of Fred et al., [29] who reported that there was increase in the plasma levels of hsCRP in patients with ESRD. Their data support the link between hsCRP and mortality in the ESRD population, with hsCRP possibly acting as a measure of atherosclerosis. They suggest that hsCRP should be considered in routine investigation of ESRD patients and using hsCRP as a valuable tool for identifying patients at high risk of cardiovascular events. Also, Annuk et al., [30] and Nasri[31] reported that hsCRP concentrations have been found to be elevated in hemodialysis patients and reflect chronic inflammation, and as an acute phase reactant, is a sensitive and independent marker of atherosclerosis. In addition, Racki et al.[32] reported that there was an increase in the

plasma levels of hsCRP in patients with chronic renal failure and serum concentration above 6.2mg/L is a strong predictor of overall and cardiovascular mortality in patients with ESRD.

The causes of chronic inflammation in ESRD patients are promoted by several factors, which may be related or not related to dialysis. In dialysis patients, there is a combination of an impaired immune response related to the uremic state and persistent immune/inflammatory responses, resulting in persistent immune system stimulation, low-grade systemic inflammation, and altered cytokine balance. This may characterize the uremic state, which may translate into an increased risk of developing vascular disease[33].

In the present study, there was significant increase in the levels of ESR in the first and second hours in comparison with that of controls. This result was in agreement with Borawsk et al.[34] who found that ESR could be useful in diagnosing of inflammation in hemodialysis patients as it was increased in patients with chronic renal failure and correlated positively with CRP and inversely with serum albumin. Also, Brouillard et al.[9] concluded that ESR was increased in most of patients with chronic renal failure.

There was significant positive correlation between plasma levels of BNP and hsCRP with the severity of renal involvement that was detected by ultrasonography. Fred et al.[29] and Anwaruddin et al.[27] found a significant inverse relationship between renal function and BNP in patients with chronic renal failure. Also, Codoqnotto et al.[35] found that BNP and hsCRP were increased in patients with uremia and there was inverse relationship between the plasma levels of both markers and glomerular filtiration rate. On the other hand, Zoccali.[22] found that hsCRP was unrelated to GFR and progression of disease. This lack of association does not necessary imply a lack of causation. Indeed circulating hsCRP may be inadequate marker of the severity of renal inflammation. In that regards, it was noting that other markers as BNP were apparently useful for monitoring progression of renal disease [22]. The relationship between plasma levels of BNP and hsCRP with the severity of renal affection, which was detected by degree of ultrasonographic echogenisity were not studied by several researchers and need progressive studies.

In the present study there was a highly significant positive correlation between plasma levels of BNP and hsCRP and highly significant negative correlation between the previous two parameters with serum albumin and Hb levels. This results was in agreement with that of Ortega et al.[36] who found that the correlation between BNP and hsCRP was stronger in predialysis patients and both parameters were inversely correlated with creatinine clearance, serum albumin and Hb levels.

It is important to note that the combination of two biomarker, hsCRP and BNP was almost informative and these biomarkers, at least, in part, reflect overlapping pathologic processes. Also, they possess the major characteristics required for a

marker to be recommended for wide use in clinical practice, namely, they should provide independent information in risk or prognosis beyond established risk factors and they should be easy to measure[37]. Recently completed and ongoing trials will provide specific answers to the hypothesis that BNP and hsCRP might be a guide to treatment in ESRD patients[20]. If positive, these trials will constitute a definitive argument for the widespread use of these biomarkers in the dialysis populations. In the specific case of ESRD, BNP and hsCRP may, also, have other practical implications because the measurement of circulating levels of these substances may be useful also for comparing the cardiovascular burden of diverse dialysis populations[20].

Conclusion:

The present results suggest a link between left ventricular pressure and inflammation in patients with chronic renal failure. The importance of strict volume control in these patients, in order to reduce left ventricular pressure and therefore inflammation, should be considered. The combination of the two biomarkers were to identify patients at particularly high risk and identify a potential role for these biomarkers to be incorporated into diagnostic and therapeutic strategies aimed at detection and treatment of atherosclerotic complications.

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دراسة مدى الارتباط بين الناتريوريتك ببتيد– ب و البروتين النشط –ج شديد الحساسية فى مرضى الفشل الكلوى المزمن

سحر الديك محمد السيد، محمد حسن مصطفى ورمضان عبد العال سيد*

قسمى الكيمياء الحيوية الطبية والباطنة العامة*

لقد وجد أن هناك ارتباطآ وثيقآ بين حدوث امراض القلب و الأوعية الدموية فى مرضى الفشل الكلوى المزمن حيث انه من اهم أسباب زيادة معدلات الوفيات فى هؤلاء المرضى. و بالأضافة الى عوامل الخطورة التقليدية ، يوجد هناك الكثير من العوامل التى قد يكون لها دور فى تطور امراض القلب فى مرضى الفشل الكلوى المزمن. من هذه العوامل زيادة معدل الناتريوريتك ببتيد–ب و البروتين النشط– سى. الناتريوريتك ببتيد– ب يعتبر من الهرمونات التى يتم افرازها عن طريق بطين القلب نتيجة لزيادة فى حجم الدم الذى يؤدى الى زيادة فى الضغط على البطين مما يؤدى الى زيادة الناتريوريتك ببتيد– ب. اما البروتين النشط–سج شد

يد الحساسية يعتبر من الدلائل التى تزيد نتيجة لحدوث التهابات مزمنة و يزيد فى مرضى الفشل الكلوى المزمن. الهدف من الدراسة: دراسة العلاقة بين زيادة الضغط فى بطين القلب (يدل عليه زيادة معدل الناتريوريتك ببتيد– ب) و الالتهابات المزمنة (يدل عليه زيادة معدل البروتين النشط– سج شديد الحساسية) فى مرضى الفشل

الكلوى المزمن مع ايجاد العلاقة بين هذه الدلالات و مدى درجة الاصابة فى الكلى التى يمكن تحديدها عن طريق استخدام الاشعة التليفزيونية على الكلى.

و قد اجريت هذه الدراسة على ٣٨ مريضآ يعانون من فشل كلوى مزمن قبل عمل غسيل بريتونى لهم و قد تم تقسيمهم الى خمس مجموعات على اساس درجه اصابه الكلى التى تم تحديدها عن طريق الاشعة التليفزيونية هذا بالاضافة الى المجموعة الضابطةو تتكون من ١٥ من الاصحاء. تم قياس معدل الزيادة فى الدلالات الكيميائية المختلفة وقد اظهرت النتائج ما يلى:

(١) هناك زيادة ذات دلالة احصائية فى معدل كل من الناتريوريتك ببتيد – ب و البروتين النشط –ج شديد الحساسية و سرعة ترسيب الدم فى جميع المرضى الذين يعانون من فشل كلوى مزمن بالمقارنة بالمجموعة الضابطة.

(٢) هذه الزيادة فى الدلالات الكيميائية لها علاقة بمدى درجة الاصابة فى الكلى و التى تم تحديدها عن طريق الاشعة التليفزيونية على الكلى.

(٣) هناك ارتباطا ايجابيا ذو دلالة احصائية بين معدل الزيادة فى كل من الناتريوريتك ببتيد – ب و البروتين النشط – سج شديد الحساسية و هناك ايضا ارتباطا ايجابيا ذو دلالة احصائية بين معدل الزيادة فى هذه الدلالات الكيميائية و مدى درجة الاصابة فى الكلى التى تم تعيينها عن طريق الاشعة التليفزيونية.

ولذلك يمكن أستنتاج وجود علاقة وثيقة بين معدل الزيادة فى كل من الناتريوريتك ببتيد– ب و البروتين النشط – ج شديد الحساسية و هذا يدل على وجود علاقة بين زيادة الضغط داخل بطين القلب مع وجود التهاب مزمن فى مرضى الفشل الكلوى المزمن ومن هنا يمكن توضيح اهمية العمل على تقليل الزيادة فى حجم الدم فى هؤلاء المرضى مما يؤدى الى تقليل الضغط فى البطين و بالتالى تقليل حدوث التهابات مزمنة فى الكلى ممايحسن من الحالة المرضية لهؤلاء المرضى.