

Correlation between High Sensitivity C-Reactive Protein and Incident Type 2 Diabetes among Newly Diagnosed Renal Impairment Patients

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Abstract

Background: Numerous markers of inflammation like C-reactive protein (CRP) are raised considerably in the diabetic population. The levels of these markers also associate with the severity of diabetes and presence of various complications. **Objective:** The objective was to study the correlation between CRP and glycemic control in type 2 diabetic patients who were newly diagnosed renal impairment and to evaluate if good glycemic control reduces the CRP values. **Patients and Methods:** This work was performed from June 2018 to January 2019. Forty-five type 2 diabetic patients who were newly diagnosed renal impairment are included after proper screening, between 48 and 60 years of age at the National Diabetic Center/Al-Mustansiriyah University. They were equated with 45 healthy subjects as control group. Then, relevant tests were done which are involved: fasting serum glucose, glycated hemoglobin (HbA1c), urea, creatinine, serum lipid profile, and serum high sensitivity CRP (hs-CRP). **Results:** A significant increase in serum urea and creatinine was found in diabetic patients as paralleled to the controls. Furthermore, there was a significant difference in hs-CRP in diabetics as compared to the controls (13.95 ± 5.42 mg/l vs. 2.0 ± 1.16 mg/l, $P = 0.01$, odds ratio = 1.157). Well glycemic control is foremost to the decline in HbA1c moreover caused a reduction in CRP levels when HbA1c was ≤ 7 . **Conclusions:** It has been concluded that higher levels of CRP can be a prognostic element for the progress of type 2 diabetes. Also, it has a positive correlation with HbA1c, reflecting the glycemic status of the patient.

Keywords: C-reactive protein, diabetes mellitus, glycated hemoglobin, renal impairment

INTRODUCTION

Diabetes mellitus (DM) has extended epidemic scopes worldwide. Diabetic kidney disease is the main reason of morbidity and mortality in diabetes.^[1] Actually, the excess mortality of diabetes happens mostly in individuals with diabetes and proteinuria, and consequences not only from end-stage renal disease (ESRD) but also from cardiovascular disease (CVD), with the latter being principally mutual in patients.^[2]

Renal failure is a systemic disease and induces a slow and progressive deterioration of kidney function improved by numerous influences counting autoimmune diseases, diabetes, cancer, and toxic chemicals.^[3] It is generally a consequence of complications arising from other severe medical situations.^[4]

Increased urea and creatinine levels represent kidney defects. Serum creatinine level is usually construed as a degree of the

glomerular filtration rate (GFR) which is used as an index of renal function in the clinical field.^[5]

In greatest patients, the first sign of diabetic nephropathy is moderately improved urinary albumin excretion (UAE), i.e., 30–300 mg/24 h in a spot urine sample also termed microalbuminuria. Patients who progress albuminuria, i.e., >300 mg/24 h also termed macro- or clinical albuminuria, are at principally great risk for increasing a failure in renal function.^[6]

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Nevertheless, up to 40% of patients with moderate albuminuria return to normoalbuminuria. Furthermore, up to 50% of patients with type 1 or type 2 diabetes experience a deterioration in GFR, although the presence of only moderate or even normoalbuminuria. Consequently, high UAE is not an essential precondition for the progress of diabetic nephropathy.^[7]

In DM, hyperglycemia causes the excess of glucose to associate with free amino acids on circulating or tissue proteins. This nonenzymatic reaction yields advanced glycosylation end-products (AGEs) through an Amadori rearrangement. Good glycemic control can lessen the occurrence of nephropathy.^[8] However, accumulating evidence indicates that inflammation may show a central intermediary part in the pathogenesis of type 2 DM (T2DM), thus connecting diabetes to a number of generally parallel conditions supposed to invent through inflammatory mechanisms. It has been recommended that C-reactive protein (CRP) is associated with T2DM.^[9] It is synthesized in the liver and one of the greatest sensitive acute phase reactants after tissue damage of inflammation which triggers the typical complement pathway due to the inflammatory response. Raised levels of CRP and poor glycemic control lead to macrovascular events and lead to raised diabetes risk in the future.^[10]

Therefore, this work was undertaken to find an association between high sensitivity-CRP (hs-CRP) and glycemic control in type 2 diabetics who were newly diagnosed renal impairment and to evaluate if good glycemic control reduces the CRP values.

PATIENTS AND METHODS

This study was performed from June 2018 to January 2019. Fortyfive patients with T2DM who were newly diagnosed as renal impairment and visiting the National Diabetic Center/Mustansiriyah University, their age ranged from 48 to 60 years. They were included after proper screening. All diabetic patients were classified as nephropathy when UAE: 30–300 mg/24 h and they were matched with 45 healthy subjects as control group. Patients with malignancy, any infection, pregnancy, current smoking history, and/or history of intake of steroids were excluded. Then, relevant tests were done which are involved: body mass index (BMI), waist circumference (WC), waist-hip ratio (WHR), systolic- and diastolic blood pressure (SBP and DBP), fasting serum glucose (FSG), glycated hemoglobin (HbA1c) using Bio-Rad VARIANT hemoglobin A1C, urea, creatinine, lipid profile including (serum total cholesterol [TC], triacylglycerol [TAG], high-density lipoprotein-cholesterol [HDL-C], and low-density lipoprotein-cholesterol [LDL-C]). Serum hs-CRP was also determined by CUSABIO kit, China. Tests with a CRP level of >10 mg/l are often due to acute conditions.

Statistical analysis

Data were done by means $\pm$ standard deviation. The *t*-test was used to compare between the two groups. Correlation

coefficient and odds ratio (OR) for hs-CRP also were measured. The substantial value is deliberated when $P < 0.05$.

RESULTS

Clinical and anthropometric characteristics among patients and controls are revealed in Table 1. There was a substantial rise ($P = 0.01$) in WHR and SBP in diabetic patients as paralleled to the control group. No substantial variance was detected in age, BMI, WC, and DBP between the two groups.

Table 2 shows the metabolic parameters for patients and controls. There was a substantial rise ($P = 0.001$) in FSG in diabetic patients as paralleled to the control group. There was an elevation in HbA1c in diabetic patients as paralleled to the control, but it was not substantial. There was a substantial increase ($P < 0.05$) in serum TC, TAG, LDL-C, and VLDL in diabetic patients as paralleled to the control group. While there was a substantial reduction in serum HDLC in diabetic patients comparable to controls.

Table 1: Clinical and anthropometric characteristics among patients and controls

Parameters	Mean $\pm$ SD		P
	Patients (n=45)	Control (n=45)	
n (male/female)	22/23	22/23	
Age (years)	52.39 $\pm$ 8.13	43.55 $\pm$ 5.70	0.07
BMI (kg/m ²)	32.48 $\pm$ 3.93	25.07 $\pm$ 2.30	0.05
WC (cm)	90.32 $\pm$ 2.25	86.15 $\pm$ 2.21	0.05
WHR	1.92 $\pm$ 0.05	0.80 $\pm$ 0.02	0.01*
SBP (mmHg)	145.29 $\pm$ 0.75	121.34 $\pm$ 0.85	0.01*
DBP (mmHg)	90.74 $\pm$ 0.52	87.88 $\pm$ 0.50	0.08
Duration of DM (years)	5.84 $\pm$ 2.5		

* $P=0.01$ significant, ** $P=0.001$ highly significant. BMI: Body mass index, WC: Waist circumference, WHR: Waisthip ratio, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, DM: Diabetes mellitus, SD: Standard deviation

Table 2: Metabolic parameters in patients and controls

Parameters	Mean $\pm$ SD		P
	Patients (n=45)	Control (n=45)	
FSG (mg/dl)	190.86 $\pm$ 4.05	89.25 $\pm$ 2.36	0.001**
HbA1c (%)	10.41 $\pm$ 2.15	4.82 $\pm$ 0.31	0.05
TC (mg/dl)	224.89 $\pm$ 19.25	152.63 $\pm$ 15.71	0.01*
TAG (mg/dl)	182.34 $\pm$ 18.35	93.75 $\pm$ 10.12	0.001**
HDLC (mg/dl)	44.43 $\pm$ 7.77	62.58 $\pm$ 5.41	0.01*
LDLC (mg/dl)	143.98 $\pm$ 7.81	71.30 $\pm$ 13.37	0.01*
VLDL (mg/dl)	36.47 $\pm$ 3.67	18.75 $\pm$ 3.08	0.01*
TC/HDLC ratio	5.06 $\pm$ 2.42	2.43 $\pm$ 2.70	0.06
TAG/HDLC ratio	4.12 $\pm$ 2.34	1.50 $\pm$ 1.85	0.08
LDLC/HDLC ratio	3.24 $\pm$ 1.02	1.14 $\pm$ 2.42	0.06

* $P=0.01$ significant, ** $P=0.001$ highly significant. FSG: Fasting serum glucose, HbA1c: Glycated hemoglobin, TC: Total cholesterol, TAG: Triacylglycerol, HDLC: Highdensity lipoproteincholesterol, LDLC: Lowdensity lipoproteincholesterol, VLDL: Very lowdensity lipoprotein, SD: Standard deviation

There was an elevation in serum lipid ratios, but they were not significant.

Table 3 shows the renal function tests for both groups. There was a substantial rise ($P = 0.01$) in serum urea and creatinine in diabetic patients as paralleled to the control group.

The hs-CRP levels in the patients' group and controls are described in Table 4. There was a substantial increase ($P = 0.01$, OR = 1.157) in hs-CRP in diabetics as compared to the controls.

Gender differences in hs-CRP value for patients and controls are demonstrated in Figure 1. There was a substantial rise in serum hs-CRP in females as paralleled to males in all groups.

The means $\pm$ SD of hs-CRP levels and HbA1c in the patients' group are demonstrated in Table 5. There was a reduction in hs-CRP when the HbA1c value was reduced. Furthermore, there was a substantial positive correlation between hs-CRP and BMI, FSG, HbA1c, TC, TAG, LDL-C, urea, creatinine, while there was a substantial negative correlation between serum hs-CRP and serum HDL-C in diabetic patients [Table 6].

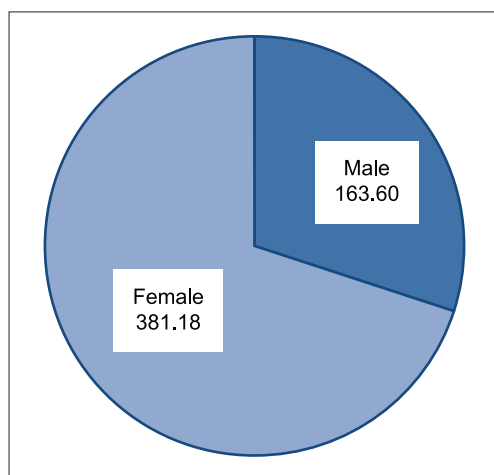


Figure 1: Gender differences in high sensitivity-C-reactive protein value for patients and controls

Table 3: Renal function tests in patients group and controls

Parameters	Mean $\pm$ SD		P
	Patients (n=45)	Control (n=45)	
Urea (mg/dl)	47.93 $\pm$ 12.196	25.32 $\pm$ 2.95	0.01*
Creatinine (mg/dl)	1.93 $\pm$ 0.40	0.30 $\pm$ 0.26	0.01*

* $P=0.01$ significant, . SD: Standard deviation

Table 4: The high sensitivityCreactive protein levels in patients' group and controls

Parameters	Mean $\pm$ SD		OR	P
	Patients (n=45)	Control (n=45)		
hsCRP (mg/l)	13.95 $\pm$ 5.42	2.0 $\pm$ 1.16	1.157	0.01*

* $P=0.01$ significant. SD: Standard deviation, hsCRP: High sensitivityCreactive protein, OR: Odds ratio

DISCUSSION

Nephropathy occurs as a result of DM. It has been stated that diabetes control deteriorated with longer period of the disease, with neuropathy, retinopathy, renal complications, CV complications, and foot ulcers.^[11] It can happen in both type 1 and 2 DM. The occurrence and pathology of nephropathy differ in both forms of diabetes.^[12] Although the occurrence of diabetic nephropathy was deliberated to be considerably developed in type 1, more data propose that the renal risk is equal in both diabetes.^[13]

In this study, there was a rise in BMI in diabetic patients as matched to the control and this is in covenant with the study of Pan *et al.*, 2017.^[14]

Raised blood pressure (BP) is an additional vital liberated risk influence for nephropathy. Lower BP was related with decreased risk for development from moderate to severe albuminuria or ESRD. Furthermore, in patients with T2DM, lower BP was connected with deterioration from moderate albumin to normoalbuminuria.^[15]

The patients had greater HbA1c levels corresponded with the controls, and adjustment for the Hb1Ac levels would be problematic due to its striking variance between patients and controls.^[16]

In the current study, WHR, FSG, TC, TAG, and LDL-C were found to be considerably higher in diabetic patients when paralleled with the control group. Nevertheless, serum HDL-C

Table 5: The high sensitivity-C-reactive protein levels and glycated hemoglobin in patients group

HbA1c (%)	hs-CRP (mg/l)
≤ 7	6.0 $\pm$ 5.22
7-8	10.50 $\pm$ 7.18
8-9	19.20 $\pm$ 2.50

hs-CRP: High sensitivity-C-reactive protein, HbA1c: Glycated hemoglobin

Table 6: Correlation coefficients between high sensitivity-C-reactive protein and other parameters in patients' group

hs-CRP (mg/l)	Correlation coefficient (r)
BMI (kg/m ²)	0.32
FSG (mg/dl)	0.71**
HbA1c (%)	0.26
TC (mg/dl)	0.64*
TAG (mg/dl)	0.72**
HDL-C (mg/dl)	-0.74*
LDL-C (mg/dl)	0.78*
Urea (mg/dl)	0.45*
Creatinine (mg/dl)	0.36

* $P=0.01$, ** $P=0.001$. BMI: Body mass index, FSG: Fasting serum glucose, HbA1c: Glycated hemoglobin, TC: Total cholesterol, TAG: Triacylglycerol, HDL-C: High-density lipoprotein-cholesterol, LDL-C: Low-density lipoprotein-cholesterol, hs-CRP: High sensitivity-C-reactive protein

was significantly low in the diabetics when equated to the controls. Dyslipidemia seems to play a part in the pathogenesis of nephropathy. It has been shown that diabetic patients tend to have a higher BMI and tend to be dyslipidemic, with high serum TAG and low HDL-C concentrations, which were in agreement with the earlier studies.^[17,18]

It has been reported that diabetic nephropathy is a complication due to the long duration of diabetes and connected it with hypertension and elevated levels of serum urea and creatinine.^[19] The certain reason of diabetic nephropathy is indefinite, but several hypothesized processes are hyperglycemia, AGEs, and cytokines triggering.^[20] Although it has been identified that poor glycemic control is one of the main influences accountable for diabetic nephropathy, there are limited facts obtainable about the correlation of serum urea and creatinine levels with the glycemic stage (HbA1c levels) in diabetic subjects.^[21]

In this revision, there was a statistical important elevation in serum urea and creatinine levels in the diabetic patients' group as paralleled to the healthy control subjects and this is in accordance with the earlier study done by Mohan *et al.*, 2013, who have reported a rise in serum urea and creatinine levels in the diabetic population.^[22]

Assumed that improved UAE is the greatest corporate feature of diabetic nephropathy, determining albuminuria is a necessary constituent in the identification of diabetic nephropathy. Rendering to the most current recommendations delivered by the American Diabetes Association, UAE should be restrained at minimum yearly in type 1 diabetic patients with duration ≥ 5 years and in all patients with T2DM,^[23] regarding to this study, means of DM duration was 2.51 ± 5.84 years. Longer duration of diabetes results in micro- and macrovascular injury. Hence, hyperglycemia can donate to the accompanying renal and microvascular complications.^[24]

An increase in serum urea and creatinine levels with the increasing FSG level in this study evidently indicates that hyperglycemia causes some injury to the kidney but the nephrogenic effect is more effectively characterized by serum creatinine levels than blood urea levels. There is a connection which is well established between DM and elevated creatinine levels called nephropathy due to DM. The higher creatinine levels or more from the normal value is the severity of nephropathy.^[25]

In the present study, patients with renal impairment develop hypoalbuminemia, which is in accordance with the study of Ahmed *et al.*, 2010, which proposed a complex setting of conditions, with systemic inflammatory response as a major cause, enduring, other factors such as malnutrition and overhydration can also play a relevant role.^[26]

Among the several biomarkers that have been proposed for CV risk stratification, hs-CRP appears to donate to the identified people at risk of developing CVD.^[27] Data from this study confirm that the high levels of hs-CRP and OR = 1.157 are associated with diabetes by high concentration in diabetic

patients paralleled to the lowest concentration in the controls. In accordance with the present finding, Abdelnabi and Sadek, 2018, reported that the median hs-CRP levels were considerably higher in diabetic patients as paralleled to nondiabetic.^[28]

In this study of Iraqi diabetic patients, raised baseline serum CRP levels were related with an elevated risk of T2DM. Nevertheless, when stratified by baseline HbA1c levels, it has been found that hs-CRP was positively related with T2DM among those already with high HbA1c levels (8%–9%), but not in those with low HbA1c levels (incident diabetes). Consequently, elevated hs-CRP levels might be by-products of hyperglycemia, rather than directly contributing to the progress of incident T2DM. The stronger association between hs-CRP levels and glycemia in Iraqi females compared to males may be clarified by the larger accumulation of subcutaneous fat in females than in males, which is paralleled with earlier study.^[29] Furthermore, serum hs-CRP levels elevate with BMI in adults and are greater in females than males at all higher levels of BMI.^[30]

The primary physiological irregularities in T2DM related to these metabolic defects stay unclear. It is considered that inflammation has a central intermediary part in the T2DM pathogenesis, thus connecting DM with a number of frequently coexisting disorders assumed to initiate through inflammatory mechanism. Recent research recommends that patients with raised basal level of CRP are at an increased risk of DM, hypertension, and CVD. Raised inflammatory biomarker as CRP has been detected in obese patients with T2DM,^[31] which is in parallel with the current data.

In addition, in this study, there was an association between higher levels of hs-CRP in diabetic patients with the highest value of WHR, FSG, TC, TAG, serum urea, and creatinine levels, which is in agreement with the earlier study.^[32]

CONCLUSIONS

It has been concluded that higher levels of hs-CRP can be a prognostic element for the development of DM. It also has positive correlation with HbA1c, reflecting the glycemic status of the patient. This study also suggests that a good planning to monitor complications of ESRD among diabetic patients can be done by measuring the hs-CRP concentration in addition to other recommended biochemical factors.

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Conflicts of interest

There are no conflicts of interest.

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