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Inflammatory Markers in Chronic Kidney Disease

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Abstract

Background: Despite recent advances in chronic kidney disease (CKD) and end-stage renal disease (ESRD) management, morbidity and mortality in this population remain exceptionally high. Persistent, low-grade inflammation has been recognized as an important component of CKD, playing a unique role in its pathophysiology and being accountable in part for cardiovascular and all-cause mortality, as well as contributing to the developmentof protein-energy wasting. Material and methods; This study was conducted on CKD patients attending OPD & IPDof Civil Hospital tarn taran. The participants having age more than 18 years & less than 60 years. We assessed 120individuals out of 60 are normal healthy individuals comprising the control groups & rest 60 is of CKD cases. SerumCRP (mg/dl) concentration was measured by Latex agglutination test & ESR (mm/hour) was measured by Wintrobe's method. Serum Creatinine (mg/dl), Urea (mg/dl) & Uric acid (mg/dl) concentration was measured by Modified Jaffe'smethod, Urease & Uricase method respectively. Results: - In the present study, serum CRP & ESR was increased in CKD patients. The mean serum CRP levels of CKD patients & controls were 33.55 ± 22.8 & 2.07 ± 6121 respectively ($p< 0.001$), highly significant result was observed. Mean level of serum ESR (40.25 ± 14.93) of cases shows statistically significant differences as compared with the mean of serum ESR of controls (13.50 ± 3.421).

CRP & ESR are the markers used to evaluate kidney disease, however, each of these has its own limitation. The use of these inflammatory biomarkers may better assess overall patients' risk & be able to stage patients more appropriately.

Keywords: *Chronic Kidney disease, Erythrocyte Sedimentation Rate, - C-reactive protein*

Introduction

Chronic kidney disease (CKD) generally causes reduction of the Glomerular Filtration Rate which indicates decreasing number of functioning nephrons [1]. Over the past 15 years, there has been an exponential growth of interest in inflammation in CKD and End-stage renal disease (ESRD) [2,3]. CKD has become a public health problem. The definition of CKD was introduced by the National Kidney Foundation in 2002 and latter adopted by the international group Kidney Disease Improving Global Outcomes in 2004 [4]. CKD is defined as abnormal kidney structure and functions persisting greater than 3 months. This can be determined either by evidence of kidney damage (presence of persistent albuminuria) or by decreased GFR [5,6]. Symptoms of kidney disease often developed only in advanced stages. The most commonly reported symptoms were weakness, decreased urine output, poor appetite, dyspnea, sleeping, bone or joint pain, breathlessness. Nearly 30% of CKD in our country are due to diabetic nephropathy and it is thus the single most common cause of Chronic renal failure [7]. It can be classified on the basis of estimated glomerular filtration rate (eGFR), low risk of progression to kidney failure start from stage 3. Data from the American National Health and Nutrition examination survey demonstrate that in the period 1999 to 2004 the prevalence of CKD stages 1–4 increased Significantly when compared with the survey period 1988–1994 (13.1 vs. 10.0%) due to this high prevalence it is also associated with increases in diabetes mellitus and hypertension. Hypertension and diabetes are the crucial cause of CKD. ESRD is defined by an eGFR <15ml/min/1.73m2. Inflammation is an essential part of CKD.

Inflammation is present in a wide spectrum of patients with CKD [8]. Chronic inflammation in the body can be measured by the increased level of inflammatory markers. Biomarkers of inflammation Adenosine deaminase (ADA) Activity, C-reactive protein (CRP), Erythrocyte sedimentation rate (ESR) could be increased as the kidney function decreases [9].

Materials & Methods

This study was conducted on CKD patients attending OPD & IPD of Civil Hospital tarn taran. The participants having age more than 18 years & less than 60 years. We assessed 120 individuals out of 60 are normal healthy individuals comprising the control groups & rest 60 is of CKD cases.

Sample Technique/Method

Total 5ml of blood was collected from each, 3ml was collected in a plain vial & remaining was collected in EDTA vial. Blood sample were subjected to centrifugation with the speed of 3000rpm for 10 min. to obtain serum for the estimation of C- reactive protein (CRP), Serum Uric acid, Serum Urea & Serum Creatinine sample of EDTA vial was directly used for the estimation of ESR.

In all subjects, these renal function and inflammatory serum markers were quantified: Serum C-reactive protein (mg/dl) concentration was measured by Latex agglutination test, Erythrocyte Sedimentation Rate (mm/hour) was measured by Wintrobe's method, Serum Creatinine (mg/dl) was measured by modified Jaffe's method, Serum Urea (mg/dl) concentration was estimated by Urease method & Serum Uric acid (mg/dl) concentration was measured by uricase method.

Results and Observations

The present study was Conducted in Department of Biochemistry, Civil Hospital Tarn Taran with an objective to study of a inflammatory markers in chronic kidney disease for this purpose, a total of 120 sample were taken.

Table 1: Comparison of Common Parameter between Cases & Controls

Biochemical Parameter	Control Mean ± sd	Cases Mean ± sd	P- value
Urea	*27.384 ± 9.299*	*138,2 ± 81.341*	*.000*
Creatinine	*.8964± .299*	*5.278 ±4.168*	*.000*
Uric acid	*4.326 ±1.753*	*7.866± 2.923*	*.000*

Table 2: Comparison of CRP & ESR Between Cases & Controls

Biochemical parameter	CONTROLS MEAN± SD	CASES MEAN ±SD	p- VALUE
CRP	*2.071± 6122*	*33.56 ±20.81*	*.000*
ESR	*13.51 ±3.422*	*40.25± 14.93*	*.000*

Table 3: Pearson Correlation Coefficient (R – Value) Of CRP With Various Biochemical Parameters (Age, Weight, Urea, Uric Acid, Creatinine, Esr)

Biochemical parameter	P – VALUE	r- VALUE
Age	*.267*	*.147*
Weight	*.433*	*.103*
Urea	*.000*	*.493*
Creatinine	*.001*	*.424*
Uric acid	*.088*	*.224*
Esr	*.004*	*.382*

Discussion

Patients who develop kidney problems usually have no symptoms early on, although the condition puts them at risk of developing more serious kidney disease. It is important to take steps to protect kidneys before the problem advances. The present study examined inflammatory markers in blood sample and correlate their levels with estimated eGFR for the assessment of early renal damage in CKD patients and compare it with that of controls. It was carried out in Civil hospital tarn taran. In the present study, CRP & ESR was increased in CKD patients. CRP is a acute phase protein that is a member of the pentraxin. It is a pattern recognition protein that are an integral part of the innate immune system. It is synthesized in the liver in response to inflammatory cytokines & assists in complement binding & phagocytosis by macrophages. Studies

have shown that epithelial cells of renal epithelium can also produced CRP under certain circumstances. Synthesis of CRP in liver is triggered by pro – inflammatory cytokines released from monocytes & macrophages. The pro – inflammatory response leads to secretion of IL - 1β & Tumour necrosis factor α which further results in the release of IL – 6, a messenger cytokine which stimulate liver to secrete CRP. In chronic inflammatory condition, CRP can rise as much as 50 to 100 mg/l within 4 to 6 hours. CRP levels double every 8 hours & peak 36 to 50 hours after the onset of inflammation or injury. Mild increases in CRP b/w 2 mg/l to 10 mg/l are considered to be metabolic inflammation. Levels of CRP fall quickly because of its short half-life (4 to 7 hours) once inflammation subsides.

Conclusions

CRP & ESR are the markers used to evaluate kidney disease, however, each of these has its own limitation. The use of these inflammatory biomarkers may better assess overall patients risk & be able to stage patients more appropriately.

Funding

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Conflict of interest

None declared.

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