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Research Article

SERUM PARATHYROID HORMONE AND ITS ASSOCIATION WITH RENAL FUNCTION IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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A B S T R A C T

Introduction: Chronic Kidney Disease (CKD) is a global health problem which involves progressive loss of renal function over a span of time. Kidneys play a major role in homeostasis of various metabolites and a loss of kidney function may lead to their severe derangement. One such derangement is an increase in serum phosphorus levels followed by decreased calcium levels. This imbalance in the levels of serum calcium and phosphorus further leads to hyperparathyroidism. **Aim:** The present study was planned to assess the serum parathyroid hormone (PTH) levels of patients with CKD (stage 4 & 5) and to explore its association with the declining renal function. **Materials and Methods: 50** CKD patients (stage 4 & 5) were enrolled for the study based on pre defined inclusion and exclusion criteria. 50 age and gender matched healthy controls were also included. Renal function tests (RFT) i.e. urea. Creatinine, uric acid and eGFR and iPTH were estimated for all enrolled subjects. Results obtained were compared among CKD patients and healthy controls. Further the correlation of iPTH with urea, creattinine, uric acid and eGFR was also evaluated**. Result:** Serum iPTH levels were significantly higher in the CKD patients group (P= 0.000). iPTH also showed a significant correlation with urea (r= 0.362), creatinine (r= 0.475) and eGFR (r= -0.503). **Conclusion:** Decline of kidney function leads to increased S. iPTH levels and an imbalance of Ca and Phosphorus homeostasis. This may result in development of complications like bone-mineral disease. Early screening of PTH levels in CKD patients can be helpful in timely patient management and in averting such complications.

INTRODUCTION

Chronic Kidney Disease (CKD) is a worldwide public health problem with serious adverse health consequences for affected individual. It is a progressive loss in renal function over a period of time, and it may lead to one of its recognized complications such as cardiovascular disease, anemia or pericarditis [(1-2)]. In particular, cardiovascular events and mortality increase as the estimated glomerular filtration rate (eGFR) declines below 60 ml/min [(3)]. In such patients, cardiovascular disease is 10- to 20-fold higher than the general population, representing at least half of the 15–25% per year mortality rate [(4)].

WHO has identified CKD as the 12th major cause of death and the 17th cause of disability worldwide [(5)]. With the decline of kidney function, a series of biochemical derangements occur. One such derangement is the increase in plasma phosphorus levels and simultaneous decrease in plasma calcium and calcitriol levels. Reduction in calcitriol further contributes to a reduction in intestinal calcium absorption. All these factors contribute to the development of secondary hyperparathyroidism[(6)]. Secondary hyperparathyroidism is characterized by hyperplasia of parathyroid gland and increased synthesis of parathyroid hormone [(7)]. Persistently increased secretion of PTH would results in bone disorder, increased risk for cardiovascular disease, decreased immune function and an overall decreased quality of life in patients with CKD [(8-9)]. Therefore identification of patients at risk and evaluating for hyperparathyroidism is imperative because early intervention may slow or arrest consequence of this complication.

The present study was planned to estimate serum levels of iPTH and its association with markers of renal function in CKD patients.

MATERIALS AND METHODS

Fifty diagnosed patients of chronic kidney disease, age 20 to 60 years, visiting the Out Patient Department of Nephrology, Mahatma Gandhi Medical College and Hospital, were enrolled in the study after informed and written consent. Approval from

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the Institutional Ethics Committee was also obtained prior to the study. Patients with acute renal failure, primary hyperparathyroidism and those undergone thyroid and parathyroid surgeries were excluded from the study group. Patients on vitamin D supplementation were also not included. Fifty age and sex matched healthy subjects constituted the control group.

All participants underwent physical examinations including anthropometric assessments followed by biochemical assessments.

Blood samples were collected using standard aseptic technique and analyzed for Serum urea, creatinine, uric acid on VITROS 5600 and serum iPTH on VITROS ECI.

The Glomerular filtration rate (eGFR) was calculated by using Cockeroft and Gault formula.

For males= [(140-age in years)x(weight in kg)] ÷ 72 X serum creatinine
For Females= 0.85 X [(140-age in years)x(weight in kg)]÷ 72 X serum creatinine
Results obtained were presented as mean± SD for the case and control groups and compared statistically using SPSS software.

RESULTS

Of the total fifty CKD patients, thirty one patients (62%) had eGFR less than 15ml/min/1.73m^2 i.e. Stage 5 and 19 patients had eGFR between 15 to 29ml/min/1.73m^2 i.e. Stage 4 **(Figure 1).**

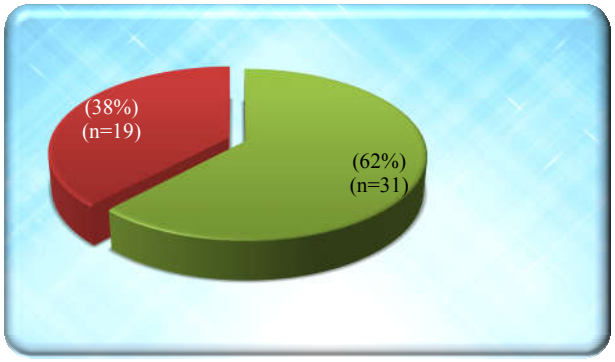

Figure 1 Distribution of the CKD patients on the basis of eGFR

Mean age of CKD patients (38.98 ± 10.32 years) was comparable with healthy subjects.

The mean values of renal profile parameters are presented in Table 1. As expected, the parameters (Urea, Creatinine and Uric acid) were significantly higher in CKD patients as compared to healthy subjects. The mean serum iPTH levels were significantly higher in CKD population (301.773 ± 218.172 pg/ml) as compared to age-matched healthy subjects with normal kidney function (41.190 ± 16.211pg/ml). Out of fifty patients, 44 patients (88%) had abnormal iPTH levels and only 06 of them had iPTH within normal range (7.5-53.5 pg/mL) (Table 3). Further, the correlation of renal profile parameters with serum iPTH levels was also evaluated (Table 2). It was observed that, increased level of serum iPTH is negatively correlated with eGFR (r= -0.503) [Figure 2(a)]. However, serum iPTH exhibited a positive correlation with serum urea (0.362) [Figure 2(b)] and creatinine (0.475) [Figure 2(c)]. No significant association was observed between serum iPTH and Uric acid levels [Figure 2(d)].

Table 1 Distribution of variables between CKD patients and Control group

Variables	CKD patients	Control Group	P-value
Age (years)	38.98 ± 10.32	38.28 ± 13.72	NS
eGFR (ml/min)	14.31 ± 4.19	106.37 ± 13.01	0.000
S. Urea (mg/dl)	109.34 ± 48.26	26.32 ± 5.17	0.001
S. Creatinine (mg/dl)	8.56 ± 2.90	0.72 ± 0.13	0.000
S. Uric acid (mg/dl)	7.870 ± 2.570	4.218 ± 1.474	0.000
S. iPTH (pg/ml)	301.73 ± 218.17	41.19 ± 16.21	0.000

Table 2 Correlation of renal profile parameters with iPTH levels

Variables	Pearson Correlation Coefficient (r)	*p*-value
eGFR	**-0.503**	**0.000*****
Urea	0.362	0.009**
Creatinine	0.475	0.001***
Uric Acid	0.245	0.087

Table 3 Distribution of the CKD patients on the basis of serum iPTH levels

iPTH (pg/ml) (7.5-53.5 pg/ml)	CKD Patients	
	No. of cases(n)	%
Abnormal	44	88
Normal	6	12
Total	50	100

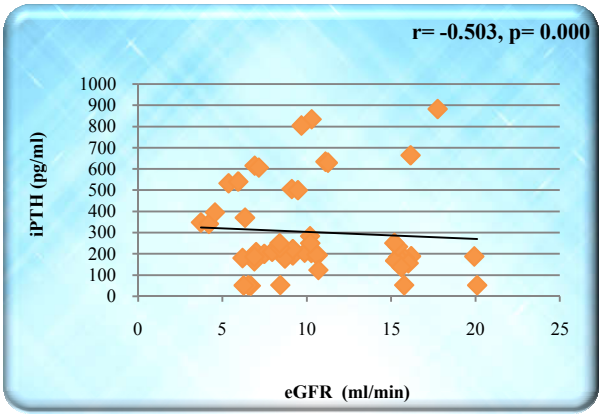

Figure 2 (a) Correlation of eGFR with iPTH levels

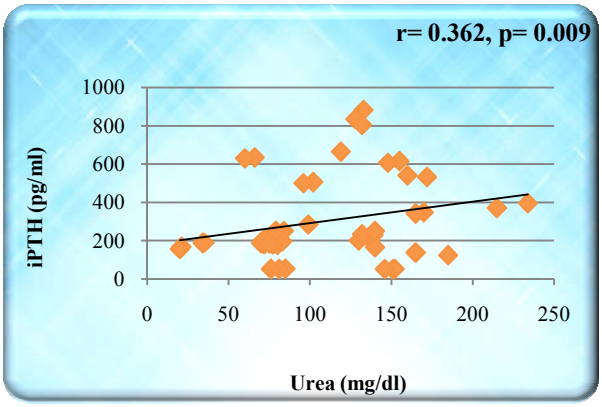

Figure 2 b Correlation of blood urea with iPTH levels

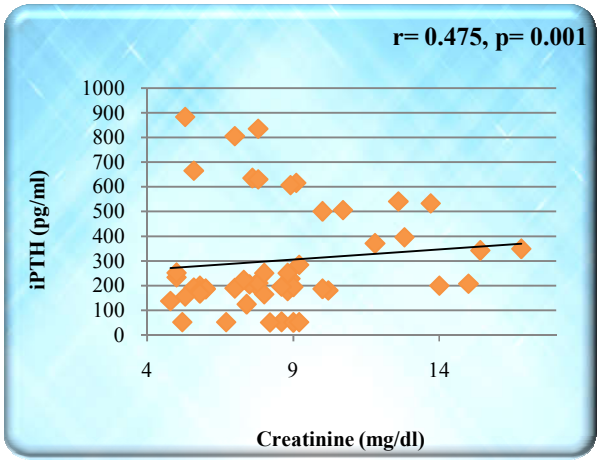

Figure 2 c Correlation of serum creatinine with iPTH levels

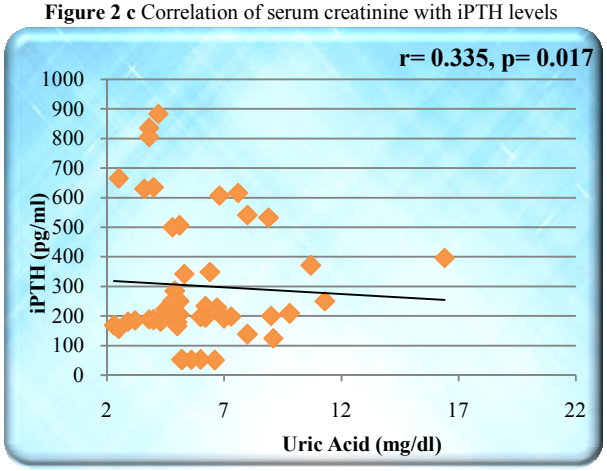

Figure 2 d Correlation of serum uric acid with iPTH levels

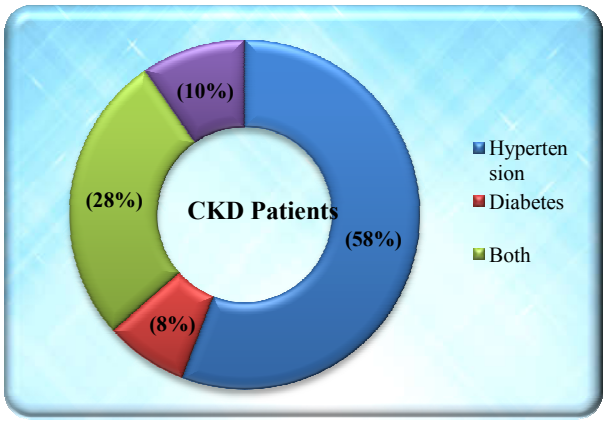

Figure 3 Distribution of the CKD patients on the basis of Comorbid Conditions

The distribution of co-morbid conditions in CKD patients was also evaluated. The study reported hypertension as the most common co-morbid condition associated with CKD. 58 % of the CKD patients were hypertensive (Figure 3).

DISCUSSION

Patients enrolled in the study were suffering from stage 4 or 5 CKD. The iPTH levels were observed to be significantly higher among CKD patients. Our findings are in accordance with previous studies [10-13].

The correlation analysis exhibited that increased serum iPTH has a strong association with estimated by eGFR and creatinine. Serum Urea and Uric acid were also observed to have a significant association with iPTH levels.

The development of secondary HPT results from many factors, including deficiency of calcitriol, retention of phosphorus, a decrease in the activation of the calcium-sensing receptor (CaR) in the parathyroid gland, and skeletal resistance to the calcemic effect of PTH. As kidney function declines, so does phosphorus excretion, thus causing plasma phosphorus levels to rise while plasma calcium and calcitriol levels decrease.

In a recent study done by Tripathi V *et al* 2015; it was reported that CKD patients with high (≥400 pg/mL) iPTH have 8.93 times the risk of developing intimal thickness (IT) of ≥60 µm as compared with patients with low (<400 pg/mL) iPTH (P-value <0.05).

Epidemiological studies have shown that damage of large arteries is a major contributing factor to morbidity and mortality in patients with chronic kidney disease (CKD) and in those with end-stage renal disease (ESRD). Further, they concluded that hyperparathyroidism is one of the mechanisms of uremic toxicity and important cause of ongoing vascular damage that may contribute to higher vascular events in CKD patients. Anderson *et al* 2011; found that elevated iPTH is associated with a higher prevalence and incidence of CV risk factors and predicts a greater likelihood of prevalent and incident, including mortality.

CONCLUSION

The present study identified hypertension as the most common comorbidity in CKD. Estimation of serum iPTH; evels is recommended at early stage of renal failure. This would be helpful in identification of patients at risk of developing CV complications. An early diagnosis of hyperparathyroidism can guide proper treatment of patient and shall help the clinician in management of any anticipated biochemical derangement.

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