



Assessment of Serum Calcium, and Phosphorus Levels in Chronic Kidney Disease

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ABSTRACT

Chronic kidney disease (CKD) is one of the major public health problems. Unlike acute renal failure, which happens quickly and suddenly, chronic renal failure happens gradually - over a period of weeks, months, or years - as the kidneys slowly stop working, leading to end-stage renal disease (ESRD). A prominent hallmark of CKD that may contribute to this increased risk is a disturbed electrolyte homeostasis, including calcium (Ca^{2+}) and phosphorus (Pi) deregulation. The severity of symptoms caused by hypocalcaemia will depend on the magnitude of hypocalcaemia and the rate of rise in serum calcium. Currently, there is limited evidence on the impact of the metabolisms of calcium and phosphorus abnormalities in patients with chronic kidney disease not receiving dialysis. Hence, it was proposed to evaluate the levels of calcium and phosphorus in the above said patients.

Keywords: Chronic kidney disease (CKD), calcium, phosphorus.

INTRODUCTION

Chronic kidney disease (CKD) is a major public health issue with a high prevalence currently affecting millions of people worldwide.¹ CKD results in significant morbidity and mortality and highly associates with the development of cardiovascular events, the primary cause of death in CKD patients.²⁻⁴ A prominent hallmark of CKD that may contribute to this increased risk is a disturbed electrolyte homeostasis, including calcium (Ca^{2+}) and phosphorus (Pi).⁵ Gastrointestinal symptoms are the most common, including nausea, vomiting, constipation and abdominal pain. Patients may also complain of difficulty in concentrating, fatigue, lethargy, and muscle weakness. Hypercalcemia can induce nephrogenic diabetes insipidus with resulting Polyuria that worsens the hypocalcaemia because of volume depletion. Although cardiac arrhythmias are rare, they are more likely with digitalis toxicity. Hypercalcemia, especially in the setting of volume depletion can lead to acute renal failure. Hyperphosphatemia can occur from increased intestinal absorption, cellular release or rapid intracellular to extracellular shifts, or decreased renal excretion.⁶ There is limited evidence on the impact of the metabolism of calcium and phosphorus abnormalities in patients with chronic kidney disease not receiving dialysis. Hence, it was proposed to evaluate the levels of calcium and phosphorus in the above said patients.

MATERIALS AND METHODS

The case control study was carried out at Shri Sathya Sai Medical College and Research Institute. A total of 50 normal persons of the age group of 30 – 60 yrs and both sexes without CKD were enrolled as control group. Age

and sex matched 50 Patients with CKD were treated as a study group. Blood samples were collected from the above two groups and analysed for serum calcium⁷ and serum phosphorus levels.⁸ All the parameters were analysed using standard kits in Semi-Auto analyser (Bio-systems BTS 350).

Statistical Analysis

The values were expressed as Mean \pm Standard deviation and the findings were analyzed by Student "t" test. "P" value less than 0.05 was considered statistically significant. Data analysis was performed using SPSS, version 17.0.

RESULTS AND DISCUSSION

In the present study, the calcium levels showed a decline and elevated levels of phosphorous was observed when compared with the control ones (Table 1). These levels were statistically significant ($P > 0.005$ & $P > 0.001$) when compared with the control values.

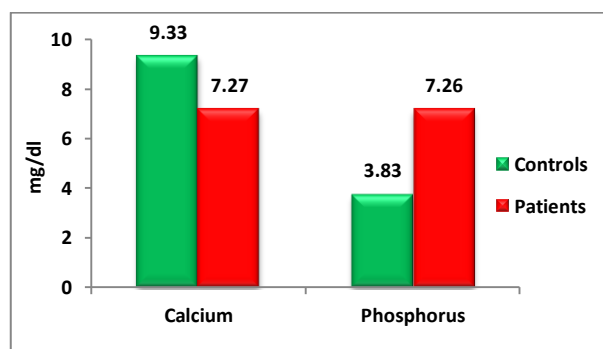


Figure 1: Calcium and phosphorus levels in control and patients.

Table 1: Biochemical parameters in CKD

Biochemical Parameters	Group	Mean \pm SD	Significance
Calcium	Controls	9.33 \pm 0.48	P < 0.005
	Patients	7.27 \pm 0.47	
Phosphorus	Controls	3.83 \pm 0.73	P < 0.001
	Patients	7.26 \pm 0.60	

These aberrations could be due to increased tissue release of phosphorus which is commonly seen in acute renal failure, further aggravating hyperphosphatemia. Acute hyperphosphatemia generally does not cause symptoms unless there is precipitation of phosphorus with calcium leading to symptoms of hypocalcaemia. Renal excretory function plays an important role in maintaining the balance of calcium and phosphate. As per the reference of Adeera Levin et al⁹, progressive CKD, results in the development of hyperphosphatemia and hypocalcemia which corroborates with the present study. As renal function declines in chronic kidney disease, calcitriol deficiency is promoted because the kidney is a site of the 1α -hydroxylation of 25-hydroxyvitamin D to its active form, 1,25-dihydroxyvitamin D (calcitriol) which could produce hypocalcaemia. Renal biopsy showed calcification due to calcium-phosphate precipitates in the interstitium and tubules.¹⁰ It was reported that there is hypocalcaemia in hypertensive patients.⁷ In this study also, hypocalcaemia may be explained on the basis of the development of hypertension in CKD.

CONCLUSION

It may be concluded from this study that there exists hypocalcaemia and hyperphosphatemia in CKD. Evaluations of these parameters help to maintain the balance which may be of use in treating the CKD patients.

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