



Original Article

Value of Calcium and Phosphorous in chronic kidney disease patients under hemodialysis: A retrospective study

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ABSTRACT

Background: Derangements of mineral metabolism occur during early stages of chronic kidney disease (CKD). Mineral metabolism has emerged as important predictors of morbidity and mortality in dialysis patients. This study aims to evaluate the values of calcium and phosphorous in reference to practice guidelines issued by National Kidney Foundation Dialysis Outcomes Quality Initiative (K/DOQI) in patients undergoing hemodialysis in KIST Medical College Teaching Hospital.

Materials and Methods: It was a retrospective study which included 101 patients who underwent hemodialysis in KIST Medical College Teaching Hospital over a period of three months (October 2010 to December 2010). The laboratory parameters of serum calcium, and phosphorus were evaluated and were compared with that of recommended values by K/DOQI guidelines.

Results: The mean values for serum calcium, phosphorus, and calcium-phosphorus product were 8.79 ± 1.48 mg/dL, 5.30 ± 1.32 mg/dL and 46.62 ± 1.96 mg²/dL² respectively. Forty (40%), 53 (53%) and 76 (76%) cases of CKD patients met K/DOQI target value for calcium, phosphorous and calcium-phosphorous product respectively. Twenty one percent of the patients meet the three K/DOQI guidelines and 14% did not meet any of the recommended guidelines by K/DOQI.

Conclusion: To achieve all the K/DOQI guidelines multi-disciplinary team approach is crucial. More extensive multicenter studies need to be carried out. It is necessary to develop our national guidelines in future.

INTRODUCTION

A chronic kidney disease (CKD) is emerging as one of the major public health problems across the world and its incidence is rising day by day.¹ CKD may lead to end-stage renal disease (ESRD) and is also associated with increased

risk of cardiovascular disease, heart failure, and increased healthcare expenditures.²

Mineral metabolism has emerged as important predictors of morbidity and mortality in dialysis patients.³ Disturbances in calcium and phosphorus is commonly observed in patients with CKD. Renal osteodystrophy, a metabolic bone disease usually accompanies CKD. It is the result of hyperparathyroidism secondary to hyperphosphatemia

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Table 1: Value of laboratory parameters in HD patients (n=101)

Variables	Mean	SD
Calcium (mg/dL)	8.79	1.48
Phosphorus (mg/dL)	5.3	1.32

Table 2: Comparison of laboratory values with K/DOQI target ranges for mineral metabolism

Factor and K/DOQI ranges	Patient percentage†
Serum Calcium (mg/dL)	
<8.4	29
8.4-9.5‡	40
>9.5	32
Serum Phosphorous (mg/dL)	
<3.5	6
3.5-5.5‡	53
>5.5	42
Calcium-Phosphorous Product (mg ² /dL ²)	
<55‡	76
55-72	23
> 72	2

†, percentages are roundup to the nearest figure
‡, K/DOQI target value

Table 3: Achievement of K/DOQI guideline targets for mineral metabolism

Status	Patients (%)
None of the targets achieved	14
At least one of the target achieved	87
At least two of the target achieved	59
Three of three target considered achieved	21

combined with hypocalcaemia, both of which are due to decreased excretion of phosphate by the damaged kidney. Healthy kidneys convert inactive vitamin D to its active metabolite, 1,25 dihydroxycholecalciferol. If renal functioning is affected, the production of 1,25 dihydroxycholecalciferol reduces that leads to hypocalcaemia because less calcium is absorbed from the intestine. In addition, the renal clearance of phosphorus decreases with a deteriorating kidney function and phosphorus concentrations increase. Hypocalcaemia in its turn leads to an increased production of parathyroid hormone (PTH) by the parathyroid glands, which is called secondary hyperparathyroidism.⁴

There is no documented data related to mineral metabolism outcomes in Hemodialysis (HD) patients in Nepal. Several expert panels have issued management guidelines for the management of altered mineral metabolism in dialysis patients. Among them, the guidelines published in 2003 by K/DOQI, recommended the serum levels of phosphorus

between 3.5-5.5 mg/dl; serum calcium 8.4-9.5 mg/dl and calcium-phosphorus product, < 55 mg²/dl², and serum PTH, between 150-300pg/ml.⁵ This study aims to evaluate the values of calcium and phosphorous in reference to practice guidelines recommended by K/DOQI in patients undergoing HD in KIST Medical College Teaching Hospital (KISTMCTH), Nepal.

MATERIALS AND METHODS

We retrospectively evaluated the laboratory parameters of serum calcium, and phosphorus from 101 (62 male and 39 female) patients who underwent HD at KISTMCTH over three months period from October 2010 to December 2010. The calcium and phosphorous evaluation was done by using Biochemical semi-analyzer (STATFAX 3000). The values of serum calcium, phosphorus and calcium-phosphorus product were compared with K/DOQI guidelines.⁵

RESULTS

Out of 101 patients, 62 were male and 39 were female. Age ranged from 20 to 80 years. Average age of the study population was 48.36±17.94 years. Seventy two percent of the patients were between the ages of 20-60 years. The mean duration of HD session was 3.95 ± 0.42 hours. The mean number of HD session per week was 2.08 ± 0.46 days. Patients were given phosphate binders (calcium carbonate and/or calcium acetate and/or lanthanum carbonate) depending upon their status of serum calcium level, phosphorous level and clinical condition.

Serum value of calcium and phosphorus is presented in Table 1. Table 2 depicts the comparison of laboratory tests results in patients on HD with K/DOQI target ranges for mineral metabolism. Twenty one percent of the patients met the three considered guidelines successfully and 14 % did not meet any of the recommended guidelines as shown in Table 3.

DISCUSSION

Abnormal mineral metabolism start early in course of CKD and ESRD, and is usually accompanied by profound changes in mineral metabolism that leads to clinical problems such as bone diseases, musculoskeletal symptoms and growth retardation. Recent study has found a strong association between mortality and abnormal mineral metabolism; presumably mediated by vascular calcification.⁶ The disturbances include calcium, phosphate, vitamin D, and PTH homeostasis. So, mineral metabolism outcomes are gaining importance in HD treatment. Hyperphosphatemia is one of the major factors responsible for alterations in mineral and bone metabolism in dialysis patients.⁷ In our study, the control of serum phosphorus values was adequate in 53% of patients undergoing HD, while 42% of the patients presented with hyperphosphatemia. These findings are

comparable to the study in Iran by Mahdavi-Mazdeh et al.⁸ Hyperphosphatemia occurs as a consequence of diminished phosphorous filtration and excretion with the progression of CKD. Decrease phosphorus excretion can initially be overcome by increase secretion of PTH which decreases proximal phosphate reabsorption. Hence phosphorus levels are usually within normal range until glomerular filtration rate (GFR) falls below approximately 30mL/min or CKD-4 according to K/DOQI classification. In more advanced stage of CKD, the urinary excretion of phosphorus can no longer keep pace with the obligatory intestinal phosphate absorption resulting in hyperphosphatemia. The recent finding suggests that elevated serum phosphorus is associated with an increase risk for cardiovascular mortality and morbidity among dialysis patients.⁹ The consequences of hyperphosphatemia include the development and progression of secondary hyperparathyroidism and a predisposition to metastatic calcification when the product of serum calcium and phosphorus is elevated. Both of these conditions may contribute to the substantial morbidity and mortality seen in patients with ESRD.⁶ In our study, 29% were hypocalcemic (< 8.4) and the K/DOQI guidelines for calcium was achieved in 40 % of the patients. However 32% patients had their calcium level above K/DOQI guidelines. Similarly the product of serum calcium and phosphorus was elevated in 24 %.

A study by Schwartz et al showed an association between higher levels of serum phosphorus and calcium-phosphorus product with an unfavorable renal outcome.¹⁰ In their study higher serum phosphorus was associated with significantly higher risk for progression of CKD, even after adjustment for multiple potential confounders. The association of higher serum phosphorus with progressive CKD was more accentuated in patients with higher serum calcium levels also supports the hypothesis that tissue calcification may be the reason behind the observed associations. This association seems now to involve active steps at the cellular and subcellular levels, with hyperphosphatemia shown to be associated with increased expression of osteoblast-specific proteins in vascular smooth muscle cells.¹¹⁻¹³ Reynolds et al showed that higher ambient serum calcium level led to more significant phosphorus-driven calcification of vascular smooth muscle *in vitro*.¹⁴

In this study, most of patients were in age group between 20-60 years of age. In developed world, mean age of CKD-5 leading to ESRD is between 60-63 years. In our study, average age of presentation was around 48 years which is similar to median age of 43 years in India.¹⁵

In this study, only 21 % of the patients met the three considered guidelines of mineral metabolism outcomes after dialysis successfully and 14 % did not meet any of the recommended guidelines. This is similar to other studies.^{9,16}

Regular dialysis treatment is unable to remove all the

phosphorous ingested with a diet containing sufficient amount of protein to avoid malnutrition. In this regards, our patients were taking phosphate binders such as calcium carbonate, calcium acetate and lanthanum carbonate to decrease dietary phosphorous depending upon their serum calcium and phosphorus levels. The treatment with these calcium-based phosphate binders is not free from complications such as hypercalcaemia. The non-calcium phosphate binder such as lanthanum carbonate is an effective and well tolerated agent for the treatment of hyperphosphatemia in patients with ESRD. Therefore, the strict maintenance of serum calcium and phosphorus levels within the ranges recommended by the K/DOQI guidelines is difficult to achieve due to these multifactorial reasons.¹⁷⁻²⁰

Craver et al argue and emphasize on the fact that data from CKD patients from one population may not apply to other population due to differences in social, ethnic and health system characteristics as well as nutritional habits.²¹

CONCLUSION

To achieve all the K/DOQI guidelines, multi-disciplinary team approach is crucial. We emphasize on the need of large scale multi-centre study of mineral metabolism of CKD patients in Nepal. This will be helpful in establishing our own local guidelines in future.

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