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Research Article

ALTERATIONS IN PARATHORMONE, CALCIUM, AND PHOSPHORUS LEVELS IN CKD PATIENTS ON MAINTENANCE HEMODIALYSIS IN A HOSPITAL SETTING IN PUNJAB

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ABSTRACT

Objective: Many people who have severe chronic kidney disease (CKD) will eventually develop kidney failure and will require dialysis. The control of parathormone (PTH), phosphorus, and calcium metabolism is one of the objectives in an adequate dialysis protocol. Therefore, we conducted this study to describe alterations in PTH, calcium, and phosphorous homeostasis in patients with CKD on hemodialysis in our center. Our study also aimed to find an association between hormonal and biochemical abnormalities in CKD patients, who have been on hemodialysis for ≥5 months and comparing the results obtained with that recommended by Kidney Disease Improving Global Outcomes (KDIGO) guidelines.

Methods: This was a hospital-based cross-sectional observational study. The study population of 330 patients (>18 years) on maintenance hemodialysis coming to dialysis Unit of Department of Medicine of Gian Sagar Hospital, Ramnagar (Patiala), over a period of 3 years (2012-2015), were enrolled in the study. Each patient was considered only once for the study. In addition, biochemical analysis of serum intact PTH (iPTH), corrected calcium, phosphorus, total alkaline phosphatase (tALP), serum creatinine, blood urea, serum albumin, and hemoglobin of all cases was done using fully automated equipment. All statistical analyses were performed using SPSS statistical software, version 17.

Results: The study population of 330 patients comprised adults, mainly illiterate (54.5%) predominantly belonging to the rural (66.4%) strata with a mean age of 52.67±15.05 (range: 25-98 years). The abnormality in the laboratory profile of the patients was found to be hyperparathyroidism in 40.3% as compared to hypoparathyroidism in 33.5% and normal iPTH levels in 26.2%. Hypocalcaemia was detected in 50.6% and hyperphosphatemia in 62.1% of the patients. There was statistically significant association of serum iPTH, with corrected calcium and phosphorus (P=0.032 and P=0.035, respectively). Corrected calcium was also significantly associated with phosphorus (P=0.001) and tALP (P=0.007).

Conclusion: We showed in the present study that disorders of mineral metabolism are common in hemodialysis patients and that only a small proportion adheres to the targets as advised in the KDIGO guidelines for bone metabolism and disease in CKD. We demonstrated that these disorders are associated with important negative clinical outcomes, such as increased all-cause lack survival, more muscle and bone problems. Our findings, therefore, support a strict control of mineral metabolism in dialysis patients. Further research and progress in this area are required to establish a more rational approach with a view toward improving patient outcomes.

Keywords: Parathormone, Calcium, Phosphorus, Hemodialysis.

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INTRODUCTION

Calcium and phosphorus are minerals that are of great importance for the composition of bones and the regulation of several processes in the body [1]. The kidney plays a leading role in maintaining calcium and phosphorus homeostasis in collaboration with other organs, i.e., the parathyroid gland, intestines, and bones. Thus, along with the progression of chronic kidney disease (CKD), various abnormalities of mineral and bone metabolism develop such as hyperphosphatemia, hypocalcemia [2], and CKD has a negative effect on parathyroid gland, associated with an increased risk of PTH, namely secondary hyperparathyroidism [3]. These are commonly observed alterations in patients with CKD which can result in significant consequences [2].

Alterations in the control mechanisms for calcium and phosphorus homeostasis occur early in the course of CKD and progress as kidney functions deteriorate; if left untreated, these alterations can result in significant consequences, making disturbances of bone and mineral metabolism a hallmark of CKD [4].

Disturbances in mineral metabolism in CKD which result in multisystem disorder have now been given a different identity as CKD-MBD (CKD-mineral and bone disorder) by Kidney Disease Improving Global Outcomes (KDIGO) 2009 [5]. The KDIGO is an international initiative with a key mission of developing evidence-based clinical practice guidelines for the prevention, diagnosis, evaluation, and treatment of CKD and has added "MBD to CKD to be now called as CKD-MBD" [6]. It has been an area of intense interest and as well as controversy [7].

CKD-MBD, the new terminology used for renal osteodystrophy and renal bone disease, is essentially a broad clinical disorder which is a manifestation of any one or combination of various abnormalities in clinical setting as follows:

- Abnormalities of biochemical parameters (calcium, phosphorus, parathyroid hormone).
- Intact parathormone (iPTH) and Vitamin D metabolism.
- Abnormalities in bone turnover, mineralization, volume, linear growth, or strength.
- Calcification of the vasculature or other soft tissues [5].

These are closely inter-related and together make major contribution to morbidity and mortality of patients with CKD receiving dialysis [8]. The KDIGO [5] for bone metabolism and disease in CKD (USA) recommend that, in stage 5 CKD, the target levels for calcium (Ca) (corrected for serum albumin), phosphorus (P), calcium × phosphorus (Ca × P) product, and PTH levels should be maintained at 8.5-10.5 mg/dl, 2.5-4.5 mg/dl, <55 mg^2/dl^2, and 100-300 pg/ml, respectively.

Many people who have severe CKD will eventually develop kidney failure and will require dialysis. Despite remarkable advances in the technical ability to provide maintenance dialysis, the mortality rate of patients on long-term dialysis has remained unacceptably high and not significantly improved over the past decade. The control of PTH, phosphorus, and calcium metabolism is one of the objectives in an adequate dialysis protocol.

At present, there is more stress on calcium, phosphorus, and other minerals for regular monitoring in dialysis patients, but the aim of the treatment must be control of phosphate retention, maintaining serum calcium concentration within the normal range (standard), with avoidance of hypercalcemia and prevention of excess PTH secretion. PTH levels should be evaluated regularly in hemodialysis patients, and awareness regarding PTH abnormalities should be there among the treating physicians also.

Furthermore, estimation of PTH was used as the main biochemical indicator along with calcium and phosphorus as compared to previous studies which only stress upon mineral (Ca and P) derangements in patients with CKD.

Therefore, we conducted this study to describe alterations in PTH, calcium, and phosphorous homeostasis in patients with CKD on hemodialysis in our center. Our study also aimed to find an association between hormonal and biochemical abnormalities in patients with CKD, who have been on hemodialysis for ≥5 months in the dialysis unit at Gian Sagar hospital, and comparing the results obtained with that recommended by the KDIGO guidelines.

CKD-MBD still remains a major problem; hence, improving the ways of detection is the key to tackling this issue, so this study might help in devising strategies in improving the clinical outcome of patients with CKD.

METHODS

Source of data

This study was a hospital-based, cross-sectional, observational study.

Research setting and participants

The study was conducted in the Department of Physiology of Gian Sagar Medical College and Hospital, in close collaboration with Dialysis Unit-Department of Medicine and Department of Biochemistry. The study was based on data collected on patients coming to dialysis unit of Department of Medicine, Gian Sagar Medical Hospital, Ramnagar, Patiala.

All the CKD patients on maintenance hemodialysis coming to Dialysis Unit of Department of Medicine of Gian Sagar Medical College and Hospital, Ramnagar (Patiala), over a period of 3 years from December, 2012, to December, 2015 (total enrolled patients - 330), were enrolled in this study. Each patient was considered only once for the study. Non-random sampling method was used in this study.

Ethical approval and informed consent

This study was approved by the Ethical Committee of Gian Sagar Medical College and Hospital. Informed consent was obtained from all the patients before the initiation the study. All the patients were duly informed about the purpose of asking questions as per pro forma attached with consent form, details of blood sample collection, risk factors, and precautions.

Inclusion criteria

All the patients (>18 years) on maintenance hemodialysis coming to dialysis unit of Department of Medicine of Gian Sagar Medical College and Hospital, Ramnagar (Patiala), during the study period of 3 years (December, 2012 - December, 2015), were enrolled in the study. Each patient was considered only once for the study. Both men and women were included in this study.

Method of collection of data

The study was started after obtaining proper approval from the Institutional Research Ethics Committee. Patients were selected based on the inclusion and exclusion criteria. Approach to a patient began by taking informed consent, after proper medical examination. The detailed medical history as well as baseline demographic data such as age (yrs.), gender (M/F), weight (kg), height (cm), body mass index, religion, educational status (literate/illiterate), and environmental status (urban/rural) were recorded as per pro forma. Dietary history of the patients was obtained. Detailed history regarding the intake of phosphate binders, both calcium based and non-calcium based, and Vitamin D analogs was taken.

Blood sample was collected for laboratory investigations (such as serum calcium, serum phosphorous, serum alkaline phosphatase, serum iPTH, serum albumin, blood urea, serum creatinine, and hemoglobin). Blood samples were collected with aseptic precautions after obtaining informed consent from the patients. 5 ml of blood was taken from the antecubital vein of the patients under full aseptic conditions. Upon clotting, serum was separated out for the estimation of biochemical parameters. All the laboratory parameters were done on fully automated equipment, standardized in Gian Sagar Hospital.

Normal values of serum calcium (corrected for albumin) and phosphate were defined as 8.5-10.5 mg/dl and 2.5-4.5 mg/dl, respectively. PTH level >300 pg/ml (2 times the upper limit of the assay) was labeled as hyperparathyroidism (high bone turnover). The detailed data related to CKD-MBD were collected in pro forma and fed into custom-built database. Observations and results were compiled at the end of the study. The control of CKD-MBD was assessed in the backdrop of the KDIGO guidelines.

Statistical analysis

All the demographic and laboratory parameters were analyzed using Statistical Package for Social Sciences package version 17.0. Descriptive statistics such as range, mean, and standard deviation were used to describe continuous variables while numbers and percentages were used to present discreet variables. Chi-square test and analysis of variance (ANOVA) with post-hoc tukey's HSD tests were used to test the association between clinical and laboratory parameters, and Pearson's coefficient of correlation was used to assess the inter-relationship between various examined laboratory markers. The result was statistically significant when $p<0.05$.

RESULTS AND DISCUSSION

CKD-related MBD (CKD-MBD) is a worldwide challenge in hemodialysis patients [9]. CKD is characterized by the decline of kidney function within long period [10]. The decline of kidney function causes a range of complications including metabolic abnormalities, endocrine complications, and increased risk of cardiovascular diseases. These complications, if not managed appropriately, may implicate on the prolonged length of stay in the hospital and increased mortality rate [7,11].

The mineral and endocrine functions disrupted in CKD are critically important in the regulation of both initial bone formation during growth (bone modeling) and bone structure and function during adulthood (bone remodeling) [5]. As a result, bone abnormalities are found almost universally in patients with progressive loss of renal function in CKD which necessitates the eventual use of dialysis (stage 5D) [5].

The present study was a hospital-based, cross-sectional, observational study. The study population of 330 patients comprised adults, mainly illiterate (54.5%) predominantly belonging to the rural (66.4%) strata and Sikh community (62.7%), with a mean age of 52.67±15.05 years (range: 25-98 years) (Table 1), of whom most patients were taking vegetarian diet (58.2%).

In the present study, out of 330 patients, 159 patients were between the age group of 41-60 years, which suggests that incidence of CKD

increases with advanced age may be because of the ignorance regarding regular medical checkup (Table 2).

The mean age of our study population was almost similar to other Indian studies like that of Agarwal *et al.* [12] (42±13 years) and Prasad and Murthy [13] (49.3 years with range 17-80) and Valson *et al.* [14] (46.6±13.4 years).

However, higher mean age was reported in a Western study (74±15 years) by Craver *et al.* [15] and by Szeto *et al.* [16] (60±12 years).

Male preponderance in the present study (65.2%) (Table 1) was observed with the study having 215 males and 115 females. One of the main reasons for these differences in gender of patients may be that, in India, more male patients visit hospitals than females because of female health negligence mainly in rural areas due to male priority or may be because of male dominance and due to financial constrains for treatment.

Males made up the majority of the CKD patients with 70.6% similar to what was reported in other studies [17-19].

Another study from Pakistan by Hussain *et al.* [20] also found male predominance with the mean age of the patients in their study as 58±16.6 years.

The predominant probable causes of CKD in our patients were as follows:

- Diabetes mellitus in 51.8%
- Drug-induced in 27.6%
- Hypertension in 14.5%
- Obstructive uropathy in 6.1% (Table 3).

Similar observations was made by Prasad and Murthy [13], who reported the cause of CKD as diabetic nephropathy in 38% of patients, hypertensive nephropathy in 28% of patients, chronic glomerulonephritis in 24% of patients, and obstructive uropathy in 6% of the patients.

Vhora *et al*. [21] observed that out of 60 CKD patients, 55% had hypertension and diabetic nephropathy together.

In a 10-year study of 368 patients with CKD in Nigeria, the etiology of renal failure was undetermined in 62%, and of the remaining patients whose etiology was ascertained, hypertension accounted for 61%, diabetes mellitus for 11%, and chronic glomerulonephritis for 5.9% [22].

In our study, drug-induced was the second common cause of renal failure in CKD patients (Table 3), ascertained with the help of medical history and patient's records, which may be because of injudicious use of non-steroidal anti-inflammatory drugs in the treatment of any kind of pain, leading to side effects on renal functioning.

In the clinical profile of the present study, the most commonly reported symptoms were pruritus (6.1%), pedal edema (40.6%), and proximal muscle weakness (13.9%). In addition, bone pain and bone fracture were found in mild proportion, i.e., 19.1% and 4.8%, respectively (Table 3). Radiological findings revealed features suggestive of bone fracture on the basis of recent medical history/records. In the present study, majority of clinical symptoms related to CKD-MBD were found absent in CKD patients undergoing dialysis, reinforcing the fact that CKD-MBD is a clinically silent disease.

Our results were in accordance with the observations of Valson *et al*. [14] who reported similar clinical symptoms such as bone pain (33.5%), proximal muscle weakness (26.2%), and pruritus (25.5%), but in high percentage as compared to our findings.

The results of our study also revealed that bone pain was significantly associated with iPTH levels (both low and high level) (χ^2=6.631 and p=0.036) (Table 4). It was observed that the percentage of CKD patients on hemodialysis of bone fracture was slightly more in hyperparathyroidism group (46.7%) as compared to hypoparathyroidism and normal parathyroidism (40.05 and 13.3%, respectively). This difference was

Table 1: Mean age versus gender

Gender	Number of dialysis patients (%)	Age (years)		
		Minimum	Maximum	Mean±SD
Female	115 (34.8)	25	85	52.15±14.48
Male	215 (65.2)	25	98	52.95±15.36
Total	330 (100.00)	25	98	52.67±15.05

VS: Versus, SD: Standard deviation

Table 2: Age-wise distribution

Age (years)	Number of dialysis patients (%)
20-40	73 (22.10)
41-60	159 (48.20)
61-80	87 (26.40)
>80	11 (3.30)
Total	330 (100.00)

Table 3: Clinical characteristics of the study patients

Clinical characteristics of patients	Number of dialysis patients n=330 (%)
Probable cause of CKD	
Diabetes mellitus	171 (51.8)
Drug-induced	91 (27.6)
Hypertension	48 (14.5)
Obstruction	20 (6.1)
Bone pain	
Absent	267 (80.9)
Present	63 (19.1)
Bone fracture	
Absent	314 (95.2)
Present	16 (4.8)
Pruritus	
Absent	310 (93.9)
Present	20 (6.1)
Relevant bone abnormalities	
Absent	328 (99.4)
Present	2 (0.6)
Intake of phosphate binders	
Not used	250 (75.8)
Used	80 (24.2)
Conscious orientation to TPP	
Absent	0 (0)
Present	330 (100.0)
Pallor	
Absent	309 (93.6)
Present	21 (6.4)
Clubbing/pseudo clubbing	
Absent	320 (97.0)
Present	10 (3.0)
Pedal edema	
Absent	196 (59.4)
Present	134 (40.6)
Bony tenderness/deformity	
Absent	322 (97.6)
Present	8 (2.4)
Proximal muscle weakness	
Absent	284 (86.1)
Present	46 (13.9)
Eyes (redness, watery, or irritation)	
Absent	286 (86.7)
Present	44 (13.3)

CKD: Chronic kidney disease, %: Percentage, n=330

not statistically significant (χ^2=1.372, P=0.503) (Table 5) although the incidence found for fracture was very low, only 5% (Table 3). PTH was also significantly associated with other physical symptoms such as proximal muscle weakness and eye problem (P=0.016 and P=0.001, respectively) (Tables 6 and 7). p<0.05 (if P is less than 0.05) was considered statistically significant.

iPTH level is directly linked with phosphorous and total alkaline phosphatase (tALP) laboratory parameters with significant p value (P=0.032 and P=0.035, respectively) as was proved with the ANOVA test of three intergroup comparisons of iPTH level that hyperparathyroidism and normal parathyroidism were more significantly associated with tALP level (P=0.002 and P=0.003, respectively) as compared to hypo-parathyroidism (P=0.987) (Table 8). Positive relationship was also found between iPTH and tALP levels (r=0.226, P=0.000) with Pearson's correlation coefficient testing (Table 9).

Alterations in biochemical profile: MBD among CKD patients undergoing hemodialysis

In the biochemical profile, the target levels for corrected calcium (corrected for serum albumin), phosphorus, calcium × phosphorus (Ca × P) product, and iPTH levels should be maintained as recommended in the KDIGO guidelines for preventive MBD in CKD patients.

However, in the present study, it was observed that alterations in biochemical profile were common in hemodialysis patients. Only a small proportion of patients adhered to the targets. Proportion of the CKD patients who were out of the target range was as follows:

- 73.8% for iPTH (target range: 100-300 pg/ml).
- 55.8% for corrected serum calcium (target range: 8.5-10.5 mg/dl).
- 63.0% for phosphorus (target range: 2.5-4.5 mg/dl) and
- 50.0% patients for tALP (target range: 60-170 IU/L) were out of the target range (Fig. 1).

These results in our study indicated that most of the hemodialysis patients reported levels for biochemical profile which were outside the recommended range as per the KDIGO guidelines.

Table 4: Association of PTH with bone pain

Presence of bone pain	iPTH level			Total
	<100	100-300	>300	
Absent	64 (29.9)	60 (28.0)	90 (42.1)	214
Present	24 (49.0)	9 (18.4)	16 (32.7)	49
Total	88 (33.5)	69 (26.2)	106 (40.3)	263

χ^2=6.631, df(degree of freedom)=2, P(Probability)=0.036, significant, iPTH: Intact parathormone

Table 5: Association of PTH with bone fracture

Presence of bone fracture	PTH level			Total
	<100	100-300	>300	
Absent	82 (33.1)	67 (27.0)	99 (39.9)	248
Present	6 (40.0)	2 (13.3)	7 (46.7)	15
Total	88 (33.5)	69 (26.2)	106 (40.3)	263

iPTH: Intact Parathormone, χ^2=1.372, df=2, p=0.503, not significant

Table 6: Association of PTH with proximal muscle weakness

Proximal muscle weakness	PTH level			Total
	<100	100-300	>300	
Absent	69 (30.3)	64 (28.1)	95 (41.7)	228
Present	19 (54.3)	5 (14.3)	11 (31.4)	35
Total	88 (33.5)	69 (26.2)	106 (40.3)	263

iPTH: Intact parathormone, χ^2=8.220, df=2, p=0.016, significant

The percentage of patients, who were in the recommended range, was less than that obtained in other studies [22,23].

The inability of the CKD patients to achieve the above-said target range may be explained by many factors.

Implementing different modalities for regulation of calcium, phosphorus, and iPTH levels in Indian population is as follows:

- Patients were using calcium-based phosphate binders only (such as calcium acetate or calcium carbonate) in our center as compared to non-calcium-based phosphate binders (such as sevelamer hydrochloride, or lanthanum carbonate, which is widely used in the USA and Europe for the treatment of hyperphosphatemia in patients with CKD [24,25].
- Moreover, in our study, only 24.2% of the CKD patients were using these binders (calcium-based phosphate binders).
- Though calcimimetic agent (cinacalcet HCl) [26] along with Vitamin D analogs may be used for controlling PTH secretion with minimal hypercalcemia and hyperphosphatemia [27], it was not advocated in our center.

In addition to this, the results of these tests are also influenced by food intake, adherence to and the timing of drug intake and dietary modifications, and differences in assay methods. This has also been documented in updated KDIGO guidelines 2016 [28].

In the current study, the laboratory profile in CKD patients was as follows:

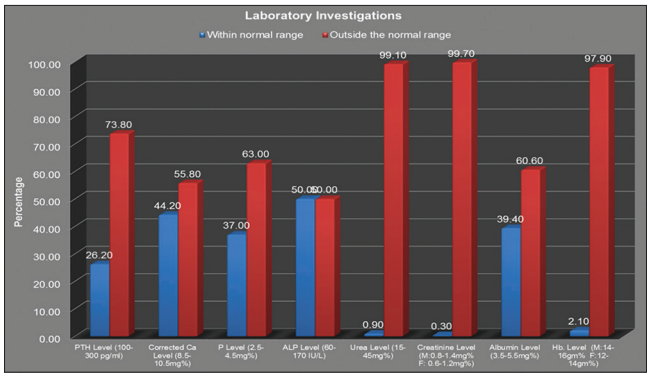

Fig. 1: Biochemical profile of chronic kidney disease patients achieving Kidney Disease Improving Global Outcomes target levels

Table 7: Association of PTH with eye problem

Eye problem	PTH level			Total
	<100	100-300	>300	
Absent	67 (29.4)	65 (28.5)	96 (42.1)	228
Present	21 (60.0)	4 (11.4)	10 (28.6)	35
Total	88 (33.5)	69 (26.2)	106 (40.3)	263

iPTH: Intact Parathormone, χ^2=13.252, df=2, p=0.001, significant, The analyzed iPTH data (total number of dialysis patients=263) were based on iPTH measurements, iPTH: Intact parathormone

Table 8: Association of PTH with tALP

PTH level	N	tALP mean±SD	ANOVA#	Comparison	p#
A. <100	88	200.83±130.06	F=8.032;	A versus B	0.987^{NS}
B. 100-300	69	191.79±141.38	p=0.000;	A versus C	0.002^{NS}
C. >300	106	377.70±540.86	NS	B versus C	0.003^{NS}

tALP: Total alkaline phosphatase, SD: Standard deviation, ANOVA: Analysis of variance. N: Number of dialysis patients #One-way ANOVA with post-hoc Tukey's HSD; NS: p>0.05, not significant

Table 9: Correlation

	Age	iPTH	Corrected _calcium	Serum phosphorus level	t-ALP
Age					
r =	1	0.041	0.072	0.037	0.049
p =		0.506	0.191	0.499	0.375
iPTH					
r =	0.041	1	0.120	−0.060	0.226**
p =	0.506		0.053^{NS}	0.335^{NS}	0.000
Corrected calcium					
r =	0.072	0.120	1	−0.149**	−0.034
p =	0.191	0.053^{NS}		0.007*	0.535
Serum phosphorus level					
r =	0.037	−0.060	−0.149**	1	−0.040
p =	0.499	0.335^{NS}	0.007*		0.473
t-ALP					
r =	0.049	0.226**	−0.034	−0.040	1
p =	0.375	0.000	0.535	0.473	

**Correlation is significant at the 0.01 level (two tailed). *correlation is significant at the 0.05 level (two tailed) r: Pearson's correlation coefficient, P=Probability; NS(Not Significant) when P≥0.05,*(Significant)when P <0.05., *p<0.05, significant

- Hyperparathyroidism in 40.3%
- Hyperphosphatemia in 62.1%
- Hypocalcaemia in 50.6%
- Elevated tALP level in 49.4% of the patients (Table 10).

Statistically, a negative but significant correlation exists between corrected calcium and phosphorus (P=0.05 level of confidence interval). Calcium and phosphate show weak insignificant relationships with iPTH (Table 9).

The majority of hemodialysis patients had highly elevated serum PTH and phosphorus levels that suggest that some of the patients had severe secondary hyperparathyroidism.

Considering the fact, it was reported that impaired phosphate excretion, with the resulting hyperphosphatemia, is one of the earliest consequences of chronic renal failure. Hyperphosphatemia, in turn, plays an important role in the development of secondary hyperparathyroidism [29,30]. Moreover, phosphate retention leads to a decrease in serum-free calcium levels (hypocalcaemia), which in turn stimulates PTH secretion [31,32].

Similar to the present study, Okoye *et al.* [8] concluded that the prevalence of various mineral bone disease abnormalities was 70% of hyper-phosphatemia and 85% of hyper-parathyroidism among the patients. Vhora *et al.* [21] reported hyperparathyroidism in 95% of CKD patients.

However, in these studies, the prevalence of hyperparathyroidism was much high as compared to our study.

Agarwal *et al.* [33] described hypocalcemia in 49.6% and hyperphosphatemia in 41.8% of CKD-5 patients and hyperparathyroidism in 39.4% of patients with CKD stage 5. He defined hyperparathyroidism (iPTH >300 pg/mL in stage 5 CKD) by using the K/DOQI guidelines 2002 [34].

Short of a bone biopsy, biochemical tests such as tALP or intact PTH can be used to evaluate bone disease.

Controlling PTH levels prevents damage to bones. In general, overactive parathyroid glands are controllable with a change in diet. Almost all foods contain phosphorus, but it is especially high in milk, cheese, peas, nuts, and peanut butter. Therefore, reducing dietary intake of phosphorus is one of the most important steps in preventing bone disease.

Table 10: Laboratory parameters in CKD patients undergoing hemodialysis

Laboratory characteristics of patients	Groups	Number of dialysis patients n=330 (%)
Serum iPTH level (pg/ml)	<100	88 (33.5)
	100-300	69 (26.2)
	More than 300	106 (40.3)
Albumin-corrected serum Ca*(mg%)	<8.5	167 (50.6)
	8.5-10.5	146 (44.2)
	More than 10.5	17 (5.2)
Serum phosphorus level (mg%)	<2.5	3 (0.9)
	2.5-4.5	122 (37.0)
	More than 4.5	205 (62.1)
Serum alkaline phosphatase level (IU/L)	<60	2 (0.6)
	60-170	165 (50.0)
	More than 170	163 (49.4)
Blood urea level (mg%)	<15	3 (0.9)
	15-45	3 (0.9)
	More than 45	324 (98.2)
Serum creatinine level (mg%)	0.6-1.4	1 (0.3)
	More than 1.4	329 (99.7)
Serum albumin level (g%)	<3.5	200 (60.6)
	3.5-5.5	130 (39.4)
Hemoglobin level (g%)	<12	323 (97.90)
	12-16	7 (2.1)

*Corrected calcium is calculated by the following formula:- {(4-patient albumin)*0.8+patient calcium level}:[4=normal albumin range]

CONCLUSION

we showed in the present study that disorders of mineral metabolism are common in hemodialysis patients and that only a small proportion adheres to the targets as advised in the KDIGO guidelines for bone metabolism and disease in CKD. We demonstrated that these disorders are associated with important negative clinical outcomes, such as increased all-cause lack survival, more muscle and bone problems. Our findings, therefore, support a strict control of mineral metabolism in dialysis patients. We would like to emphasize the importance of educating and stimulating dialysis patients to achieve optimal adherence to the treatment regimens and dietary recommendations. A renal dietician can help develop a dietary plan to control phosphorus levels in the blood. Along with it, the use of phosphate-binding agents is a must. The aim of the treatment must be control of phosphate retention, maintaining serum calcium concentration within the normal range (standard), with avoidance of hypercalcemic episodes and prevention of excess PTH secretion.

Further research and progress in this area are required to establish a more rational approach with a view toward improving patient outcomes.

Recommendations

1. Awareness of CKD and KDIGO guidelines must be raised among renal physicians, the staff dealing with hemodialysis patients, and the patients.
2. Controlling PTH levels prevents damage to bones. Usually, overactive parathyroid glands are controllable with a change in diet, dialysis treatment, or medication.
3. Studies should be done to look into the role of serum levels of Vitamin D in CKD-MBD among our dialysis unit.

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