International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 17s, 2025
https://theaspd.com/index.php

Impact Of low phosphorus containing formula On Parathyroid Hormone Level In Children On Dialysis

Mohammed Ibrahim Ahmed Eliwa[1]*, Doaa Youssef Mohamed[2], Ahmed Elsayed Wageeh[3]
[1] Pediatric Department, Faculty of Medicine, Zagazig Fever Hospital, Egypt
[2] Pediatric Department, Faculty of Medicine, Zagazig University, Egypt
[3] Pediatric Department, Faculty of Medicine, Suez University, Egypt
***Corresponding author:** Mohammed Ibrahim Ahmed Eliwa
Email: drmohamed_2@yahoo.com,

Abstract:
Background: *Children with end-stage renal disease (ESRD) undergoing dialysis often suffer from disturbances in mineral metabolism, particularly involving calcium, phosphorus, and parathyroid hormone (PTH). One of the most common complications is secondary hyperparathyroidism (SHPT), a condition driven by phosphate retention, hypocalcemia, and reduced calcitriol synthesis, leading to increased PTH secretion. Low-phosphorus containing formulas have emerged as an important nutritional strategy to reduce serum phosphate levels and, consequently, PTH levels. So we aimed to assess the effect of low phosphorus containing formula on parathyroid hormone level in children on dialysis.*
Methods: *This one- arm clinical trial was conducted at Pediatrics department, Dialysis unit, Zagazig University Hospitals, Sharqia governorate, Egypt on Children on dialysis. Parathyroid hormone level assessment was done before and after taking of low phosphorus containing formula.*
Results: *There was a significant decrease in parathyroid hormone (PTH) levels following low phosphorus-containing formula ($p<0.001$) with a 95% confidence interval for the difference ranging from 80.14 to 199.59 pg/mL.*
Conclusion: *The administration of a low phosphorus-containing formula in children on dialysis resulted in a statistically significant reduction in serum parathyroid hormone (PTH) and phosphorus levels, indicating its effectiveness in managing secondary hyperparathyroidism and hyperphosphatemia in this population.*
Keywords: *Chronic Kidney Disease (CKD), Pediatric Dialysis, Secondary Hyperparathyroidism (SHPT), Low-Phosphorus Formula.*

INTRODUCTION:

Chronic kidney disease (CKD) in children, particularly those receiving dialysis, leads to a 10-fold increase in cardiovascular morbidity and death due to a combination of conventional and uremia-related risk factors. Chronic kidney disease-mineral and bone disorders (CKDMBD) is a multi-systemic condition characterized by abnormalities in calcium, phosphorus phosphate, vitamin D metabolism, bone histology, and artery calcification **(1).**

CKD-MBD negatively impacts children's physical and mental health, lowers life expectancy, and necessitates effective management of secondary hyperparathyroidism and parathyroid hormone (PTH) levels. Data on PTH targets in kids with CKD2-4 is limited, as PTH, phosphate, and FGF23 levels rise well before end-stage renal disease, likely due to hyperphosphatemia and dysregulation in CKD **(2).**

Hyperphosphatemia, hypocalcemia, and reduced vitamin D levels in chronic kidney disease (CKD) contribute to complex mineral and bone disorders linked to CKD-MBD. Many authors aimed to improve the management of children on dialysis by evaluating the impact of low phosphorus containing formula on parathyroid hormone levels. Disrupted PTH levels have been linked to cardiovascular comorbidities, anemia, left ventricular hypertrophy, poor longitudinal growth, and even mortality in pediatric dialysis patients **(3).**

Therefore, it is evident that excessive PTH levels are harmful, but low PTH levels have also been linked to vascular calcifications and growth impairment due to the underlying adynamic bone. Therefore, in order to improve the care of these patients, it became necessary to develop an understanding of how low phosphorus containing formula affects the level of parathyroid hormone in children on dialysis **(4, 5).**

So we aimed to assess the effect of low phosphorus containing formula on parathyroid hormone level in children on dialysis.

Patients and Methods

This one- arm clinical trial was conducted at Pediatrics department, Dialysis unit, Zagazig University Hospitals, Sharqia governorate, Egypt on Children on dialysis.

Inclusion criteria:

- Age from 2 to 18 years.
- Both sexes.
- Children on dialysis for at least one year .
- Hyperphosphatemia with hyperparathyroidism.

Exclusion criteria:

- Hypoparathyroidism.
- Rhabdomyolysis.
- Severe infection (sepsis).
- Physical trauma (crush injury). **(6).**

Sampling/Experiment design (describing study groups): one study group consists of 45 cases at least with adding 10-15 % to compensate for possible drop out then total sample size will be 52 cases.

Sample size estimation: Using PASS 15 program for sample size calculation **(7)**, setting power at 90% and alpha error at 0.05, it is estimated that sample size of 45 patients will be needed to detect a statistically significant difference in PTH level before and after intervention, corresponding to an expected medium effect size (Cohen's d = 0.5) **(8)** ,using a two-sided paired t-test. **(9)**. For expected 10-15% drop out rate , sample was increased to 52 patients.

Methods:

All children were subjected to:

- Complete history taking including personal, complaint, present, past, perinatal, developmental, dietetic, vaccination, drug and family history.
- Full clinical either general or local examination for all systems by inspection, palpation, percussion, and auscultation.
- Routine laboratory investigations. (CBC - Serum Calcium and phosphorous - Kidney function tests- Serum electrolytes: Na, K - Random blood glucose).
- Parathyroid hormone level assessment before and after taking of low phosphorus containing formula such (Renastart formula).

Outcomes assessment: We expect that there is an impact of low phosphorus containing formula on parathyroid hormone level in children on dialysis.

Statistical Analysis

The collected data was tabulated, and statistically analyzed using SPSS program (Statistical Package for Social Sciences) software version 26.0, Microsoft Excel 2019.

Descriptive statistics were done for numerical parametric data as mean±SD (standard deviation) and minimum & maximum of the range and for numerical non parametric data as median and 1^{st}& 3^{rd} inter-quartile range, while they were done for categorical data as number and percentage.

Student t-test: For normally distributed quantitative variables, to compare between two studied groups. **Mann Whitney test:** used for abnormally distributed quantitative variables, to compare between two studied groups. **Paired t-test**; used for comparison between two related samples with normally distributed data. **Wilcoxon Rank test** was used to compare two related samples, matched samples, or to conduct a paired difference test of repeated measurements on a single sample to assess whether their population mean ranks differ with not-normally distributed data.

The level of significance was taken at P value <0.05 is significant, otherwise is non-significant. The p-value is a statistical measure for the probability that the results observed in a study could have occurred by chance.

RESULTS

Table (1): Demographic data among the studied cases.

		Studied patients (N= 52)	
		N	%
Gender	Male	26	50.0%
	Female	26	50.0%
Age (years)	Mean± SD	11.48± 3.35	
	Median (Range)	11.5 (5.5- 18)	
Residence	Rural	26	50.0%
	Urban	26	50.0%

SD: standard deviation, n: number, %: percentage,

This table shows demographic among the studied cases. There was equal gender distribution, comprising 26 males (50.0%) and 26 females (50.0%). The mean age of studied cases was 11.48 ± 3.35 years, with a median age of 11.5 years and range from 5.5 to 18 years. Residence was also evenly distributed, with 26 patients (50.0%) residing in rural areas and 26 (50.0%) from urban areas. **[Table 1].**

Table (2): Causes of CKD among the studied cases.

		Studied patients (N= 52)	
		N	%
Causes of CKD	Congenital anomalies of the kidney and urinary tract (CAKUT)		
	▪ Neurogenic bladder	7	13.5%
	▪ Congenital anomaly of kidney	2	3.8%
	▪ Single /absent kidney	3	5.8%
	▪ Joubert syndrome	1	1.9%
	▪ Atrophied kidney	4	7.7%
	Hereditary nephropathies		
	▪ Nephronophthisis	3	5.8%
	▪ Poly cystic kidney	2	3.8%
	▪ Congenital nephrosis	1	1.9%
	Glomerular diseases:		
	▪ Focal segmental glomerulosclerosis	4	7.7%
	▪ Thrombotic microangiopathy	1	1.9%
	▪ SLE	1	1.9%
	▪ CKD 2ry to lupus nephritis	1	1.9%
	▪ Atypical HUS	2	3.8%
	Nephrotic syndrome	8	15.4%
	Chronic parenchymal disease	2	3.8%
	Unexplained RF	11	21.2%
	Obstructive uropathy	6	11.5%
	Environmental and socioeconomic factors		
	▪ Nephrocalcinosis	1	1.9%

*More than one disease may found in same patient

The most common identified cause of CKD was unexplained renal failure, accounting for 21.2% of cases. Congenital anomalies of the kidney and urinary tract (CAKUT) were collectively the most prevalent defined etiology (32.7%), with neurogenic bladder being the most frequent CAKUT subtype (13.5%). Hereditary

nephropathies such as nephronophthisis and polycystic kidney disease represented 11.5% of cases. Glomerular diseases were identified in 17.3%, including focal segmental glomerulosclerosis and lupus-related nephritis. Nephrotic syndrome was noted in 15.4% of patients. Other contributors included obstructive uropathy (11.5%), chronic parenchymal disease (3.8%), and a smaller proportion attributed to environmental and socioeconomic factors such as nephrocalcinosis (1.9%). **[Table 2].**

Table (3): Demographic data among the studied cases.

		Studied patients (N= 52)
Dialysis period (years)	Mean± SD	4.17± 3.61
	Median (Range)	3 (5 months- 15)

SD: standard deviation, n: number, %: percentage,

This table shows that the mean duration of dialysis was 4.17 ± 3.61 years. The median duration was 3 years, with a range from 5 months to 15 years. **[Table 3].**

Table (4): Comparison of PTH (parathyroid hormone) and kidney functionbefore and after low phosphorus-containing formula among children underwent dialysis.

	Studied patients							Difference (95%CI)	Wilcoxon Signed Rank Test	
	Mean	±SD	Median	IQR		Range			Test value	p- value
PTH (pg/mL)										
Before formula	369.28	±437.69	169.00	71.15	558.0	4.7	1871.0	139.86 (80.14-199.59)	6.275	< 0.001**
After formula	229.42	±338.22	101.70	39.45	280.0	4.1	1691.0			
Creatinine (mg/dL)										
Before formula	7.96	±7.02	6.80	5.68	8.85	2.40	55.00	0.799 (-1.19- 2.78)	0.498	0.619
After formula	7.16	±2.08	7.40	5.90	8.45	2.40	11.90			
BUN (mg/dL)										
Before formula	51.89	±13.42				27.0	89.0	1.99 (-6.67- 2.68)	0.856	0.396
After formula	53.88	±13.98				27.0	82.3			

P value> 0.05 is not significant, *P value< 0.05 is significant, **P value< 0.01 is highly significant.

SD: Standard deviation, IQR: Inter-quartile range

The median Interquartile Range (IQR) PTH level before the formula was 169 pg/mL (71.15- 558) pg/mL. After the formula, the median (IQR) was 101.7 pg/mL (39.45-280) pg/mL. There was a significant decrease in parathyroid hormone (PTH) levels following low phosphorus-containing formula (p<0.001) with a 95% confidence interval for the difference ranging from 80.14 to 199.59 pg/mL. The mean BUN level before the low phosphorus-containing formula was 51.89±13.42 mg/dL, compared to 53.88 ± 13.98 mg/dL after the

formula. Blood urea nitrogen (BUN) levels in the studied patients showed no statistically significant change after the formula (p>0.05). The median (IQR) creatinine level before the formula was 6.8 (5.68-8.85) mg/dL. After the formula, the median (IQR) was 7.4 (5.9-8.45) mg/dL. Creatinine level measured before and after formula administration showed no statistically significant change among the studied patients (p>0.05) **[Table 4].**

Table (5): Comparison of calcium and phosphorus level before and after low phosphorus-containing formula among children underwent dialysis.

		Studied patients				Difference (95%CI)	Paired T Test	
		Mean	±SD	Range			Test value	p- value
Ca^{+2} (mg/dL)	Before formula	9.23	±1.14	4.80	12.0	0.31 (-0.64- 0.01)	1.940	0.058
	After formula	9.54	±1.16	6.20	12.4			
Phosphorus (mg/dL)	Before formula	6.97	±1.79	3.40	13.07	1.26 (0.99- 1.53)	6.153	<0.001**
	After formula	5.72	±1.62	2.60	11.50			

P value> 0.05 is not significant, *P value< 0.05 is significant, **P value< 0.01 is highly significant.
SD: Standard deviation

The mean Ca^{+2} level changed from 9.23±1.14 mg/dL before the formula to 9.54±1.16 mg/dL after. Serum calcium (Ca^{+2}) levels measured before and after formula administration showed no statistically significant change among the studied patients (p>0.05). The median (IQR) phosphorus level before the formula was 6.95 (5.80- 7.68) mg/dL. After the formula, the median (IQR) was 5.70 (4.70- 6.35) mg/dL. There was a significant decline in phosphorus level following low phosphorus-containing formula (p<0.001) with a difference of 1.26 mg/dL & 95% confidence interval for the difference ranging from 0.99 to 1.53 **[Table 5].**

Table (6): Comparison of Hemoglobin and albumin level before and after low phosphorus-containing formula among children underwent dialysis.

	Studied patients							Difference (95%CI)	Wilcoxon Signed Rank Test	
	Mean	±SD	Median	IQR		Range			Test value	p- value
Hemoglobin (g/dL)										
Before formula	9.77	±1.00	9.60	9.05	10.4	7.8	13.3	0.054 (-0.43- 0.32)	0.239	0.811
After formula	9.82	±1.03	9.70	9.10	10.4	8.3	13.4			
Albumin (g/dL)										
Before formula	4.21	±0.61	4.13	3.90	4.5	3.2	7.4	0.044 (-0.13- 0.22)	0.260	0.795
After formula	4.17	±0.38	4.25	3.85	4.5	3.5	4.9			

P value> 0.05 is not significant, *P value< 0.05 is significant, **P value< 0.01 is highly significant.
SD: Standard deviation, IQR: Inter-quartile range

The median (IQR) albumin level before the formula was 4.13 (3.90- 4.5) g/dL. After the formula, the median (IQR) was 4.25 (3.85-4.5) g/dL. Serum albumin level measured before and after low phosphorus-containing formula administration showed no statistically significant change among the studied patients (p>0.05). The median hemoglobin level increased slightly from 9.60 g/dL before the formula to 9.70 g/dL after. Serum hemoglobin level measured before and after low phosphorus-containing formula administration showed no statistically significant change among the studied patients (p>0.05) **[Table 6].**

Table (7): Relation between gender with different biochemical data.

		Gender						Test value	P-value
		Male (N= 26)			Female (N= 26)				
		Mean	SD	Median	Mean	SD	Median		
PTH (pg/mL)	Before formula	285.53	±421.1	123.0	453.03	445.96	369.0	Z_{MWU}= 2.251	**0.024***
	After formula	185.14	±317.1	61.0	273.70	358.80	141.5	Z_{MWU}=1.922	0.055
BUN (mg/dL)	Before formula	51.22	±15.20	49.0	52.56	11.63	51.75	T= 0.357	0.723
	After formula	54.35	±11.71	54.5	53.42	16.16	49.15	T= 0.240	0.812
Creatinine (mg/dL)	Before formula	8.55	±9.69	6.6	7.36	2.41	6.80	Z_{MWU}=0.769	0.442
	After formula	7.41	±2.10	7.55	6.91	2.07	7.05	T= 0.861	0.393
Ca^{+2} (mg/dL)	Before formula	9.32	±1.28	9.36	9.14	0.99	9.10	T= 0.569	0.572
	After formula	9.48	±1.37	9.45	9.60	0.94	9.65	T= 0.376	0.709
Phosphorus (mg/dL)	Before formula	7.01	±1.46	6.85	6.94	2.09	7.0	Z_{MWU}=0.156	0.876
	After formula	5.75	±1.45	5.94	5.69	1.79	5.55	Z_{MWU}=0.439	0.660
Albumin (g/dL)	Before formula	4.17	±0.41	4.10	4.25	0.77	4.25	Z_{MWU}=0.229	0.819
	After formula	4.14	±0.38	4.20	4.19	0.39	4.30	Z_{MWU}=0.524	0.600
Hemoglobin (g/dL)	Before formula	9.82	±1.01	9.85	9.71	1.01	9.50	Z_{MWU}=0.770	0.441
	After formula	9.92	±1.26	9.65	9.72	0.75	9.80	Z_{MWU}=0.055	0.956

p>0.05 is non-significant, *p≤0.05 is significant, **p≤0.01 is highly significant,
SD: standard deviation, T: Student T Test, ZMWU: Z value of Mann Whitney U test, PTH: Parathyroid hormone

Among the 52 studied patients (26 males and 26 females), there were no significant gender-based differences in most laboratory parameters, including BUN, creatinine, calcium, phosphorus, albumin, and hemoglobin levels, either before or after the introduction of the formula as well as PTH after formula. However, a significant

difference was observed in parathyroid hormone (PTH) levels before the formula (p=0.024), with females showing higher median PTH levels compared to males (369 vs. 123 pg/mL). **[Table 7].**

Table (8): Relation between age with different biochemical data.

		Age/ years	
		r	**P-value**
PTH (pg/mL)	**Before formula**	0.068	0.632
	After formula	-0.001	0.995
BUN (mg/dL)	**Before formula**	-0.145	0.304
	After formula	-0.281	**0.044***
Creatinine (mg/dL)	**Before formula**	0.090	0.526
	After formula	0.220	0.117
Ca^{+2} (mg/dL)	**Before formula**	0.058	0.681
	After formula	0.143	0.312
Phosphorus (mg/dL)	**Before formula**	-0.134	0.342
	After formula	-0.090	0.526
Albumin (g/dL)	**Before formula**	0.036	0.801
	After formula	0.211	0.133
Hemoglobin (g/dL)	**Before formula**	-0.097	0.492
	After formula	-0.053	0.710
Dialysis periods (years)		0.443	**0.001****

p>0.05 is non-significant, *p≤0.05 is significant, **p≤0.01 is highly significant,
r: Pearson correlation coefficient,

The correlation analysis between patients' age and various biochemical parameters revealed that most markers, including PTH, creatinine, calcium, phosphorus, albumin, and hemoglobin levels, showed no significant association with age either before or after the introduction of the formula ($p > 0.05$). On the other hand, BUN level after the formula showed a statistically significant negative correlation with age ($r = -0.281$, $p = 0.044$), suggesting that older patients had lower BUN levels post-intervention. Also, there was a strong positive correlation between age and dialysis duration ($r = 0.443$, $p = 0.001$), **[Table 8, Figure 1].**

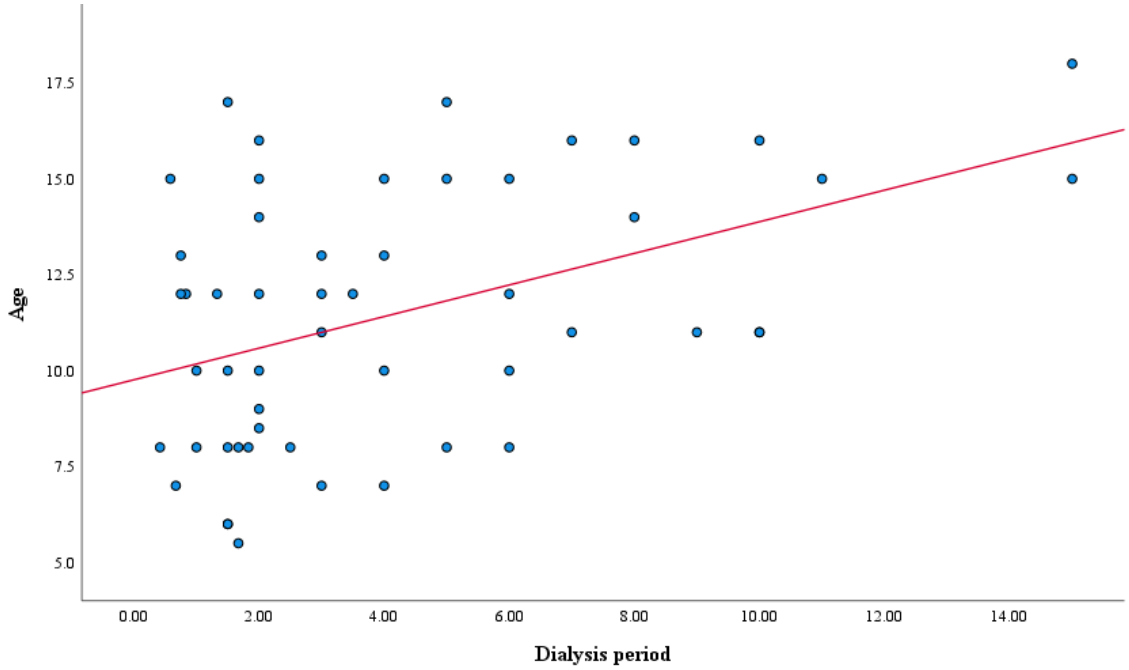

Figure (1): Scatter-plot showing significant positive correlation between age and dialysis period.

Discussion

Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD), defined by KDIGO in 2006, is a systemic condition affecting mineral metabolism due to impaired kidney function. It disrupts the kidney–bone–vascular axis, leading to imbalances in calcium, phosphorus, parathyroid hormone (PTH), and vitamin D. These imbalances contribute to bone abnormalities and soft tissue and vascular calcifications. Hyperphosphatemia is a key factor in the development of secondary hyperparathyroidism, renal osteodystrophy, and cardiovascular complications, significantly increasing mortality risk in CKD patients, particularly those on dialysis. Management includes dietary phosphorus restriction, phosphate binders, and dialysis, though dietary control remains most effective. Both high and low PTH levels are harmful—excess PTH causes bone disease, while low PTH is linked to vascular calcification and growth issues. Thus, understanding the impact of low-phosphorus formulas on PTH levels is essential in managing children on dialysis.

In our study, the demographic distribution was balanced with an equal number of males and females (26 each, 50.0%) and an evenly split residential background (50.0% rural and 50.0% urban). The mean age was 11.48 ± 3.35 years, ranging from 5.5 to 18 years. In contrast, the study by **Sharaf et al. (10),** which was a prospective case-control study involving 30 children with end-stage renal disease (ESRD) on regular hemodialysis and 20 age- and sex-matched healthy controls, reported a higher prevalence of CKD in males (60%) compared to females (40%) **(10)**.

However, similar to our findings, their study found no statistically significant difference in age or sex between cases and controls. The slight variation in gender distribution in Sharaf's study may reflect a smaller sample size or differing regional patterns of CKD prevalence, while our study's equal distribution strengthens the representativeness and potential generalizability of our findings across both sexes and residential backgrounds.

In our study, the mean duration of dialysis was 4.17 ± 3.61 years, with a median of 3 years and a wide range from 5 months to 15 years. This indicates that a substantial proportion of our pediatric patients have been maintained on dialysis for prolonged periods. When compared to other international studies, this duration appears notably longer.

Levy Erez et al. (11), in a single-center study from Israel involving 110 children on chronic dialysis, reported a considerably shorter median dialysis duration of 1.46 years.

Similarly, **Chan et al. (12)**, in a comprehensive 20-year territory-wide analysis of 147 pediatric patients on kidney replacement therapy, observed a median time to kidney transplantation of 3.7 years. These differences may reflect variations in healthcare infrastructure, availability of donor organs, or national transplant policies. The extended dialysis duration in our population may also suggest challenges in access to transplantation services or delays in referral, underscoring the need for health system improvements to reduce time on dialysis and improve outcomes.

In our study, there was a significant decrease in parathyroid hormone (PTH) and phosphorus levels following low phosphorus-containing formula ($p<0.001$).

These findings align with previous studies indicating that hyperphosphatemia is a primary stimulus for elevated PTH levels in chronic kidney disease (CKD). In children, where bone growth and mineral metabolism are especially dynamic, uncontrolled SHPT not only contributes to skeletal deformities and growth retardation but is also associated with increased cardiovascular risk, including vascular calcification and left ventricular hypertrophy.

Previous studies highlighting the phosphate-regulating role of dietary phosphorus intake. High phosphorus intake is known to stimulate PTH secretion, primarily through the induction of mild hypocalcemia or a direct effect on the parathyroid glands.

Our findings are consistent with those of Portale et al. reported reductions in serum PTH following dietary phosphorus restriction in both healthy individuals and patients with chronic kidney disease **(13)**. Similarly, Isakova et al. demonstrated that lowering dietary phosphorus led to a decline in fibroblast growth factor 23 (FGF23) and PTH levels, emphasizing the endocrine interplay in phosphate homeostasis. **(14)**.

Compared to these earlier studies, the magnitude of PTH reduction in our study was notably larger, which may be attributable to differences in baseline phosphorus intake, the phosphorus content of the formula used, or the population studied.

There was no statistically significant change after the formula regarding blood urea nitrogen (BUN), Creatinine, serum calcium (Ca^{+2}), albumin, hemoglobin levels. These results suggest that dietary phosphorus restriction can effectively reduce parathyroid hormone (PTH) levels without adversely affecting other key biochemical parameters.

Similarly, a study by **Guida et al. (15)** examined the effects of dietary phosphate restriction in dialysis patients. The researchers observed decreases in serum phosphate and intact PTH levels without significant alterations in serum albumin, calcium, potassium, or body composition, suggesting that phosphorus-controlled diets can be implemented safely without compromising nutritional health **(15)**.

Moreover, a study by **Shichiri et al. (16)** assessed the impact of a low-protein, very-low-phosphorus diet on patients with diabetic renal insufficiency and proteinuria. The results demonstrated significant reductions in serum urea nitrogen and urinary excretion of protein, creatinine, urea nitrogen, and phosphate. Importantly, there were no significant changes in serum creatinine, protein, albumin, calcium, or hemoglobin levels, indicating that the diet effectively managed phosphorus levels without adversely affecting other biochemical parameters.

In our study, a significant gender-based disparity was observed in pre-formula PTH levels, with females demonstrating notably higher median levels compared to males (369 vs. 123 pg/mL, $p = 0.024$). This finding is consistent with **Laster et al., (17)** and **Nan et al., (18)** suggesting that gender may influence PTH regulation in pediatric dialysis populations.

Laster et al. (17) analyzed a cohort of 661 children on dialysis and reported significantly elevated PTH levels in African-American and Hispanic females compared to their Caucasian counterparts, with increases of 38% and 28.8%, respectively. These results emphasize the combined influence of gender and ethnicity on mineral metabolism in chronic kidney disease (CKD) patients.

Furthermore, **Nan et al. (18)** established age- and gender-specific reference intervals for biochemical markers in healthy three-year-old children, identifying gender as a significant determinant of PTH levels even in non-CKD populations.

There is no statistically significant correlation was found between age and parathyroid hormone (PTH), creatinine, calcium, phosphorus, albumin, and hemoglobin levels ($p > 0.05$). These findings are supported by **Li et al. (19)** emphasized that while age-specific reference ranges are clinically relevant, individual biochemical values such as PTH, calcium, and phosphorus are more strongly affected by the underlying pathology and therapeutic interventions. Additionally, clinical practice points from the ESPN CKD-MBD and Dialysis Working Group similarly underscore the limited role of age in determining these parameters in pediatric CKD populations.

Conversely, a significant positive correlation was identified between age and dialysis duration ($r = 0.443$, $p = 0.001$), suggesting that older children had longer exposure to dialysis. This observation is consistent with findings from **Abd Alati et al. (20)**, who reported that serum levels of trace elements, particularly lead, were positively associated with the duration of dialysis in pediatric end-stage renal disease patients. The study implies that older patients, by virtue of longer disease duration, are more likely to have undergone prolonged dialysis therapy.

CONCLUSION

The administration of a low phosphorus-containing formula in children on dialysis resulted in a statistically significant reduction in serum parathyroid hormone (PTH) and phosphorus levels, indicating its effectiveness in managing secondary hyperparathyroidism and hyperphosphatemia in this population. Importantly, this intervention did not adversely affect kidney function markers (BUN, creatinine), serum calcium, albumin, or hemoglobin levels, suggesting it is a safe and well-tolerated approach. These findings support the therapeutic value of phosphorus restriction via specialized nutritional formulations in pediatric dialysis patients, with potential benefits for bone and cardiovascular health.

REFERENCES:

1. Fusaro, M., Barbuto, S., Gallieni, M., Cossettini, A., Re Sartò, G. V., Cosmai, L., Cianciolo, G., La Manna, G., Nickolas, T., Ferrari, S., Bover, J., Haarhaus, M., Marino, C., Mereu, M. C., Ravera, M., Plebani, M., Zaninotto, M., Cozzolino, M., Bianchi, S., Messa, P., ... Tripepi, G. (2023). Real-world usage of chronic kidney disease–mineral bone disorder (CKD-MBD) biomarkers in nephrology practices. Clinical Kidney Journal, 17(1), sfad290.
2. Capossela, L., Ferretti, S., D'Alonzo, S., Di Sarno, L., Pansini, V., Curatola, A., Chiaretti, A., & Gatto, A. (2023). Bone disorders in pediatric chronic kidney disease: A literature review. *Biology, 12*(11), 1395.
3. Goodbred, A. J., & Langan, R. C. (2023). Chronic kidney disease: Prevention, diagnosis, and treatment. *American Family Physician, 108*(6), 554–561.
4. Khan, M., Sundar, S., Rampure, S., Siddini, V., & Bhat, V. (2024). Comparison between values of iPTH and wPTH in patients with chronic kidney disease. *Journal of the Egyptian Society of Nephrology and Transplantation, 24*(1), 45.
5. Wijaya, E., Solek, P., Rachmadi, D., Rahayuningsih, S. E., Tarigan, R., & Hilmanto, D. (2024). Effect of executive function on health-related quality of life in children with chronic kidney disease. *International Journal of Nephrology and Renovascular Disease, 17*, 17–28.
6. Chen, L., He, J.-X., Chen, Y.-Y., Ling, Y.-S., Lin, C.-H., & Guan, T.-J. (2018). Intensified treatment of hyperphosphatemia associated with reduction in parathyroid hormone in patients on maintenance hemodialysis. *Renal Failure, 40*(1), 15–21.
7. Hintze, J. L. (2017). PASS 15 Power Analysis and Sample Size Software. NCSS, LLC.
8. Cohen, J. (1988). Statistical Power Analysis for the Behavioral Sciences (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates.
9. Lakens, D. (2021). Calculating and reporting effect sizes: A practical primer for psychologists. European Journal of Psychological Assessment, 37(1), 1-16.
10. Sharaf, A., El-Kenawy, S., Taha El-Keiy, M., Mohammed Rabeea, Ų., & Soliman Hammad, K. (2020). Evaluation of some trace elements and parathyroid hormone levels in children with chronic renal failure on regular hemodialysis. *Al-Azhar Journal of Pediatrics, 23*, 629–647.
11. Levy Erez, D., Krause, I., Dagan, A., Cleper, R., Falush, Y., & Davidovits, M. (2016). Impact of Pediatric Chronic Dialysis on Long-Term Patient Outcome: Single Center Study. *International journal of nephrology, 2016*(1), 2132387.

12. Chan, E. Y. H., Yap, D. Y. H., Wong, W. H. S., Ho, T. W., Tong, P. C., Lai, W. M., Chan, T. M., & Ma, A. L. T. (2022). Demographics and long-term outcomes of children with end-stage kidney disease: a 20-year territory-wide study. *Nephrology*, *27*(2), 171-180.
13. Portale, A. A., Halloran, B. P., & Morris, R. (1987). Dietary intake of phosphorus modulates the circadian rhythm in serum concentration of phosphorus: Implications for the renal production of 1,25-dihydroxyvitamin D. *The Journal of Clinical Investigation, 80*, 1147–1154.
14. Isakova, T., Gutiérrez, O. M., & Wolf, M. (2009). A blueprint for randomized trials targeting phosphorus metabolism in chronic kidney disease. *Kidney International, 76*, 705–716.
15. Guida, B., Piccoli, A., Trio, R., Laccetti, R., Nastasi, A., Paglione, A., et al. (2011). Dietary phosphate restriction in dialysis patients: A new approach for the treatment of hyperphosphataemia. *Nutrition, Metabolism and Cardiovascular Diseases, 21*, 879–884.
16. Shichiri, M., Nishio, Y., Ogura, M., & Marumo, F. (1991). Effect of low-protein, very-low-phosphorus diet on diabetic renal insufficiency with proteinuria. *American Journal of Kidney Diseases, 18*, 26–32.
17. Laster, M., Soohoo, M., Streja, E., Elashoff, R., Jernigan, S., Langman, C. B., Norris, K. C., Salusky, I. B., & Kalantar-Zadeh, K. (2019). Racial-ethnic differences in chronic kidney disease-mineral bone disorder in youth on dialysis. *Pediatric Nephrology*, *34*, 107-115
18. Nan, J., Li, X., Zhang, X., Zheng, R., Men, S., Guo, G., Tian, Y., & Dong, J. (2022). Reference Intervals and Regularity Analysis of Biochemical Makers in 3-Year-Old Children. *Clinical Laboratory*, *68*(3).
19. Li, J., He, D., Zhao, W., Wu, X. a., Luo, M., Wang, Y., Yan, M., Niu, W., & Li, P. (2021). Association of mineral metabolism biomarkers with chronic kidney disease in Chinese adults. *Clinical and Experimental Nephrology*, *25*, 760-770.
20. Abd Alati, A. E., Abd, A.-H. A. E.-M., El-Hamid, S. A. E.-H., Yousef, D. M., Abd El-Gawad, E. R., & Zakaria, R. M. (2024). Serum Level of Trace Elements in Pediatric Patients with End Stage Renal Disease on Hemodialysis. The Egyptian Journal of Hospital Medicine (April 2024), 95, 2134-2141.