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Original Article

Parathyroid hormone, pulmonary artery pressure and right ventricular function in patients undergoing/awaiting hemodialysis

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Abstract

Background: Cardiovascular disease is a leading cause of death in chronic kidney disease (CKD) patients, with pulmonary hypertension (PH) and right ventricular (RV) dysfunction being especially detrimental. Secondary hyperparathyroidism (SHPT), common in CKD-mineral and bone disorder (CKD-MBD), has been linked to vascular problems, but its specific role in pulmonary vascular disease and RV function in hemodialysis (HD) patients remains unclear.This study examined the relationship between serum intact parathyroid hormone (iPTH), pulmonary artery pressure (PAP), and RV function in CKD patients (both on HD and pre-dialysis). The hypothesis was that higher iPTH correlates with increased PAP and worse RV function.

Methods: A total of 115 CKD patients (69.6% HD, 30.4% pre-dialysis) were enrolled. Clinical data included iPTH, calcium, phosphorus, and albumin. Echocardiography measured pulmonary artery systolic pressure (PASP), tricuspid annular plane systolic excursion (TAPSE), and fractional area change (FAC). Statistical analyses adjusted for confounders.

Results: PH prevalence was 60%. Severe PH patients had significantly higher mean iPTH (P=0.001). In the HD group, elevated iPTH strongly linked to severe PH (p<0.001); each unit iPTH increase raised PH risk by 1.01-fold (P=0.004). However, no direct iPTH–RV function association was found.

Conclusion: Elevated iPTH and longer HD duration are significant PH risk factors in CKD, particularly in HD patients. These findings underscore the need for CKD-MBD management and suggest that PTH-lowering therapies may reduce PH risk and improve cardiovascular outcomes. Further research is needed to identify other contributors to RV dysfunction in CKD.

Keywords: Parathyroid hormone, Pulmonary hypertension, Right ventricular function, Hemodialysis, Chronic kidney disease.

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Chronic kidney disease (CKD), which affects over 10% of the global adult population, poses a significant public health challenge, with cardiovascular complications being the leading contributor to morbidity and mortality (1). Among these, pulmonary hypertension (PH) and right ventricular (RV) dysfunction are especially severe. In patients undergoing hemodialysis (HD), PH arises from a complex interplay of factors, including uremic toxins, systemic inflammation, fluid overload, and vascular remodeling (2). However, the detailed mechanisms driving PH in this population remain poorly understood, hindering efforts to develop targeted therapies (3). Secondary hyperparathyroidism (SHPT), a frequent and often severe manifestation of CKD-mineral and bone disorder (CKD-MBD), has garnered increasing attention as a potential contributor to systemic vascular pathology (4).

Elevated parathyroid hormone (PTH) levels are implicated in processes such as vascular calcification, endothelial dysfunction, and inflammation mechanisms that may plausibly connect SHPT to the development of both PH and RV dysfunction. Despite the biological plausibility of this connection, current evidence on the relationships between SHPT, PH, and RV function remains inconsistent and constrained by significant methodological limitations (5). The results of the study by Havoshki et al. showed that there is a possible relationship between hyperparathyroidism, serum calcium levels, and PAH in hemodialysis patients (6). Since secondary hyperparathyroidism is frequently diagnosed in ESRD patients undergoing hemodialysis and this can lead to the occurrence of PH, which leads to irreversible heart failure and death in these patients, it is important to consider this association and timely evaluate these patients using echocardiography.

Despite studies, many questions remain about the prevalence and pathogenesis of pulmonary hypertension in dialysis patients (7). Furthermore, PAH is associated with an increased risk of cardiovascular complications in hemodialysis patients (8). Risk factors for PAH in hemodialysis patients include left ventricular systolic and diastolic dysfunction, age, and smoking (9, 10). Most prior studies investigating these associations are cross-sectional, limiting their ability to establish temporal relationships or infer causation (11, 12). Additionally, many of these studies lack comprehensive echocardiographic evaluations and fail to adjust for critical confounding factors, making it difficult to isolate SHPT's role in PH pathogenesis (13, 14). Therefore, the aim of this study was to investigate the relationship between serum parathyroid hormone (PTH) levels, right ventricular function, and pulmonary arterial pressure in patients with chronic hemodialysis.

Methods

Study design and population: This prospective, observational cohort study examined the relationships between serum intact parathyroid hormone (iPTH) levels, pulmonary arterial pressure (PAP), and right ventricular (RV) function in a cohort of 115 adult patients diagnosed with stage 4 or 5 chronic kidney disease (CKD). Conducted at a specialized nephrology and cardiology center from January 2022 to December 2023, the study included individuals undergoing maintenance hemodialysis (HD) for at least three months as well as those receiving pre-dialysis care. Exclusion criteria aimed to reduce potential confounding factors and included congenital heart disease, active malignancy, significant chronic lung disease, known pulmonary arterial hypertension (PAH) from other causes, and medications directly influencing pulmonary artery pressure. Predialysis patients were defined as GFR less than 30 Ml/Min/1.73m2 (15). In dialysis patients, IPTH levels are 2-9 higher than the standard measurement range (16).In predialysis patients, the IPTH level is 2-3.3 higher than the standard value (17). Pulmonary arterial blood pressure was divided into 4 groups after measuring pulmonary artery systolic blood pressure using a cut-off point of 35 mmHg. sPAP < 34 mmHg was normal and healthy in terms of pulmonary arterial hypertension, sPAP between 35 and 45 mmHg was defined as mild PAH, between 45 and 60 mmHg as moderate, and greater than 61 mmHg as severe (18).

Data collection: At baseline, demographic information such as age, sex, race/ethnicity, comorbidities (e.g., diabetes, hypertension, coronary artery disease), and dialysis duration (where applicable) were collected for each participant. Fasting venous blood 4ml were obtained to measure serum iPTH, calcium, phosphorus, and albumin levels. Serum iPTH concentrations were analyzed using a chemiluminescent enzyme immunoassay, with reagents and instruments calibrated per manufacturer specifications (Serum iPTH levels were measured using COBAS411 equipment and a ROCH kit (Tavan Afarin Health Development Company) manufactured in Iran.).

Transthoracic echocardiography was performed using the high-resolution Vivid E95 ultrasound system (GE Healthcare, Chicago, IL). Echocardiographic parameters included pulmonary artery systolic pressure (PASP) as a surrogate for PAP, RV function metrics such as tricuspid annular plane systolic excursion (TAPSE) and fractional area change (FAC), and left ventricular ejection fraction (LVEF). A certified echocardiographer interpreted all echocardiographic measurements following established guidelines.

Outcome measures: The primary outcome was the relationship between serum iPTH levels and PASP, used as a surrogate for PAP. Secondary outcomes explored the correlations between iPTH and RV function metrics (TAPSE and FAC), subgroup analyses based on dialysis status (HD vs. pre-dialysis), and the relationship between iPTH levels and pulmonary hypertension severity, defined by established PASP thresholds.

Statistical analysis: Data analysis was performed using SPSS Version 28.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as means ± standard deviations (SD) or medians with interquartile ranges (IQR), depending on distribution, while categorical data were presented as frequencies and percentages. Correlations were evaluated

using Pearson's or Spearman's coefficients, depending on data normality. Multivariable linear and logistic regression models assessed independent associations between iPTH levels, PAP, and RV function, adjusting for confounders such as age, sex, comorbidities, and dialysis duration. Missing data were addressed using multiple imputation techniques based on a fully conditional specification model. Sensitivity analyses were conducted to confirm the robustness of findings across imputation models. Statistical significance was set at a two-tailed p-value of < 0.05.

Materials: Standard clinical reagents were sourced from reputable vendors and used by manufacturer's instructions. Laboratory analyses were performed using calibrated, quality-controlled equipment. Echocardiographic evaluations adhered to a standardized protocol guided by institutional and professional standards.

Results

This prospective cohort study meticulously examined the intricate relationships between serum intact parathyroid hormone (iPTH) concentrations, pulmonary artery pressure (PAP) as indexed by pulmonary artery systolic pressure (PASP), and right ventricular (RV) function in a cohort of 115 patients with chronic kidney disease (CKD) (table 1). The findings unveiled a compelling association between elevated iPTH levels and the presence of severe pulmonary hypertension (PH), particularly within the hemodialysis (HD) subpopulation. To ensure a comprehensive analysis, results were further stratified by key demographic and clinical variables.

Study population characteristics: The study cohort had a mean age of 62.3±12.4 years. A substantial proportion, 69.6% (n = 80), were receiving maintenance HD, with an average dialysis duration of 8.4±1.7 years. The overall prevalence of PH, determined by established PASP thresholds, was 60% (n = 69). Within the PH population, 31.3% were classified as having mild, 25.2% moderate, and 3.5% severe PH. Notably, PH prevalence was significantly lower in patients undergoing HD compared with those in the pre-dialysis group (p < 0.001). This suggests a potentially important difference in the disease presentation across different CKD stages.

Primary outcomes: Pulmonary hypertension and Ventricular dysfunction: A robust association was observed between severe PH and moderate dysfunction of both the right and left ventricles (p < 0.001), highlighting the interconnectedness of cardiopulmonary sequelae in this cohort.

- iPTH and PH severity: Patients exhibiting severe PH displayed significantly elevated mean iPTH concentrations (P = 0.001), as well as longer durations of dialysis (p < 0.001). The association between iPTH levels and PH severity was particularly striking in the HD subpopulation (p < 0.001), while the same association did not attain statistical significance in the pre-dialysis cohort (P = 0.093), suggesting a differential impact of SHPT in the presence of chronic HD.
- Demographic and Clinical Subgroup Analyses: Elevated iPTH levels showed significant associations with severe PH across multiple clinically relevant subgroups, including patients over the age of 60 (P = 0.001), both male (P = 0.030) and female individuals (P = 0.001), and those with diagnoses of diabetes (P = 0.001) or hypertension (P= 0.002). This underscores the broad impact of SHPT in this complex patient population.
- Correlations: PASP demonstrated significant positive correlations with iPTH concentrations (r = 0.787, p < 0.001), dialysis duration (r = 0.731, p < 0.001), serum calcium (r = 0.578, p < 0.001), serum phosphorus (r = 0.222, P= 0.017), and serum albumin (r = 0.234, P = 0.012). Furthermore, iPTH concentrations were also positively correlated with both dialysis duration (r = 0.652, p < 0.001) and serum calcium concentrations (r = 0.661, p < 0.001), further illuminating the inter-related nature of these biochemical and clinical variables.

Secondary outcomes:

- iPTH and Ventricular function: In contrast to our primary findings, no significant associations were detected between serum iPTH concentrations and measures of either RV or left ventricular (LV) function (p > 0.05).
- iPTH in Pre-dialysis vs. Hemodialysis patients: Serum iPTH concentrations did not differ significantly between pre-dialysis and HD patients when stratified by ventricular function (p > 0.05), reinforcing the complexity of the relationship between PTH, renal replacement therapy, and ventricular dysfunction.
- Multivariable Logistic Regression Analysis (figure 1): Following adjustment for potential confounders, a multivariable logistic regression analysis revealed that each unit increase in serum iPTH was associated with a 1.01-fold increase in the odds of PH (P = 0.004). Furthermore, dialysis duration emerged as an independent predictor of PH, with each additional year of dialysis increasing the odds of PH by 2.40-fold (P= 0.003). Conversely, surprisingly, higher serum calcium concentrations were associated with a reduced risk of PH (P= 0.029).

Consistency with hypotheses: These results are consistent with our primary hypothesis, thereby supporting the assertion that elevated iPTH concentrations represent an independent and significant risk factor for increased PAP and PH in CKD patients, especially those receiving HD. Detailed demographic data, PH prevalence information, and comprehensive analyses of associated risk factors are available in the supplementary tables and figures. For all analyses, statistical significance was defined as a p-value of < 0.05.

Table 1. Comparative clinical and biochemical characteristics of predialysis and hemodialysis patients

Parameter	Predialysis (n=15)	Hemodialysis (n=80)	P-value
Age (years)	60±12	64±13	0.045
Sex (M/F)	8/7	52/28	0.231
Body Mass Index (kg/m²)	23.5±2.1	24.2±2.3	0.120
Duration of Dialysis (years)	-	8.44±1.65	-
Hypertension (%)	66.7	75	0.322
Diabetes Mellitus (%)	53.3	60	0.478
Systolic Pulmonary Artery Pressure (mmHg)	34.5±5.3	41.2±6.4	0.012
Pulmonary Hypertension (%)	46.7	57.5	0.090
Cardiac Output (L/min)	5.2±0.6	4.8±0.7	0.015
Left Ventricular Ejection Fraction (%)	55±5	50±6	0.004
Serum Albumin (g/dL)	3.8±0.3	3.6±0.4	0.050
Serum Blood Urea Nitrogen (mg/dL)	19.4±6.5	58.7±18.6	<0.001
Serum Creatinine (mg/dL)	2.5±0.7	10.2±3.5	<0.001
Serum Phosphorus (mg/dL)	4.1±0.5	5.0±0.8	0.018
Calcium × Phosphorus Product (mg²/dL²)	10.3±2.1	11.8±2.6	0.033
Hemoglobin (g/dL)	11.5±1.2	10.8±1.5	0.210
Parathyroid Hormone (pg/mL)	229.16±89.15	229.96±85.39	0.990

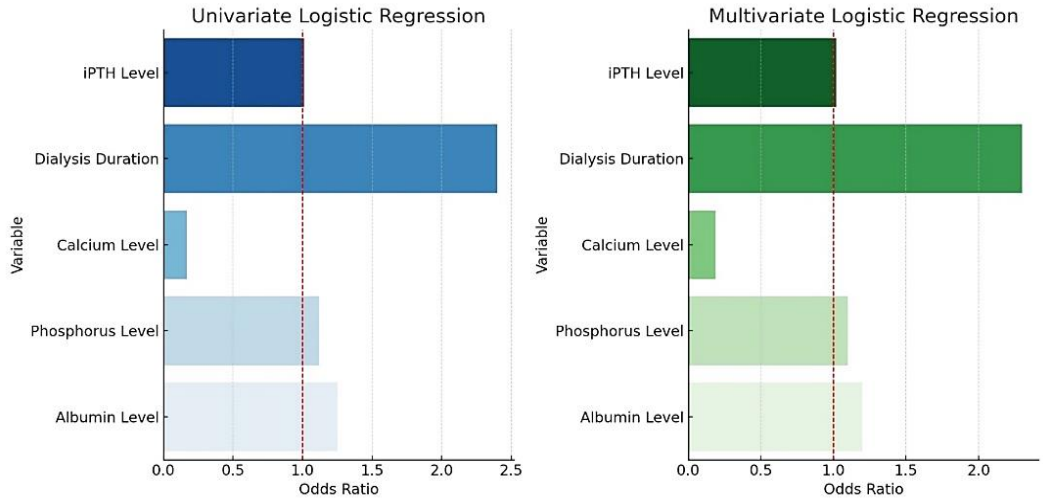

Figure 1. Logistic regression analysis of patients with pre-hemodialysis and hemodialysis

Discussion

The results of the study showed that there is a positive correlation between serum IPTH and PAH levels, and with increasing severity of pulmonary arterial hypertension, parathyroid hormone levels also increase. According to the results of our study, the mean parathyroid hormone level was significantly higher in patients with severe PAH. In similar studies, Mortazavi Moghadam et al. (19), Tudoran et al. (20), Havoshki et al. (6), Genctoy et al. (21) and Baradaran et al. (22) found a moderate correlation between IPTH and PAH in patients undergoing hemodialysis.

The effect of high serum PTH concentration on pulmonary artery pressure may be through increasing the calcium content of arterial smooth muscle cells. In fact, parathyroid hormone is known to increase calcium entry into many cells. In a study by Carracedo et al., parathyroid hormone was found to be a strong independent predictor of death among hemodialysis patients, independent of cardiovascular disease (23). In a study by Eskandarian et al., no significant difference in serum PTH levels was observed between patients with and without pulmonary hypertension (24). Amin et al. (25) and Hayati et al. (26) also found no significant association between PAH and serum parathyroid hormone levels in hemodialysis patients.

Published data on the prevalence of pulmonary arterial hypertension in hemodialysis patients and its mechanisms are insufficient. The reason for such different results in the above studies could be due to more recent findings that suggest dysfunction of vascular endothelial cells as the main pathogenesis of pulmonary hypertension in hemodialysis patients. In the present study, the prevalence of PAH was higher in predialysis patients than in hemodialysis patients (60% vs. 42.5%). It is worth noting that PAH was reported in all predialysis patients, and this finding is clinically important.Various articles state that predialysis and hemodialysis patients are equally susceptible to PAH (9,27). However, in our study, the prevalence of PAH in predialysis patients was 2.3 times higher than in hemodialysis patients. A possible explanation for this phenomenon maybe the increase in pressure following the absence of ultrafiltration in predialysis patients and the increase in cardiac output, which leads to an increase in pulmonary artery pressure (28). However, it is important to acknowledge that further studies are needed to establish a definitive correlation. The prevalence of PAH in hemodialysis patients in the studies of Ramasubbu et al. (29), Fabian et al. (30), and Eskandarian et al. (24) has ranged from 20% to 58%. The prevalence obtained in our study is also higher than the values reported in the above studies and the study of Saad et al., who reported a prevalence of PAH in hemodialysis patients ranging from 12% to 49% (27). According to the results of the study by Nagaraju et al., this variation in prevalence can be attributed to various factors, such as the specific threshold used to define PH, the inclusion criteria used in the different studies (which may include subjects with congestive heart failure or other comorbidities associated with increased pulmonary artery pressure). The specific extracorporeal dialysis method used (hemodialysis or peritoneal dialysis) may also be influential (31). Another study finding was the association between dialysis duration and PAH, with an increase in dialysis duration increasing the risk of PAH by 2.4 times. Tudoran et al. also reported a strong correlation between longer dialysis duration and increased severity of PAH (20), which is in line with the findings of the study. One of the clinically important points in our study was that a significant association was also reported between moderate right and left ventricular function and severe pulmonary arterial hypertension, but in multivariate analysis, mild right ventricular function increased the risk of PAH by 10.11 times. In the study by Reque et al., a significant association was observed between the presence of PAH and systolic dysfunction and diastolic dysfunction (32). PAH appears to be caused by pulmonary vascular calcification and decreased distensibility, followed by right ventricular failure in hemodialysis patients. The findings of this study show that there was no statistically significant difference between individuals with PAH and those without PAH in terms of age, gender, hypertension, diabetes mellitus, serum calcium, albumin, and phosphorus levels. The results obtained were consistent with the findings of Nagaraju et al. (31), Shang et al. (33), Mehta et al. (34), and Tarrass et al. (35). Blood pressure and its changes during dialysis are among the common disorders in dialysis patients, and understanding the factors affecting it can be effective in the prognosis of individuals. The existence of a correlation between parathyroid hormone and pulmonary artery blood pressure allows for accurate estimation of left and right ventricular function and pulmonary artery pressure without the risks of invasive procedures such as right heart catheterization. The prevalence of pulmonary arterial hypertension in our study was relatively high, which indicates the need to pay attention to pulmonary arterial hypertension in hemodialysis patients. Using the results of this study, early diagnosis of pulmonary arterial hypertension by correcting the underlying factor before the occurrence of right ventricular failure can reduce the cardiac mortality rate of patients undergoing hemodialysis and predialysis. It is suggested that a study be conducted in hemodialysis and predialysis patients with normal and

increased serum PTH levels and its relationship with pulmonary arterial blood pressure by measuring pulmonary arterial blood pressure through catheterization. The limitations of this study are the measurement of pulmonary arterial blood pressure with Doppler echocardiography instead of catheterization, which can reduce the accuracy of the values obtained, and the lack of examination of anemia variables, heart rate, and blood pressure, which can affect pulmonary arterial blood pressure. In conclusion, this study provides robust evidence supporting the independent associations of elevated iPTH concentrations and prolonged dialysis duration with PH in CKD patients, particularly those on HD. Our findings underscore the critical need for early detection and targeted management strategies for SHPT to mitigate the risk of pulmonary vascular disease and potentially improve cardiovascular outcomes in this high-risk population. This study also establishes a solid foundation for future research to further elucidate the complex pathobiology of PH in CKD, refine existing therapeutic strategies, and explore novel therapeutic options for patients affected by this challenging condition.

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Authors' contribution: Authors wrote the main manuscript text prepared tables. All authors reviewed and approved the final manuscript. It is important to note that no generative AI technologies were used in the creation of this work. Authors wrote the main manuscript text prepared tables. All authors reviewed and approved the final manuscript. It is important to note that no generative AI technologies were used in the creation of this work.

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