Open Access

ISSN: 2663-9491 e-ISSN: 2789-6897

ORIGINAL ARTICLE

Parathormone as a Risk Factor for Cardiac Dysfunction in Individuals Undergoing Hemodialysis

DOI: 10.5281/zenodo.17377844

Shanjida Sultana Juthy[1], Samira Khatun[2], Madhabi Karmaker[3], Khaleda Akhter[4], Farnaz Nobi[5]

Received: 10 Oct 2025
Accepted: 13 Oct 2025
Published: 17 Oct 2025

Published by:
Gopalganj Medical College, Gopalganj, Bangladesh

Correspondence to
Shanjida Sultana Juthy

ORCID
https://orcid.org/0009-0005-0616-6196

ABSTRACT

Background: *Elevated parathyroid hormone (iPTH) levels have been associated with adverse cardiovascular outcomes in hemodialysis patients. This study aims to explore the relationship between elevated iPTH levels and cardiac dysfunction, with a focus on left ventricular ejection fraction (LVEF), left ventricular hypertrophy (LVH), and other cardiovascular markers.* ***Objectives:*** *To explore the role of parathyroid hormone (PTH) as a potential risk factor for cardiac dysfunction in individuals undergoing hemodialysis.* ***Methods & Material:*** *A total of 215 hemodialysis patients were enrolled and categorized into elevated iPTH and normal iPTH groups. Demographic data, dialysis duration, biochemical markers, and cardiovascular parameters, including LVEF, LVH, B-type natriuretic peptide (BNP), and carotid intima-media thickness (CIMT), were assessed. Logistic regression and multivariate linear regression were used to evaluate the relationship between iPTH and cardiovascular outcomes.* ***Result:*** *The elevated iPTH group had significantly lower LVEF (50.1% vs. 56.4%, p<0.001), higher prevalence of LVH (70% vs. 48.2%, p=0.002), and elevated BNP and CIMT levels (p<0.001). Multivariate analysis revealed that iPTH levels were independently associated with reduced LVEF and increased cardiovascular risk (adjusted OR: 2.34, p=0.012). Serum calcium and phosphorus imbalances were also significant predictors of cardiovascular dysfunction.* ***Conclusion:*** *Elevated iPTH levels are significantly associated with adverse cardiovascular outcomes, including reduced LVEF, increased LVH, and elevated BNP and CIMT, in hemodialysis patients. These findings highlight the critical role of iPTH in cardiovascular pathology and suggest that controlling iPTH levels could improve cardiovascular health in this population.*

Keywords: *Elevated parathyroid hormone, cardiovascular dysfunction, hemodialysis, left ventricular ejection fraction, left ventricular hypertrophy*

(The Insight 2025; 8(2): 296-301)

1. *Assistant Professor, Kidney Foundation Hospital & Research Institute, Dhaka, Bangladesh*
2. *Dialysis Medical Officer, Department of Nephrology, Rajshahi Medical College Hospital, Rajshahi, Bangladesh*
3. *Junior Consultant, Department of Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh*
4. *Major (Assistant Professor), Combined Military Hospital, Dhaka, Bangladesh*
5. *Assistant Professor, Department of Nephrology, Kidney Foundation Hospital and Research Institute, Dhaka, Bangladesh*

INTRODUCTION

Parathormone or Parathyroid hormone (PTH) is a critical regulator of calcium and phosphate homeostasis, exerting significant effects on bone metabolism [1]. In patients undergoing hemodialysis (HD), altered PTH levels have been implicated in various cardiovascular complications, including cardiac dysfunction [2]. This introduction explores the association between PTH and cardiac dysfunction in individuals undergoing HD, highlighting the underlying mechanisms and clinical implications. Chronic kidney disease (CKD) is a progressive condition that often necessitates renal replacement therapy, with hemodialysis being the most prevalent and widely used modality for patients who reach end-stage renal disease (ESRD) [3]. However, patients undergoing HD often experience significant disturbances in mineral metabolism, leading to secondary hyperparathyroidism. Elevated levels of PTH are commonly seen in these individuals and have been linked to several adverse cardiovascular outcomes, contributing to a heightened risk of cardiovascular morbidity and mortality in this population [4]. A study demonstrated that a clear association between increased PTH concentrations and higher mortality rates among HD patients in the Gulf Cooperation Council countries, underscoring the impact of this hormone on

ISSN: 2663-9491 e-ISSN: 2789-6897

long-term outcomes [5]. The pathophysiological mechanisms connecting PTH to cardiac dysfunction in HD patients are complex and multifactorial. PTH exerts its effects not only on bone metabolism but also on myocardial contractility, vascular tone, and cardiac remodeling [6]. PTH serves as a critical bridge between bone metabolism and cardiovascular disease, affecting both bone and heart tissues. Additionally, elevated PTH levels may contribute to left ventricular hypertrophy (LVH), a precursor to heart failure [7]. Echocardiographic techniques were utilized to assess left ventricular structure and function in end-stage renal disease patients, providing insights into cardiac alterations [8]. Furthermore, PTH interacts with other hormones and factors that modulate cardiovascular health [9]. For instance, fibroblast growth factor 23 (FGF23) and vitamin D are involved in mineral metabolism and have been linked to cardiac outcomes in CKD patients. The interplay between these factors complicates the assessment of PTH's direct effects on the heart [10]. Clinically, it has become evident that monitoring PTH levels in HD patients is essential for identifying those at higher risk for cardiac dysfunction. The role of fragmented QRS complexes on electrocardiograms as a marker of subclinical left ventricular dysfunction in chronic kidney disease patients, indicating the need for comprehensive cardiac screening in this population [11]. Additionally, interventions aimed at reducing PTH levels, such as the use of phosphate binders and vitamin D analogs, have demonstrated promise in mitigating cardiovascular risks and improving patient outcomes [12]. However, PTH affects renal phosphate transporters, offering potential avenues for therapeutic intervention [13]. Elevated PTH levels in HD patients are closely linked to cardiac dysfunction through various direct and indirect mechanisms [14]. This study aims to delve deeper into the role of PTH as a risk factor for cardiac dysfunction among individuals undergoing hemodialysis, providing evidence to inform clinical practices and therapeutic interventions.

METHODS & MATERIALS

This was a cross-sectional observational study conducted at [Institution Name] between [start date] and [end date] in the Department of [Department Name]. The study was approved by the institutional ethics committee, and written informed consent was obtained from all participants. A total of 215 adult patients who were undergoing regular hemodialysis were included in the study. Participants were categorized into two groups based on their parathormone (PTH) levels: PTH Elevated (N=130) and PTH Normal (N=85).

Inclusion Criteria

- Adults aged ≥18 years.
- Hemodialysis patients with at least 6 months of dialysis history.
- Stable clinical status, with no major acute complications in the past month.

Exclusion Criteria

- Severe acute or chronic infections.
- Active malignancy.
- Severe liver dysfunction (e.g., cirrhosis).
- Recent myocardial infarction or stroke.
- Pregnancy.

Clinical, Biochemical, And Cardiac Assessment

Baseline demographic and clinical data, including age, sex, duration of hemodialysis, blood pressure, and comorbidities such as hypertension and diabetes mellitus, were recorded. Blood samples were collected before dialysis for biochemical analysis, which included parathormone (PTH), serum calcium, phosphorus, albumin, and B-type natriuretic peptide (BNP) levels. PTH was measured using an enzyme-linked immunosorbent assay (ELISA), with levels >65 pg/mL considered elevated. Serum calcium and phosphorus were determined using colorimetric assays, while BNP was analyzed using a high-sensitivity ELISA kit.

Cardiac function was assessed using transthoracic echocardiography to measure left ventricular ejection fraction (LVEF) and detect left ventricular hypertrophy (LVH), defined as a left ventricular mass index >115 g/m^2 in men and >95 g/m^2 in women. Carotid intima-media thickness (CIMT) was evaluated using ultrasound, and electrocardiography (ECG) was performed to assess cardiac rhythm abnormalities. Dialysis parameters, including dialysis vintage (total duration of dialysis in months) and dialysis adequacy (measured as Kt/V), were recorded.

Data Collection

Data were collected through structured patient interviews, clinical examinations, and medical record reviews. Demographic and clinical characteristics were obtained during patient visits, while laboratory parameters were analyzed using standard biochemical techniques. Cardiac function assessments were performed by trained cardiologists using echocardiography and ultrasound, ensuring consistency in measurements. Dialysis-related parameters were extracted from medical records, including dialysis duration and adequacy. All data were systematically recorded in a secure electronic database, with regular quality checks to ensure accuracy and completeness.

Statistical Analysis

Data analysis was conducted using SPSS software version 26 (IBM Corporation, Armonk, NY). Continuous variables were presented as mean ± standard deviation (SD) or median (interquartile range), while categorical variables were expressed as frequencies and percentages. Comparisons between groups (elevated vs. normal PTH levels) were performed using the Student's t-test for continuous variables and the chi-squared test for categorical variables. Univariate and multivariate logistic regression analyses were conducted to determine the association between PTH levels and cardiac dysfunction, adjusting for potential confounders such as age, gender, dialysis vintage, and comorbidities. Results were reported as odds ratios (OR) with 95% confidence intervals (CI), and a p-value <0.05 was considered statistically significant.

ISSN: 2663-9491 e-ISSN: 2789-6897

RESULT

A total of 215 patients participated in this study. The mean age of participants with elevated iPTH was 59.1±10.8 years), compared to 57.2±11.6 years) for those with normal iPTH (p=0.321). The gender distribution was similar, with 64.62% of participants with elevated iPTH being male, compared to 57.65% in the normal iPTH group (p=0.402). Dialysis duration was significantly longer for the elevated iPTH group (52.8 ± 27.1 months) compared to the normal iPTH group (41.5±24.9 months, p=0.012). The prevalence of hypertension was higher in the elevated iPTH group (86.15%) compared to the normal iPTH group (77.65%, p=0.098). Diabetes Mellitus was more prevalent in the elevated iPTH group (53.08%) than the normal iPTH group (38.82%, p=0.043) (Table I). Table II presented the cardiac dysfunction parameters based on iPTH levels. The mean Left Ventricular Ejection Fraction (LVEF) was significantly lower in participants with elevated iPTH (50.1±8.9) compared to the normal iPTH group (56.4±9.2, p<0.001). Left Ventricular Hypertrophy (LVH) was more common in the elevated iPTH group (70% compared to 48.2% with normal iPTH, p=0.002). BNP levels were significantly higher in the elevated iPTH group (511±230 pg/mL compared to 301±180 pg/mL with normal iPTH, p<0.001). Carotid Intima-Media Thickness (CIMT) was also significantly higher in the elevated iPTH group (0.89 ± 0.23 mm compared to 0.74±0.19 mm with normal iPTH, p<0.001). Logistic regression showed a significant association between elevated iPTH and cardiac dysfunction (adjusted OR 2.34, p=0.012). Dialysis duration greater than 48 months was also associated with cardiac dysfunction (adjusted OR 1.82, p=0.028) (Table III). Table IV represented that biochemical and hemodialysis parameters showed that serum calcium was lower in the elevated iPTH group (8.1±0.8 mg/dL) compared to the normal iPTH group (8.8±0.7 mg/dL, p<0.001). Serum phosphorus was higher in the elevated iPTH group (5.5±1.3 mg/dL compared to 4.8±1.0 mg/dL with normal iPTH, p<0.001), as was the calcium-phosphorus product (45.9±10.1 mg^2/dL^2 compared to 41.7±8.5 mg^2/dL^2 with normal iPTH, p=0.004). Multivariate regression analysis found each 10 pg/mL increase in iPTH was associated with a decrease in LVEF (β = -0.28, p = 0.002) (Table V). Spearman's correlation showed significant negative correlations between iPTH and LVEF (ρ = -0.46, p<0.001) and positive correlations with LVH, BNP, CIMT, phosphorus, and calcium-phosphorus product (Table VI).

Table – I: Demographic and clinical characteristics of study population

Variable	iPTH Elevated (N=130)		iPTH Normal (N=85)		P-Value
	n	%	n	%	
Age (years)					
Mean ± SD	59.1 ± 10.8		57.2 ± 11.6		0.321
Gender					
Male	84	64.62	49	57.65	0.402
Female	46	35.38	36	42.35	
Dialysis Duration (months)					
Mean ± SD	52.8 ± 27.1		41.5 ± 24.9		0.012
Hypertension	112	86.15	66	77.65	0.098
Diabetes Mellitus	69	53.08	33	38.82	0.043

Table – II: Cardiac dysfunction parameters based on iPTH levels

Cardiac Dysfunction Parameter	iPTH Elevated (N=130) (Mean ± SD)	iPTH Normal (N=85) (Mean ± SD)	P-Value
Left Ventricular Ejection Fraction (LVEF)	50.1 ± 8.9	56.4 ± 9.2	<0.001
Left Ventricular Hypertrophy (LVH, %)	91 (70.00%)	41 (48.20%)	0.002
B-Type Natriuretic Peptide (BNP, pg/mL)	511 ± 230	301 ± 180	<0.001
Carotid Intima-Media Thickness (CIMT, mm)	0.89 ± 0.23	0.74 ± 0.19	<0.001

Table – III: Association between elevated iPTH and cardiac dysfunction (Logistic Regression Analysis)

Risk Factor	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	P-Value
iPTH > 65 pg/mL	2.85 (1.61–4.98)	2.34 (1.21–4.53)	0.012
Hypertension	1.79 (1.02–3.14)	1.45 (0.78–2.73)	0.189
Diabetes Mellitus	1.52 (1.04–2.82)	1.38 (0.93–2.67)	0.213
Dialysis Duration (>48 months)	2.11 (1.41–3.88)	1.82 (1.19–3.52)	0.028

Table – IV: Biochemical and hemodialysis parameters of the study population

Parameter	iPTH Elevated (N=130)	iPTH Normal (N=85)	P-Value
Serum Calcium (mg/dL)	8.1 ± 0.8	8.8 ± 0.7	<0.001
Serum Phosphorus (mg/dL)	5.5 ± 1.3	4.8 ± 1.0	<0.001
Calcium-Phosphorus Product (mg^2/dL^2)	45.9 ± 10.1	41.7 ± 8.5	0.004
Serum Albumin (g/dL)	3.7 ± 0.5	4.1 ± 0.6	<0.001
Dialysis Efficiency (Kt/V)	1.34 ± 0.22	1.41 ± 0.25	0.047

ISSN: 2663-9491 e-ISSN: 2789-6897

Table – V: Multivariate Linear Regression for Predictors of Left Ventricular Dysfunction (LVEF%)

Predictor	β-Coefficient	Standard Error	P-Value	Adjusted R^2
iPTH Level (per 10 pg/mL increase)	-0.28	0.09	0.002	0.31
Age (per 1-year increase)	-0.21	0.07	0.008	
Hypertension	-2.34	1.12	0.044	
Serum Phosphorus (mg/dL)	-0.91	0.28	0.003	
Dialysis Vintage (months)	-0.15	0.04	0.001	

Table – VI: Spearman's Correlation Between iPTH Levels and Cardiac Dysfunction Parameters

Variable	Spearman's ρ	P-Value
Left Ventricular Ejection Fraction (LVEF, %)	-0.46	<0.001
Left Ventricular Hypertrophy (LVH, %)	0.41	<0.001
B-Type Natriuretic Peptide (BNP, pg/mL)	0.48	<0.001
Carotid Intima-Media Thickness (CIMT, mm)	0.39	<0.001
Serum Calcium (mg/dL)	-0.33	0.001
Serum Phosphorus (mg/dL)	0.37	<0.001
Calcium-Phosphorus Product (mg^2/dL^2)	0.36	<0.001
Dialysis Duration (months)	0.25	0.009

DISCUSSION

Elevated parathyroid hormone (iPTH) levels have emerged as a significant risk factor for cardiac dysfunction in individuals undergoing hemodialysis. The relationship between parathyroid hormone (PTH) levels and cardiac dysfunction in individuals undergoing hemodialysis has garnered significant attention due to its potential implications for managing cardiovascular risk in this high-risk population. In our study, we examined 215 hemodialysis patients, with a focus on the effects of elevated intact parathyroid hormone (iPTH) levels on cardiac dysfunction. This study aimed a strong association between elevated iPTH and various markers of cardiac dysfunction, including left ventricular ejection fraction (LVEF), left ventricular hypertrophy (LVH), and serum biomarkers such as B-type natriuretic peptide (BNP) and carotid intima-media thickness (CIMT). These findings suggest that iPTH could be a key risk factor for cardiac abnormalities in this cohort. The baseline characteristics of the two treatment groups were comparable, with a mean age of 59.1±10.8 years in the elevated iPTH group and 57.2±11.6 years in the normal iPTH group. In a similar study conducted by *Li et al.*, the mean age of patients with elevated iPTH was 58.3 years, which is consistent with the age range observed in our study, suggesting that iPTH levels tend to increase with age, particularly in individuals with chronic kidney disease (CKD) [15]. Gender distribution was also comparable between groups, with 64.62% males in the elevated iPTH group and 57.65% in the normal iPTH group. In a study by *Tan J et al*., a male predominance was observed in hemodialysis patients, which is similar to the gender distribution in our STUDY [16].Furthermore, the elevated iPTH group had a significantly longer dialysis duration (52.8±27.1 months) compared to the normal iPTH group (41.5 ± 24.9 months, p=0.012), reflecting prolonged exposure to the metabolic abnormalities associated with CKD. This finding is in line with a study by *Zhang et al*., where longer dialysis duration was associated with increased iPTH levels and cardiovascular complications in hemodialysis patients [17]. Diabetes mellitus was more prevalent in the elevated iPTH group (53.08%) compared to the normal iPTH group (38.82%, p=0.043), consistent with prior research linking elevated iPTH levels with an increased risk of diabetes in hemodialysis patients [18]. The results of our study also demonstrated a clear link between elevated iPTH levels and the severity of cardiac dysfunction. Specifically, the mean LVEF was significantly lower in the elevated iPTH group (50.1±8.9) than in the normal iPTH group (56.4 ± 9.2, p<0.001). This is in line with previous findings suggested by *Bollerslev J et al*. that hyperparathyroidism is associated with adverse effects on cardiac function, particularly through mechanisms such as increased calcium-phosphate product and vascular calcification [19]. In our study, left ventricular hypertrophy (LVH) was more prevalent in the elevated iPTH group (70%) compared to the normal iPTH group (48.2%, p=0.002), which further supports the hypothesis that high iPTH levels contribute to structural cardiac changes, as LVH is a well-established marker of cardiac strain in dialysis patients [20]. B-type natriuretic peptide (BNP) levels, a marker of heart failure, were significantly higher in patients with elevated iPTH (511 pg/mL compared to 301 pg/mL, p<0.001). A study by *Maisel et al.,* demonstrated that elevated BNP levels are commonly associated with heart failure and other forms of cardiac stress, and the increase in BNP seen in this cohort may reflect the subclinical cardiac dysfunction associated with elevated iPTH levels [21]. Similarly, carotid intima-media thickness (CIMT), a marker of atherosclerosis and vascular dysfunction, was significantly higher in patients with elevated iPTH (0.89 mm compared to 0.74 mm, p<0.001), further suggesting that elevated iPTH may contribute to the development of both myocardial and vascular dysfunction in this population [22]. Logistic regression analysis revealed that elevated iPTH (>65 pg/mL) was independently associated with an increased risk of cardiac dysfunction, with an adjusted odds ratio (OR) of 2.34 (95% CI: 1.21–4.53, p=0.012). This association remained significant after adjusting for other potential confounders, such as hypertension, diabetes mellitus, and dialysis duration. These findings corroborate

previous studies that have identified elevated iPTH as an independent risk factor for adverse cardiac outcomes in hemodialysis patients.[23] Additionally, dialysis duration of more than 48 months was also significantly associated with cardiac dysfunction (adjusted OR: 1.82, 95% CI: 1.19–3.52, p=0.028), underscoring the role of long-term dialysis exposure in the development of cardiovascular complications [24]. Further analysis through multivariate linear regression confirmed that iPTH levels were a significant predictor of LVEF, with each 10 pg/mL increase in iPTH corresponding to a 0.28% decrease in LVEF (p=0.002). Other significant predictors of reduced LVEF included age, hypertension, serum phosphorus, and dialysis vintage. These results are consistent with literature suggesting that calcium-phosphate imbalances and PTH excess may directly contribute to cardiac dysfunction through both direct and indirect mechanisms [25]. Additionally, iPTH levels were negatively correlated with serum calcium (ρ=-0.33, p=0.001) and LVEF (ρ=-0.46, p<0.001), while being positively correlated with LVH (ρ=0.41, p<0.001), BNP (ρ=0.48, p<0.001), CIMT (ρ=0.39, p<0.001), and serum phosphorus (ρ=0.37, p<0.001), reinforcing the notion that iPTH acts as a central mediator of cardiovascular risk in hemodialysis patients. In terms of biochemical markers, the elevated iPTH group exhibited significantly lower serum calcium levels (8.1 mg/dL compared to 8.8 mg/dL, p<0.001) and higher serum phosphorus levels (5.5 mg/dL compared to 4.8 mg/dL, p<0.001), both of which are known to contribute to vascular calcification and cardiac dysfunction in CKD patients [26]. The calcium-phosphorus product, an important indicator of calcification risk, was significantly higher in the elevated iPTH group (45.9 mg^2/dL^2 compared to 41.7 mg^2/dL^2, p=0.004), further supporting the role of mineral imbalances in the pathogenesis of cardiovascular disease in this population. Additionally, the lower serum albumin levels and slightly reduced dialysis efficiency (Kt/V) observed in the elevated iPTH group may reflect poor nutritional status and suboptimal dialysis, both of which are recognized risk factors for cardiac dysfunction in dialysis patients [27].

LIMITATIONS OF THE STUDY

Despite the valuable insights provided by this study, several limitations should be acknowledged. Firstly, the cross-sectional design of the study limits the ability to establish causality between elevated iPTH levels and cardiovascular dysfunction. Longitudinal studies are needed to determine the long-term effects of iPTH on cardiovascular outcomes. Secondly, the sample size, while adequate for preliminary findings, may not be sufficiently large to capture all potential confounders or to generalize the results to a broader population of hemodialysis patients. Additionally, the study did not account for the potential impact of medications (such as calcium or phosphate binders) on iPTH levels and cardiovascular function, which could influence the observed associations. Furthermore, the lack of detailed data on nutritional status, physical activity, and other lifestyle factors may have contributed to residual confounding. Finally, the study was conducted in a single center, limiting the external validity of the results.

CONCLUSION AND RECOMMENDATIONS

In conclusion, our study demonstrates a significant association between elevated iPTH levels and adverse cardiovascular outcomes in hemodialysis patients. Elevated iPTH was linked to lower left ventricular ejection fraction (LVEF), higher left ventricular hypertrophy (LVH), increased B-type natriuretic peptide (BNP), and greater carotid intima-media thickness (CIMT), suggesting a direct impact on both myocardial and vascular dysfunction. Additionally, longer dialysis duration, diabetes mellitus, and mineral imbalances (including lower serum calcium and higher phosphorus) were identified as contributing factors to cardiovascular risk. Our findings highlight the critical role of managing iPTH levels and optimizing dialysis adequacy to mitigate cardiac dysfunction in this population. These results support the need for strategies aimed at controlling mineral metabolism and improving dialysis efficiency to improve long-term cardiovascular outcomes in hemodialysis patients. Further research is required to explore potential therapeutic interventions targeting elevated iPTH to prevent cardiovascular complications in this high-risk group.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee.

REFERENCES

1. *Lombardi G, Ziemann E, Banfi G, Corbetta S. Physical activity-dependent regulation of parathyroid hormone and calcium-phosphorous metabolism. International journal of molecular sciences. 2020 Jul 29;21(15):5388.*
2. *Ganesh SK, Stack AG, Levin NW, Hulbert-Shearon T, Port FK. Association of elevated serum PO4, Ca× PO4 product, and parathyroid hormone with cardiac mortality risk in chronic hemodialysis patients. Journal of the American Society of Nephrology. 2001 Oct 1;12(10):2131-8.*
3. *Price IN, Wood AF. Chronic kidney disease and renal replacement therapy: an overview for the advanced clinical practitioner. British Journal of Nursing. 2022 Feb 10;31(3):124-34.*
4. *Isakova T, Xie H, Yang W, Xie D, Anderson AH, Scialla J, Wahl P, Gutiérrez OM, Steigerwalt S, He J, Schwartz S. Fibroblast growth factor 23 and risks of mortality and end-stage renal disease in patients with chronic kidney disease. Jama. 2011 Jun 15;305(23):2432-9.*
5. *Al Salmi I, Bieber B, Al Rukhaimi M, AlSahow A, Shaheen F, Al-Ghamdi SM, Al Wakeel J, Al Ali F, Al-Aradi A, Al Hejaili F, Al Maimani Y. Parathyroid hormone serum levels and mortality among hemodialysis patients in the gulf cooperation council countries: results from the DOPPS (2012–2018). Kidney360. 2020 Oct 1;1(10):1083-90.*
6. *Kempson SA, Lotscher M, Kaissling BR, Biber J, Murer HE, Levi MO. Parathyroid hormone action on phosphate transporter mRNA and protein in rat renal proximal tubules. American Journal of Physiology-Renal Physiology. 1995 Apr 1;268(4):F784-91.*
7. *Goettsch C, Iwata H, Aikawa E. Parathyroid hormone: critical bridge between bone metabolism and cardiovascular disease. Arteriosclerosis, thrombosis, and vascular biology. 2014 Jul;34(7):1333-5.*
8. *Habib SA, Hasan AA, Abd Elaziz OH, Ibrahim AH, Ali TA, Kotb FM, Khalaf H, Omar MA, Said IF, Shokr AM, Elkhouly AA. Comparing*

Left Ventricular Structure and Functions in End-Stage Renal Disease Using Conventional Echocardiography and 2D Speckle Tracking Echocardiography. Cureus. 2024 Jun 22;16(6).

9. *Tomaschitz A, Ritz E, Pieske B, Rus-Machan J, Kienreich K, Verheyen N, Gaksch M, Grübler M, Fahrleitner-Pammer A, Mrak P, Toplak H. Aldosterone and parathyroid hormone interactions as mediators of metabolic and cardiovascular disease. Metabolism. 2014 Jan 1;63(1):20-31.*
10. *Anderson JL, Vanwoerkom RC, Horne BD, Bair TL, May HT, Lappé DL, Muhlestein JB. Parathyroid hormone, vitamin D, renal dysfunction, and cardiovascular disease: dependent or independent risk factors?. American heart journal. 2011 Aug 1;162(2):331-9.*
11. *Kahyaoğlu M, Geçmen Ç, Çelik M, Yılmaz Y, Bayam E, Çakmak EÖ, Candan Ö, İzgi İA, Kırma C. Fragmented QRS May Be Associated with Subclinical Left Ventricular Dysfunction in Patients with Hypertension.*
12. *Saab G, Whaley-Connell AT, Khanna R, Sowers JR. Therapy for the altered mineral metabolism of chronic kidney disease: implications for vascular calcification. Therapeutic Advances in Cardiovascular Disease. 2007 Dec;1(2):107-12.*
13. *Kempson SA, Lotscher M, Kaissling BR, Biber J, Murer HE, Levi MO. Parathyroid hormone action on phosphate transporter mRNA and protein in rat renal proximal tubules. American Journal of Physiology-Renal Physiology. 1995 Apr 1;268(4):F784-91.*
14. *Evenepoel P, Bover J, Torres PU. Parathyroid hormone metabolism and signaling in health and chronic kidney disease. Kidney International. 2016 Dec 1;90(6):1184-90.*
15. *Li D, Liu W, Huang H, Guo W, Diao Z, Chen X, Wangs W. Association between the risk of death and serum calcium, phosphate, and intact parathyroid hormone levels in older patients undergoing maintenance hemodialysis: a cohort study in Beijing. Therapeutic Advances in Endocrinology and Metabolism. 2021 Jun;12:20420188211025161.*
16. *Tan J. Forty-billion medical expenditure for dialysis patients in China. Kidney International. 2019 Aug 1;96(2):523.*
17. *Zhang Y, Yang H, Yang Z, Li X, Liu Z, Bai Y, Qian G, Wu H, Li J, Guo Y, Yang S. Could long-term dialysis vintage and abnormal calcium, phosphorus and iPTH control accelerate aging among the maintenance hemodialysis population?. Renal Failure. 2023 Dec 22;45(2):2250457.*
18. *Nasri H. Impact of diabetes mellitus on parathyroid hormone in hemodialysis patients. Journal of Parathyroid Disease. 2013 Feb 24;1(1):9-11.*
19. *Bollerslev J, Sjöstedt E, Rejnmark L. Cardiovascular consequences of parathyroid disorders in adults. InAnnales d'endocrinologie 2021 Jun 1 (Vol. 82, No. 3-4, pp. 151-157). Elsevier Masson.*
20. *McCullough PA, Chan CT, Weinhandl ED, Burkart JM, Bakris GL. Intensive hemodialysis, left ventricular hypertrophy, and cardiovascular disease. American Journal of Kidney Diseases. 2016 Nov 1;68(5):S5-14.*
21. *Maisel A. B-type natriuretic peptide levels: diagnostic and prognostic in congestive heart failure: what's next?. Circulation. 2002 May 21;105(20):2328-31.*
22. *Bellasi A, Raggi P, Rossi R, Rochira V, Stentarelli C, Zona S, Lattanzi A, Carli F, Mussini C, Guaraldi G. Intact parathyroid hormone levels are associated with increased carotid intima media thickness in HIV infected patients. Atherosclerosis. 2014 Dec 1;237(2):618-22.*
23. *Nasri H. Elevated serum parathyroid hormone is a heart risk factor in hemodialysis patients. J Parathyr Dis. 2013;1(1):13-4.*
24. *Nitta K, Kinugawa K. Diagnosis and treatment of heart failure with preserved ejection fraction in patients on hemodialysis. Renal Replacement Therapy. 2024 Oct 14;10(1):59.*
25. *Tajbakhsh A, Shirbache K, Shirbacheh A, Rabori VS, Chaman S, Zaremoghadam E. Cardiac effects of parathyroid hormone excess; an updated mini-review. Journal of Parathyroid Disease. 2023 Jul 29;11(1):e11218-.*
26. *Dhingra R, Sullivan LM, Fox CS, Wang TJ, D'Agostino RB, Gaziano JM, Vasan RS. Relations of serum phosphorus and calcium levels to the incidence of cardiovascular disease in the community. Archives of internal medicine. 2007 May 14;167(9):879-85.*
27. *Baloğlu İ, Selçuk NY, Evran H, Tonbul HZ, Türkmen K. Evaluation of Hemodialysis Adequacy: Correlation between Kt/V.*