

The comparison study of BMD between patients with normocalcemic and hypercalcemic hyperparathyroidism after kidney transplantation and its predictors

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Abstract

Introduction

Persistent hyperparathyroidism is a well-established complication following kidney transplantation with a significant impact on bone health. This condition can lead to bone thinning and an elevated risk of fractures. However, a comparison between normocalcemic and hypercalcemic hyperparathyroidism is less well-studied.

Objective

This study aims to compare bone mineral density (BMD) values among post-kidney transplant patients with normocalcemic hyperparathyroidism and hypercalcemic hyperparathyroidism. The secondary objective is to explore clinical factors that predict BMD values and BMD changes over time in this population.

Method

This cross-sectional study included patients who were more than 1 year post-kidney transplant and had persistent hyperparathyroidism documented more than 2 times. Medical history, laboratory results and BMD values were collected.

Results

This study included a total of 130 patients, 64 (49.2%) had normocalcemic hyperparathyroidism and 66 (50.8%) had hypercalcemic hyperparathyroidism. 72 patients underwent BMD measurement. Patients in the hypercalcemic hyperparathyroidism group had higher iPTH (141.1 vs 87.3 pg/mL, $P = 0.004$), higher corrected calcium (10.8 vs 9.7 mg/dL, $P < 0.001$), lower phosphate (2.7 vs. 3.2 mg/dL, $P < 0.001$), and longer dialysis vintage (6.4 vs 4.3 years, $P = 0.010$) when compared to those in the normocalcemic group. Mean BMD values in the hypercalcemic vs normocalcemic groups at all sites were similar. Female sex and longer dialysis vintage independently predicted lower BMD at multiple sites. In longitudinal analysis ($n = 19$), BMD changes did not differ significantly between the two groups at any skeletal site, although patients with hypercalcemia showed a trend toward greater cortical bone loss.

Conclusion

Among post-kidney transplant patients with persistent hyperparathyroidism, BMD and BMD changes were similar between those with hypercalcemic and normocalcemic hyperparathyroidism. Dialysis vintage and female sex were primary predictors of lower BMD.

Background

Currently, there is an increasing number of patients with end-stage renal disease, creating significant global public health challenges. The most effective treatment for end-stage renal disease at present is

kidney transplantation⁽¹⁾. With the increasing number of patients undergoing kidney transplantation⁽²⁾, persistent hyperparathyroidism has become a common issue, affecting up to 50% of post-kidney transplant cases⁽³⁾. This includes both hypercalcemic and normocalcemic cases. The pathogenesis of persistent hyperparathyroidism after kidney transplant, despite a well-functioning kidney allograft, is attributed to the slow turnover rate of parathyroid cells and the persistence of hyperplastic glands⁽⁴⁾.

Persistent hyperparathyroidism has various impacts on organs and systems, including the kidneys, leading to the formation of kidney stones⁽⁵⁾ and potential deterioration of transplanted kidney function^(6, 7). It also disrupts mineral homeostasis, resulting in high blood calcium levels, low blood phosphate levels^(8, 9), and significantly increased mortality rates in patients with persistent hyperparathyroidism⁽¹⁰⁾.

The skeletal system is one of the well-established organs affected by hyperparathyroidism, leading to reduced bone density, bone thinning^(11–13), and increased fracture risk⁽¹⁴⁾ particularly in patients with markedly elevated PTH levels (> 150 pg/mL)⁽¹⁵⁾. Furthermore, kidney transplant recipients receiving immunosuppressive medications, particularly corticosteroids cause a deterioration in bone health, characterized by bone loss, decreased bone mineral density (BMD), and an increased risk of fracture⁽¹³⁾. These findings highlight the significant of persistent hyperparathyroidism following kidney transplantation and its bone associated complications. However, most of the available data focus on patients with persistent hyperparathyroidism and high serum calcium levels. Studies on patients with persistent hyperparathyroidism and normal blood calcium levels are limited⁽¹⁶⁾, despite their relatively high prevalence. A previous study found that as high as 45% of patients with normal blood calcium levels after kidney transplantation had high levels of parathyroid hormone⁽¹⁷⁾. Therefore, there is currently a lack of data on disease progression, such as changes in bone mass, fracture risk, kidney deterioration, or mortality rates, and it remains unclear how disease progression or prognostic factors differs in patients with hypercalcemic versus normocalcemic persistent hyperparathyroidism after kidney transplantation.

Methods

Objective

The primary objective is to compare BMD of post-kidney transplant patients with normocalcemic hyperparathyroidism and hypercalcemic hyperparathyroidism. The secondary objective is to investigate clinical factors that predict BMD and BMD changes in post-kidney transplant patients with normocalcemic and hypercalcemic hyperparathyroidism.

Study Patients

We conducted a cross-sectional study at Ramathibodi Hospital, a tertiary referral center in Bangkok, Thailand. Eligible patients were included if they met all of the following criteria at the time of enrollment: kidney transplant performed more than 1 year prior, age over 18 years, and intact parathyroid hormone

(iPTH) level exceeding 65 pg/mL on at least two occasions, with a minimum interval of 2 weeks between each test.

Exclusion criteria comprised an estimated glomerular filtration rate (eGFR) below 60 ml/min/1.73 m², pregnancy within 1 year of BMD measurement, and other bone-associated diseases such as Cushing's disease, Paget's disease, and thyroid stimulating hormone (TSH) level less than 0.3 uIU/mL. A flowchart of subject recruitment is presented in Fig. 1. We reviewed the database from 1 January 2010 to 31 December 2023.

This study was approved by the Institutional Review Board and by the Committee on Human Rights Related to Research Involving Human Subjects, Faculty of Medicine, Ramathibodi Hospital, Mahidol University. Informed consent was waived by the IRB due to the nature of the study.

Defining terms

Patients were divided into two groups according to corrected calcium levels. The mean calcium levels were calculated by averaging calcium levels from 6 months before and after recruitment. Patients with hypercalcemic hyperparathyroidism had mean corrected serum calcium levels of ≥ 10.2 mg/dL, whereas those with normocalcemic hyperparathyroidism had mean corrected serum calcium levels of < 10.2 mg/dL.

Alcohol drinking was defined as consuming 3 or more units of standard alcohol drinking daily. A unit of standard alcohol drinking was 10 grams of alcohol.

Biochemical measurement

All laboratory tests were assessed at the central laboratory of Ramathibodi Hospital. The latest value of each laboratory results before recruitment were used in the analysis.

Corrected calcium used the following formula: corrected calcium (mg/dL) = measured total calcium (mg/dL) + $0.8 \times (4.0 - \text{serum albumin [g/dL]})$

The average levels of calcium obtained six months before and after BMD measurement were used to investigate the association between those values and BMD and BMD changes.

BMD assessment

Patients underwent BMD assessment at the lumbar spine (L1–L4 vertebrae), hip (femoral neck (FN) and total hip (TH)) and/or 1/3 radius. All measurement procedures operating in the fast array mode were performed by technologists using the same Hologic Discovery W DXA scanner (Hologic, Bedford, MA) on all female patients and GE-Lunar iDXA scanner (GE/Lunar, Madison, WI) on all male patients. Quality

assurance procedures using a spine phantom were performed daily. Lumbar spine (L1-L4), TH, FN, and one-third radius T-scores were analyzed using Asian population reference databases supplied by the manufacturer.

BMD change used the following formula: $\text{BMD change (g/cm}^2\text{/year)} = [\text{BMD latest value (g/cm}^2\text{)} - \text{BMD earliest value (g/cm}^2\text{)}] \div \text{duration between the BMD measurements (years)}$.

Thailand-specific Fracture Risk Assessment Tool (FRAX) was used to calculate the predicted 10-year probability of hip fracture and major osteoporotic fracture (MOF).

Statistical Analysis

Continuous variables were reported as the mean with standard deviation (SD) for normal distribution and median with interquartile range (IQR) for non-normal distribution and categorical variables were reported as number and percentage. The differences in variables between patients with normocalcemic and hypercalcemic hyperparathyroidism were determined by student's t-test or Mann–Whitney U test for continuous variables and Chi-square or Fisher's exact test for categorical variables. Univariate and multivariate linear regression analyses were used to determine the relationship between all variables and both the recent (the latest) BMD values and changes in BMD values.

A sample size calculation was performed by using two independent means.

$$n = (Z_{\alpha/2} + Z_{\beta})^2 \cdot 2 \cdot \sigma^2 / d^2$$

where $Z_{\alpha/2}$ is the critical value of the normal distribution at $\alpha/2$ (for a confidence level of 95%, α is 0.05 and the critical value is 1.96), Z_{β} is the critical value of the normal distribution at β . (for a power of 80%, β is 0.2 and the critical value is 0.84), σ^2 is the population variance, and d is the group difference.

With a desired power of 80% and a confidence interval of 95%, a minimum sample size of 21 patients per group was estimated based on anticipated effect sizes and standard deviations observed in previous studies^(16, 18).

All statistical analyses were performed using STATA version 18.0 (StataCorp LLC; College Station, TX, USA). *P* value of 0.05 was set as the threshold for statistical significance.

Results

Baseline Characteristic

A total of 706 kidney transplant recipients with persistent elevation of iPTH more than one year after transplantation were identified. Of these, 130 patients were included: 64 patients (49.2%) had normocalcemic hyperparathyroidism and 66 patients (50.8%) had hypercalcemic hyperparathyroidism. The mean age of the study population was 47.9 ± 11.2 years, with a mean BMI of 24.2 ± 4.8 kg/m², and

36.2% of the participants were female. The median time since transplantation was 89.5 months (range, 66–124 months), and the median dialysis vintage prior to transplantation was 5 years (range, 3–7 years). The median iPTH and the mean corrected calcium were 111.8 pg/mL (84.8–173.8) and 10.2 ± 0.7 mg/dL, respectively.

Of the 130 included patients, 72 patients (55.4%) underwent BMD measurement at least once during follow-up. Baseline characteristics were similar between patients who did and did not undergo BMD assessment. Among the 72 patients with BMD data, 33 patients (45.8%) had normocalcemic hyperparathyroidism and 39 patients (54.2%) had hypercalcemic hyperparathyroidism. The mean age in this group was 49.5 ± 11.3 years, the mean BMI was 25.1 ± 4.8 kg/m², and 40.3% of the participants were female (Table 1).

Table 1
Baseline characteristic and bone mineral density of participants with stratified calcium levels

Characteristic	Total (n = 72)	Normocalcemic hyperPTH (n = 33)	Hypercalcemic hyperPTH (n = 39)	P value
Age (years)	49.5 (11.3)	48.1 (11.9)	50.7 (10.8)	0.322
Female, n (%)	29 (40.3)	14 (42.4)	15 (38.5)	0.733
BMI (kg/m ²)	25.1 (4.8)	26.2 (4.9)	24.1 (4.5)	0.051
Previous fracture, n (%)	6 (8.3%)	1 (3.0%)	5 (12.8%)	0.134
Parental hip fracture, n (%)	8 (11.1%)	2 (6.1%)	6 (15.4%)	0.210
Smoking, n (%)	1 (1.4)	0 (0%)	1 (2.6%)	0.354
Alcohol drinking, n (%)	4 (5.6%)	1 (3.0%)	3 (7.7%)	0.390
Time after KT (months)	107.5 (80.0-156.0)*	108.0 (81.0-133.0)*	107.0 (78.0-169.0)*	0.844
Dialysis vintage (years)	5.4 (3.5)	4.3 (3.7)	6.4 (3.0)	0.010
Laboratory results mean (SD)				
Corrected calcium (mg/dL)	10.3 (0.7)	9.7 (0.4)	10.8 (0.5)	< 0.001
Albumin (g/L)	40.7 (3.2)	41.1 (3.1)	40.4 (3.4)	0.454
iPTH (pg/mL)	114.7 (87.0-186.9)*	87.3 (80.9-123.2)*	141.1 (108.9-229.5)*	0.004
Phosphate (mg/dL)	2.9 (0.5)	3.2 (0.5)	2.7 (0.5)	< 0.001
25(OH)D: 25-hydroxyvitamin D level, BMI: body mass index, CaCO ₃ : calcium carbonate, DLP: dyslipidemia, FRAX: fracture risk assessment tool, GFR: glomerular filtration rate, HT: hypertension, hyperPTH: hyperparathyroidism, KT: kidney transplant, MI: myocardial infarction, MOF: major osteoporotic fracture, iPTH: intact parathyroid hormone, RA: rheumatoid arthritis, T2DM: type2 diabetes mellitus				
^a n = 52, ^b n = 23, ^c n = 29, ^d n = 62, ^e n = 29, ^f n = 33, ^g n = 67, ^h n = 34				
*Median (IQR) for continuous variables				

Characteristic	Total (n = 72)	Normocalcemic hyperPTH (n = 33)	Hypercalcemic hyperPTH (n = 39)	P value
GFR (ml/min/1.73 m ²)	80.8 (14.2)	81.0 (15.7)	80.7 (13.0)	0.929
25(OH)D (ng/mL)	30.6 (11.9) ^a	29.1 (11.3) ^b	31.7 (12.5) ^c	0.434
Drug-related fracture risk, n (%)				
Alendronate	18 (25.0%)	5 (15.2%)	13 (33.3%)	0.076
CaCO ₃	5 (6.9%)	2 (6.1%)	3 (7.7%)	0.786
Vitamin D	13 (18.1%)	6 (18.2%)	7 (17.9%)	0.980
Underlying disease, n (%)				
T2DM	23 (31.9%)	10 (30.3%)	13 (33.3%)	0.783
HT	67 (93.1%)	31 (93.9%)	36 (92.3%)	0.786
DLP	42 (58.3%)	20 (60.6%)	22 (56.4%)	0.719
Stroke	3 (4.2%)	2 (6.1%)	1 (2.6%)	0.459
MI	4 (5.6%)	3 (9.1%)	1 (2.6%)	0.228
RA	1 (1.4%)	1 (3.0%)	0 (0.0%)	0.274
BMD mean (SD), g/cm²				
L1-L4	0.930 (0.179)	0.938 (0.182)	0.924 (0.178)	0.750
Femoral neck	0.709 (0.145)	0.705 (0.100)	0.711 (0.177)	0.853
Total hip	0.794 (0.159)	0.789 (0.112)	0.797 (0.192)	0.831
25(OH)D: 25-hydroxyvitamin D level, BMI: body mass index, CaCO ₃ : calcium carbonate, DLP: dyslipidemia, FRAX: fracture risk assessment tool, GFR: glomerular filtration rate, HT: hypertension, hyperPTH: hyperparathyroidism, KT: kidney transplant, MI: myocardial infarction, MOF: major osteoporotic fracture, iPTH: intact parathyroid hormone, RA: rheumatoid arthritis, T2DM: type2 diabetes mellitus				
^a n = 52, ^b n = 23, ^c n = 29, ^d n = 62, ^e n = 29, ^f n = 33, ^g n = 67, ^h n = 34				
*Median (IQR) for continuous variables				

Characteristic	Total (n = 72)	Normocalcemic hyperPTH (n = 33)	Hypercalcemic hyperPTH (n = 39)	P value
1/3 radius	0.663 (0.120) ^d	0.674 (0.125) ^e	0.653 (0.116) ^f	0.497
T-score				
L1-L4	-1.2 (-2.0 - -0.2)	-1.1 (-1.9 - -0.1)	-1.3 (-2.3 - -0.3)	0.560
Femoral neck	-1.4 (-2.1 - -0.5)	-1.3 (-1.9 - -0.9)	-1.3 (-2.2 - -0.5)	0.811
Total hip	-0.9 (-1.8 - -0.1)	-0.9 (-1.6 - -0.5)	-1.1 (-1.9 - -0.0)	0.799
1/3 radius	-1.9 (-2.9 - -1.0) ^d	-1.5 (-2.8 - -0.5) ^e	-2.4 (-2.9 - -1.5) ^f	0.236
FRAX median (IQR) %				
MOF	2.8 (1.9–4.5) ^g	2.6 (1.9–4.2)	3.3 (2.0-5.8) ^h	0.107
Hip	0.7 (0.2–1.6) ^g	0.6 (0.2–1.1)	0.8 (0.2–1.9) ^h	0.086
25(OH)D: 25-hydroxyvitamin D level, BMI: body mass index, CaCO ₃ : calcium carbonate, DLP: dyslipidemia, FRAX: fracture risk assessment tool, GFR: glomerular filtration rate, HT: hypertension, hyperPTH: hyperparathyroidism, KT: kidney transplant, MI: myocardial infarction, MOF: major osteoporotic fracture, iPTH: intact parathyroid hormone, RA: rheumatoid arthritis, T2DM: type2 diabetes mellitus				
^a n = 52, ^b n = 23, ^c n = 29, ^d n = 62, ^e n = 29, ^f n = 33, ^g n = 67, ^h n = 34				
*Median (IQR) for continuous variables				

Patients with hypercalcemic hyperparathyroidism had a longer dialysis vintage compared with those with normocalcemic hyperparathyroidism (6.4 vs 4.3 years, $P = 0.010$). They also had higher iPTH levels (141.1 vs 87.3 pg/mL, $P = 0.004$) and higher corrected calcium levels (10.8 vs 9.7 mg/dL, $P < 0.001$). In contrast, serum phosphate levels were lower in the hypercalcemic group (2.7 vs 3.2 mg/dL, $P < 0.001$). Other clinical characteristics and laboratory parameters were comparable between the two groups. Additionally, the prevalence of bone-related comorbidities and the use of medications that affect bone metabolism were similar (Table 1).

BMD value and predictors for BMD values

The mean recent BMD values were 0.930 g/cm² at the lumbar spine (L1-L4), 0.709 g/cm² at the femoral neck, 0.794 g/cm² at the total hip, and 0.663 g/cm² at the one-third radius. There were no differences in BMD value and T-score at any skeletal site between patients with hypercalcemic and normocalcemic hyperparathyroidism (Table 1). Similarly, FRAX estimates for both hip fractures and major osteoporotic fractures (MOFs) did not differ between the two groups.

To further explore factors associated with recent BMD, univariate and multivariate linear regression analyses were performed, as shown in Table 2 (n = 72). Overall, female sex was consistently associated with lower BMD across multiple skeletal sites, including the lumbar spine (L1-L4), femoral neck, total hip, and one-third radius. This association remained significant after adjusting for multiple variables. Longer dialysis vintage was associated with lower BMD at all skeletal sites in univariate analysis; however, after multivariate adjustment, the association remained significant only at the femoral neck, total hip, and one-third radius, but not at the lumbar spine (L1–L4). For BMI, higher BMI was associated with higher BMD at weight-bearing sites, specifically at the femoral neck and total hip, in univariate analysis. However, this association did not persist in multivariate analysis. Vitamin D supplement showed a weak inverse association with total hip BMD in univariate analysis but was not retained in the final multivariate model. Similarly, calcium carbonate use was associated with lower BMD at the one-third radius in univariate analysis, but this association was not included in the multivariate model.

Table 2
Univariate and multivariate linear regression analysis of clinical risk factors associated with BMD values
(n = 72)

Site	Univariate linear regression		Multivariate linear regression	
	β (95% CI)	P value	β (95% CI)	P value
L1-L4				
Female	-0.154 (-0.232 - -0.076)	< 0.001	-0.154 (-0.232 - -0.076)	< 0.001
Dialysis vintage	-0.010 (-0.022–0.001)	0.093		
Vitamin D	-0.100 (-0.208 - -0.006)	0.066		
Femoral neck				
Female	-0.099 (-0.165 - -0.033)	0.004	-0.096 (-0.159 - -0.032)	0.003
BMI (kg/m ²)	0.007 (0.000–0.014)	0.038		
Dialysis vintage (years)	-0.012 (-0.022 - -0.002)	0.011	-0.012 (-0.021 - -0.002)	0.010
Total hip				
Female	-0.102 (-0.175 - -0.030)	0.006	-0.099 (-0.169 - -0.030)	0.006
BMI	0.008 (0.000–0.015)	0.036		
Dialysis vintage (years)	-0.014 (-0.024 - -0.003)	0.012	-0.013 (-0.023 - -0.003)	0.011
Vitamin D	-0.085 (-0.181–0.010)	0.079		
1/3 radius^a				
Female	-0.120 (-0.174 - -0.066)	< 0.001	-0.119 (-0.172 - -0.067)	< 0.001
Dialysis vintage (years)	-0.008 (-0.017–0.001)	0.070	-0.008 (-0.016–0.000)	0.041
CaCO ₃	-0.114 (-0.223 - -0.006)	0.040		
BMI: body mass index, CaCO ₃ : calcium carbonate, DLP: dyslipidemia, FRAX: fracture risk assessment tool, MOF: major osteoporotic fracture, iPTH: intact parathyroid hormone				
^a n = 62				

BMD changes and predictors for BMD changes

Among the 72 patients who underwent BMD measurements, 19 had repeat assessments and were included in the longitudinal analysis. Annual changes in BMD at each skeletal site (g/cm²/year) are shown in Supplementary Table 1. Overall, BMD changes did not differ significantly between patients with hypercalcemic and normocalcemic hyperparathyroidism at any skeletal site.

Discussion

This study demonstrates that the prevalence of normocalcemic hyperparathyroidism was approximately half among post-kidney transplant patients with persistent hyperparathyroidism. Additionally, the analysis revealed no differences in BMD between the two groups at the lumbar (L1-L4), hip, and 1/3 radius. Consistent with traditional and CKD-specific risk factors, female sex and longer dialysis vintage were identified as the significant predictors of lower BMD values. Of interest, data of BMD changes in a subset of the participants revealed that higher corrected calcium levels may be link to cortical bone loss at femoral neck and 1/3 radius. We hypothesized that individuals who have undergone kidney transplantation and have hypercalcemic hyperparathyroidism would experience more severe bone loss than those who have normocalcemia. The study with a larger sample size is required to validate these preliminary results.

Our findings on the prevalence of normocalcemic and hypercalcemic hyperparathyroidism are consistent with those previous reported^(9, 15, 17, 19, 20) which prevalence of normocalcemic hyperparathyroidism is about 20–50% while prevalence of hypercalcemic hyperparathyroidism is about 35–60% in persistent hyperparathyroidism after kidney transplant. However, our results regarding BMD values differ from those of Setterberg et al.⁽¹⁶⁾, who observed lower BMD in patients with hypercalcemic hyperparathyroidism compared to those with normocalcemia. Our study revealed similar BMD values between the two groups. The observed differences in BMD values between our study and that of Setterberg et al.⁽¹⁶⁾ may be due to variations in ethnicity and sex distribution. Our study included a lower proportion of females (40%) compared to Setterberg et al.⁽¹⁶⁾, who reported a 50% female representation. Since female is a known predictor of lower BMD values, this difference in patient demographics might partially explain the observed discrepancies in BMD findings between the two studies. Additionally, these discrepancies may also be influenced by variations in other underlying medical disorders or medications related to bone health.

Although annual BMD changes did not differ significantly between the two groups, patients with hypercalcemia exhibited a numerical tendency toward more pronounced BMD reduction at the femoral neck and total hip compared to normocalcemic individuals. Cortical-predominant bone is particularly susceptible to hyperparathyroidism-mediated turnover; however, the lack of statistical significance at these sites and the 1/3 radius was likely constrained by the limited sample size (n = 19). These observations should therefore be considered hypothesis-generating.

Parathyroid hyperplasia is a common finding in patients with end-stage renal disease. Even following a kidney transplant, the hyperplastic glands may not always resolved. Consequently, optimal iPTH levels in

post-kidney transplants patients is not well documented. Previous study reported that mean iPTH after kidney transplant ranged about 65–125 pg/mL^(17, 21, 22). In addition, higher iPTH related with long duration of dialysis vintage, high pre-transplant iPTH and calcium levels, cinacalcet use and parathyroid gland size^(22, 23). It is possible that mildly elevated iPTH with normal calcium levels (normocalcemic hyperparathyroidism) would be a normal response. Conversely, high calcium levels with elevated iPTH would indicate that the hyperplastic glands have not resolved, and this elevated iPTH levels may cause high bone remodeling and significant bone loss in these particular patients. Therefore, in persistent hyperparathyroidism after kidney transplantation, calcium levels would guide the optimal management in maintaining bone health.

This study was one of the first trial to study about the BMD changes and clinical predictors of bone health in normocalcemic and hypercalcemic hyperparathyroidism after kidney transplant. Despite its strength, this study also had some limitations. First, the number of subjects was limited in single center. There were 130 patients who participated in the study, but only 72 completed the BMD assessment, with only 19 patients having data on BMD changes. Second, this is retrospective cross-sectional study and might have some selection biases and confounding factors. For example, clinician possibly trended to measure BMD in patients with clinical clues such as history of fractures or patient requested.

In summary, our study provides preliminary findings into understanding bone health in post-kidney transplant patients, which are crucial for managing their mineral and bone disorders. Although the BMD values of the hypercalcemic and normocalcemic hyperparathyroidism group were not different, patients with sustained hypercalcemia may warrant closer monitoring for potential accelerated cortical bone loss. To validate these preliminary results, a larger sample size prospective study is required. Fracture should also be considered as an outcome of interest in future research. The ultimate goal is to optimize management strategies to reduce bone fractures and improve long-term bone associated outcomes in this specific group of patients. In patients with persistent hyperparathyroidism, monitoring calcium levels may be essential in order to determine the optimal management for maintaining bone health.

Conclusions

We found that among persistent hyperparathyroidism after kidney transplant patients, BMD results of hypercalcemic and normocalcemic hyperparathyroidism were similar. Lower BMD values were independently predicted by female sex and longer pre-transplant dialysis vintage. The preliminary finding suggests that higher corrected calcium levels may be associated with greater bone loss in cortical-predominated bone, which warrants validation in larger, long-term prospective cohorts.

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board and the Committee on Human Rights Related to Research Involving Human Subjects, Faculty of Medicine Ramathibodi Hospital, Mahidol University (COA. MURA2023/45). Due to the nature of the study and the use of anonymized clinical data, the requirement for informed consent was waived by the Institutional Review Board.

Consent for publication

Not applicable.

Availability of data and materials

Data will be made available on request.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

G.S. and H.N. conceived and designed the study, analyzed and interpreted the patient data, and drafted the main manuscript text. S.D. contributed to the study design, interpretation of data, and critical revision of the manuscript. C.S., S.A., N.S., and R.K. acquired the clinical and radiological data and contributed to participant evaluations. B.O. contributed to the data analysis, supervised the project, and critically revised the manuscript. All authors reviewed and approved the final manuscript.

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Data Availability

The datasets generated and/or analysed during the current study are not publicly available due to patient privacy and ethical restrictions, but are available from the corresponding author on reasonable request.

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Figures

Figure 1. Study flow chart

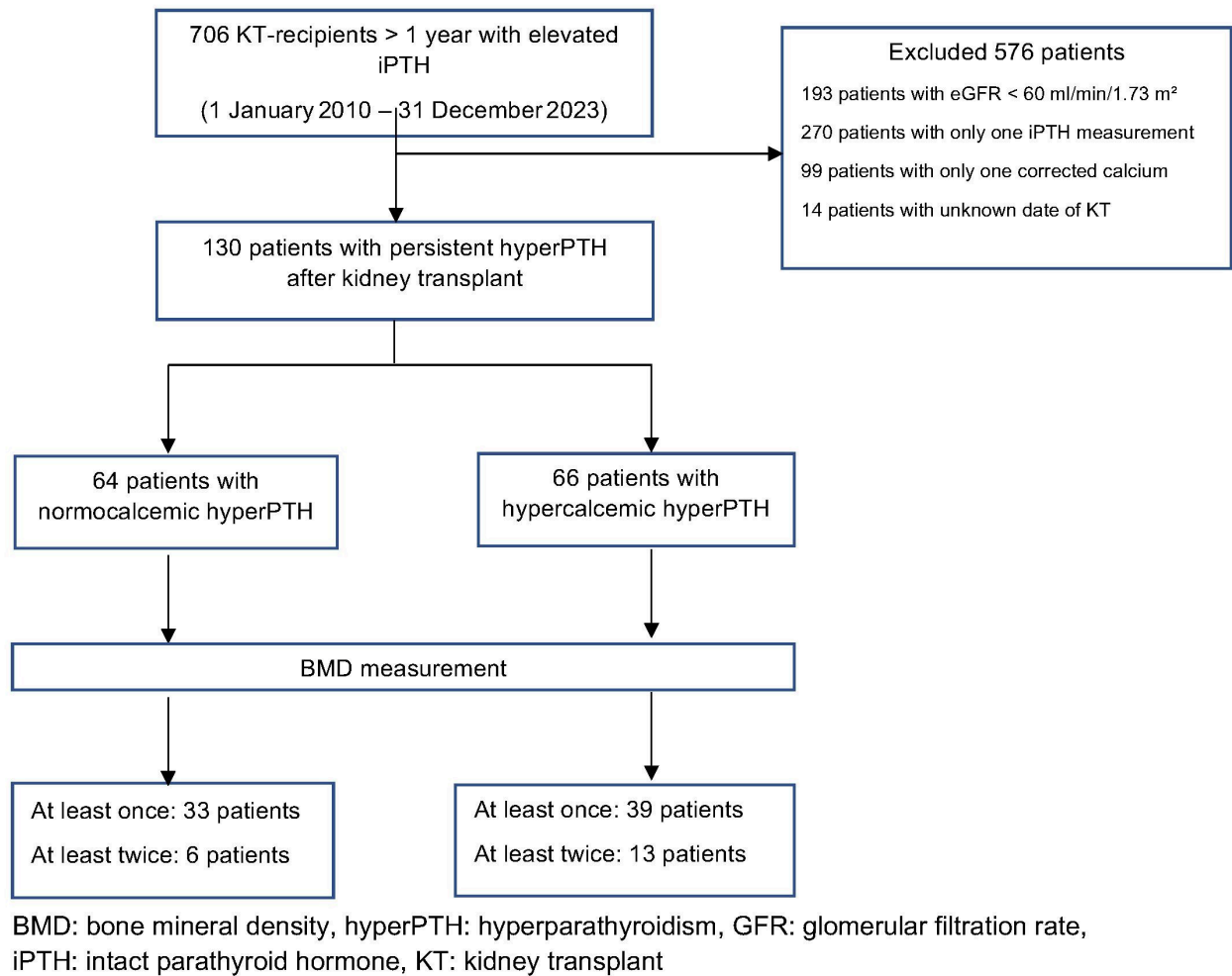


Figure 1

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