ORIGINAL ARTICLE

Associated Factors of Long-Term Severe Hypocalcemia after Total Parathyroidectomy with Forearm Auto-Transplantation in Patients with Renal Hyperparathyroidism

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Objective: To identify patient-specific factors associated with long-term severe hypocalcemia following total parathyroidectomy with forearm auto-transplantation (TPTx+AT) in patients with renal hyperparathyroidism.

Materials and Methods: A retrospective study was conducted on 70 patients who underwent TPTx+AT at Thammasat University Hospital between January 2013 and October 2023. The clinical and laboratory data of these patients were analyzed. Multivariable logistic regression was used to identify independent predictors of long-term severe hypocalcemia. Analyzed variables included age, preoperative serum calcium, parathyroid hormone, alkaline phosphatase, clinical of pruritus, bone pain, and history of postoperative intravenous calcium supplementation.

Results: Long-term severe hypocalcemia occurred in 26 of 70 patients (37.1%). Logistic regression revealed that a history of intravenous calcium administration was significantly associated with increased odds of long-term severe hypocalcemia (OR 10.29, 95% CI 1.70 to 60.32, p=0.01). Younger age was also identified as a significant predictor (OR 0.93 per year, 95% CI 0.88 to 0.98, p=0.01).

Conclusion: Younger age and postoperative intravenous calcium supplementation are independent predictors of long-term severe hypocalcemia following TPTx+AT. Identifying high-risk patients may facilitate proactive postoperative monitoring and individualized calcium management.

Keywords: Hyperparathyroidism; Secondary; Parathyroidectomy; Transplantation; Autologous; Hypocalcemia; Postoperative complications; Renal insufficiency; Chronic; Calcium

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Secondary and tertiary hyperparathyroidism (also referred to as renal hyperparathyroidism) are significant complications of chronic kidney disease (CKD). Severe renal hyperparathyroidism can lead to refractory pruritus, bone pain, muscle weakness, progressive soft tissue calcification, kidney stones, constipation, spontaneous long bone fractures, psychosis, and even an increased risk of death[1]. Parathyroidectomy becomes necessary in approximately 15% of patients after 10 years of dialysis and in about 38% of patients after 20 years of ongoing dialysis therapy[2]. Successful parathyroidectomy helps lower serum parathyroid hormone (PTH) levels and alleviates clinical symptoms such as bone pain and pruritus[3,4].

Hypocalcemia is a common postoperative complication of parathyroidectomy. In patients with renal hyperparathyroidism, the incidence of post-parathyroidectomy hypocalcemia ranges between 72% and 97%, despite frequent monitoring of serum calcium levels and adjustments in calcium and vitamin D supplementation[5].

Hypocalcemia following parathyroidectomy can be classified by severity. Mild hypocalcemia is defined as a corrected serum calcium level between 7.5 and 8.5 mg/dL, often managed with oral calcium supplementation alone. Severe hypocalcemia is defined as a corrected serum calcium level of 7.5 mg/dL or below, typically requiring high-dose oral calcium supplementation exceeding 2 g of elemental calcium per day, and in acute settings, intravenous (IV) calcium administration. When severe hypocalcemia persists beyond one year postoperatively despite

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adequate supplementation, it is considered equivalent to permanent hypoparathyroidism, a chronic and debilitating condition that significantly impairs patients' quality of life and necessitates long-term medical management.

Preoperative serum calcium, alkaline phosphatase (ALP), and PTH levels are known risk factors for severe postoperative hypocalcemia in short-term studies (within 72 hours post-surgery)[5-8]. However, no previous studies have focused on the long-term complications of parathyroidectomy, particularly severe hypocalcemia, in Thailand.

Materials and Methods

This was a single-center, retrospective cross-sectional study conducted at Thammasat University Hospital, Thailand. The study included patients with renal hyperparathyroidism who underwent total parathyroidectomy with forearm auto-transplantation (TPTx+AT) between January 2013 and October 2023. Retrospective data were collected from the medical records.

Clinical data included gender, age, comorbidities, body weight, height, body mass index (BMI), mode of renal replacement therapy, duration of dialysis, bone pain, severe pruritus, bone deformity or fracture, preoperative medications, discharge medications, postoperative 6-month medications, postoperative 1-year medications, and relevant laboratory data (corrected calcium, phosphate, ALP, and serum PTH levels at preoperative, immediate postoperative, 6-month postoperative, and 1-year postoperative time points).

Inclusion criteria were adult patients with renal hyperparathyroidism who underwent TPTx+AT and experienced a reduction in postoperative PTH levels at 24 hours of more than 80% compared with preoperative levels. Exclusion criteria included non-dialysis CKD patients, patients who had undergone kidney transplantation, and those with incomplete clinical data.

This study was approved by the Human Research Ethics Committee of Thammasat University No. 1 (Faculty of Medicine) (Certificate of Approval No. MTU-EC-SU-0-210/67). The requirement for informed consent was waived due to the retrospective nature of the study.

The primary outcome was to identify factors associated with postoperative long-term hypocalcemia following TPTx+AT. The secondary outcome was to determine the incidence of postoperative long-term severe hypocalcemia in this population.

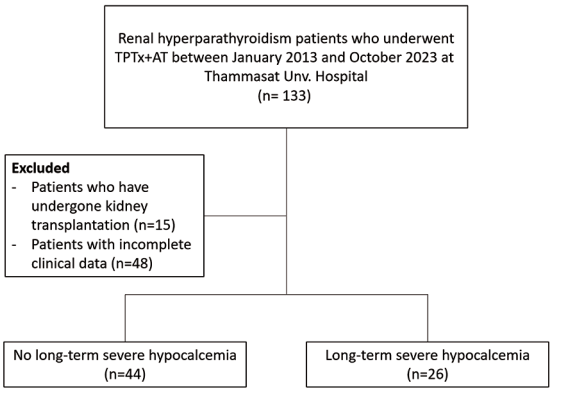

Figure 1. The flow diagram of the study identification and inclusion and exclusion process.

Long-term severe hypocalcemia was defined as a serum calcium level less than or equal to 7.5 mg/dL, requiring elemental calcium supplementation of more than 2 g/day and persisting for at least one year postoperatively. This condition was considered equivalent to permanent hypocalcemia[9].

Statistical Analysis: continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on data distribution. Categorical variables were presented as proportions and percentages. Comparisons between groups were performed using the chi-square test for categorical variables and the dependent t-test for continuous variables. A p-value of less than 0.1 was used as a cutoff for multivariable logistic regression, and a p-value of less than or equal to 0.05 was considered statistically significant. All statistical analyses were conducted using Stata/SE version 17 (StataCorp LLC, College Station, TX, USA).

Results

One hundred thirty-three patients with renal hyperparathyroidism who underwent TPTx+AT between January 2013 and October 2023 were initially included. Of these, 15 patients who had undergone kidney transplantation and 48 patients with incomplete clinical data were excluded, leaving 70 patients for analysis. Among these, 26 patients developed long-term severe hypocalcemia (Figure 1). The demographic and clinical characteristics of the patients are shown in Table 1.

The incidence of long-term severe hypocalcemia after TPTx+AT in this study was 37.1% (Table 2). Patients in the hypocalcemia group were significantly younger and more likely to be less than or equal to

Table 1. Demographic and clinical characteristics of the study

Characteristics	All patients (n=70)
Female sex; n (%)	37 (52.9)
Age at time of surgery (years); mean±SD	49.79±14.04
BMI; mean±SD	23.22±3.64
Mode of renal replacement therapy; n (%)	
Hemodialysis	64 (91.4)
Peritoneal dialysis	6 (8.6)
Duration of dialysis (years); mean±SD	6.89±4.14
Underlying renal disease; n (%)	
Diabetes mellitus	12 (11.14)
Hypertension	15 (21.43)
Chronic glomerulonephritis	7 (10.00)
Congenital renal disease	1 (1.42)
Renal cystic disease	4 (5.71)
Unknown	22 (31.43)
Symptoms; n (%)	
Bone pain	46 (65.71)
Fracture	6 (8.57)
Pruritis	14 (20.00)
Preoperative laboratory testing	
Corrected calcium (mg/dL); mean±SD	9.8±0.87
Phosphate (mg/dL); mean±SD	5.03±2.03
ALP (IU/L); median (IQR25-75)	357 (151 to 460)
PTH (pg/mL); median (IQR25-75)	2,066 (1,566 to 2,545)
History of IV calcium; n (%)	35 (50.00%)
Postoperative hypocalcemic symptoms; n (%)	5 (7.14)

BMI=body mass index; ALP=alkaline phosphatase; PTH=parathyroid hormone; IV=intravenous; SD=standard deviation; IQR=interquartile range

45 years of age (61.5% versus 27.3%, p=0.006). There were no significant differences in sex distribution, BMI, dialysis modality, dialysis duration, or underlying renal disease between the groups. Preoperatively, the hypocalcemia group had significantly lower serum corrected calcium levels (9.55±0.84 versus 10.02±0.85 mg/dL, p=0.032). Median preoperative PTH levels were higher in the hypocalcemia group (2,294 pg/mL versus 1,946 pg/mL), but this did not reach statistical significance (p=0.079). Postoperative serum corrected calcium, phosphate, and PTH levels at 24 hours did not differ significantly between groups. In addition, the history of IV calcium use was more frequent in the long-term severe hypocalcemia group (76.9% versus 34.1%, p=0.001).

In the multivariate logistic regression model (Table 3), two variables remained independently associated with long-term severe hypocalcemia. Age less than or equal to 45 years was a strong predictor (OR 30, 95% CI 2.24 to 543.11, p=0.01), as was a history of IV calcium administration (OR 10.71, 95% CI 1.74 to 65.71, p=0.01).

Discussion

This study investigated the incidence and associated factors of long-term severe hypocalcemia following TPTx+AT in renal hyperparathyroidism patients. The incidence of long-term severe hypocalcemia in this study was 37.1%, which is clinically significant and highlights the importance of understanding risk factors to optimize postoperative management.

Two independent predictors of long-term severe hypocalcemia were identified: younger age and a history of IV calcium administration. Younger patients were significantly more likely to develop long-term severe hypocalcemia compared with older patients. One plausible explanation is that younger patients often exhibit higher bone turnover, resulting in a more pronounced calcium shift from the circulation into bone following the abrupt postoperative decline in PTH levels. This mechanism is consistent with the pathophysiology of hungry bone syndrome (HBS), a well-described cause of profound hypocalcemia following parathyroidectomy, particularly in patients with severe renal hyperparathyroidism. Previous studies[8] have also demonstrated that higher bone remodeling activity correlates with the severity of postoperative hypocalcemia in the short term (within 72 hours).

The history of IV calcium supplementation was also significantly associated with the development of long-term severe hypocalcemia. This finding may reflect a preexisting calcium imbalance or more severe disease, both of which predispose patients to persistent postoperative hypocalcemia[4]. IV calcium administration is a common therapeutic response to acute HBS, and its association with long-term severe hypocalcemia in this study suggests that patients requiring early aggressive calcium replacement may continue to have dysregulated calcium homeostasis for prolonged periods. However, as the diagnosis of HBS was not systematically recorded in the medical charts, the authors were unable to establish a definitive causal relationship.

Interestingly, preoperative biochemical markers such as serum calcium, PTH, and ALP levels were not independently associated with long-term severe hypocalcemia in the multivariate analysis. This contrasts with previous short-term studies[5,7,8,10], which identified high preoperative calcium, PTH, and ALP as predictors of early postoperative

Table 2. Comparison of clinical data of patients with and without long-term severe hypocalcemia

Characteristics	Long-term severe hypocalcemia (n=70)		p-value
	Yes (n=26)	No (n=44)	
Female sex; n (%)	16 (61.54)	21 (47.73)	0.21
Age at time of surgery (years); mean±SD	44.46±14.07	52.93±13.19	0.018
Age ≤45 years; n (%)	16 (61.54)	12 (27.27)	0.006
BMI; mean±SD	23.43±3.81	23.09±3.57	0.694
Mode of renal replacement therapy, n (%)			
Hemodialysis	25 (96.15)	39 (88.64)	
Peritoneal dialysis	1 (3.85)	5 (11.36)	0.30
Duration of dialysis (years); mean±SD	6.69±3.54	7±4.5	0.763
Underlying renal disease; n (%)			
Diabetes mellitus	4 (15.38)	8 (18.18)	0.764
Hypertension	6 (23.08)	9 (20.45)	0.796
Chronic glomerulonephritis	3 (11.54)	3 (6.82)	0.5
Congenital renal disease	0 (0.00)	1 (2.227)	-
Renal cystic disease	1 (3.85)	3 (6.82)	0.609
Unknown	6 (24.00)	16 (36.36)	0.293
Symptoms; n (%)			
Bone pain	18 (69.23)	28 (63.64)	0.634
Fracture	3 (11.54)	3 (6.82)	0.5
Pruritis	2 (7.69)	12 (27.27)	0.063
Preoperative laboratory testing			
Corrected calcium (mg/dL); mean±SD	9.55±0.84	10.02±0.85	0.032
Phosphate (mg/dL); mean±SD	5.06±1.61	5.02±2.25	0.944
ALP (IU/L); median (IQR25-75)	433.8±358.66	314.43±281.06	0.143
ALP ≥420 IU/L; n (%)	11 (42.31)	10 (22.73)	0.088
PTH (pg/mL); median (IQR25-75)	2,294.5 (1,579.58 to 2,956.5)	1,946 (1,221.83 to 2,080.78)	0.079
Postoperative laboratory testing			
Corrected calcium (mg/dL); mean±SD	8.79±1	9.02±0.95	0.327
Phosphate (mg/dL); mean±SD	3.17±1.54	3.11±1.34	0.872
PTH (pg/mL); median (IQR25-75)	85 (33.25 to 148.73)	54 (33 to 97)	0.694
History of IV calcium; n (%)	20 (76.92)	15 (34.09)	0.001
Postoperative hypocalcemic symptoms; n (%)	3 (12.00)	2 (4.55)	0.268
Duration of hospital admission (day); median (IQR25-75)	8 (6 to 10)	6 (4 to 8)	0.083
Discharge medication; median (IQR25-75)			
Calcium carbonate (mg/day)	10,500 (8,250 to 12,000)	9,000 (4,500 to 12,000)	0.191
Calcitriol (mcg/day)	2.5 (1.75 to 4)	2 (2 to 4)	0.247

BMI=body mass index; ALP=alkaline phosphatase; PTH=parathyroid hormone; IV=intravenous; SD=standard deviation; IQR=interquartile range

Table 3. Multiple logistic regression model of risk factors for long-term severe hypocalcemia

Variables	Odds ratio	95% confidence interval	p-value
Age at time of surgery (years)	1.06	0.97 to 1.15	0.18
Age ≤45 years	30.00	2.24 to 543.11	0.01
Pruritis	0.23	0.33 to 1.51	0.13
Pre-operative corrected calcium (mg/dL)	6.59	0.38 to 113.45	0.19
Pre-operative ALP ≥420 IU/L	2.68	0.42 to 17.17	0.30
Pre-operative PTH (pg/mL)	1.00	0.99 to 1.001	0.62
History of IV calcium	10.71	1.74 to 65.71	0.01
Duration of hospital admission (days)	0.87	0.71 to 1.073	0.2

ALP=alkaline phosphatase; PTH=parathyroid hormone; IV=intravenous

hypocalcemia. This discrepancy warrants further exploration. In most published series of renal hyperparathyroidism, preoperative hypercalcemia has been identified as a paradoxical protective factor against severe postoperative hypocalcemia and HBS[8,11]. This contradictory finding is explained by the pathophysiology of tertiary hyperparathyroidism: patients with autonomous PTH hypersecretion and preoperative hypercalcemia tend to have adynamic or less metabolically active bone due to chronic calcium excess, which results in reduced skeletal uptake of calcium after parathyroidectomy and therefore a lower risk of HBS. In contrast, patients with secondary hyperparathyroidism who maintain normal or low serum calcium despite high PTH levels have markedly under-mineralized bone osteoid under conditions of high bone turnover, and upon abrupt removal of the PTH stimulus, their skeleton rapidly and hungrily remineralizes, resulting in profound and prolonged hypocalcemia. Similarly, elevated preoperative ALP, a surrogate marker of high osteoblastic activity and bone turnover, and markedly elevated PTH have been shown to significantly predict HBS and early severe hypocalcemia in renal hyperparathyroidism, as confirmed by a recent risk-scoring study demonstrating ALP greater than 150 U/L and PTH greater than 1,000 pg/mL as the strongest predictors[8,11]. The absence of these associations in the multivariate analysis of long-term hypocalcemia, rather than contradicting the existing literature, likely reflects a genuine mechanistic difference between short-term and long-term outcomes. While the short-term hypocalcemia nadir is driven by the acute skeletal calcium uptake during bone remineralization, the determinants of whether hypocalcemia persists beyond one year are governed by additional factors, including graft function, the degree of eventual skeletal remineralization completion, and the adequacy of long-term supplementation regimens. This suggests that different mechanisms may govern short-term versus long-term calcium regulation following parathyroidectomy. While short-term hypocalcemia is driven by rapid calcium redistribution, long-term hypocalcemia may reflect the interplay of bone remineralization dynamics, graft function, and chronic calcium-vitamin D supplementation patterns.

Beyond patient-level factors, several surgical variables may also influence the risk of long-term hypocalcemia following TPTx+AT and warrant consideration. First, the volume of parathyroid tissue transplanted during autotransplantation is a critical determinant of graft function. Studies have recommended transplanting 50 to 60 mg of parathyroid tissue, typically consisting of 10 to 20 small fragments of approximately one mm^3 each, into the brachioradialis muscle of the non-dominant forearm to optimize the likelihood of functional engraftment[12,13]. Insufficient transplanted tissue volume may result in graft failure or subnormal PTH secretion, thereby impairing calcium homeostasis and contributing to persistent hypocalcemia. Conversely, the selection of a nodular or fibrotic gland fragment for autotransplantation may reduce graft viability. Second, the technique of autotransplantation itself has been discussed in the literature. Both the minced tissue injection method and the thin-slice implantation technique have been described, with comparable reported outcomes. However, the quality of tissue handling and the avoidance of ischemic injury to the graft remain paramount. Third, surgeon experience plays a significant role in the outcomes of parathyroid surgery. Studies have consistently demonstrated that higher-volume surgeons and high-volume centers achieve better perioperative outcomes, including lower rates of persistent hypocalcemia and surgical complications[9,14]. In the context of TPTx+AT for renal hyperparathyroidism, the surgeon's ability to identify and completely excise all hyperfunctioning parathyroid tissue, including the supernumerary glands, while selecting the most appropriate gland for transplantation, is a technically demanding skill that directly affects long-term calcium outcomes. In the present study, gland volume, specific autotransplantation technique, and surgeon caseload were not systematically recorded, representing important limitations that future prospective studies should address.

Vitamin D status is another factor that may contribute to the development and persistence of long-term hypocalcemia following parathyroidectomy. Vitamin D plays a significant role in intestinal calcium absorption and bone mineral metabolism, and its deficiency can impair calcium homeostasis in the postoperative period. In patients with CKD, impaired renal hydroxylation of 25-hydroxyvitamin D to its active form, 1,25-dihydroxyvitamin D (calcitriol), is a well-recognized mechanism underlying disordered calcium and phosphate metabolism. Following parathyroidectomy, the abrupt loss of PTH-driven stimulation of renal 1-alpha-hydroxylase further diminishes calcitriol production, potentially aggravating hypocalcemia. Moreover, vitamin D insufficiency, even in the

absence of frank deficiency, may amplify the severity of HBS by limiting intestinal calcium absorption at a time of heightened skeletal demand during bone remineralization. Several studies have demonstrated that preoperative vitamin D deficiency is associated with more severe and prolonged postoperative hypocalcemia after parathyroidectomy[15,16]. Accordingly, preoperative optimization of vitamin D status, along with adequate supplementation of active vitamin D analogs in the postoperative period, may represent an important strategy for mitigating long-term severe hypocalcemia in this patient population. In the present study, serum vitamin D levels were not routinely measured preoperatively; therefore, the authors were unable to assess the independent contribution of vitamin D status to long-term hypocalcemia. Future prospective studies should include systematic assessment of vitamin D levels and evaluate the impact of preoperative vitamin D optimization and standardized postoperative active vitamin D supplementation protocols on long-term calcium outcomes after TPTx+AT.

This study has limitations. First, the sample size was small, which may limit the statistical power to detect associations with less common variables. Second, some patients had incomplete clinical or laboratory data, which may introduce bias. Third, due to the retrospective nature of the study, serum vitamin D levels were not routinely measured in most patients prior to surgery, precluding assessment of their potential influence on postoperative calcium homeostasis. Fourth, the timing of laboratory measurements was not uniform across patients, potentially affecting the accuracy of biochemical trend analysis. Future prospective studies with standardized protocols, including routine preoperative vitamin D assessment and larger sample sizes, are warranted to confirm these findings and elucidate the underlying mechanisms.

Conclusion

In this single-center retrospective study, younger age and a history of IV calcium administration were identified as independent predictors of long-term severe hypocalcemia after TPTx+AT in renal hyperparathyroidism patients. Preoperative biochemical markers, including calcium, PTH, and ALP levels, were not significantly associated with this complication. Early identification of at-risk patients may allow for closer postoperative monitoring, tailored calcium-vitamin D supplementation strategies, and proactive long-term follow-up to prevent complications. Future prospective studies with larger sample sizes and standardized postoperative protocols are warranted to validate these findings and further elucidate the mechanisms underlying long-term calcium regulation following parathyroidectomy.

What is already known about this topic?

- Hypocalcemia is a common complication after parathyroidectomy in renal hyperparathyroidism patients, with short-term incidence ranging from 72% to 97%.
- Preoperative biochemical markers such as high PTH, ALP, and calcium levels are known predictors of early postoperative hypocalcemia.

What does this study add?

- First study to identify predictors of long-term severe hypocalcemia (persisting for one year or longer) following total parathyroidectomy in a Thai population.
- Younger age and requirement for postoperative IV calcium supplementation are independent predictors of long-term severe hypocalcemia, while preoperative biochemical markers are not significantly associated with this long-term complication.

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Authors' contributions

All authors meet the ICMJE authorship criteria. PR: Conceptualization, study design, data collection, data analysis and interpretation, and original manuscript drafting. SS: Conceptualization, clinical supervision, and manuscript critical revision. WT: Conceptualization, study design, surgical procedures, data interpretation, manuscript critical revision and final approval, and corresponding author responsibilities. All authors approved the final version and are jointly accountable for all aspects of the work.

Data availability statement

The de-identified dataset supporting the findings of this study is available from the corresponding author upon reasonable request, subject to ethics committee approval and institutional data governance requirements. Public sharing of the dataset is

restricted due to patient privacy concerns and the retrospective nature of the study.

Ethics approval and consent to participate

This study was approved by the Human Research Ethics Committee of Thammasat University No. 1 (Faculty of Medicine) (Certificate of Approval No. MTU-EC-SU-0-210/67) and was conducted in accordance with the Declaration of Helsinki. The requirement for individual informed consent was waived by the ethics committee due to the retrospective nature of the study and the use of de-identified data from existing medical records.

Clinical trial registration

Not applicable. This study is a retrospective observational study and is not a clinical trial; therefore, clinical trial registration is not required.

Use of artificial intelligence

An artificial intelligence (AI)-assisted tool was used solely to support English language editing and manuscript writing during the preparation of this article. Specifically, Claude AI (claude-sonnet-4-6, Anthropic, 2025) was used to assist with sentence refinement, grammar checking, and English language expression. All scientific content, including study design, data collection, statistical analysis, interpretation, and conclusions, was conducted exclusively by the authors. The authors take full responsibility for the integrity, accuracy, and originality of all content. Claude AI is not listed as an author per the ICMJE authorship criteria.

Conflicts of interest

The authors declare no conflict of interest.

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