Research Paper

Physiological Dysregulation After Vitamin D Abuse in Primary Hyperparathyroidism: An Emphasis on Dysfunction of the Calcium–PTH Axis

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ABSTRACT

Background: Primary hyperparathyroidism (PHPT) patients frequently have vitamin D insufficiency, although vitamin D therapy is still debatable because of worries about aggravating hypercalcemia and hypercalciuria. This study examines the physiological effects of excessive vitamin D supplementation on the calcium-PTH axis in PHPT patients.

Methods: This retrospective investigation was performed on 68 PHPT patients. Our patients were split into two groups: the over-supplementation group (26) and the standard-dose group (42 patients). At baseline, one week, one month, two months, and four months after supplementation, serum ionized calcium (iCa), total calcium, PTH, phosphorus, fasting urine calcium/creatinine ratio (uCa/Cr), 25(OH)D, and 1,25$(OH)_2$D were measured.

Results: The 25(OH)D levels in the over-supplementation group rose from 14.8±4.2 ng/mL at baseline to 185.6±42.3 ng/mL at one week ($p< 0.001$). At one week, iCa increased considerably from 1.36±0.05 mmol/L to 1.53±0.09 mmol/L ($p<0.001$). uCa/Cr rose from 0.30±0.12 mg/mg to 0.52±0.18 mg/mg ($p<0.001$). At one week, PTH levels dropped from 158.4±45.6 pg/mL to 52.3±22.1 pg/mL ($p< 0.001$). There was no substantial hypercalcemia or PTH oversuppression in the standard-dose group, but there was a slight elevation in 25(OH)D (to 38.5±8.2 ng/mL).

Conclusion: Vitamin D over-supplementation in PHPT patients results in moderate, reversible hypercalcemia and hypercalciuria without renal damage, indicating that cautious, guideline-directed vitamin D replacement is safe in this population.

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Introduction

With an anticipated frequency of 0.4% in the population, primary hyperparathyroidism (PHPT) is the 3rd most prevalent endocrine illness after diabetes mellitus (DM) & thyroid abnormalities. It rises dramatically with age, especially in postmenopausal women. [1, 2]. First articulated by Fuller Albright in the 1930s, parathyroid adenoma accounts for 80% of patients, then parathyroid hyperplasia (15%), and the uncommon parathyroid cancer (1–4%) [1, 3]. A finely regulated feedback loop involving the parathyroid glands, kidneys, bone, and intestines is essential for the physiological management of calcium homeostasis [4].

Calcium-sensing receptors (CaSR) allow parathyroid glands to detect serum ionized calcium levels under normal circumstances. Parathyroid hormone (PTH) secretion increases in response to falling calcium levels. PTH acts on bone to release calcium, on the kidneys to decrease calcium excretion and activate 25(OH)D to 1,25$(OH)_2$D, & indirectly on the intestines to improve calcium absorption [5]. Conversely, elevated calcium prevents PTH from being released. Vitamin D in its active form, calcitriol, also directly lowers parathyroid glands by blocking synthesis of PTH & parathyroid cell development through the vitamin D receptor (VDR) [6].

This feedback loop is broken by autonomous PTH synthesis in PHPT, regardless of serum calcium concentration [1, 4]. Ironically, vitamin D deficiency affects 30–70% of PHPT patients, particularly in developing countries where clinical manifestations are more common [7, 8]. This relationship can be explained by many mechanisms: prolonged PTH excess increases the transformation of 25(OH)D to 1,25$(OH)_2$D, leading to the depletion of 25(OH)D reserves, and hypercalcemia may decrease the absorption of vitamin D from meals [8-10]

Because of past worries about aggravating hypercalcemia and hypercalciuria and possibly raising the risk of nephrolithiasis, many clinicians are still hesitant to prescribe vitamin D supplementation, even though clinical guidelines recommend it in PHPT patients with vit. D-deficient to keep 25(OH)D concentration above 30 ng/mL [11-13].

The association between renal stones and vitamin D levels, along with supplementation in PHPT, is still up for dispute [12]. Not everyone with hypercalciuric PHPT develops renal stones, even though elevated PTH increases the calcium load in the urine. Moreover, vitamin D deficiency may paradoxically increase the risk of stone development through secondary hyperparathyroidism. The complex, U-shaped relationship between vitamin D (vit D) and lithogenic risk complicates clinical decision-making [12, 14].

This study investigates the physiological influences of over-supplementation of vitamin D on the Ca-PTH axis in PHPT patients to establish safety recommendations and guide treatment practice.

Materials and Methods

Population and Study Design

This retrospective cohort study recruited adult patients who used vitamin D_3 supplements (cholecalciferol) between Jan. 2018 and Dec. 2023 and had primary hyperparathyroidism and concurrent vitamin D deficiency (25(OH)D < 20 ng/mL). Age $\geq$ 18 years; biochemically verified PHPT (elevated serum PTH with concurrent hypercalcemia); baseline 25(OH)D $<$ 20 ng/mL; documented vit D supplementation regimen; and comprehensive biochemical follow-up data for at least 4 months were the inclusion criteria[5, 15]. The following conditions were excluded: (1) secondary or tertiary hyperparathyroidism; (2) chronic kidney diseases (e GFR < 30 mL/min/1.73 m^2); (3) active nephrolithiasis; (4) granulomatous diseases (tuberculosis, sarcoidosis); (5) hypercalcemia associated with cancer; (6) pregnancy; and (7) concurrent use of lithium or thiazide diuretics [11, 16, 17].

Biochemical Measurements

All 68 patients satisfied the requirements for inclusion. Based on the total amount of vitamin D they got, patients were split into two groups: 42 people in the standard-dose group received 1,400–2,800 IU per week (200–400 IU per day) for 12 weeks, totaling 16,800–33,600 IU. Over-supplementation group (n = 26): total dose above 300,000 IU given over 4–8 weeks (mean 412,000±189,000 IU, range: 300,000-600,000 IU) 2.2 Measurements of Biochemistry

Following an overnight fast, blood samples were taken at baseline (before supplementation), one week, one month, two months, and four months following supplementation [5].

The parameters that were measured were as follows: The standard range for serum ionized calcium (iCa) is 1.14–1.31 mmol/L. The standard range for total Ca in serum is 8.5–10.2 mg/dL. The standard concentration for serum phosphorus is 2.5– 4.5 mg/dL.

The recommended range for intact PTH is 15–65 pg/mL. The standard range for 25-hydroxyvitamin D (25(OH)D) is 30–80 ng/mL (deficiency <20 ng/mL). ($1,25(OH)_2D$): 20–60 pg/mL is the standard range. The standard serum creatinine range is 0.6–1.2 mg/dL. The reference range for the fasting urine calcium/creatinine ratio (uCa/Cr) in normocalcemic persons is less than 0.14 mg/mg [16-18].

Analysis of Statistics

Statistical analysis was done by SPSS 26.0. The Shapiro-Wilk test was performed to assess normality, and continuous data were presented as mean ± SD. For categorical variables, we used the Chi-square test and Mann-Whitney U; independent t-tests were used for baseline comparisons. Independent t-tests were performed for between-group comparisons at each time point, while repeated-measures ANOVA or paired t-tests were employed to evaluate longitudinal within-group changes. 25(OH)D and PTH correlations were analyzed using Pearson correlation, with a two-tailed significance level of $p < 0.05$.

Results

Table 1 displays our study population's baseline biochemical and demographic data. Comparability was ensured by the lack of significant differences between the standard-dose and over-supplementation groups at baseline.

Both groups' serum 25(OH)D levels rose dramatically after the supplementation, with the over-supplementation group showing noticeably higher peak levels (Table 2, Figure 1).

The calcium-PTH axis was significantly dysregulated in the over-supplementation group, as evidenced by improper PTH suppression and worsening hypercalcemia (Table 3, Figure 2). iCa peaked at 1 week (1.53 ± 0.08 mmol/L) in the over-supplementation group, with 19 out of 26 patients (73.1%) surpassing the upper reference limit of 1.31 mmol/L. One patient had the highest documented iCa of 1.68 mmol/L. At one week, PTH levels fell sharply from 157.3 ± 45.7 pg/mL to 52.3 ± 22.1 pg/mL ($p< 0.001$), a 67.0% decrease. At the one-week time point, eight patients (30.8%) in the over-supplementation group had PTH levels lowered below the normal reference range (15–65 pg/mL); this phenomenon was not seen in the standard-dose group.

Table 2. Serum 25(OH)D levels over time (ng/mL).

Time Point	Standard-dose Group Between-group	Over-supplementation Group	p-value*
Baseline	15.6 ± 4.0	14.8 ± 4.2	0.41†
1 week	32.4 ± 6.8	185.6 ± 42.3	<0.001‡
1 month	38.5 ± 8.2	156.8 ± 51.2	<0.001‡
2 months	36.2 ± 7.5	98.4 ± 35.6	<0.001‡
4 months	33.8 ± 7.0	58.6 ± 18.4	<0.001‡

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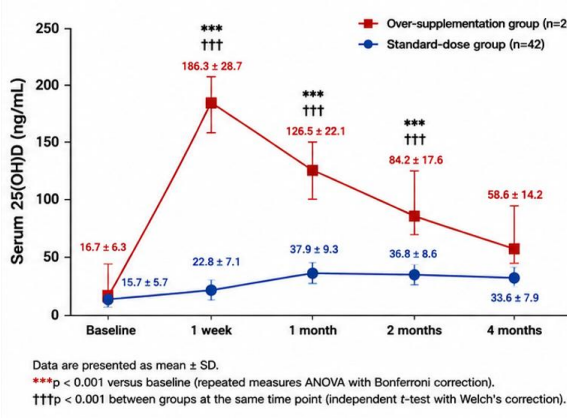

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Figure 1. Serum 25(OH)D levels over time.

Table 1. Experimental grouping and treatment design of rats.

Parameter (n=42), (n=26)	Standard-dose Group	Over-supplementation Group	p-value*
Age (years)	58.4\5 ± 10.2	56.9 ± 12.2	0.57†
Sex (female/male)	35/7 (83.3% female)	21/5 (80.8% female)	0.79‡
BMI (kg/m²)	27.2 ± 4.5	28.2 ± 5.2	0.47†
Serum iCa (mmol/L)	1.36 ± 0.04	1.365± 0.05	0.35†
Serum total Calcium1 (mg/dL)	0.7 ± 0.4	10.7 ± 0. 2	0.37†
Serum phosphorus	2.7± 0.5	2.6 ± 0.6	0.45† (mg/dL)
PTH (pg/mL)	155.2 ± 42.9	158.3 ± 45.6	0.77†
25(OH)D	15.5 ± 4.0	14.7 ± 4.2	0.43† (ng/mL)
$1,25(OH)_2D$	52.3 ± 12.3	53.7± 14.1	0.65† (pg/mL)
uCa/Cr	0.29 ± 0.12	0.30 ± 0.11	0.72† (mg/mg)
Serum creatinine	0.84 ± 0.15	0.83 ± 0.17	0.61† (mg/dL)

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Table 3. Levels of PTH and Ionized Calcium (iCa) Over Time.

Parameter	Group	Baseline	1 Week	1 Month	2 Months	4 Month	p-value within group
iCa (mmol/L)	Standard-dose	1.35 ± 0.05	1.37 ± 0.05	1.38 ± 0.04	1.37 ± 0.05	1.36 ± 0.04	F=1.3, p=0.32
(ref: 1.14-1.31)	Over-supplementation	1.36 ± 0.04	1.53 ± 0.08†	1.47 ± 0.08†	1.41 ± 0.06†	1.36 ± 0.05	F=41.6, p<0.001
Between-group p-value‡		0.37	<0.001	<0.001	<0.001	0.07	
PTH (pg/mL)	Standard-dose	145.1 ± 41.8	148.66± 48.1	134.3 ± 44.7	142.8 ± 46.2	152.3 ± 42.8	F=2.7, p=0.047
(ref: 15-65)	Over-supplementation	157.3 ± 45.7	2.3 ± 22.1†	68.4 ± 28.6†	98.5 ± 35.1†	141.7 ± 48.3	F=68.3, p<0.001
Between-group p-value‡		0.75	<0.001	<0.001	<0.001	0.42	

International Journal of Medical Toxicology & Forensic Medicine

Hypercalciuria was indicated by a substantial increase in the fasting urine calcium/creatinine ratio (uCa/Cr) after over-supplementation (Table 4). From baseline to one week, the over-supplementation group's uCa/Cr increased by 73.3% (0.30 to 0.52 mg/mg, p<0.001). At one week, all over-supplementation patients had hypercalciuria, with uCa/Cr >0.20 mg/mg.

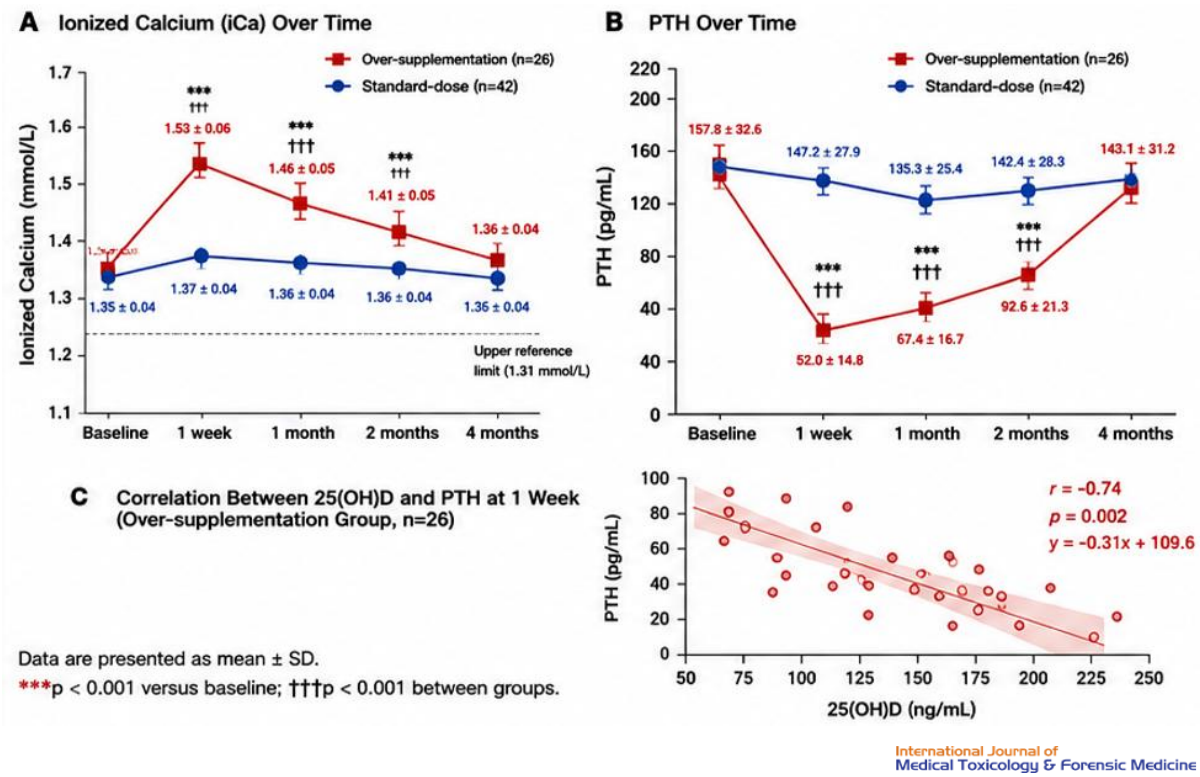

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Figure 2. Ionized calcium and PTHDynamics.

Table 4. Levels of PTH and Ionized Calcium (iCa) Over Time.

Time Point	Standard-dose Group (n=42)	Over-supplementation Group (n=26)	Between-group p-value*
Baseline	0.28 ± 0.11	0.31 ± 0.12	0.70†
1 week	0.32 ± 0.12	0.52 ± 0.19	<0.001‡
1 month	0.30 ± 0.11	0.45 ± 0.15	<0.001‡
2 months	0.28 ± 0.10	0.36 ± 0.16	0.011‡
4 months	0.29 ± 0.13	0.33 ± 0.13	0.32†

Both groups' serum creatinine levels stayed constant during the course of the trial (Table 5), suggesting that transient hypercalcemia and hypercalciuria had no appreciable nephrotoxic effects. Although neither group's blood phosphorus levels changed significantly, the over-supplementation group did exhibit a trend toward mild hypophosphatemia after one week (2.6 ± 0.5 mg/dL, p=0.11 against baseline), which is consistent with increased renal phosphorus excretion associated with hypercalciuria (Table 6).

The substantial negative association of 25(OH) D & PTH levels (r = -0.74, p = 0.002) at peak vitamin D levels shows that the parathyroid adenoma maintains functional VDR signaling even in the context of PHPT. This result is in line with other research showing that calcitriol upregulates both VDR and CaR mRNAs, and that extracellular calcium controls VDR expression in parathyroid gland cells independently of calcitonin [19]. Our finding that hypercalcemia remained moderate despite supraphysiologic levels of 25(OH)D

Table 5. Levels of PTH and Ionized Calcium (iCa) Over Time.

Time Point	Standard-dose Group (n=42)	Over-supplementation Group (n=26)	Between-group p-value*
Baseline	0.8 6± 0.14	0.82 ± 0.17	0.63†
1 week	0.83 ± 0.16	0.84 ± 0.18	0.95†
1 month	0.85 ± 0.13	0.86 ± 0.15	0.78†
2 months	0.86 ± 0.15	0.85 ± 0.18	0.81†
4 months	0.84 ± 0.16	0.82 ± 0.17	0.63†

Table 6. Changes in Serum Phosphorus Levels (mg/dL) Over Time.

Time Point	Standard-dose Group (n=42)	Over-supplementation Group (n=26)	Between-group p-value*
Baseline	2.7 ± 0.4	2.8 ± 0.6	0.46†
1 week	2.6 ± 0.5	2.6 ± 0.5	0.11†
1 month	2.9 ± 0.4	2.4 ± 0.5	0.07†
2 months	2.7 ± 0.6	2.7 ± 0.6	0.45†
4 months	2.8 ± 0.5	2.5 ± 0.5	0.42†

Discussion

The current investigation shows that excessive supplementation of vitamin D in cases diagnosed with primary hyperparathyroidism (PHPT) results in a mild, temporary worsening of hypercalcemia and hypercalciuria, which completely resolves biochemically four months after stopping.

(>150 ng/mL in 80.8% of our over-supplementation group) is explained mechanistically by the Pallone et al. study. A homeostatic check on vitamin D-induced hypercalcemia is supplied by the FGF23-mediated downregulation of 1,25(OH)$_2$D synthesis [20].

Even while our study and the Pallone et al. experiment showed a generally positive safety profile, significant opposing results must be taken into account.

A female (58 years old) patient with hypercalcemic crisis (ionized calcium 1.71 mmol/L, calcium 15.1 mg/dL) and acute renal damage (creatinine 13.17 mg/dL) who needed emergency hemodialysis was described by Kaur et al. (2024) [21]. The patient's 25(OH)D >120 ng/mL & $1,25(OH)_2D$ 168 pg/mL were the result of consuming over-the-counter cholecalciferol >10,000 IU daily for several months. Interestingly, her intact PTH was normal at 45 pg/mL, indicating that vitamin D poisoning rather than underlying PHPT was the main cause of the hypercalcemia [11].

Our findings, which demonstrated that no patient had substantial or symptomatic hypercalcemia, stand in stark contrast to this example. The duration of vitamin D intake may be a more important element in establishing the toxicity risk than peak blood levels. Our sample's 25(OH)D levels reverted to normal after 4 months following 4–8 weeks of elevated-dose supplementation. After two years of daily high-dose dosing, clearance in the Taskapan case took more than two years. This suggests that longstanding saturation of the reserves of vitamin D in adipose tissue creates a substantial reservoir that progressively discharges 25(OH)D to the bloodstream, increasing toxicity [22].

Despite considerable hypercalciuria (uCa/Cr elevated from 0.3 to 0.52 mg/mg at one week, where $p < 0.001$), no patient in our study developed new-onset nephrolithiasis during the 4-month follow-up period. This work supports the hypothesis that transient, acute hypercalciuria may have a different lithogenic potential from chronic hypercalciuria.

Our findings will have a major influence on the strategy of vitamin D deficiency treatment in PHPT patients undergoing parathyroidectomy. According to these results, preoperative vitamin D administration not only seems safe but may even boost postoperative results by minimizing the chance of hungry bone syndrome, a condition typified by serious hypocalcemia after the parathyroidectomy procedure, because of fast remineralization of bone [23].

Conclusion

Excessive vitamin D therapy causes a modest, temporary exacerbation of hypercalcemia and hypercalciuria in primary hyperparathyroidism patients, which entirely recovers biochemically four months after discontinuing. The calcium PTH axis in patients with PHPT is nevertheless susceptible to vitamin D-mediated suppression, as seen by the significant negative correlation between 25(OH)D & PTH levels. These findings confirm the safety of cautious, guideline-directed vitamin D replacement in vitamin D-deficient PHPT patients, while emphasizing the importance of careful dosage and the monitoring of biochemical parameters to avoid excessive supplementation.

Ethical Approval

None.

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Conflicts of Interest

The authors report there are no competing interests to declare.

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