

# Efficacy of High vs. Conventional Ergocalciferol Dose for Increasing 25-Hydroxyvitamin D and Suppressing Parathyroid Hormone Levels in Stage III-IV CKD with Vitamin D Deficiency/Insufficiency: A Randomized Controlled Trial

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**Background:** Vitamin D deficiency/insufficiency is common in chronic kidney disease (CKD) patients and it contributes to secondary hyperparathyroidism, which occurs early in CKD. It is not clear whether the Kidney Disease Outcomes Quality Initiative (K/DOQI) recommended doses of ergocalciferol are adequate for correction of vitamin D insufficiency and hyperparathyroidism.

**Objective:** To evaluate the parathyroid hormone (PTH)-lowering effect, safety, and tolerability of high-dose ergocalciferol compared with conventional-dose ergocalciferol in CKD subjects.

**Material and Method:** We enrolled CKD stage III-IV patients who had 25-hydroxyvitamin D (25-OH-D) level <30 ng/mL. The patients were randomized into two groups, control group treated with ergocalciferol as recommended by K/DOQI guidelines, and treatment group treated with double dosage of ergocalciferol from the recommendation. We compared serum 25-OH-D, intact-PTH, phosphate, calcium, and bone biomarker levels, during the 8-week intervention.

**Results:** Sixty-eight patients were included (34 controls and 34 treatments). Baseline characteristics of both groups were similar except calcium level  $9.12 \pm 0.56$  mg/dL in control group and  $9.44 \pm 0.38$  mg/dL in treatment group ( $p = 0.009$ ), but not clinically significant. At the end of the 8-week, the mean 25-OH-D level significantly increased from  $20.99 \pm 6.68$  to  $33.41 \pm 8.92$  ng/mL in the treatment group ( $p = 0.001$ ) and increased from  $20.84 \pm 7.21$  to  $23.42 \pm 7.89$  ng/mL in the control group ( $p = 0.026$ ). There was also a significantly greater increase of 25-OH-D levels in the treatment group. Additionally, PTH levels significantly decreased from  $90.75 \pm 67.12$  to  $76.40 \pm 45.97$  at 8 weeks ( $p = 0.024$ ) in the treatment group, and there was no change in the control group ( $97.14 \pm 83.52$  vs.  $101.13 \pm 95.03$  pg/mL,  $p = 0.546$ ). Serum calcium, phosphate, and adverse effects did not significantly change in either group throughout the study.

**Conclusion:** In addition to improving vitamin D levels, oral high-dose ergocalciferol was safe and had a beneficial effect in decreasing PTH in patients with stage III-IV of CKD.

**Keywords:** Hyperparathyroidism, Chronic kidney disease, Ergocalciferol

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Chronic kidney disease-Bone metabolism disease (CKD-BMD) is the important factors in patients with kidney disease and it is related to increased morbidity and mortality rate in patients with CKD. This condition is characterized by alteration of calcium and phosphate metabolism especially phosphate retention and calcitriol deficiency resulting in hyperparathyroidism. Calcitriol or 1, 25-hydroxycholecalciferol (1, 25-OH-D) is produced

in renal tubular cells by 1-alpha hydroxylation of 25-hydroxycholecalciferol. In the decline of the renal function, especially in chronic tubulointerstitial disease, calcitriol deficiency will develop. A reduction of serum 25-hydroxyvitamin D (25-OH-D), the substrate for the kidney's generation of calcitriol (1, 25-OH-D), produces secondary hyperparathyroidism (2°HPT) in individuals with normal kidney function<sup>(1,2)</sup>, and may aggravate 2°HPT in those with CKD and decreased kidney function<sup>(3,4)</sup>. Recently, 25-OH-D insufficiency and deficiency is more common and associated with the level of kidney function in the CKD population especially advanced stage of CKD<sup>(5)</sup>. Moreover, the deficiency of 25-OH-D was found to be associated with

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cardiovascular mortality in CKD<sup>(6,7)</sup>. In patients with CKD, nutritional vitamin D deficiency and insufficiency can both be prevented by supplementation with vitamin D<sub>2</sub> (ergocalciferol). The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF/KDOQI) guideline in patients with CKD and those requiring dialysis is to receive a modest supplementation with ergocalciferol to raise serum 25-OH-D levels to 30 to 60 pg/mL. In addition, they might have an increase in the plasma levels of 1, 25-OH-D or lower the elevated serum levels of intact parathyroid hormone (PTH)<sup>(8)</sup>. However, it is not clear whether the KDOQI recommended doses of ergocalciferol are adequate for correction of vitamin D insufficiency and hyperparathyroidism. In addition, high dose ergocalciferol supplement successively increased serum 25-OH-D levels in the prospective observational study and there was no other side effect associated with the treatment in predialysis CKD<sup>(9)</sup>. Thus, there was limitation of evidence for dosage of ergocalciferol supplement in CKD population. The present study examined the effect of short-term high dose ergocalciferol therapy on serum 25-OH-D, PTH levels, serum calcium, and phosphate compared with conventional dose ergocalciferol as KDOQI recommendation.

### Material and Method

This was an 8-week open labeled, randomized, controlled study conducted in Outpatient, Department of Medicine, Phramongkutklao Hospital. Data was collected between October 2013 and February 2014. The study was approved by the Institutional Review Boards of the Phramongkutklao Hospital and College of Medicine. Inclusion criteria into the study were age greater than 18 years, CKD stage III-IV (estimated GFR 15-60 ml/min/1.73 mm<sup>2</sup> by CKD-EPI creatinine base formula), serum 25-OH-D less than 30 ng/dL. Exclusion criteria were primary or tertiary hyperparathyroidism, hypoparathyroidism, receiving vitamin D analogue within four weeks, serum calcium greater than 10.5 mg/dL, serum phosphate greater than 5.5 mg/dL, chronic liver disease, malabsorption syndrome, malignancy, chronic illness whose life expectancy was less than six months. All patients gave informed written consent. Eligible patients were randomized by a block of four randomization assigned to two groups. The sample size calculated from previous study was to determine the mean difference of conventional dose and high dose at least 10 pg/mL<sup>(9)</sup>. To test the hypothesis, approximately 28 persons per

group and plus dropout rate 20%. Finally, 68 patients were included into the present study.

### Clinical protocol

The patients in the conventional group was received ergocalciferol according to KDOQI recommendation<sup>(10)</sup>, that were classified by 25-OH-D level (50,000 IU/week x 8 weeks if 25-OH-D <5 ng/mL, 50,000 IU/week x 4 weeks then 50,000 IU/month if 25-OH-D 5-15 ng/mL and 50,000 IU/months x 2 months if 25-OH-D 16-30 ng/mL). For treatment with high dose ergocalciferol, the patients will received the double dose of ergocalciferol according to KDOQI recommended dose. All patients were advised to take the medicine weekly before bedtime and were monitor the medication prescription based on pill counts.

### Clinical laboratory measurements

Laboratory testing including serum 25-OH-D, serum intact PTH, serum phosphate, serum calcium, serum albumin, complete blood count (CBC), blood urea nitrogen and serum creatinine were performed in the both groups at baseline and at 8-week. The method to measure serum 25-OH-D and intact PTH were the electrochemiluminescence binding assay. It is intended for use on Elecsys and Cobas e 601 immunoassay analyzers. The principle test of 25-OH-D and intact PTH used competitive principle and sandwich principle method.

### Safety monitoring

Adverse events that were or were not considered to be related to treatment were monitored four weeks. The patients were questioned in a systematic way about their experiences concerning adverse events during the previous eight weeks.

### Statistical analysis

Statistical analyses were performed using the SSPS 16.1 software program. Continuous variables between study and control groups were compared with unpaired t-tests. Continuous variables between baseline and at the end of study for each group of patients were compared using paired t-tests. Chi-square test was used to examine the categorical outcome. Statistical significance was taken as  $p < 0.05$ .

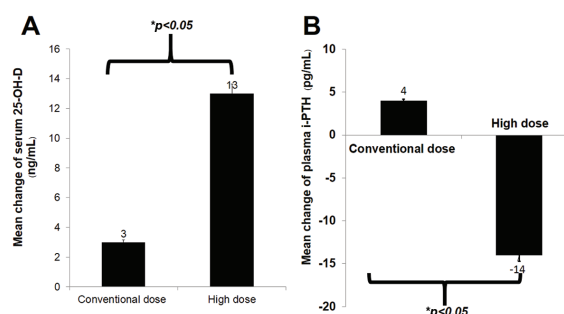
### Results

Eighty-eight patients in outpatient department were screened for possible study enrollment. Sixty-eight patients were eligible according to the entry criteria.

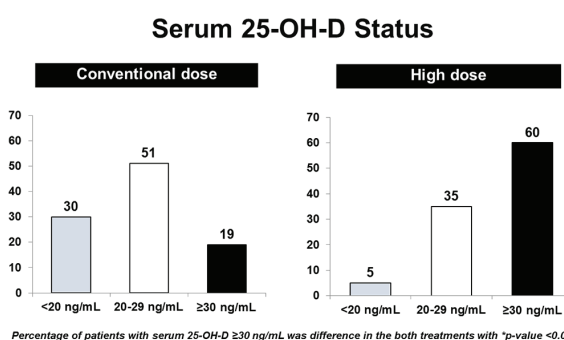
Thirty-four patients were assigned to the high dose ergocalciferol group and remainder were assigned to conventional dose ergocalciferol group. All the patients adhered to the ergocalciferol prescription based on pill counts. The patients' baseline characteristics are shown in the Table 1. Baseline characteristics of the both groups were similar except serum calcium level was significantly higher in the high dose ergocalciferol group ( $9.44 \pm 0.38$  vs.  $9.12 \pm 0.56$  mg/dL,  $p = 0.009$ ). At the end of the 8-week, the mean serum 25-OH-D level significantly increased from  $20.99 \pm 6.68$  to  $33.41 \pm 8.92$  ng/mL in the high dose ergocalciferol group ( $p = 0.001$ ) and increased from  $20.84 \pm 7.21$  to  $23.42 \pm 7.89$  ng/mL in the conventional dose ergocalciferol group ( $p = 0.026$ ). There was also a significantly greater increase of 25-OH-D levels in the high dose ergocalciferol group (Fig. 1A).

Additionally, serum PTH levels significantly decreased from  $90.75 \pm 67.12$  to  $76.40 \pm 45.97$  at 8-weeks ( $p = 0.024$ ) in the high dose ergocalciferol group, and there was no change in the conventional dose ergocalciferol group. ( $97.14 \pm 83.52$  vs.  $101.13 \pm 95.03$  pg/mL,  $p = 0.546$ ). There was a significantly greater decrease in serum intact PTH in the high dose ergocalciferol group as compared to the conventional dose ergocalciferol group (Fig. 1B).

Moreover, in comparison to baseline status, the high dose ergocalciferol group displayed significantly increased percentage of normalization of serum 25-OH-D levels than conventional dose ergocalciferol group (25-OH-D level  $>30$  ng/mL: 60% vs. 19%,  $p < 0.05$ ) (Fig. 2). Serum calcium, and phosphate did not significantly change in either group throughout the study and this parameters were shown in Table 2.



**Fig. 1** Mean changes in serum 25-OH-D levels and plasma PTH after treatment.



**Fig. 2** Serum 25-OH-D status after treatment with high dose ergocalciferol compared with conventional dose ergocalciferol.

During the 8-week study period, the patients with serum calcium between 10.2 and 10.5 mg/dL were 5.8% and serum phosphate between 4.5-5 mg/dL were 2.9% in the high dose ergocalciferol group, but no significant difference was detected in the both groups. No patients had any serious adverse events during the study.

**Table 1.** Baseline characteristics of the study population

	Conventional dose (n = 34)	High dose (n = 34)	p-value
Male	18	18	0.895
Age (years)	$65.9 \pm 15.5$	$66.7 \pm 15.4$	0.852
Serum creatinine (mg/dL)	$2.07 \pm 0.92$	$2.16 \pm 1.03$	0.711
Glomerular filtration rate (mL/min/1.73 m <sup>2</sup> )	$35.3 \pm 15.6$	$33.1 \pm 13.3$	0.522
Intact-PTH (pg/mL)	$97.7 \pm 80.3$	$90.7 \pm 65.4$	0.812
25-OH-D (ng/mL)	$20.8 \pm 7.3$	$20.9 \pm 7.8$	0.552
Serum calcium (mg/dL)	$9.12 \pm 0.56$	$9.44 \pm 0.38$	0.009
Serum phosphate (mg/dL)	$3.58 \pm 0.54$	$3.46 \pm 0.59$	0.378
Diabetes mellitus	60.6%	70.6%	0.390

25-OH-D = 25-hydroxyvitamin D; intact-PTH = intact-parathyroid hormone  
Data are mean  $\pm$  SD and percentage

**Table 2.** Serum calcium and phosphate after treatment

	Conventional dose (n = 34)	High dose (n = 34)
Serum calcium level (mg/dL)	10.05±0.53	9.59±0.44
Phosphate level (mg/dL)	3.4±0.51	3.8±0.36
Serum calcium >10.2 mg/dL	-	2 (5.8%)
Serum phosphate >4.5 mg/dL	-	1 (2.9%)
Serum calcium >10.5 mg/dL and/or serum phosphate >5 mg/dL	-	-

Data are mean ± SD and percentage, no significant difference was detected in the both groups

## Discussion

Ergocalciferol is a synthetic derivative of 25-OH-D and is recommended for the treatment of vitamin D deficiency, vitamin D resistant rickets, and hypophosphatemia in the general population and CKD patients. K/DOQI 2003 clinical practice guidelines for MBD-CKD recommended supplementation with ergocalciferol dose as general population in CKD patients whose 25-OH-D level less than 30 ng/mL<sup>(10)</sup>. Our study demonstrated that high dose ergocalciferol had higher efficacy for increasing 25-OH-D and decreasing intact PTH level in patient with CKD than conventional dose ergocalciferol after eight weeks of treatment.

The KDOQI guidelines recommend treatment of vitamin D deficiency starting with CKD stage III, though no data are available showing an impact on serum PTH concentrations<sup>(10)</sup>. Previous studies demonstrated that standard dose ergocalciferol/cholecalciferol therapy did not appear to have benefits in serum PTH levels in stage III of CKD<sup>(11,12)</sup>. In addition, previous retrospective study demonstrated that initiation of treatment with ergocalciferol had no significant effect on phosphorus and PTH in patients with CKD stage V<sup>(13)</sup>. In contrast to one report in patients with early CKD treated with 50,000 IU of ergocalciferol once weekly for 12 weeks and once monthly thereafter for a total of six months showed that an increase in 25-OH-D level greater than 5 ng/mL (>12 nmol/L) was associated with a significant likelihood of a greater than 30% decrease in plasma intact PTH level<sup>(14)</sup>. Similar to our outcomes, our study revealed that oral high-dose ergocalciferol in the short term study was safe and had a beneficial effect in decreasing serum PTH and achieving serum 25-OH-D level in early stage of CKD patients. It was believed that high dose of 25-OH-D restoration resulted in an increase in 1, 25-OH-D production, and thus the suppression of PTH.

In the present study, there were no side effects associated with the treatment. Serum calcium and phosphate had no significant changes in all study patients. This finding was similar to other studies in CKD patients that no adverse consequences were observed with high dose 25-OH-D supplements<sup>(13,15)</sup> and low incidence of hypercalcemia and hyperphosphatemia<sup>(16)</sup>. However, the present study was limited by the short duration of follow up and no apparent treatment related benefits in prevent of cardiovascular events and impaired renal function in CKD patients. Additional research is needed to confirm these results and determine the optimal levels of serum 25-OH-D.

In conclusion, the present study results showed that ergocalciferol administration had a favorable effect on serum PTH levels and increase serum 25-OH-D levels in patients with CKD stage III and IV. Low incidence of adverse effects including hypercalcemia and hyperphosphatemia was detected.

## What is already known on this topic?

Vitamin D insufficiency and deficiency are more common in the CKD population especially advanced stage of CKD. Moreover, the deficiency of 25-OH-D was found to be associated with all-cause and cardiovascular mortality in CKD. K/DOQI 2003 clinical practice guidelines recommended supplementation with ergocalciferol dose as general population in CKD patients whose 25-OH-D level less than 30 ng/mL. However, it is not clear whether the KDOQI recommended doses of ergocalciferol are adequate for correction of vitamin D insufficiency and hyperparathyroidism.

## What this study adds?

Double standard dose ergocalciferol had higher efficacy for increasing 25-OH-D and decreasing intact PTH level in patient with CKD stage III and IV

than standard dose ergocalciferol after 8 weeks of treatment. Low incidence of adverse effects including hypercalcemia and hyperphosphatemia was detected in the double standard dose ergocalciferol.

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#### Potential conflicts of interest

None.

#### References

1. Bouillon RA, Auwerx JH, Lissens WD, Pelemans WK. Vitamin D status in the elderly: seasonal substrate deficiency causes 1,25-dihydroxycholecalciferol deficiency. *Am J Clin Nutr* 1987; 45: 755-63.
2. Ooms ME, Roos JC, Bezemer PD, van der Vijgh WJ, Bouter LM, Lips P. Prevention of bone loss by vitamin D supplementation in elderly women: a randomized double-blind trial. *J Clin Endocrinol Metab* 1995; 80: 1052-8.
3. Eastwood JB, Stamp TC, Harris E, de Wardener HE. Vitamin-D deficiency in the osteomalacia of chronic renal failure. *Lancet* 1976; 2: 1209-11.
4. Cannell JJ, Hollis BW, Zasloff M, Heaney RP. Diagnosis and treatment of vitamin D deficiency. *Expert Opin Pharmacother* 2008; 9: 107-18.
5. Satirapoj B, Limwannata P, Chaiprasert A, Supasyndh O, Choovichian P. Vitamin D insufficiency and deficiency with stages of chronic kidney disease in an Asian population. *BMC Nephrol* 2013; 14: 206.
6. Hutchinson MS, Grimnes G, Joakimsen RM, Figenschau Y, Jorde R. Low serum 25-hydroxyvitamin D levels are associated with increased all-cause mortality risk in a general population: the Tromso study. *Eur J Endocrinol* 2010; 162: 935-42.
7. Melamed ML, Michos ED, Post W, Astor B. 25-hydroxyvitamin D levels and the risk of mortality in the general population. *Arch Intern Med* 2008; 168: 1629-37.
8. National Kidney Foundation. K/DOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. *Am J Kidney Dis* 2003; 42 (4 Suppl 3): S1-201.
9. Trakarnvanich T, Chalapipat O, Disthabanchong S, Kurathong S, Praditpornsilpa K, Stitchantrakul W, et al. Effect of high dose ergocalciferol in chronic kidney disease patients with 25-hydroxyvitamin D deficiency. *J Med Assoc Thai* 2010; 93: 885-91.
10. Bailie GR, Massry SG. Clinical practice guidelines for bone metabolism and disease in chronic kidney disease: an overview. *Pharmacotherapy* 2005; 25: 1687-707.
11. Zisman AL, Hristova M, Ho LT, Sprague SM. Impact of ergocalciferol treatment of vitamin D deficiency on serum parathyroid hormone concentrations in chronic kidney disease. *Am J Nephrol* 2007; 27: 36-43.
12. Chandra P, Binongo JN, Ziegler TR, Schlanger LE, Wang W, Someren JT, et al. Cholecalciferol (vitamin D3) therapy and vitamin D insufficiency in patients with chronic kidney disease: a randomized controlled pilot study. *Endocr Pract* 2008; 14: 10-7.
13. Blair D, Byham-Gray L, Lewis E, McCaffrey S. Prevalence of vitamin D [25(OH)D] deficiency and effects of supplementation with ergocalciferol (vitamin D2) in stage 5 chronic kidney disease patients. *J Ren Nutr* 2008; 18: 375-82.
14. Al Aly Z, Qazi RA, Gonzalez EA, Zeringue A, Martin KJ. Changes in serum 25-hydroxyvitamin D and plasma intact PTH levels following treatment with ergocalciferol in patients with CKD. *Am J Kidney Dis* 2007; 50: 59-68.
15. Saab G, Young DO, Gincherman Y, Giles K, Norwood K, Coyne DW. Prevalence of vitamin D deficiency and the safety and effectiveness of monthly ergocalciferol in hemodialysis patients. *Nephron Clin Pract* 2007; 105: c132-8.
16. Kandula P, Dobre M, Schold JD, Schreiber MJ Jr, Mehrotra R, Navaneethan SD. Vitamin D supplementation in chronic kidney disease: a systematic review and meta-analysis of observational studies and randomized controlled trials. *Clin J Am Soc Nephrol* 2011; 6: 50-62.

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การศึกษาแบบทดลองสุ่มเปรียบเทียบประสิทธิผลของ *ergocalciferol* ชนิดรับประทานขนาดสูงและขนาดมาตรฐาน ต่อการเพิ่มของระดับของ *25-hydroxyvitamin D* และการลดลงของระดับพาราไทรอยด์ฮอร์โมนในเลือดในผู้ป่วยโรคไตเรื้อรังระยะ 3-4 ที่มีภาวะพร่องวิตามินดี

ปรมัตต์ ธิมาไชย, อุบลัมภ์ ศุภสินธุ์, อำนวย ชัยประเสริฐ, บัญชา สติระพจน์

ภูมิหลัง: ภาวะพร่องวิตามินดี พบได้บ่อยในผู้ป่วยที่มีโรคไตเรื้อรังและพบร่วมกับการเพิ่มขึ้นของระดับพาราไทรอยด์ฮอร์โมน โดยเฉพาะโรคไตเรื้อรังระยะท้าย ขนาดของ *ergocalciferol* รับประทานตามคำแนะนำของสมาคมโรคไตตาม *Kidney Disease Outcomes Quality Initiative (K/DOQI)* ในผู้ป่วยไตเรื้อรังมีผลต่อการเปลี่ยนแปลงของระดับ *25-hydroxyvitamin D* และระดับพาราไทรอยด์ฮอร์โมนไม่ชัดเจน

วัตถุประสงค์: เพื่อศึกษาเปรียบเทียบประสิทธิผลระหว่างการให้ *ergocalciferol* ชนิดรับประทานขนาดสูงเป็นสองเท่าและขนาดตามที่ *K/DOQI* แนะนำ ต่อการลดลงของระดับพาราไทรอยด์ฮอร์โมน และความปลอดภัยของการรักษาในผู้ป่วยโรคไตเรื้อรัง

วัสดุและวิธีการ: ผู้เข้าร่วมการศึกษาคือผู้ป่วยโรคไตเรื้อรังระยะ 3 และ 4 ที่มีระดับ *25-hydroxyvitamin D* ในเลือดต่ำกว่า 30 นาโนกรัม/มิลลิลิตร ผู้ป่วยถูกสุ่มเป็นสองกลุ่ม โดยกลุ่มควบคุมให้ *ergocalciferol* ชนิดรับประทานในขนาดตามที่ *K/DOQI* แนะนำ และในกลุ่มทดลองเพิ่มขนาด *ergocalciferol* เป็นสองเท่า จากที่ *K/DOQI* แนะนำ ตรวจวัดระดับ *25-hydroxyvitamin D* พาราไทรอยด์ฮอร์โมน แคลเซียม และฟอสเฟตในเลือดก่อนและหลังการรักษา โดยติดตามเป็นเวลา 8 สัปดาห์

ผลการศึกษา: ผู้ป่วยโรคไตเรื้อรังจำนวน 68 ราย มีกลุ่มควบคุมและกลุ่มทดลอง กลุ่มละ 34 ราย ข้อมูลพื้นฐานของผู้ป่วยสองกลุ่มไม่มีความแตกต่างกัน ยกเว้นค่าเฉลี่ยของระดับแคลเซียมในเลือดของกลุ่มทดลองมีค่า  $9.12 \pm 0.56$  มิลลิกรัม/เดซิลิตร สูงกว่ากลุ่มควบคุมที่มีค่า  $9.44 \pm 0.38$  มิลลิกรัม/เดซิลิตร ( $p = 0.009$ ) แต่อยู่ในเกณฑ์ปกติซึ่งไม่มีนัยสำคัญทางคลินิก เมื่อสิ้นสุดการศึกษาที่ 8 สัปดาห์ ค่าเฉลี่ยของระดับ *25-hydroxyvitamin D* ในเลือดของทั้งสองกลุ่มเพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติ โดยในกลุ่มทดลองเพิ่มจาก  $20.99 \pm 6.68$  เป็น  $33.41 \pm 8.92$  นาโนกรัม/มิลลิลิตร ( $p = 0.001$ ) และในกลุ่มควบคุมเพิ่มจาก  $20.84 \pm 7.21$  เป็น  $23.42 \pm 7.89$  นาโนกรัม/มิลลิลิตร ( $p = 0.026$ ) แต่ระดับ *25-hydroxyvitamin D* ในเลือดเพิ่มขึ้นอย่างมีนัยสำคัญในกลุ่มทดลองเมื่อเทียบกับกลุ่มควบคุม นอกจากนี้ค่าเฉลี่ยของพาราไทรอยด์ฮอร์โมนในเลือดลดลงเฉพาะในกลุ่มทดลอง โดยลดจาก  $90.75 \pm 67.12$  เป็น  $76.40 \pm 45.9$  พิโคกรัม/มิลลิลิตร อย่างมีนัยสำคัญทางสถิติ ( $p = 0.024$ ) ส่วนในกลุ่มควบคุมกลับพบว่า ค่าเฉลี่ยของพาราไทรอยด์ฮอร์โมนไม่มีการเปลี่ยนแปลงคือ จาก  $97.14 \pm 83.52$  เป็น  $101.13 \pm 95.03$  พิโคกรัม/มิลลิลิตร ( $p = 0.546$ ) จากการศึกษาพบว่าไม่พบการเปลี่ยนแปลงของระดับแคลเซียมหรือฟอสเฟตในเลือดอย่างมีนัยสำคัญทางสถิติ และไม่พบผลข้างเคียงอื่น ๆ

สรุป: นอกจากการเพิ่มระดับ *25-hydroxyvitamin D* ในเลือดแล้ว *ergocalciferol* ขนาดสูงมีความปลอดภัย และมีประโยชน์ในการลดระดับพาราไทรอยด์ฮอร์โมนในเลือด ซึ่งคาดว่ามีความปลอดภัยต่อภาวะเมแทบอลิซึมของกระดูกในผู้ป่วยโรคไตเรื้อรังระยะที่ 3-4

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