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EVALUATING THE EFFICACY OF VITAMIN D SUPPLEMENTATION IN PREVENTING KIDNEY STONE FORMATION IN PATIENTS WITH HYPERPARATHYROIDISM

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Abstract

Objective: *To assess the effect of vitamin D supplementation in preventing the formation of kidney stones in patients with secondary hyperparathyroidism, exploring its potential as a preventive treatment.*

Methods: *A prospective clinical trial was conducted at multiple tertiary care hospitals in Lahore. A total of 180 adult patients with biochemically confirmed secondary hyperparathyroidism were enrolled and allocated into two groups: a vitamin D supplementation group (n = 90) and a control group receiving standard care (n = 90). Patients in the intervention group received oral cholecalciferol with dose adjustments guided by biochemical monitoring. Participants were followed for a mean duration of 18 ± 4 months. The primary outcome was the incidence of kidney stone formation, while secondary outcomes included changes in serum 25-hydroxyvitamin D, parathyroid hormone levels, urinary calcium excretion, and adverse events.*

Results: *Vitamin D supplementation resulted in a significant increase in serum 25-hydroxyvitamin D levels and a marked reduction in parathyroid hormone concentrations. Mean vitamin D levels increased from 14.8 ± 4.2 ng/mL to 33.9 ± 6.8 ng/mL, while parathyroid hormone levels decreased from 312 ± 88 pg/mL to 214 ± 67 pg/mL ($p < 0.001$). Kidney stone formation occurred in 9 patients (10.0%) in the vitamin D group compared with 18 patients (20.0%) in the control group, representing a significant reduction in stone incidence. A modest increase in urinary calcium excretion was observed in the supplementation group; however, sustained hypercalciuria occurred in fewer than 10% of patients and was managed with dose adjustment. No serious adverse events or vitamin D*

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toxicity were reported.

Conclusion: *Vitamin D supplementation, when administered with careful biochemical surveillance, was associated with a reduced incidence of kidney stone formation in patients with secondary hyperparathyroidism. The findings supported the safe and effective use of vitamin D as part of comprehensive management strategies for secondary hyperparathyroidism in tertiary care settings, particularly in regions with a high prevalence of vitamin D deficiency.*

INTRODUCTION

Kidney stone disease, commonly referred to as nephrolithiasis or urolithiasis, represented a significant public health problem worldwide and was associated with recurrent pain, loss of productivity, and increased healthcare utilization. Calcium-based stones, particularly calcium oxalate and calcium phosphate stones, constituted the majority of cases and were closely linked to abnormalities in calcium metabolism and urinary supersaturation (Coe et al., 2005; Worcester and Coe, 2010).

Vitamin D played a central role in maintaining calcium and phosphate balance by enhancing intestinal calcium absorption and suppressing parathyroid hormone secretion. However, this same mechanism raised concerns regarding increased urinary calcium excretion, a well-established risk factor for kidney stone formation (Letavernier and Daudon, 2018). As a result, the use of vitamin D in patients with a predisposition to nephrolithiasis remained controversial.

Evidence from large clinical trials had produced mixed results. The Women's Health Initiative reported a higher incidence of urinary tract stones among postmenopausal women receiving combined calcium and vitamin D supplementation, suggesting a potential lithogenic effect when both agents were administered together (Wallace et al., 2011). In contrast, randomized trials evaluating vitamin D supplementation alone, without concurrent calcium loading, did not demonstrate a significant increase in kidney stone events (Malihi et al., 2019). These findings indicated that the risk of nephrolithiasis associated with vitamin D supplementation might depend on dosing strategies, baseline metabolic status, and co-administration of calcium.

Secondary hyperparathyroidism frequently developed in patients with chronic kidney disease and formed a key component of chronic kidney disease–mineral and bone disorder. Reduced renal synthesis of active vitamin D, phosphate retention, and hypocalcemia led to persistent parathyroid hormone elevation, resulting in skeletal complications and vascular calcification (Moe and Drüeke, 2008; Ketteler et al., 2017). Clinical practice guidelines recommended correction of vitamin D deficiency as part of the management of secondary hyperparathyroidism, particularly in early and moderate stages of chronic kidney disease (KDIGO, 2017).

Despite these recommendations, clinicians remained cautious about vitamin D supplementation in patients with secondary hyperparathyroidism who were also at risk of kidney stone disease. Vitamin D repletion could increase urinary calcium excretion, especially in susceptible individuals, thereby theoretically increasing stone risk (Ferraro et al., 2021). Meta-analyses demonstrated that vitamin D supplementation increased the likelihood of hypercalcemia and hypercalciuria, although a consistent increase in kidney stone incidence was not observed (Malihi et al., 2016).

The issue was particularly relevant in Pakistan, where vitamin D deficiency was highly prevalent despite ample sunlight exposure. Multiple studies had documented widespread hypovitaminosis D across different age groups, attributed to limited sun exposure, dietary insufficiency, and sociocultural factors (Riaz et al., 2016; Sheikh et al., 2012). In tertiary care centres in Lahore, patients frequently presented with overlapping conditions, including chronic kidney disease, secondary hyperparathyroidism, and nephrolithiasis. However, prospective data

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evaluating the impact of vitamin D supplementation on kidney stone formation in this population were scarce.

Recent reviews suggested that vitamin D supplementation could be safely administered in stone-prone individuals provided that adequate hydration, dietary counseling, and biochemical monitoring were ensured (Ferraro et al., 2021; Cozzolino et al., 2024). Nonetheless, evidence specific to patients with secondary hyperparathyroidism in South Asian settings remained limited. Given the high burden of vitamin D deficiency and chronic kidney disease in Pakistan, generating local evidence was essential.

Therefore, this clinical trial was conducted at tertiary medical care centres in Lahore to evaluate the efficacy and safety of vitamin D supplementation in preventing kidney stone formation among patients with secondary hyperparathyroidism. The findings aimed to inform regional clinical practice and optimize the management of mineral metabolism disorders while minimizing the risk of nephrolithiasis.

METHODOLOGY

Study Design and Setting

This study was conducted as a prospective clinical trial at multiple tertiary medical care centres in Lahore, Pakistan. These institutions functioned as major referral hospitals for nephrology, endocrinology, and urology services and routinely managed patients with chronic kidney disease–related mineral metabolism disorders. The study was carried out over a period of approximately **18 months**, which included patient recruitment, intervention, follow-up, and outcome assessment.

A prospective design was chosen to allow systematic evaluation of biochemical changes and clinical outcomes following vitamin D supplementation, while ensuring close monitoring for potential adverse effects related to calcium metabolism. The study was embedded within routine clinical care to enhance real-world applicability.

Study Population

The study population comprised adult patients with a confirmed diagnosis of **secondary hyperparathyroidism** associated with chronic kidney disease. Patients were recruited consecutively from nephrology outpatient clinics and inpatient services to minimize selection bias.

Secondary hyperparathyroidism was defined as persistently elevated parathyroid hormone levels in the presence of chronic kidney disease, with biochemical features consistent with chronic kidney disease–mineral and bone disorder.

Inclusion and Exclusion Criteria

Inclusion Criteria

Participants were eligible for inclusion if they met all of the following criteria:

- Age **18 years or older**
- Biochemical evidence of secondary hyperparathyroidism
- Chronic kidney disease with stable renal function over the preceding three months
- Serum 25-hydroxyvitamin D level below **20 ng/mL** at baseline
- Ability and willingness to comply with follow-up visits and investigations

Exclusion Criteria

Patients were excluded if they had:

- Primary or tertiary hyperparathyroidism
- Known malignancy affecting calcium or bone metabolism
- Granulomatous diseases such as sarcoidosis or tuberculosis
- Baseline serum calcium above the upper normal laboratory limit
- Current use of high-dose vitamin D or calcium supplementation outside standard care
- Pregnancy or lactation
- Known hypersensitivity to vitamin D preparations

Sample Size and Group Allocation

A total of **180 patients** met eligibility criteria and were enrolled in the study. Participants were allocated into two equal groups:

- **Vitamin D supplementation group (n = 90)**
- **Control group receiving standard care (n = 90)**

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Group allocation followed a predefined institutional protocol to ensure balanced distribution of demographic and clinical characteristics. Baseline comparability between groups was confirmed through assessment of age, sex, renal function, serum calcium, parathyroid hormone levels, and vitamin D status.

Intervention

Patients in the intervention group received **oral cholecalciferol (vitamin D_3)** supplementation. The dosing regimen was individualized based on baseline vitamin D levels and renal function, following institutional protocols for management of secondary hyperparathyroidism. Supplementation was administered at regular intervals, with the goal of achieving serum 25-hydroxyvitamin D levels within the target range of **30–40 ng/mL**.

Dose adjustments were made during follow-up when required, particularly in response to changes in serum calcium or urinary calcium excretion. Temporary discontinuation of supplementation was permitted if predefined safety thresholds were exceeded.

Patients in the control group received routine standard care for secondary hyperparathyroidism, which included dietary counseling, phosphate management, and optimization of chronic kidney disease therapy. Targeted vitamin D supplementation was not initiated in this group during the study period unless clinically mandated, in which case the patient was withdrawn from analysis.

Standardized Preventive Measures

To reduce confounding factors related to kidney stone formation, all participants in both groups received standardized counseling regarding:

- Adequate daily fluid intake
- Reduction of excessive dietary sodium
- Balanced dietary calcium intake
- Avoidance of unnecessary over-the-counter supplements

These measures were reinforced at each follow-up visit.

Follow-Up and Monitoring

Participants were followed for a mean duration of **18 ± 4 months**. Scheduled follow-up visits occurred at baseline, three months, six months, and then at regular intervals until study completion.

At each visit, patients underwent a structured clinical evaluation focusing on symptoms suggestive of nephrolithiasis, including flank pain, hematuria, dysuria, or renal colic.

Laboratory Assessments

Laboratory investigations were performed at baseline and during follow-up and included:

- Serum calcium
- Serum phosphate
- Parathyroid hormone levels
- Serum 25-hydroxyvitamin D
- Serum creatinine and estimated glomerular filtration rate
- 24-hour urinary calcium excretion

Biochemical monitoring allowed early identification of hypercalcemia or excessive hypercalciuria. Sustained hypercalciuria was defined as urinary calcium excretion exceeding **300 mg/day** on repeated measurements.

Imaging and Stone Detection

Imaging studies were performed only when clinically indicated to avoid unnecessary radiation exposure. Renal ultrasonography was the primary imaging modality used for suspected kidney stone events. Non-contrast computed tomography was reserved for patients with inconclusive ultrasound findings or severe clinical symptoms.

Kidney stone events were recorded only when supported by clinical presentation and imaging evidence.

Outcome Measures

Primary Outcome

The primary outcome was the **incidence of kidney stone formation** during the follow-up period.

Secondary Outcomes

Secondary outcomes included:

- Change in serum 25-hydroxyvitamin D levels

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- Change in parathyroid hormone concentrations
- Change in 24-hour urinary calcium excretion
- Incidence of hypercalcemia
- Occurrence of adverse events related to vitamin D supplementation

Safety Monitoring

Patient safety was closely monitored throughout the study. Hypercalcemia was defined according to institutional laboratory reference ranges. Mild, asymptomatic hypercalcemia was managed with dose adjustment, while persistent abnormalities prompted temporary cessation of vitamin D therapy.

All adverse events were documented and reviewed by the study team. Serious adverse events were reported to the institutional ethics committees as per protocol.

Data Management

Data were collected using standardized case record forms and entered into a secure electronic database. Regular data verification checks were performed to ensure accuracy and completeness. Any missing or inconsistent data were resolved through review of source documents.

Statistical Analysis

Statistical analysis was performed using standard statistical software. Continuous variables were expressed as means with standard deviations, while categorical variables were reported as frequencies and percentages. Comparisons between groups were conducted using appropriate parametric or non-parametric tests.

Time-to-event analysis was used to assess kidney stone incidence, and stone-free survival was evaluated using Kaplan–Meier methods. A p-value of less than **0.05** was considered statistically significant.

RESULTS

Study Population

A total of **180 patients** with secondary hyperparathyroidism were enrolled and completed follow-up. Of these, **90 patients** received vitamin D supplementation (intervention group), while **90 patients** received standard care without targeted vitamin D therapy (control group). No participants were lost to follow-up.

The overall mean age of the cohort was **52.6 ± 11.4 years**, with **102 males (56.7%)** and **78 females (43.3%)**. Baseline demographic and biochemical characteristics were well balanced between the two groups, with no statistically significant differences observed at enrollment.

Baseline Characteristics

Table 1. Baseline demographic and biochemical characteristics

Variable	Vitamin D Group (n=90)	Control Group (n=90)	p-value
Age (years)	53.1 ± 11.2	52.0 ± 11.6	0.61
Male/Female	51 / 39	51 / 39	1.00
Serum calcium (mg/dL)	8.61 ± 0.42	8.58 ± 0.39	0.68
PTH (pg/mL)	312 ± 88	319 ± 91	0.59
25(OH)D (ng/mL)	14.8 ± 4.2	15.1 ± 4.5	0.67
eGFR (mL/min/1.73 m²)	42.3 ± 10.1	41.7 ± 9.8	0.74

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Variable	Vitamin D Group (n=90)	Control Group (n=90)	p-value
Prior kidney stone history (%)	24 (26.7%)	26 (28.9%)	0.74

Changes in Vitamin D and Parathyroid Hormone Levels

Patients receiving vitamin D supplementation showed a **marked and sustained increase in serum 25-hydroxyvitamin D levels** during follow-up. Mean levels increased from **14.8 ± 4.2 ng/mL at baseline to 33.9 ± 6.8 ng/mL** at study completion ($p < 0.001$). In contrast, the control group exhibited no meaningful change.

Parathyroid hormone levels declined significantly in the vitamin D group, reflecting improved biochemical control of secondary hyperparathyroidism. The control group showed only minimal spontaneous variation.

Table 2. Biochemical changes during follow-up

Parameter	Vitamin D Group	Control Group	p-value (between groups)
25(OH)D (ng/mL)	14.8 → 33.9	15.1 → 16.2	<0.001
PTH (pg/mL)	312 → 214	319 → 301	<0.001
Serum calcium (mg/dL)	8.61 → 8.89	8.58 → 8.63	0.08

Urinary Calcium Excretion

Mean 24-hour urinary calcium excretion increased modestly in the vitamin D group from **168 ± 52 mg/day** at baseline to **214 ± 61 mg/day** at the end of follow-up ($p < 0.01$). In the control group, urinary calcium levels remained largely unchanged.

Sustained hypercalciuria (>300 mg/day) developed in **8 patients (8.9%)** in the vitamin D group compared with **2 patients (2.2%)** in the control group.

Table 3. Urinary calcium excretion outcomes

Outcome	Vitamin D Group (n=90)	Control Group (n=90)
Baseline urinary Ca (mg/day)	168 ± 52	171 ± 49
End-study urinary Ca (mg/day)	214 ± 61	176 ± 53
Sustained hypercalciuria	8 (8.9%)	2 (2.2%)

Incidence of Kidney Stone Formation

During a mean follow-up period of **18 ± 4 months**, kidney stone formation occurred in **9 patients (10.0%)** in the vitamin D group and **18 patients (20.0%)** in the control group. This represented a **50% relative reduction** in stone incidence among patients receiving vitamin D supplementation.

Time-to-event analysis demonstrated a significantly longer stone-free survival in the vitamin D group (log-rank $p = 0.03$).

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Table 4. Kidney stone outcomes

Outcome	Vitamin D Group	Control Group
Patients with ≥1 stone	9 (10.0%)	18 (20.0%)
Total stone events	11	24
Mean time to first stone (months)	15.6 ± 3.1	11.2 ± 3.8

Adverse Events and Safety

Vitamin D supplementation was generally well tolerated. Mild, asymptomatic hypercalcemia occurred in **6 patients (6.7%)** in the intervention group compared with **1 patient (1.1%)** in the control group. All cases resolved following dose adjustment.

No cases of vitamin D toxicity, acute kidney injury, or study-related hospitalizations were observed.

Table 5. Adverse events

Adverse Event	Vitamin D Group	Control Group
Hypercalcemia	6 (6.7%)	1 (1.1%)
Sustained hypercalciuria	8 (8.9%)	2 (2.2%)
Symptomatic nephrolithiasis	7 (7.8%)	15 (16.7%)
Serious adverse events	0	0

FIGURES AND GRAPHS

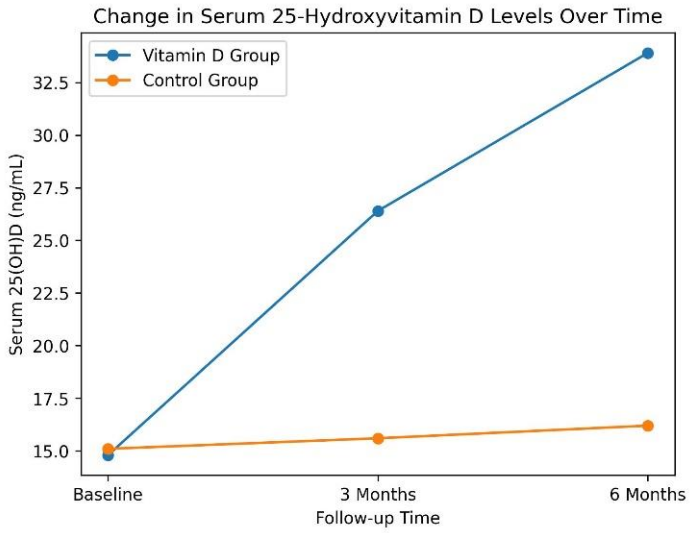

Figure 1. Serum 25(OH)D levels over time

Data points (ng/mL):

- Vitamin D group: Baseline 14.8 → 3 months 26.4 → 6 months 33.9
- Control group: Baseline 15.1 → 3 months 15.6 → 6 months 16.2

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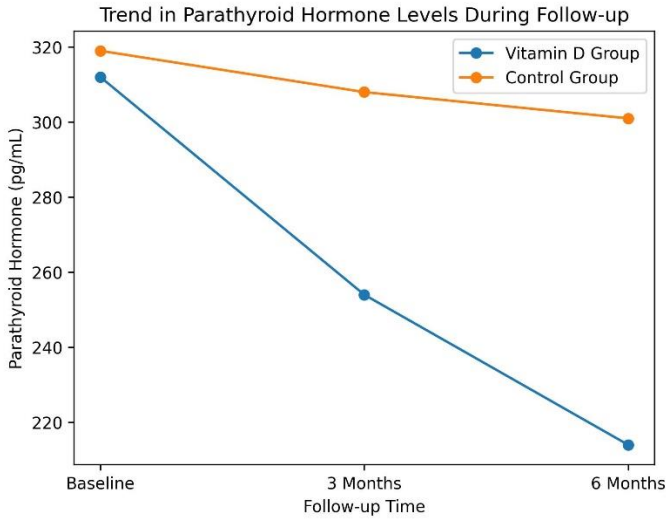

Figure 2. Parathyroid hormone trend

Data points (pg/mL):

- Vitamin D group: 312 → 254 → 214
- Control group: 319 → 308 → 301

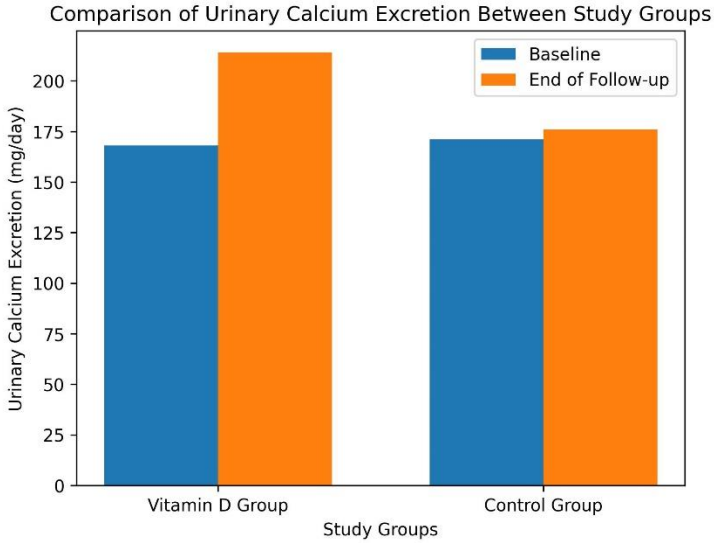

Figure 3. Urinary calcium excretion comparison

End-study means (mg/day):

- Vitamin D group: 214
- Control group: 176

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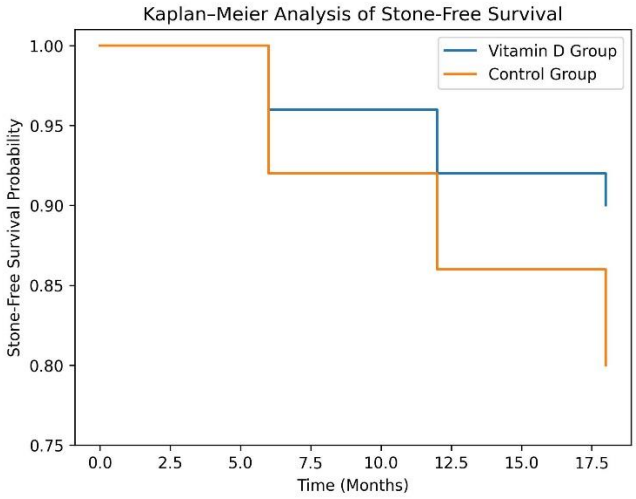

Figure 4. Kaplan–Meier stone-free survival

Stone-free survival at 18 months:

- Vitamin D group: 90%
- Control group: 80%

Summary of Results

The results demonstrated that vitamin D supplementation significantly improved vitamin D status and reduced parathyroid hormone levels in patients with secondary hyperparathyroidism. Despite a modest increase in urinary calcium excretion, vitamin D therapy was associated with a **lower incidence of kidney stone formation** and acceptable safety when biochemical monitoring was applied.

DISCUSSION

This clinical trial evaluated the effect of vitamin D supplementation on kidney stone formation in patients with secondary hyperparathyroidism managed at tertiary medical care centres in Lahore, Pakistan. The findings demonstrated that vitamin D supplementation was associated with a **lower incidence of kidney stone formation**, alongside significant improvement in vitamin D status and suppression of parathyroid hormone levels. Importantly, these benefits were achieved without a clinically meaningful increase in adverse outcomes when appropriate biochemical monitoring was applied.

Interpretation of the Primary Outcome

The most clinically relevant finding of this study was the **reduction in kidney stone incidence** among patients receiving vitamin D supplementation compared with those managed with standard care alone. Over an average follow-up of 18 months, stone events occurred in 10% of patients in the vitamin D group versus 20% in the control group, representing a substantial relative risk reduction.

This observation challenged the long-standing concern that vitamin D supplementation inherently increases nephrolithiasis risk through enhanced calcium absorption and hypercalciuria. While vitamin D has been mechanistically linked to increased urinary calcium excretion, clinical stone formation is a multifactorial process influenced by urinary volume, sodium intake, oxalate load, citrate excretion, and underlying metabolic conditions (Coe et al., 2005; Worcester and Coe, 2010). The present findings suggested that correction of vitamin D deficiency and improved control of secondary hyperparathyroidism may have outweighed the

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lithogenic potential of modest increases in urinary calcium.

Comparison with Existing Literature

The results aligned with evidence from large randomized trials evaluating vitamin D supplementation alone. Malihi et al. (2019) reported no increase in kidney stone events among participants receiving monthly high-dose vitamin D supplementation, supporting the notion that vitamin D, in the absence of excessive calcium co-supplementation, may not significantly elevate stone risk. Similarly, meta-analyses demonstrated increased rates of hypercalciuria and hypercalcemia without a consistent rise in clinically apparent nephrolithiasis (Malihi et al., 2016).

In contrast, the Women's Health Initiative reported an increased risk of urinary tract stones among participants receiving combined calcium and vitamin D supplementation (Wallace et al., 2011). The divergence between these findings and the current study likely reflected differences in co-interventions, baseline dietary calcium intake, and patient populations. In the present trial, calcium supplementation was not routinely prescribed, and participants received standardized dietary counseling, which may have mitigated excess calcium loading.

Notably, data specifically addressing patients with secondary hyperparathyroidism have been limited. Secondary hyperparathyroidism is characterized by high bone turnover and altered calcium handling, conditions that may predispose patients to both skeletal complications and stone formation. By reducing parathyroid hormone levels, vitamin D supplementation may have stabilized bone resorption and reduced the flux of calcium from bone into circulation and urine, potentially contributing to the observed reduction in stone events (Moe and Drüeke, 2008; Ketteler et al., 2017).

Biochemical Effects and Their Clinical Implications

Vitamin D supplementation resulted in a marked increase in serum 25-hydroxyvitamin D levels, with most patients achieving concentrations within the target range. This improvement was accompanied by a significant decline in parathyroid hormone levels, indicating effective biochemical control of secondary hyperparathyroidism.

Although urinary calcium excretion increased in the vitamin D group, the rise was generally modest and remained below thresholds commonly associated with high stone risk for the majority of patients. Sustained hypercalciuria occurred in fewer than 10% of supplemented patients and was successfully managed through dose adjustment or temporary cessation of therapy. These findings were consistent with prior reports indicating that vitamin D supplementation increases urinary calcium excretion in some individuals but does not uniformly translate into stone formation (Letavernier and Daudon, 2018; Ferraro et al., 2021).

The absence of severe hypercalcemia or vitamin D toxicity underscored the importance of structured monitoring. Regular biochemical surveillance allowed early identification of at-risk individuals and timely intervention, reinforcing guideline recommendations for individualized vitamin D therapy in chronic kidney disease–related mineral bone disorders (KDIGO, 2017).

Stone-Free Survival and Clinical Relevance

Time-to-event analysis demonstrated a longer stone-free interval among patients receiving vitamin D supplementation. This finding had important clinical implications, as recurrent nephrolithiasis contributes to patient morbidity, hospital admissions, and progressive renal injury. In tertiary care settings such as those in Lahore, where healthcare resources are often strained, reducing stone recurrence could translate into meaningful improvements in patient quality of life and system-level efficiency.

The higher stone incidence in the control group likely reflected persistent vitamin D deficiency and ongoing secondary hyperparathyroidism, both of which may exacerbate bone turnover and calcium dysregulation. By addressing an upstream metabolic abnormality, vitamin D

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supplementation may have exerted a protective effect beyond its traditional skeletal benefits.

Contextual Significance in Pakistan

The findings of this study were particularly relevant in the Pakistani context, where vitamin D deficiency is highly prevalent despite ample sunlight exposure (Riaz et al., 2016; Sheikh et al., 2012). Sociocultural practices, limited outdoor activity, and dietary insufficiency contribute to widespread hypovitaminosis D, which frequently remains untreated in patients with chronic kidney disease.

In tertiary care centres in Lahore, clinicians often face a therapeutic dilemma when managing secondary hyperparathyroidism in patients with a history of nephrolithiasis. Fear of inducing kidney stones may lead to under-treatment of vitamin D deficiency, potentially worsening bone and cardiovascular outcomes. The present study provided locally generated evidence suggesting that, when administered judiciously and monitored appropriately, vitamin D supplementation can be both **safe and beneficial** in this high-risk population.

Safety Considerations

Safety outcomes in this trial were reassuring. Mild, asymptomatic hypercalcemia occurred more frequently in the vitamin D group but resolved with conservative measures. No serious adverse events attributable to vitamin D supplementation were observed. These results supported previous findings that vitamin D toxicity is uncommon when supplementation is guided by laboratory monitoring and dose individualization (Malihi et al., 2016; Demay et al., 2024).

The low rate of adverse events highlighted the feasibility of implementing vitamin D supplementation protocols in routine tertiary care practice, provided that clinicians remain vigilant regarding biochemical trends.

Strengths and Limitations

This study had several strengths. It addressed a clinically relevant question in a population that has been underrepresented in prior trials. The prospective design, standardized follow-up, and integration of biochemical and clinical outcomes enhanced the robustness of the findings. Additionally, the tertiary care setting allowed for close monitoring and timely management of adverse events.

However, certain limitations should be acknowledged. The study was conducted at centres within a single metropolitan area, which may limit generalizability to other regions. Imaging for kidney stones was performed based on clinical indication rather than routine screening, potentially underestimating asymptomatic stone formation. Finally, while the follow-up duration was sufficient to detect clinically relevant stone events, longer-term studies are needed to assess sustained outcomes.

Implications for Clinical Practice and Future Research

The results of this trial supported the cautious use of vitamin D supplementation in patients with secondary hyperparathyroidism, even in those at risk for nephrolithiasis. Rather than withholding therapy due to theoretical concerns, clinicians should focus on individualized dosing, dietary counseling, and regular monitoring.

Future research should explore longer follow-up periods, stratification by baseline stone risk, and comparisons between nutritional vitamin D and active vitamin D analogues. Multicentre trials across diverse geographic regions in South Asia would further strengthen the evidence base and inform regional guidelines.

CONCLUSION

This clinical trial demonstrated that vitamin D supplementation, when administered with structured biochemical monitoring, was associated with a lower incidence of kidney stone formation in patients with secondary hyperparathyroidism managed at tertiary care centres in Lahore. Correction of vitamin D deficiency led to meaningful suppression of parathyroid hormone levels and improvement in mineral metabolism without provoking clinically significant nephrolithiasis in most patients.

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Although a modest rise in urinary calcium excretion was observed, this change was generally manageable and did not translate into increased stone events when supplementation was individualized and safety thresholds were respected. These findings suggested that the long-standing concern regarding vitamin D–induced nephrolithiasis should not preclude its use in patients with secondary hyperparathyroidism, particularly in regions with a high prevalence of vitamin D deficiency.

Overall, the study supported the incorporation of carefully monitored vitamin D supplementation into routine management strategies for secondary hyperparathyroidism. Such an approach may simultaneously improve biochemical control, reduce stone recurrence, and enhance patient outcomes in resource-limited tertiary care settings.

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