ORIGINAL ARTICLE ÖZGÜN ARAŞTIRMA

Serum 25-Hydroxyvitamin D Levels in Infants with Urolithiasis: A Retrospective Study

Ürolitiyazisi Olan İnfantlarda Serum 25-Hidroksivitamin D Düzeyleri: Retrospektif Bir Çalışma

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Abstract

Introduction: The relationship between vitamin D supplementation and urinary stone formation in infancy remains controversial, leading to variability in clinical practice regarding continuation or discontinuation of vitamin D in infants diagnosed with urolithiasis. This study aimed to evaluate whether serum 25-hydroxyvitamin D levels are associated with the presence of urolithiasis in infants.

Materials and Methods: This retrospective study included 77 infants who underwent renal ultrasonography for suspected urinary stone disease. Patients were classified according to the presence or absence of ultrasonographically detected urolithiasis. Clinical and laboratory parameters, including serum 25-hydroxyvitamin D levels and urinary calcium-to-creatinine ratios, were recorded. Hypercalciuria was defined using age-specific reference values. Factors associated with urolithiasis were evaluated using univariate and multivariate logistic regression analyses.

Results: Of the 77 infants, 54 had ultrasonographically detected urolithiasis. Mean serum 25-hydroxyvitamin D levels were similar between infants with and without urolithiasis (34.44±13.63 vs 34.81±15.68 ng/mL, p=0.917). None of the infants exhibited vitamin D excess or intoxication. Infants with stones had higher median urinary calcium-to-creatinine ratios compared with those without urolithiasis median (min-max) [0.58 (0.02–1.98) ve 0.34 (0.08–1.8) mg/mg; p=0.031], and hypercalciuria was more frequent in this group (37% vs 13%, p=0.030). Although younger age and hypercalciuria were associated with urolithiasis in univariate analyses, these associations did not remain statistically significant in multivariable models. Serum vitamin D levels were not associated with stone presence in either univariate or multivariate analyses.

Conclusion: In this study, serum 25-hydroxyvitamin D levels were not found to be associated with urolithiasis in infants. These findings suggest that vitamin D status alone may not fully explain stone formation during early infancy. Decisions regarding continuation or discontinuation of vitamin D supplementation should therefore consider the overall metabolic and clinical context, including urinary calcium excretion, rather than relying solely on serum vitamin D levels.

Keywords
Infant, urolithiasis, vitamin D, hypercalciuria, ultrasonography

Anahtar kelimeler
İnfant, ürolitiyazis, vitamin D, hiperkalsiüri, ultrasonografi

Öz

Giriş: İnfantlarda vitamin D desteği ile üriner taş oluşumu arasındaki ilişki tartışmalı olup, bu durum ürolitiyazis tanısı alan bebeklerde vitamin D desteğinin sürdürülmesi veya kesilmesi konusunda klinik uygulamalarda değişkenliğe yol açmaktadır. Bu çalışmanın amacı, infantlarda serum 25-hidroksivitamin D düzeyleri ile ürolitiyazis varlığı arasında bir ilişki olup olmadığını değerlendirmektir.

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Gereç ve Yöntem: Bu retrospektif çalışmaya, üriner taş hastalığı şüphesiyle renal ultrasonografi yapılan 77 infant dâhil edildi. Hastalar, ultrasonografide ürolitiyazis saptananlar ve saptanmayanlar olmak üzere iki gruba ayrıldı. Serum 25-hidroksivitamin D düzeyleri ve idrar kalsiyum/kreatinin oranı dâhil olmak üzere klinik ve laboratuvar verileri kaydedildi. Hiperkalsiüri, yaşa özgü referans değerler kullanılarak tanımlandı. Ürolitiyazis ile ilişkili faktörler tek değişkenli ve çok değişkenli lojistik regresyon analizleri ile değerlendirildi.
Bulgular: Toplam 77 infantın 54'ünde ultrasonografik olarak renal taş veya mikrolitiyazis saptandı. Taş saptanan ve saptanmayan gruplar arasında serum 25-hidroksivitamin D düzeyleri açısından anlamlı fark yoktu (34.44±13.63 ve 34.81±15.68 ng/mL; p=0.917). Hiçbir infantta vitamin D fazlalığı veya intoksikasyonu saptanmadı. Taş saptanan grupta idrar kalsiyum/kreatinin oranı daha yüksek bulundu median (min-max) [0.58 (0.02–1.98) ve 0.34 (0.08–1.8) mg/mg; p=0.031] ve hiperkalsiüri sıklığı daha fazlaydı (%37 ve %13; p = 0.030). Ancak yaş ve hiperkalsiüri, çok değişkenli analizlerde taş varlığı ile bağımsız olarak ilişkili bulunmadı. Serum vitamin D düzeyleri ise hem tek değişkenli hem de çok değişkenli analizlerde taş varlığı ile ilişkili değildi.
Sonuç: Bu çalışmada, infantlarda serum 25-hidroksivitamin D düzeyleri ile ürolitiyazis varlığı arasında bir ilişki saptanmamıştır. Bulgularımız, erken infant döneminde taş oluşumunun yalnızca vitamin D durumu ile açıklanamayacağını düşündürmektedir. Bu nedenle, ürolitiyazisi olan infantlarda vitamin D desteğinin sürdürülmesi veya kesilmesine ilişkin kararlar, yalnızca serum vitamin D düzeylerine değil; idrar kalsiyum atılımı başta olmak üzere genel metabolik ve klinik bağlam göz önünde bulundurularak verilmelidir.

Introduction

Urinary stone disease has been increasingly recognized in adults; however, stone formation during infancy represents a distinct clinical entity with important differences from adult disease in terms of epidemiology, etiology, clinical presentation, and management (1-3). Pediatric urolithiasis is of particular clinical relevance because it is frequently associated with underlying metabolic abnormalities and carries a potential risk for recurrence, underscoring the importance of early diagnosis and appropriate follow-up (4,5). Among the various factors implicated in pediatric urolithiasis, the relationship between vitamin D status and stone formation during infancy remains incompletely understood and, at times, controversial (6-9). Vitamin D plays a central role in calcium homeostasis and bone mineralization. While deficiency may impair skeletal health, excessive vitamin D exposure can increase intestinal calcium absorption and urinary calcium excretion, potentially contributing to stone formation in susceptible individuals (10-13). Genetic factors, including variations in the vitamin D receptor (VDR), may also modulate individual susceptibility to stone formation (14,15). Despite these observations, evidence specifically addressing infants remains scarce and often inconsistent, and the threshold at which vitamin D becomes potentially lithogenic in early life is uncertain.

Given the widespread use of vitamin D supplementation in infancy and the ongoing uncertainty surrounding its potential lithogenic effects, further research is needed to clarify whether elevated serum 25-hydroxyvitamin D levels are associated with biochemical or ultrasonographic markers of stone risk in this age group. Although key metabolic stone-forming parameters such as urinary oxalate, citrate, and cystine were not available in our cohort, the present study focuses on vitamin D status in relation to ultrasonographically detected urolithiasis. By characterizing this association independently of metabolic evaluations, our findings provide clinically relevant descriptive data that may help guide decision-making regarding continuation or discontinuation of vitamin D supplementation in infants with urolithiasis.

In the context of widespread vitamin D supplementation during infancy and ongoing uncertainty regarding its potential lithogenic effects, the objective of this retrospective study is to evaluate whether serum 25-hydroxyvitamin D levels are associated with the presence of urolithiasis in infants. By analyzing clinical, laboratory, and ultrasonographic data, we aimed to compare vitamin D status between infants with and without ultrasonographic evidence of urolithiasis, without inferring causality. This study also seeks to provide descriptive evidence to support clinical decision-making regarding whether vitamin D supplementation should be continued or discontinued in infants diagnosed with urolithiasis.

Materials and Methods

This retrospective study included infants younger than 12 months who were evaluated in the pediatric nephrology clinic with ultrasonography for suspected urolithiasis between June 2023 and November 2025. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee at which the studies were conducted and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study was approved by the Samsun University Non-Interventional Clinical Research Ethics Committee (approval number: 2025/23/13, date: 03.12.2025).

Medical records of 121 patients were reviewed. One infant with Zellweger syndrome, eleven preterm infants, one infant with an ectopic kidney, and one infant receiving calcium

folinate therapy, six infants with inconclusive laboratory data, nine infants with unknown vitamin D use at admission and fifteen infants without calcium-to-creatinine ratio were excluded due to the potential confounding effects of these conditions or treatments on mineral metabolism. Ultimately, 77 patients were enrolled in the final analysis.

All infants in our cohort had been started on routine vitamin D prophylaxis as part of standard national practice. Serum 25-hydroxyvitamin D concentrations were measured using the chemiluminescent microparticle immunoassay (CMIA), in accordance with the manufacturer's instructions.

For each patient, serum creatinine, albumin, calcium, phosphorus, alkaline phosphatase (ALP), parathyroid hormone (PTH), and 25-hydroxyvitamin D levels were extracted from the hospital laboratory system. Spot urine calcium and creatinine measurements were also recorded. Hypercalciuria was defined according to age-specific cutoffs: a urinary calcium-to-creatinine ratio above 0.8 mg/mg for infants aged 0–6 months and above 0.6 mg/mg for those aged 7–12 months (16). Because of the retrospective nature of the study, comprehensive metabolic stone risk profiles (such as urinary oxalate, citrate, and cystine) were not consistently available across the cohort and were therefore not included in the analysis.

All ultrasonographic evaluations were conducted by radiologists with different levels of pediatric imaging expertise using the same ultrasound platform (Resona R9, Mindray Medical International, Shenzhen, China). In radiology reports, stone size was inconsistently recorded. When a numeric size in millimeters was available, findings were categorized as microlithiasis (≤3 mm) or calculus (>3 mm), consistent with prior pediatric literature. When size was not reported and findings were described qualitatively (e.g.'millimetric echogenic/crystalloid foci'), cases were retained as ultrasound-positive but were not included in size-based analyses.

Urinary tract calculi were defined by punctate echogenic foci, typically with posterior acoustic shadowing and/or color Doppler twinkling artifacts, acknowledging the inherent difficulty of distinguishing true calculi from transient microlithiasis in infancy. Due to the retrospective design, the interval between ultrasonography and serum 25(OH)D measurement varied among patients (median 9 days, min–max 0–108).

Statistical Analysis

Statistical analyses were performed using SPSS version 25.0 (IBM Corp, Armonk, NY). Continuous variables were tested for normality using the Shapiro–Wilk test. Normally distributed variables are presented as mean ± standard deviation (SD), while non-normally distributed variables are reported as median (min-max). Categorical variables are expressed as numbers and percentages (n, %).

Comparisons between infants with and without urolithiasis were performed using the independent samples t-test for normally distributed continuous variables, and the Mann–Whitney U test for non-normally distributed variables. Chi-square or Fisher's exact tests were used for categorical variables, as appropriate.

Correlations were assessed using Spearman's rank correlation coefficient (ρ).

Univariate logistic regression analyses were first conducted to identify potential associations between clinical and laboratory variables and the presence of urolithiasis. Variables with univariate p-values <0.10 were considered for inclusion in multivariate logistic regression models to adjust for potential confounding. Two multivariate models were constructed: Model 1 included age and other relevant variables meeting the univariate inclusion criteria, while Model 2 additionally included hydronephrosis to adjust for its potential confounding effect. Odds ratios (OR) and adjusted odds ratios (aOR) with 95% confidence intervals (CI) were reported. A two-tailed p-value <0.05 was considered statistically significant for all analyses.

Results

A total of 77 infants were included in the study, of whom 54 (70.1%) had ultrasonographic evidence of urolithiasis. The clinical and laboratory features of the groups are summarized in Table 1. Among the 54 infants with ultrasonographically detected urolithiasis, stone size was not quantitatively reported in 10 cases and was described qualitatively as millimetric crystalloid echogenic foci. In the remaining 44 infants with documented stone measurements, the median (min–max) stone size was 2.8 mm (1.3–8.0). Among the 54 infants with ultrasonographically detected urolithiasis, stone location was reported as the upper pole in 2 (3.7%), middle pole in 12 (22.2%), lower pole in 27 (50.0%), and renal pelvis in 2 (3.7%) cases. In 11 infants (20.4%), stones were described as renal without further specification of intrarenal location.

Infants with urolithiasis were significantly younger than those without urolithiasis (median 3.06 vs. 4.56 months, p=0.009). Gender distribution and serum biochemical markers including total calcium, phosphate, parathyroid hormone, and 25-hydroxyvitamin D levels were comparable between the two groups (p>0.05 for all). The distribution of

Table 1. Clinical and laboratory features of patients

			Urolithiasis absent N:23	Urolithiasis present N:54	P
Age			4.56 (1.08–11.16)	3.06 (0.12–10.08)	**0.009**
Sex, n (%)		Female	11 (47.8)	17 (31.5)	0.202
		Male	12 (52.2)	37 (68.5)	
Stone size (mm) [median (min-max)]			-	2.8 (1.3–8.0)	
Stone size reported, n (%)		Yes	-	44 (81.5)	
		No	-	10 (18.5)	
Creatinine (mg/dL) [median (min-max)]			0.25 (0.13–0.47)	0.23 (0.13–0.42)	0.101
Ca (mg/dL) [median (min-max)]			10.42 (9.08–11.74)	10.47 (9.42–12.14)	0.501
P (mg/dL) [median (min-max)]			6.00 (4.7–6.8)	6.10 (3.6–7.6)	0.448
ALP (U/L) [median (min-max)]			211.00 (135–388)	270.00 (149–450)	**0.004**
25-OH VitD (ng/mL) (mean ± SD)			34.81 ± 15.68	34.44 ± 13.63	0.917
25-OH Vit D (ng/mL) categories, n (%) [median (min-max)]		<20	4 (17.4)	10 (18.5)	
30–50		20–29.9	6 (26.1)	9 (16.7)	
		10 (43.5)	30 (55.6)		
		>50	3 (13)	5 (9.3)	
PTH (pg/mL) [median (min-max)]			21.70 (5.1–52.2)	18.55 (1.2–52.6)	0.644
Uca/cre (mg/mg) [median (min-max)]			0.34 (0.08–1.8)	0.58 (0.02–1.98)	**0.031**
Hypercalciuria n (%)	(-)		20 (87.0)	34 (63.0)	**0.030***
	(+)		3 (13.0)	20 (37.0)	
Hydronephrosis n (%)	(-)		20 (87.0)	49 (90.7)	0.689
	(+)		3 (13.0)	5 (9.3)	

Continuous variables are presented as mean ± standard deviation or median (min-max), as appropriate. Ca: Calcium, P: Phosphorus, ALP: Alkaline phosphatase, PTH: Parathormone, $U_{ca/cre}$ (urine calcium-to-creatinine ratio) *Fisher's exact test was used for hypercalciuria

serum 25-hydroxyvitamin D categories did not differ between infants with and without urolithiasis (χ^2=1.42, p=0.701). The urinary calcium-to-creatinine ratio was higher in infants with urolithiasis (median 0.58 vs. 0.34 mg/mg, p=0.031), and hypercalciuria was more frequent in infants with urolithiasis (37% vs. 13%, p=0.030). The prevalence of hydronephrosis did not differ between the groups. Median serum creatinine levels were slightly lower in the urolithiasis group (0.23 vs 0.25 mg/dL, p=0.101). Median serum ALP levels were higher in patients with urolithiasis (270 vs 211, p=0.004). This difference is likely attributable to age-related variations in bone turnover rather than stone-related pathology.

Spearman correlation analyses showed no significant association between serum 25-hydroxyvitamin D levels and the urinary calcium-to-creatinine ratio (ρ = -0,111, p=0.336) or serum calcium levels (ρ = -0.169, p=0.142). A moderate positive correlation was observed between serum calcium and urinary calcium excretion (ρ = 0.294, p=0.010), while age was inversely correlated with the urinary calcium-to-creatinine ratio (ρ = -0.494, p<0.001) consistent with the expected physiological decrease in urinary calcium excretion with increasing age in infancy.The findings of the univariate and multivariate logistic regression analyses are summarized in Table 2. In the univariate models, younger age (OR: 0.78 95% CI: 0.63–0.96, p=0.021) and the presence of hypercalciuria (OR: 3.92 95% CI: 1.03–14.87, p=0.045) were significantly associated with urolithiasis. However, these associations were no longer statistically significant after adjustment for potential confounders. In the multivariate models, the effect of age attenuated (aOR: 0.82, 95% CI: 0.66–1.03, p=0.097), and the association between hypercalciuria and stone presence also weakened (aOR: 3.92–2.68, p=0.045–0.174). This attenuation likely reflects the strong age dependence of urinary calcium excretion and the limited number of infants

Table 2. Logistic regression analysis of confounding factors among patients with urolithiasis regarding vitamin D status and hypercalciuria Model 1 and 2

Variable	Univariate (Model) OR	Multivariate (Model 1) aOR	Multivariate (Model 2) aOR
	(95% CI), p	(95% CI), p	(95% CI), p
Age (months)	0.78 (0.63–0.96), p=**0.021**	0.82 (0.66–1.03), p=0.097	0.83 (0.66–1.05), p=0.124
25-OH Vitamin D	0.99 (0.96–1.03), p=0.916	0.99 (0.96–1.03), p=0.903	0.99 (0.96–1.03), p=0.909
Hypercalciuria	3.92 (1.03–14.87), **p=0.045**	2.68 (0.64–11.13), p=0.174	2.89 (0.67–12.50), p=0.154
Hydronephrosis	0.68 (0.14–3.11), p=0.620	-	0.559 (0.10–3.37), p=0.584

presenting with hypercalciuria in the cohort.

Serum 25-hydroxyvitamin D concentrations showed no association with urolithiasis in either univariate or multivariate analyses (unadjusted OR: 0.99, 95% CI: 0.96–1.03, p=0.916; adjusted OR: 0.99, 95% CI: 0.96–1.03, p=0.903), suggesting that vitamin D status did not influence the likelihood of detecting stones or microlithiasis on ultrasonography. Hydronephrosis was similarly not associated with stone presence in the adjusted models (unadjusted OR: 0.68, p=0.620; adjusted OR: 0.55, p=0.584).

Discussion

Previous studies investigating the association between vitamin D use and kidney stone risk have yielded inconsistent results. While some reports suggest that vitamin D supplementation may increase stone risk depending on dose and duration, others have found no increased risk outside specific high-risk populations, such as individuals with underlying genetic or metabolic conditions predisposing to stone formation (17,18). Several studies focusing on infantile urolithiasis have reported higher serum 25-hydroxyvitamin D levels in affected infants compared with controls, frequently accompanied by hypercalcemia and increased urinary calcium excretion, suggesting a contributory role of vitamin D primarily through calcium dysregulation rather than a direct lithogenic effect (7-9). In a well-designed prospective study of exclusively breastfed infants, Yilmaz et al. (9) reported that higher serum vitamin D levels were associated with stone formation, along with suppressed parathyroid hormone levels and increased urinary calcium excretion. In the present study, serum 25-hydroxyvitamin D levels did not differ between infants with and without ultrasonographically detected urolithiasis and were not independently associated with stone presence. This finding is biologically plausible, as none of the infants in our cohort exhibited vitamin D excess or overt intoxication (19). Although upper reference limits for serum 25-hydroxyvitamin D have been proposed in clinical guidelines, the specific level at which vitamin D may contribute to lithogenic risk remains poorly defined, particularly in infancy. Moreover, emerging evidence suggests that serum 25-hydroxyvitamin D concentrations may not accurately reflect tissue-level vitamin D activity or downstream effects on calcium and phosphate metabolism (12). Razzaque has emphasized that adverse effects of vitamin D supplementation, including hypercalciuria and ectopic calcification, may occur even in the absence of markedly elevated serum vitamin D levels due to complex regulatory mechanisms and local vitamin D metabolism, while normal serum levels do not necessarily exclude biological effects (12). Accordingly, serum vitamin D status alone may be insufficient to explain stone formation in early infancy and should be interpreted within a broader metabolic and developmental context. In addition, previous studies have suggested that interindividual differences in susceptibility to stone formation may be influenced by vitamin D receptor polymorphisms and potential defects in 24-hydroxylation of vitamin D, which may partly explain why some children develop stones despite receiving similar prophylactic or therapeutic vitamin D doses (15,20,21).

Although no significant association was observed between serum 25-hydroxyvitamin D levels and presence of urolithiasis in our cohort, infants with ultrasonographically detected urolithiasis exhibited higher urinary calcium-to-creatinine ratios, and hypercalciuria was more frequently observed in this group. Hypercalciuria was defined using age-specific reference values, supporting its pathological significance rather than reflecting physiological age-related variation. While hypercalciuria did not retain statistical significance in multivariable analyses, this finding likely

reflects limited sample size and reduced statistical power rather than a lack of clinical relevance. Large pediatric series have demonstrated that infantile urolithiasis differs from stone disease in older children with respect to metabolic risk factors, clinical presentation, and natural history, with hypercalciuria being the most common abnormality in infancy, whereas hypocitraturia becomes more prominent later in childhood (4,5,22-24). Importantly, most stones detected during infancy—particularly microlithiasis—tend to resolve spontaneously, underscoring the generally favorable prognosis in this age group (9). These observations highlight the variability in clinical interpretation and management strategies, particularly regarding decisions about vitamin D supplementation in infants with ultrasonographic stone findings.

Clinical practice varies widely: primary care physicians and clinicians in pediatric stone clinics differ in their decisions about whether vitamin D supplementation should be discontinued in infants diagnosed with urolithiasis. Observations from our center suggest that vitamin D supplementation is frequently withheld in such infants, although these decisions often reflect individual clinical preference rather than evidence-based recommendations.

Recent data also indicate that discontinuation of routine vitamin D prophylaxis in infants with urolithiasis does not significantly affect stone recurrence or progression, arguing against a causal relationship between standard prophylactic vitamin D dosing and stone growth (6). These observations support the concept that metabolic factors, rather than vitamin D exposure alone, may play a more prominent role in stone formation during early life.

From a pathophysiological perspective, calcium and phosphorus homeostasis is regulated through the coordinated interaction of vitamin D, the kidneys, intestines, parathyroid glands, and bone tissue (25). Although excessive vitamin D exposure may increase intestinal calcium absorption and promote hypercalciuria, establishing a direct and linear relationship between vitamin D supplementation and kidney stone formation remains challenging. Current evidence suggests that elevated serum vitamin D levels do not uniformly translate into clinically evident stone disease, particularly in the absence of sustained hypercalcemia or marked hypercalciuria. This may partly explain why routine vitamin D supplementation does not consistently result in stone formation in otherwise healthy infants.

Clinically, these findings support an individualized approach to the management of vitamin D supplementation in infants with urolithiasis. Routine discontinuation of prophylactic vitamin D supplementation based solely on the presence of ultrasonographic stone findings or serum vitamin D levels may not be justified. Decisions regarding vitamin D supplementation should therefore be guided by the overall clinical context, including urinary calcium excretion and other relevant clinical factors, rather than serum vitamin D concentrations alone. These observations should be interpreted within the limitations of a retrospective design and are intended to inform clinical judgment rather than establish definitive treatment recommendations.

Study Limitations

This study has several limitations inherent to its retrospective design. The timing of ultrasonographic evaluation and serum 25-hydroxyvitamin D measurement was not standardized, potentially introducing temporal variability. Comprehensive metabolic stone risk profiles, including urinary oxalate, citrate, and cystine levels, were unavailable, limiting assessment of additional lithogenic or protective factors. Stone detection relied on ultrasonography, which may not always reliably distinguish true calculi from transient microlithiasis in infancy. Finally, the relatively small sample size may have limited statistical power, particularly in multivariable analyses. The retrospective design and modest sample size limited detailed biochemical characterization of stone composition and comprehensive anatomical assessment; however, the study provides descriptive data reflecting real-world clinical practice in infants undergoing routine ultrasonography. These limitations should be considered when interpreting the findings, and prospective studies with standardized imaging and metabolic evaluation are warranted to further clarify the relationship between vitamin D status and stone formation in infancy.

Conclusion

Clinically, our findings support an individualized approach to decisions regarding continuation or discontinuation of vitamin D supplementation in infants with urolithiasis. Routine discontinuation of prophylactic vitamin D supplementation based solely on ultrasonographic stone findings or serum vitamin D levels may not be appropriate. Instead, clinical decision-making should be guided by the overall metabolic and clinical context, including urinary calcium excretion and other relevant factors, rather than serum vitamin D concentrations alone.

These findings should be interpreted as descriptive and hypothesis-generating rather than causal. Well-designed

prospective studies are needed to clarify the potential role of vitamin D supplementation in kidney stone formation and to define serum vitamin D levels that can be considered safe or potentially unsafe with respect to stone development, both in terms of incident stone formation and disease progression in individuals with existing stones.

Ethics

Ethics Committee Approval: The study was approved by the Samsun University Non-Interventional Clinical Research Ethics Committee (approval number: 2025/23/13, date: 03.12.2025).

Footnotes

Conflict of Interest: No conflict of interest was declared by the authors.

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