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Vitamin D Status and Proteinuria Severity in Children with Steroid-Sensitive and Steroid-Resistant Nephrotic Syndrome

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KEYWORDS

Nephrotic syndrome, children, vitamin D, proteinuria, steroid-sensitive nephrotic syndrome, steroid-resistant nephrotic syndrome

ABSTRACT:

Introduction: Vitamin D deficiency is a common metabolic abnormality in children with nephrotic syndrome (NS), primarily caused by urinary loss of vitamin D–binding protein (VDBP). The severity of deficiency may differ depending on steroid responsiveness, with steroid-resistant nephrotic syndrome (SRNS) patients showing more prolonged proteinuria compared to steroid-sensitive nephrotic syndrome (SSNS).

Objective: To compare serum vitamin D levels and proteinuria severity between patients with SRNS and SSNS.

Methods: A cross-sectional study was conducted at Dr. Wahidin Sudirohusodo Hospital, Indonesia, from January to March 2025, involving 36 children aged 1–18 years diagnosed with NS (18 SRNS and 18 SSNS). Serum vitamin D levels were measured, and urinary protein was assessed using a semi-quantitative dipstick and protein–creatinine ratio (PCR). Data were analyzed using appropriate statistical tests to compare vitamin D and proteinuria severity between groups.

Results: Serum vitamin D levels were significantly lower in SRNS compared to SSNS ($p = 0.033$), and all SRNS patients were vitamin D deficient. Almost all SRNS patients (94.4%) had urine PCR ≥ 0.50 g/g, compared to 77.8% in SSNS ($p = 0.148$). Semi-quantitative analysis showed that heavy proteinuria (4+) was more common in SRNS (61.1%) than in SSNS (38.9%) ($p = 0.478$). Among SRNS children, those with nephrotic-range proteinuria had markedly lower vitamin D levels than those with non-nephrotic proteinuria ($p = 0.005$).

Conclusion: Children with SRNS exhibit significantly lower serum vitamin D levels and more severe proteinuria than those with SSNS. Persistent nephrotic-range proteinuria is strongly associated with reduced vitamin D levels. Routine monitoring and appropriate vitamin D supplementation are recommended, particularly for SRNS patients.

1. Introduction

Nephrotic syndrome (NS) is the most frequent glomerular disorder in children, characterized clinically by massive proteinuria, hypoalbuminemia, hyperlipidemia, and edema (Albar & Bilondatu, 2019). Based on steroid response, NS is classified into steroid-sensitive nephrotic syndrome (SSNS) and steroid-resistant nephrotic syndrome (SRNS). While most pediatric patients with NS achieve remission with corticosteroid therapy, approximately 10–20% of patients exhibit resistance and are diagnosed with SRNS (Dewi, Suarta, & Nilawati, 2019). This distinction is

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clinically important because SRNS patients experience prolonged proteinuria, require more aggressive immunosuppressive therapy, and have a significantly higher risk of progression to chronic kidney disease (CKD) (Popa, Balgradean, & Croitoru, 2022).

A common and clinically significant metabolic abnormality in children with NS is vitamin D deficiency. The pathogenesis is multifactorial, but the most substantial contribution arises from urinary loss of vitamin D–binding protein (VDBP) due to increased glomerular permeability, which depletes circulating 25-hydroxyvitamin D (25(OH)D) (Selewski et al., 2016). Previous studies have consistently shown an inverse correlation between the severity of proteinuria and serum vitamin D levels (Kogon et al., 2023). However, the extent of vitamin D deficiency may differ significantly between SRNS and SSNS patients, influenced by factors such as the degree of glomerular injury, disease chronicity, and cumulative exposure to long-term immunosuppressive treatments (Bennett et al., 2016; Omar et al., 2025).

Vitamin D deficiency represents a serious clinical issue because vitamin D plays a crucial role in calcium and phosphate homeostasis, bone mineralization, and immune regulation (Yati, Batubara, & Suryawan, 2018). In children, prolonged vitamin D deficiency is associated with poor linear growth, bone deformities such as rickets, cardiovascular risk, and impaired immune function (Kogon et al., 2023; Yati et al., 2018). Accordingly, both the International Pediatric Nephrology Association (IPNA) and the Kidney Disease: Improving Global Outcomes (KDIGO) 2021 guidelines recommend vitamin D supplementation for patients receiving long-term corticosteroid therapy (Trautmann et al., 2020; KDIGO, 2021). Nonetheless, current clinical recommendations generally do not specify dosage variations based on NS phenotypes, such as SSNS or SRNS.

Despite global recognition of vitamin D deficiency in NS, there remains a lack of comparative data from Indonesia assessing vitamin D status among pediatric patients categorized by steroid responsiveness. Addressing this knowledge gap is essential for refining local management strategies. Therefore, this study was designed to compare serum vitamin D levels and proteinuria severity between children with SRNS and those with SSNS, and to further examine the relationship between serum vitamin D levels and the degree of proteinuria.

2. Methods

Study Design and Setting

This cross-sectional study was conducted in the Mother and Child Center at Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, from January to March 2025. The study population consisted of pediatric patients aged 1–18 years who were diagnosed with nephrotic syndrome (NS) based on the criteria established by the *Kidney Disease: Improving Global Outcomes* (KDIGO, 2021) and the *Konsensus Tata Laksana Sindrom Nefrotik Idiopatik pada Anak* (Trihono et al., 2012). All participants had received treatment for at least two months. Patients were classified as steroid-resistant nephrotic syndrome (SRNS) if no remission was achieved within 4–6 weeks of initial corticosteroid treatment and as steroid-sensitive nephrotic syndrome (SSNS) if complete remission occurred (Trautmann et al., 2020).

Patients who met the inclusion criteria were consecutively recruited from both outpatient and inpatient services. Exclusion criteria included the use of medications known to interfere with vitamin D metabolism (such as antiepileptic or anti-tuberculosis drugs), impaired renal function (estimated glomerular filtration rate < 60 mL/min/1.73 m^2), impaired liver function (elevations in aspartate aminotransferase and alanine aminotransferase enzymes), malabsorption symptoms, or severe malnutrition. Ethical approval for this study was obtained from the Ethics Committee of the Faculty of Medicine, Universitas Hasanuddin, Makassar, Indonesia. Written informed consent was obtained from parents or legal guardians of all participants prior to enrollment.

Data Collection Procedures

Demographic and anthropometric data were recorded through structured clinical assessments and laboratory evaluations using standardized forms. Nutritional status was categorized according to the WHO Child Growth Standards (for ages 1–5 years) and CDC Growth Charts (for ages 5–18 years). Serum vitamin D [25(OH)D] levels were measured using the chemiluminescent immunoassay (CLIA) method from blood samples

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collected in the morning and processed within one hour of collection. Vitamin D status was categorized according to the Indonesian Pediatric Society criteria as deficient (<20 ng/mL), insufficient (20–30 ng/mL), or sufficient (>30 ng/mL) (Yati, Batubara, & Suryawan, 2018).

All subjects were known to be currently receiving routine vitamin D supplementation of 400 IU. Urinary protein was analyzed from morning spot urine specimens using both semi-quantitative dipstick testing (graded as negative, trace, 1+, 2+, 3+, or 4+) and quantitative measurement of the protein–creatinine ratio (PCR) using an automated urine chemistry analyzer. Based on semi-quantitative proteinuria grading, proteinuria levels of "3+" and "4+" were classified as nephrotic range. According to established guidelines, a urine PCR less than 2.0 g/g indicates a normal result, while a urine PCR greater than or equal to 2.0 g/g indicates nephrotic-range proteinuria (Huang et al., 2020). However, the urine analyzer available at our center could only detect a maximum measurable value of ≥0.50 g/g.

Statistical Analysis

All data were processed and analyzed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA). The normality of numerical variables was assessed using the Shapiro–Wilk test. Data with a normal distribution were presented as mean ± standard deviation (SD), while non-normally distributed data were expressed as median (minimum–maximum). Comparisons between the SSNS and SRNS groups were performed using the independent t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Categorical variables were analyzed using the Chi-square test. A p-value < 0.05 was considered statistically significant.

3. Results

Subject Characteristic

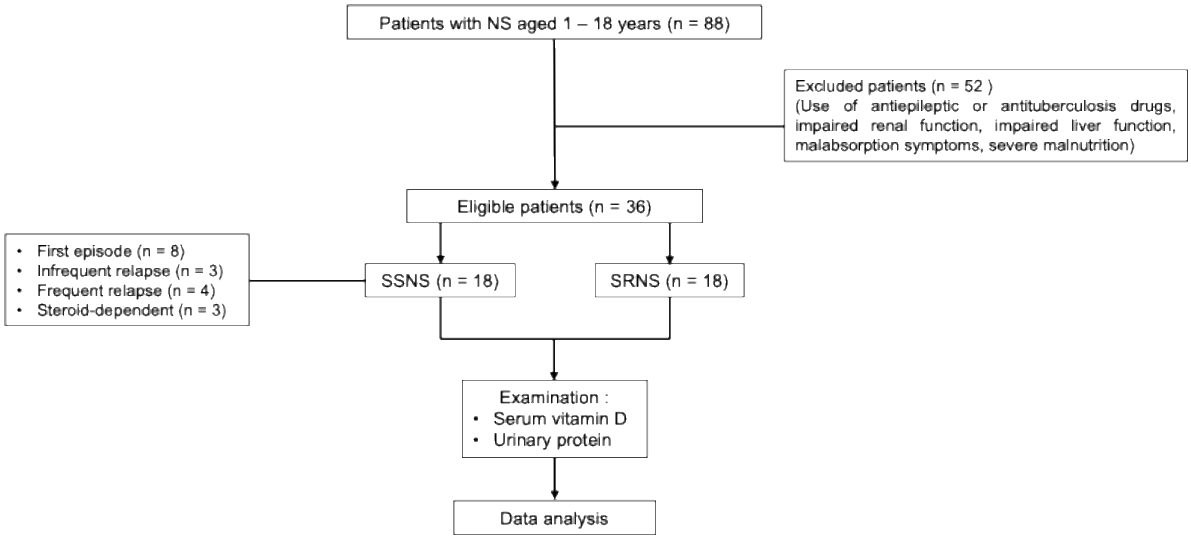

Figure 1. Research schematic diagram

A total of 88 children aged 1–18 years diagnosed with NS were initially screened **(Figure 1)**. Of these, 52 patients were excluded due to the use of antiepileptic or antituberculosis medications, presence of renal or liver disorders, symptoms of malabsorption, or severe malnutrition. Thirty-six eligible patients met the inclusion criteria and were enrolled in the study, consisting of 18 patients with SSNS and 18 with SRNS. The SSNS group consisted of 8 first-episode cases, 3 with infrequent relapses, 4 with frequent relapses, and 3 who were steroid-dependent. All enrolled participants

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underwent examinations, including serum vitamin D and urinary protein measurements. The collected data were then analyzed to compare biochemical parameters between the SSNS and SRNS groups. The mean age of SRNS patients was 12.17 ± 4.60 years, slightly older than SSNS (9.78 ± 5.36 years). The sex distribution was similar, though males predominated in SSNS (77.8%) (Table 1). Most children had normal nutritional status (61.1% SRNS; 72.2% SSNS). (**Table 1**).

Table 1. Characteristics of study subjects

Variable	SRNS (n = 18)	SSNS (n = 18)
Age (years), mean ± SD	12.17 ± 4.60	9.78 ± 5.36
Sex, n (%)		
Male	9 (50.0)	14 (77.8)
Female	9 (50.0)	4 (22.2)
Nutritional status, n (%)		
Undernourished	1 (5.6)	2 (11.1)
Normal	11 (61.1)	13 (72.2)
Overweight	6 (33.3)	3 (16.7)

Comparison of Serum Vitamin D and Proteinuria Severity

As shown in **Table 2**, serum vitamin D levels were significantly lower in SRNS than SSNS (median 7.0 ng/mL vs 15.0 ng/mL; p = 0.033). All SRNS patients (100%) had vitamin D deficiency, while 22.2% of SSNS were insufficient and 77.8% deficient (p = 0.034). Regarding urinary protein, almost all SRNS patients (94.4%) had a urine PCR ≥ 0.50 g/g, whereas this proportion was 77.8% in SSNS (p = 0.148). Semi-quantitative analysis further demonstrated that heavy proteinuria (4+) was more frequent in SRNS (61.1%) than in SSNS (38.9%) (p = 0.478).

Table 2. Comparison of serum vitamin D levels, vitamin D status, urine PCR, and semi-quantitative proteinuria grading in patients with SRNS and SSNS

Variable	SRNS (n = 18)	SSNS (n = 18)	p-value
Serum vitamin D (ng/mL), median (min–max)	7.00 (2.00 – 17.00)	15.00 (3.00 – 23.00)	**0.033[a]**
Vitamin D Status, n (%)			**0.034[b]**
Normal	0 (0)	0 (0)	
Insufficient	0 (0)	4 (22.2)	
Deficient	18 (100)	14 (77.8)	
Urine PCR, n (%)			0.148[b]
<0.20 g/g	1 (5.6)	4 (22.2)	
0.20 – 0.49 g/g	0 (0)	0 (0)	
≥ 0.50 g/g	17 (94.4)	14 (77.8)	

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Semi-quantitative urine protein, n (%)			0.478[b]
Negative Trace	1 (5.6) 0 (0)	4 (22.2) 0 (0)	
1+	2 (11.1)	1 (5.6)	
2+	3 (16.7)	4 (22.2)	
3+	1 (5.6)	2 (11.1)	
4+	11 (61.1)	7 (38.9)	

[a]Mann-Whitney test

[b]Chi-square test

Vitamin D Levels by Proteinuria Grading

Table 3 and Table 4 demonstrate the comparison of serum vitamin D levels according to urinary protein excretion in children with SRNS and SSNS. As shown in **Table 3**, patients with SRNS who had nephrotic range proteinuria exhibited markedly lower serum vitamin D concentrations compared with those in the non-nephrotic range (median 4.00 ng/mL [2.00–9.00] vs 11.0 ng/mL [6.00–17.00]; p = 0.005). This result indicates a significant inverse association between the severity of proteinuria and vitamin D status among SRNS patients. In contrast, among the SSNS group, serum vitamin D levels also tended to be lower in the nephrotic range subgroup (median 7.00 ng/mL [3.00–20.00]) than in the non-nephrotic subgroup (median 18.0 ng/mL [4.00–23.00]); however, this difference did not reach statistical significance (p = 0.062).

As presented in **Table 4**, when serum vitamin D levels were compared based on quantitative proteinuria, a significant difference (p = 0.022) was observed among SSNS patients. Children with urine PCR ≥ 0.20 g/g had markedly lower median serum vitamin D levels (4.00 ng/mL [3.00–15.00]) compared to those with urine PCR < 0.20 g/g (16.0 ng/mL [8.00–20.00]). Among SRNS patients, the difference in vitamin D levels between the two urine PCR categories was not statistically significant (p = 0.382), likely due to the small number of subjects in the non-nephrotic PCR subgroup (n = 1).

Table 3. Comparison of serum vitamin D levels based on semi-quantitative proteinuria grading in patients with SRNS and SSNS

Group	**Serum Vitamin D (ng/mL)**		**p-value**
	Non-nephrotic Range	**Nephrotic Range**	
SRNS, median (min–max)	11.0 (6.00–17.00)	4.00 (2.00–9.00)	**0.005[a]**
SSNS, median (min–max)	18.0 (4.00–23.00)	7.00 (3.00–20.00)	0.062[a]

[a]Mann-Whitney test

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Table 4. Comparison of serum vitamin D levels based on quantitative proteinuria grading in patients with SRNS and SSNS

Group	Serum Vitamin D (ng/mL)		p-value
	Urine PCR < 0.20 g/g	Urine PCR ≥ 0.20 g/g	
SRNS, median (min–max)	n = 1 (9.00)	7.00 (2.00–17.00)	0.382[a]
SSNS, median (min–max)	16.0 (8.00–20.00)	4.00 (3.00–15.00)	**0.022**[a]

[a]Mann-Whitney test

4. Discussion

We observed a significantly higher prevalence of vitamin D deficiency among patients with SRNS compared with those with SSNS. The SRNS group had a median serum vitamin D concentration of only 7.0 ng/mL, which was notably lower than that of the SSNS group. Furthermore, all SRNS patients met the criteria for vitamin D deficiency despite receiving daily vitamin D supplementation of 400 IU. These findings highlight that the more refractory disease course seen in SRNS may predispose patients to a more severe depletion of vitamin D. Our analysis of proteinuria severity provides further insight into the relationship between urinary protein loss and vitamin D status. Among patients with SRNS, those presenting with nephrotic range proteinuria (semi-quantitative "3+" and "4+" category) had markedly lower median serum vitamin D level compared with those exhibiting non-nephrotic proteinuria. In contrast, although patients with SSNS also demonstrated a tendency toward lower vitamin D levels with increasing proteinuria, the difference did not reach statistical significance. When proteinuria was quantified using the urine PCR, SSNS patients with PCR ≥ 0.20 g/g showed significantly lower serum vitamin D levels than those with PCR < 0.20 g/g, whereas no statistically significant difference was observed among SRNS patients, likely attributable to the small number of subjects in the non-nephrotic PCR subgroup.

The association between heavy proteinuria and reduced vitamin D levels is biologically plausible. In nephrotic syndrome, massive urinary protein losses involve not only albumin but also VDBP and the albumin-bound fraction of 25(OH)D, resulting in decreased circulating concentrations of total vitamin D metabolites (Bennett et al., 2016). Moreover, the persistent glomerular leakage characteristic of SRNS likely causes greater and more sustained losses of VDBP–25(OH)D complexes, further exacerbating vitamin D deficiency. This mechanism may explain why all children in our SRNS group exhibited vitamin D deficiency. In contrast, children with SSNS, who experience intermittent proteinuria and periods of remission, may retain a partial ability to restore vitamin D stores, thereby demonstrating a gradient in vitamin D levels corresponding to proteinuria severity (Selewski et al., 2016; Yang et al., 2021). These findings are consistent with prior studies reporting an

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inverse relationship between proteinuria and serum vitamin D concentrations in pediatric nephrotic syndrome. Bennett et al. demonstrated that urinary VDBP excretion was markedly increased in patients with SRNS (Bennett et al., 2016). Banerjee et al. further observed significantly lower free and total vitamin D levels during active disease compared to remission in steroid-sensitive cases (Banerjee et al., 2020). Maji et al. reported that nearly 80% of children with nephrotic syndrome were vitamin D deficient with lower levels in those with longer duration of illness, like children who were frequent relapsing (FRNS) or delayed in achieving remission (SRNS) (Maji et al., 2022). Persistent deficiency in SRNS may also reflect longer proteinuria duration and chronic corticosteroid exposure, which can impair vitamin D metabolism (Lee et al., 2021; Beins & Dell, 2015). Our study of children with NS showed no individuals achieving a vitamin D level considered 'adequate' by most international standards (>30 ng/mL). Given that both groups are at risk, the findings emphasize that nephrotic syndrome in children is associated with profound disturbance of vitamin D status beyond what might be expected in otherwise healthy children.

From a clinical standpoint, these results highlight the importance of routine vitamin D assessment and individualized supplementation in children with NS, particularly those with SRNS and sustained nephrotic-range proteinuria. Optimizing vitamin D status may not only support skeletal health but also exert immunomodulatory and podocyte-protective effects, potentially contributing to improved disease outcomes (Sîrbe et al., 2022; Adamantidi et al., 2024; Shi et al., 2018). This study has several limitations. The cross-sectional design restricts the ability to establish causal relationships between variables. Additionally, the study was conducted at a single tertiary hospital, which may limit the generalizability of the findings to broader populations. The relatively small sample size, particularly within specific subgroups, may have reduced the statistical power and contributed to some non-significant results. Future multicenter studies with larger cohorts and longitudinal follow-up are recommended to better assess the causal relationship between vitamin D status and disease severity, as well as to evaluate the impact of vitamin D correction on clinical outcomes.

5. CONCLUSION

Children with SRNS exhibit significantly lower serum vitamin D concentrations and more severe proteinuria compared with those with SSNS, indicating that the persistence and intensity of protein loss play a crucial role in worsening vitamin D deficiency. The continuous urinary loss of vitamin D–binding protein (VDBP) and albumin-bound 25(OH)D in SRNS patients leads to a sustained depletion of circulating vitamin D metabolites, which may further impair bone metabolism, immune regulation, and podocyte stability. Persistent nephrotic-range proteinuria, as commonly observed in SRNS, amplifies this loss, creating a cycle of vitamin D deficiency and disease progression. Consequently, these findings emphasize the importance of routine monitoring of serum vitamin D levels and individualized supplementation strategies, particularly for SRNS patients who are at greater risk of prolonged deficiency. Tailored

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vitamin D replacement therapy, alongside optimal management of proteinuria, may help improve not only skeletal outcomes but also the overall renal prognosis and quality of life in affected children.

6. AUTHORS' CONTRIBUTION

Conceptualization, R.A.S. and S.R.; methodology, software, validation, formal analysis, investigation, resources, data curation, R.A.S., S.R., and E.A.; writing original draft preparation, R.A.S., S.R., E.A.; writing review and editing, M.M., J., U.; visualization, R.A.S.; supervision, S.R. and J; project administration, E.A.; funding acquisition, R.A.S. All authors have read and agreed to the published version of the manuscript.

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