Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(5), 3132-3139 | ISSN:2251-6727

Effect of Vitamin D3 Supplementation on Serum Interleukin 6 and Hepcidin Levels in Child with Chronic Kidney Disease

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(Received: 25 August 2025 Revised: 27 September 2025 Accepted: 14 October 2025)

KEYWORDS

Vitamin D3, Hepcidin, Interleukin 6, Chronic Kidney Disease, Children

ABSTRACT:

Introduction: Chronic kidney disease (CKD) is a global health issue, leading to various complications. Chronic inflammation is common in children with CKD shown by increased interleukin 6 and hepcidin level and decreased vitamin D level. Low vitamin D levels also contribute to hepcidin and interleukin 6 homeostasis.

Objective: To know the effect of vitamin D3 supplementation on serum hepcidin and interleukin 6 levels in children with CKD.

Methods: A prospective cohort design was conducted in Wahidin Sudirohusodo Hospital from September to December 2024. Samples were divided into two groups based on GFR. Complete blood count, vitamin D, interleukin 6, and hepsidin level examination were perform before and after supplementation. The data were analyzed with Mann-Whitney Test and Logistic Biner Regression.

Results: Total of 43 children (2 – 18 years old) were included in this study. Characteristics of age, gender, nutritional status, and diagnosis between groups were not significantly different ($p > 0.05$). After supplementation, vitamin D levels significantly increased in both groups ($p = 0.045$), while hepcidin levels and interleukin 6 levels don't decreased in both group I ($p = 0,56$; p value = 0,082).

Conclusion: Vitamin D 2000 IU supplementation for 6 months increases vitamin D levels in children with CKD but not with hepcidin and interleukin 6 levels.

1. Introduction

Every year, the prevalence and mortality rates of chronic kidney disease (CKD), a condition that also affects children, continue to rise (Becherucci et al., 2016; Van Biljon & Meyers, 2015). Chronic inflammation is one of the fundamental processes of CKD, leading to reduced erythropoiesis and impaired iron mobilization and absorption due to the liver's increased synthesis of hepcidin (Lee et al., 2019). Interleukin-6 (IL-6) is one of the inflammatory cytokines produced as a result of CKD and serves as an inducer of hepcidin (Charlebois & Pantopoulos, 2021). Administration of vitamin D3 has been shown to regulate the expression of hepcidin and IL-6, which promotes hepcidin synthesis (Icardi et al., 2013; Lestari, n.d.). However, limited research has been conducted on how vitamin D3 supplementation affects interleukin and hepcidin levels among pediatric CKD patients in Indonesia.

Globally, in 2019, 13.4% of the world's population was estimated to have CKD, with a significant proportion being children. In Indonesia, data collected from 14 hospitals between 2007 and 2009 showed that 150 children were diagnosed with CKD (Kementerian Kesehatan RI, 2018). Children with CKD often experience vitamin D deficiency. Coccia et al. (2017) found that the prevalence of vitamin D deficiency among pediatric CKD patients in the U.S. and Europe ranged between 30% and 50%. Similarly, a study conducted in Ohio among 182 children aged 5 to 21 years with CKD reported that 50% suffered from vitamin D deficiency.

Patients with CKD frequently exhibit chronic inflammation, characterized by low vitamin D levels and elevated hepcidin and IL-6 concentrations (Meireles et al., 2016). This occurs due to uremic conditions and metabolic acidosis that lead to oxidative and carbonyl stress, which are highly proinflammatory, thereby triggering immune dysregulation and inflammatory

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JCHR (2025) 15(5), 3132-3139 | ISSN:2251-6727

activation in CKD (Akchurin & Kaskel, 2015). CKD patients undergoing dialysis are prone to thrombotic events and infections such as catheter-related bloodstream infections, dialysis access site infections, and peritonitis, all of which can trigger further inflammatory responses (Akchurin et al., 2019). Additionally, intestinal dysbiosis and increased translocation of intestinal bacteria and their components into the bloodstream lead to systemic inflammation. High levels of circulating cytokines also occur due to decreased renal clearance (Akchurin et al., 2019).

Interleukin-6 is among the inflammatory cytokines generated under these pathological conditions. The expression of HAMP (Hepcidin Antimicrobial Protein) mRNA, which encodes hepcidin, is primarily triggered by inflammatory responses and iron levels. When IL-6 binds to its receptor in hepatocytes, JAK1/2 kinase phosphorylates STAT3, which then translocates to the nucleus to initiate hepcidin transcription (Wrighting & Andrews, 2006; Su et al., 2017). When 25(OH)D is converted into 1,25(OH)D—the active form of vitamin D3—glomerular mitochondrial activity increases. However, in CKD, this synthesis is inhibited, resulting in impaired immunomodulatory effects of vitamin D3.

Vitamin D3 supplementation has been shown to decrease hepcidin mRNA expression and the release of pro-inflammatory cytokines IL-6 and IL-1β (Smith et al., 2017). The immunomodulatory effects of vitamin D occur through several mechanisms, including paracrine feedback pathways that enhance T-helper cell function and reduce inflammation. Prior studies have demonstrated that vitamin D3 is safe and leads to reduced memory B cells, increased regulatory T cells (Tregs), and decreased Th1 and Th17 effector cells (Subandiyah et al., 2019). In cultures of human peripheral blood-derived CD4 T cells, the active form of vitamin D3 has also been shown to elevate IL-10 and Foxp3+ expression.

It is believed that vitamin D3 contributes to achieving hepcidin balance by regulating inflammatory factors—particularly IL-6—thereby improving renal inflammation (Chau & Kumar, 2012; Goyal et al., 2018). Considering that vitamin D3 may help control the inflammatory response in CKD, this study is expected to enrich scientific knowledge, provide a reference for future research, and highlight the importance of vitamin D3 supplementation and nutritional enhancement, particularly for children suffering from CKD.

2. Methods

This study is an analytic observational study with a prospective cohort design aimed at determining the effect of vitamin D3 supplementation on serum levels of vitamin D, hepcidin, and interleukin-6 in children with chronic kidney disease (CKD) (Becherucci et al., 2016; Van Biljon & Meyers, 2015). The research was conducted in the pediatric nephrology inpatient and outpatient wards at Wahidin Sudirohusodo Hospital from September to December 2024. Subjects were recruited using a consecutive sampling technique, involving children aged 2–18 years diagnosed with CKD who were admitted to or treated in the pediatric nephrology wards (Lee et al., 2019; Coccia et al., 2017).

Blood samples (3 cc) were drawn from the patients' peripheral veins, centrifuged for 30 minutes, and then placed in a cooler box containing ice packs at a temperature of 2–8°C before being transported to the laboratory for analysis (Libowo et al., 2020; Esfandiar et al., 2019). The laboratory analyses were conducted to assess changes in serum vitamin D, hepcidin, and interleukin-6 levels after supplementation (Akchurin & Kaskel, 2015; Smith et al., 2017; Goyal et al., 2018).

Data analysis was performed using the Mann-Whitney test and Binary Logistic Regression to determine the statistical significance of vitamin D3 supplementation effects (Pistis et al., 2023; Kamboj et al., 2023). This study was reviewed and approved by the Ethical Committee of the Faculty of Medicine, Hasanuddin University (Lestari, 2024).

3. Results

Forty-three pediatric patients with chronic kidney disease (CKD) aged between 2 and 18 years participated in this study (Becherucci et al., 2016; Van Biljon & Meyers, 2015). The subjects were categorized into two groups based on their glomerular filtration rate (GFR). Group I consisted of 22 children with GFR ≥ 90 mL/min/1.73 m², while Group II consisted of 21 children with GFR < 90 mL/min/1.73 m². Both groups showed no significant differences in terms of age, gender, nutritional status, or diagnosis ($p > 0.05$) (Libowo et al., 2020; Rai, 2019). The characteristics of both groups are shown in Table 1.

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Table 1. Characteristics of Research Subjects

Characteristics	Group I (n=22)	Group II (n=21)	ρ-value
Gender	Male 9 (40.9%) / Female 13 (59.1%)	Male 11 (52.4%) / Female 10 (47.6%)	0.654
Age (years), Mean (SD)	12.23 (5.40)	13.63 (4.56)	0.214
Nutritional status	Underweight 3 (13.6%), Good 15 (68.2%), Overweight 4 (18.2%)	Underweight 4 (19.0%), Good 16 (76.2%), Overweight 1 (4.8%)	0.377
Comorbidities	Lupus nephritis, steroid-resistant nephrotic syndrome, non-steroid resistant nephrotic syndrome, rapidly progressive glomerulonephritis, nephrotic syndrome, relapsing nephrotic syndrome, CAKUT	–	0.056

Before vitamin D supplementation, the median vitamin D levels were 21.00 ng/mL in the group with GFR ≥ 90 mL/min/1.73 m² and 16.00 ng/mL in the group with GFR < 90 mL/min/1.73 m², with no significant difference (ρ = 0.089). The median hepcidin levels were 1293.7 ng/mL and 1178.12 ng/mL, respectively, also showing no significant difference (ρ = 0.421). Similarly, the interleukin-6 (IL-6) levels showed no significant difference between the two groups (ρ = 0.576) (Akchurin et al., 2019; Goyal et al., 2018; Smith et al., 2017). These comparisons are shown in Table 2.

Table 2. Comparison of Serum Levels of Vitamin D, Hepcidin, and Interleukin-6 Before Supplementation

Characteristic	GFR ≥ 90 mL/min/1.73 m² Median (Min–Max)	GFR < 90 mL/min/1.73 m² Median (Min–Max)	ρ-value
Vitamin D (ng/mL)	21.00 (8.00–53.00)	16.00 (4.00–30.00)	0.089
Hepcidin (ng/mL)	1293.7 (71.78–2402.47)	1178.12 (38.58–8523.04)	0.421
Interleukin-6 (pg/mL)	133.85 (0–780.01)	134.90 (0–688.41)	0.576

After supplementation with 2000 IU of vitamin D_3, median vitamin D levels increased to 30.00 ng/mL in the GFR ≥ 90 mL/min/1.73 m² group and 26.00 ng/mL in the GFR < 90 mL/min/1.73 m² group, showing a significant difference (ρ = 0.047). Hepcidin and IL-6 levels, however, did not show statistically significant differences between the groups (ρ = 0.560 and 0.082, respectively) (Icardi et al., 2013; Atkinson et al., 2017; Kamboj et al., 2023). The results are summarized in Table 3.

Table 3. Comparison After Vitamin D_3 Supplementation

Characteristic	GFR ≥ 90 mL/min/1.73 m² Median (Min–Max)	GFR < 90 mL/min/1.73 m² Median (Min–Max)	ρ-value
Vitamin D (ng/mL)	30.00 (22.00–62.00)	26.00 (18.00–54.00)	0.047

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Hepcidin (ng/mL)	835.37 (1.11–2207.47)	746.81 (0.42–5495.89)	0.560
Interleukin-6 (pg/mL)	113.95 (0–785.30)	154.35 (–6.83–520.65)	0.082

Following supplementation, there was an overall improvement in vitamin D status in both groups (Coccia et al., 2017; Esfandiar et al., 2019; Dyussenova et al., 2021). Among children with GFR ≥ 90 mL/min/1.73 m², 7 initially had vitamin D deficiency (5 improved to insufficiency and 2 reached sufficiency), while 9 with insufficiency improved to sufficiency in 4 cases. In children with GFR < 90 mL/min/1.73 m², 8 were initially deficient, with 7 improving to insufficiency and 1 reaching sufficiency (ρ = 0.002 and ρ = 0.000, respectively). These results are presented in Table 4.

Table 4. Changes in Vitamin D Status After Supplementation

Pre-supplementation	Post-supplementation	Total	ρ-value
GFR ≥ 90 – Deficiency	5 insufficiency / 2 sufficiency	7	0.002
GFR ≥ 90 – Insufficiency	5 insufficiency / 4 sufficiency	9	
GFR ≥ 90 – Sufficiency	0 insufficiency / 5 sufficiency	5	
GFR < 90 – Deficiency	7 insufficiency / 1 sufficiency	8	0.000
GFR < 90 – Insufficiency	7 insufficiency / 6 sufficiency	13	
GFR < 90 – Sufficiency	0 insufficiency / 1 sufficiency	1	

Changes in the median vitamin D levels were 9.00 ng/mL in the group with GFR ≥ 90 mL/min/1.73 m² and 10.00 ng/mL in the group with GFR < 90 mL/min/1.73 m², with no significant difference (ρ = 0.421). Similarly, changes in hepcidin and IL-6 levels showed no significant differences between groups (ρ = 0.903 and 0.590, respectively) (Pistis et al., 2023; Chau & Kumar, 2012; Su et al., 2017). The comparative results are summarized in Table 5.

Table 5. Changes in Serum Vitamin D, Hepcidin, and Interleukin-6 After Supplementation

Characteristic	GFR ≥ 90 mL/min/1.73 m² Median (Min–Max)		GFR < 90 mL/min/1.73 m² Median (Min–Max)	ρ-value
Vitamin D (ng/mL)	9.00 (3.00–38.00)		10.00 (3.00–35.00)	0.421
Hepcidin (ng/mL)	458.33 (–1521.36–4548.15)		431.31 (–7799.21–974.35)	0.903
Interleukin-6 (pg/mL)	19.90 (–113.47–161.58)		19.45 (–167.76–216.72)	0.590

4. Discussion

Chronic kidney disease (CKD) is characterized by structural damage to the kidneys and a decline in glomerular filtration rate (GFR) to less than 60 ml/min per 1.73 m² for more than three months (Becherucci et al., 2016). In this survey, the 13–18 age group represented the largest proportion in both groups, consistent with the findings of Libowo, Widiasta, and Rachmadi (2020), who reported that 80.65% of pediatric CKD patients were aged between 10 and 18 years. Because creatinine generation correlates with muscle mass, children and adolescents typically show higher

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creatinine levels as they age, resulting in different estimated GFRs (eGFR) compared with adults (Rai, 2019).

In this study, 52.4% of boys and 47.6% of girls were in the first group of CKD-affected children, compared with 40.9% of boys and 59.1% of girls in the second group. These findings align with the research of Pradeep et al. (2019), which indicated an almost equal gender distribution among pediatric CKD patients (50.8% male and 49.2% female). Similarly, Libowo, Widiasta, and Rachmadi (2020) found that 54.37% of respondents were male. The majority of CKD children in both groups had good nutritional status, consistent with Metasyah and Hidayati (2023), who reported that childhood obesity does not increase CKD risk. Most children with GFR ≥ 90 mL/min/1.73 m² were diagnosed with lupus nephritis, whereas those with GFR < 90 mL/min/1.73 m² were classified in CKD stages 3–5. Libowo, Widiasta, and Rachmadi (2020) also found that steroid-resistant nephrotic syndrome accounted for 58.25% of pediatric CKD cases, while Wenderfer, Orjuela, and Dionne (2023) reported that lupus nephritis contributed to 15% of juvenile CKD cases.

Both groups of CKD children in this study showed elevated interleukin-6 (IL-6) and hepcidin levels, along with vitamin D deficiency or insufficiency. Children with GFR ≥ 90 mL/min/1.73 m² had lower IL-6, higher vitamin D, and increased hepcidin compared to those with GFR < 90 mL/min/1.73 m². Coccia et al. (2017) observed that 32% of CKD patients under 19 years had vitamin D deficiency, and Lee et al. (2023) demonstrated that deficiency prevalence increases as CKD progresses. Vitamin D insufficiency is often linked to dietary restrictions and reduced physical activity among children with severe CKD (Dyussenova et al., 2021). Moreover, proteinuria contributes to the urinary loss of vitamin D metabolites, impairing 25(OH)D transport and activation (Esfandiar, Shakiba, & Mirzaei, 2019). Uremia, acidosis, and high phosphate or FGF-23 levels further reduce 1-α hydroxylase activity, decreasing active vitamin D synthesis (Icardi et al., 2013).

Children with CKD undergoing dialysis often exhibit elevated hepcidin levels (Santos-Silva et al., 2019; Aras et al., 2021). Inflammation stimulates hepcidin through cytokines such as IL-1β and IL-6 via the JAK/STAT3 and BMP-6/SMAD pathways (Wrighting & Andrews, 2006). Endoplasmic reticulum stress can further enhance hepcidin expression (Charlebois & Pantopoulos, 2021). Akchurin and Kaskel (2015) found that children receiving hemodialysis produced more IL-6 than healthy peers, confirming that inflammation and decreased renal clearance elevate cytokine levels. Uremic conditions amplify oxidative and carbonyl stress, exacerbating inflammation (Su, Lei, & Zhang, 2017; Kristina Kardani et al., 2021). Additionally, CKD patients—especially those on dialysis—are more susceptible to infections and thrombosis, contributing to chronic inflammatory stimuli (Fitzgerald et al., 2016).

Vitamin D supplementation (2000 IU) raised serum vitamin D levels in both groups in this study but did not affect IL-6 or hepcidin concentrations. CKD children with GFR ≥ 90 mL/min/1.73 m² showed a greater improvement than those with GFR < 90 mL/min/1.73 m². Similar observations were made by Iyengar et al. (2022) and Nadeem et al. (2021), who reported that children with glomerular disease required higher vitamin D doses over longer periods to achieve optimal levels. Panwar et al. (2018) demonstrated that vitamin D affects hepcidin directly by suppressing HAMP gene transcription and indirectly by inhibiting pro-inflammatory cytokines that stimulate hepcidin synthesis (Smith et al., 2017). However, Pistis et al. (2023) and Kamboj et al. (2023) found no significant reduction in hepcidin after supplementation, likely due to persistent inflammation and iron therapy in CKD.

Vitamin D deficiency and inflammation commonly coexist in dialysis patients (Meireles et al., 2016). Vitamin D supplementation at 50,000 IU twice weekly for 12 weeks improved vitamin D levels and reduced IL-6 and CRP (Meireles et al., 2016). This supplementation also enhanced CYP27B1 and vitamin D receptor expression in monocytes, which are key to immune regulation (Coccia et al., 2017). Vitamin D therapy can suppress IL-6 and IL-1β secretion (Subandiyah, Ghofar, & Fitri, 2019) and downregulate inflammatory pathways including TGF, PKC, MAPK1, TNFα, and IFN-γ (Kashani et al., 2018). By improving liver function and lipid metabolism, vitamin D reduces inflammation and mortality risk (Sharif, 2022). However, in CKD patients with GFR < 90 mL/min/1.73 m², IL-6 levels increased after 2000 IU vitamin D for six weeks, indicating that higher doses and longer duration may be necessary.

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Atkinson et al. (2018) noted that while CRP levels declined after 12 weeks of vitamin D therapy, IL-6 levels did not significantly change. Similarly, no notable reduction in hepcidin levels was observed. Despite these findings, vitamin D may still influence inflammation, though the effects are limited by small sample sizes and low baseline cytokine levels. Overall, short-term vitamin D supplementation appears to have minimal impact on inflammatory status in CKD children. IL-6 levels may also be affected by dialysis processes, steroid therapy, and infection risk in nephrotic syndrome (Altemose et al., 2018).

5. RESEARCH LIMITATIONS

This study has limitations in that it did not conduct an adjustment analysis related to confounding factors such as diet, sun exposure, calcium levels, and the incidence of infection. This study also only conducted a supplementation study at one dose, namely 2000 IU, and a relatively short measurement period after the intervention.

6. CONCLUSION

Vitamin D supplementation effectively increases vitamin D levels in children with CKD but not with inflammatory status presented with hepcidin and interleukin-6. Vitamin D supplementation with longer duration and higher dosage should be considered in next research in children with CKD.

7. AUTHORS' CONTRIBUTION

Jusli Aras, Syarifuddin Rauf, and Ema Alasiry made significant contributions to the conception and design of the manuscript, while Nicholas Redly was involved in the acquisition, analysis, and interpretation of the data. All authors participated in drafting the manuscript, and Nicholas Redly provided critical revisions. Each author reviewed and approved the final version of the manuscript. All authors contributed equally to the manuscript and confirmed their approval of the final version.

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