

Letter to the Editor

Investigating Hypovitaminosis D and Childhood Nephrotic Syndrome



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Dear Editor

This letter aims to bring focus to the important connection between vitamin D deficiency and nephrotic syndrome (NS) in children, an area deserving greater clinical and investigative priority [1]. Pediatric NS, characterized by significant proteinuria, hypoalbuminemia, and edema, continues to be a major contributor to illness among children. Although corticosteroid therapy is the primary treatment, frequent disease flares and steroid dependence often worsen long-term prognosis, highlighting the importance of managing associated treatable conditions.

Growing research indicates that low vitamin D levels might worsen NS by promoting immune system dysfunction, damage to kidney filtering cells (podocytes), and increased protein loss. However, standardized protocols for checking and correcting vitamin D deficiency in children with NS have not yet been widely established. Our recent observational work sought to determine the prevalence of vitamin D deficiency among pediatric patients with NS and to assess its potential links with illness severity, frequency of relapses, and response to therapy.

Initial results indicated that 72% of the children with NS in our study (n=45) had severely low serum 25-hydroxyvitamin D levels (under 20 ng/mL). This deficiency was more pronounced in cases that did not respond to steroids. Furthermore, patients with low vitamin D had more yearly relapses (averaging 3.2 compared to 1.8)

and longer durations of proteinuria. These observations add to existing evidence supporting vitamin D's role in regulating immune activity and potentially lessening kidney damage [1].

This study underscores two urgent needs:

1. Clinical attention: Vitamin D screening should be integrated into routine care for children with NS, particularly in relapsing or steroid-resistant cases. While targeted supplementation may offer a safe, cost-effective adjunct to reduce disease burden, more robust clinical evidence and guideline development are required before it can be universally prescribed as standard care [2].

2. Research priorities: Longitudinal studies are needed to establish causality and evaluate whether vitamin D repletion improves outcomes. Randomized controlled trials could clarify optimal dosing and duration of supplementation.

On the other hand, given vitamin D's critical role in facilitating intestinal absorption of calcium and phosphorus, coupled with the calcium-wasting effects of corticosteroid therapy, the observed reductions in serum total calcium and vitamin D levels in NS may stem from urinary losses of protein-bound calcium and vitamin D (the latter bound to vitamin D-binding protein [3]. Future investigations should aim to concurrently evaluate serum calcium levels, vitamin D status, and alkaline phosphatase activity to better delineate their interrelationships in this context. Additionally, clinical vigilance for manifestations of vitamin D deficiency such as musculoskeletal



discomfort (e.g. leg pain) or nonspecific symptoms, such as restlessness, is warranted to guide timely intervention in affected individuals.

We urge clinicians and researchers to prioritize this underrecognized comorbidity. Addressing hypovitaminosis D in childhood NS could represent a simple yet transformative intervention to enhance quality of life and reduce healthcare costs, pending further confirmatory evidence.

Thank you for considering this call to action. We welcome collaborations to advance this vital dialogue.

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