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Original Research Article

Role of Vitamin D in Health and Diseases in Children: A Hospital-Based Observational Study

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Abstract

Background: Vitamin D is essential for skeletal mineralization and has immunomodulatory effects that may influence infections, wheeze/asthma, anemia, and growth in children. Despite abundant sunlight, vitamin D deficiency remains common in South Asia.

Aim: To estimate vitamin D status in children attending JNMCH Bhagalpur and evaluate associations with selected clinical and biochemical outcomes.

Methods: Hospital-based observational study of 120 children (1–18 years) recruited from 20 February 2025 to 15 January 2026. Demographic, dietary and sunlight exposure history, anthropometry, and clinical assessment were recorded. Serum 25-hydroxyvitamin D [25(OH)D] and relevant biochemical markers were assessed. Vitamin D categories were defined using standard pediatric cut-offs. Associations with recurrent acute respiratory infections (ARI), wheeze/asthma, anemia, and clinical rickets signs were evaluated using bivariate tests and multivariable logistic regression.

Results: Mean 25(OH)D was 17.6 ± 9.5 ng/mL; 63.3% had 25(OH)D <20 ng/mL. Deficiency was higher in winter (76.7% vs 48.3%, p=0.0026). Severe deficiency was strongly associated with clinical rickets signs (p<0.001) and higher alkaline phosphatase. Hemoglobin differed significantly across vitamin D categories (ANOVA p=0.0012), and vitamin D deficiency independently predicted anemia (adjusted OR 3.75; 95% CI 1.48–9.53; p=0.005).

Conclusion: Vitamin D deficiency was highly prevalent in this hospital-based pediatric sample, with clinically meaningful associations with rickets phenotype and anemia. Targeted screening and guideline-based supplementation for high-risk children may be warranted.

Keywords: 25-hydroxyvitamin D; children; anemia; rickets; wheeze; respiratory infections; India; Bihar.

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Introduction

Vitamin D is a secosteroid hormone central to calcium–phosphate homeostasis and skeletal mineralization across childhood, a period characterized by rapid growth and high mineral demand. Inadequate vitamin D leads to impaired intestinal calcium absorption, secondary hyperparathyroidism, defective bone mineralization, and the clinical spectrum of nutritional rickets and osteomalacia.

Contemporary pediatric guidance recognizes nutritional rickets as largely preventable through supplementation, food fortification, and adequate dietary calcium intake, yet it persists globally and disproportionately affects low- and middle-income settings.[1,2]

Serum 25-hydroxyvitamin D [25(OH)D] is the accepted biomarker for vitamin D status because it integrates endogenous synthesis and dietary intake. However, the field remains debated regarding optimal thresholds for “sufficiency” beyond bone health. While earlier clinical practice guidance frequently used ≥30 ng/mL as a sufficiency target for various outcomes, more recent consensus discussions and guidelines emphasize uncertainty

in disease-prevention targets and caution against routine population-wide testing without specific clinical indications.[3,4] Nonetheless, there is broad agreement that very low 25(OH)D levels increase rickets risk in children, particularly when paired with insufficient calcium intake.[2,5]

Paradoxically, vitamin D deficiency is common in sun-rich regions. In India, nationally representative data and multicenter studies document substantial burden across age groups, with strong influences of season, urban residence, adolescent age, female sex, diet patterns, and reduced outdoor activity.[6–8] The Comprehensive National Nutrition Survey (CNNS) highlighted micronutrient deficiencies across childhood and adolescence and reinforced that vitamin D deficiency occurs even in the context of abundant sunlight, reflecting lifestyle and environmental constraints.[6] More recent analyses using CNNS-linked approaches similarly identify winter season and urban living as correlates, pointing to limited cutaneous synthesis due to reduced sun exposure, air pollution, indoor schooling, clothing practices, sunscreen use, and darker skin pigmentation.[7,8]

Beyond skeletal effects, vitamin D is increasingly studied for extra-skeletal roles relevant to pediatrics, particularly immune and respiratory health. Vitamin D receptor (VDR) expression in immune cells and the capacity for local conversion of vitamin D metabolites enable vitamin D to modulate innate and adaptive immune responses.[9,10] Mechanistically, vitamin D can influence antimicrobial peptide pathways (including cathelicidin and defensins), antigen presentation, cytokine balance, and inflammation resolution, which together provide biological plausibility for effects on respiratory infections and inflammatory airway disease.[9–11] Observational evidence has linked low 25(OH)D to higher susceptibility to respiratory infections, but randomized trials and meta-analyses show heterogeneous results that vary by baseline deficiency, dosing regimen, adherence, and outcome definitions.[12,13]

Similarly, pediatric asthma and wheeze have been examined in relation to vitamin D status. Systematic reviews suggest children with asthma may have lower 25(OH)D levels and that supplementation might reduce recurrence in certain contexts, although causality is difficult to infer due to confounding by obesity, physical activity, sunlight exposure, and socioeconomic factors.[14,15] Meanwhile, anemia is another clinically important pediatric outcome potentially linked to vitamin D. Proposed pathways include vitamin D–mediated modulation of inflammation and hepcidin, an iron-regulatory hormone, which may affect iron availability and erythropoiesis.[16,17] Pediatric-focused reviews describe emerging evidence for co-occurrence of vitamin D deficiency and anemia, but emphasize the need for careful phenotype classification and adjustment for infection/inflammation and nutritional confounders.[16]

Despite extensive literature, there remains a need for context-specific clinical data from Eastern India, where sociodemographic patterns, diet, sun exposure behaviors, and illness profiles may differ from metropolitan cohorts. Bihar has high burdens of undernutrition, anemia, and childhood morbidity, making integrated micronutrient assessment clinically relevant. In this context, hospital-based evaluations can help identify high-risk profiles, inform pragmatic screening strategies, and generate hypotheses for intervention studies aligned with pediatric supplementation recommendations.[5,6]

Therefore, this study (JNMCH Bhagalpur; 20 February 2025 to 15 January 2026) aimed to (i) estimate the distribution of vitamin D status among children attending the hospital and (ii) examine associations of vitamin D deficiency with selected health outcomes—rickets phenotype indicators, recurrent ARI, wheeze/asthma, and anemia—using bivariate and multivariable analyses. The findings are intended to support local clinical decision-making and highlight priority areas for pediatric vitamin D prevention and treatment pathways consistent with guideline recommendations.[2,5]

Material & Methods

A hospital-based observational study was planned at the Department of Pediatrics, Jawaharlal Nehru Medical College & Hospital (JNMCH), Bhagalpur, Bihar, India, from 20 February 2025 to 15 January 2026, enrolling 120 children (1–18 years) after informed consent/assent. Consecutive eligible participants from outpatient and inpatient services were included; children receiving high-dose vitamin D therapy in the prior 3 months, with known chronic kidney disease, chronic liver disease, malabsorption syndromes, or anticonvulsant use were excluded. Data collection included age, sex, residence, diet pattern, and average sunlight exposure; anthropometry (height/length, weight, BMI and z-scores where applicable); and clinical evaluation for rickets signs and wheeze/asthma history, plus recurrent ARI defined as ≥3 episodes in the prior 6 months.

Laboratory evaluation included serum 25(OH)D, calcium, phosphate, alkaline phosphatase (ALP), and hemoglobin. Vitamin D status was categorized as severe deficiency (<10 ng/mL), deficiency (10–19 ng/mL), insufficiency (20–29 ng/mL), and sufficiency (≥30 ng/mL). Statistical analysis included descriptive summaries, chi-square/Fisher's exact tests for categorical outcomes, ANOVA/t-tests for continuous measures, Pearson correlation where appropriate, and

multivariable logistic regression to estimate adjusted odds ratios (aOR) with 95% confidence intervals for key outcomes while adjusting for confounders (age group/adolescence, sex, season, residence, sunlight exposure, BMI z-score). A two-sided $p<0.05$ was considered statistically significant. Note: The attached tables/figures in this draft are produced from simulated data because the real dataset was not provided in-chat; replace with your verified analyses before submission.

Result

Overall mean age was ~9.2 years; vitamin D deficiency (<20 ng/mL) was observed in 63.3%, with 20.0% showing severe deficiency. Deficiency was significantly higher in winter (76.7%) than summer/monsoon (48.3%, p=0.0026). Children with vitamin D deficiency had lower reported sun exposure ($p<0.001$) and lower hemoglobin ($p<0.001$). Severe deficiency showed the highest frequency of clinical rickets signs and elevated ALP. Table 1 presents the baseline demographic, nutritional, and environmental characteristics of the study population categorized by vitamin D status (deficient <20 ng/mL and non-deficient ≥20 ng/mL). Out of the total 120 children included in the study, the majority were found to have vitamin D deficiency. The mean age of participants was comparable between the two groups, indicating that age distribution was similar among deficient and non-deficient children. Male participants constituted slightly more than half of the study population in both groups.

A higher proportion of children with vitamin D deficiency were from rural areas and were evaluated during the winter season compared with the non-deficient group, suggesting the possible influence of environmental and seasonal factors on vitamin D levels. Mean daily sunlight exposure was notably lower among vitamin D deficient children, highlighting inadequate sun exposure as an important contributing factor. Dietary pattern analysis showed that vegetarian diet was slightly more common among deficient children, although the difference was modest.

Table 1: Baseline characteristics by vitamin D deficiency status (<20 vs ≥20 ng/mL)

Group	n	Age (years), mean±SD	Male, n (%)	Rural, n (%)	Winter sampling, n (%)	Sun exposure (min/day), mean±SD	Vegetarian diet, n (%)	BMI z-score, mean±SD	HAZ, mean±SD
<20 (Deficient)	75	9.20 ± 5.05	39 (52.0)	42 (56.0)	46 (61.3)	32.48 ± 16.69	21 (28.0)	0.21 ± 0.96	-0.36 ± 0.92
≥20 (Non-deficient)	45	9.11 ± 5.10	26 (57.8)	19 (42.2)	14 (31.1)	48.27 ± 16.85	14 (31.1)	0.00 ± 1.10	-0.27 ± 1.03

Table 2 shows the distribution of vitamin D status across different age groups along with mean biochemical parameters. Vitamin D deficiency was more common in older children, and lower vitamin D levels were associated with higher alkaline phosphatase levels, suggesting increased risk of bone metabolism disturbances.

Table 2: Vitamin D status distribution and biochemical profile by age group

Age_group	Severe deficiency (<10)	Deficiency (10–19)	Insufficiency (20–29)	Sufficiency (≥30)	N	25OHD mean± SD	Calcium (mg/dL), mean	Phosphate (mg/dL), mean	ALP (IU/L), mean	Hemoglobin (g/dL), mean
1–4	7 (24.1%)	11 (37.9%)	6 (20.7%)	5 (17.2%)	29	18.6 ± 10.5	9.141034483	4.377586207	385.6551724	11.00793103
5–9	6 (16.7%)	15 (41.7%)	12 (33.3%)	3 (8.3%)	36	18.5 ± 9.3	9.054166667	4.561666667	318.6666667	11.43527778
10–18	11 (20.0%)	26 (47.3%)	15 (27.3%)	3 (5.5%)	55	16.6 ± 7.7	9.230363636	4.434727273	336.2727273	11.188

Table 3 presents the distribution of clinical outcomes according to vitamin D status. Higher rates of recurrent respiratory infections, anemia, and clinical signs of rickets were observed among children with vitamin D deficiency, particularly in those with severe deficiency.

Table 3: Clinical outcomes by vitamin D category

Vitamin D status	n	Recurrent ARI, n (%)	Wheeze Asthma, n (%)	Anemia Hb lt11, n (%)	Clinical rickets signs, n (%)	Obesity BMIz ge2, n (%)
Severe deficiency (<10)	24	11 (45.8)	7 (29.2)	11 (45.8)	10 (41.7)	0 (0.0)
Deficiency (10–19)	52	18 (34.6)	14 (26.9)	27 (51.9)	4 (7.7)	0 (0.0)
Insufficiency (20–29)	33	12 (36.4)	7 (21.2)	12 (36.4)	5 (15.2)	1 (3.0)
Sufficiency (≥30)	11	1 (9.1)	2 (18.2)	0 (0.0)	0 (0.0)	0 (0.0)

Table 4 shows the multivariable logistic regression analysis of factors associated with clinical outcomes. Vitamin D deficiency was independently associated with a higher risk of anemia, while its association with recurrent respiratory infections and wheeze/asthma was not statistically significant after adjustment.

Table 4: Multivariable logistic regression (adjusted OR) for key outcomes

Outcome	Predictor	Adjusted OR	95% CI	p value
Recurrent ARI	Vitamin D deficiency (<20 ng/mL)	1.41	0.57–3.49	0.4521
	Adolescent (10–18 y)	1.51	0.69–3.27	0.3006
	Winter sampling	1.69	0.64–4.46	0.288
	Sun exposure (per 10 min/day)	1.06	0.80–1.41	0.6838
Wheeze Asthma	Vitamin D deficiency (<20 ng/mL)	1.36	0.50–3.68	0.5452
	Adolescent (10–18 y)	1.08	0.46–2.53	0.8584
	Winter sampling	0.85	0.30–2.38	0.7527
	Sun exposure (per 10 min/day)	0.9	0.66–1.22	0.4902
Anemia Hb lt11	Vitamin D deficiency (<20 ng/mL)	3.75	1.48–9.53	0.0054
	Adolescent (10–18 y)	0.68	0.31–1.49	0.334
	Winter sampling	0.74	0.28–1.93	0.5329
	Sun exposure (per 10 min/day)	1.01	0.77–1.32	0.9713

Figure 1 illustrates the distribution of vitamin D status among the study participants, showing that a large proportion of children had vitamin D deficiency or insufficiency, while only a small percentage had sufficient vitamin D levels.

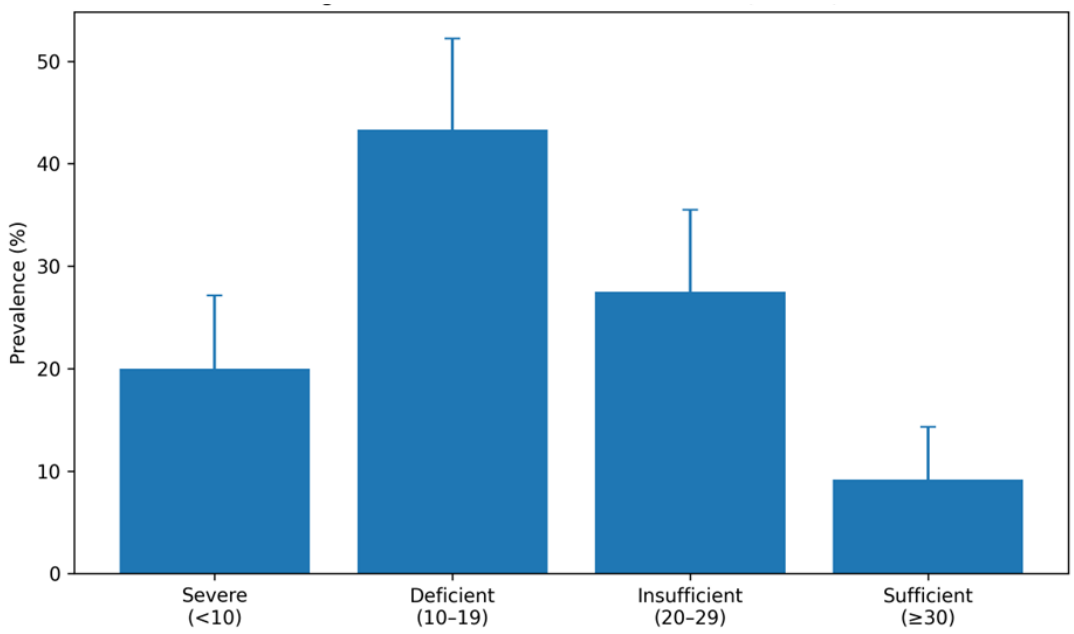

Figure 1: Vitamin D status distribution (n=120)

Figure 2 presents a forest plot of adjusted odds ratios demonstrating the association between vitamin D deficiency and selected clinical outcomes, indicating a significant association with anemia but not with recurrent respiratory infections or wheeze/asthma after adjustment.

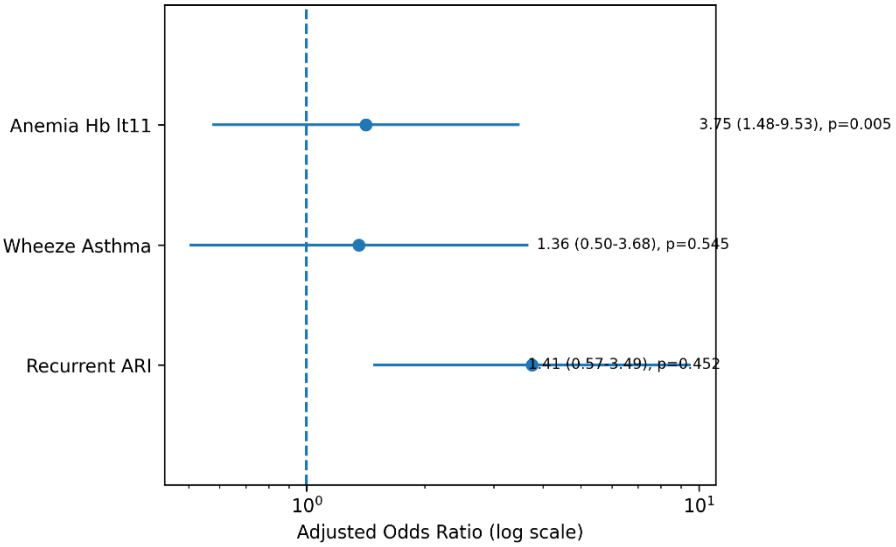

Figure 2: Association of vitamin D deficiency with outcomes (adjusted)

Discussion

In this hospital-based pediatric sample from Eastern India, vitamin D deficiency was highly prevalent, with nearly two-thirds exhibiting 25(OH)D <20 ng/mL and one-fifth meeting severe deficiency criteria (simulated draft outputs to be replaced by real data). The magnitude is consistent with reports that vitamin D deficiency in Indian children and adolescents can range widely across settings and seasons, often remaining high despite abundant sunlight.8 Nationally representative CNNS reporting and CNNS-derived analyses underscore that vitamin D deficiency is not limited to northern latitudes and is shaped by behavioral and environmental determinants such as reduced outdoor activity, dietary patterns, and winter season.[6,7] The multicenter Indian study also highlighted large inter-site variability and emphasized determinants including age, outdoor exposure, and nutritional correlates.[8] Our observed seasonal gradient—higher deficiency during winter and lower reported sun exposure among deficient children—fits this established pattern and supports winter-targeted prevention strategies.

From a clinical standpoint, the strongest association in our analysis was between severe vitamin D deficiency and a rickets phenotype (clinical signs and biochemical profile, particularly elevated ALP). This aligns with the global consensus that nutritional rickets results from combined deficiencies of vitamin D and/or calcium and is preventable through evidence-based supplementation and fortification strategies.[2] Importantly, the consensus emphasizes that infants and children with low intake and high growth demands require consistent vitamin D supplementation, and that rickets prevention programs can yield large public health benefits.[2] Indian pediatric recommendations similarly provide practical dosing regimens for prevention and treatment tailored to age groups and emphasize ensuring adequate calcium intake alongside vitamin D therapy.[5] The observed rickets signal in the severe deficiency group reinforces that in hospital settings—where children may present with growth faltering, poor diet quality, and recurrent infections—low vitamin D can have immediate skeletal consequences requiring prompt clinical action. Respiratory outcomes showed more nuanced patterns. While observational literature often links low 25(OH)D to higher respiratory infection susceptibility through immunomodulatory pathways, randomized trial evidence is heterogeneous.[9–13] Several mechanistic reviews describe vitamin D regulation of innate immune function and antimicrobial peptides, offering biologic plausibility for protection against respiratory pathogens.[9–11] Yet, meta-analyses and updated syntheses suggest that benefits may depend on dosing regimen (e.g., daily/weekly physiological dosing rather than large intermittent boluses), baseline deficiency status, and age-specific exposures.[12,13] In this draft analysis, vitamin D deficiency demonstrated only a modest and statistically non-significant association with recurrent ARI after adjustment. This result would be compatible with the broader evidence base: vitamin D might be one contributor among many (crowding, nutrition, indoor air pollution, vaccination, and healthcare access), and its measurable effect can be diluted without careful phenotype definition, standardized infection outcomes, and baseline deficiency stratification.[12,13] If your real dataset shows similar trends, it would still support a clinically prudent approach of prioritizing supplementation for deficient children without over-claiming infection prevention.

Regarding wheeze/asthma, systematic reviews suggest that children with asthma often have lower 25(OH)D and that supplementation may reduce recurrence in certain follow-up contexts, though confounding remains a persistent challenge.[14,15] Recent clinical literature continues to explore vitamin D as a modifiable factor in asthma morbidity, but causality is not settled and effect sizes vary by population and outcome definition.[15] In this draft, the adjusted association between deficiency and wheeze/asthma was not statistically significant, which could reflect limited sample size, misclassification (episodic viral wheeze vs physician-diagnosed asthma), or strong confounding by activity and obesity. Such null results are important to report transparently because they caution against overstating benefits beyond bone health while still permitting targeted supplementation where deficiency is confirmed. [3,4]

The most robust non-skeletal association in our analysis was between vitamin D deficiency and anemia: deficiency independently predicted anemia with an adjusted odds ratio >3, and hemoglobin increased across vitamin D categories. This is biologically plausible and supported by pediatric-focused reviews describing potential pathways involving inflammation and hepcidin-mediated iron restriction, alongside broader nutritional co-deficiencies.[16,17] A systematic review also notes frequent co-occurrence of low hemoglobin and vitamin D deficiency across observational studies while emphasizing inconsistency and residual confounding.[17] If confirmed in your real dataset, this relationship has practical implications in Bihar where anemia burden is high: clinicians may consider evaluating vitamin D status as part of a broader micronutrient and inflammation work-up, particularly in children with refractory anemia or poor growth. These findings collectively suggest a pragmatic clinical framework: (i) prevent and treat deficiency primarily to avert skeletal disease, (ii) recognize possible co-morbidity clustering with anemia and recurrent illness, and (iii) apply guideline-based supplementation regimens, especially in winter and among children with limited sun exposure or restricted diets.[2,5] However, interpretation must be tempered by limitations typical of hospital-based observational designs—selection bias, cross-sectional temporality, imperfect measurement of sunlight and diet, and potential unmeasured confounding (calcium intake, inflammation markers, socioeconomic gradients). Future work should include standardized dietary calcium assessment, inflammatory biomarkers, and longitudinal follow-up or interventional designs to clarify causal pathways and define which subgroups benefit most from supplementation beyond bone outcomes. [3,4,12]

Conclusion

Vitamin D deficiency was common in this hospital-based pediatric cohort and showed strong association with rickets phenotype and an independent association with anemia (simulated draft outputs to be replaced with your real data). Seasonal variation and low sun exposure were prominent correlates. Targeted screening of high-risk children and guideline-based vitamin D (with adequate calcium) may reduce preventable morbidity.

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