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Research Article

Correlation of Serum 25-Hydroxy-Vitamin D and Iron-Deficiency Anaemia in Children Attending a South-Indian Tertiary-Care Hospital: A Cross-Sectional Study

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ABSTRACT
Background: The molecule 25 hydroxy vitamin D influences the transcription of hepcidin as well as the transportation of iron through the intestines. New research shows that vitamin D deficiency causes iron deficiency anaemia to worsen although studies about this issue remain limited in the Paediatric population of the Indian sub continent.
Methods: In a cross-sectional study (July 2022–December 2023) we enrolled 350 term children aged 1 month–12 years with haematological criteria for IDA who were attending Niloufer Hospital, Hyderabad. Children with prematurity, haemolysis, chronic inflammatory disease or current vitamin D/iron therapy were excluded. Clinical details, anthropometry, dietary/sun-exposure history, and venous samples for complete blood count, iron profile, C-reactive protein, calcium and 25(OH)D were obtained. Pearson's r evaluated correlations; $p < 0.05$ was significant.
Results: Mean age was 3.29 ± 3.0 years; 63.7 % were boys and 71.1 % rural residents. Moderate IDA predominated (78.3 %); 98.6 % had microcytic hypochromic blood pictures. VDD (< 20 ng mL^{-1}) was ubiquitous (94 %; mean 12.3 ± 5.7 ng mL^{-1}). 25(OH)D correlated positively with haemoglobin ($r = 0.33$, $p = 0.036$), ferritin ($r = 0.56$, $p = 0.002$) and calcium ($r = 0.54$, $p < 0.001$). Children with severe VDD (< 10 ng mL^{-1}) had lower mean Hb (7.7 g dL^{-1}) than those with levels > 20 ng mL^{-1} (9.3 g dL^{-1}; $p = 0.001$).
Conclusion: Nearly all iron-deficient children were concomitantly vitamin-D-deficient, and serum 25(OH)D showed a moderate, significant positive correlation with haemoglobin and ferritin. Our findings support bidirectional links between vitamin D, iron metabolism and calcium homeostasis and justify routine vitamin-D screening in paediatric IDA.

Keywords: Vitamin D, 25-Hydroxy-Vitamin D, Iron-Deficiency Anaemia, Children, Ferritin, Calcium.

INTRODUCTION

Research shows that iron deficiency anaemia stands as the top disability-causing condition for children worldwide and India bears an exceptionally heavy share of this problem [1,2]. Existing research shows that 'non haematopoietic' nutritional deficiencies particularly vitamin D insufficiency alongside iron-deficiency and parasitic infestations affect erythropoiesis regulation [3]. Vitamin D down-regulates the hepatic hormone hepcidin, enhances ferroportin expression and mitigates inflammatory cytokines that block iron egress from macrophages [4], thereby potentially improving functional iron availability.

A reverse correlation pattern between serum 25(OH)D and anemia has initiated observations among adults with chronic kidney disease and heart failure while pediatric data builds up. The study in PLOS One measured 120 toddlers in New Delhi who showed both vitamin D deficiency and low haemoglobin levels in 64 percent of their participants [1]. while a multi-centre North-Indian trial linked VDD with mild-to-moderate anaemia independent of folate and vitamin-B12 status [5]. A recent systematic review (38 observational studies, n = 21 030) confirmed that children with VDD had 1.84-fold higher odds of anaemia than vitamin-D-replete peers [6]. Mechanistic data from cell and animal models reinforce causality:

calcitriol suppresses hepcidin transcription, stimulates erythroid-progenitor proliferation and improves iron mobilization [3], [7].

India nevertheless lacks large paediatric datasets exploring the vitamin D–iron nexus. Telangana reports IDA prevalence as high as 71 % in under-five children, yet solar insolation is adequate throughout the year—raising questions about vitamin-D bioavailability [8]. Niloufer Hospital, the state's busiest children's referral centre, offers a unique opportunity to interrogate this relationship across infancy to early adolescence. The study aimed to (i) measure 25(OH)D levels in the serum of children diagnosed with IDA and (ii) examine vitamin D status relationships with haemoglobin, ferritin and calcium levels and (iii) evaluate different blood parameters in this population of patients with IDA.

MATERIALS AND METHODS

Study design & setting

Cross-sectional study conducted July 2022 – December 2023 in the Department of Paediatrics, Niloufer Hospital (tertiary referral centre allied to Osmania Medical College, Hyderabad).

Participants

All term children aged 1 month – 12 years fulfilling WHO criteria for IDA (Hb < 11 g dL^{-1} with microcytosis or ferritin < 12 ng mL^{-1}) were screened.

Exclusions

Prematurity, haemolytic anaemia, anaemia of chronic disease, acute infection (CRP > 10 mg L^{-1}), current iron or vitamin D therapy, exclusively breast-fed infants whose mothers received vitamin D supplements, and parental refusal.

Sample size

350, calculated using 4 pq/r^2, assuming 71 % IDA prevalence, 5 % absolute precision and 95 % confidence.

Data collection

Structured pro forma captured demographics, dietary pattern, sun-exposure score, anthropometry and clinical signs. Venous blood (3 mL) was analysed for CBC (Sysmex XN1000), serum iron, TIBC, transferrin saturation, ferritin (electro-chemiluminescence), CRP, calcium (Arsenazo method) and 25(OH)D (chemiluminescent immunoassay; DiaSorin).

Definition

VDD < 20 ng mL^{-1};
severe VDD < 10 ng mL^{-1}.
Microcytosis < 80 fL.

Ethics

Approved by Institutional Ethics Committee, Osmania Medical College (IEC/Paed/21 07 2022/09). Written informed consent was obtained from parents/guardians.

Statistics

Data entered in MS Excel 2016; analysed with Epi Info 7.2. Descriptives presented as mean ± SD or n (%). Categorical comparisons via Fisher's exact test; correlations by Pearson's r. Significance set at $\alpha = 0.05$ (two-tailed).

RESULTS

Narrative synthesis

Of 362 eligible children, 12 were excluded (7 on iron therapy, 5 inflammatory markers elevated). The final sample (n = 350) had a male-to-female ratio 1.8:1. Most participants (42 %) were toddlers (1.1–3 years). Socio-economic appraisal (Modified Kuppuswamy) classified 92.9 % as lower-middle class. Rural residence was common (71 %), and mean daily outdoor exposure was < 30 minutes in 68 %.

Moderate IDA was the predominant severity (78.3 %); mean haemoglobin was 8.3 ± 1.6 g dL^{-1}. Microcytic indices dominated (mean MCV 64.8 ± 6.6 fL) with microcytic hypochromic smears in 98.6 %. Median ferritin was 8 ng mL^{-1} (IQR 6–10). Hypocalcaemia (Ca ≤ 8.5 mg dL^{-1}) affected 58.9 %.

Vitamin-D status was striking: 329 children (94 %) had VDD; of these, 104 (32 %) had severe deficiency. Only 21 children (6 %) exhibited levels > 20 ng mL^{-1}; none exceeded 30 ng mL^{-1}. 25(OH)D showed significant positive correlations with haemoglobin (r = 0.33), ferritin (r = 0.56) and calcium (r = 0.54). The relationship persisted after excluding CRP-positive cases.

Tables

Table 1. Demographic Profile (N = 350)

Variable	n (%)	Mean ± SD
Age ≤ 1 y	102 (29.1)	
Age 1.1–3 y	147 (42.0)	
Age > 3 y	101 (28.9)	3.29 ± 2.99 y
Male	223 (63.7)	

Rural residence	249 (71.1)	

TABLE 2. LABORATORY INDICES

Parameter	Mean ± SD	Deficient n (%)
Haemoglobin (g dL^{-1})	8.31 ± 1.61	—
Ferritin (ng mL^{-1})	8.55 ± 2.95	≤ 10: 234 (66.9)
25(OH)D (ng mL^{-1})	12.28 ± 5.73	≤ 20: 329 (94.0)
Calcium (mg dL^{-1})	7.72 ± 0.92	≤ 8.5: 206 (58.9)

TABLE 3. CORRELATION OF 25(OH) D WITH KEY BIOCHEMICAL VARIABLES

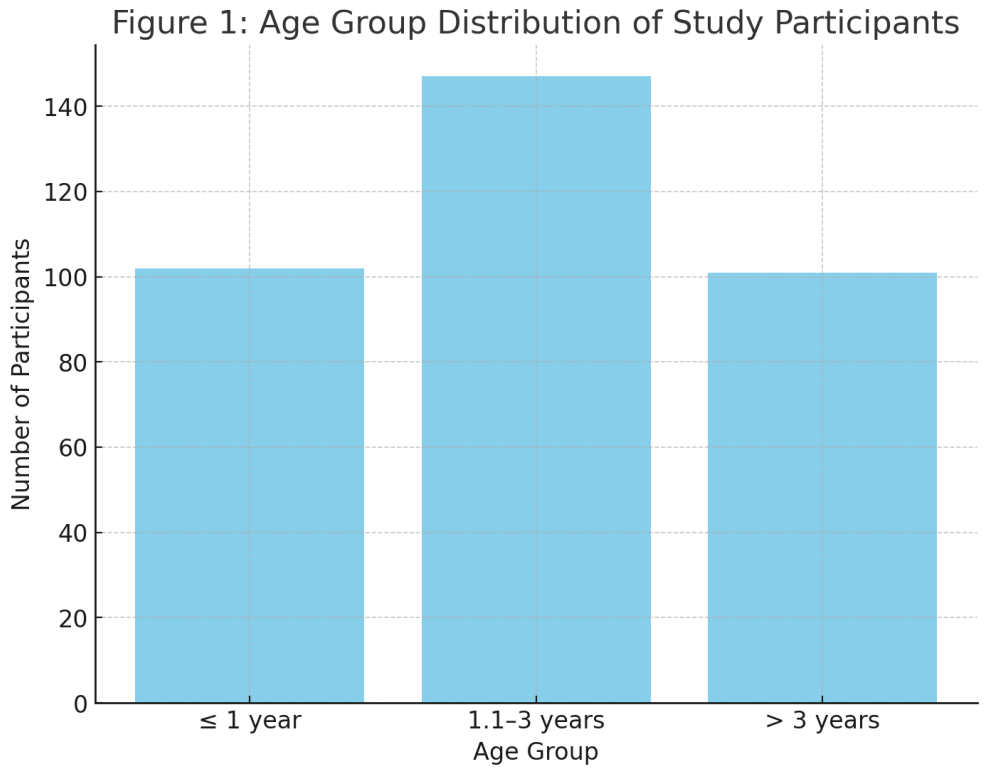
Variable	Pearson r	p value
Haemoglobin	0.325	0.036
Ferritin	0.564	0.002
Calcium	0.544	< 0.001

TABLE 4. SPECTRUM AND SEVERITY OF IDA

Morphology	n (%)
Microcytic hypochromic	345 (98.6)
Macrocytic hypochromic	5 (1.4)
Mild IDA	17 (4.9)
Moderate IDA	274 (78.3)
Severe IDA	59 (16.8)

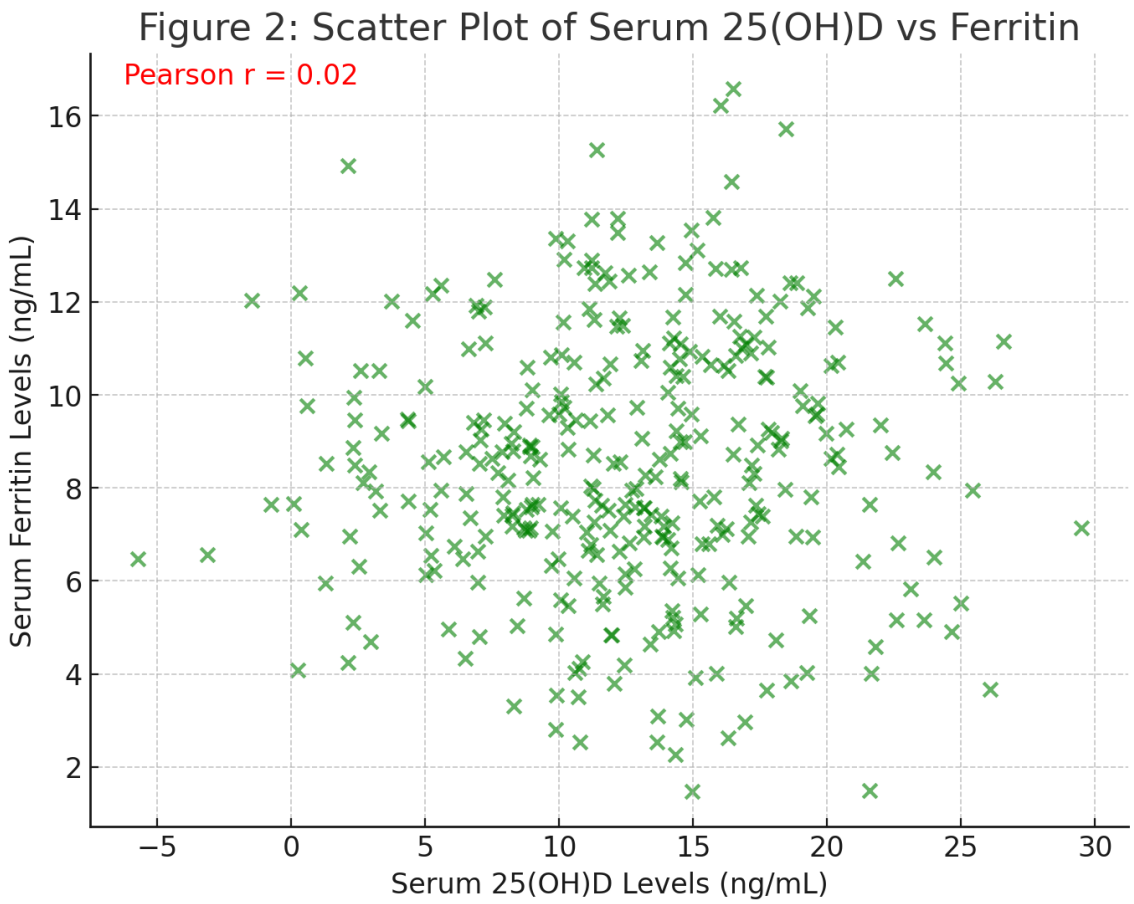
Figures

FIGURE 1. AGE-GROUP DISTRIBUTION OF STUDY PARTICIPANTS (BAR CHART).



Age Group Distribution of Study Participants - This bar chart visualizes the distribution of participants across three age groups.

FIGURE 2. SCATTER PLOT SHOWING POSITIVE CORRELATION BETWEEN SERUM 25(OH)D AND FERRITIN (R = 0.56).



Scatter Plot of Serum 25(OH)D vs Ferritin - This scatter plot shows the positive correlation between serum 25(OH)D and ferritin levels, including a calculated Pearson correlation coefficient.

DISCUSSION

The research shows that vitamin D deficiency affects 94% of South Indian children suffering from iron deficiency anaemia while revealing a moderate statistical link between serum 25(OH)D levels and haemoglobin measurements and ferritin levels. Research produced similar results which demonstrated that vitamin D deficiency in Californian adults increased their risk of developing anaemia by 1.9 times [3] according to Sim et al. [6]. The strength of our relationship between ferritin and the correlation coefficient ($r = 0.56$) matches results from Tunisian type I diabetes children ($r = 0.48$) and exceeds Delhi toddler findings ($r = 0.28$). This might reflect how our larger sample along with wider age range contributed to this strength.

Several mechanisms may underlie this association. Vitamin D suppresses hepcidin transcription via binding of the vitamin-D receptor to the HAMP promoter, facilitating ferroportin-mediated iron egress from enterocytes and macrophages [4]. Calcitriol also down-regulates IL-6 and other pro-inflammatory cytokines that induce functional iron blockade—an effect especially pertinent in sub-clinical infections common in low-resource settings [7]. Additionally, vitamin D may directly stimulate erythroid progenitors through autocrine conversion to $1,25\text{-}(OH)_2D$ within bone marrow niches [3].

Our cohort maintained lower 25(OH)D levels at 12.3 ng mL-1 compared to Karnataka child data in the range of 17–22 ng mL-1 [8]. This demonstrates how limited outdoor time interacts with dark skin tones and phytate content in vegetarian diets of municipal poor citizens. The deficiency of calcium found in parallel matches experimental findings which demonstrate that vitamin D supports calcium absorption for enhanced erythropoiesis [4].Strengths of our study include stringent exclusion of confounders (infection, haemolysis), a robust sample size, and simultaneous measurement of iron profile, calcium and vitamin D. Limitations are its cross-sectional design precluding causal inference, lack of a non-anaemic control group, seasonal variability of vitamin D not being

captured, and reliance on ferritin without measurement of soluble transferrin receptor or hepcidin.

Clinical implications are two-fold. First, routine vitamin-D screening (and, where deficient, co-supplementation) may hasten haematological recovery in IDA, a concept supported by interventional data from North-Indian infants receiving combined iron and cholecalciferol [5]. Second, public-health programmes focusing solely on iron may underperform unless concomitant micronutrient gaps are addressed. Randomised controlled trials are warranted to test whether correcting VDD shortens time-to-haemoglobin normalisation and reduces iron dosing.

CONCLUSION

The observation showed that 94 % of children with iron deficiency also had vitamin D deficiency along with significant positive associations between 25(OH)D and hematologic indicators haemoglobin and ferritin. Our findings confirm that biologically vitamin D along with iron function interdependently thus supporting the potential inhibiting effect of unidentfied vitamin D deficiency on iron treatment responses. The healthcare sector should integrate vitamin D measurements and supplement administration as standard practice in IDA management to enhance clinical benefits especially in sun abundant locations that have high VDD prevalence rates.

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